SECOND EDITION

Avian Medicine and Surgery in Practice

Companion and Aviary Birds



Bob Doneley

CRC Press
Taylor & Francis Group

Downloaded From Vet-eBooks.com

Avian Medicine and Surgery in Practice

Companion and Aviary Birds

SECOND EDITION

BOB DONELEY, BVSc, FANZCVS (Avian Medicine), CMAVA

Registered Specialist in Bird Medicine
Head of Service, Small Animal Hospital
School of Veterinary Science
University of Queensland, Australia

CRC Press
Taylor & Francis Group
Boca Raton London New York

CRC Press is an imprint of the
Taylor & Francis Group, an **informa** business

Downloaded From Vet-eBooks.com

CRC Press
Taylor & Francis Group
6000 Broken Sound Parkway NW, Suite 300
Boca Raton, FL 33487-2742

© 2016 by Taylor & Francis Group, LLC
CRC Press is an imprint of Taylor & Francis Group, an Informa business

No claim to original U.S. Government works
Version Date: 20160219

International Standard Book Number-13: 978-1-4822-6019-9 (eBook - PDF)

This book contains information obtained from authentic and highly regarded sources. While all reasonable efforts have been made to publish reliable data and information, neither the author[s] nor the publisher can accept any legal responsibility or liability for any errors or omissions that may be made. The publishers wish to make clear that any views or opinions expressed in this book by individual editors, authors or contributors are personal to them and do not necessarily reflect the views/opinions of the publishers. The information or guidance contained in this book is intended for use by medical, scientific or health-care professionals and is provided strictly as a supplement to the medical or other professional's own judgement, their knowledge of the patient's medical history, relevant manufacturer's instructions and the appropriate best practice guidelines. Because of the rapid advances in medical science, any information or advice on dosages, procedures or diagnoses should be independently verified. The reader is strongly urged to consult the relevant national drug formulary and the drug companies' and device or material manufacturers' printed instructions, and their websites, before administering or utilizing any of the drugs, devices or materials mentioned in this book. This book does not indicate whether a particular treatment is appropriate or suitable for a particular individual. Ultimately it is the sole responsibility of the medical professional to make his or her own professional judgements, so as to advise and treat patients appropriately. The authors and publishers have also attempted to trace the copyright holders of all material reproduced in this publication and apologize to copyright holders if permission to publish in this form has not been obtained. If any copyright material has not been acknowledged please write and let us know so we may rectify in any future reprint.

Except as permitted under U.S. Copyright Law, no part of this book may be reprinted, reproduced, transmitted, or utilized in any form by any electronic, mechanical, or other means, now known or hereafter invented, including photocopying, microfilming, and recording, or in any information storage or retrieval system, without written permission from the publishers.

For permission to photocopy or use material electronically from this work, please access www.copyright.com (http://www.copyright.com/) or contact the Copyright Clearance Center, Inc. (CCC), 222 Rosewood Drive, Danvers, MA 01923, 978-750-8400. CCC is a not-for-profit organization that provides licenses and registration for a variety of users. For organizations that have been granted a photocopy license by the CCC, a separate system of payment has been arranged.

Trademark Notice: Product or corporate names may be trademarks or registered trademarks, and are used only for identification and explanation without intent to infringe.

Visit the Taylor & Francis Web site at
http://www.taylorandfrancis.com

and the CRC Press Web site at
http://www.crcpress.com

Downloaded From Yet-eBooks.com

CONTENTS

Preface xxiii
Abbreviations xxv
US and international unit (blood values) conversion xxvii

CHAPTER **1** **CLINICAL ANATOMY AND PHYSIOLOGY** **1**

INTRODUCTION 1
THE SKIN, FEATHERS, NAILS AND BEAK 1
THE SKELETON 4
- General 4
- Skull 5
- Vertebrae 6
- Ribs 6
- Sternum 6
- Pectoral girdle 6
- Wings 6
- Pelvic girdle 7
- Legs 7

THE DIGESTIVE TRACT 7
- Oropharynx 7
- Oesophagus and crop 9
- Proventriculus and ventriculus 10
- Intestinal tract 10
- Pancreas 11
- Liver 11
- Cloaca 12

THE URINARY SYSTEM 13
THE RESPIRATORY TRACT 15
- Upper respiratory tract 15
- Lower respiratory tract 16

THE REPRODUCTIVE TRACT 20
- Female reproductive tract 20
- Egg 21
- Male reproductive tract 22
- Reproductive physiology 22

Downloaded From Vet-eBooks.com

CARDIOVASCULAR SYSTEM 24
General 24
Function 25
Blood cells 26
NERVOUS SYSTEM 29
The brain 29
Cranial nerves 30
The spinal cord 31
Spinal nerves 32
Autonomic nervous system 32
ENDOCRINE GLANDS 33
Pituitary 33
Thyroid 34
Parathyroid gland 34
Ultimobranchial glands 35
Adrenal glands 35
Pancreas 36
ORGANS OF THE SPECIAL SENSES 38
Eye 38
Ear 40
Chemical senses 41
THE IMMUNE SYSTEM 42
FURTHER READING 43

CHAPTER **2** **HUSBANDRY, GROOMING AND NUTRITION** **45**

HUSBANDRY 45
Pet birds 45
Aviary birds 48
Grooming birds 54
Nutrition 56
Lorikeets 60
FURTHER READING 61

CHAPTER **3** **THE PHYSICAL EXAMINATION** **63**

UNDERSTANDING THE MASKING PHENOMENON 63
EXAMINATION ROOM EQUIPMENT 64
HISTORY TAKING 64
Signalment 64
Origin of the bird 65
Husbandry 65
Nutrition 66
Behaviour 67
Reproductive history 68

Downloaded From Vet-eBooks.com

Previous medical history 68
Presenting problem 68
THE DISTANT EXAMINATION 68
The bird 68
The cage 69
Faecal examination 69
THE PHYSICAL EXAMINATION 71
Handling and restraint 71
Weight recording 72
Auscultation 73
Body condition 73
Skin and plumage 73
Head 75
Crop 76
Body 76
Wings 77
Legs 77
Neurological assessment 78
FURTHER READING 79

CHAPTER **4** **CLINICAL TECHNIQUES** **81**

DIAGNOSTIC TECHNIQUES 81
Blood collection and handling 81
Microbiology 83
Cytology 84
Parasitology 84
Diagnostic imaging 85
Endoscopy 85
Cardiology 85
TREATMENT TECHNIQUES 85
Heating 86
Fluid therapy 86
Nutritional support 89
Respiratory support 90
Administering medications 91
Immobilising limb fractures using external coaptation 92
FURTHER READING 94

CHAPTER **5** **DIAGNOSTIC IMAGING** **95**

RADIOLOGY 95
Equipment 95
Contrast 96
Restraint and positioning 96

Downloaded From Vet-eBooks.com

Normal anatomy 97
Radiographic abnormalities 99
ULTRASOUND 104
COMPUTERISED TOMOGRAPHY (CT) 105
FLUOROSCOPY 106
MAGNETIC RESONANCE IMAGING (MRI) 107
FURTHER READING 107

CHAPTER **6** **ENDOSCOPY** **109**

EQUIPMENT 109
CARING FOR THE EQUIPMENT 112
Handling 112
Cleaning 112
Sterilisation/disinfection 113
OPERATING THE ENDOSCOPE 113
BIOPSY COLLECTION 113
ENDOSCOPIC APPROACHES 113
Tracheal approach 113
Coelomic approach 114
Ventral coelomic approach 115
Choanal approach 116
Cloacal approach 116
Approach to the upper gastro-intestinal tract 116
FURTHER READING 116

CHAPTER **7** **INTERPRETING DIAGNOSTIC TESTS** **117**

CHOOSING WHICH TESTS TO USE 117
HAEMATOLOGY 118
Erythrocytes 118
Leucocytes 120
Thrombocytes 121
Assessing blood coagulation 122
PROTEIN ELECTROPHORESIS 122
Submission of samples for Protein Electrophoresis 123
Interpretation of results 123
CLINICAL BIOCHEMISTRY 124
Normal variation between species and individuals 124
Artefacts 125
Biochemical analysis by organ system 126
LIPIDS 128
SEROLOGY 129
Using serology 129
Scenarios involving the use of serology 132

Downloaded From Vet-eBooks.com

PCR TESTING 133
CULTURES 133
CYTOLOGY 134
Classification of cell types 135
Classification of cellular responses 135
Cytology of commonly sampled fluids and tissues 136
Cytology of the alimentary tract 137
Cytology of the respiratory tract 139
Cytology of internal organs 141
FURTHER READING 144

CHAPTER **8** **SUPPORTIVE THERAPY** **145**

DEHYDRATION 145
Clinical presentation 145
Management 145
HYPOTHERMIC SHOCK 145
Clinical presentation 145
Management 145
CATABOLISM 146
Clinical presentation 146
Management 146
RESPIRATORY COMPROMISE 146
Clinical presentation 146
Management 146
ANALGESIA 146
Clinical signs of pain 146
Management 147
BLOOD LOSS 147
Clinical presentation 147
Management 147
HOSPITAL CARE 148
Security 148
Warmth 148
Biosecurity 149
Feeding 149
Psychological care 149
FURTHER READING 149

CHAPTER **9** **DIFFERENTIAL DIAGNOSES** **151**

CHANGE IN DROPPINGS 151
Diarrhoea 151
Change in colour of the faecal portion 151
Enlarged faecal portion 151

Downloaded From Vet-eBooks.com

Frank blood in the droppings 151
Whole seed in the droppings 152
Change in the colour of the urates 152
Malodorous droppings 152
Polyuria 152
APPETITE AND THIRST 152
Increased appetite 152
Decreased appetite 152
Polydypsia 152
Decreased thirst 152
VOMITING 152
WEIGHT LOSS 153
POSTURE 153
Fluffed, immobile, eyes closed, both legs on perch 153
Fluffed, immobile, eyes closed, head tucked under one wing, only one leg on perch 153
Tail pointing down, perpendicular to cage floor, while wings remain at a normal angle 153
Tail bobbing up and down in an exaggerated movement 153
Head held down but bird is looking up, wings spread out, tail spread 153
Wing droop 154
Sitting on the floor of the cage, body upright, perhaps panting 154
Neurological signs (ataxia; fitting; paralysis/paresis; tremor) 154
Bilateral leg paresis/paralysis 154
Unilateral leg paresis/paralysis 154
One wing held out to the side, resting on the floor or perch 154
FEATHERS AND SKIN 154
Generalised feather loss 154
Feathering gradually darkening or becoming 'greasy-looking' 154
Broken primary feathers on wings and/or tail 154
'Stress lines' (horizontal breaks in the feather vane) 155
Long straw-like feathers over the thighs or other feathers still encased in keratin sheaths 155
Abnormally coloured feathers 155
Ragged looking plumage without pruritus 155
Continued growth of flight, tail and contour feathers in budgerigars unable to fly 155
Pruritis 155
Feathers missing on the head 156
Greenish discoloration of skin 156
Flaky, dry skin 156
Feather cysts 156
Polyfolliculosis 156
Enlarged uropygial (preen) gland 156

Downloaded From Vet-eBooks.com

Yellow subcutaneous deposits 156
Self-mutilation of African lovebirds (*agapornis* spp.) 156
Feather damaging behaviour 156
WINGS 156
Blood on feathers 156
Wing drooping 157
Swellings 157
Wings held away from the body 157
Green discoloration 157
FEET AND LEGS 157
Limping 157
Swollen joints 157
Missing nails or toes 157
Overgrown nails 157
Abnormal shape or direction of the legs 157
Hyperkeratosis of the scaled part of the leg 157
Self-mutilation of foot and toes 158
BEAK 158
Overgrown beak 158
Beak twisted to the left or right 158
Upper beak inside the lower beak 158
Inability to close beak properly 158
Flakes of keratin on the beak 158
White, crusty, honeycombed lesions on the beak 158
Nares unequal in size 158
Thickening and hypertrophy of cere of budgerigar hens 158
Nares blocked or staining/matting of feathers above nares 158
EYES 158
Feather loss around eyes 158
Eyelid abnormalities 159
Thickening and hyperaemia of the conjunctiva 159
Exophthalmos 159
Enophthalmos 159
Corneal changes 159
Hyphema 159
Cataracts 159
FACE 159
Swellings on the face 159
Matting/staining of feathers below and caudal to eye 159
Matting of the feathers over face and head 160
Small nodules on the facial skin of macaws 160
BODY 160
Prominent keel bone (‘going light’) 160
Twisted keel bone 160

Downloaded From Vet-eBooks.com

Split keel bone (non-traumatic) 160
Ulcerative lesion on the cranial end of the keel 160
Overabundance of pectoral muscle mass (i.e. 'cleavage') along keel 160
Enlarged coelom 160
Subcutaneous emphysema 160
Split in skin between vent and tail 160
Subcutaneous masses 160
PAEDIATRICS 161
Crop not emptying, or slow to empty 161
Cervical emphysema 161
Erythematous skin 161
Pallor 161
Overly large head 161
Bruising on the skin 161
Feathers not growing normally 161
Swollen toes 161
Thin toes 161
Vomiting 161
Refusing to eat 161
Reddened skin or scab over the crop 161
FURTHER READING 161

CHAPTER **10** **DISEASES OF THE SKIN AND FEATHERS** **163**

CONGENITAL DISORDERS 163
'Feather duster' or 'chrysanthemum' syndrome in budgerigars 163
'Straw feather' in canaries 163
Feather cysts in canaries 163
'Porcupine feathers' in homer and fantail pigeons 164
Baldness in lutino cockatiels 164
NUTRITIONAL DISORDERS 164
Feather quality 164
Feather colour 164
Skin changes 164
ENDOCRINE DISORDERS 165
Hypothyroidism 165
Delayed moulting 166
Trauma 166
BACTERIAL INFECTIONS 167
FUNGAL INFECTIONS 168
VIRAL INFECTIONS 169
Psittacine beak and feather disease 169
Avian Polyomavirus 171
Avian poxvirus 171
Papillomavirus 172

Downloaded From Vet-eBooks.com

PARASITIC INFECTIONS 172
Mites 172
Lice 173
Fleas 173
Flies 174
Ticks 174
Management 174
NEOPLASTIC AND PSEUDONEOPLASTIC CONDITIONS 174
Lipomas 174
Neoplasia of the uropygial gland 175
Other neoplasms 175
Xanthomas 176
TOXIC CONDITIONS 176
CONDITIONS AFFECTING THE UROPYGIAL GLAND 176
Impaction 176
Infection and abscesses 176
Neoplasia 176
IATROGENIC TRAUMA 176
FEATHER DAMAGING BEHAVIOUR AND OTHER SELF-MUTILATING CONDITIONS 177
Polyfolliculosis/polyfolliculitis 177
Self-mutilation in *Agapornis* species 178
Cockatiel feather mutilation syndrome 178
Quaker mutilation syndrome 178
Feather damaging behaviour directed towards other birds 179
Feather damaging behaviour directed towards the bird itself 180
FURTHER READING 183

CHAPTER **11** **DISORDERS OF THE BEAK AND CERE** **187**

MALFORMATION 187
TRAUMA 189
HYPEREXTENSION OF THE MAXILLA 191
OVERGROWN MAXILLA 192
CNEMIDOCOPTES 192
INFECTION 193
Bacterial and fungal infections 193
Viral infections 194
NEOPLASIA 194
KERATIN FLAKES ON THE BEAK 194
DISORDERS OF THE CERE 195
Hypertrophy 195
Colour change 195
Distortion 195
FURTHER READING 196

Downloaded From Vet-eBooks.com

CHAPTER **12** **DISORDERS OF THE EYE** **197**

ASSESSING THE EYE 197
Distant examination 197
Physical examination 197
DISORDERS OF THE EYE 199
Lids and periorbital region 199
Globe and orbit 200
Conjunctiva 201
Cornea 201
UVEA 202
Acute uveitis 202
Chronic uveitis 202
LENS 202
Cataracts 202
Retina 203
FURTHER READING 203

CHAPTER **13** **DISORDERS OF THE EAR** **205**

OTITIS EXTERNA 205
OTITIS MEDIA AND OTITIS INTERNA 206
FURTHER READING 206

CHAPTER **14** **DISEASES OF THE LEGS, FEET AND TOES** **207**

MALFORMATIONS 207
Introduction 207
Coxofemoral subluxation 208
ANGULAR LIMB DEFORMITIES 208
Leg rotation 208
Bowed legs 209
PATHOLOGICAL FRACTURES 209
TOE ANTEROFLEXION 210
SLIPPED TENDON (PEROSIS) 210
SWOLLEN JOINTS 210
Articular gout 210
Luxation of the joint 211
Arthritis 211
PODODERMATITIS 211
CONSTRICTED TOE SYNDROME 213
BILATERAL PARESIS OR PARALYSIS 214
Spinal trauma or neoplasia 214
Obturator paralysis 214
Lorikeet paralysis syndrome 214

Downloaded From Vet-eBooks.com

Barraband (or *Polytelis*) paralysis syndrome 215
Bilateral leg trauma 215
Lead toxicosis 215
UNILATERAL PARESIS OR PARALYSIS 215
Unilateral trauma 215
Sciatic nerve compression 215
Spinal neoplasia 215
HYPERKERATOSIS 215
SELF-MUTILATION OF THE FEET AND TOES 216
TOE TAPPING IN ECLECTUS PARROTS 216
LEG BAND CONSTRICTION 217
TOE NECROSIS 217
FROST BITE 218
OVERGROWN NAILS 219
MISSING NAILS AND TOES 219
FURTHER READING 219

CHAPTER **15** **DISORDERS OF THE MUSCULOSKELETAL SYSTEM** **221**

SKELETAL DISORDERS 221
HEREDITARY, CONGENITAL AND DEVELOPMENTAL 221
Spinal bifida 221
Kyphosis 221
Scoliosis 221
NUTRITIONAL/METABOLIC 221
Osteomalacia 221
Nutritional secondary hyperparathyroidism 221
Osteopetrosis or polyostotic hyperostosis 221
Osteomyelitis 223
Neoplasia 223
JOINTS 224
Luxation 224
Swollen joints 225
MUSCLE DISORDERS 225
CONGENITAL 225
Muscular dystrophy 225
Arthrogryposis 225
NON-INFLAMMATORY 225
Muscle atrophy 225
Vitamin E and selenium deficiency 225
Ionophore toxicosis (monensin, lasalocid, salinomycin, narasin) 225
INFLAMMATORY: NON-INFECTIOUS 225
Trauma 225
Exertional/capture myopathy 226

Downloaded From Vet-eBooks.com

INFLAMMATORY: INFECTIOUS 226
Viral 226
Bacterial 226
Fungal 226
Parasites 226
Sarcocystis 226
Leukocytozoon 227
NEOPLASTIC 227
TENDONS AND LIGAMENTS 227
Tendon contracture ('joint disease') 227
Slipped tendon (perosis) 227
Tendonitis 227
FURTHER READING 228

CHAPTER **16** **DISEASES OF THE GASTROINTESTINAL TRACT** **229**

THE OROPHARYNX AND CROP 229
Candidiasis 229
Trichomoniasis 230
Hypovitaminosis A 231
Poxvirus infection 231
Internal papilloma disease 231
Crop stasis 231
Thermal injuries 232
Crop perforations 232
Ingluvoliths and other foreign bodies in the crop 233
PARASITES 233
Capillaria contorta (hair worm, thread worm) 233
Spiruroid worms (Spiruroidea) 233
PROVENTRICULUS AND VENTRICULUS 233
FUNGAL INFECTIONS 233
Macrorhabdus 233
Viral infections 235
Parasitic infections 238
Foreign bodies 238
Neoplasia 239
DISORDERS OF THE INTESTINAL TRACT 239
Ileus 239
Parasites 239
Capillaria (hair worm, thread worm) 240
Heterakis (caecal worm) 241
Cestodes (tapeworms) 241
Coccidia 241
Cryptosporidia 242
Giardia 242

Downloaded From Vet-eBooks.com

Cochlosoma 242
Hexamita (*Spironucleus* spp.) 242
Histomonas meleagridis 243
Microsporidiosis (encephalitozoonosis) 243
Mycobacteria 243
Bacterial enteritis 245
Escherichia coli 245
Clostridium spp. 245
Salmonella spp. 245
DISORDERS OF THE CLOACA 246
Prolapse 246
Internal papilloma disease (IPD) 247
Cloacoliths 249
Cloacal atony 249
Cloacitis 249
Neoplasia 249
FURTHER READING 250

CHAPTER **17** **DISORDERS OF THE LIVER** **251**

OVERVIEW OF LIVER DISEASE 251
AETIOLOGY OF LIVER DISEASE 251
Congenital 251
Trauma 251
Metabolic/nutritional 251
Toxic 252
Parasitic 252
Infectious 253
Neoplastic 255
Idiopathic 255
CLINICAL SIGNS 255
Cholestasis 255
Inadequate conversion of ammonia to uric acid and urea 255
Protein synthesis deficits 255
Abnormal carbohydrate and fat metabolism 256
Failure of Kupffer cell activity 256
Portal hypertension 256
Inadequate or inappropriate metabolism of drugs and chemicals 256
Other clinical signs 256
DIAGNOSIS 256
Clinical pathology 256
Diagnostic imaging 256
TREATMENT 257
Supportive care 257

Downloaded From Vet-eBooks.com

Treatment of the specific condition 257
Creation of an environment favourable to regeneration 258
CHLAMYDIOSIS 259
Clinical signs in man 262
PACHECO'S DISEASE 262
FURTHER READING 264

CHAPTER **18** **DISORDERS OF THE PANCREAS** **265**

EXOCRINE 265
Pancreatic insufficiency 265
Pancreatitis 266
Pancreatic neoplasia 267
ENDOCRINE 267
Diabetes mellitus 267
FURTHER READING 269

CHAPTER **19** **DISEASES OF THE RESPIRATORY SYSTEM** **271**

UPPER RESPIRATORY TRACT 271
Sinusitis 271
Rhinitis 272
Ruptured cervicocephalic air sac 272
Choanal atresia 273
LOWER RESPIRATORY TRACT 274
Tracheal obstruction 274
Tracheitis 277
Pulmonary parenchyma disease 278
Air sac disease 279
Aspergillosis 280
FURTHER READING 283

CHAPTER **20** **DISORDERS OF THE CARDIOVASCULAR SYSTEM** **285**

CARDIAC DISEASE 285
Overview 285
Congenital heart disease 285
Endocardial disease 285
Myocardial disease 285
Pericardial disease 286
Arrhythmias 286
Congestive heart failure 287
ATHEROSCLEROSIS 292
HYPERTENSION 295
FURTHER READING 295

Downloaded From Vet-eBooks.com

CHAPTER **21** **DISORDERS OF THE LYMPHATIC AND HAEMATOPOIETIC SYSTEMS** **297**

OVERVIEW 297
Thymic cysts 297
Premature thymic and/or bursal atrophy 297
Splenic atrophy 297
Splenomegaly 297
Lymphosarcoma 298
DISEASES OF THE BONE MARROW 299
PRIMARY DISORDERS OF THE IMMUNE SYSTEM 300
Immune suppression 300
IMMUNE-MEDIATED DISORDERS 300
Allergies 300
Immune-mediated haemolytic anaemia 300
Membranous glomerulonephropathy 301
Transfusion reactions 301
FURTHER READING 301

CHAPTER **22** **DISORDERS OF THE NERVOUS SYSTEM** **303**

INTRODUCTION 303
HISTORY 303
The bird 303
The problem 303
DISTANT EXAMINATION 303
Mental status 303
Posture 303
Flight 303
Gait 304
Other 304
PHYSICAL EXAMINATION 304
DIAGNOSTIC TESTS 304
CENTRAL NERVOUS SYSTEM DISORDERS 304
Congenital/hereditary conditions 304
VIRAL DISEASES 304
Paramyxovirus 1, 2, 3 and 5 304
Proventricular dilatation disease (Avian Bornavirus; PDD) 305
West Nile virus 305
Avian Polyomavirus 305
Herpesvirus 305
Adenovirus 305
Togavirus 305
Other viruses 306

Downloaded From Vet-eBooks.com

BACTERIAL INFECTIONS 306
FUNGAL DISEASES 306
CHLAMYDIA 306
PROTOZOAL DISEASES 306
Sarcocystis falcatula 306
Toxoplasma gondii 306
Leukocytozoon 306
NEMATODES 306
TRAUMA 307
CEREBROVASCULAR ACCIDENTS 307
TOXINS 307
Lead toxicosis 307
Botulism 309
Organophosphates and carbamates 309
Chlorinated hydrocarbon (DDT)/organochlorines 310
Dimetridazole 310
Levamisole 310
Nitrofurazone 310
NUTRITIONAL DEFICIENCIES 310
Vitamins 310
Calcium 311
NEOPLASIA 311
Pituitary adenomas 311
Primary brain tumours 311
EPILEPSY/SEIZURES 311
PERIPHERAL NERVOUS SYSTEM DISORDERS 313
Trauma 313
Renal neoplasia 313
Horner's syndrome 314
Obturator paralysis 314
Neuralgia 314
FURTHER READING 315

CHAPTER **23** **DISORDERS OF THE REPRODUCTIVE TRACT** **317**

THE MALE REPRODUCTIVE TRACT 317
Congenital 317
Non-inflammatory 317
Orchitis 317
Neoplasia 317
Phallic prolapse 318
Aggression 318
THE FEMALE REPRODUCTIVE TRACT 319
THE OVARY 319

Downloaded From Vet-eBooks.com

Congenital abnormalities 319
Oophoritis 319
Ovarian cysts 320
Neoplasia 320
THE OVIDUCT 321
Congenital abnormalities 321
Cystic hyperplasia 321
Salpingitis and metritis 321
Yolk-related peritonitis 323
Egg binding (dystocia) 323
Ectopic eggs 325
Retained eggs 326
Chronic or excessive egg laying 326
INVESTIGATING REPRODUCTION PROBLEMS IN THE AVIARY 327
Introduction 327
Lack of egg production 328
Infertile eggs 328
Embryonic death 329
FURTHER READING 331

CHAPTER **24** **DISORDERS OF THE URINARY SYSTEM** **333**

RENAL DISEASE 333
Mechanism of renal disease 333
Inflammatory conditions 333
Non-inflammatory conditions 334
UROLITHIASIS 338
GOUT 339
ZINC TOXICOSIS 340
FURTHER READING 341

CHAPTER **25** **BEHAVIOURAL PROBLEMS** **343**

INTRODUCTION 343
PRINCIPLES 343
UNDERSTANDING BIRD BEHAVIOUR 343
HOW DO BEHAVIOURAL PROBLEMS DEVELOP? 344
HISTORICAL APPROACH TO TREATING BEHAVIOURAL PROBLEMS 345
NEW CONCEPTS 346
Birds are not people, or dogs, or cats 346
The best results are obtained when a bird wants to do the behaviour that the owner desires 347
A successful treatment may be a reduction in an unwanted behaviour, rather than a complete cessation of it 347

Downloaded From Vet-eBooks.com

Successful behavioural change in companion parrots starts with behavioural change in the parrot owner 348
BASIC STEPS TO IMPLEMENT IN A BEHAVIOUR-MODIFICATION PROGRAMME 348
Basic training 348
Normalise social interactions 349
Avoid unwanted behaviours 349
Replacement of unwanted behaviours with acceptable behaviours 349
FURTHER READING 351

CHAPTER **26** **INCUBATION OF EGGS** **353**

WHY INCUBATE EGGS? 353
WHEN TO COLLECT EGGS 353
STORING EGGS PRIOR TO INCUBATION 353
SANITATION OF THE EGGS 353
EQUIPMENT 354
INCUBATION PARAMETERS 354
Temperature 354
Humidity 354
Ventilation 355
Turning and positioning 355
MONITORING 355
HYGIENE 355
RECORD KEEPING 356
EMBRYONIC DEVELOPMENT 356
Early term 356
Mid term 356
Late term 356
HATCHING 356
PROBLEMS WITH INCUBATION 357
EGG NECROPSY 357
FURTHER READING 358

CHAPTER **27** **PAEDIATRICS** **359**

INTRODUCTION 359
EXAMINATION OF THE CHICK 360
History 360
Physical examination 360
Diagnostic testing 362
COMMON PROBLEMS 362
Stunting 362
Crop stasis (‘sour crop’) 363
Thermal injuries to the crop 364
Crop perforations 364

Downloaded From Vet-eBooks.com

Orthopaedic problems 364
Beak malformations 364
Omphalitis 364
'Wry Neck' 365
Foreign Bodies 365
Infectious disease 365
Avian Polyomavirus (APV) 366
Adenovirus 368
Psittacine Beak and Feather Disease 368
FURTHER READING 369

CHAPTER **28** **ANALGESIA AND ANAESTHESIA** **371**

ANALGESIA 371
Signs and effects of pain 371
Principles of analgesia 371
Analgesics commonly used in avian practice 372
ANAESTHESIA 373
What makes avian anaesthesia different? 373
Anatomy 373
Metabolism 374
Heat loss 374
Chronically ill patients 374
Inhalation anaesthetic agents 374
Intravenous or intramuscular agents 375
Anaesthetic technique 376
Anaesthetic emergencies 381
FURTHER READING 382

CHAPTER **29** **SURGERY** **385**

INTRODUCTION 385
PRE-SURGICAL ASSESSMENT AND CONDITIONING 385
Physical examination 385
Diagnostics 385
Conditioning the patient 386
Minimising anaesthetic time 386
SURGICAL PREPARATION AND PATIENT SUPPORT PROCEDURES 386
Preparation of the surgical site 386
Minimising tissue trauma and blood loss 387
COMMON SOFT TISSUE SURGICAL PROCEDURES 389
Ingluviotomy 389
Crop fistula repair 390
Left lateral coeliotomy 390
Ventral midline coeliotomy 390

Downloaded From Vet-eBooks.com

Pancreatic biopsy 394
Proventriculotomy 394
Ventriculotomy 394
Coelomic hernia repair 395
Cloacopexy 395
Cloacoplasty/ventoplasty 396
Cloacotomy 396
Salpingohysterectomy 397
Orchidectomy 397
Enucleation 398
Repairing skin lacerations 398
ORTHOPAEDICS 399
General considerations 399
Bone healing 399
Principles of orthopaedic surgery 400
Types of fracture repair 400
Selection of means of fixation 403
Post-operative management and complications 406
Approaches to the bones of the wing 406
Approaches to the bones of the leg 412
Joint surgery 417
FURTHER READING 418

CHAPTER **30** **ONCOLOGY** **421**

INTRODUCTION 421
TUMOUR CLASSIFICATION 421
Diagnosis 421
Treatment options 422
FURTHER READING 424

Appendix 1: Formulary **425**
Appendix 2: Reference Intervals for Commonly Kept Companion Birds **447**
Appendix 3: Biological Values for Some Common Companion Bird Species **449**

Downloaded From Vet-eBooks.com

PREFACE

Well, a lot of things have happened since I wrote the first edition of *Avian Medicine and Surgery in Practice*. I completed my transition from private clinical practice to the role of clinical academic at The University of Queensland, my children have grown up and left home, but life seems to be just as busy. So it came as a surprise when Jill Northcott of CRC Publishing invited me to write a second edition of my book.

The first thing I did was go back and read the reviews written by my colleagues around the world. While these reviews had kind things to say, they did highlight some glaring omissions and errors in the first edition. How did I miss cardiovascular anatomy and neuroanatomy in the first chapter? So, to Alex Rosenwax, Michelle Barrows, Julia Whittington and Scott Echols I owe a great vote of thanks. The changes contained in this second edition are largely due to their astute observations and constructive comments.

Other thanks go to my avian medicine colleagues all around the world, all of whom contribute every day to the body of knowledge we call avian medicine and surgery. You generously share your wealth of knowledge and experience through discussion lists, conferences, and published papers. Without your efforts I could not have written this book and the field of avian medicine would be barren and lifeless.

I must also thank my colleagues at the UQ Veterinary Medical Centre. Working with world-class small animal surgeons, internists, anaesthetists, radiologists, general practitioners, and nursing staff has greatly broadened my understanding of veterinary medicine and encouraged me to re-examine my approach to understanding and caring for my patients. The pathology staff at the UQ School of Veterinary Science's Veterinary Laboratory Services have helped me to make sense of those cases I could not help, and deepened my understanding of pathogenesis and pathology. I am a far better veterinarian for having worked with all of you.

To Jill Northcott and the staff of CRC Publishing, thank you for your faith and trust in asking me to write a second edition. To say I was a little surprised and daunted by the task is an understatement. Having your hard drive crash when you have nearly finished is a little off-putting as well. But you never doubted I would come through, and I thank you for that.

I cannot conclude this foreword without acknowledging the constant support, love and encouragement I get every day from Maree, my wife of over 30 years, and Liz and Pat, our children. Words cannot say how I feel. As with anything I have done, I could not have written this book without you.

Bob Doneley
Associate Professor, Avian and Exotic Pet Medicine
School of Veterinary Science
The University of Queensland
Gatton, Queensland
Australia

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

ABBREVIATIONS

ACE	angiotensin converting enzyme
ACH	acetylcholine
ACTH	adrenocorticotrophic hormone
ADH	antidiuretic hormone
ALD	angular limb deformity
ALP	alkaline phosphatase
ALT	alanine aminotransferase
APP	avian pancreatic polypeptide
AST	aspartate aminotransferase
ATP	adenosine triphosphate
AV	atrioventricular
AVT	arginine vasotocin
BELISA	blocking enzyme-linked immunosorbent assay
bpm	beats per minute
BUN	blood urea nitrogen
CAM	chorioallantoic membrane
CBC	complete blood count
CDC	Centre for Disease Control
CF	complement fixation
CK	creatine kinase
CNS	central nervous system
COX	cyclo-oxygenase
CRF	corticotropin-releasing factor
CT	computed tomography
DIC	disseminated intravascular coagulation
DMSO	dimethyl sulfoxide
ECG	electrocardiogram
ECL	electrochemiluminescent
ELISA	enzyme-linked immunosorbent assay
ESF	external skeletal fixation
FA	fluorescent antibody
FSH	follicle stimulating hormone
GABA	gamma-amino butyric acid
GFR	glomerular filtration rate
GGT	gamma glutamyl transferase
GLDH	glutamate dehydrogenase
GnRH	gonadotrophin-releasing hormone
HCG	human chorionic gonadotropin
IFA	immunofluorescent antibody
IPD	internal papilloma disease
IPPV	intermittent positive pressure ventilation
LDH	lactate dehydrogenase
LH	luteinizing hormone
LHRH	luteinizing hormone releasing hormone
LPS	lipopolysaccharide
MCH	mean corpuscular haemoglobin
MCHC	mean corpuscular haemoglobin count
MCV	mean cell volume
MRI	magnetic resonance imaging
MSH	melanotropic hormone
NSAID	non-steroidal anti-inflammatory drug
PAS	periodic acid–Schiff
PBFD	psittacine beak and feather disease
PCR	polymerase chain reaction
PCV	packed cell volume
PDD	proventricular dilatation disease
PET	positron emission tomography
PG	prostaglandin
PMV	paramyxovirus
PP	pancreatic polypeptide
PsHV	psittacid herpesvirus
PT	prothrombin time
PTFE	polytetrafluoroethylene
PTH	parathyroid hormone
RBC	red blood cell
SGOT	serum glutamate oxaloacetate transaminase
SGPT	serum glutamate pyruvate transaminase
SPF	specific pathogen-free
STC	spontaneous turkey cardiomyopathy
STH	somatotropic hormone
TG	thyroglobulin
TIF	tie-in fixator

Downloaded From Vet-eBooks.com

TRH	thyrotropin releasing hormone
TSH	thyroid-stimulating hormone
UDCA	ursodeoxycholic acid
USG	urine specific gravity
VPC	ventricular premature contraction
WBC	white blood cell

Downloaded From Vet-eBooks.com

US AND INTERNATIONAL UNIT (BLOOD VALUES) CONVERSION

	CONVENTIONAL (US) UNIT	CONVERSION FACTOR	SI UNIT
Haematology			
Red blood cell count	10^6/µl	1	10^{12}/l
Haemoglobin	g/dl	0.1	g/l
MCH	pg/cell	1	pg/cell
MCHC	g/dl	0.1	g/l
MCV	μm^3	1	fl
Platelet count	10^3/µl	1	10^9/l
White blood cell count	10^3/µl	1	10^9/l
Plasma chemistry			
Alkaline phosphatase	u/l	1	IU/l
ALT (SGPT)	u/l	1	IU/l
Albumin	g/dl	10	g/l
Ammonia (NH_4)	µg/dl	0.5871	µmol/l
Amylase	u/l	1	IU/l
AST (SGOT)	u/l	1	IU/l
Bilirubin	mg/dl	17.1	µmol/l
Calcium	mg/dl	0.2495	mmol/l
Carbon dioxide	mEq/l	1	mmol/l
Chloride	mEq/l	1	mmol/l
Cholesterol	mg/dl	0.02586	mmol/l
Cortisol	µg/dl	27.59	nmol/l
Creatine kinase	u/l	1	IU/l
Creatinine	mg/dl	88.4	µmol/l
Fibrinogen	mg/dl	0.01	g/l
Glucose	mg/dl	0.05551	mmol/l
Iron	µg/dl	0.1791	µmol/l
Lipase			
Sigma Tietz	u/dl	280	IU/l
Cherry Crandall	u/l	1	IU/l
Lipid, total	mg/dl	0.01	g/l
Osmolality	mOsm/kg	1	mmol/kg
Phosphate (as inorganic P)	mg/dl	0.3229	mmol/l
Potassium	mEq/l	1	mmol/l
Protein, total	g/dl	10	g/l
Sodium	mEq/l	1	mmol/l
Thyroxine (T_4)	µg/dl	12.87	nmol/l
Urea nitrogen	mg/dl	0.357	mmol/l*
Uric acid	mg/dl	59.48	µmol/l

Modified from: *The Merck Veterinary Manual* (1998) 8th edn. Merck and Co., Whitehouse Station, NJ as adapted from *The SI Manual in Health Care* (1981) Metric Commission, Canada.

*Urea.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 1

CLINICAL ANATOMY AND PHYSIOLOGY

INTRODUCTION

Although veterinarians are taught avian anatomy in veterinary school, if one is unfamiliar with its clinical relevance it is always worth refreshing one's memory. This chapter will highlight those features of avian anatomy and physiology that are relevant to the clinician. It will not seek to be a comprehensive review of the subject. The focus in this chapter is on companion birds and, as such, anatomical structures such as the phallus (found in ratites and waterfowl) will not be discussed. Without a basic understanding of anatomy and physiology, it becomes difficult to understand the pathophysiology of disease and how treatment will affect the patient as a whole.

THE SKIN, FEATHERS, NAILS AND BEAK

Avian skin is attached both to the underlying muscles and, more tightly, to the skeleton. It consists of the thin epidermis (only 10 cell layers deep) and the thicker underlying dermis. Feather follicles originate from the dermis. True glands are absent from much of the skin, although epidermal cells may secrete a lipoid sebaceous material. The external ear has glands that secrete a waxy material, but the only other gland found on the skin is the uropygial gland. This gland, absent in ratites, many pigeons, woodpeckers and some parrots (*Amazona*, *Anodorhynchus* and *Cyanospitta*), is a bi-lobed gland on the dorsum of the tail. It secretes a lipoid sebaceous material, spread over feathers during grooming, which may assist in waterproofing, cleaning and reducing skin infections. It is thought it may also have a role in sex identification; uropygial secretions may play a role in ultraviolet reflectivity, which in turn may be used by birds for sex differentiation.

Scales, raised areas of highly keratinised epidermis separated by folds of less keratinised skin, cover the non-feathered part of the leg, known as the podotheca. The claws, which enclose the terminal phalanx of each digit, are made up of two plates: the strongly keratinised dorsal plate enclosing dorsal and lateral aspects of the phalanx, and the softer ventral plate forming the sole of the claw. The dorsal plate grows faster than the ventral plate, therefore the nails curve downwards.

The bones of the upper and lower jaw are covered in horny keratin, called rhamphotheca (**Fig. 1.1**); the mandibular rhamphotheca is known as the gnathotheca and the maxillary rhamphotheca is called the rhinotheca. The dorsal midline of the rhinotheca is the culmen and the ventral midline of the gnathotheca is the gonys. The cutting edge of both the upper and lower beak is the tomia, while the soft tissue between the mandibular rami is the inter-ramular region (**Fig. 1.2**). Histologically the rhamphotheca resembles skin, with the dermis attached to the periosteum of the underlying bone. The epidermis is modified, in that the stratum corneum is thickened and hardened, as the cells contain free calcium phosphate and crystals of hydroxyapatite. The neurological innervation of the upper beak is the ophthalmic and maxillary divisions of the trigeminal nerve, while the mandibular division of the trigeminal nerve innervates the lower beak. The cere, the fleshy area around the nares, is found only in owls, parrots and pigeons.

The unique structure of avian skin is the feathers. These arise from feather follicles, arranged in tracts around the body known as pterylae. The featherless skin between these tracts is called apterylae. Each follicle is a cylindrical pit in the skin, lined with epidermis and dermis. At the base of the follicle is the dermal papilla, a small mound of dermis that enters the proximal shaft of the feather (the calamus)

Downloaded From Vet-eBooks.com



Figure 1.1 **A red-tailed black cockatoo. Note that the keratin covering its beak (the rhamphotheca) is histologically similar to skin.**

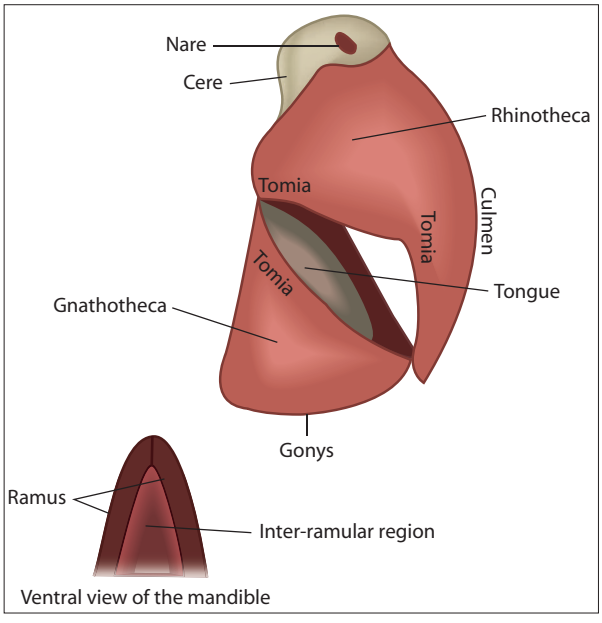


Figure 1.2 **Labelled diagram of a bird beak with key parts and their locations.**

through a small hole known as the inferior umbilicus. The epidermis covering the dermal papilla is continuous with the calamus and with a thin layer of epidermis lining the follicle (**Fig. 1.3**).

Each feather shaft consists of the calamus, embedded in follicle, and the rachis, the main shaft beyond the calamus. They are distinguished by the

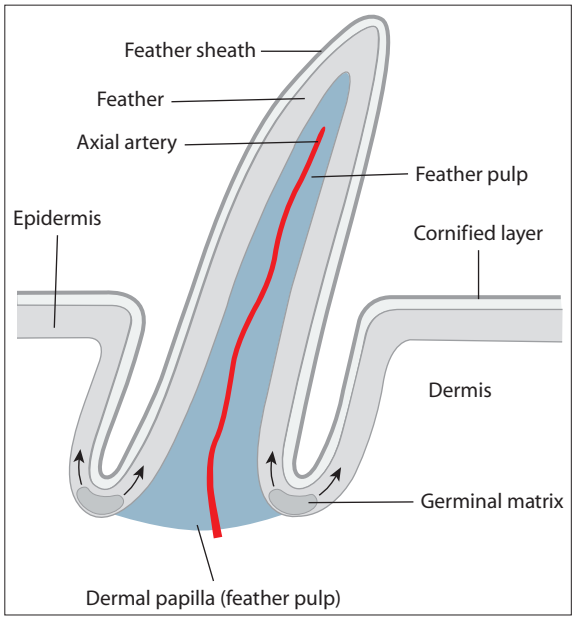


Figure 1.3 **Diagram of the internal structures of the feather follicle.**

distal (superior) umbilicus, a small opening into the shaft found at the junction of the rachis and calamus. Occasionally there is an after feather (the hypopenna), a small extra feather on the rim of the distal umbilicus (**Figs 1.4, 1.5**).

Coming off the rachis are the barbs and coming off the barbs are the barbules, filaments that interlock to form the vane (**Fig. 1.6**). This is the pennaceous region. The vanes are asymmetrical, with the external vane narrower than the internal vane. On the dorsal wing the external vane of one feather overlaps the internal vane of the next. Just below the vane is the plumaceous region, where a few downy barbs fail to interlock.

Within the calamus of an immature feather is the pulp, a loose reticulum of mesoderm with an axial artery and vein. This pulp retracts as the feather matures, leaving pulp caps (empty chambers within the calamus).

There are seven types of feathers (**Fig. 1.7**): contour, semiplume, down, powder down, hypopenna, filoplume and bristle.

- Contour feathers include the flight feathers and the body feathers. The flight feathers on the tail are called the retrices. The flight feathers on the wings are known as the remiges: the primaries (9–11)

Downloaded From Vet-eBooks.com

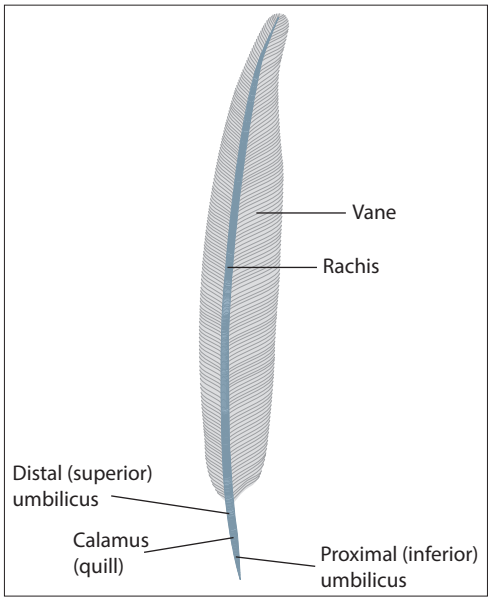


Figure 1.4 Diagram of the external parts of a feather.

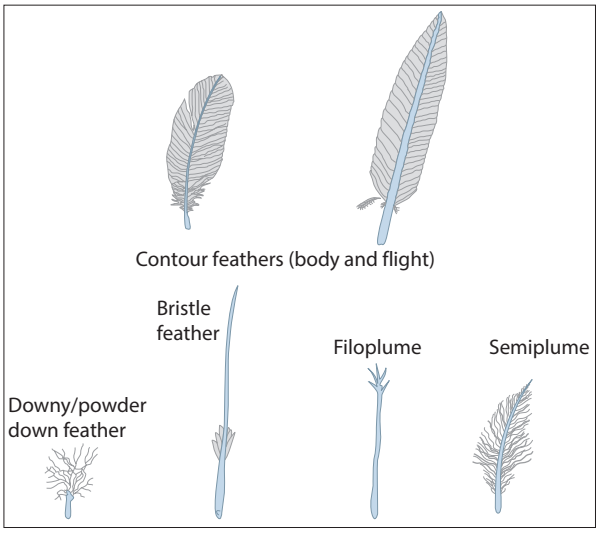


Figure 1.7 Illustration of the range and shapes of different types of feather.

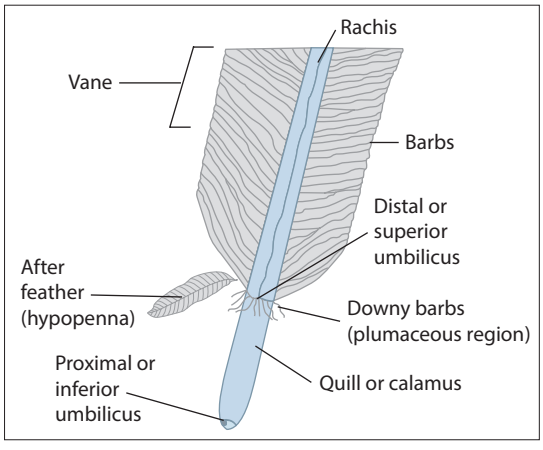


Figure 1.5 Closer view of the calamus and rachis parts of a feather.

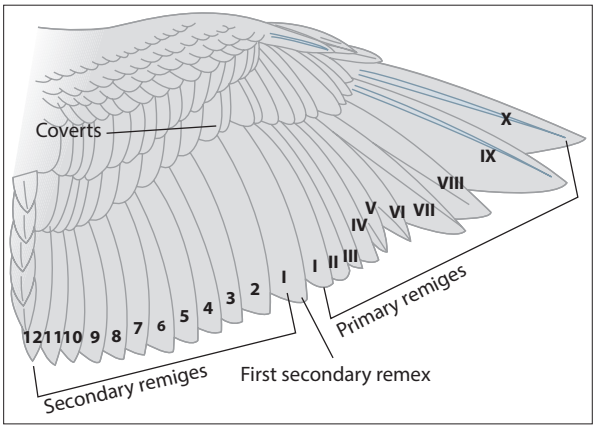


Figure 1.8 Schematic diagram of the location and types of feathers that make up a parrot wing.

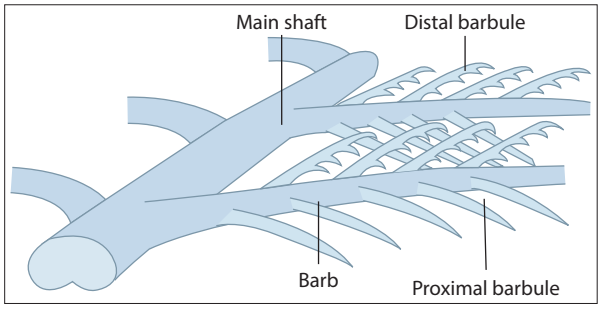


Figure 1.6 Closer view of the pennaceous portion of a feather.

in total arise from the periosteum of the metacarpus; the secondaries (6–32) in total arise from the periosteum of the ulna; and the tertiaries arise from the humeral area. Overlying them are the coverts (**Fig. 1.8**).

- Semi-plume feathers have a wholly fluffy vane, with the rachis longer than barb. They lie along pterylae margins, acting as insulation.
- Down feathers are also wholly fluffy, but the rachis is either absent or shorter than the longest barb. Distribution varies between species.

Downloaded From Vet-eBooks.com

- Powder down feathers are structured like down feathers, although some are semi-plumes or contour feathers. They shed a fine waxy powder, which is actually keratin flakes. This powder forms a waterproofing coat over the contour feathers and may play a role in keeping the bird clean. Powder down feathers are usually grouped in patches (e.g. on the thigh), although some species have them widely distributed. They are found in herons, parrots, toucans, pigeons and bowerbirds.
- Hypopennae (1–5) in total are small feathers projecting from the distal umbilicus of pennaceous and plumaceous feathers (after feathers). They are usually not associated with retrices or longer remiges.
- Filoplumes have a long fine shaft with a tuft of short barbs/barbules at the end. They possibly have a sensory/proprioceptive role and are found close to the follicles of contour feathers.
- Bristles have a stiff rachis, with either a few barbs at the proximal end or no barbs at all. They are found around the mouth, nares and eyes, and possibly have a tactile function.

The colour of feathers is the result of the combination of pigments and feather structure. Carotenoids or psittacins (yellow pigments absorbed from the diet, including reds, oranges and pinks) create the foreground colour. Melanins are the grey pigments (including black, grey and brown) that create the background and also the foreground colour. Each feather barb has a cortex (an outer layer) containing either carotenoid pigments (psittacins) or melanin pigments. If melanin is in the cortex, it is known as foreground colour and produces black, greys, dark browns and chestnut reds. This is the marking seen in many birds. The barb also has a medulla, which only ever contains melanin (background melanin). All of these pigments are distributed in different layers and, when combined with special features of barb structure that affect the passage of light, produce the spectrum of colours seen.

Moulting, the shedding of old, worn feathers and the renewal of plumage, is a regular event. It is controlled by a wide range of factors including thyroid activity, reproductive hormones, photoperiod, body condition, age and diet. After a series of juvenile moults to attain adult plumage, most birds go on to moult one to two times annually. These moults, often referred to as the prenuptial and postnuptial moults, occur in spring and autumn respectively. The pattern of moulting is orderly and in the following progression (with some overlap): the inner primaries; the outer primaries; the secondaries and tail feathers; and finally the body contour feathers. It is usually bilaterally symmetrical and is paced so as to avoid loss of flight capacity at any time.

When it is time to moult an old feather, a proliferation of epidermal cells at the base of the follicle (the epidermal collar) separates the old feather from the dermal papilla and allows it to shed. These epidermal cells then start to group themselves into two series of spiral barb ridges. The tips of these ridges end along a longitudinal line on the ventral aspect of the feather (the seam). On the dorsal side of feather the epidermis thickens to form the rachis. Within this structure is the dermal core, consisting of the axial blood vessels, with mesoderm around them. As it grows the feather emerges from the follicle as a pointed projection with a dermal core and an epidermal cover (sheath). This sheath then progressively ruptures, freeing the barbs that have separated along the seam and allowing the feather to open. Much of the increased grooming activity seen in birds at this time is to remove this sheath.

Because birds lack sweat glands, they rely on evaporative heat loss from the respiratory tract and heat transfer through apterylae (the featherless tracts of skin) to cool their bodies. To do this, many birds hold their feathers close to the body and may extend their wings, exposing the apterylae. Conversely, to retain body heat when ill or cold, they fluff their feathers up to trap body heat against the skin. (Ostriches do the reverse, i.e. they raise their feathers to promote heat loss and hold them close to conserve body heat.)

THE SKELETON

General

Bones serve two major functions: they provide structural support for the muscular system and they act as a reservoir for calcium and phosphorus (**Fig. 1.9**). Although the structural make-up of bone is similar across all animal species, there are some specific

Downloaded From Vet-eBooks.com

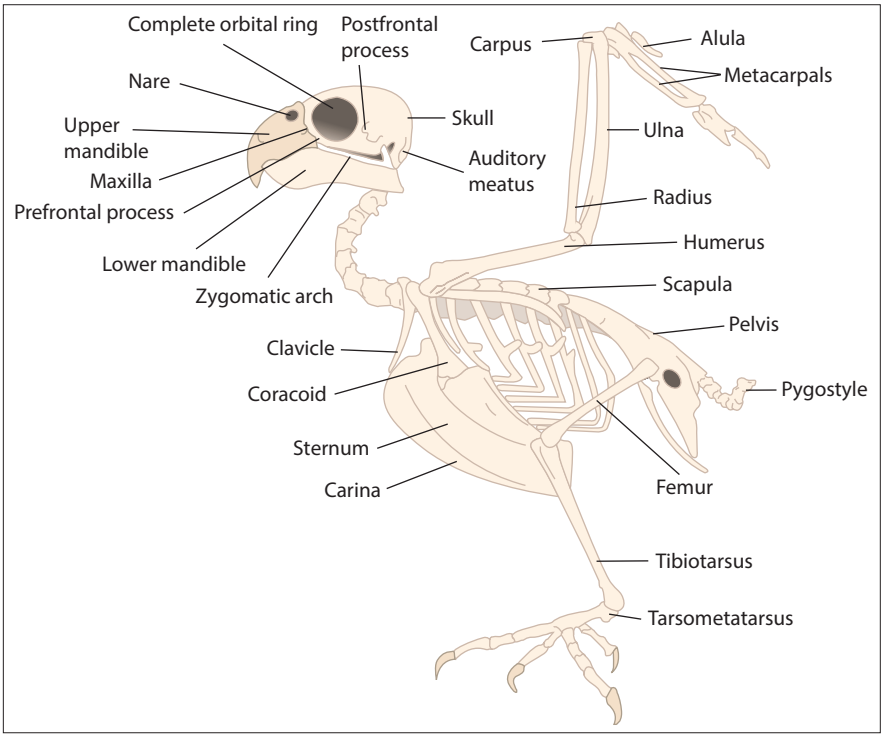


Figure 1.9 **Labelled diagram of the skeleton of a bird.**

differences between mammalian and avian bones. The requirement for flight means that birds have evolved with bones that are lightweight, but aerodynamically strong. They have thin brittle cortices and wide medullas that may, in some species and some bones, be pneumatic. Under the influence of oestrogen during the breeding season, many hens will lay down medullary bone (extra bone in the medullary cavities of the long bones) to form a calcium reservoir for egg production.

The blood supply to bones arises from periosteal, medullary, metaphyseal and epiphyseal vessels. The periosteal blood supply is the predominant source of blood to the bone and its disruption, either by trauma or surgical repair of a fracture, may result in delayed healing or even complete failure to heal (a non-union).

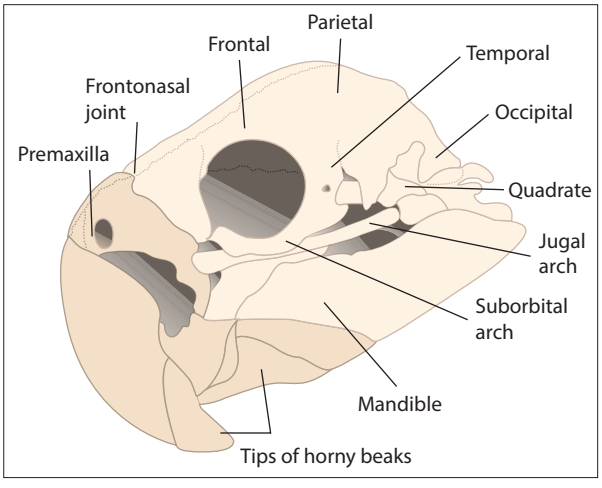


Figure 1.10 **Labelled diagram of a psittacine, or parrot, skull.**

Skull

The upper jaw of psittacine birds consists of three bones: the premaxilla, the nasal bone and the maxilla (**Fig. 1.10**). Together they form a rigid block hinged to the braincase by the prokinetic craniofacial joint (**Fig. 1.11**). The elastic zone of this joint allows movement of the upper jaw. The palate, made up of the palatal processes of the premaxillary and maxillary bones, the palatine bones, the vomers, jugal arches, pterygoids and the quadrate bones, is not a complete shelf between the oral and the nasal cavities. The left and right quadrate bones articulate between the mandible, the braincase, the jugal arches and

Downloaded From Vet-eBooks.com

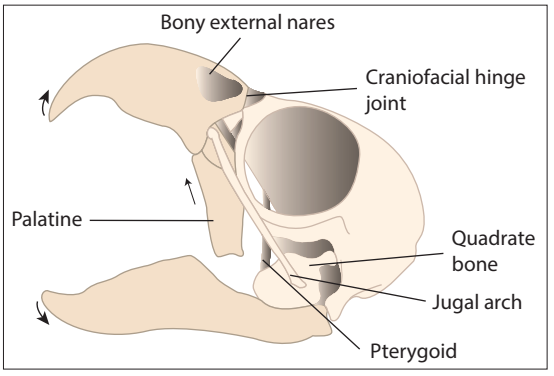


Figure 1.11 Diagram illustrating the movement (prokinesis) which occurs in the bones of the skull to enable feeding.

the pterygoid bones. Rostral rotation pushes the jaw open and *vice versa*. The eye orbit is complete only in parrots. There is a very thin inter-orbital septum with the orbital nerves running through the caudal edge of this septum. The lower jaw consists of two mandibular rami fused at a symphysis.

Vertebrae

The requirements of flight have limited the flexibility of the avian spinal column. The most mobile part is the cervical spine, made up of 11–12 vertebrae in parrots. They provide sufficient flexibility for a bird to reach its tail and uropygial gland. The notarial vertebrae, carrying the ribs, are somewhat flexible in parrots, but are fused in many other species to form the notarium. A slightly flexible notarial-synsacral joint connects the notarium to the synsacrum, made up of 10–23 fused notarial, lumbar, sacral and coccygeal vertebrae. It is followed by five to eight free coccygeal vertebrae and then the pygostyle (four to ten fused coccygeal vertebrae), which supports the tail retrices.

Ribs

There are three to nine pairs of ribs, each with a dorsal vertebral component (made of bone) and a ventral sternal component (made of ossified cartilage). There is an uncinate process (caudodorsal process) on the vertebral ribs.

Sternum

The size of the sternum increases with increasing flight or swimming abilities, as it serves as the attachment of the pectoral muscles needed for flight. There is a prominent ventral medial keel (the carina) in many species.

Pectoral girdle

The pectoral girdle is made up of the scapulae, the coracoids and the clavicles. The scapula is strongly attached to the ribs and, in some species, reaches to the ilium. The coracoid is massive in most birds, functioning to hold the wing away from the sternum during flight. The clavicles fuse ventrally to form the furcula. In many parrots they are united only by cartilage or fibrous tissue. The ventral part of the furcular is attached to the apex of the sternal keel by ligaments. The clavicles serve as a transverse spacer, bracing the wings apart, and for attachment of muscles that produce the downstroke of the wings. These three bones come together at the canalis triosseus (foramen triosseum, triosseal canal), through which the tendon of the supracoracoid muscle passes. This muscle lifts the humerus for the upstroke of the wing. The glenoid cavity, formed by the scapula and coracoid, is directed laterally and allows abduction and adduction of the wing.

Wings

The humerus, in most pet species, is a pneumatic bone; the lateral diverticulum of the clavicular air sac enters through the pneumatic foramen on the medial side of the greater tuberosity. In flight the dorsal edge of the humerus becomes the trailing edge of the wing, demonstrating the range of movement that the humerus is capable of, a range of movement that includes elevation, depression, protraction, retraction and dorsal and ventral rotation. The ulna is larger than the radius. The secondary flight feathers are anchored to the ulna by ligaments. The 'wrist' joint is formed by the radial carpal bone (cranial) and the ulnar carpal bone (caudal) articulating with the carpometacarpus. This consists of the metacarpal bones and the digits. The major and minor metacarpals are fused proximally and distally with an interosseus space. There are three digits: the alular digit with one phalanx; the minor digit, also with one phalanx; and the major digit with two phalanges (**Fig. 1.12**).

Downloaded From Vet-eBooks.com

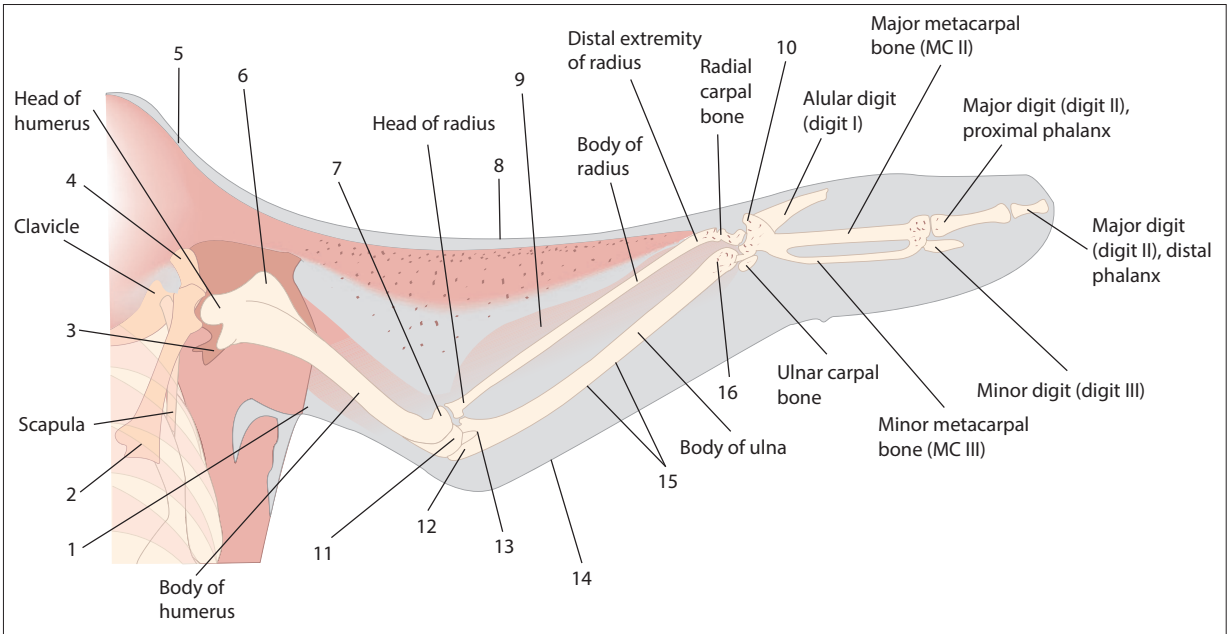


Figure 1.12 **Labelled diagram of a bird wing showing the location and names of the bones.**

Pelvic girdle

The pelvic girdle contains the ilium, ischium and pubis, all partially fused with each other and the synsacrum. The girdle is incomplete ventrally for passage of large fragile eggs (the pelvic symphysis is present only in ostriches and rheas).

Legs

The femur is a stout and relatively short bone that slopes cranially to bring the legs forward towards the centre of gravity. A patella is present in most birds. The tibiotarsus is formed by the fusion of the tibia and the proximal row of tarsal bones; the hock joint, therefore, is actually an intertarsal joint. The fibula extends two-thirds of the way down the tibiotarsus, to which it is fused. The reduction in its size limits rotation of the leg. The tarsometatarsus, formed by the fusion of the distal row of tarsal bones to the three main metatarsal bones of digits II, III and IV, is usually shorter than the tibiotarsus except in long-legged birds. There are four digits in parrots: I has two phalanges and is usually directed backwards; II has three phalanges; III has four phalanges; and IV has five phalanges. The fourth digit is directed caudally in parrots (**Figs 1.13a** and **b**).

THE DIGESTIVE TRACT

Oropharynx

The choana is a median fissure in the palate connecting the oropharynx to the nasal cavity. The palate is usually ridged laterally and rostrally to the choana, and is associated with the dehusking of seed and other foods. Caudal to the choana and palate is the infundibular cleft, a slit-like opening in the midline that is common to the right and left pharyngotympanic (Eustachian) tubes.

Downloaded From Vet-eBooks.com

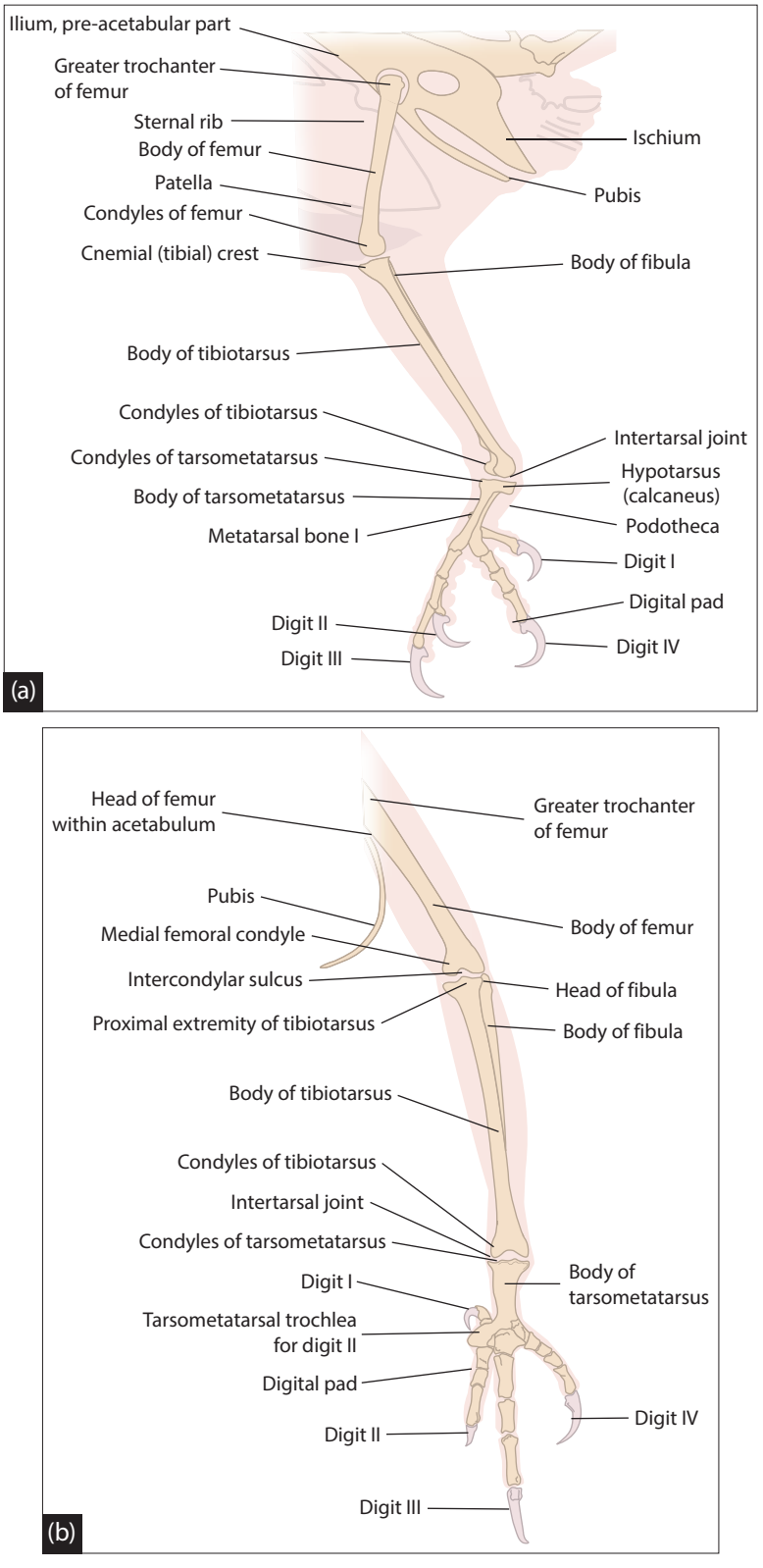


Figure 1.13 **Labelled diagrams of a parrot leg as seen in a lateral view (a) and craniocaudal view (b).**

Downloaded From Vet-eBooks.com

This cleft cannot be closed by atmospheric pressure, making changes in altitude while flying possible. There is also lymphatic tissue (the pharyngeal tonsil) abundant in wall of the cleft.

The tongue is supported by the hypobranchial (hyoid) apparatus. It has many adaptations for collecting and manipulating food and for swallowing. Only parrots have intrinsic muscles within the tongue. Just caudal to the tongue is the laryngeal mound, which carries the glottis. It has several rows of backward-pointing papillae to aid in swallowing.

The salivary glands are found in: the roof of the oropharynx (maxillary, palatine and sphenopterygoid glands); the angle of the mouth and cheeks; and the floor of the oropharynx (mandibular, lingual, and cricoarytenoid glands). They are best developed in birds that evolved on a dry diet.

Oesophagus and crop

The oesophagus is thin-walled and distensible, with a relatively greater diameter than that found in mammals (**Figs 1.14a** and **b**). Longitudinal folds on the internal surface allow for this distension; the size and degree of these folds is proportional to the size of the food particles swallowed. The oesophagus is lined with incompletely keratinized squamous epithelial cells and mucus glands (especially in the distal oesophagus).

The crop is an enlargement of the oesophagus found in many (but not all) birds (e.g. it is very prominent in parrots, but very small in most passerines). Its lining is similar to that of the oesophagus, except that it has no mucus glands. It serves as a food storage area for birds that eat rapidly and then move to a safer area to give the food time to pass further down the digestive tract. The exit from the crop into the

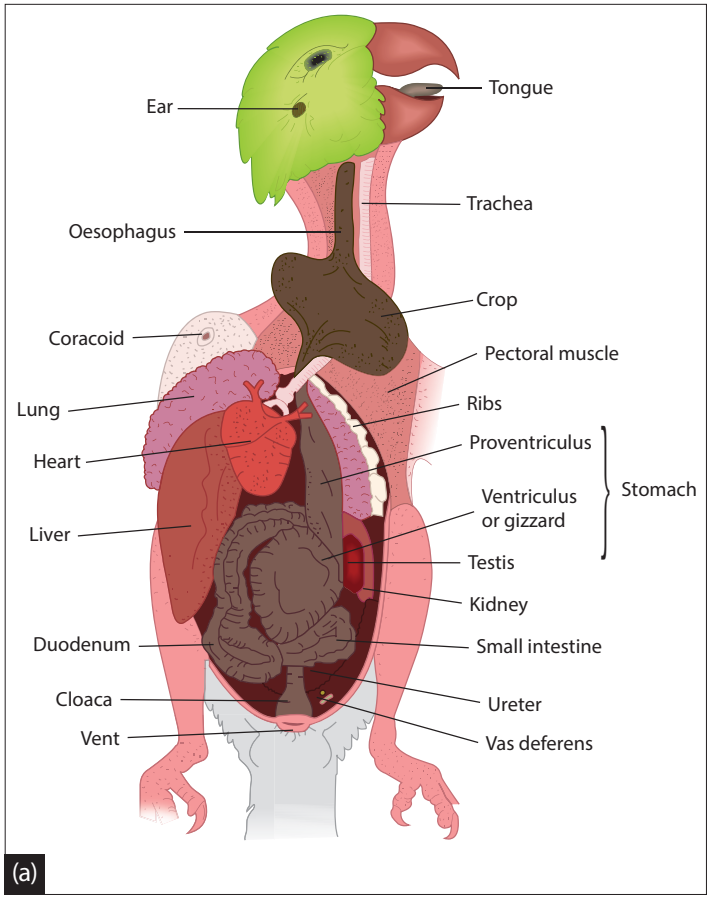


Figure 1.14 **Labelled diagrams illustrating the key components of the gastrointestinal tract viewed ventrally (a).** ***(Continued)***

Downloaded From Vet-eBooks.com

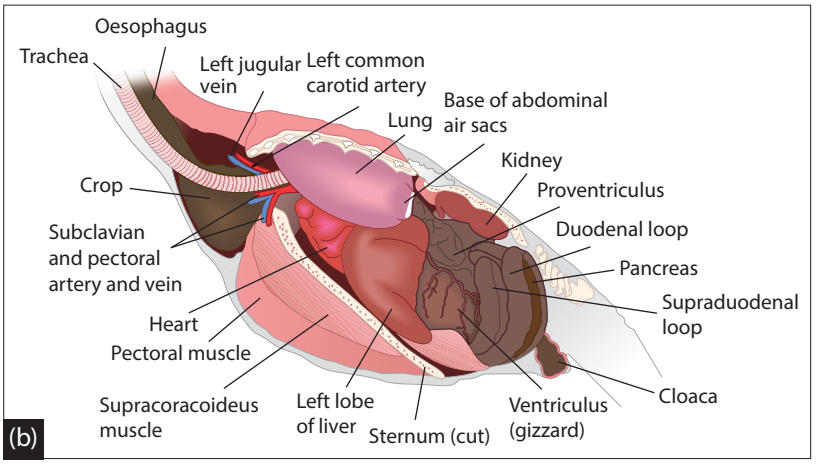


Figure 1.14 (Continued) **Labelled diagrams illustrating the key components of the gastrointestinal tract viewed laterally (b).**

distal oesophagus is at the level of the thoracic inlet, on the right side of the midline of the neck. A fold of crop tissue lies over the exit, which then makes an S-shaped turn into the distal oesophagus.

Proventriculus and ventriculus

There is no obvious boundary between the distal oesophagus and the proventriculus, other than a lack of internal folds. The proventriculus is lined with mucus-secreting columnar epithelial cells. Within the laminar propria are the gastric glands, multi- or unilobular glands lined with tall columnar mucus cells. They discharge into an alveolus, which then drains into the central cavity of the lobule. Secondary ducts collect from different glands, which then empty into a primary duct and this empties into the proventricular lumen. These glands may be present throughout the proventriculus or contained in defined tracts or areas. They produce hydrochloric acid and pepsin.

Between the proventriculus and the gizzard is the intermediate zone, a variably developed region with a microscopic structure somewhere between the two. It may narrow to form an isthmus between the proventriculus and gizzard.

The gizzard (or ventriculus) varies in size and shape between species. Those species that eat soft food (e.g. lorikeets) have smaller, rounder gizzards, which can be somewhat difficult to distinguish from the proventriculus. Other species have a thickened, biconvex gizzard, with a wall consisting of smooth muscle bands, rich in myoglobin. Asymmetrical arrangement of these muscle bands results in both rotatory and crushing movements when the gizzard contracts. The gizzard is lined with simple columnar epithelium, with crypts containing exits for tubular glands in the lamina propria. These tubular glands secrete hard vertical rods that interconnect laterally for greater strength. Between them is a softer horizontal matrix of carbohydrate–protein complex secreted by the cells of the epithelium and crypts. This matrix hardens with the effect of hydrochloric acid. The vertical rods project slightly out from the horizontal matrix. This layer of rods and matrix is known as the cuticle or koilin layer. When combined with the asymmetrical rotatory grinding of the ventricular muscles, these rods and the matrix are very effective at crushing and grinding food into a soft pulp.

There is also a pyloric region connecting the gizzard to the duodenum. Its lining is microscopically intermediate between gizzard and duodenum. Its function is unclear.

Intestinal tract

The duodenum forms a narrow U-shape on the right side of the gizzard, with the descending duodenum proximal to the ascending duodenum. It is lined with mucus-secreting goblet cells. The bile and pancreatic ducts often open near to each other in the distal end of the ascending duodenum. There may be two

Downloaded From Vet-eBooks.com

bile ducts (the common hepatoenteric duct and the cystoenteric duct) and two to three pancreatic ducts.

The jejunum and ileum are usually arranged in a number of narrow U-shaped loops at the edge of the dorsal mesentery on the right side of the coelomic cavity. The vitelline (Meckel's) diverticulum is the short blind remnant of the yolk sac; it can be used to differentiate jejunum from ileum.

The large intestine is very short, separated from the ileum by the ileorectal sphincter. In some species, caeca arise from the rectum at the junction of the rectum with the ileum. Their form and size vary, and they are reduced or absent in parrots, swifts and pigeons (see **Figs 1.14a** and **b**).

Pancreas

The avian pancreas consists of three lobes. The dorsal and ventral lobes are supported and separated by the pancreatic artery within the duodenal loop, and the splenic lobe runs more laterally up to the spleen, as an extension of the ventral lobe (see **Figs 1.14a** and **b**). The pancreas has both endocrine and exocrine functions. While the amount of endocrine tissue is proportionally greater than that of mammals, over 99% of the pancreatic mass has an exocrine function. The exocrine pancreas consists of compound tubuloacinar glands divided into lobules. These glands secrete amylase, lipase, proteolytic enzymes and sodium bicarbonate into the ascending duodenum via pancreatic ducts. Pancreatic secretion, which is at a higher rate than that of mammals, is controlled by both neural and hormonal mechanisms. Immediately a bird starts eating, pancreatic secretion begins, apparently via a vagal reflex. Distension of the proventriculus stimulates a hormonal response involving a vasoactive intestinal polypeptide; this results in pancreatic secretion. Diet can also affect the rate of secretion, with diets high in fat and carbohydrates increasing the activity of amylase and lipase.

Liver

The avian liver consists of the right and left lobes joined cranially in the midline. The right lobe is larger than the left, with each lobe having several small processes. The liver is enclosed in a thin and slightly elastic capsule of connective tissue, allowing its expansion. Blood is supplied to the liver by the right and left hepatic arteries and hepatic portal veins. The hepatic arteries arise from the coeliac artery, while the portal veins drain blood from the proventriculus, ventriculus, duodenum, pancreas, intestines and cloaca. Two hepatic veins join the caudal vena cava cranial to the liver, draining blood away from the liver.

Terminal portal venules and arterioles empty into sinusoids between plates of hepatocytes. The low pressure in these sinusoids allows the hepatocytes to absorb molecules from the blood. Phagocytic Kupffer cells are also present in the sinusoids, collecting particulate matter and microorganisms. The now 'filtered' blood drains into the hepatic veins and on to the heart. The oxygenated arterial blood maintains the viability of the hepatocytes. Bile canaliculi form between three to five hepatocytes and drain into a bile ductule.

A portal triad of arteriole, portal venule and bile ductule, along with associated hepatocytes, bile canaliculi and sinusoids, forms the basic functional unit of the liver: the hepatic acinus. Hepatocytes close to these portal triads are said to be 'periportal'. Those further away, near the hepatic venules, are called 'periacinar'. The intermediate area is termed the 'midzone'. The hepatocytes in these different areas, although morphologically the same, are biochemically different and react differently to incoming chemicals and metabolites.

Bile is produced by hepatocytes and enters the bile canaliculi and then the ductules in the portal triad, which then empty into the interlobular ducts. These in turn form the right and left hepatic ducts, which join to become the common hepatoenteric duct emptying into the duodenum. A branch of the right hepatic duct either forms the right hepatoenteric duct (emptying into the duodenum) or, in those birds with a gall bladder, the hepatocystic duct entering the gall bladder. (Pigeons, most parrots and ostriches do not have gall bladders.) From there the cystoenteric duct runs to the duodenum. Birds thus have two bile ducts emptying into the duodenum.

The liver has several functions in the body:

- Digestion. Bile contains bile acids, synthesised in the liver from cholesterol. (In birds the primary bile acid is chenodeoxycholic acid.)

Downloaded From Vet-eBooks.com

In the distal duodenum these bile acids emulsify fat, facilitating its digestion. Bile acids are then resorbed in the jejunum and ileum and recirculated through the liver. Bile also plays a role in the digestion of carbohydrates and protein. It contains amylase and helps to activate pancreatic amylase and lipase in the duodenum. Because of the lack of biliverdin reductase and glucuronyl transferase in birds, the primary bile pigment is biliverdin, giving avian bile its characteristic green colour.

- Carbohydrate metabolism. The portal blood supply, carrying nutrient-rich blood from the gastrointestinal tract, supplies the liver with these nutrients before any other major organs. Hepatic enzymes carry out glycogenesis, protein synthesis and lipogenesis in the well-fed bird. The glycogen, protein and triglycerides produced in the liver enter the circulation and are used (or stored) throughout the body. If a bird is fasted (for any reason), the resultant hypoglycaemia stimulates glucagon production, which in turn activates liver enzymatic pathways to produce glucose through glycogeolysis, gluconeogenesis and lipolysis. The liver therefore plays a major role in carbohydrate metabolism.
- Metabolism of metabolites, drugs and chemicals. The liver, through its microsomal drug-metabolizing enzyme system in the periacinar hepatocytes, processes both endogenous metabolites and exogenous chemicals. Hydrophobic, lipid-soluble molecules (which are difficult to eliminate) are converted by the liver to hydrophilic, water-soluble molecules and excreted in bile and urine. This is done in two phases; in the first, enzymes modify the molecules by oxidation or reduction; in the second they are enzymatically conjugated with other molecules to become sufficiently water soluble. The best example of this is the synthesis of urea and uric acid from protein in the liver.
- Protein synthesis. The liver is the primary site of synthesis of a range of essential proteins:
- Albumin.
- Fibrinogen, prothrombin and clotting factors I, II, V, VI, VII, IX and XII.
- Molecules involved in the transport of metals, hormones, and lipids (e.g. ceruloplasmin and macroglobulins).
- Antimicrobial effect. The Kupffer cells in the sinusoids are important in the clearance of microorganisms entering the portal circulation in cases of intestinal infections or surgery. They also play a role in the detoxification of bacterial endotoxins.

Cloaca

The cloaca is the common exit for the gastrointestinal, urinary and reproductive tracts. As a point of terminology, the cloaca is the chamber; its opening to the skin is the vent. The cloaca is divided internally by two mucosal folds into three compartments: the coprodeum, the urodeum and the proctodeum (**Fig. 1.15**). This structure is similar in all birds; the main variation is the presence or absence of the phallic structures of the proctodeum.

The coprodeum is the most cranial, and largest, compartment. There is no distinction between rectum and coprodeum (except in the ostrich, which has a rectocoprodeal fold, and Anatidae, where there is an abrupt change in gross appearance of the mucosa). Some species have villi and folds on the mucosa; others have none.

The urodeum is the middle and smallest compartment, separated from the other two compartments by two circular mucosal folds. The coprourodeal fold is a cranial annular fold that stretches to become a thin diaphragm if the coprodeum is full of faeces; it may close during egg laying to prevent defecation while the bird is laying an egg. The uroproctodeal fold is a caudal, semicircular dorsolateral fold that fades out ventrally. The urogenital ducts open into the urodeum on the dorsolateral mucosa (the ureters dorsally, the genital ducts laterally). The ureter opens via a simple opening; the ductus deferens opens via a conical papilla. In immature hens a homologue of the ductus deferens papilla may be present, but it disappears with maturity. In the mature hen the left oviduct opens ventrally and laterally relative to the left ureter. There may be a small mound at its opening. In immature birds it is covered with a membrane that disappears at maturity.

Downloaded From Vet-eBooks.com

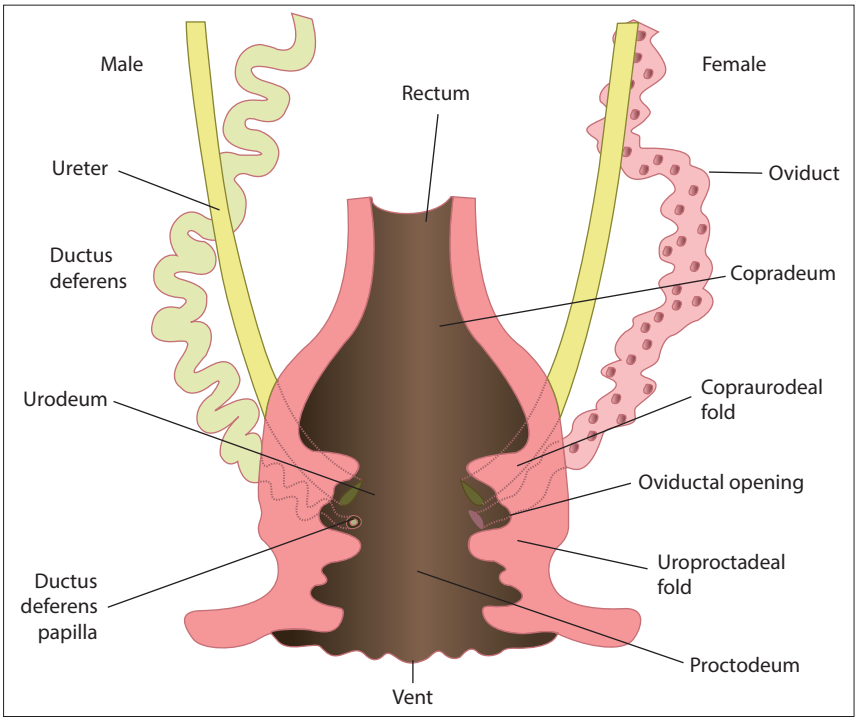


Figure 1.15 **Schematic diagram illustrating both the male and female components of the cloaca.**

The proctodeum is the short caudal compartment between the lips of the vent and the uroproctodeal fold. In immature birds an opening in the dorsal wall leads into the cloacal bursa.

The vent is a transverse slit guarded by dorsal and ventral lips. This horizontal arrangement is the reason why purse-string sutures are unsuitable to close the vent in birds.

THE URINARY SYSTEM

The kidneys lie in the renal fossae of the synsacrum, each divided into three divisions: the caudal, middle and cranial divisions (**Fig. 1.16**). (Note: These are not lobes.) The distinctions between these divisions are not always clear. The spinal nerves and sacral plexus pass through the kidneys between the middle and caudal divisions. The surface of the kidney is covered in rounded projections, the renal lobules.

Each renal lobule is a pear-shaped elongated piece of tissue wedged between the interlobular veins and enclosed by its perilobular collecting tubules. At the tapering end of the lobule the collecting tubules converge to form the medullary collecting tubules (medullary region or cone of the lobule); this tapering end also contains the nephronal loops (loops of Henle) of the medullary nephrons. The wide part of the lobule is the cortical region; it contains nephrons of both cortical and medullary nephrons (but not the medullary nephronal loops).

Several lobular medullary regions converge into a single cone-shaped assembly of collecting tubules in a connective tissue sheath (known as the medullary region of a renal lobe) draining into a single collecting duct. Several of these ducts combine to form a secondary branch of the ureter.

Although there is a lobular cortex and medulla, there are no distinct renal cortical and medullary regions because both lobes and lobules are embedded in tissue at differing depths in the kidney. There is a higher number of medullary regions in birds that conserve water and, therefore, a smaller volume of cortical regions. This implies a higher proportion of mammalian-type nephrons and, therefore, a better counter-current concentration.

There are two types of nephron: cortical (or reptilian) nephrons with no nephronal loop; and medullary (or mammalian) nephrons with a nephronal

Downloaded From Vet-eBooks.com

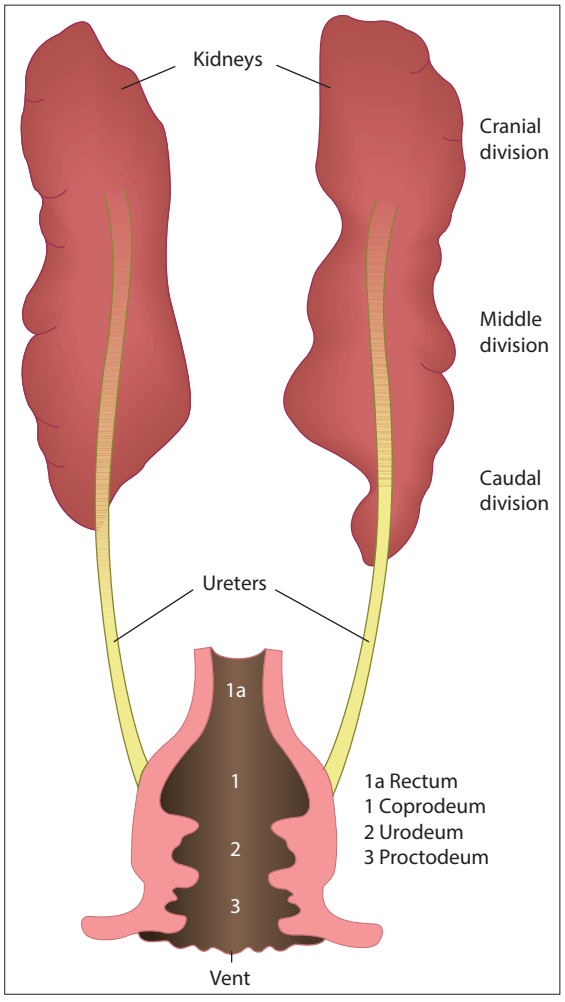


Figure 1.16 **Labelled diagram of the location of the key components of a bird's urinary system.**

loop penetrating the medullary region. Both types start with a renal corpuscle: a glomerular capsule (Bowman's) enclosing the glomerulus (tuft of capillaries). Collecting tubules lie both superficially on the surface of the cortical region (perilobular) and within the medullary region (medullary). Several form a collecting duct, which then forms a secondary branch of the ureter.

The arterial blood supply to the kidneys comes from the cranial, middle and caudal renal arteries. The kidney also receives a venous supply via the cranial and caudal renal portal veins, which form a venous ring encompassing both kidneys. Blood enters this ring from the external iliac vein, the ischiadic veins, the internal iliac veins and the caudal mesenteric veins. Afferent renal veins come off the ring and enter the renal parenchyma to become the interlobular veins. The renal portal valve is located in the common iliac vein; when it is open (adrenergic stimulation), blood is diverted into the caudal vena cava and away from the kidney.

The ureter starts within the depth of the cranial division and continues caudally in a groove on the ventral surface of the middle and caudal divisions. It receives primary branches, which in turn receive secondary branches from the collecting ducts draining several renal lobes. The ureter opens into the urodeum.

The urine: plasma osmolar ratio in most birds can only reach 2.0–2.5, compared with 25–30 in mammals. Birds will excrete 1% of filtered water, compared with mammals who excrete less than 0.1%. The concentration ability of the avian kidney lies with the mammalian nephrons; because most birds have more reptilian nephrons than mammalian nephrons, they do not produce concentrated urine. This is believed to be, in part, due to the need for water to transport the more viscous uric acid through renal tubules. Uric acid, the end product of protein metabolism in birds, is produced in the liver and removed from the blood by a combination of filtration in the glomerulus (10%) and tubular secretion in the proximal part of the nephron (90%).

Urine production is controlled by arginine vasotocin (AVT), the avian equivalent of antidiuretic hormone (ADH). Increased plasma osmolality stimulates the hypothalamus to produce AVT. This in turn constricts afferent arterioles of the reptilian nephrons (reducing the glomerular filtration rate [GFR]) and increases the permeability of the collecting ducts of the mammalian nephrons. The end result is decreased urine production and therefore decreased plasma osmolarity.

Water resorption also occurs in the rectum during retrograde flushing from the cloaca. Up to 15% of urine water can be resorbed in this manner, but this is reduced by polyuria and stress-induced defecation. If the urine is too concentrated, a concentration gradient across the rectal mucosa cannot be achieved and so resorption would be limited. But, as urine osmolality increases, birds are able gradually to increase plasma osmolality, thus preserving the urine: plasma osmolar ratio

Downloaded From Vet-eBooks.com

and allowing water resorption. Birds with functional salt glands can decrease plasma osmolality by excreting salt, but this is not applicable to parrots.

Effective osmoregulation therefore requires:

- Normal plasma osmolalarity.
- Sufficient functional nephrons.
- Normal production of, and response to, AVT.
- Efficient cloacal water resorption.

THE RESPIRATORY TRACT

Upper respiratory tract

The size and shape of the nares (nostrils) is variable between species. They are located at the top of the beak (except in the kiwi, where they are located at the tip of the beak). In parrots and pigeons (and owls) they are located within the fleshy cere. Just inside the nares is the operculum, a cornified flap of tissue.

The nasal septum is partly bony and partly cartilaginous. It is complete in parrots and many other species, and separates the nasal cavity to the level of the choana. Within the nasal cavity are the nasal conchae (turbinates), usually divided into three parts: the scroll-like highly vascular rostral turbinates with their stratified squamous epithelial lining; the middle turbinates, also scroll-like but with a mucociliary lining; and the caudal turbinates with an olfactory epithelial lining innervated by the olfactory nerve. The vascularity of the turbinates assists in the control the rate of water and heat loss from the body. The infraorbital sinus network is connected to the nasal cavity in the middle and caudal regions of the nasal cavity. However, the connections between the nasal cavity and the sinuses are such that it is difficult to sample the sinuses by a nasal flush.

The infraorbital sinuses are located around the eye, the upper beak, the mandible and the pneumatised sections of skull (**Fig. 1.17**). They are classified as rostral (in the maxillary rostrum or bill), periorbital/preorbital (rostral to the orbit), infraorbital (medial to the eye), mandibular (mandibular rostrum) and postorbital (surrounding the opening of the ear). The right and left sides communicate and have diverticula in Anseriformes, Psittaciformes and insectivorous Passeriformes. They do not communicate in non-insectivorous Passeriformes.

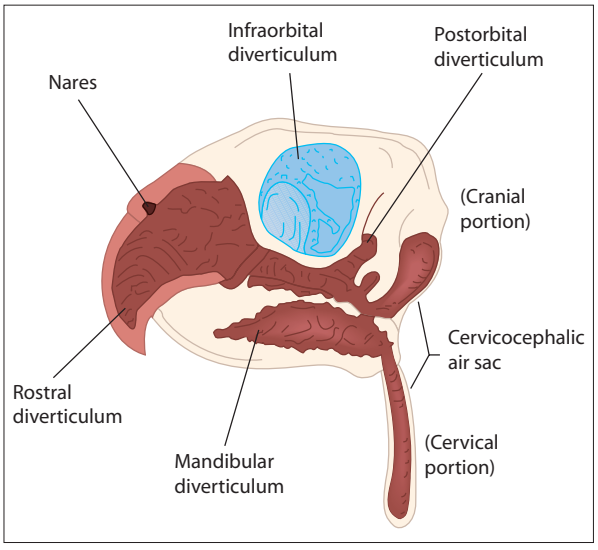


Figure 1.17 Labelled diagram of the sinus network within a psittacine skull.

Each infraorbital sinus has five diverticula and two chambers:

- Rostral diverticulum. Within the beak, surrounded by premaxillary bone.
- Maxillary chamber. Underneath the nares, lateral to the rostral diverticulum, extending back to the preorbital diverticulum.
- Preorbital diverticulum. In the space between the nares and the rostral aspect of the orbit. It is bordered medially by the nasal cavity and dorsolaterally by the nasal and frontal bones. It connects to the suborbital chamber.
- Suborbital chamber. Beneath the bony orbit. It is bordered medially by the caudal extent of the nasal cavity and laterally by the jugal and prefrontal bones.
- Infraorbital diverticulum. Above the suborbital chamber, extending behind the suborbital arch and medial to the eye. It is the largest diverticulum.
- Postorbital diverticulum. This has two parts: the caudal portion is the preauditory diverticulum, extending caudally to the bony orbit and bound by the temporal and quadrate bones laterally; and the cranial portion is immediately caudal to the orbit.
- Mandibular diverticulum. Communicating with the maxillary portion near the postorbital diverticulum. Occupies the mandibular bone.

Downloaded From Vet-eBooks.com

In addition the cervicocephalic air sac communicates with the most caudal aspect of the infraorbital sinus. This air sac does not play a role in gas exchange, nor does it communicate with the lower respiratory tract. It has two divisions: the cranial cephalic (from occipital region to just behind the cere), which is not found in many species, including the macaw; and the cervical (from the tympanic area and extending in two columns bilaterally down the neck). It is thought to play several roles: insulation for heat retention, control of buoyancy, reducing the force of impact with water in fish-eating birds, and support of the head during sleep or flight. Jugular venepuncture may result in blood entering this air sac and appearing as epistaxis.

The upper respiratory tract serves several functions: it provides a sense of smell; it filters airborne debris; it plays a role in thermoregulation; and it plays a role in water conservation.

Lower respiratory tract

Birds do not have a true larynx as such, but rather have a glottis. This has no role in voice production; its main role is preventing food passing into the trachea. There are four cartilage structures in the glottis: the cricoid, a scoop-shaped cartilage with left and right lateral wings; the procricoid, a small cartilage that articulates with the cricoid wings on the dorsal midline; and the two arytenoids that form the margins of the glottis. The glottis is located in the laryngeal mound behind the tongue. During inspiration the glottis is raised to the choana and opened, allowing air to be inspired without opening the mouth.

Unlike mammals, the avian trachea is composed of complete cartilaginous rings, each shaped like a signet ring, with the broad part forming the left and right walls, alternately. These rings therefore partially overlap each other. They may be ossified in passerines and some larger species. The tracheal lumen diameter progressively reduces caudally, but is still larger than that of a comparative mammal: the typical avian trachea is 2.7 times longer and 1.29 times wider than that of comparably sized mammals. This means that the avian tracheal dead space is 4.5 times greater than that of comparably sized mammals. Birds compensate for this with a low respiratory frequency (one-third of that of mammals) and an increased tidal volume (four times that of mammals). It is important to understand this anatomy when intubating birds. Non-cuffed tubes are preferable, but if cuffed tubes are used they should not be inflated, and smaller tubes than at first appreciated are necessary to prevent iatrogenic trauma to the tracheal lining caudal to the glottis.

Most companion birds will have a tracheobronchial syrinx: the last of the tracheal rings fuse into a syringeal box, which joins to the first of the bronchial rings. (There are also tracheal and bronchial syrinxes in other species, e.g. storks and owls.) The syrinx consists of a number of variably ossified cartilages and vibrating soft structures.

The syringeal cartilages consist of:

- The tympanum. A direct continuation of the trachea, formed by the fusion of several tracheal rings. It is commonly ossified.
- The tracheal syringeal cartilages. C-shaped flattened cartilages, attached to the pessulus at one end and free at the other.
- The pessulus. A wedge-shaped cartilage with the blade lying dorsoventrally, dividing the airway vertically.
- The bronchial syringeal cartilages. Three to five paired C-shaped rings forming the divided part of the syrinx.

The vibrating structures of syrinx include:

- The paired medial tympaniform membranes that form the medial surface of the divided part of syrinx, held between free ends of bronchial syringeal cartilages.
- The paired lateral tympaniform membranes that form the membranous areas between the cartilages on the lateral aspect of the syrinx.
- The lateral labium. A pad of elastic tissue projecting into the lumen of the syrinx from the cartilage of the lateral wall.
- The medial labium projecting into the lumen from the pessulus.

The syrinx is controlled by both intrinsic and extrinsic muscles. The number of intrinsic muscles varies between species: songbirds have five pairs; parrots have only two pairs; and some ratites and Galliformes

Downloaded From Vet-eBooks.com

have none at all. There are three sets of extrinsic muscles: one pair of cleidohyoid muscles, from the clavicle to the glottis, which pulls the trachea caudally and relaxes the muscles around the syrinx; the tracheolateral muscle, from the caudal trachea to the syrinx, enclosing the trachea ventrally and laterally, which tenses the syrinx; and the sternotracheal muscle, from the craniolateral sternum to the trachea, just cranial to the syrinx, which fuses with the tracheolateral muscle.

There are two theories on how birds vocalise. The first holds that vibration of the tympaniform membranes produces sound; the second that compression of the bronchial elements against the median parts of syrinx forms narrow slots through which air is forced during expiration, causing whistling sounds.

Avian lungs are not lobed as are mammalian lungs. Approximately one-quarter of the lung volume is enclosed between ribs; avian lungs weigh about the same as those of mammals (on a weight basis), but are more compact and take up 50% as much space as in mammals. They extend from the first to the seventh rib in Psittaciformes, but may extend to the ilia in some species (**Figs 1.18a** and **b**).

Each lung receives one of the two primary bronchi, formed by the bifurcation of the trachea at the syrinx. The bronchus enters the lung ventrally and obliquely at the junction of the cranial and middle third of the lung and then passes dorsolaterally to the lung surface and turns caudally to its opening into the abdominal air sac. The bronchi have a well-developed internal, circular, smooth muscle layer and longitudinally orientated smooth muscles. Acetylcholine, pilocarpine and histamine induce contraction and atropine blocks these effects. Each primary bronchus gives off four groups of secondary bronchi:

- Mediodorsal (seven to ten). Originate from the dorsal wall of the primary bronchus and are located over the costal surface of the lung.
- Lateroventral (eight). Arise from ventral wall of the primary bronchus and are located in the ventral part of the costal surface of the lung; they enter the abdominal and caudal thoracic air sac.
- Laterodorsal (variable number). Arise from the lateral wall of the primary bronchus and extend laterally towards the costal surface.
- Medioventral (four to six). Arise from the dorsomedial wall of the cranial third of the primary bronchus and run medially on the ventral (septal) surface of the lung, servicing three-quarters of the septal surface of the lung. They are the largest of the secondary bronchi.

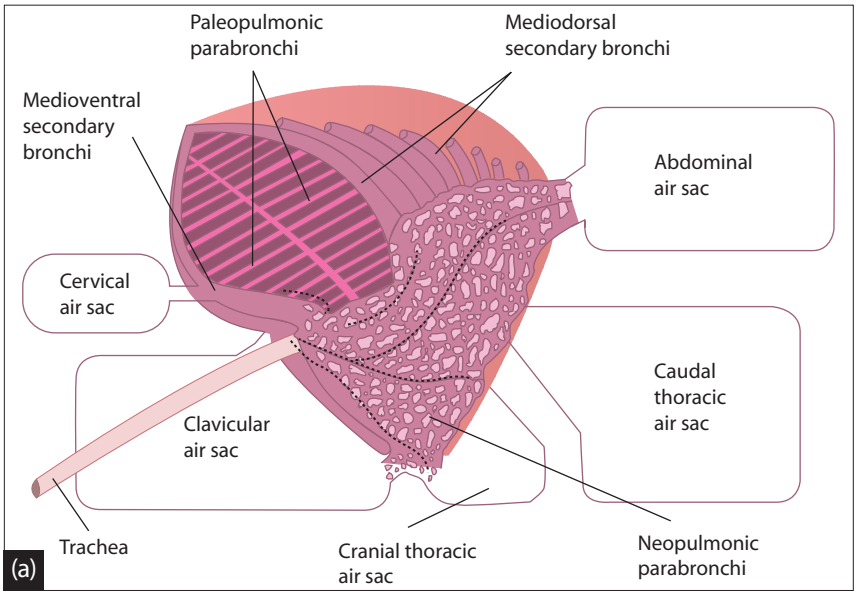


Figure 1.18 **Schematic drawings of the avian lower respiratory tract showing the key components as seen in a lateral view (a).** ***(Continued)***

Downloaded From Vet-eBooks.com

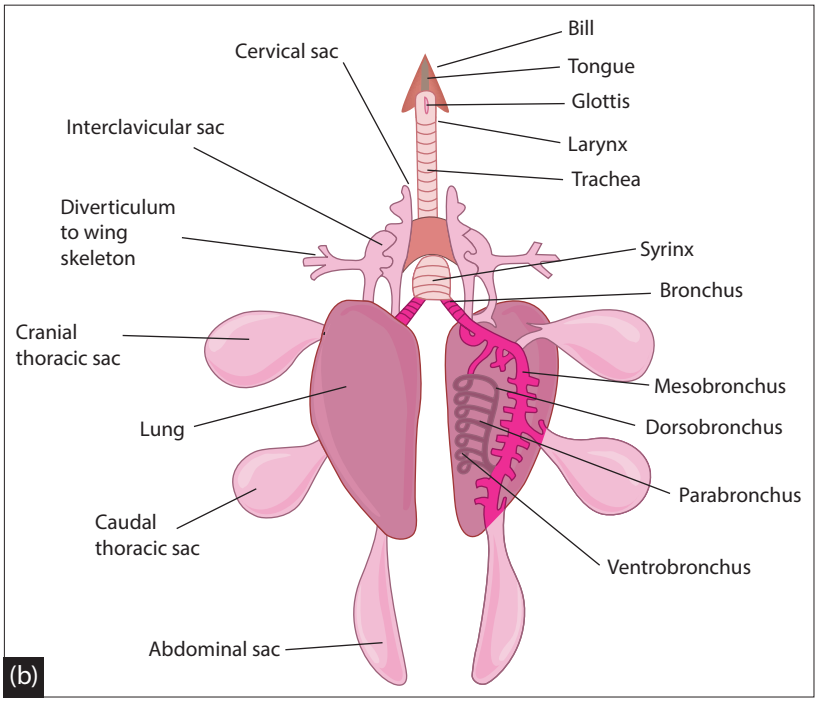


Figure 1.18 (Continued) **Schematic drawings of the avian lower respiratory tract showing the key components as seen in a ventral view (b).**

The secondary bronchi give rise to the parabronchi (tertiary bronchi), which anastomose with other parabronchi. They are divided into two groups: the paleopulmonic and the neopulmonic parabronchi. The paleopulmonic parabronchi come off the mediodorsal and medioventral secondary bronchi. They form the medioventral–mediodorsal system in the cranial and dorsal region of the lung, making up about two-thirds of the lung. Air flows unidirectionally, caudal to cranial, in this region of the lung, which is the major site of gas exchange and is more efficient than the neopulmonic lung. The remainder of the lung (ventrolateral) is the neopulmonic region. It is most advanced in chickens, pigeons and passerines; absent in emus and penguins; and minimal in storks, cormorants, cranes, ducks, gulls, owls and buzzards. Air in this region changes direction with each phase of breathing (i.e. it is bidirectional).

The parabronchi are uniform in diameter throughout the lung and lined with simple squamous epithelium. The inner lining of these parabronchi is pierced by numerous openings into individual chambers, called atria. Atria are pocket-like polygonal cavities, lined with flat or cuboidal epithelium and coated in surfactant. The openings into the atria are surrounded by smooth muscle with parasympathetic and sympathetic innervation. At the bottom of each atrium are infundibula: openings that lead to air capillaries. These air capillaries branch and freely anastomose with each other; their small diameter means that the pressure gradient for oxygen diffusion is greater than in mammals. They are intimately entwined with a network of blood capillaries, making them the site of gaseous exchange.

Extending from the lungs are the air sacs. Embryos have six pairs, two of which fuse in most birds at, or soon after, hatching to form the clavicular air sac. Adult birds, therefore, have nine air sacs: the unpaired clavicular and the paired cervical, anterior thoracic, posterior thoracic and abdominal air sacs. (Chickens and some other species fuse their cervical air sacs, leaving them with eight air sacs.)

The clavicular air sac is a large unpaired and complicated sac occupying the thoracic inlet and extending into the extrathoracic diverticula (the humerus,

Downloaded From Vet-eBooks.com

coracoid, scapula and clavicle) and the intrathoracic diverticula (around the heart and along the sternum). The first, second and third medioventral bronchi form the main connections to the clavicular air sac.

The cervical air sacs arise from the first medioventral bronchus. They form two median chambers lying between lungs and dorsal to the oesophagus, leading into a pair of vertebral diverticula on each side of the vertebral column, one inside the neural canal and one outside. They also invade the vertebrae.

The cranial thoracic air sacs arise from medioventral secondary bronchi and lie dorsolaterally in the coelom. The caudal thoracic air sacs are found caudal to the cranial thoracic air sacs and arise from lateroventral secondary bronchi and primary bronchi.

The abdominal air sacs arise from lateroventral secondary bronchi and primary bronchi and lie between the caudal thoracic air sacs. They are the most variable in size, but are often the largest air sacs. They carry air to leg and pelvic bones through perirenal and femoral diverticula.

Although the mesenteric oblique septum separates the cranial and caudal coelomic cavities, there is no muscular diaphragm to aid in respiration. Instead, birds rely on the movement of the ribs and sternum to move air through the respiratory tract. During inspiration the external intercostal muscles pull the ribs cranially, laterally and ventrally. At the same time the sternum, coracoids and furcula move ventrally and cranially, pivoting at the shoulder joint. Combined, these movements have the effect of increasing the coelomic volume. Expiration is simply a reversal of these movements, using the internal intercostal muscles and the coelomic muscles.

The air moves through the airways on a two-breath cycle (**Fig. 1.19**):

- First inspiration. Air moves through the trachea into the primary bronchus and neopulmonic region, and then into the caudal air sacs. Some air may enter the paleopulmonic region and start gaseous exchange.
- First expiration. Air moves from the caudal air sacs into the paleopulmonic region; a small volume of air (12%) escapes from the caudal air sacs through the bidirectional neopulmonic region to escape through the trachea.
- Second inspiration. Air moves from the paleopulmonic region into the cranial air sacs.
- Second expiration. Air moves from the cranial air sacs out through the bronchi and trachea; a small volume of air (12%) escapes from the caudal air sacs through the bidirectional neopulmonic region to escape through the trachea.

As mentioned earlier, gaseous exchange occurs in the air capillaries. The cross-current arrangement between parabronchial air flow and parabronchial blood capillaries in the paleopulmonic region provides a highly efficient system of gaseous exchange,

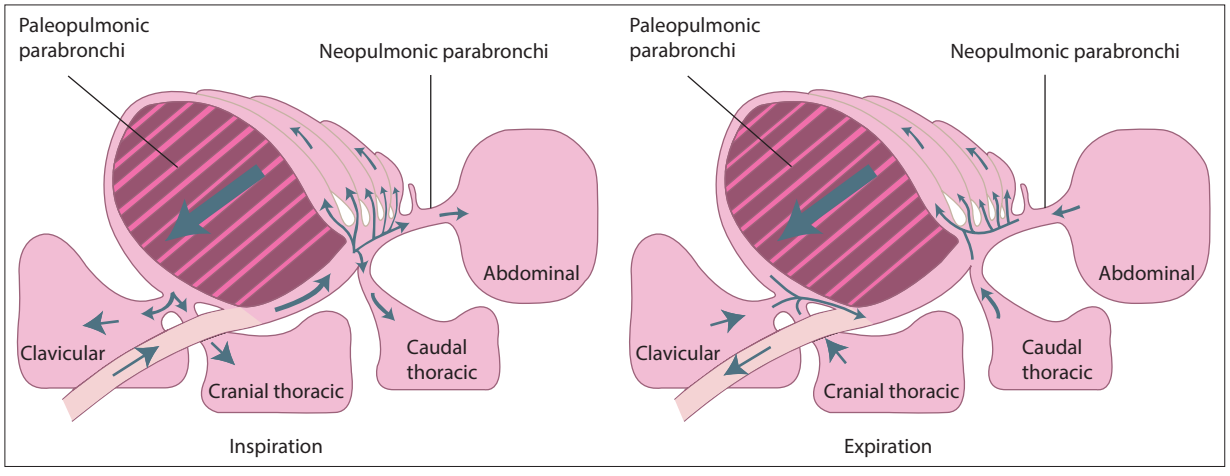


Figure 1.19 **Schematic diagrams illustrating the pattern of air flow through the respiratory tract during inspiration and expiration.**

Downloaded From Vet-eBooks.com

so efficient that birds need less ventilation to achieve a higher level of oxygenation of blood than mammals.

THE REPRODUCTIVE TRACT

Female reproductive tract

The avian embryo has two ovaries and two oviducts. During incubation the left gonadal region receives more germ cells than the right, leading to asymmetrical development. The right ovary and oviduct usually regress, so most birds have only a left ovary and oviduct (with the exception of the kiwi and some raptors) (**Fig. 1.20**).

The ovary is located beside the cranial division of the left kidney, adjacent to the adrenal gland.

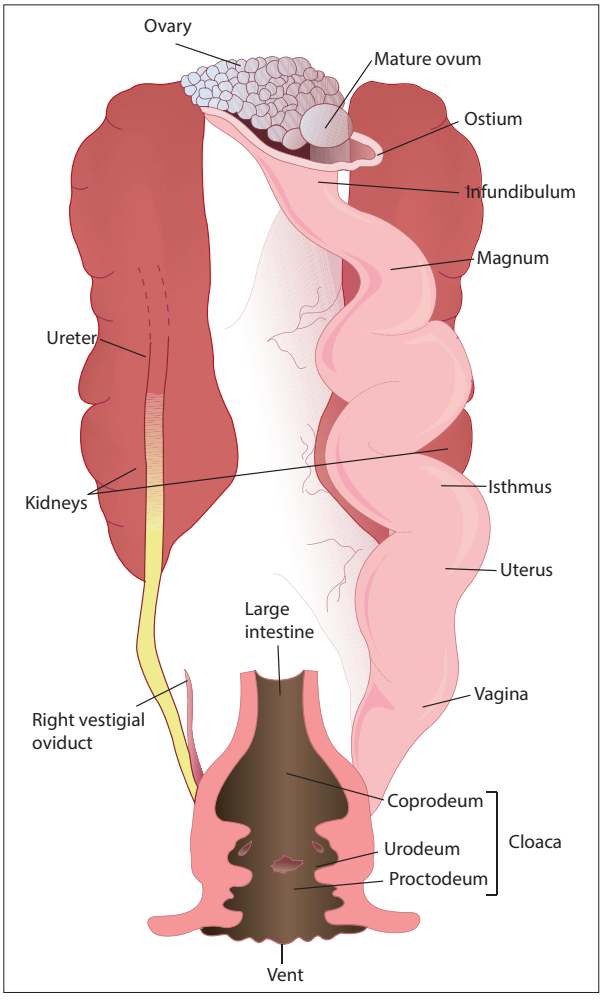


Figure 1.20 **Schematic diagram of the female reproductive tract viewed ventrally.**

The ovarian blood supply enters the ovarian hilus where it is in close contact with the dorsal coelomic wall. The arterial supply comes from the ovario-oviductal branch of the left cranial renal artery, while venous drainage is via two ovarian veins directly into the caudal vena cava. This vascular anatomy makes ovariectomy a difficult and dangerous procedure to undertake, requiring optical magnification and specialised ligating instruments.

In seasonal laying birds (e.g. parrots) three phases of ovarian growth can be recognised:

- Prenuptial acceleration. At the beginning of the breeding season the ovary begins to enlarge.
- Culmination phase. Ovulation and egg laying commences.
- Refractory phase. With egg laying completed, the ovary reduces in size.

Each follicle, containing an oocyte surrounded by the wall of the follicle, is suspended by stalk, which possesses smooth muscle, blood vessels and nerves. The wall of the follicle is very vascular and innervated with cholinergic and adrenergic fibres. Running across the surface is the stigma, a meridional band, which is less vascular and has no connective tissue or smooth muscle. Vitellogen or yolk, consisting of protein and lipid, is synthesised in the liver (vitellogenesis) and enters the follicle as it develops.

During ovulation the wall of the follicle splits along the stigma, releasing the yolk and oocyte. The follicle then shrinks to a thin-walled sac, which quickly regresses and is absorbed. No corpus luteum is formed, although the regressing follicle may secrete progesterone for the first 24 hours, affecting oviposition and nesting behaviour.

The oviduct can be divided into five regions: the infundibulum, the magnum, the isthmus, the uterus (shell gland) and the vagina.

The infundibulum resembles a funnel with a thin wall; its opening, an elongated slit, faces into the ovarian pocket formed by the left abdominal air sac. The infundibulum tapers rapidly into a tubular part (the chalaziferous region) with a thickened wall with higher mucosal folds. The infundibulum is reasonably motile, moving to envelop the developing follicle and capturing it as ovulation occurs. Fertilisation

Downloaded From Vet-eBooks.com

occurs in the infundibulum before albumen (thick albumen immediately around the yolk, and the chalaza at each end of the yolk) is laid down by the chalaziferous glands in the tubular region. The egg passes through the infundibulum in 15 minutes.

From the infundibulum the developing egg passes into the magnum, the longest and most coiled part of the oviduct. This transition is marked by a sudden great enlargement of the mucosal folds. There are numerous tubular glands in these folds, which secrete albumen. Passage through the magnum takes three hours, during which time the egg acquires albumen, sodium, magnesium and calcium.

The next part of the oviduct, the isthmus, is short and reduced in calibre, with folds less prominent than those in the magnum. After a short band of demarcating tissue without glands, the wall of the isthmus has tubular glands similar to those of the magnum. Passage through the isthmus is slow, taking 75 minutes, during which protein is added to the albumen and the shell membranes (inner and outer) are added.

There is no distinct separation between the isthmus and the uterus (shell gland). This part is relatively short, but divided into two areas, the initial short, narrow 'red region' and a larger pouch-like region. In the uterus the longitudinal folds are transected by transverse furrows, forming leaf-like lamellae. The egg stays in the uterus for 20 hours: plumping (the addition of watery solutions) occurs in the first eight hours, and then the egg shell is formed and calcified over another 15 hours.

The vagina, S-shaped due to smooth muscle and connective tissue, is separated from the uterus by a sphincter. The mucosal folds of the vagina are thin and low and it has a thick muscle wall. There are no secretory glands; however, near the sphincter are the spermatic fossulae, crypts that act as a storage site for sperm for up to several weeks. Immature birds have a membrane covering the entrance of the vagina into the cloaca; tearing of this membrane can account for the presence of blood on the shell of the first egg laid.

The oviduct is suspended from the dorsal wall of the coelom by the dorsal mesosalpinx. A ventral mesosalpinx extends ventrally from the oviduct, but has a free margin. Smooth muscle in both ligaments is continuous with smooth muscle layers of the oviductal wall and caudally the smooth muscle in the ventral ligament condenses into a muscle chord fused with the ventral surface of the uterus and vagina. These ligaments may help to move the egg along the oviduct, especially in the magnum.

As with the ovary, there is marked seasonal growth and differentiation of the oviduct under the influence of the neuroendocrine system.

Egg

Lying on the surface of the yolk is the germinal disc containing the blastoderm (if fertilised) or the blastodisc (if unfertilised). Underlying the germinal disc is the 'white yolk' or latebra, which is less dense than the yellow yolk and therefore will always be uppermost regardless of the orientation of the egg. It is made up of protein (two-thirds) and fat (one-third). The yellow yolk (two-thirds fat and one-third protein) is encased in four layers of yolk membrane, which, while mechanically strong, forms a water- and salt-permeable membrane between the yolk and albumen.

Albumen is less viscous than yolk and it contains protein (ovomucin). The amount of ovomucin determines whether it is dense or thin albumen. There is a dense chalaziferous layer around the yolk, which is continuous with the chalazae at each end of the egg that merge with the shell membranes. The chalazae therefore suspend yolk in the middle of the egg (**Fig. 1.21**). Beyond this chalaziferous layer are three

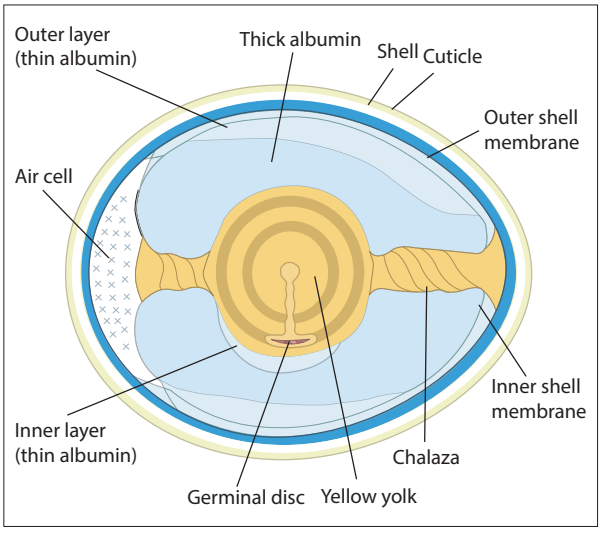


Figure 1.21 **Labelled diagram of a cross-section of an egg.**

Downloaded From Vet-eBooks.com

more layers of albumen: thin inner and outer layers, and a dense layer between them. Albumen contributes to the aqueous environment of the embryo, has antibacterial components, and is a source of nutrition for the embryo.

The egg shell has three layers: the shell membranes, the testa and the cuticle. There are two shell membranes, each composed of several layers of fibre. The inner layer is fused to the chalazae as described above. The outer layer is fused to the testa. They separate at the blunt end to form the air cell. The testa is made up of an organic matrix of fine fibres and an inorganic solid component of calcite (crystalline calcium carbonate). The organic matrix is made up of a thin inner mamillary layer, containing conical knobs embedded in the outer shell membrane, and a thick outer spongy layer. The inorganic component has a thin inner layer corresponding to the mamillary layer and a thick outer palisade layer corresponding to the spongy layer. Pores run through all the layers, through which gaseous and water exchange occurs. Surrounding the entire shell is the outer cuticle, a continuous organic layer that reduces water loss and is somewhat resistant to bacteria. It also has a water repellent effect. Not all species have a cuticle on their shells.

Male reproductive tract

Like the female embryo, the male embryo initially develops a larger left testicle. Unlike the right ovary, however, the right testicle does not regress, so that while the left testis is often larger than the right in the immature bird, this changes after maturity so that both are similarly sized. Suspended by the mesorchium, the testicles are surrounded, but not cooled, by the abdominal air sacs. The bulk of the testis is made up of thousands of convoluted seminiferous tubules with numerous anastomoses. There is no lobulation as is seen in mammals, as there are no septa present. The size of the testicle increases with sexual activity due to increased length and diameter of the seminiferous tubules and a greater number of interstitial cells. The testicle is covered in the tunica albuginea, but there is no pampiniform plexus. The seminiferous tubules are lined by spermatogenic epithelium made up of germ cells and sustentacular cells (Sertoli cells), which provide mechanical support for the germ cells, produce steroid hormones and may have a phagocytic role. Between the tubules are the interstitial cells (cells of Leydig), which produce androgenic hormones, especially testosterone. There may also be melanocytes present in the interstitial spaces of some species, giving the testis a black coloration.

There are three phases of spermatogenesis: the multiplication of spermatogonia; their growth into primary spermatocytes; and then the maturation of primary spermatocytes into secondary spermatocytes and then spermatids, which then develop into spermatozoa. These mature spermatozoa detach and pass through a short straight tubule into the rete testis, a thin-walled irregular channel on the dorsomedial aspect of the testis, adjacent to the epididymis. (The rete testis is not present in all species.)

The epididymis lies on the dorsomedial side of the testis and is relatively small compared with that in mammals. It enlarges during sexual activity, but has no distinct head, body and tail because the efferent ductules, arising from the rete testis, enter along its entire length. They lead into connecting ductules and finally into the epididymal duct.

The ductus deferens runs from the epididymis to the cloaca, entering the cloaca at the urodeum (**Fig. 1.22**). At the urodeum it enters the receptacle of the ductus deferens, a spindle-shaped dilation embedded in the cloacal muscle. In passerines the caudal end of the ductus deferens forms a mass of convolutions called the seminal glomus. This enlarges in the breeding season to push into the cloaca, forming the cloacal promontory, which pushes the vent caudally. This is the main site of spermatozoal storage in these birds.

Reproductive physiology

Most birds are seasonal breeders, with substantial variation between species in their reproductive strategies. This variation is based on the environmental cues used to trigger reproduction, the developmental stage of the chick at hatch and the extent of parental care.

Mechanisms controlling this breeding seasonality are both endogenous and exogenous. Endogenous factors are poorly understood, but are reflected in the fact that many captive birds held in constant environmental conditions still show seasonality, as do migratory birds

Downloaded From Vet-eBooks.com

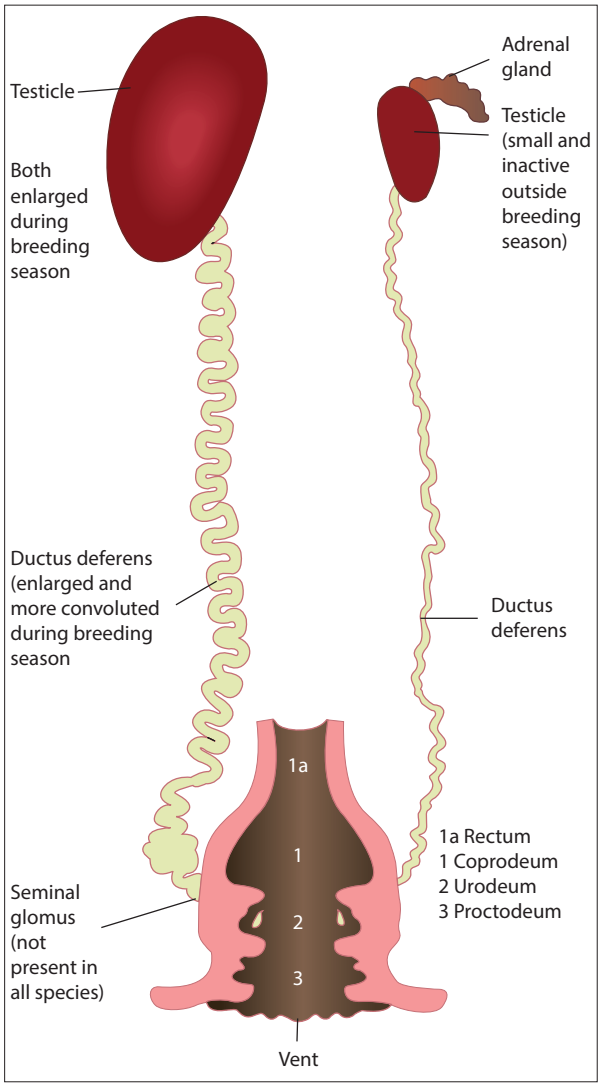


Figure 1.22 **Schematic diagram of the male reproductive tract viewed ventrally.**

going through a wide range of environmental changes and tropical birds with little variation in photoperiod. Exogenous factors are better understood. They can be either ultimate factors, which select for individuals that will breed when there are optimum conditions for offspring survival (e.g. food availability), or proximate factors that vary from year to year. These proximate factors are further broken down into:

- Initial predictive factors that initiate gonadal development in anticipation of breeding (e.g. photoperiod).
- Essential supplementary factors that supplement the initial predictive factors and initiate final stages of gonadal development. These include social cues (e.g. breeding plumage, mate availability, courtship behaviour), territorial behaviour, climate (e.g. rainfall) and nutrition (in particular an increase in fat and sugars in the diet).
- Synchronizing and integrating factors that regulate the sequence of breeding behaviour (e.g. social interaction between a pair).
- Modifying factors that can disrupt the breeding cycle (e.g. loss of a mate or disturbance of the nest site).

All of these factors have hormonal modulators. Their input into the hypothalamus has an effect on the release of gonadotrophin releasing hormone (GnRH), which in turn stimulates the pituitary to release follicle stimulating hormone (FSH) and luteinizing hormone (LH). In the hen, FSH supports ovarian and oviductal growth, gametogenesis and steroidogenesis, while LH also supports steroidogenesis. This steroidogenesis sees the release of oestrogen, which has effects on follicular and oviductal growth, calcium metabolism and vitellogenesis. Some female secondary behaviours are also influenced by oestrogen (e.g. courtship and nesting behaviours). In males FSH initiates growth of seminiferous tubules and results in increased spermatogenesis, while LH promotes development of the testosterone-producing cells of Leydig. This in turn gives rise to secondary male characteristics such as plumage changes, nesting activity, courtship behaviour and territorial behaviour.

Progesterone is produced by granulosa cells in the large follicles as they develop under the influence of LH. This in turn causes a surge in LH production from the pituitary just before ovulation. This surge of LH stimulates the production of prostaglandin (PG) F2α from ovarian follicles, causing the follicular stigma to rupture and allowing ovulation. Progesterone inhibits further ovulation and induces behavioural and physical changes associated with incubation and brood care.

PGF2α and PGE (1 and 2) are released by the F1 (first generation of follicles produced during

Downloaded From Vet-eBooks.com

ovulation) and postovulatory follicles. PGE2 and PGF2α bind at specific sites in the shell gland and vagina; PGF2α binds preferentially at the shell gland, allowing PGE2 to potentiate its effects and to allow relaxation of the vagina during oviposition. Therefore, PGE (1 and 2) allows relaxation of the uterovaginal sphincter, while PGF2α stimulates shell gland contractions. Uterine contractility stimulates AVT release from the pituitary, which stimulates further contractility and release of uterine PGs.

Eggs are successively laid until a clutch is formed: indeterminate layers continue to lay if eggs are removed, while determinate layers will only lay a set number of eggs. Incubation is performed by the hen only (in 25% of species), shared (54%), by the males only (6%) or by mixed strategies. Plasma prolactin levels are elevated in both sexes during incubation, which then has an inhibitory feedback on GnRH release.

CARDIOVASCULAR SYSTEM

General

The avian heart lies in the cranial midline of the body, within the rib cage and enclosed by the liver rather than the lungs. The basic anatomy is similar to the anatomy of the mammalian heart, with two atria and two ventricles. The right cranial vena cava and the caudal vena cava form a sinus venosus with left and right sinoatrial valves. A sinus septum separates the opening of the left crania vena cava from the openings of the other two veins. The right atrioventricular valve is muscular with no chordae tendineae, while the left atrioventricular valve is membranous (similar to mammals) but is a continuous sheet without defined cusps. The resistance to blood flow is, as with mammals, lower in the pulmonary circulation than the systemic circulation, leading to more pressure required in the left ventricle. Accordingly the left ventricular wall is thicker than the right. The aortic and pulmonary artery valves are similar to mammals, while the left and right pulmonary veins merge in the atrium to form a single vessel entering the left atrium. The aorta curves to the right, as it is derived from the right fourth arterial arch and right aorta.

The heart is enclosed in the pericardium, a semi-rigid fibrous sac that contains only a small amount of serous fluid to allow lubrication for the heart within the sac. The pericardium is adherent to the sternum, the cranial and caudal thoracic air sacs, the liver, and the hepatic peritoneum (which is, in turn, adherent to the ventral vertebral column). These multiple connections provide mechanical stability to the apex of the heart, and to several large, central blood vessels that are adherent to the pericardium. The relative rigidity of the pericardial sac acts to mechanically couple the ventricles (i.e. limit acute increases in cardiac size).

The ascending aorta gives off the coronary arteries, supplying the myocardium, and then two brachiocephalic trunks, supplying the head, wings and flight muscles. These brachiocephalic trunks give off the carotid arteries before carrying on to the wings and flight muscles. The descending aorta runs caudally and dorsally, just ventral to the vertebral column. As it does, it gives off arteries supplying the internal organs and the legs (**Fig. 1.23a**).

The arteries eventually give rise to arterioles and then capillary beds within tissue. These perfuse the tissues before entering the venous system. The avian venous system cranial to the heart differs from the mammalian system in that there are two cranial vena cavae; the right jugular vein is larger than the left, and there is an anastomosis between left and right jugular veins at the base of the head, allowing some of the venous blood to enter the larger right jugular vein (**Fig. 1.23b**).

Caudal to the heart the major features of the venous system are the renal and hepatic portal systems. Blood returning from the legs and lower intestine enters the kidneys via the external iliac veins. If the renal portal valves (in the common iliac veins) are partially closed due to parasympathetic stimulation, blood is diverted to the cranial and caudal renal portal veins and perfuses the kidneys. The cranial renal portal veins empty into the caudal vena cava via the common iliac veins. Blood flowing through the caudal portal veins eventually returns to the heart via the internal vertebral venous sinuses and the caudal mesenteric vein. On the other hand, under sympathetic stimulation the renal portal valves open and blood from the external iliac veins enters the common iliac vein and then the caudal vena cava, bypassing the kidney completely (see **Fig. 1.24**).

Downloaded From Vet-eBooks.com

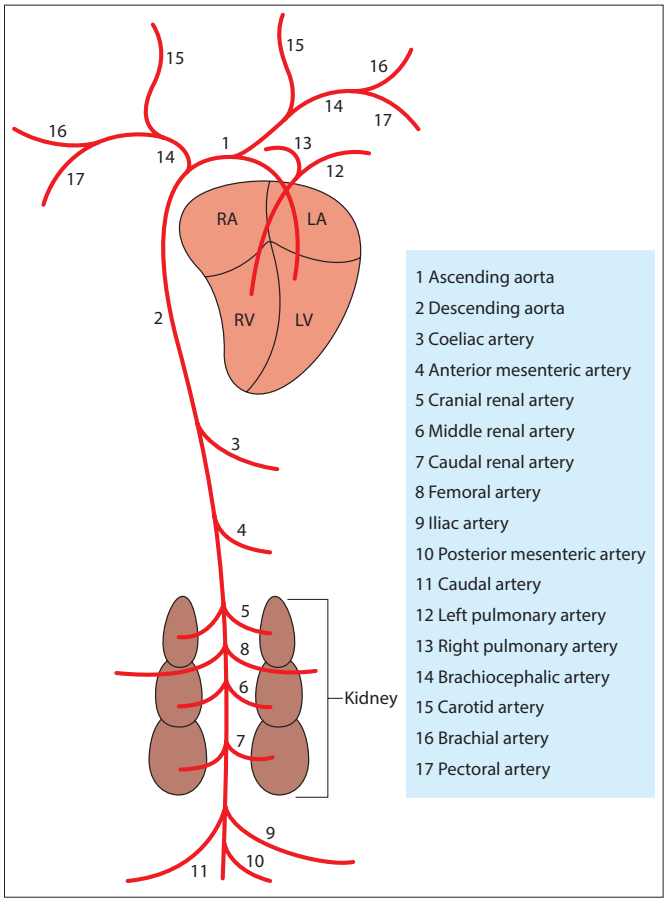


Figure 1.23 **Labelled diagrams of the major arteries (a) and minor veins (b) within the avian cardiovascular system.**

Blood returning from the legs and caudal intestinal tract in a resting bird can therefore enter either the cranial renal portal vein or the caudal renal portal vein, but not both at the same time. The control of this flow is not yet understood. Blood that enters the caudal renal portal veins will make its way into the caudal mesenteric vein and from there to the hepatic portal veins and the liver. The hepatic portal veins drain into the hepatic sinusoids, and from there to the hepatic veins. The right and left hepatic veins enter the caudal vena cava in the liver, which exits through the dorsal part of the right liver lobe to the heart.

Function

The cardiovascular system of any animal plays several major roles in the body: to deliver oxygen and metabolites throughout the tissues of the body; to remove metabolic waste products from these tissues; and to assist in thermoregulation. Birds have evolved a high-performance cardiovascular system to meet the demands imposed by flying, swimming and running.

The heart is relatively larger than that of mammals and beats rapidly (usually greater than 200 beats/minute but capable in some species, e.g. hummingbirds, of reaching 1,000 beats/minute). This is achieved by having larger numbers of myocytes with a small diameter allowing for more rapid depolarisation.

This combination of a larger heart with a fast rate gives a relatively greater cardiac output (mls/kg/minute) than mammals. When exercising (e.g. flying), birds increase their cardiac output by increasing their heart rate rather than their stroke volume (mls/beat). For example, a flying pigeon can increase its heart rate from 115 beats per minute to over 600 beats per minute. During this exercise the stroke volume decreases

Downloaded From Vet-eBooks.com

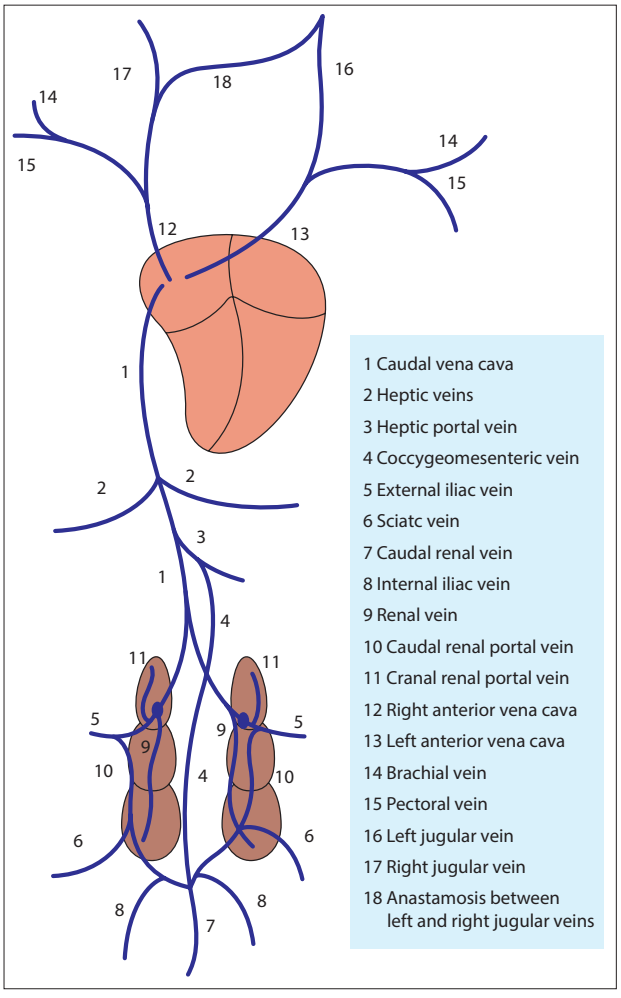


Figure 1.24 **Schematic diagram of the avian renal portal system.**

marginally, but the overall result is that cardiac output is increased more than five-fold – an output up to seven times greater that of a man or dog when all are at full exercise.

In turn, this high cardiac output requires a high arterial blood pressure (140-250 mmHg) to adequately perfuse the capillary beds under a wide range of physiological conditions. This blood pressure is maintained by the baroreflex, driven by mechanoreceptors in the arterial walls in the aorta. The primary cardiovascular response to changes in blood pressure is to alter the cardiac output by increasing or decreasing the heart rate.

Much of the heat loss in many birds is through the distal leg and feet, regulated by the rate of blood flow to these areas. In cold conditions intermittent brief periods of very great flow helps to counter the effects of frost-bite. In very hot conditions the blood flow increases as well, but allows radiant heat loss. This can increase during exercise by up to 10-15 times the amount of heat transfer when at rest.

Blood cells

Erythrocytes

Erythrocytes (**Fig. 1.25**) are elliptical in shape with a centrally located, oval nucleus. They are usually 10–15 µm in length (smaller cells are found in smaller species), compared with the typical biconcave mammalian erythrocyte, which is 6–7 µm in diameter. Typically, healthy erythrocytes have a uniform colour and size when examined in a stained blood smear. They have a relatively short half-life (28–45 days) when compared

Downloaded From Vet-eBooks.com

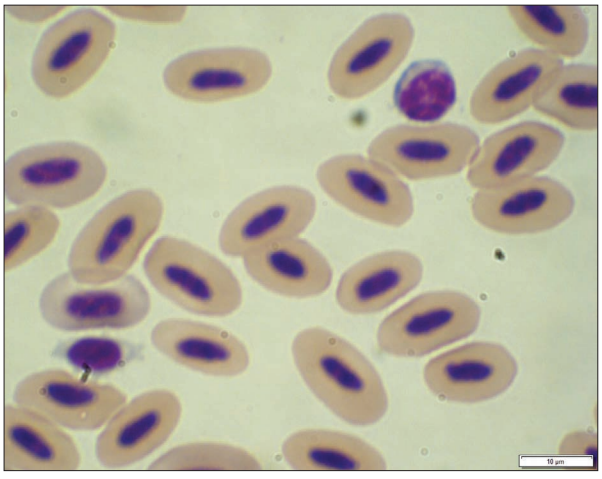


Figure 1.25 **Stained avian erythrocytes isolated from a blood sample.**

with those of mammals (120 days in man), which results in a greater number of immature erythrocytes in the peripheral circulation. The cytoplasm of these immature erythrocytes stains more basophilic than that of mature erythrocytes (polychromasia), they are non-uniform in size (anisocytosis) and the nuclear chromatin is less condensed than mature erythrocytes. In most healthy parrots they represent approximately 1–5% of the total erythrocyte count.

Erythropoiesis occurs in the bone marrow (and, in the embryonic chick, the yolk), although ectopic erythropoiesis can be occasionally found on the spleen and liver. There are seven stages of development:

- Rubriblasts are large cells with large central and round nuclei with coarse chromatin and large nucleoli. The cytoplasm is deeply basophilic with clear spaces (mitochondrial spaces).
- Prorubricytes resemble rubriblasts, but lack nucleoli and have mitochondrial spaces in the cytoplasm.
- Basophilic rubricytes have homogenous basophilic cytoplasm and round nuclei with clumped chromatin.
- Initial polychromatic rubrycites are smaller than basophilic rubrycites. The cytoplasm of these cells is basophilic to slightly eosinophilic, an indication of the beginning of haemoglobin synthesis.
- Late polychromatic rubrycites have a round to oval shape and with a more eosinophilic cytoplasm.
- Polychromatic erythrocytes resemble mature erythrocytes but are more oval in shape. Their cytoplasm is slightly basophilic and chromatin appears less condensed
- Mature erythrocytes

Erythropoiesis is controlled by a number of factors such as oxygen concentration in tissues and hormones. Hypoxia stimulates the production and release of erythropoietin from the kidneys which then stimulates erythropoiesis. Other hormones, such as androgens and adrenocorticoids, also stimulate this process. Polycythaemia, either secondary or primary, may suppress erythropoiesis.

The major function of erythrocytes is oxygen transport, but recently it has been reported that they may participate in some immune responses that contribute to host defence.

Thrombocytes

Thrombocytes (**Fig. 1.26**) are homologous with mammalian platelets. They are round to oval cells, smaller than erythrocytes, and contain an oval to rounded nucleus. The cytoplasm is light blue or colourless, often vacuolated with a few acidophilic granules. Thrombocytes are often confused with

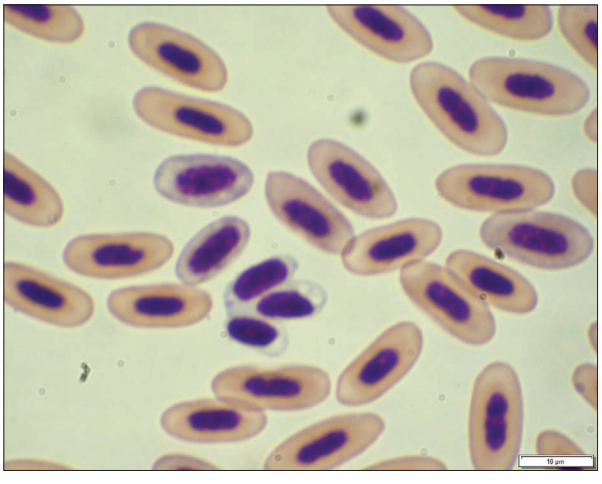


Figure 1.26 **Stained avian thrombocytes surrounded by erythrocytes. Note the cytoplasm is light blue.**

Downloaded From Vet-eBooks.com

small lymphocytes but have a tendency to clump in blood smears, particularly if non-coagulated blood is used to make the smear. Thrombocytes aggregate at the site of vascular injury and form a haemostatic plug. They may also have phagocytic abilities and probably have some function in non-specific immunity.

Heterophils

Heterophils (**Fig. 1.27**) are the avian equivalent of the neutrophil. They are rounded cells with a polymorphic nucleus (2–3 lobes) and eosinophilic rod-shaped granules within a clear cytoplasm. They are highly mobile, phagocytic, and play an important role in the body's defence mechanisms, responding to a chemotactic response within 30 minutes. Their numbers can rapidly increase during mild to moderately stressful conditions and consequently the heterophil:lymphocyte ratio can be used as an indicator of this stress. Severe stress, however, may cause a heteropaenia.

Lymphocytes

Lymphocytes (**Fig. 1.28**) may be seen in three sizes: small, medium and large. The larger cells may be less mature than the smaller lymphocytes. They are usually round in shape, although on a smear they may appear irregular in shape as they mould to surrounding cells. The nucleus:cytoplasm ratio is high, with the cytoplasm often appearing as a narrow pale blue band around the nucleus. Certain antigenic stimuli (e.g. chlamydial infections) may induce a reactive change, where the cytoplasm darkens in colour and the nucleus appears immature. Droplets of cytoplasm may protrude from the cell membrane. Lymphocytes are involved in cell-mediated and humoral immunity.

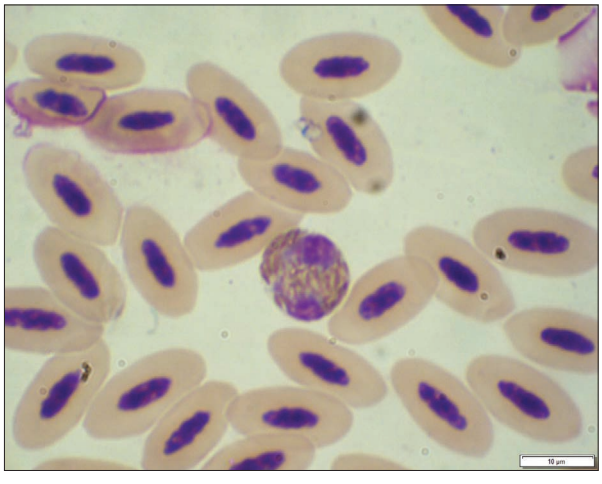


Figure 1.27 **A stained avian heterophil surrounded by erythrocytes.**

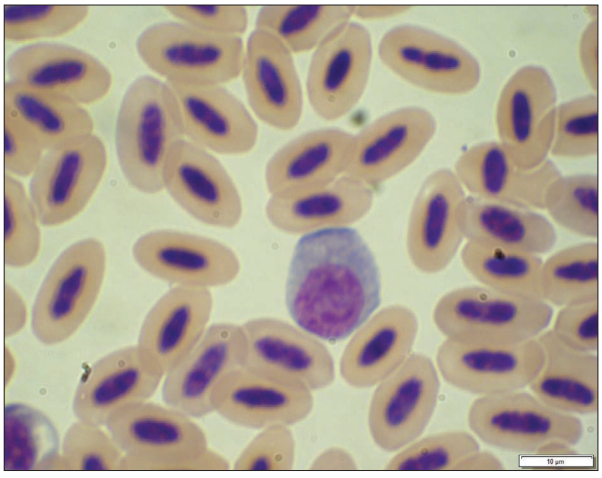


Figure 1.28 **A stained avian lymphocyte surrounded by erythrocytes.**

Eosinophils

Eosinophils are round cells with a bi-lobed nucleus and numerous eosinophilic spherical granules in a pale blue cytoplasm. Their role is unclear. Unlike mammals, their presence does not indicate parasitism. Number may increase with soft tissue injuries.

Monocytes

Monocytes (**Fig. 1.29**) are round or irregular cells with an indented 'bean-shaped' central nucleus. The cytoplasm is finely granular, with a blue-grey appearance. They may be easily confused with large lymphocytes. Monocytes are generally uncommon in peripheral blood. A monocytosis is suggestive of chronic infections such as chlamydiosis.

Basophils

Basophils (**Fig. 1.30**) are round cells with a round, centrally-located nucleus. The cytoplasm contains numerous, deeply basophilic granules that often mask the nucleus. In birds basophils appear to play an important role in early inflammatory

Downloaded From Vet-eBooks.com

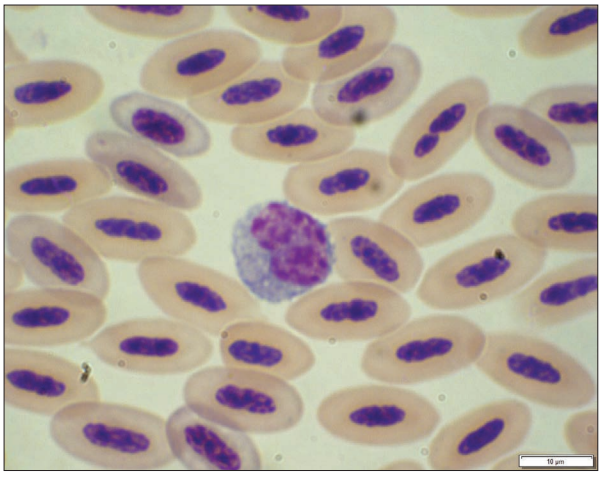


Figure 1.29 **A stained avian monocyte surrounded by erythrocytes. Note the indented 'bean-shaped' nucleus.**

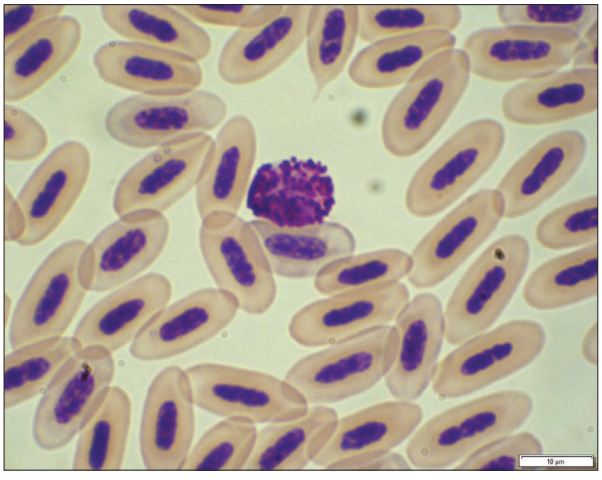


Figure 1.30 **A stained avian basophil surrounded by erythrocytes.**

and immediate hypersensitivity reactions, but differ from those in mammals by not contributing to delayed hypersensitivity.

Granulopoiesis in birds seems to follow similar stages to those seen in mammals i.e. myeloblast, promyelocyte, myelocyte, metamyelocyte and granulocytes. While the bone marrow is the primary site of granulopoiesis, other organs such as the spleen, liver, kidney, lungs, thymus, gonads, and pancreas may also be involved. Lymphopoiesis, occurring in the bursa, thymus and spleen, has three stages: the lymphoblast, the prolymphocyte, and the lymphocyte.

NERVOUS SYSTEM

The brain

In some ways the avian brain is similar to that of the mammalian brain, particularly in the hindbrain (rhombencephalon) and the midbrain (mesencephalon). Evolutionary pressures have seen the greatest differences develop in the forebrain (telencephalon and diencephalon), especially in the size and position of the neocortex. Mammals have evolved a large superficial neocortex, whereas in birds it is much smaller and deep within the cerebral hemisphere.

The hindbrain (rhombencephalon), a continuation of the spinal cord, is composed of a large medulla oblongata, a small pons, and the cerebellum. The medulla oblongata contains the cardiac, respiratory, vomiting and vasomotor centers and so deals with the autonomic (involuntary) functions of breathing, heart rate and blood pressure. Cranial nerves XII to V (inclusive) arise from the ventral and lateral aspects of the medulla. The cerebellum is attached to the dorsal aspect of the medulla and the optic lobe (below) by two cerebellar peduncles. It consists of a large median tri-lobed body (the vermis) flanked by two smaller cerebellar hemispheres. The cerebellum in many species is relatively much larger than that of mammals, reflecting the need for precise co-ordination when flying and when using a beak and tongue to manipulate objects.

The midbrain (mesencephalon) consists largely of the mesencephalic (optic) tectum, or optic lobe. This optic lobe is relatively much larger than that of mammals, reflecting the importance of vision to birds. Two cranial nerves (III and IV) arise from the ventral side of the midbrain, and the cerebellum is attached to the midbrain by the cranial cerebellar peduncle

The diencephalon is an extension of the midbrain. On the dorsal aspect the pineal gland arises from the epithalamus and sits in the triangle formed by the two cerebral hemispheres and the cerebellum. The pineal gland responds to light via the eyes and the brain, and plays a role in diurnal and season rhythm in birds. Ventrally the optic chiasma leads to the left and right optic tracts; immedialtely caudal to this is the ventral surface of the hypothalamus and the hypophysis. The thalamus lies within the diencephalon and acts as a relay for afferent pathways ascending into the cerebral hemispheres. As such it plays a role in vision and

Downloaded From Vet-eBooks.com

hearing, as well as other functions. The hypothalamus lies within the ventral portion of the diencephalon, continuous with the neurohypophysis and then the hypophysis. It dominates all autonomic functions such as thermoregulation, respiration, circulation, thirst, appetite, reproduction and behaviour.

The telencephalon consists of the cerebral hemispheres and the olfactory bulb. The olfactory bulb is relatively small, and protrudes from the rostral forebrain. The left and right cerebral hemispheres, separated dorsally by the median fissure, are almost smooth (particularly when compared to the mammalian brain). The thin cortex overlies a large amount of grey matter which forms the bulk of the hemisphere. (In contrast, the mammalian cortex is much thicker.) Caudally the cerebral hemispheres overlap the optic lobe of the midbrain. The cerebral hemispheres play a key role in the development of complex behaviours in birds, especially those involving learning.

The brain is enclosed by the same three meninges enclosing the spinal cord i.e. the dura, arachnoid and pia maters. The dura is attached to the periosteum except where the dural venous sinuses separate the dura and periosteum. Dural folds separate the forebrain from the optic lobes and the optic lobes from the cerebellum. The subarachnoid space expands slightly at the base of the cerebellum dorsal to the medulla oblongata, forming the cisterna magna. Although CSF can be obtained from this cistern at the foramen magna, this procedure is extremely hazardous due to the proximity of the vertebral venous sinus.

Cranial nerves

As with mammals, birds possess 12 pairs of cranial nerves.

1. ***Olfactory nerve* (I):** a sensory nerve connecting the nasal epithelium to the olfactory bulb of the forebrain via the olfactory foramen (there is no cribriform plate as there is in mammals). Both nasal cavities are innervated by separate branches which combine into a single nerve at the nasal bone; this then runs along the dorsal border of the inter-orbital septum to the olfactory foramen and then the olfactory lobe.
2. ***Optic nerve* (II):** a sensory nerve from the retina to the optic chiasma, the optic nerve is the largest of the cranial nerves, again reflecting the visual natures of birds. It penetrates the skull at the optic foramen, decussates at the optic chiasma and continues as the optic tract to the thalamus. The optic nerves are relatively short, and care must be taken when enucleating one eye that the optic nerve of the other eye is not avulsed accidentally.
3. ***Oculomotor nerve* (III):** a motor nerve arising from the midbrain and exiting the cranium either via the optic foramen or the oculomotor foramen. After exiting the nerve divides into a dorsal branch (upper eyelid levator muscle and the dorsal rectus muscle) and a ventral branch (ventral rectus, medial rectus and ventral oblique muscles). The ventral branch also gives off the iridociliary nerve which, after receiving a branch of the trigeminal nerve (V), provides parasympathetic innervation to the iris and ciliary body.
4. ***Trochlear nerve* (IV):** a motor nerve arising from the dorsal midbrain, exiting through the trochlear foramen into the orbit and innervates the dorsal oblique muscle
5. ***Trigeminal nerve* (V):** a nerve arising from the brainstem at the caudal edge of the optic lobe. It divides almost immediately into the ophthalmic nerve and the combined maxillary and mandibular nerves. The *ophthalmic nerve* is the main sensory nerve of the nasal cavity and the wall of the eye ball. It supplies the iris and ciliary body (along with the Oculomotor nerve III), the nasal mucosa and the edge and tip of the upper beak, the palate, the upper eyelid and the adjacent skin, and the rostral nasal cavity. The combined maxillary and mandibular nerves separate either before or immediately after exiting the skull through the maxillaomandibular foramen and sometimes the mandibular foramen. The *maxillary nerve*, also a sensory nerve, divides into three branches: the supraorbital nerve supplying the upper eyelids and crest; the infraorbital nerve supplying the lower eyelids; and the nasopalatine nerve supplies the lacrimal glands and the glands of the nasal mucosa. The *mandibular nerve*, primarily a sensory nerve, innervates the muscles of mastication, the skin along the mandible, and the floor of the oral cavity.

Downloaded From Vet-eBooks.com

6. ***Abducent nerve* (VI):** this motor nerve arises from the rostral medulla oblongata and exits the skull rostrally through the abducent foramen. It innervates the lateral rectus muscle and the nictitating membrane muscles.
7. ***Facial nerve* (VII):** this primarily motor nerve arises from the ventrolateral medulla oblongata, enters the internal acoustic meatus and then passes along the facial canal. As it exits the facial nerve foramen it divides into the palatine nerve and the hyomandibular nerve. The *palatine nerve* divides into a dorsal branch (ethmoidal ganglion, innervating the nictitating membrane gland, salt gland, and nasal glands) and a ventral branch (sphenopalatine ganglion, innervating nasal glands and caudal palate). The *hyomandibular nerve* innervates depressor muscles of the mandible and the stylohyoid muscle.
8. ***Vestibulocochlear nerve* (VIII):** a sensory nerve arising from the medulla oblongata, this nerve is divided into the *vestibular part* and *the cochlear part*. It receives sensation from the ear and vestibular apparatus.
9. ***Glossopharyngeal nerve* (IX):** this nerve arises from the ventrolateral medulla oblongata with the vagus and accessory nerves. It divides into three branches: the *lingual nerve* (sensory from the epithelium of the tongue and the taste buds, motor to the sublingual salivary gland); the *laryngopharyngeal nerve* innervates the muscles of the larynx and the pharynx; and the *descending oesophageal nerve* innervates the cervical oesophagus and trachea.
10. ***Vagus nerve* (X):** this nerve arises from the medulla oblongata and exits the skull through the foramen. It runs down the neck alongside the jugular vein and, after passing through the thoracic inlet, divides into: the *nerve to the carotid body*; *glandular filaments* to the thymus, thyroid, ultimobranchial gland, and parathyroid gland; an *aortic nerve*; the *cranial cardiac nerve*; the *recurrent nerve*, which innervates the crop, oesophagus, tracheal muscles and syringeal muscles; the *pulmoesophageal nerve* (oesophagus and lungs); *pulmonary rami* (lungs); and the *caudal cardiac nerves*. The left and right vagal trunks then unite to innervate the proventriculus, ventriculus, duodenum, and liver.
11. ***Accessory nerve* (XI):** this nerve arises from both the medulla oblongata and the cranial spine. The spinal part passes rostrally through the foramen magnum and anastomoses with the rest of the nerve, which then exits the skull with the vagus nerve. It innervates the cucillaris capitus muscle (the avian equivalent to the trapezius muscle).
12. ***Hypoglossal nerve* (XII):** arising from the ventral medulla oblongata and exiting via the hypoglossal foramen, this nerve anastomoses with the first cervical spinal nerve to form the *hypoglossocervical nerve*, and then with the vagus and glossopharyngeal nerves. It then divides into the descending cervical nerve (tracheal muscles), the laryngolingual ramus (tongue muscles) and the tracheal ramus (syringeal muscles). This is a motor nerve.

The spinal cord

The internal anatomy of the spinal cord in birds is similar to that of mammals, with a central butterfly of grey matter surrounded by white matter. Unlike mammals, the avian spinal cord is the same length as the neural canal, with no *cauda equina* at its termination. The dorsal and ventral roots of the spinal nerves pass separately through the dura, coming together in the intervertebral foramina and exiting laterally rather than caudally. The cord diameter increases at the cervical enlargement (associated with the brachial plexus) and the lumbosacral enlargement (associated with the lumbosacral plexus). The cervical enlargement is greater than the lumbosacral enlargement in flying birds such as parrots, but smaller in non-flighted birds such as poultry and ratites. The dorsal column of white matter in the lumbosacral enlargement is divided sagitally by the rhomboidal sinus, a cleft occupied by the gelatinous body (glial cells rich in glycogen); its function remains unclear. There may be a similar glycogen body in the brachial region.

The spinal cord is enclosed, protected and supported by three meninges–the dura, the arachnoid and the pia mater. The *dura mater* is the thick outer meninge; initially fused with the periosteal lining of the vertebral canal at the foramen magna, it is then separated from the periosteum by an epidural space extending along the cervical and notarial region. This space is filled with a gelatinous material and,

Downloaded From Vet-eBooks.com

on the dorsal side, the vertebral venous sinus. The dura then fuses with the periosteum from the caudal notarium to the caudal end of the canal, although the venous sinus intervenes irregularly between the dura and the periosteum. The *arachnoid mater* is a thin, delicate layer lying in close proximity to the dura, while the *pia mater* is attached to the spinal cord. The space between these two meninges is the subarachnoid space, occupied by fine denticulate ligaments suspending the spinal cord from the dura and a small amount of cerebrospinal fluid (CSF).

The spinal cord derives its blood supply from the vertebral arteries (cervical and cranial notarial regions) and intersegmental branches of the descending aorta (from the level of the heart caudally). Coming off the intersegmental arteries are the vertebromedullary arteries, which divide and penetrate the dura. These arteries also feed into three longitudinal arteries (the ventral spinal artery and two dorsolateral spinal arteries). The dorsolateral spinal arteries supply the white matter on the dorsal cord, while the ventral spinal artery gives off branches (the sulcal arteries) supplying the bulk of the cord. Venous drainage is via the vertebral venous sinus.

Spinal nerves

The spinal nerves emerge laterally between the vertebrae, with the total number varying between species (due to the species-variance in the number of vertebrae). Each nerve has a dorsal (afferent) and a ventral (efferent) root, which combine to form a mixed spinal nerve. The size of the spinal roots and nerves is determined by the relative importance of the nerves (e.g. in flying birds the nerves in the brachial plexus region are larger than those in the lumbosacral plexus). Each spinal nerve divides into three: the meningeal branch (innervating the spinal meninges; the dorsal ramus (innervating the epaxial muscles and dorsal skin); and the ventral ramus (innervating the hypaxial muscles and ventral skin, as well as forming the brachial and lumbosacral plexuses).

The nerves of the wing arise from the brachial plexus, formed by the ventral rami of 4–5 notarial spinal nerves, known as the roots of the plexus. These roots combine to form 2–3 short trunks of the plexus, which then divide into a dorsal and ventral division. The dorsal divisions unite to form a dorsal fascicle and the ventral divisions form a ventral fascicle. The dorsal fascicle forms the peripheral nerves that innervate the dorsal (extensor) muscles of the wing; these nerves include the radial nerve and the axillary nerve, and serve to elevate the wing. The ventral fascicle innervates the ventral (flexor) muscles and include the large pectoral nerves, the medioulnar nerve, the ulnar nerve, and the median nerve; these nerves serve to lower the wing. The ventral flexor also gives rise to the supracoracoid nerve which activates the upstroke of the wing through its innervation of the supracoracoid muscle.

The lumbosacral plexus is formed by the ventral rami of eight spinal nerves and is divided into the lumbar plexus (three roots, one of which is shared with the sacral plexus) and the sacral plexus (six roots, including one from the lumbar plexus). These plexuses then give rise to the peripheral nerves of the leg – the femoral nerve and its branches come off the lumbar plexus and the sciatic nerve comes off the sacral plexus. The roots of the lumbar plexus are in contact with the dorsal surface of the cranial division of the kidney, while the roots of the sacral plexus and the start of the sciatic nerve are embedded in the medial division. Compression of these nerves by renal enlargement will result in paresis or paralysis of the affected leg.

The pudendal and caudal plexuses supply the nerves which innervate the tail, vent sphincter, and cloaca. The pudendal plexus is located in region of, and within, the caudal renal division.

Autonomic nervous system

The autonomic nervous system acts as a control system functioning largely below the level of consciousness. It affects heart rate, digestion, respiration rate, salivation, pupillary size, and other functions. Whereas most of its actions are involuntary, some, such as breathing and pupillary size, work in tandem with the brain and peripheral nerves. The autonomic nervous system in birds, as with mammals, has two divisions: the craniosacral (parasympathetic) system which serves to conserve body resources; and the thoracolumbar (sympathetic) system which induces physiological responses appropriate to flight.

Downloaded From Vet-eBooks.com

These work together to preserve homeostasis of the internal organs via dual innervation which is both afferent and efferent, eliciting a specific organ function or response (e.g. cardiac function, reproductive activity, gastrointestinal integrity, renal function, pain response, and pancreatic and hepatobiliary function).

Parasympathetic system

The cranial division of this system is formed by cranial nerves III, VII, IX and X. They distribute efferent pathways to the eye, the glands of the orbit, the nasal cavity, the salivary glands, the heart, the lungs, and the digestive tract as far as the duodenum. The sacral division is formed by the pudendal spinal plexus and pudendal nerve and supplies the rest of the digestive tract, the urogenital tract, and the cloaca. The parasympathetic system is mediated by the neurotransmitter acetylcholine, and is primarily concerned with conserving and restoring a steady state in the body.

Sympathetic system

This system is based on a chain of paravertebral ganglia (the sympathetic chain) running from the base of the skull to the tail. The first (and largest) ganglia distributes sympathetic fibres to the eye, the glands of the head, and many of the large vessels in the head. These ganglia give off the cardiac nerve, the splanchnic nerves and the intestinal nerve. The sympathetic system is mediated by catecholamines (epinephrine, norepinephrine and dopamine), and is concerned with 'fight or flight' response.

ENDOCRINE GLANDS

Pituitary

Also known as the hypophysis, the pituitary gland is attached to the ventral surface of the diencephalic part of the brainstem (the hypothalamus) immediately caudal to the optic chiasma. It has two components: the adenohypophysis, arising from embryonic stomodaeum; and the neurohypophysis, arising from the diencephalon.

The adenohypophysis has only two components; there is no pars intermedia as in mammals. The pars tuberalis is the smaller part of the adenohypophysis and covers part of the neurohypophysis, carrying portal vessels from there to the pars distalis. The pars distalis makes up the bulk of the adenohypophysis, lying ventral and rostral to the neurohypophysis. Seven types of secretory cells have been identified: alpha, beta, gamma, delta, epsilon, eta and kappa. They secrete at least seven hormones:

- FSH (beta cells). Stimulates ovarian follicular growth and secretion of oestrogen by the ovary; in males stimulates tubular growth of the testes and spermatogenesis.
- Thyroid-stimulating hormone (TSH) (delta cells). Controls the thyroid gland; under the control of thyrotropin releasing hormone (TRH).
- LH (gamma cells). Causes ovulation; in males stimulates interstitial cells to produce androgens. Controlled by luteinizing hormone releasing hormone (LHRH).
- Prolactin (eta cells). Causes broodiness (perhaps by suppressing release of the gonadotrophin hormones FSH and LH). Prolactin increases with norepinehprine, serotonin and histamine; it also produces hyperglycaemia and stimulates hepatic lipogenesis. Broodiness can be terminated by oestrogen (chickens) suggesting that oestrogens prevent release of prolactin from the pituitary.
- Somatotropic hormone (STH) (alpha cells). Regulates body growth. Also known as growth hormone.
- Adrenocorticotrophic hormone (ACTH) (epsilon cells). Regulates adrenal corticosteroid production. Presumably released when corticotropin releasing factor (CRF) is released. Stimulates adrenal cortical cells to produce and release corticosterone and other glucocorticoids.
- Melanotropic hormone (MSH) (kappa cells). Function unknown.

Releasing factors are formed in the hypothalamic nuclei and travel to the median eminence in some of the axons of the hypothalamohypophyseal tract. From there they enter the primary capillary plexus and, via the portal vessels, enter the secondary capillary plexus in the pars distalis and cause these cells to release their hormones.

Downloaded From Vet-eBooks.com

Thyroid

The paired thyroid glands are located on either side of the trachea on the ventral–lateral aspect of the neck just above the thoracic inlet and adhering to the common carotid artery just above the junction of the common carotid with the subclavian artery. They are medial to the jugular vein.

The gland is encapsulated by reticular connective tissue. Its follicles are composed of a single layer of endodermal epithelium of varying height, depending on the state of activity (secretory rate). Depending on the secretory state, the follicles may be filled with, or completely devoid of, colloid, which is a homogeneous fluid of protein gel composed of an iodinated protein, thyroglobulin (TG) (the storage form of thyroid hormones). During activity the amount of colloid is reduced and the secretory cells become taller. Between the follicles are connective tissue stroma, interfollicular cells and a rich blood supply.

The avian thyroid is unique in its lack of calcitonin cells; they are located separate from the thyroid gland in the ultimobranchial gland. Doves and pigeons appear to be exceptions, and are similar to the rat, with calcitonin cells found within the follicular epithelium.

Thyroid hormones (T_3 and T_4) are synthesised in a process similar to that in mammals. Iodide is concentrated within the thyroid, the so-called iodide trap. A peroxidase system within the thyroid converts the iodide to iodine and a second enzyme system is responsible for combining the iodinated tyrosines within the polypeptide chain of TG to form T_3 and T_4. Thyroid hormones are released from the thyroid as the predominant amino acid T_4. Once in the blood they are bound to protein. Both T_3 and T_4 are bound to serum albumin, and the binding affinity of albumin for T_3 and T_4 is the same. There is no thyroxine-binding globulin in avian species as there is in mammals. This binding of T_4 to albumin is evidently weak compared to that in man, resulting in more, free T_4 in avian blood than in human or most mammalian blood. The half-lives of T_3 and T_4 are very short (measured in hours) and almost identical for both forms. The principal route of excretion of T_3 and T_4 is via bile and urine.

The function of the thyroid is governed by the concentration of the circulating thyroid hormones and their effects on the hypothalamic-controlled pituitary release of TSH. A decrease in the amount of circulating thyroid hormones to a level below metabolic requirements prompts the neuroendocrine-controlled anterior pituitary to increase the release of TSH. TSH stimulates the thyroid and produces both hypertrophy (increased cell size) and hyperplasia (an increase in cell numbers), together with accelerated formation or secretion of T_4.

Thyroid hormones play a major role in regulating the oxidative metabolism of birds and thus regulate heat production in response to changes in environmental temperature. Any pronounced alteration in thyroid function is reflected in an altered metabolic rate. Seasonal profiles of circulating T_4 and T_3 in birds suggest that T_4 seems to be associated with reproduction and moult, whereas T_3 is associated with calorigenesis and lipogenesis, especially during migration.

The size of the thyroid is influenced by several variables such as age, sex, climatic conditions, diet, activity, species and hypophysectomy. An iodine deficiency produces goitre (enlargement) due to cellular hyperplasia as a result of TSH stimulation. Low environmental temperatures increase thyroid activity and thus thyroid size. In primary hypothyroidism there is a loss of follicles resulting either from thyroiditis or atrophy, while in secondary or tertiary hypothyroidism the thyroid follicles are distended with colloid and the lining epithelial cells become flattened. In hyperthyroidism a diffusely hyperplastic epithelium may be observed, with little or no colloid present and possibly with lymphocytic infiltration.

Parathyroid gland

In the chicken there are four parathyroid glands slightly caudal to the thyroid. A pair of glands is found on each side of the midline. Each pair represents an anterior and posterior lobe, which are often fused. The cranial gland is usually slightly larger. In the chicken the left parathyroid gland is not in contact with the thyroid gland, while the right-sided cranial lobe lies next to the thyroid gland.

Each parathyroid is encapsulated by connective tissue and is composed mainly of chief cells (very similar to those of the rat). Oxyphil cells are absent

Downloaded From Vet-eBooks.com

in many avian species. It may be assumed that the parathyroid chief cell in avian species is responsible for synthesis, packaging and secretion of parathyroid hormone (PTH).

PTH plays a major role in the regulation of blood calcium. It is secreted in response to hypocalcaemia and its effects appear to target the kidney and the bones. The initial response (within 30 minutes) is to decrease calcium excretion through the kidneys by increasing tubular resorption of calcium. It also causes an increased excretion of urinary phosphate. Renal tubular secretion appears to play a role in the response, although decreased tubular resorption of phosphate also plays a part, at least in the laying hen.

A third renal effect is the activation of vitamin D3 through the conversion of 25-hydroxycholecalciferol to 1,25-dihydroxycholecalciferol. Vitamin D3 elevates plasma calcium and inorganic phosphorus by increasing small intestinal absorption of these minerals. It also works with PTH to increase bone resorption and decrease calcium excretion.

Ultimobranchial glands

The left and right ultimobranchial glands lie caudodorsal to the caudal lobe of the parathyroid gland. They are small, flattened, irregularly shaped and unencapsulated glands.
They have four major components:

- C cells. Eosinophilic cells arranged in scattered groups and chords.
- Parathyroid nodules. Encapsulated accumulations of parathyroid tissue. Cords of parathyroid tissue grow from these nodules, penetrate between the C cells, and link up with the vesicles.
- Vesicles make up a large proportion of the gland and are lined by secretory epithelium. They accumulate a carbohydrate–protein secretion in their lumen.
- Lymphoid tissue foci of lymphoid cells and thymus tissue.

The C cells secrete calcitonin, which blocks the transfer of calcium from bone to blood. However, in contrast to its action in mammals, calcitonin does not induce a hypocalcaemia in normocalcaemic birds. It appears, rather, to control hypercalcaemia and to protect the skeleton from excessive calcium resorption. Its mode of action is still unclear.

Adrenal glands

The paired avian adrenal glands are located anterior and medial to the cranial division of the kidney. They are flattened and lie close together, even fusing in some species. Their arterial blood supply comes from branches of the renal artery, and each gland has a single vein draining into the posterior vena cava. There is evidence in some species, including the domestic fowl, of an adrenal portal system between the glands and the muscles of the lateral coelomic wall. Sympathetic nerves reach the cranial and caudal ganglia on the pericapsular sheath of the adrenal glands. Non-myelinated fibres originate from these ganglia and penetrate the gland. Each fibre innervates up to three chromaffin cells.

In birds, adrenal zonation is less clear than in mammals, with two zones, a subcapsular zone and an inner zone. Cortical and chromaffin tissue is intermingled in birds, with clusters or strands of chromaffin cells distributed throughout the cortical tissue. The adrenal cortical tissue is divided into a subcapsular zone, which is about 20–40 cells thick and produces aldosterone, and the more extensive inner zone, which produces corticosterone. Cortical tissue accounts for 70–80% of the avian adrenal gland. The cortical cells are arranged in numerous cords, with each being composed of a double row of parenchymatous inter-renal cells. The cords radiate from the centre of the gland, branching and anastomosing frequently, and loop against the inner surface of the connective tissue capsules. The arrangement of specific cell types along the cords results in some structural zonation. The cortical cells release primarily corticosterone and a smaller amount of aldosterone, along with some cortisol and cortisone (the levels of both decline as the bird reaches maturity). As in mammals, the secretion of corticosterone is primarily regulated by ACTH (corticotropin) that is released from the pituitary gland in response to CRF or AVT.

The control of aldosterone secretion in birds is believed to be similar to that of mammals (i.e. via the

Downloaded From Vet-eBooks.com

renin–angiotensin system), although some control via the hypothalamus and pituitary gland is believed to occur. Renin is released from the juxtaglomerular cells of the kidney in response to various stimuli including low sodium concentrations and reduced blood volume. The renin acts on angiotensinogen to form angiotensin I, which is converted to angiotensin II. Aldosterone secretion is in turn stimulated by angiotensin II. In contrast to mammals, birds do not release aldosterone in response to elevated extracellular potassium concentrations. Therefore, aldosterone increases when blood volume decreases, sodium increases or under the influence of ACTH. Avian adrenocortical hormones may have both mineralocorticoid and glucocorticoid properties and play important roles in regulating metabolism, stress responses, reproduction, moulting, and electrolyte homeostasis.

Chromaffin tissue constitutes about 15–20% of adrenal tissue. The chromaffin cells are in close association with blood spaces and appear to be more abundant in the middle of the gland. Two distinct types of chromaffin cells exist in the avian adrenal, releasing epinephrine and norepinephrine, respectively. These can be differentiated cytochemically and ultrastructurally, the latter including differences in the size and shape of the cytoplasmic neurosecretory granules. Chromaffin cells store and release the catecholamine hormones, either epinephrine or norepinephrine. The release, and presumably also the synthesis, of epinephrine and norepinephrine are separately controlled by the cholinergic innervation of the avian adrenal gland; in addition, hormones such as ACTH, corticosterone and aldosterone influence their synthesis and release. Their effects include changes to carbohydrate and lipid metabolism, cardiovascular parameters and the release of other hormones.

Pancreas

The anatomy of the pancreas has been discussed earlier (see p. 11). Like pancreatic tissue in all other vertebrates, most (99%) of the organ is devoted to the synthesis and secretion, through well-formed ducts, of digestive enzymes. The remaining 1–2% of the pancreas is endocrine and has no functional association with the pancreatic ducts. An extension of the ventral pancreatic lobe, which runs from the most superior portion of the lobe to the side of the spleen, is frequently referred to as the splenic lobe. This portion of the pancreas represents about 1–2% of the total wet weight of the organ and is without an exocrine duct.

The majority of the pancreatic islet cells are in the splenic lobe. These islet clusters synthesise and release their peptide products directly into the bloodstream. Pancreatic hormones (released in response to absorbed nutrients, to cholinergic input and probably to hormonal stimulation) include insulin, glucagon, pancreatic polypeptide (PP) and somatostatin.

Two types of endocrine islets have been described. The larger (and more numerous) islets appear to be composed predominantly of the glucagon (A cell) type, but also contain some B, D and PP cells. D cells (somatostatin) frequently occupy a central position within the glucagon islets. The smaller (and less numerous) islets, documented to be predominantly B cells that synthesise and release insulin, are scattered throughout the pancreas, although B islets residing in the splenic lobe are very large compared with the those found elsewhere in the pancreas. Distribution of PP cells appears to be without preference for any single lobe. Thus, they are fairly uniformly distributed in islets, as PP cell clusters and as single cells throughout the entire acinar pancreas.

The proportion of cell types varies between species: in carnivorous birds the proportions are approximately 70% B cells, 20% A cells, 9% D cells and 1% PP cells; in granivorous birds the proportions have changed to 37% B cells, 50% A cells, 12% D cells and 1% PP cells.

A islets, containing predominantly glucagon-secreting cells, secrete glucagon, which is a powerful hepatic glycogenolytic agent. Glucagon levels in avian plasma have been reported to be at least 10–80 times higher than in mammals, and pancreatic tissue glucagon concentrations are 2–4 times higher in the various avian species studied. It therefore appears to be the dominant pancreatic hormone in granivorous birds. It is a powerful catabolic hormone, stimulating gluconeogenesis, glycogenolysis and lipolysis. Its release is triggered by free fatty acids, cholecystokinin and somatostatin, while insulin inhibits it.

Downloaded From Vet-eBooks.com

The insulin:glucagon ratio is usually 1:2, thus favouring catabolic reactions and ensuring a continuous supply of energy to sustain higher metabolic rate.

Insulin is synthesised within the B cells. Once in the bloodstream this hormone acts primarily as an anabolic agent to increase the availability of glucose transport carriers, allowing easier transfer of glucose into the cell. It inhibits gluconeogenic processes and may be involved in lipogenesis. Insulin is not antilipolytic in birds as it is in mammals, and it is known to decrease glucagon secretion in birds. Avian plasma levels of insulin are much higher than in mammals. Its secretion is not triggered by glucose; rather, it appears to be more sensitive to cholecystokinin, glucagons and a mixture of absorbed amino acids. Carnivorous birds are thought to be more insulin dependent than granivorous species, although it is still important in granivorous birds.

The PP cell is identified as the sole source of avian pancreatic polypeptide (APP). Circulating levels of APP in the well-fed bird approximate 6–10 ng/ml, a level that decreases about 50% after an overnight fast. These values are 40–60 times greater than those found in mammals, including man. In addition to inhibiting gastrointestinal motility and secretions, APP exerts certain metabolic effects in birds; it stimulates gastrin release and mobilises liver glycogen, but has no effects on plasma glucose levels. It is primarily involved in lipogenesis, having an antilipolytic effect. Its levels rise sharply after a meal and it induces a sense of satiety.

Somatostatin is synthesised and secreted by the D cells. The D cell represents almost 30% of the cell population of dark (glucagon) islets, but only half of this population in insulin islets. The possibility exists that neural elements, with which the D cell appears to be well endowed (in the chicken), play a major role in regulating somatostatin release. Somatostatin depresses glucagon secretion and may act as the regulator of glucagon and insulin, 'fine tuning' their release. It also slows the absorption of nutrients, especially glucose and lipids, and inhibits lipolysis.

Growth hormone, thyroid hormones, prolactin and catecholamines are also involved in carbohydrate metabolism.

In many ways, carbohydrate metabolism in birds is similar to that in mammals. Differences include the hormonal control in granivorous birds, the absorption of glucose and gluconeogenesis. The end-product of digestion is glucose, which is then absorbed (usually passively) across the gut wall and either utilised locally by the enterocytes or enters portal circulation. It is then metabolised, aerobically or anaerobically, to produce adenosine triphosphate (ATP), which is used for energy. Glycogenesis (the formation of glycogen from glucose) occurs when there is excess glucose to body needs. Glycogen is synthesised in the liver and then stored in liver and muscle. This is controlled by glucagon (which controls liver glycogen stores) and epinephrine (which affects liver and muscle).

Gluconeogenesis is the formation of glucose from other molecules (usually lactate or glycerol) when there is insufficient intake of glucose. This occurs primarily in the liver, although there may be slight renal involvement. The transition to gluconeogenesis is rapid, usually beginning several hours postprandially. Carnivorous birds may exhibit continuous gluconeogenesis from amino acids, regardless of whether fed or not. This allows carnivorous birds to eat less frequently than granivorous birds.

Fasting or starvation induces catabolism; insulin levels are low while the glucagon levels are high. The glucagon stimulates lipolysis, as fat is preferentially mobilised during starvation. Glycogenolysis is also stimulated; hepatic glycogen is utilised first, but may be all gone within hours. Skeletal muscle stores are then used, especially in carnivorous birds. At this time blood glucose levels start to fall, stimulating gluconeogenesis, which begins after several days. Blood glucose levels then rise again. (In carnivorous birds, constant gluconeogenesis means that hepatic glycogen stores are usually untouched.) If the starvation continues, gluconeogenesis induces more protein catabolism to produce the amino acids needed for the process.

Hypermetabolism occurs when there is an increased demand for nutrients (e.g. sepsis, trauma, severe illness, surgery, pain, or hypotension), leading to an associated increase in metabolic rate due to effect of catecholamines, glucocorticoids and glucagon, but at the same time there is a reduction in food

Downloaded From Vet-eBooks.com

intake or absorption. In this situation fat oxidation cannot meet demands for energy requirements and so body proteins are broken down for gluconeogenesis. This results in increased susceptibility to disease, delayed healing and wound dehiscence.

ORGANS OF THE SPECIAL SENSES

Eye

The size of eye is extremely large in relation to the head, particularly when compared with that of mammals; in many birds the two eyes together outweigh the brain. Large eyes equal a large image projected on the retina, which contributes to visual acuity. The globe can be one of three basic shapes. Flat globes are found in the majority of diurnal birds with narrow heads. The short distance between the cornea and the retina means that the image thrown onto the retina is relatively small, with corresponding low visual acuity. Globular globes are found in diurnal birds with wider heads, such as insectivorous wing-feeders, crows and diurnal birds of prey. The cone-shaped eyeball results in greater visual acuity. Tubular globes are found in nocturnal birds of prey; the elongated shape gives the greatest visual acuity.

The lower eyelid is thinner, more extensive and more mobile than the upper lid. In most species the eyelids only close when sleeping, therefore the nictitating membrane, lying beneath the eyelids on the nasal side of eye, is responsible for blinking. Tears are produced by the Harderian gland and the lacrimal gland, which is present inferior and lateral to the globe. The tears drain into the conjunctival sac on the bulbar surface of the lower lid and then exit via the inferior and superior nasolacrimal puncta at the medial canthus. Meibomian glands are absent in birds. In budgerigars and others, a nasal or salt gland lies in the orbit dorsomedial to the globe and the duct of this gland pierces the frontal bone and enters the nasal cavity. Hyperplasia of this gland may occur in waterfowl given drinking water high in salt. Modified feathers (filoplumes) are present near the eyelid margin and have a protective and tactile function.

The cornea is small compared to the rest of the eyeball. It is small in underwater swimmers and more extensive and more strongly curved in species such as eagles and owls with globular or tubular eyes. It consists of five layers: an anterior (outer) stratified squamous epithelium; an anterior (outer) limiting lamina (Bowman's membrane), not always differentiated in birds and not found in mammals; the substantia propria, consisting of bundles of collagen fibres, which forms the great bulk of the corneal wall; a posterior limiting lamina (Descemet's membrane); and a posterior (inner) layer of simple cuboidal epithelium.

The sclera is reinforced by a continuous layer of hyaline cartilage which, in the zone nearest the cornea, is modified into a ring of 10–18 small, roughly quadrilateral, overlapping bones called the scleral ossicles. The ossicles strengthen the eyeball and provide attachments for the ciliary muscles. In large eyes the scleral ossicles can be pneumatic. In many species, including falcons, hummingbirds, woodpeckers and passerines, the scleral cartilage around the optic nerve is ossified, forming a U-shaped bone called the os nervi optici. The scleral venous sinus (canal of Schlemm) is conspicuous in some species, but small or almost invisible in others; it lies at the limbus (junction between the cornea and sclera). The trabecular reticulum, or pectinate ligament, in this region (a wide-meshed plexus of connective tissue fibres) joins the limbus to the iris and to the ciliary body. The spaces between these fibres form the spaces of the irido-corneal angle (spaces of Fontana) through which the aqueous humor drains into the scleral venous sinus.

The uvea, the vascular part of the eye wall, consists of the choroid, the ciliary body and the iris. The choroid is a thick, highly vascular, darkly pigmented layer coating the retina. A tapetum lucidum is only found in a few nocturnal species. The choroid continues as the ciliary body and the iris. The ciliary body suspends the lens by the zonular fibres; it also forms small folds (ciliary processes), which are pressed against the rim of the lens by the ciliary muscles. The iris is dark in most species, but highly coloured in some. It forms a round aperture in most species. The ciliary muscles and the sphincter and dilator muscles of the iris are striated muscles, in contrast to the smooth muscle of mammals.

The retina arises as a direct continuation of the brain. It consists of an external, non-sensory single

Downloaded From Vet-eBooks.com

layer of cuboidal epithelium containing pigment (pigment epithelium) and an internal transparent and thicker neuroepithelium (sensory retina) containing several types of neurones and glial cells. Rods and cones are present in birds, serving similar functions as in mammals. The retina is thick compared to other vertebrates, and contains an array of photoreceptors and several possible combinations of areas and foveas specialised for more acute (and often stereoscopic) vision. It is completely devoid of blood vessels and derives its nutrients from both capillaries within the choroid, external to the pigment epithelium, and the well vascularised pecten with the vitreous body.

Areas are circumscribed thickenings of the sensory retina involving thinner and longer visual cells that improve the resolving power and are, therefore, associated with improved visual acuity in birds. There is almost always one central area, but often two or even three distinct areas (a circular lateral area and a horizontal linear area) are present. Foveas are depressions within either a central or lateral area or both. Not all areas have foveas, but foveas are only found within an area. Visual cell density is greater in the fovea than elsewhere in an area, and its shape acts to magnify the retinal image and increase its resolution. Although some species have no fovea, most have one or two. The location, depth and relative position of these foveas exhibit considerable variation depending on the species. The presence of three distinct retinal areas (central, lateral and linear), two of which (central and lateral) possess a fovea, is a unique avian adaptation that permits the formation of three separate and distinct visual fields (visual tridents), two lateral monocular fields (one for each eye) and a central binocular field.

Birds are able to detect a spatial frequency much higher than that of mammals: 160 frames/second compared with 60 frames/second in humans. This higher spatial frequency can cause problems under artificial light, which has a frequency of 100–120 frames/second and can therefore produce a stroboscopic effect, which may attribute to some behavioural disorders. High-frequency lights are therefore recommended for indoor birds.

The pecten, unique to birds, arises from the site of exit of the optic nerve and projects a variable distance into the vitreous body in the ventral and temporal quadrant of the fundus. It consists almost exclusively of capillaries and extravascular pigmented stromal cells; there are no muscular or neurological tissues. It can be conical, vaned or pleated. The pleated pecten is the most common structure, arising as a single accordion-pleated lamina held in place at its free (apical) end by a more heavily pigmented crest or bridge of tissue. The mass, shape, number and arrangement of pleats, their extent of pigmentation and their relationship to the ventral ciliary body varies considerably in different birds. The size of the pecten and number of pleats do not coincide with size of eye, but seem to be related to the behaviour of the bird towards illumination and its general level of activity. Active diurnal birds with a high visual acuity and monocular vision have a larger and more pleated pecten, while those nocturnal species with poor vision usually have a smaller pecten of simpler morphology. It may play a number of roles: nutrition of the retina; secretion of glycosaminoglycans and other products; regulation of intraocular pressure through the secretion of fluids; light absorption, which would reduce internal reflections that may possibly interfere with the production of a clear image; the perception of movement; and perhaps orientating birds in space by acting as a sextant, casting a shadow upon the retina to permit the latter to estimate the angular position and movement of the sun.

The lens of birds is much softer than that of mammals due to a fluid-filled lens vesicle between the annular pad and the body of the lens. This softness allows rapid accommodation. The lens is optically clear, rendering ultraviolet radiation visible. An annular pad runs around the equator of the lens, adjacent to the ciliary processes. This is well developed in diurnal predators, but reduced in nocturnal species and flightless species. It has extensive equatorial thickening for contact with the ciliary body. Contraction of the ciliary muscles thrusts the ciliary body inward against the annular pad, transmitting the stress directly to the softer lenticular centre. This occurs evenly along the entire extent of the equatorial lens, allows for flexible accommodation and focuses light directly onto the retina. The transparency of

Downloaded From Vet-eBooks.com

the avian lens allows the passage of a broader spectrum of light than mammals, allowing birds to see not only blue, green and red, but also ultraviolet and fluorescent.

Both the anterior and posterior chambers of the eye are filled with aqueous humor, which is responsible for regulating intraocular pressure and maintaining the proper shape and rigidity of the eyeball. It is continually secreted into the posterior chamber by the ciliary body and then circulates through the pupil and into the anterior chamber. It then drains into the scleral venous sinus through the spaces of Fontana at the iridocorneal angle (**Fig. 1.31**).

The attainment of exceptional avian visual abilities is the result of both the structure and function of the eye. The positioning of the eyes allows, in many birds, a wide field of vision. The large size of the eye permits the formation of a large retinal image. The actions of the cornea, lens and aqueous humor provide a superb accommodative apparatus, increasing the depth of focus. The retina acts as a parabolic reflector, allowing all-round visual acuity, unlike mammals, which only have central vision acuity. And finally, the presence of elaborate and diverse retinal areas and foveas provide a high degree of acute monocular and binocular vision.

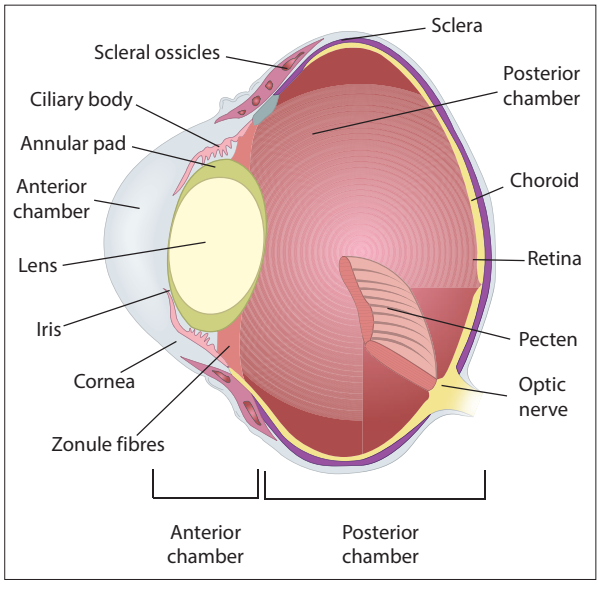


Figure 1.31 **Schematic diagram of the structure and key components of the avian eye.**

Ear

Birds have a keen sense of hearing and a high degree of equilibration. Their excellent voice production and remarkable ability to imitate sounds has inferred an exceptional degree of sound analysis (pitch discrimination) within a wide range of auditory frequencies. Their aerial mode of life demands sonic acuity, in addition to a well-coordinated balance and position sense. In many ways the avian ear closely resembles the mammalian ear, but it is simpler in structure and reptilian in design. It includes three separate but contiguous anatomical segments (the external, middle and inner ears). These develop completely independently and from different embryonic primordia and then combine to form a synchronised functional unit.

The external ear collects sound waves from the outside air and conducts them to the middle ear. It consists of a collecting device, the external acoustic meatus (a small aperture, nearly always circular, which opens externally on the side of the head), and a simple conducting tube. It terminates medially as a partition, the tympanic membrane (eardrum), completely separating the external ear from the middle ear. It is covered by specialised contour feathers (the ear coverts) in most birds; the width of the feathers is related to sound localisation and their fine structure varies in accordance with auditory efficiency. The coverts lying on the rostral aspect of the meatus reduce the drag caused by turbulence in flight and thus diminish the masking of sound by noise generated from turbulence in the external ear; the barbs of these ear coverts lack barbules; the sound waves are not obstructed. On the caudal aspect of the meatus these specialised feathers combine into a tight funnel, which is particularly enlarged in songbirds, parrots and falcons.

The middle ear is an air-filled, ossicle-containing space (tympanic cavity) that receives the sound waves as mechanical vibrations of the tympanic membrane in its lateral wall and transfers them in an amplified form to the inner ear at its medial wall. It is directly continuous with the pharynx via the pharyngotympanic tube (Eustachian tube), which enters the pharynx at the interfundibular cleft. The middle ear also communicates with a large group of accessory air cavities occupying the surrounding skull bones and then extending into the mandible and beak in

Downloaded From Vet-eBooks.com

some species. The right and left tympanic cavities communicate with each other via interconnecting air sinuses; this has been implicated in the transference of pressure fluctuations emanating at the round window.

In contrast to mammals, birds transfer sound with a single skeletal element, the columella, which extends medially across the tympanic cavity to form a direct connection between the tympanic membrane and the fluid within the inner ear. The columella is homologous to the mammalian stapes; the mammalian incus and maleus are homologous to the avian quadrate and articular bones, respectively. The tympanic membrane and columella function as a mechanical transformer that matches the impedance of air and inner ear fluid, facilitating the transfer of sound energy. The tension in the tympanic membrane is altered by the columellar muscle, which attaches to the columella and to the tympanic membrane itself. Vibrations of the tympanic membrane are carried to the perilymph of the inner ear by the extracolumella cartilage in contact with the tympanic membrane and the rod-like bony columella, which is implanted medially in the vestibular window. The cochlear (round) window lies near the vestibular window; it is in contact with the scala tympani of the inner ear.

The inner ear is responsible for the initial analysis and characterization of the sound vibrations and for maintaining equilibrium. It consists of two very complex, fluid-filled components or labyrinths, one membranous, the other bony. The membranous labyrinth includes several non-auditory receptive areas composing the vestibular labyrinth and a single organ of hearing, the cochlear labyrinth. The vestibular labyrinth is a series of canals and ducts filled with endolymph and surrounded by perilymph. It is then encased within the bony labyrinth. The cochlea is a relatively short and slightly curved tube containing the cochlear duct (scala media) filled with endolymph. It ends at the lagena, which contains a group of sensory cells; afferent nerve fibres from this area appear to end in the auditory centres of the medulla.

Movement of endolymph within the vestibular labyrinth provides the bird with its proprioception. Vibrations carried through the cochlear duct are detected in the brain as sound.

Chemical senses

The olfactory capabilities of birds have been controversial for years. Research conducted over the last two decades has indicated that birds possess olfactory systems whose complexity and development vary widely among species. They possess nasal conchae, but lack a vomeronasal (Jacobsen's) organ. The turbinates of the third nasal chonchae possess olfactory epithelium; the peripheral terminals of the olfactory nerves lie in this epithelium and communicate with the olfactory bulbs of the brain. The sense of smell is well developed in kiwis, New World vultures, albatross and petrels; it is moderately developed in poultry, pigeons and most birds of prey; and poorly developed in songbirds. It is possible that development of a sense of smell is related to food sources (e.g. vultures are carrion feeders that are led to the general area of food by olfaction; once in the general area they rely on vision to find the food source).

The function of a sense of taste (gustation) is to encourage ingestion of nutrients, to discriminate among foods that are available and to avoid toxic foodstuffs. As such, gustation in a particular species can be expected to complement digestion, metabolism and the dietary requirements of that species. Taste receptors (buds) are found in close association with the salivary glands at the base of the tongue and the floor of pharynx. Some are found in other areas and the number of buds and their distribution may change over time. The glossopharyngeal nerves innervate posterior buccal and pharyngeal areas. Cutaneous and taste information is carried by both nerves. The relationship between the number of taste buds and taste behaviour is not clearly defined; the relatively poor taste acuity in birds may be related to the small number of sensory cells.

Most avian species demonstrate little or no interest in common sugars except for parrots, hummingbirds and nectar feeders. These birds will actively select sugar solutions. While many birds kept on salt-free diets will actively pursue salt when offered, few will drink salt water at a concentration greater than its kidney can handle; many will die of thirst rather than drink. In parrots the salt threshold appears to be 0.35%. There is a wide range of tolerance for sour tastes, as there is for bitter foods. The temperature of the food is an important factor; many

Downloaded From Vet-eBooks.com

birds will reject food that is significantly higher than their body temperature.

The common chemical sense is relatively primitive; the senses of taste and olfaction are later differentiations. The major component of the common chemical sense is the trigeminal system. Irritants such as ammonia and acids stimulate free nerve endings of the nasal chamber, mouth and eyelids. It differs between species. For example, pigeons are indifferent to strong ammonia that can affect other birds and parrots can consume capsicum peppers that other birds cannot.

THE IMMUNE SYSTEM

The major lymphoid organs found in birds are the thymus and bursa of Fabricius and, to a lesser extent, the spleen and disseminated lymphoid tissue. Other than some waterfowl, birds do not have discrete lymph nodes.

The thymus is found in the neck, often in multiple sites extending from the angle of the jaw to the thoracic inlet. It consists of lobules of epithelial cells, each covered by a connective tissue capsule. Each lobule has an outer dark cortex and an inner light medulla. Lymphocytes are most dense in the cortex, while thymic corpuscles (islands of reticular tissue known as Hassal's corpuscles) are present in the medulla. The thymus is at its largest in the sexually immature bird. It serves as the source of T lymphocytes, which are the circulating cells responsible for cell-mediated immune responses. Approximately 65% of the mononuclear cells in the spleen and 80% of the mononuclear cells in blood of chickens are T cells.

The bursa of Fabricius is a dorsal median diverticulum of the proctodeal region of the cloaca. It contains a central cavity that forms a single large cavity opening into the proctodeum. The internal wall of the bursa is folded and covered by simple columnar or pseudostratified columnar epithelium. Lymphoid nodules are located between these epithelial folds. Each nodule has a cortex and a medulla, separated by a basement membrane and epithelial cells. Lymphocytes, plasma cells and macrophages are found in the cortex, while lymphoblasts and lymphocytes are found in the medulla. In the embryo the medulla is the first lymphoid organ to produce immunoglobulins. Mature B lymphocytes, responsible for humoral immunity, migrate from the bursa to peripheral/secondary lymphoid tissue. This occurs as early as day 17 of incubation. It also produces a hormone, bursapoietin, which stimulates the movement of B cells from the yolk sac to the bursa, and induces maturation of bone marrow cells. The bursa involutes as the bird ages; in parrots this may take as long as 18–20 months, compared with chickens where the bursa has involuted by 2–3 months of age.

The spleen is found on the right side of the junction between the proventriculus and the ventriculus. It varies in size and shape; it may be round, elongated or slightly triangular, depending on species of bird. There are no well-defined trabeculae; instead there is a basic network of reticular fibres and cells. Lymphoid tissue, known as white pulp, surrounds the arteries and is responsible for lymphopoiesis. Numerous venous sinuses are present, surrounded by lymphocytes, macrophages and elements of circulating blood. This red pulp is responsible for the phagocytosis of aged erythrocytes. Both white and red pulp contribute towards antibody production. The spleen does not function as a significant blood reservoir.

Disseminated lymphoid tissue is found in the Harderian gland in the third eyelid, throughout the alimentary tract (caecal tonsils, oropharynx, small intestine, and caudal oesophagus) and as solitary nodules in all organs and the bone marrow. It responds to antigens similarly to the spleen.

The immune system serves two primary purposes: it clears infection from the body and it then develops a pathogen-specific resistance to protect the bird from future infections. It does this through a combination of non-specific defences (including barriers such as the skin and mucosa, and the innate immune system—macrophages, heterophils, thrombocytes and complement) and specific defences, including humoral (B cells) and cell-mediated immunity (T cells).

Macrophages (in tissue) and monocytes (in blood) identify and consume damaged cells and foreign materials. They are attracted to sites of inflammation by lymphokines produced by damaged cells. Once this phagocytosis is complete, more lymphokines

Downloaded From Vet-eBooks.com

are released by the macrophages/monocytes, which attract B and T cells.

B cells produce antibodies (proteins that coat and neutralise an invading pathogen and 'mark' infected cells so that the immune system can identify and destroy them). An antigen, usually a protein on the pathogen, may activate a B cell directly or initially bind an accessory cell that leads to the activation of T and then B cells. Either way, the B cell then differentiates into the antibody-producing plasma cell. Antibody response can be either a primary response, stimulated the first time the body is infected with a specific pathogen, or a secondary response to subsequent reinfections. A primary response is characterised by a lengthy (1–2 week) latent phase during which immunocompetent cells are activated, leading to a progressive increase in circulating antibody. This initial antibody is usually IgM; it declines after reaching a peak. A secondary response is characterised as a shortened latent period, with a peak of antibody production, usually IgG (although some species utilise IgY), occurring earlier and at higher levels. Young birds originally passively derive both IgM and IgG (IgY) from the egg yolk and albumen, but an active humoral system begins to develop approximately two weeks after hatching, reaching maturity at 4–6 weeks of age.

Following the humoral response is the cell-mediated response. T cells originate in the thymus, and therefore cell-mediated immunity is dependent on the normal development of the thymus. Lymphokines (chemotactic factor, thrombocyte migratory inhibitory factor, interleukins 1 and 2, several types of interferons and lymphocytotoxin) attract both B and T cells to an area of inflammation. There are three types of T cell: T helper cells, which release chemical signals controlling other cells involved in the immune response; T cytotoxic cells, which destroy infected and damaged cells; and T suppressor cells, which modulate the effect on the immune system, preventing over-stimulation and autoimmune damage.

FURTHER READING

Jones MP (1994) Avian immunology: a review. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 333–336.

King AS, McLelland J (1984) *Birds: Their Structure and Function*, 2nd edn. Baillière Tindall, London.

Korbel RT (2002) Avian ophthalmology: principles and application. In: *Proceedings of the World Small Animal Veterinary Association Congress 2002*.

Whittow G (ed) (1999) *Sturkie's Avian Physiology*, 5th edn. Academic Press, San Diego.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

HUSBANDRY, GROOMING AND NUTRITION

Although many veterinarians are comfortable and familiar with the husbandry of companion animals such as dogs and cats, many are much less comfortable, or familiar, with the world of bird keeping. But without an understanding of how birds are kept and fed, it is difficult to assess conditions that lead to disease or make recommendations as to how to maintain a bird's health and happiness. There are some simple and subtle differences between the husbandry and nutrition of pet birds and aviary birds and the veterinarian needs to be aware of them.

HUSBANDRY

Pet birds

Birds have been kept as pets for many hundreds of years, but there has been a surge of popularity for them in the last 50 years. A large number of species are kept as pets, with interactions between owner and pet ranging from a bird that becomes a part of the household to a bird that rarely leaves its cage, serving more as a decoration than a pet. They all share a common feature in that their living arrangements nearly always involve life in a cage for all, or part, of their day. These cages have a direct effect on the health of the occupant, and it is important that veterinarians include an assessment of the cage and the owner's husbandry as part of their initial workup of a patient.

The cage

While the size of the cage must obviously reflect the size of the bird it houses, it must also reflect the bird's lifestyle. A bird that is allowed free range of the household, spending only a small amount of time in the cage each day, can do well in a smaller cage than a similar-sized bird that spends most of its time in the cage (**Fig. 2.1**). As a general rule, the minimum size of the cage should be large enough so that the bird can stretch its wings out without touching the sides and sit on a perch without the tail touching the floor of the cage. If the bird has restricted out-of-cage time, the cage must be large enough for the bird to engage in normal behaviours such as grooming, playing, and moving. Most birds will do better in a cage that is longer, rather than high, as they fly or hop in a horizontal direction rather than vertical.



Figure 2.1 **A clear example of an inappropriately small cage for this size of bird if it never has the opportunity to leave it.**

The cage should be constructed from material that is strong enough to withstand the power of the bird's beak; is easy to clean; and is non-toxic if ingested. Plastic-coated wire is commonly used for small bird cages while powder-coated bars, or even stainless steel, are used for larger cages. Small-gauge

Downloaded From Vet-eBooks.com

wire (e.g. chicken wire netting) is easily chewed and ingested by large parrots, resulting in heavy-metal intoxication, and should be avoided.

The floor of the cage should be lined with an easily replaced, non-toxic substrate. Newspaper or paper towel are appropriate; sand, wood shavings, corn cob bedding and crushed nut shells are not. The latter style of cage liners can cause problems if ingested and may harbour fungal spores and bacteria. Many cages have a wire floor; this is appropriate so long as it is cleaned regularly to prevent spilt food and dried faecal material adhering to the wire.

Unless very large, most cages are placed on a stand so that the bird is able to perch at a height level with a person's chest. Placing the cage on the floor, with human companions and predators such as dogs and cats looming over the top of the bird may lead to it becoming fearful. The stand should be strong and stable enough to hold the weight of the cage and bird, and should ideally be mobile.

Cage furniture

Perches, while giving the birds somewhere to sit, should not be so numerous that the bird cannot move freely in the cage. They should be positioned so that they do not sit above food and water dishes as faecal contamination will otherwise occur. The perches should be destructible i.e. it is normal for parrots to chew on them. Using metal pipe to avoid replacing perches is not a sound practice. Cotton fibre perches have to be monitored carefully, as gastrointestinal obstructions following ingestion of these fibres is becoming a more common diagnosis. Perches should be irregular in shape and diameter, to exercise the bird's feet and the diameter should be such that the bird's nails just meet when gripping the perch. The perches should be removed and replaced when they are soiled or badly damaged. Natural non-toxic branches usually make the best perches. The provision of a concrete 'grooming perch' can assist the bird in keeping its beak clean and blunting of the nails. The practice of covering perches with sandpaper should be discouraged as it can lead to abrasions of the plantar aspect of the foot and subsequent pododermatitis.

Feed and water bowls should be of a material suitable for the species; plastic dishes are fine for a canary, but a cockatoo or macaw will destroy them with ease. Stainless steel, glass, or glazed ceramic bowls are generally suitable for most species. Galvanised water dishes, coated in zinc and often soldered with lead, can be highly toxic to birds (**Fig. 2.2**). Terracotta dishes, although keeping water cool, are both absorbent and difficult to clean. The practice of placing water and feed dishes side by side should be avoided, as many parrots are fond of dunking their food in water, leading to contamination and rapid bacterial overgrowth in the water.

The provision of nest boxes and other sleeping/privacy shelters in a cage is a contentious subject. While some species (e.g. conures and small macaws) prefer a secure sleeping area, other birds will see it as a stimulus for reproductive behaviour. Each situation may have to be weighed on its own merits but if egg laying is not desired, it may be best to remove potential nesting sites.

Many owners provide their birds with toys to relieve boredom. Some of these toys are suitable for the inquisitive but destructive nature of parrots; others are not. Toys with cotton fibres should be avoided as ingestion of these fibres is a common cause of gastrointestinal obstruction (**Figs 2.3, 2.4**). Cheaply made plastic toys or toys with metallic components are easily destroyed and ingested, presenting the possibility of toxicosis or obstruction. Alternatives include non-toxic seed pods (e.g. pine cones, eucalyptus nuts), natural browse, blocks of soft untreated wood, and small cardboard boxes.



Figure 2.2 **A water dish constructed using lead solder.**

Downloaded From Vet-eBooks.com



Figure 2.3 **Examples of commercially available cotton fibre toys.**



Figure 2.4 **An obstruction largely composed of cotton fibres which had to be removed from a parrot's proventriculus.**

Positioning

While pet birds can enjoy interacting with their human companions, they can also become stressed by constant noise and movement, lack of privacy and security, irregular diurnal cycles and the possibility of the cage been knocked over. Indoor cages should be positioned with these stressors in mind. Some suggestions include: position the cage in a corner of a room, so that at least two approaches to it are secure; keep the cage off main traffic routes through the house, lessening close contact with people and other pets; and having a day cage and a night cage, so that birds can be 'put to bed' (rather than being covered) at nightfall.

Management

Feeding practices: Birds should be fed at least daily; it is a common practice to fill a feed bowl and leave it till empty. This practice, while labour saving, is misguided; empty seed husks can be mistaken for seed, leaving the bird to starve, and food left for long periods of time becomes contaminated with water and faeces, and attracts insects and mice. Fresh water should also be provided every day.

Cleaning: Cleaning of both the cage and the cage furniture needs to be done frequently and regularly. Cleaning should be aimed at removing organic material and then, where possible, disinfection should follow with a non-irritant chemical (**Fig. 2.5**).

Environmental enrichment: This means providing animals under managed care with environmental stimuli. Numerous studies have shown that brain activity is increased when environmental enrichment is provided, and that the animals' health improves. This in turn leads to fewer behavioural problems, increased production and better longevity. Enrichment has two goals. The first is to increase the number of natural behaviours a bird exhibits. These include foraging, positive social behaviour, and an increase in physical activity. The second is to decrease the number of unnatural or unwanted behaviours an animal exhibits. These might include pacing, self-mutilation and companion mutilation, appetite loss, obesity or aggression.

Downloaded From Vet-eBooks.com



Figure 2.5 **Example of poor cage hygiene which must be avoided.**

Suggestions for ways of providing environmental enrichment include:

- Foraging behaviours – providing an environment where a bird needs to spend time looking for food (**Fig. 2.6**). As an example, ground foraging birds (such as many Australian parrots) enjoy 'fossicking' in a wide tray with food items dispersed amongst a substrate of non-toxic items (e.g. gravel, paper cat litter pellets).
- Toys that can be played with, and even destroyed. Care must be taken that these toys are not dangerous or toxic if swallowed (e.g. fibre, plastic, metals; see **Fig. 2.4**).



Figure 2.6 **A suitable activity gym for birds that will encourage foraging for food.**

- Natural browse is an ideal enrichment tool, as it allows birds to engage in a natural chewing behaviour that can entertain them for hours.
- External stimuli can be provided by placing a cage or aviary in a position where the bird can view what is happening around it, while at the same time feeling safe and secure.
- Providing bathing opportunities – either with a body of water or a sprinkler system (please note that some birds prefer a dust bath).

Aviary birds

Aviculture is the practice of keeping birds in captivity, normally within the confines of an aviary, for hobby, display, research, conservation or income purposes. It is a popular hobby around the world, with operations ranging from a small backyard aviary with a handful of budgerigars to large commercial operations producing birds for the pet market or other hobbyists. Veterinary involvement in aviculture is, unfortunately, predominantly limited to 'fire brigade' medicine – diagnosing and treating disease in individuals and flocks. Aviculturists, perhaps unaware of what veterinarians have to offer, rarely seek advice from them or engage in preventative management and medicine. Aviculturists are not solely to blame for this state of affairs – veterinarians must accept responsibility for not educating clients about preventative medicine and not promoting it. Both parties must move towards each other to promote the better management of avicultural collections and the health of the birds within those collections.

General

Local council regulations often stipulate where a backyard structure such as an aviary can be built and how big it can be. Although not an obvious veterinary concern, it can impact on suggestions for alterations and extensions. Similarly, another consideration is the amount of land that is available. Ideally the open side of aviaries, pens and lofts should face the Equator i.e. north in the southern hemisphere and south in the northern hemisphere. This maximises exposure to direct sunlight throughout the day. This helps to keep enclosures dry and allows the birds to engage in

Downloaded From Vet-eBooks.com

normal daytime activities. The closed side of the aviary, on the other hand, should face towards the direction of prevailing weather, offering some protection against rain and wind.

Enclosure design and construction

The aviary frame can be of wood, metal or polyvinyl chloride (PVC) construction. While wood is the cheapest and easiest to work with, it has several disadvantages: the birds can chew it; it is often treated with preservatives that can be toxic; it cannot be disinfected; and it can harbour external parasites such as red mite (*Dermanyssus* spp.). Metal frames require specialised construction techniques, while PVC frames can be lightweight and prone to unwanted movement.

There are several requirements for aviary wire: it must be strong enough to withstand chewing by the species in the enclosure; it must be small enough to prevent wild birds entering the enclosure; it must be rust proof, but not so heavily or poorly galvanised that the zinc poses a toxic threat to the birds; and it should be 'see through' (this can be achieved by painting it black – the silver colour of wire reflects light back into the eyes of the viewer). See *Table 2.1* below for suggested mesh apertures and wire thicknesses for different bird sizes.

Aviary roofs can be either complete or partial i.e. the roof is either solid across the entire length or it is partially solid with the rest being wire. The latter design allows sunshine and rainfall to reach the birds, allowing a more natural effect, but the disadvantage is cats, wild birds, possums and rats can run along the top. Some aviculturists reach a compromise by having a small, wire-roofed 'porch' or landing where the birds can get into the rain, and then withdraw into cover if a threat presents.

Aviaries can be either full flight or suspended, depending on the floor – full flight aviaries have solid floors, while suspended aviaries have wire floors raised above the ground (**Figs 2.7, 2.8**). Dirt floors are cheap, but they are impossible to clean and allow access to the aviary by mice, rats or snakes. Concrete is expensive and permanent (the aviary can't be moved), but is easy to clean and maintain. Sand and gravel are used by some as a compromise between concrete and dirt. This has to be raked regularly to turn it over, and replaced every few months. Concerns for hygiene have to be weighed up against environmental enrichment – some birds, especially Australian species, like to forage for food on the ground, and this is of great psychological benefit to them. Although suspended



Figure 2.7 **An outdoor aviary which allows birds full flight.**

Table 2.1 **Suggested aviary mesh apertures and wire thickness for different bird sizes**

BIRD SIZE	MESH APERTURE (MM)	WIRE THICKNESS (MM)
Small (20–80 g)	12 × 12	0.7–0.8
Medium (80–200 g)	12–25 × 12	1.24
Medium–large (200–500 g)	12–25 × 12	1.6–1.8
Large (>500 g)	25–75 × 25–50	1.24–2.00

Downloaded From Vet-eBooks.com



Figure 2.8 **Example of an outdoor suspended aviary.**



Figure 2.9 **An outdoor aviary with safety porch used to prevent bird escape.**

aviaries are easy to clean they are becoming less popular, especially for larger species, as the impact on the birds' psychological wellbeing is becoming clearer.

Regardless of the aviary design, at some stage someone will have to enter it – to clean it, to recover a bird, to check nest boxes, and so on. This means opening a door, thus providing an escape route for a bird. It is never a good idea to allow a captive bird to go free; not only does it lack the skills needed to survive, it may be harbouring disease that could be devastating to native birds. It can also be financially disastrous – some birds are worth a lot of money and for this reason, responsible aviculturists often have a safety porch or walkway associated with their aviary so that they can always maintain a closed door between the birds and the outside world (**Fig. 2.9**).

Aviary furniture

It is important to encourage aviary birds to exercise. To this end, perches should not be so numerous that the bird does not see the need to fly. This can be done by placing only one perch at each of the aviary or by running a perch along one wall. As with pet bird cages perches should be positioned so that they do not sit above food and water dishes and should be appropriately sized for the birds being kept. Natural non-toxic branches are ideal.

The design of nesting sites varies between species – canaries, many finches and pigeons prefer an open nest, while parrots prefer a deep hollow. Aviculturists have come up with numerous designs for nest boxes: A-frames, L-frames, Z-frames, vertical boxes, and horizontal boxes (**Fig. 2.10**). The preferred design varies between species, and often between individuals, and so it is a sound practice to offer birds a choice of nest box designs and locations, allowing them to select their preferred box (the other boxes can be removed).



Figure 2.10 **Example of an horizontal nest box suitable for macaws.**

Downloaded From Vet-eBooks.com

Nest boxes should be positioned so that the bird feels safe entering and leaving it. In particular, sun shining directly into the entrance hole will frequently discourage birds from using them. A privacy shelter frequently overcomes this issue. This can be as simple as a partition in front of the nest box, screening it from view.

Nest boxes should be removed, cleaned thoroughly and left open to ventilate during the winter months. This prevents/minimises fungal contamination, especially with *Aspergillus* spores which can cause pneumonia in both adult birds and chicks.

Pest control

Bird enclosures are difficult to keep scrupulously clean; food and faeces attract insects and mice, which in turn attract rats and snakes. Small birds attract larger birds – especially raptors, owls and corvids. Design and management features that can minimise these problems include:

- Fully roofing aviaries, and ensuring there are no overhanging branches.
- Suspended aviaries minimise pest problems, but this may be compromising the birds' psychological welfare.
- Concrete floors are easily cleaned and maintained, but can be expensive to build.
- Fine mesh wire, sometimes doubly wired, can help to restrict access by rats and snakes. Mice, however, can get through almost anything.
- Rat walls – concrete barriers below ground level to a depth of 30 cm – can stop rats and mice burrowing into an aviary.
- Regular cleaning of the aviary floor and avoiding the use of feed hoppers – large dishes of feed that are topped up when empty – minimise the amount of waste that attracts pests.
- Careful use of mice/rat traps and baits can assist, but care must be taken to ensure that the birds do not become inadvertent victims.

Management

The following principles help aviculturists and veterinarians make a clear distinction between avicultural management (flock health) and the management of an individual pet bird. These principles are:

1. *Aviculture is farming:* Farming, by definition, is productivity-oriented animal stewardship. Many correlations can be drawn between the goals of the dairy, pork, poultry and beef industries and aviculture.
2. *Productivity is the lifeblood of the farm:* A farm is kept viable when there is a product that results from the investment in establishing a collection and effort to maintain it. Without production, numerous difficulties are experienced.
3. *Time and cost must always be justified:* Justification for time and cost in each avicultural effort varies immensely between individual owners. Regardless of the individual farm's manner of justification, there must always be thought applied to this principle. Physical time and expense invested in new management efforts must not compromise current maintenance efforts. Otherwise, ramifications from those distractions can pose a significant threat to the overall effectiveness of the farm.
4. *The flock always takes precedence over the individual:* Flock health must be focused on the group rather than the individual. Individually diseased birds should still be diagnosed and treated, but the emphasis must always be on prevention of the reoccurrence of the problem(s) in the future. No single bird should be prioritised above the entire collection.
5. *Culling and replacement are fundamental for improvement:* The concept of culling and stock rotation is a fundamental part of any progressive animal production oriented operation. Based on the aviculturist's goals and objectives, those individual birds not meeting desired goals should be culled by sale or physical removal. When working with particularly rare or valuable species, culling should be a final management step when efforts to identify limiting factors to production have failed. (Note that culling does not include relocation within the aviary or further evaluation to determine the cause of poor production.)

Downloaded From Vet-eBooks.com

The closed aviary concept

The fundamental concept behind maintaining a healthy avicultural collection is the closed aviary concept. It is a system of management principles by which aviculturists should be able to maintain their aviaries in a disease-free state. The basis of these principles is to firstly establish a disease-free flock, and then to maintain it that way by preventing the introduction and spread of disease.

The first step is to establish a flock that is healthy. This is usually accomplished through an inspection of the aviary by an avian veterinarian. This visit, discussed later, focuses on two key areas: 1) the aviary infrastructure and management and 2) the birds – their age, health and productivity.

Once the aviary has been certified disease-free by the veterinarian, it can be maintained that way using the following steps:

1. The aviary should be divided into five geographically separate, areas: food storage; breeding birds; nursery and juveniles; isolation; and quarantine (see **Fig. 2.11**). The only way a bird enters or re-enters the aviary is through the quarantine area. Quarantine is not an isolation area (which is used for sick birds in the collection). Quarantine is for recently purchased birds and those returning from shows, breeding loans and other trips to the outside world (**Fig. 2.12**). During the 30–45 day period in quarantine, all birds must pass diagnostic tests to be accepted into the collection. Depending on the aviculturist's resources and previous disease problems, these tests may be as simple as casual observation or as complex as PCR, cultures, and blood tests.
2. After passing quarantine, the bird may enter the breeding flock with the ultimate goal of producing offspring. The offspring will be housed in the nursery and juvenile bird area, and eventually be sold or retained as breeding stock.
3. Occasionally disease may strike even the closed aviary. In these cases, affected birds are placed in isolation until they recover and have passed the same diagnostic tests used in quarantine. Isolated birds should not be housed with quarantined birds as they are still members of the closed aviary.
4. All supplies (food and water), people and equipment entering the aviary should be clean and free of disease. The person feeding should wear protective clothing that is not worn outside the aviary. Food should be stored in a clean, separate area that is hygienic and rodent-proof.

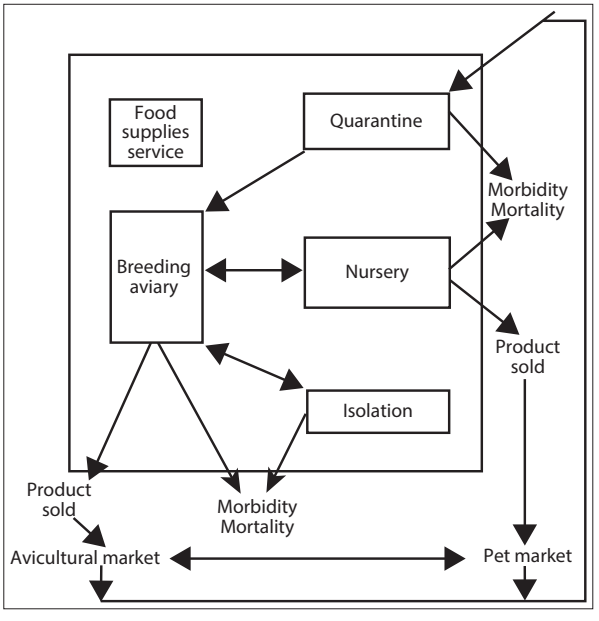


Figure 2.11 **Diagram illustrating the closed aviary concept showing appropriate bird flow between five geographically separated areas.**



Figure 2.12 **Suitable caging arrangements in a quarantine room.**

Downloaded From Vet-eBooks.com

5. Human traffic flow should move in a one-way direction from the outside to the feed storage, then to the breeding birds, the nursery and juveniles, then to the isolation area and finally to the quarantine area. This pathway follows from the 'cleanest' birds to the 'dirtiest' birds and prevents unnecessary cross-contamination.

The aviary visit

The aviary visit is a key element of ensuring flock health. It serves two purposes: to establish a baseline for the flock's health, and then to monitor the flock's status and improvements. It can be divided into three components: 1) the initial assessment of the client and the collection, 2) the physical visit and 3) the preparation of a report for the client.

The initial client assessment: It is not an uncommon experience for a veterinarian, after been asked to give advice, to become frustrated at the client's perceived unwillingness to make fundamental management and structural changes to their collection in order to follow the advice. At the same time the client becomes frustrated with the veterinarian's perceived lack of understanding of the realities of their circumstances. This shared frustration usually arises because, in the early stages of the collection assessment, the veterinarian failed to carry out an appreciation of the aviculturist's desires, plans, abilities and limitations. This must be addressed before any further assessment or planning is performed.

This initial assessment can either be done face to face, or (ideally) through a questionnaire filled in prior to this visit. Areas that should be explored include:

- What are the client's goals or objectives?
- What does the client hope to achieve through this assessment process?
  - Identify major problems from the client's perception.
  - What does the client expect a veterinary inspection and advice to achieve?
- What is the client's level of experience and expertise in bird keeping?
- A description of the collection:
  - Type of birds.
  - Numbers held.
  - Age and sex of each individual bird.
  - Grouping of the birds (pairs, colonies).
- A description of the collection infrastructure:
  - Aviary layout (a map is very useful).
  - Cage construction.
  - Food storage.
  - Quarantine and isolation facilities.
- Management:
  - Diet.
  - Prophylactic treatments.
  - Daily management, including traffic flow around the collection.
  - Purchasing policy and quarantine process.
- What records are available?
- Collection performance over the last 3–5 years:
  - Breeding successes or failures.
  - Losses and medical problems.
  - Other issues.

After collating this information, the veterinarian should be able to analyse the data so that a clear picture of the client's abilities, expectations and limitations emerges, as does an idea of the current situation within the flock. With this information in hand, the next step is the aviary visit.

The visit: The visit is for the collection of information and data, not for decision making and planning. It is important that both the veterinarian and the client understands that it is only part of the collection assessment. Before physically walking through the collection, the veterinarian and the client should sit down and discuss the answers to the questionnaire and the veterinarian's assessment of that data. It is important that both parties agree on the information and interpretation and are clear on each other's intentions, abilities and limitations.

While inspecting the collection, the veterinarian needs to have three things in mind:

1. The information already gathered in the initial assessment.
2. The principles behind avicultural management.
3. The closed aviary concept.

The inspection can also be broken down into three components: 1) the initial walk through,

Downloaded From Vet-eBooks.com

2) biosecurity assessment and 3) monitoring; and an inspection of the records.

The initial 'walk through' inspection focuses on:

- Layout of the complex.
- Individual cage design and construction:
  - flooring
  - roofing
  - frame
  - wire
  - furniture.
- Water sources.
- Food sources and storage.
- Rodent and pest proofing and control.
- The nursery, including the incubation, hand-rearing and juvenile-holding facilities.
- Biosecurity measures, such as cleaning, disinfection, visitor limitations, boots and sometimes protective garments, and equipment usage.

Biosecurity assessment and monitoring utilises tests to evaluate the flock for the presence of disease, and may include:

- Faecal examinations.
- Cultures.
- DNA tests for diseases such as PBFD, polyomavirus and *Chlamydia*.
- Blood tests (haematology, biochemistry).
- Necropsies.

The collection's records are also examined. These records should include:

- Breeding records:
  - identity and pedigree
  - productivity
  - incubation
  - paediatrics
  - overall productivity analysis.
- Medical records.
- Sample contractual agreements between the aviculturist and purchasers/sellers of birds.

The final step – the report: To complete the aviary visit a written report should be prepared for the aviculturist. This is the most important part of the process and, while it needs to be prepared promptly, it should be prepared thoughtfully and thoroughly. It should include the following details:

- Date of the visit and qualifications of the veterinarian.
- Background and objectives of the collection.
- Description of the collection including the species and number of birds held.
- Aviary design and layout, including a map and aviary construction.
- Biosecurity measures.
- Nursery management.
- Diet, including food storage and preparation.
- Individual bird health and results of diagnostic testing.
- State of record keeping and an analysis of the productivity of the collection.
- Summary of the findings.
- Recommendations.
- Follow-up.

Follow-up: It is important to realise that the visit is not the final step in the process of maintaining a healthy collection. It is suggested that an annual visit be conducted, with subsequent reports using the original report as a baseline for change and improvement.

Grooming birds

Clients will frequently present their birds for grooming, namely wing trimming, nail trimming, and beak trimming.

Wing trimming

Wing trimming is a grooming technique designed to keep birds safe when indoors (see **Figs 2.13a** and **b**). It is not a means of preventing flight – rather, it lessens the bird's ability to gain height and speed while in flight. It does not mean that the owner can take the bird outside without restraint. Some simple rules for wing trimming include:

- Do not trim a bird's wings until it has learnt to fly. It is only by flying that the bird can learn how to land – the most difficult part of flight. More birds are injured by clumsy landings than anything else associated with flight.

Downloaded From Vet-eBooks.com

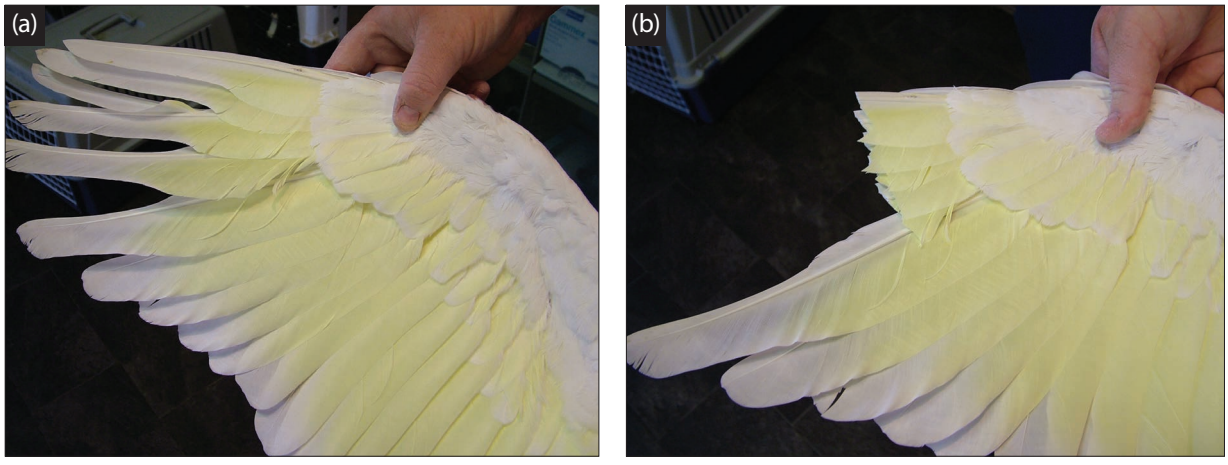


Figure 2.13a,b **A cockatoo wing before and after a correct wing trimming procedure.**

- Trim only the primary feathers – the long feathers coming off the metacarpus from the carpus to the wing tip. The bird needs its secondary and tertiary feathers to slow down and land safely.
- Trim the same number of feathers on both wings – one-winged clips unbalance the bird and make a bad landing more likely.
- Never guarantee the effect of a wing trim – the trimmed feathers will moult out and be replaced, and when this will happen cannot be predicted.
- Trim 5 mm below the edge of the coverts – this avoids cutting through a 'blood quill' - a newly erupted feather with blood vessels and nerves in the shaft.

Common injuries associated with poorly performed wing trims include sternal trauma, tail-split injuries, and wing-tip trauma.

Nail trimming

Trimming a bird's nails is usually performed to avoid scratches and lacerations to the owner's hands and arms while handling it. Occasionally birds with malnutrition and/or liver disease will develop overgrown nails, requiring trimming. In these cases the nails are often deformed and soft or flaky. Birds with nails like this should be investigated for underlying problems.

The nail consists of a hard keratin dorsal plate and a softer keratin ventral plate encasing the last phalange and the growth area of the nail (the nail bed). The dorsal plate grows faster than the ventral plate giving the nail its characteristic curved shape. The nails are normally trimmed naturally by perching on branches of varying diameters, shapes and textures. Lack of opportunity to perch naturally can result in overgrown sharp nails.

The nails can be trimmed with either small 'cat' nail trimmers or, in larger birds, a Dremel® hobby grinder (**Fig. 2.14**). The nail is trimmed to a level which is a few millimetres below the ventral plate.



Figure 2.14 **Demonstration of bird nail trimming technique using a Dremel® hobby grinder.**

Downloaded From Vet-eBooks.com

If bleeding occurs it can be stopped by applying digital pressure for a few minutes, or by pressing the nail into dry Condy's crystals (potassium permanganate) or a wet bar of soap.

Beak trimming

Although many clients will present their bird for a beak trim, it is important to emphasise that this is not a routine grooming procedure. Birds will keep their beak in a normal shape and length by chewing on branches and other hard wooden structures. Overgrown beaks can result from lack of such chewing opportunities or, more commonly, from malocclusion or chronic liver disease. Birds presented for the first time for a beak trim should be thoroughly evaluated for such problems.

It must also be remembered that some birds (e.g. long-billed corella) have a naturally long beak. It is not inappropriate to check what length a normal beak should be in an unfamiliar species.

Although the beaks of small birds (e.g. cockatiels and budgerigars) can be trimmed with nail clippers, larger birds usually require trimming with a Dremel® hobby grinder to both shape and shorten the beak. Care must be taken with soft or flaky beaks not to inadvertently damage the beak. In severely overgrown beaks, especially those with malocclusion, general anaesthesia may be required to achieve a good result without unduly stressing the bird.

Nutrition

Poor nutrition is the underlying cause of many of the health problems seen in both pet and aviary birds; in fact, most avian veterinarians consider malnutrition to be the major problem affecting the health of birds today. For this reason it is important that veterinarians treating birds have a sound and practical understanding of the nutrition of pet and aviary birds.

Nutrients can be classified into six categories: 1) protein, 2) fats, 3) carbohydrates, 4) vitamins, 5) minerals and 6) water. Bird owners frequently feed seed, vegetables, fruit, supplements, and treats ('human food'). Some feed a mix of these foodstuffs, hoping to achieve a balance of nutrients. Others, however, either lacking knowledge or simply frustrated by the selectivity of their bird, feed only one or two food items. The popularity of commercially manufactured formulated diets (which removes the bird's ability to selectively eat an unhealthy diet) is rising, but by no means universal.

Seed

Captive parrots are commonly fed a predominantly or even all-seed diet, believed by many to be a 'natural-diet (**Fig. 2.15**). Parrots are commonly thought to be 'seed-eaters'. This belief fails to take into consideration that there are some 365 species of parrot, found naturally in four continents and numerous islands. They have evolved in a myriad of geological and climatic conditions, with a wide range of very different foodstuffs available to them. The natural diet of wild parrots includes many different plants and plant parts, as well as invertebrates and sometimes even meat from animal carcasses. Their diet changes based on seasonal availability, and their requirements change according to their life stage and physiological status.

It is believed that parrots evolved in Gondwanaland some 50 million years ago. However, agricultural crops were not planted in Australia until some 200 years ago so the grains found in commercial seed mixes are not indigenous to the natural habitat of Australian parrots. In fact, many of these grains originate from the American continent and thus are not natural food items for African or Asiatic species either. Although many wild parrots have adapted to, and even taken advantage of, the introduction of agricultural crops into their habitat, they do not feed



Figure 2.15 **Example of a commercially available all-seed, high-fat bird food mix.**

Downloaded From Vet-eBooks.com

on these crops exclusively, browsing on a variety of food items and, perhaps more importantly, foraging over large areas of land to find enough food. In fact, field studies have indicated that most wild parrots spend more than 50% of their day foraging for food, see **Fig. 2.16**. This exercise 'burns up' much of the excess energy contained in agricultural grain. It has been estimated that the energy requirement of a wild bird is more than 1.5 times that of a bird in captivity.

Not only are all seed diets unnatural, they are also nutritionally unbalanced. The fat content of seed varies between 20% and 50% (some nuts contain an even higher proportion). Although ample carbohydrates are present, they are often surfeit to the bird's energy requirements. The total protein content of seed is often suitable for adult birds (but not for growing chicks), however, the amino acid composition is not suitable for maintaining long-term health. Seed is deficient in vitamins A (including beta-carotene, its precursor), D, and K. Seed usually contains adequate amounts of vitamin E (if fresh) and, although certain seed types may be marginal in individual B vitamins, if a mix of seed is offered, requirements for these vitamins may be met. Most seeds have an excess of phosphorous but are deficient in other minerals including calcium, manganese, iron and zinc. Iodine and selenium may also be deficient, depending upon the geographical region of production. The effects of the high fat content can amplify the effects of the low calcium through saponification in the intestinal tract making the calcium unavailable for absorption.

In summary, dry seed diets have: excessive fat, carbohydrates and phosphorous; marginal protein; adequate vitamin E; and are deficient in in amino acids, calcium, available phosphorus, sodium, manganese, zinc, iron, vitamins A, D, K, and B12, riboflavin, pantothenic acid, choline, and available niacin. While they can sustain life, a dry seed diet is not suitable for long-term health, growth or reproduction.



Figure 2.16 **Wild galahs foraging for food.**

In an attempt to improve the nutritional value of seed, some bird owners will soak and/or sprout the seed. This process increases the water content (diluting the energy), lowers the fat content and, if sprouted, increases the protein and vitamin content. These advantages have to be weighed against the risks of bacterial or fungal overgrowth during the process, resulting in fermentation of the seed. Some aviculturists minimise this risk by adding chlorhexidine (or even household bleach) to the mix. Thorough rinsing is necessary before feeding.

Vegetables

In an attempt to balance a seed diet and to more closely simulate the diet of a wild parrot, many owners add vegetables to their birds' diet. However, many of these domestic vegetables are deficient in energy, calcium and other nutrients, and are often higher in water, than their wild counterparts. People can thrive on a balanced vegetarian diet, but it should be remembered that compared to birds, humans grow very slowly and have a low reproductive output. Birds fed an exclusively vegetable diet will display signs of poor growth, loss of condition, and poor reproductive success.

On the plus side, vegetables fed as a supplement to a seed diet can dilute the energy content while providing some nutrients not found in seed (**Fig. 2.17**). They also provide a form of enrichment when fed in large pieces. This does, however, require that the bird eat the vegetables – when given free choice many birds will eat seed in preference to vegetables, often to the complete exclusion of the vegetables. Feeding strategies, such as limiting access to seed to a short period morning and night, may be needed to overcome this dietary preference.

Fruit

Many of the preceding comments regarding the feeding of vegetables apply to fruit as well, although

Downloaded From Vet-eBooks.com



Figure 2.17 **A cockatiel eating a mix of vegetables, pellets and seed.**

fruits tend to be higher in energy and water than vegetables. Many arid zone Australian parrots (e.g. some cockatoos, cockatiels, and budgerigars) have not evolved with fruit as part of their diet, and show little interest in it. Other, more tropical, parrots such as eclectus, lorikeets, and Asiatic, South American and African parrots relish fruit in their diet. Some fruits, such as avocado, may be poisonous to birds, and should be avoided. Citrus fruits can promote iron absorption, leading to iron storage disease in susceptible species (e.g. mynahs, toucans and lorikeets); minimal amounts should be fed, if at all. In general, fruit should be restricted to a small component of the diet (<10%) or even regarded as a treat rather than a regular food item.

Supplements

The diets consumed by free-living birds can rarely be duplicated in captivity because the vast range of seeds and other food items are not usually available in sufficient quantities. Even if these food items, or very similar ones, could be obtained, they may still not be nutritionally adequate. This is because wild birds eat large amounts of food to meet their energy requirements, but their intake of amino acids, vitamins and minerals remains constant. Captive birds eat relatively less food, and if eating a 'wild' diet, they will become deficient in these nutrients. For this reason, supplementation is necessary for birds fed a diet based on seed and vegetables.

As mentioned previously, the level of calcium in seed diets is low, and even in vegetables with reasonable quantities, the availability can be extremely variable. Calcium found in plants is often less digestible than other sources because of the formation of poorly digestible complexes with phytic and oxalic acid, or saponification when fed with high-fat diets. In the wild, many birds supplement their diet with mollusc shells, eggshells, and calciferous grit. In captivity, a calcium supplement is required when seed and vegetables are fed as the predominant diet. This supplementation is available in several forms: cuttlebone, shell grit, calcium powder and calcium syrup, each with its own advantages and disadvantages. Cuttlebone is relished by many smaller parrots, but destroyed by larger parrots with little ingestion. Shell grit is readily consumed by all parrots but, if over-eaten, may lead to a grit impaction of the proventriculus and ventriculus. (This can be prevented by adding only a small amount to the food every few days, rather than providing a separate bowl of grit.) Calcium powder is often unpalatable, and has to be mixed in well with the food. Calcium syrup often has sugars added to improve its palatability, but this can lead to bacterial overgrowth if food and water dishes are not washed daily. Care must be taken not to over-supplement calcium; in some species such as budgerigars, excessive levels can be toxic.

Similarly, vitamin and other mineral supplementation is also required for birds on seed and/or vegetable-based diet. Many birds appear to be able to balance energy, amino acid, and calcium levels in their diets by selecting among dietary items, but there is little evidence they can select for adequate intake of many other nutrients. Vitamin and mineral supplements are frequently offered in the form of powder and, liquids to be added to the food or water. The effect is often lost when added to a dry seed mix, as the vitamin coatings on the husk are lost when the bird removes and eats the kernel. Vitamin-mineral pellets added to a seed mix are often of little value, as birds often ignore them. Vitamins and minerals added to the water may not achieve its intent, as these solutions are often very unstable in water and rates of water consumption are extremely variable. Some bird owners will attempt to compensate for this variable intake by over-supplementing the diet with

Downloaded From Vet-eBooks.com

vitamins and minerals. Unfortunately this practice may result in excessive intake and subsequent toxicosis. Vitamin A and D over-supplementation, in particular, have been reported in parrots with sometimes fatal effects.

Formulated diets

In an attempt to overcome the inadequacies and deficiencies posed by feeding seed and vegetables, formulated diets have been developed for birds as they have for other species. The processing binds the dietary ingredients together so that the bird cannot select individual components. These ingredients are typically ground grains (such as corn) and ground legumes (such as soybean meal or peanut meal), with vitamins, minerals, vegetable oil, and purified amino acids added to meet known (or estimated) requirements. These ingredients are then either pelletised or extruded. Pellets are made by heating the mix to 70–80°C and passing it through holes using a roller. When the mix emerges as cylindrical particles of a constant diameter, a turning knife cuts the pellets at pre-set lengths. Extruded diets, on the other hand, are made by forcing the mix, under pressure and at high temperatures (between 90–180°C), through an extruder. Water is added in the extruder as steam for a 'dwell' time of anywhere from 30 seconds to several minutes. The food will take on the shape of the holes in the extruder plates. The water in the mix instantaneously evaporates when it leaves the extruder, along with the intracellular water in the plant cells and bacteria, rupturing the plant cells and killing infectious agents if present. The resultant extruded diet is hydrolysed and sterile, but may have lower nutritional value. This last problem is overcome by using more nutrients in the pre-mix before the extrusion to compensate for expected losses. Extruded diets are usually more palatable than pelleted diets.

Many aviculturists and pet bird owners resist the concept of formulated diets, arguing that these diets are unnatural, too expensive, boring for the bird, or that their birds just won't eat them. The first two arguments can be dealt with through client education; the third can be overcome by adding vegetables and fruit to the diet and the use of foraging activities to make the bird 'work' for its food.

The last argument, that the birds won't eat the new diet, is more problematic. Many birds are neophobic (scared of new things); others simply don't recognise the new diet as food. Forcing the birds to convert to a new diet by withholding other foods does not work in many cases; small parrots, in particular, may starve to death if this approach is attempted. There are a few simple guidelines to think about when planning to convert a bird to a healthier diet:

- If there is more seed in the cage than the bird will eat in a day, it will not touch new foods
- Foods offered in a deep dish are unattractive to most birds. Conversely, foods offered in a flat dish or on a flat surface such as a table top will often be explored, picked up and eaten
- Birds learn by mimicry; if they see other birds (or their owner) eating the new foods, they are more likely to try it themselves. If they are closely bonded to the owner, they may accept new foods when offered to them by hand
- Conversion may take a few minutes or a few months.

Strategies utilising these guidelines include: hand feeding the bird with the new foods, giving plenty of praise and encouragement while doing so; if the bird comes out of its cage, placing the new foods on a table top and encouraging the bird to pick them up and try them; placing the food on a flat dish at the bottom the cage and removing all seed for 6–8 hours; and putting the bird beside another bird that already eats the formulated diet.

If these strategies do not work, a more calculated approach may be required:

1. Determine how much seed the bird eats in a day. This is done by measuring how much seed is placed in the cage in the morning, and then how much is left the next day. The difference between the two amounts is the amount eaten. Repeat this exercise for 2–3 days to get an average daily seed consumption.
2. Once it is known how much seed the bird eats each day, only that amount of seed is provided, mixed with an equal amount of pellets plus vegetables made into a mash.

Downloaded From Vet-eBooks.com

3. Feed this mash fresh each day, but each day remove a small amount of seed. This makes the bird slightly hungrier each day and as it searches through the mash looking for the seed, it will hopefully start to eat the pellets and vegetables. Over a period of several weeks seed can be eliminated from the diet.

During the conversion process the owner should weigh the bird each day. If there is more than a 10% weight loss, or if the bird starts to look unwell, it should be offered seed immediately. If the bird returns to normal weight and behaviour, it should be given a few days to recuperate before starting the process again.

Lorikeets

Lorikeets and lories are brightly coloured, active nectarivorous parrots native to Australia and Oceania. Long popular as aviary birds, they are becoming more popular as pets but have unique dietary requirements. In the wild these birds require a lot of energy. They fly swiftly and powerfully through their habitat and are constantly moving from flower to flower whilst feeding (**Fig. 2.18**). Their natural diet (nectar, pollen, manna, honeydew, lerp, insects and fruit) reflects this high-energy usage, and their anatomy reflects this high-energy diet (brush tongue; small, relatively weak ventriculus; and a rapid gastrointestinal transit time). Nectar is a sugar-rich, liquid food source, but is low in amino acids, vitamins, and trace minerals. Pollen protoplasm is composed of highly digestible protein and contains a diverse amino acid profile, and is therefore a good food source for lorikeets. Manna is a sugary exudate from damaged eucalypt leaves or woods; honeydew is the sugary excretion of nymphal stages of aphids, coccids, and insects belonging to the family Psyllidae (plant lice); and lerp is a waxy material secreted as a protective scale by psyllids. However, given the high carbohydrate and low protein content of these latter foods, it is likely that they are used only as alternatives to nectar. Nectarivorous birds almost certainly obtain a good supply of protein from insects.

Attempts to replicate this diet in captivity have met with varying degrees of success. Many of these artificial diets are high in sugar and do not account for the decreased energy output associated with captivity. As well, many do not match the amino acid composition of pollen and insects; the result is often obesity, poor growth, and decreased reproductive performance. Several attempts have been made to produce lower energy diets for lorikeets, but palatability remains an issue for many birds (**Fig. 2.19**).

Generally, captive lorikeets are fed a small amount of liquid formula once or twice daily and *ad lib* dry food, supplemented with fruit, vegetables and sometimes sprouts. Some larger lorikeets such as the Rainbow lorikeet (*Trichoglossus haematodus*) will consume seed, but this should not be considered



Figure 2.18 **A wild scaly-breasted lorikeet foraging freely.**



Figure 2.19 **Examples of foodstuffs for pet birds which try to replicate the wild diet of lorikeets.**

Downloaded From Vet-eBooks.com

a suitable diet for these birds. More research needs to be done to determine the ideal diet of captive lorikeets and lories; veterinarians should not assume that commercially manufactured diets are nutritionally adequate.

FURTHER READING

Gartrell BD (2000) The nutritional, morphologic, and physiologic basis of nectarivory in Australian birds. *Journal of Avian Medicine and Surgery*. **14**(**2**):85–94.

Harper EJ, Skinner ND (1998) Clinical nutrition of small Psittacines and Passerines. *Seminars in Avian and Exotic Pet Medicine*. **7**(**3**):116–127.

Kalma ID, Janssen GPJ, Moons CPH (2010) Guidelines and ethical considerations for housing and management of Psittacine birds used in research. *ILAR*. **51**(**4**):409–423.

Koutsos EA, Matson KD, Klasing KC (2001) Nutrition of birds in the order Psittaciformes. A Review. *Journal of Avian Medicine and Surgery*. **15**(**4**):257–275.

Speer BL (1991) Avicultural medical management. An introduction to basic principles of flock medicine and the closed aviary concept. *Veterinary Clinics of North America. Small Animal Practice*. **21**(**6**):1393–1404.

Stahl S, Kronfeld D (1998) Veterinary nutrition of large Psittacines. *Seminars in Avian and Exotic Pet Medicine* **7**(**3**):128–134.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

THE PHYSICAL EXAMINATION

UNDERSTANDING THE MASKING PHENOMENON

A common misconception held by many bird owners (and many veterinarians) is that birds are not very resistant to illness. To the novice it often appears that birds show signs of illness one day, are at the bottom of their cage the next, and dead the day after. This misconception has stemmed from two sources.

Firstly, many of the birds seen in practice are only a few generations descended from wild birds. As such, they still retain many of the protective instincts inherited from their forebears. As many of the species kept as companions are relatively low on the food chain, quite a few of these instincts have been developed to avoid drawing the attention of predators. One such instinct is often known as the masking phenomenon. Predators are naturally drawn to prey that looks or behaves differently from others. Unusual colouring, weakness and lameness can single out a bird and make it a draw card for a predator. A natural instinct is therefore to avoid looking 'different': a sick bird will make a determined effort to look healthy, even when there are no predators around (**Fig. 3.1**). The classical 'sick bird look' signs we usually associate with illness (fluffed up, eyes closed, lethargic) only develop when the bird is no longer capable of masking these signs (**Fig. 3.2**). Many of the patients presented to veterinarians are well past the initial stages of their illness and are now decompensating rapidly.

There are many subtle changes in a bird's behaviour or appearance which are indications of a health problem. While these signs are often discernible by experienced owners and veterinarians, they are just as often overlooked by those with less experience. Missing these early signs, when combined with the bird's efforts to mask obvious clinical signs, invariably leads to the late detection of illness and the presentation of the bird *in extremis*. It is important that veterinarians learn to recognise these early signs of illness, and then educate their clients so that illnesses can be detected before becoming too advanced.



Figure 3.1 **An Slatey-Head parrot demonstrates the 'masking phenomenon'. It was eating just minutes before it died.**

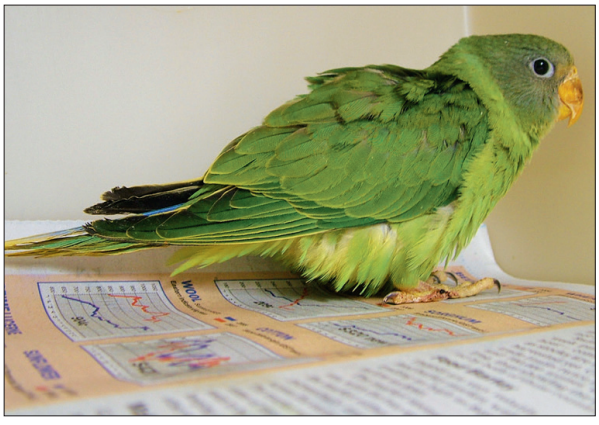


Figure 3.2 **The signs of 'sick bird look' in an Slatey-Head parrot; it is fluffed up and lethargic.**

Downloaded From Vet-eBooks.com

The masking phenomenon and the ease with which early clinical signs can be overlooked highlight the importance of regular health examinations for the companion bird. Long-standing conditions such as malnutrition can be detected and corrected before the bird begins to decompensate and show signs of overt illness.

EXAMINATION ROOM EQUIPMENT

Appropriate equipment for use in the examination room includes:

- A supply of freshly laundered towels of different sizes (or paper towels) for restraining birds.
- Scales capable of weighing in grams, preferably with a detachable T-perch (to allow birds to perch on while being weighed), and a container in which to weigh smaller birds.
- A training perch for the bird to perch on while being examined.
- Clinical equipment such as a stethoscope, a focal light source, magnifying loupes, needles and syringes, blood collection bottles and culture swabs.
- Alcohol for wetting feathers down for a closer examination of the skin and underlying fat and muscle.
- Treats to reward pet birds and make the experience more enjoyable (or at least, less stressful).

The use of heavy gloves to catch and restrain birds should be discouraged. With these gloves on, the clinician cannot be sensitive to small movements of the bird, and can easily hurt or even kill the patient.

Ensure the room is escape-proof, and that clinic staff do not enter the room unexpectedly. Avoid stressful sights and sounds such as dogs, cats and other potential predators.

HISTORY TAKING

The collection of an accurate and thorough history of a patient is as crucial to making a diagnosis as the physical examination and appropriate diagnostic testing. A good history can alert the clinician to likely problems and allow him/her to focus on likely possibilities and refine the rest of the diagnostic approach. History taking can be divided into:

- Background history including:
  - signalment
  - origin of the bird
  - husbandry
  - nutrition
  - behaviour
  - reproductive history
  - previous medical history.
- Presenting problem.

Signalment

The first step in obtaining a history is to gain as much information about the bird itself as possible. A good receptionist or technician can often obtain such information, but the clinician needs to be familiar with birds in general, as often clients are not aware of some basic facts about their bird (such as species, sex and age). Things that need to be ascertained include:

- The species of bird. The clinician needs to be able to recognise common species and have access to literature that will enable him/her to identify other species. Be aware of local names that may differ from those in the literature. Knowing the species, its behaviour and its dietary requirements can offer the clinician vital clues to likely problems.
- The age of the bird can offer other clues. As a general rule, juveniles are more likely to suffer from infectious diseases and nutritional deficiencies, while adult birds are more likely to suffer from neoplasia, chronic malnutrition and degenerative conditions.
- The sex of the bird. Many parrots are sexually monomorphic and require DNA or surgical sexing. Do not accept the owner's assertion of their bird's sex, unless they have proof of sex identification (e.g. a history of egg laying or a certificate of sex identification that can be correlated with this bird). Knowing the sex of a bird can be vital, as many conditions, including behavioural problems, can be linked to the bird's sex.

Downloaded From Vet-eBooks.com

For example, coelomic hernias are almost non-existent in male birds, while being relatively common in females; yolk-related peritonitis is obviously only seen in hens; cocks do not become egg-bound.

Origin of the bird

- How long have the clients owned this bird? Birds that have been in the owner's possession for many years, with no recent exposure to other birds, are less likely to have infectious diseases. Recently obtained birds are more likely to have been in close contact with other birds and, as such, have possibly been exposed to infectious diseases.
- Where did the owner obtain the bird? With experience, the clinician will be able to identify 'problem sources' of birds in the local area (e.g. a certain breeder or a pet shop). Developing a working knowledge of the quality of the sources of pet birds in your area can be a key element of patient evaluation in your practice.
- Is the bird aviary bred or wild caught? Wild-caught parrots, although less common in recent times, still appear occasionally in the pet market. The behaviour and diseases of such a bird may well be linked to its source.
- Is the bird hand reared or parent reared? The health of juvenile birds is dependent to a large extent on the health and nutrition of the parents. Hand-reared birds have two additional factors to be aware of, namely the quality of the hand-rearing formula being fed and the skill of the person doing the rearing. Parent-reared birds tend to be more difficult to tame, unless they are handled on a regular basis before fledging. Hand-reared birds, on the other hand, while usually more closely bonded to people, often have behavioural disorders associated with poor socialisation skills, especially if reared in isolation. If a large number of chicks from different sources are reared in the one facility, there is a higher probability of the spread of diseases such as polyomavirus and *Chlamydia*. All of these factors need to be assessed during the history collection, especially when examining juvenile birds.

Husbandry

- How has the bird been managed? Is it an aviary bird, a companion bird or a zoological specimen? An assessment of a companion bird's husbandry must include the cage, the environment around the cage and the bird's interaction with its environment (**Figs 3.3, 3.4, 3.5a** and **b**).
- Ascertain whether the cage in the examination room is the bird's permanent cage or simply a transport cage. If the latter, ask the owner to



Figure 3.3 **Aviaries that allow for full flight enable birds to have greater movement in all directions and provide a more 'natural' environment. Good hygiene practices are essential to prevent health issues amongst the birds such as infections and parasitism.**



Figure 3.4 **Suspended aviaries are usually parasite free, but it can be argued that they limit birds' ability to exhibit normal behaviours.**

Downloaded From Vet-eBooks.com





Figure 3.5a,b **An example of an inappropriately sized and poorly cleaned cage.**

describe the permanent cage or to bring along photographs or video images.

- Where is the cage located in the house? Is it exposed to toxins such as burning Teflon®, cigarette smoke or household plants? Does the bird get any privacy? Is it kept isolated, away from family activities? Does it get a good night's sleep or is it forced to stay up with its owner watching television all night? Does the bird get access to direct, unfiltered sunlight on a daily basis? If the bird is kept outside, is it safe from predators (including wild birds) and from vectors of diseases such as *Sarcocystis*?
- Does the bird come out of its cage? How long does it have outside the cage each day? Are its wings clipped? Is it supervised when out of the cage? Does it interact with other animals and birds?
- Does the client have other birds? Any other pets?

See Chapter 2, Husbandry, Grooming and Nutrition for a full discussion on the husbandry of both companion and aviary birds.

Nutrition

Underlying many health problems in pet birds is a common thread of malnutrition. For many generations, bird owners have accepted as fact that birds eat seed, and that is all they need (**Fig. 3.6**). Birds, as with many other animals, have a preference for high-fat diets. Given a choice between seed, vegetables, formulated diets and fruits, nearly all birds will consume the seed first. Given this preference, it is not surprising to hear many bird owners state, 'All he will eat is the seed, so that is all that I give him.' See **Figs 3.7** and **3.8**.

It is therefore important to ascertain:

- What does the owner feed the bird and what does the bird actually eat? The clinician needs to be aware that there are major species differences in dietary requirements with, for example, some species having a higher requirement for fat than others. There is no one diet to suit all avian species, any more than there is one diet to suit all mammalian species.

Downloaded From Vet-eBooks.com



Figure 3.6 Wild short-billed corellas feeding on bamboo. Contrary to popular belief, wild birds eat a wide range of foods, and not just seeds.



Figure 3.7 Example of a commercially available all-seed, high-fat bird food mix.



Figure 3.8 A cockatiel eating a mix of vegetables, pellets and seed.

- How much food is being consumed? (Some birds on an excellent diet will still eat too much and gain excessive weight.)
- Is the food prepared fresh daily?
- Are dishes cleaned each day?
- Does the bird dunk food into its water dish, creating a nutrient-rich broth ideal for bacterial contamination?
- Does the bird get any treats such as food off their owner's plate?
- Are vitamin and mineral supplements being offered?

Paediatric cases open another spectrum of questions. What hand-rearing formula is being used? How is it prepared? Are the manufacturer's recommendations being followed? Has anything extra been added to an already balanced diet? At what temperature has it been fed? How much has been fed, and how often? How has the chick been fed: syringe, crop tube or spoon? How are these utensils cleaned and disinfected?

See Chapter 2, Husbandry, Grooming and Nutrition for a full discussion on the nutrition of companion and aviary birds.

Behaviour

A behavioural history is becoming increasingly more important as pet birds move out of their cages and more into their owners' lives. Just as countless dogs and cats are euthanased every year because of behavioural problems, many birds suffer the same fate, or are transferred from household to household, for the same reasons.

As psittacine behaviour is determined to a large extent by the interaction between the bird, its owners and its environment, questioning must focus on these areas:

- How many hours per day does the bird spend alone?
- What does the bird see and hear when it is alone?
- Does the bird spend time with other birds or other pets?
- Are toys provided for the bird? Does it utilise those toys?

Downloaded From Vet-eBooks.com

The bird's interaction with its owners (human flock) will need to be clarified:

- Who is the primary caretaker?
- Who does the bird seem to prefer?
- Does the bird dislike anyone?
- How tame is the bird?
- Does it readily step up on to a proffered hand?
- Does it always try to move up on to a person's shoulder? Is this allowed or encouraged?
- Does the bird talk?
- Does it like to be petted? Where?
- How does it react to different family members?
- How does it react to strangers?

Reproductive history

It is important to ascertain if the bird has displayed any reproductive activity. If it is a hen, has it engaged in nesting or territorial behaviour, or has it laid eggs? If a cock bird, has it displayed courtship behaviour towards another bird or even the owner?

Previous medical history

The patient's medical history should be examined. Has the bird been ill before? Who saw it, what did they diagnose, and how was it treated? Has it had remedies supplied by pet shops or breeders? Is the bird being presented for a second opinion or a specialist referral? If appropriate, permission to obtain copies of medical records from the previous (or referring) veterinarian should be requested.

Presenting problem

- What exactly is the problem? This needs to be clarified with the client, as confusion often exists between owners and veterinarians over the precise description of clinical signs.
- When did it start?
- What effect is the problem having on the bird's appetite, thirst, urination, defecation, and behaviour?
- Is this the first time it has happened?
- Are other birds affected?
- Have other treatments been tried? Who prescribed these treatments? Did they work?
- Is this condition progressing or static, or perhaps even improving?

The history taking described above is not a comprehensive review of every possible question that can be asked of the owner. As the process continues, areas of interest will become apparent and more questions may be needed to clarify these areas. The clinician must take care to ensure that he/she does not dominate the conversation; rather, ask short questions and listen carefully to the client's reply. However, the clinician must be prepared to guide the discussion, as otherwise some clients may become distracted and lose track of the original question!

THE DISTANT EXAMINATION

Although the history taking should be done before the bird is handled, the clinician should be using this time to gain some insights into the bird and its health by careful observation of the bird, its cage and its droppings.

The bird

- Most birds, no matter how ill they are, will make an effort to mask their clinical signs when first brought into the examination room. This effort will rarely last long, usually only a minute or two. Avoid disturbing the bird until it has settled, otherwise valuable clinical signs may be overlooked.
- Watch the bird's breathing. Once the bird has settled in its cage in the examination room, there should be no open-mouth breathing, marked tail bobbing or marked respiratory effort. There should be no audible respiratory noise. The presence of these signs should alert the clinician to the likelihood of respiratory compromise and great care must obviously be taken with handling such a patient.
- Look at the bird's posture. Many sick birds are hypothermic and attempt to conserve body heat and energy by fluffing their feathers, sitting still and sleeping more. These signs are often referred to as the 'sick bird look', but not all sick birds will display these signs. Evidence of a wing droop, lameness or reluctance to bear weight on one leg indicates a musculoskeletal problem. Spinal deformities can often be detected by an abnormal positioning of the tail. An upright

Downloaded From Vet-eBooks.com

position with a wide-legged stance may indicate egg-binding or a similar space-occupying lesion in the coelom. Birds holding both wings away from their body and panting are usually heat stressed.

- Examine the plumage. Normally it should be sleek and well groomed, and should be clean. Untidy or dirty plumage can indicate either that the bird is not grooming itself for some reason or that there is a feather dystrophy of some description. Discoloured feathering can reflect a variety of problems, such as Psittacine Beak and Feather Disease (PBFD), chronic liver disease, excessive handling with oily hands or malnutrition.
- Examine the beak and toenails. Overgrown or flaky beaks or overgrown and twisted nails can be associated with PBFD (in cockatoos), poor husbandry, chronic liver disease or malnutrition.
- Watch the bird defecate. Look for signs of straining or discomfort and listen for any accompanying flatulence.
- Observe the bird's behaviour, assessing how tame the bird is and whether it is showing any overt sexual display towards the owner or other people present in the room. Look at how the owner interacts with the bird, as this can give valuable clues to relationships at home.



Figure 3.9 **A cockatoo with badly damaged primary feathers. A consequence of this bird being housed in an inappropriately small cage leading to its wings striking the bars.**

The cage (see Chapter 2, Husbandry, Grooming and Nutrition pp. 45)

- Is the cage large enough (**Fig. 3.9**)?
- Is it constructed of safe materials?
- Is the floor of the cage solid or wire? Is it covered in grit, sand or paper?
- What sort of perches and dishes are in the cage, and how are they positioned?
- Are any toys appropriate for the bird and not overcrowding the cage?
- Is the bird offered the opportunity to bathe, or at least be misted, on a daily basis?

Faecal examination

Birds' droppings are made up of three components: faeces, urates and urine (**Fig. 3.10**). In a healthy bird the faecal portion should be formed and homogeneous, with little odour (except for poultry,



Figure 3.10 **Example of 'normal' droppings from a parrot.**

Downloaded From Vet-eBooks.com

waterfowl and carnivorous birds). The colour should range from brown to green. The urates should be a crisp white and slightly moist. Old droppings will have greenish urates as biliverdin leaches out of the faeces. The urine should only extend a couple of millimetres past the dropping. True polyuria should not be confused with so-called 'excitement polyuria', the excess urine produced by an excited or nervous bird. Lorikeets, due to their liquid diet, will produce large amounts of urine, which should not be mistaken for pathological polyuria. A close examination of the droppings is a valuable starting point to a clinical examination.

Some abnormalities commonly encountered include:

- Diarrhoea (unformed faecal portion).
- Undigested food in faeces (**Fig. 3.11**).
- Very bulky droppings indicate maldigestion, malabsorption, reproductively active hens, coelomic growth or pelleted diets (**Figs 3.12** and **18.1**).
- Melena or very dark droppings can indicate anorexia or intestinal haemorrhage (**Fig. 3.13**).
- Malodorous droppings are often associated with bacterial/fungal overgrowth.
- Green urates (biliverdinuria) are often indicative of liver disease (**Fig. 3.14**).
- Pink/red urates (haematuria/haemoglobinuria) are seen in cases of renal disease often associated with lead poisoning, especially in Amazon parrots and galahs (**Fig. 3.15**).
- Yellow urates can be associated with anorexia.
- Orange urates indicate that a vitamin B injection may have been given in the last few hours.
- Thick, pasty urates are seen in dehydrated birds.
- Polyuria (excessive urine – be aware that this is normal in lorikeets and birds eating a lot of fruit).
- Anuria.
- Discoloured urine: similar causes to discoloured urates (**Fig. 3.16**).
- Fresh blood indicates either a cloacal problem or oviductal disease.

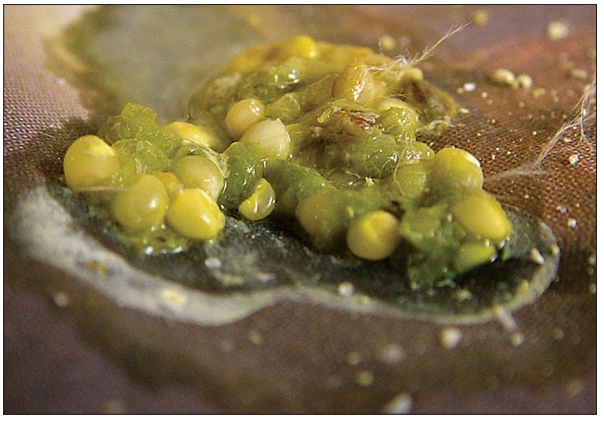


Figure 3.11 **Whole, undigested seed seen in droppings from a Sun conure could indicate a maldigestion issue.**



Figure 3.12 **Bulky droppings from a cockatiel are indicative of a number of health issues requiring investigation.**



Figure 3.13 **Very dark droppings known as melena can indicate anorexia or intestinal haemorrhage.**

Downloaded From Vet-eBooks.com



Figure 3.14 Green urates (biliverdinuria) as seen associated with these droppings are often indicative of liver disease.



Figure 3.16 Discoloured urine (biliverdinuria and polyuria) from a parrot suggests this bird has liver disease.

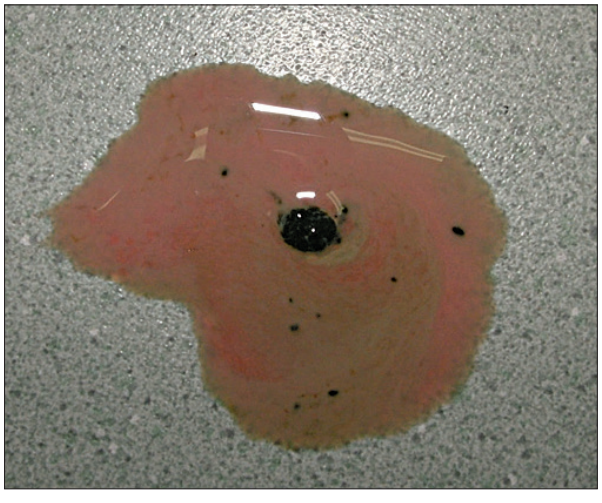


Figure 3.15 Pink or red urates as shown indicate haematuria or haemoglobinuria, often associated with lead poisoning, especially in Amazon parrots and galahs.

THE PHYSICAL EXAMINATION

A thorough, systematic physical evaluation of the patient is essential to obtaining clues about the bird's problem and diagnosis. Clinicians should develop a thorough examination protocol that they are comfortable with, and use it for every patient regardless of the reason for presentation.

Handling and restraint

At some time during every examination the veterinarian must handle, and sometimes restrain, the patient. Traditionally, it was considered acceptable to put on a heavy pair of gloves, pin the bird to the table top or the side of the cage and then drag it, struggling and screaming, into a position where it could be better examined. Undoubtedly this stress, imposed on an already compromised patient, contributed to the myth that birds were 'soft' animals, prone to dying while just being examined!

Just as it is inappropriate to muzzle and grapple with every dog and cat that comes into a veterinary surgery, it is inappropriate to use heavy-handed restraint on a companion bird. Many of these birds have learnt to trust humans and regard them affectionately. Destroying this trust through aggressive catching and handling techniques can adversely affect the bond between owner and bird.

Downloaded From Vet-eBooks.com

This relationship must be preserved and handling techniques for closely bonded birds should emphasise minimal stress and fear.

As the oils on human skin can be detrimental to the feathers of many species, a light dusting of talcum powder on the clinician's hands is appropriate before beginning an examination. This is particularly necessary for those birds that do not produce powder down (e.g. Amazons, eclectus parrots and lorikeets). Handled frequently without talcum powder, the green feathers on these birds begin to acquire a black discoloration that can become quite unsightly.

If the bird is presented in a cage or carrier, it is often more appropriate to ask the owner to remove the bird from its cage. Doing so will often relax the bird in a new environment. If the owner is unwilling (or unable) to do so, the clinician should firstly offer the bird the opportunity to step out of the cage by itself. If it is unwilling to do so, an attempt should be made to bring the bird out on the clinician's hand (if the cage door is large enough). A hand is slowly and gently introduced into the cage, with the back of the hand to the bird. If there is no aggression, the forefinger or hand is extended and placed under the bird's chest. A tame bird will usually step on to the finger or hand. If possible, one toe is restrained by gently pressing on it with a thumb against the finger, keeping the bird steady, and it is gently brought out of the cage. During this procedure the clinician should keep talking to the bird, praising it and maintaining eye contact. When the bird is out of the cage, one should continue to talk to it and praise it, scratch its ear and generally make a fuss of it.

If the bird will not step on to a hand, it is best to use a small hand (or paper) towel to gently envelop and then restrain the bird. The bird is shown the towel and allowed to get used to it. If possible, the clinician should slowly envelop the bird in the towel from below; an approach from above is potentially a very intimidating experience for a pet bird. The clinician should keep talking in a friendly voice and maintain eye contact.

Birds that are not tame can be caught using a towel as described above. These birds will rarely stay still during capture, so a quick capture is the best approach. A few seconds spent removing obstacles in the cage (food and water containers, perches, toys) is a worthwhile endeavour, as it will reduce the bird's opportunities to avoid capture.

Once the bird has been removed from the cage, the next step will be determined by how tame it is. Very tame birds can be placed on a T-perch for further examination and weighing; less tame birds may need to be examined while restrained in the towel.

At all times the clinician must be aware of the bird and how it is handling the stress of restraint and examination. As many birds are presented for evaluation of an illness, and they have been ill for some time, even being restrained can add extra burden to an already overburdened body. Collapse and death are, unfortunately, not uncommon with critically ill birds. (If the bird appears critically ill when first presented, it is often worthwhile having oxygen and mask on hand to assist with resuscitation if the bird collapses.) If there is any doubt as to the bird's ability to cope with the stress, it should be immediately returned to a perch or the cage and be allowed to regain its composure before proceeding.

Once the examination is complete the bird can be gently replaced in its cage the same way it was removed. The whole procedure should be conducted with a minimum of stress, noise and excitement.

Weight recording

A vital aspect in avian medicine is the accurate recording of the patient's weight. All birds should be weighed on each visit to the veterinarian and each day while hospitalised. An accurate weight record allows the clinician to quantify the bird's body condition (if normal weights for that species are known), to accurately calculate drug dose rates and to monitor the patient's response to therapy (if it is weighed daily). Over a period of time the clinician will also develop a working knowledge of expected body weights for different species.

Weights should be recorded to the nearest gram. As such, a veterinary practice dealing with birds

Downloaded From Vet-eBooks.com



Figure 3.17 **Using a T-perch on a set of scales allows for easy weighing of most pet birds.**

will need to invest in a good quality digital scale, readily available in many electronics or kitchenware stores.

Once practised, the weighing of an avian patient is relatively easily performed. Larger birds can be weighed by having them stand on a T-perch mounted on the digital scale (**Fig. 3.17**). Smaller birds such as budgerigars, canaries and finches may be placed inside a metal or plastic container similarly mounted on the scale.

The patient's weight must be recorded on its medical records in order to demonstrate to clients their pet's weight gain or loss, and to provide part of that patient's minimum database.

Auscultation

When to auscultate is the prerogative of the clinician, but it may be better performed before the bird is handled for too long. The heart rate is usually rapid, although that of some larger pet birds (e.g. cockatoos) can be surprisingly slow compared with stressed or smaller birds. Murmurs, arrhythmias and bradycardia are occasionally detectable.

Movement of air through the lungs and air sacs can be auscultated over the bird's back and occasionally friction rubs associated with air saculitis can be detected.

Body condition

Traditionally a bird's body condition was determined by palpation of the pectoral muscles and allocating a body score based on the muscle and fat coverage of the sternum. This technique fails to take into account that most birds do not store fat in their pectoral region and they can be carrying significant fat deposits while still having an apparently good body score. Wetting the feathers over the ventral coelom and flanks with alcohol allows visualization of subcutaneous fat deposits, seen as yellow fat under the skin rather than pinkish-red muscle. Coelomic fat deposits can be evaluated by palpation.

The combination of bodyweight recording, pectoral muscle and coelomic palpation and examination of subcutaneous fat allows for a more accurate assessment of body condition than just palpation of the pectoral muscles. Many avian veterinarians prefer not to use a Body Conditioning Scoring scheme, as there is no international consensus or consistency.

Skin and plumage

The bird's skin and feathering should be examined in detail. Attention should be paid to the following areas:

- Colour of the feathers. Normal coloration of feathers is the result of pigment deposited in the feather keratin and the reflection of light from the feathers. Abnormal coloration of feathers results from anything that affects the pigment deposition or the ultrastructure of the barbs and barbule. PBFD can cause green feathers to turn yellow and blue feathers to turn white. It will also lead to a generalised dirtiness of the feathers,

Downloaded From Vet-eBooks.com

especially in cockatoos. Chronic liver disease and/or malnutrition can cause darkening of feathers. As mentioned earlier, frequent handling of green birds by the owner can cause a black discoloration on their feathers. This is not seen in birds with powder down, presumably because the powder keeps the feathers clean.

- Tidiness of the plumage. Birds generally keep their plumage well-groomed and tidy. If the plumage is untidy, with no immediately obvious cause (e.g. recent handling), the clinician should suspect that either the bird is unable to groom itself properly, or a generalised feather dystrophy (e.g. PBFD) is present.
- Evidence of feather damage. Chewed and/or broken feathers should lead the clinician to suspect over-grooming, self-mutilation or malnutrition. Saw-toothed edges can indicate a failure to moult normally, hence old, worn feathers are being retained. It should be noted in cases of feather picking whether the feathers have been bitten off level with the skin or plucked out, or if the shaft has been chewed.
- Evidence of feather dystrophies. Retained feather sheaths, retained pulp, haemorrhage in the shaft of feathers, strictures of the calamus and twisted feathers are indicative of feather dystrophies, often of viral origin (e.g. polyomavirus, PBFDV).
- Wing clipping (if present). The wings should be examined to determine if the bird's wings have been clipped and, if so, if that clip is appropriate to the species of bird. The quality of the clip should be examined to determine if the cut ends of feathers could be bothering the bird (**Fig. 3.18**).
- Absence or presence of powder down. Powder down is produced by the powder down feathers on the thighs of many species of birds, particularly cockatoos and African grey parrots. It is easily recognised by the presence of a fine white powder on the clinician's hands and clothing after handling the bird. A lack of powder down leads to staining of the feathers and a shiny appearance of the beak and feet. The most common cause of a loss of this powder is PBFD, but it can be seen in obese birds, elderly birds, and birds that have chewed at their powder down feathers.



Figure 3.18 **Injuries to the wing of a cockatiel due to inappropriate and excessive wing trimming.**

- Moulting patterns. Most birds will typically moult heavily twice yearly, in spring and autumn, the so-called 'prenuptial' and 'postnuptial' moults. Outside of these annual moults there is a steady and progressive turnover of old feathers. Continual heavy moults or the sudden loss of many feathers is abnormal, as is the failure to moult (seen as the retention of worn and broken feathers).
- The presence of stress lines. Stress or disease at the time a feather is growing will lead to a transverse 'break' in the vane of the feather. The presence of many feathers with such stress lines is indicative of a problem in the bird's recent past. The black marks left on green-coloured birds by human skin oils (see above) should not be confused with stress lines.
- Birds that are unable to groom properly (e.g. spinal disease or wearing an Elizabethan collar) may develop overgrown powder down feathers, retained in their shafts over the thighs. This should not be confused with a feather dystrophy such as that seen with diseases such as PBFD.
- The condition of the skin. The presence of erythema, excessive scale or areas of skin trauma should be noted. This can be done by parting the feathers with a cotton bud or gently blowing on the feathers.

Downloaded From Vet-eBooks.com

- Areas of trauma. The skin should be thoroughly examined for areas of trauma, especially on the wing tips, sternum and axillae.
- Flexibility of the feather. The feather of a healthy bird on a good diet should flex, rather than bend, when the tip is drawn down towards the base, and then spring back to a normal position when released.
- Parasites. The presence of parasites on the feathers should also be noted. Microscopic examination of a freshly plucked or moulted feather may be needed.

It is important that changes in feathers and skin be recorded in a detailed manner. Veterinarians should familiarise themselves with the descriptive terminology used for the external anatomy of a bird, and use that when describing lesions. In addition, it is important that lesions be described accurately (e.g. whether feathers are been stripped, chewed, plucked or bitten off). Such precise terminology is essential when describing a case to other veterinarians and can assist in developing a clearer picture for all concerned.

The uropygial (or preen) gland is located on the dorsal base of the tail. (See Chapter 1, Clinical Anatomy and Physiology p. 1). It should be assessed for evidence of impaction, enlargement, inflammation or trauma.

Head

- Look for asymmetry arising from sinus swellings, exophthalmos, enophthalmos and trauma.
- Loss of feathers on the head can be due to a variety of conditions. Some cockatiel mutations, especially lutinos, have a bald spot behind the crest. Feather loss in other species can be associated with fungal dermatitis, *Cnemidocoptes* infection, PBFD or excessive grooming by a cage mate. Feather loss around the eyes can indicate facial rubbing associated with conjunctivitis or sinusitis.
- Matting of the feathers over the crown and nape can indicate that the bird has been vomiting (**Fig. 3.19**).
- The conformation of the beak should be assessed for the presence of congenital or acquired abnormalities such as scissor (wry) beak, prognathism and bragnathism. Trauma to the beak or localised sinus infections can result in localised anatomical abnormalities (e.g. longitudinal grooves in the keratin). Excessive keratin flaking of the beak can reflect poor nutrition or simply a lack of opportunity to rub the beak on a suitably abrasive surface (such as a cement perch). Overgrowth of the beak can occur with PBFD, *Cnemidocoptes*, misalignment of the upper and lower beaks, chronic liver disease or malnutrition. It is rarely the result of a lack of objects to chew on. (It is important to note that some species such as the Long-billed Corella naturally have elongated beaks. This should not be mistaken for an overgrown beak.) Underrunning of the ventral surface of the rhinotheca is commonly seen in cockatoos with PBFD, but can also be seen in birds with bacterial or fungal infections in the keratin.
- The cere, the fleshy skin at the top of the beak, is not present in all species. In the budgerigar cere colour can be used to sex the bird, with cocks having a blue cere and hens a brown cere. However, this will vary with the age of the bird, the colour mutation and the degree of health. Cere hypertrophy, a thickening of the brown cere in the budgerigar hen, may reflect a hyperoestrogenic state.



Figure 3.19 **A cockatiel presenting with matted head feathers suggests it may have been vomiting.**

Downloaded From Vet-eBooks.com

- The nares, the openings into the sinuses located at the top of the beak, should be symmetrical and dry. They should be open and the presence of keratin plugs (rhinoliths) is abnormal. Blockage of the nares may result in a subtle movement of the skin over the infraorbital sinuses. This may be the only clue that a blockage exists. Discharge from the nares, often seen as staining and matting of the feathers dorsal to the nares, may be an indication of upper respiratory disease. Previous or chronic episodes of sinusitis may be seen as asymmetrical enlargement of the nares, sometimes associated with a longitudinal groove in the keratin of the beak.
- The eyes should be bright and clean. Ocular discharge and loss or matting of the feathers around the eyes indicates either conjunctivitis or sinusitis. Conjunctival hypertrophy is common in chronic conjunctivitis, especially in cockatiels with *Chlamydia* infection. Focal light, magnification and fluorescein dye are needed for a detailed ocular examination. See Chapter 12, Disorders of the Eye for a detailed discussion of the ocular examination.
- Examination of the oropharynx can be accomplished by using gauze bandage or metal gags to open the mouth. The choana (the slit on the roof of the oropharynx) should be free of excessive mucus or discharge, and fringed with well-defined papillae. There should be no abscesses or diphtheritic membranes present.
- The ears can be examined by parting the ear coverts with the handle of a cotton bud or similar appliance. The ears should be open and free of discharge or erythema. Visualization of the tympanic membrane is difficult in most species without the use of an endoscope.

Crop

The crop can be palpated in most birds at the base of the neck, just cranial to the thoracic inlet. It should be carefully and gently palpated to assess if:

- Food is present (i.e. is the bird eating?).
- It feels doughy or fluid-filled, indications that crop stasis may be present.
- Ingluvoliths or other foreign objects are present.
- The crop mucosa feels thickened.

Care must be taken in critically ill birds not to force fluid or ingesta back up the neck into the oropharynx, as aspiration and death can result.

Body

Palpation of the skin over the trunk occasionally reveals the crackling of subcutaneous emphysema. While this is normal in species such as pelicans, in most species it is often the result of trauma or infection rupturing air sacs and allowing the escape of air under the skin.

The coelom in the normal bird is concave between the end of the sternum and the pubic bones. The clinician needs to distinguish between internal and external distension of the coelom. Internal distension of the coelom can be due to fat, organ enlargement, ascites, neoplasia or the presence of an egg. External distension can be due to subcutaneous fat, neoplasia (especially lipomas) or hernias. Radiography and ultrasonography may be required to distinguish between internal and external coelomic distension, and between different aetiologies of both. Coelomic pain or discomfort can occasionally be elicited by careful palpation.

The back should be carefully palpated for evidence of scoliosis, lordosis or kyphosis. As the notarial vertebrae are predominantly fused, flexibility of the spine cannot be assessed in the same way as it is in dogs and cats.

The carina of the sternum should be palpated for evidence of distortion, trauma or congenital defects such as splitting. Distortion of the carina, often indicating a history of rickets or other metabolic bone disease, should lead the clinician to recommend radiographic evaluation of the rest of the patient's skeletal system.

The cloaca can be assessed externally for enlargement and dilation (often indicative of reproductive behaviour in a hen), prolapse, ulceration or inflammation around the mucocutaneous junction, and the presence or loss of sphincter tone. Gently everting the cloaca can give a cursory examination of the mucosa, possibly revealing papillomas in

Downloaded From Vet-eBooks.com

susceptible species. A more thorough evaluation of the cloaca requires endoscopy.

The area between the cloaca and the tail should be assessed for splitting of the skin. This is commonly seen in pet parrots (especially cockatiels) and is associated with a poor diet and a poor wing clip. The wing clip causes the bird to land awkwardly, pushing its tail up as it does so. Malnutrition causes the skin to lose its elasticity. The result is that the skin in this area splits, leading to bleeding and feather picking in this area. It is also a common area for hernia in hens, often containing the oviduct or even cystic ovaries, and for lipomas in obese birds.

Wings

Each wing should be carefully extended and flexed to assess mobility and compared with the other wing. The bones and joints should be palpated for swelling or crepitus. Recent trauma may be evident as greenish discoloration of the soft tissue. This is bruising, and should not be mistaken for infection or tissue death. If the cause of a wing droop is not detectable after careful palpation, radiography is required to assess the pectoral girdle. The bones of this girdle are covered in strong muscles, and fractures are often not detectable by palpation alone. The patagium should be evaluated for loss of elasticity, trauma or scarring and for the presence of a tattoo (indicating the bird has been surgically sexed).

Legs

Each leg should be carefully palpated to detect abnormalities such as fractures, swelling (**Fig. 3.20**) and bruising, healed calluses and angular deformities of the long bones. Each joint should be extended and flexed to assess mobility and range of movement. Joints should also be examined for swelling and deposition of chalky white uric acid crystals (i.e. articular gout). Each leg should be compared with the other in all aspects (e.g. symmetry, length, swellings).

The toes should be examined for abnormalities including:

- Missing digits or nails.
- Annular constrictions.
- Swelling of interphalangeal joints, occasionally with the deposition of uric acid crystals.



Figure 3.20 **Injury and swelling of the leg of a Jenday conure due to a leg band constriction.**

- Avascular necrosis.
- Excessive thinness, especially in neonates.
- Abnormal position and conformation of the toes.
- Excessively long or twisted nails.

The plantar surface of each foot should be examined and the condition of the metatarsal pad and digital pads noted. Abnormalities seen here include loss of definition of the epidermis (seen as a shiny, reddened surface), swelling, erosions, ulcers and scabs. Pododermatitis (bumble foot) is common in raptors, but can be seen in any bird where a unilateral lameness causes more weight-bearing on the unaffected leg. This in turn can lead to pressure necrosis, infection and subsequent pododermatitis. Consequently, in cases of a unilateral lameness, the opposite leg should always be closely examined.

In some birds, especially overweight specimens, calluses and dermatitis can also be seen on the caudal aspect of the tarsometatarsus. This may be the result of improper perches and a bird that cannot perch normally because of its weight.

Occasionally, due to the nature of the injury or the disposition of the bird, a full examination may require general anaesthesia. If this is the case, radiographs can be taken at the same time, minimizing handling of the conscious (and therefore stressed) patient.

Downloaded From Vet-eBooks.com

Neurological assessment

Birds presented for any of the following problems require a thorough neurological assessment:

- Abnormal posture or behaviour.
- Abnormal mentation.
- Paresis or paralysis of any or all limbs.
- Fractures of limb bones.
- Weakness or inability to grip with one or both feet.
- Other neurological abnormalities.

The assessment should be performed methodically and logically. The order in which the assessment is performed depends on the clinical condition and cooperation of the patient. The degree of cooperation should be considered when interpreting responses to neurological tests.

The first part of the neurological assessment is to step back and observe the bird's posture, mentation, flight, gait and behaviour. (This is discussed in more detail in Chapter 22, Diseases of the Nervous System.) The bird is then caught and examined physically. In particular, the tone of the wing and leg musculature is evaluated. Muscle atrophy is suggestive of reduced innervation. The skeletal system is palpated for evidence of crepitus or other abnormalities.

A systematic evaluation of the nervous system is then conducted.

Cranial nerve assessment

- The menace response, evaluating cranial nerves II and VII, can be provoked by bringing the hand towards each eye. Normal responses include eye-blink, pulling away of the head or aggressive action of the beak.
- The pupillary light response evaluates cranial nerves II and III. It is often considered unreliable in birds because, although there is complete decussation of the optic nerves at the chiasma, and therefore no consensual pupillary light response, the thin bones of the avian skull can allow a light shone in one eye to stimulate a response in the other eye.
- Pupil size. The pupils should be symmetrical. If not, consider intraocular inflammation, ocular structural lesions or sympathetic neuropathy affecting cranial nerve III.
- A symmetrical eye-blink normally occurs when the cornea is gently touched with a moist cotton swab. If not, consider an abnormality affecting cranial nerves V and VII.
- A symmetrical eye-blink should be elicited by touching each side of face or the lateral canthus. This evaluates cranial nerves V and VII.
- The tongue should be positioned and move normally. Abnormal tongue movement or a deviated tongue indicates damage to cranial nerves IX to XII.
- Reduced beak strength when biting or eating indicates possible damage to cranial nerves V, IX, X, XI and XII.
- Nystagmus, the periodic, rhythmic, and involuntary movement of both eyeballs in unison in a vertical, horizontal or rotary direction, indicates damage to cranial nerve VIII, a cerebellar lesion or increased intracranial pressure.
- Strabismus, an involuntary deviation of one eye, can occur with damage to cranial nerves III, IV or VI and vestibular lesions.
- Horner's syndrome (enophthalmos, upper eyelid ptosis, slight elevation of the lower lid, piloerection of feathers on the affected side of head, pupil constriction and narrowing of the palpebral fissure) has been reported with intracranial lesions or lesions affecting the cervical sympathetic tract or brachial plexus. Note that miosis and third eyelid protrusion are not as obvious in birds as in mammals because of the presence of striated muscle.
- Torticollis, cervical muscle contraction producing neck torsion, has been seen with lesions involving cranial nerve XI.

Peripheral nerve assessment

Spinal reflexes, although difficult to assess objectively in birds, can help determine if a lesion is centrally or peripherally located. In most situations determining if a reflex is present or absent is sufficient, and symmetry of response should receive particular attention. Spinal reflexes require reflex arcs only to

Downloaded From Vet-eBooks.com

be intact; no other parts of the central nervous system (CNS) are involved. Spinal reflex tests include:

- Body balancing. With the wings held into the body, suspend the bird vertically and head down, then quickly rotate up to a horizontal position and observe fanning of the tail feathers. Dip the bird forward again, back to the vertical position, and observe the tail flick up. This reflex functions to maintain balance.
- Wing withdrawal. Lightly touch a wing and observe it being pulled away. This is a segmental reflex which, if present, indicates that the reflex arc and associated spinal cord segment in the cervico-notarial cord are intact. Damage may still exist higher in the CNS.
- Leg withdrawal. Lightly touch a leg and observe it being pulled away. This is again a segmental reflex; if present it indicates that the reflex arc and associated segment in the notarial spinal cord are intact.
- Vent reflex. Touch the vent mucosa with a fine object and observe it close tightly. This is also a segmental reflex that indicates that the reflex arc and associated synsacral spinal cord segment are intact.

Reactions require reflex pathways (ascending and descending fibre tracts in the spinal cord and higher centres). It is important to look closely for subtle asymmetrical deficits.

Comparison of spinal reflexes and reactions helps to localise lesions. If a reflex exists, but its corresponding reaction does not, a lesion exists within the CNS rostral to the segment involved in the reflex arc. Reactions include:

- Proprioception (wings). Note the resting wing carriage. Pull one wing out of its resting position and note the time taken for its return. Only normally innervated wings will correct displacement.
- Proprioception (legs). Observe the bird at rest. Knuckling of the foot is quickly corrected in normal birds.
- Unilateral and bilateral wing fanning should be assessed while the bird is moved up, down and from side to side, while the feet and lower back are restrained. Forceful, rhythmic fanning out of the wings should be evident and, in the bilateral test, wing movements should be simultaneous. Unilateral wing fanning will occur only in the normally innervated wing, while in the bilateral test a lesion causing loss of afferent stimulus on one side will be compensated for by afferent stimulus from the other side and normal wing movements may occur.
- Placing. With wings held into the body and the legs free, approach a horizontal surface (such as a desk top). As soon as any part of the foot touches the surface, both feet should swiftly position themselves accurately on the surface in order to support the bird's weight.
- The differentiation between pain perception and withdrawal reflex is critical. Movement of a pinched limb does not indicate that the patient is able to feel the stimulus. Some type of conscious recognition of the stimulus is required (e.g. vocalisation, attempts to escape or bite). This part of the examination is best kept till last, so as not to influence the patient's behaviour to other segments of the neurological examination. Loss of pain perception is a poor prognostic sign. Deficits in a particular wing may only be obviously detected if its counterpart is restrained.

FURTHER READING

Clippinger TL, Bennett RA, Platt SR (1996) The avian neurologic examination and ancillary neurodiagnostic techniques. *Journal of Avian Medicine and Surgery* **10(4):**221–247.

Doneley B, Harrison GJ, Lightfoot TL (2006) Maximising information from the physical examination. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing, Palm Beach, pp. 153–212.

Greenacre CB, Lusby AL (2004) Physiological responses of Amazon parrots (*Amazona* species) to manual restraint. *Journal of Avian Medicine and Surgery* **18(1):**12–18.

Platt SR (2006) Evaluating and treating the nervous system. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing, Palm Beach, pp. 493–518.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 4

CLINICAL TECHNIQUES

After the physical examination, a range of diagnostic tests can be employed to move closer to a diagnosis. At the same time the patient must be treated. Both of these procedures require the clinical techniques to achieve the best outcome. This chapter describes the clinical techniques used in diagnostic testing and administering treatment. Interpretation of these tests and supportive care are described in later chapters.

DIAGNOSTIC TECHNIQUES

The limited range of physical responses to disease that is available to birds, and their relatively small size, has increased the requirement for diagnostic testing in order to better diagnose disease and provide an appropriate treatment. The full gamut of testing available in small animal medicine is also available for avian practitioners. However, before any diagnostic test can be interpreted, it is vital that the clinician understands how to, and be able to, collect quality samples and position the patient correctly for diagnostic imaging.

Blood collection and handling

There are a number of haematological, biochemical, serological and polymerase chain reaction (PCR) tests available for avian veterinarians to utilise in the care of their patients. The basic requirement for these tests is to collect a sufficient volume of non-haemolysed blood without endangering the patient. Secondary requirements include storing the collected sample correctly and making fresh blood smears without damaging the relatively fragile nucleated erythrocytes.

Blood can be collected from the right jugular vein (**Fig. 4.1**) (the left jugular vein is accessible, but is much smaller), the basilic vein (on the medial aspect of the elbow) or the medial tibiotarsal vein (in large birds) (**Fig. 4.2**). The right jugular vein is usually preferred in companion birds because of the relatively easy access; it lies under an apteryla and, with practice, a sole operator can both restrain the bird



Figure 4.1 **The right jugular vein visible in this image can be found under an apteryla on the right side of the neck.**



Figure 4.2 **A cockatoo with an intravenous catheter placed in the medial tarsal vein.**

Downloaded From Vet-eBooks.com

and perform the venepuncture. It must be remembered that avian veins have thin walls and tear easily. Coagulation in birds usually relies on extrinsic clotting pathways requiring tissue thromboplastin, rather than the intrinsic clotting pathways utilised by mammals. Care must therefore be taken to prevent accidentally tearing the vein wall, which can lead to a rapidly fatal haemorrhage. It should be noted that occasionally a bird will bleed from the nares after jugular venepuncture. This follows blood from the jugular vein entering the cervicocephalic air sac (an extension of infraorbital sinus) after inadvertent rupture or tearing during blood collection. This haemorrhage is rarely significant, although anecdotal reports suggest that birds with a coagulopathy can suffer a fatal haemorrhage. It is advisable to apply digital pressure to the venepuncture site for 30–60 seconds to minimise haematoma formation. Using a 25–29-gauge needle minimises the iatrogenic trauma to the vein, but the smaller the needle bore the more likely haemolysis is to occur during collection. Once the needle has entered the vein, avoid using excessive pressure to draw back; this will prevent both collapsing the vein and haemolysing the sample. It is sometimes advantageous to use a heparinised syringe for blood collection (**Fig. 4.3**).

A toenail clip, once advocated as a means of blood collection in birds, is the least desirable method of blood collection because of contamination with debris and uric acid from the bird's droppings, increased likelihood of the sample clotting, increased incidence of abnormal cell distributions and the pain it causes the patient.

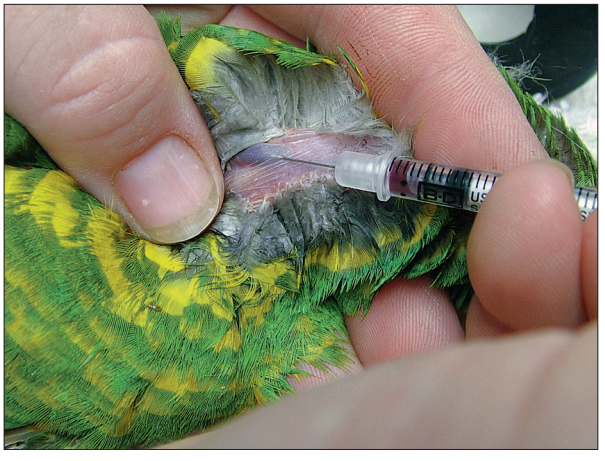


Figure 4.3 **With the right jugular vein located, blood samples can be collected as demonstrated.**

Many clients (and veterinarians) are surprised at how much blood can be safely collected from a bird. The blood volume in any given bird is approximately 10% of its bodyweight. Given that 10% of this blood volume can usually be safely collected for diagnostic testing or other purposes, an amount of blood equal to 1% of a bird's bodyweight can be safely collected (i.e. 1 ml of blood can be collected for each 100 g of bodyweight). With the modern laboratory equipment available in both external and in-house laboratories, this is usually a sufficient sample to run both haematological and biochemical assays.

Biochemistry testing is usually best performed on blood that has been collected into lithium heparin. If testing is likely to be delayed for anything more than a few hours, it should be centrifuged and the plasma decanted and submitted. The use of plain tubes that allow the blood to clot and serum to come off it is not advisable. Although serum collected in this manner has no anticoagulant in it, it has been in contact with haemolysing red blood cells for a significant time, which causes more artefactual changes. (Serum is lower in total protein, albumin and glucose; it is higher in uric acid, potassium, calcium, magnesium and phosphorus.)

Haematology, on the other hand, is usually best performed on blood that has been stored in sodium EDTA. (Note that some labs prefer lithium heparin for haematology; the clinician should consult with their local laboratory for preferences.) Fresh blood smears should always be made before the blood is placed into any anticoagulant. There are several techniques used for preparing blood smears:

- The traditional glass slide angled 'push' method, which often results in significant cell lysis.
- The angled slide 'drag' method: the angled slide is used to drag, rather than push, the blood film along the preparation slide. Cell lysis is much less common than with the previous technique.
- The coverslip method: place a 2 mm diameter drop of blood between two coverslips, allow the drop to spread close to the edges, then slide the coverslips apart horizontally. The dried

Downloaded From Vet-eBooks.com

coverslips can then be stained and mounted face down on a glass slide using mounting medium, immersion oil or a drop of adhesive.
- The coverslip on slide method: a drop of blood is placed on the glass slide and the coverslip is then placed on the drop. Once the blood has spread to the edges of the coverslip, it is then drawn horizontally along the slide. If not done gently, significant cell lysis can occur.

Microbiology

Microbiology is an essential tool in avian medicine. It can begin with a Gram stain to determine if bacteria or fungi are present in an area or organ of interest, and in what proportions. The sample is collected, rolled on to a slide, air dried and then heat-fixed with a gentle flame. Excessively thick smears should be avoided. The staining procedure for Gram stain is as follows:

- Apply crystal violet solution for 60 seconds, rinse gently with water.
- Apply Gram's iodine solution for 60 seconds, rinse gently with water.
- Decolorise with acetone or ethanol for 2–5 seconds, rinse gently with water.
- Stain with 0.1% basic fuchsin solution for 60 seconds, rinse gently and dry.

Gram-positive organisms and yeast stain deep violet and gram-negative organisms stain red. Gram-negative bacteria are predominantly rod- or coccobacilli-shaped and are usually much smaller than gram-positive rods in birds. Spirochaetes are gram-negative spiral bacteria.

Samples collected for culture and sensitivity are collected with sterile swabs, placed in a transport medium and sent to the laboratory (**Fig. 4.4**). Common sites cultured in live birds include the cloaca, choana, crop, eye, ear and skin. Less commonly sampled sites include the sinuses, the trachea, joints, air sacs and the oviduct.

Swabs used to culture the choana, crop or cloaca should be pre-moistened with either sterile saline or transport medium to minimise iatrogenic damage to mucosal surfaces (**Figs 4.5a** and **b**). Cloacal swabs are preferred to faecal swabs, as there is less

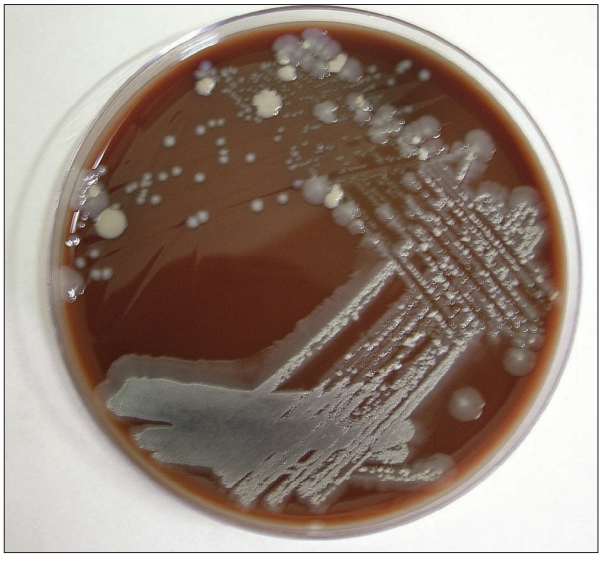


Figure 4.4 **Culture plates enable the identification in the laboratory of pathogenic organisms collected from swabs.**





Figure 4.5a,b **Cloacal swabs are preferred to faecal swabs. A moistened swab reduces iatrogenic trauma to the cloaca during the swabbing procedure shown here.**

Downloaded From Vet-eBooks.com

chance of environmental contamination. The owners should be advised that after the swab has been collected from this site the next few droppings may have blood on them. This is common and rarely of concern.

Choanal cultures are similarly collected with a pre-moistened swab. Some clinicians prefer to flush the nares with 1–2 ml of sterile saline immediately prior to swabbing the choana; this reduces oral contamination and gives a more representative sampling of the nasal cavity. A mouth speculum or gauze strips are used to hold the mouth open. The swab is gently inserted into the rostral choana.

It must be noted that the sinus and nasal cavity anatomy is such that a choanal culture may not represent the pathology occurring in the sinus. In cases of sinusitis it is therefore usually preferable to culture a sample from a sinus aspirate. This is done by inserting a 22–27-gauge needle, attached to a 3–5 ml syringe, midway between the commissure of the beak and the medial canthus of the eye, under the jugal bone. The sinus is more easily entered if the mouth is held open with a speculum during this procedure. Care must be taken to avoid iatrogenic trauma to the eye.

Culture of joints is indicated when bacterial or fungal infection is suspected based on cytology. The bird should be anaesthetised unless it is severely depressed. The skin over the joint is prepared aseptically and joint fluid aspirated using a small needle. The sample is expressed on to a swab, placed in transport media and sent to the laboratory.

Tracheal washes are useful for evaluating birds with suspected lower respiratory tract disease. The procedure can be performed on a conscious patient, but care must be taken with birds in respiratory distress. In many cases, however, anaesthesia reduces the chances of undue stress in an already compromised patient, iatrogenic trauma and contamination of the sample. A sterile catheter, long enough to reach the tracheal bifurcation, is introduced through the open glottis, taking care to avoid contamination through indirect contact with the mouth. Once the catheter is in position, sterile 0.9% saline (0.5–1.0 ml/kg) is quickly infused and immediately re-aspirated. (The lower dose rate should be used in larger birds and those with severe respiratory compromise.) Approximately 25–50% of the volume infused should be recovered and can be processed for both culture and cytology.

Cytology

Cytology is a useful tool in avian medicine. Antemortem samples can be collected from the choana, pharynx, sinuses, joints, skin, feather follicles, trachea, lungs, the crop, the cloaca, the coelom and the bone marrow. Collection techniques for the most part are similar to those described above.

Coeliocentesis is indicated in the presence of ascites or other abnormal coelomic fluid. (Often these birds present with dyspnoea, so care should be taken when handling.) The ventral midline is aseptically prepared and a 21–25-gauge catheter or needle (attached to a syringe) is inserted on the midline immediately caudal to the sternum, directed to the right side of the coelom to avoid the ventriculus. If using a catheter the stylet is removed immediately the catheter enters the coelom, and a syringe is attached. Gentle negative pressure is applied until fluid enters the syringe.

Bone marrow cytology is useful for the evaluation of haematopoiesis. Under anaesthesia the proximal cranial tibiotarsus is aseptically prepared. A small incision is made over the cnemial crest; a spinal or bone marrow needle is inserted into the shaft of the tibiotarsus with a gentle, twisting pressure, the stylet is removed and the sample is aspirated with a syringe and expelled on to a slide. A smear is then made in a manner similar to making a blood smear. For the best evaluation of the bone marrow, a peripheral blood sample collected the same day should also be submitted to the laboratory.

Fluid samples collected for cytology are often dilute, with a low cellularity; they must therefore be concentrated before being examined. It is preferable to use a gravity-based cell concentration method that avoids cell distortion. Centrifugation can be used, but often causes variable degrees of cellular distortion.

Parasitology

External parasitism can be diagnosed by direct examination of the patient and its feathers. The characteristic 'honeycomb' appearance of Cnemidocoptic mite

Downloaded From Vet-eBooks.com

infection is unmistakable, but a gentle skin scraping can be used to confirm the diagnosis. Other mites such as *Dermanyssus* spp. can be difficult to diagnose, but can usually be found after a careful examination. Close examination of the feathers may reveal lice eggs (nits) and the lice themselves. Examination of the feather shaft under magnification may be needed to diagnose quill mite (*Syringophilus* spp. and others).

Internal parasitism can be assessed by a combination of crop wash aspirate and faecal examination. Motile protozoa such as *Trichomonas* spp., *Spironucleus* spp., *Cochlosoma* spp. and *Giardia* spp. are best detected by immediate examination of a fresh crop wash aspirate or faecal material suspended in 0.9% saline solution. Protozoan oocysts and nematode eggs are sometimes detected by this method, but may require concentration methods such as faecal flotation using hypertonic solutions (zinc sulphate, sodium nitrate or sugar solutions) or faecal centrifugation.

It has been suggested by some researchers that faecal centrifugation consistently detects more parasitic ova than standard faecal flotation techniques. The technique can be performed in most veterinary clinics:

- Mix 2–5 g faeces with 10 ml faecal flotation solution and shake well until dispersed.
- Strain this fluid into a 15 ml centrifuge tube until a slight positive meniscus forms at the top.
- Place a coverslip on the tube and centrifuge it in a swinging head centrifuge at not less than 1200 rpm for five minutes.
- Let it stand for ten minutes before examination under the microscope.

Note that if a fixed head centrifuge is used, the tube should not be completely filled with the faecal solution, nor should a coverslip be applied. After centrifugation the tube is placed in a vertical rack and filled with fresh flotation solution until a meniscus forms. A coverslip is applied and the tube left to stand for ten minutes.

Cestode infections can be difficult to detect by any of the above means, as proglottid sections are often too heavy to float and may be missed on a fresh faecal smear.

Diagnostic imaging

Radiography is a valuable diagnostic tool in avian medicine; unfortunately much of its value is lost due to incorrect technique and equipment, and difficulties in interpretation of the images. Ultrasonography is less commonly used in avian medicine, mainly because of the air-filled coelomic cavity. It does, however, have a definite place in avian diagnostic imaging. Fluoroscopy, computed tomography (CT), positron emission tomography (PET) and magnetic resonance imaging (MRI) also play their roles in avian diagnostic imaging and are becoming more frequently utilised. Diagnostic imaging is discussed in more detail in Chapter 5, Diagnostic Imaging.

Endoscopy

Endoscopy was first used in avian medicine in the 1970s and 1980s to determine the sex of parrots. It was quickly realised that the unique avian anatomy, with its air-filled body cavities, made birds ideal candidates for endoscopy. Endoscopy is discussed in detail in Chapter 6, Endoscopy.

Cardiology

Radiography, ultrasound, Doppler and ECGs play an important role in the diagnosis of heart disease. These techniques are described in detail in Chapter 5, Diagnostic Imaging pp. 101 and 105 and Chapter 20, Diseases of the Cardiovascular System p. 289.

TREATMENT TECHNIQUES

Once a patient's condition has been diagnosed, or the clinician is waiting for test results for a diagnosis, treatment can be given both to support the patient and to treat the causative problem. Supportive care, sometimes overlooked, is a critical part of the treatment for a patient that is often more ill than many other species that are presented to veterinarians. Sick birds, on top of their original problem, are often hypothermic, dehydrated and hypoglycaemic. Obviously, sending such a patient home with some antibiotic powder to place in its drinking water is unlikely to help it much.

Because so many of the patients presented to veterinarians are in a critical condition, treatment (and diagnostic testing) has to be done carefully, in

Downloaded From Vet-eBooks.com

limited stages, to avoid 'overwhelming' the patient. Following a quick assessment to identify immediate life-threatening problems (e.g. uncontrolled haemorrhage or severe dyspnoea), it is often appropriate to commence supportive care before attempting diagnostic testing.

Heating

Birds are particularly sensitive to hypothermia because of their small size and higher surface area to volume ratio. It is for this reason that sick birds sit still (to conserve energy) and fluff their feathers (to retain body heat). When presented with such a patient, it is prudent to actively warm the bird before attempting any further treatment or diagnosis. As a general rule, ill birds should be maintained at a temperature of approximately 25–30°C, with a relative humidity of about 70%. Heat can be provided using an incubator, brooder, heat lamp (**Fig. 4.6**), circulating warm water blanket, hot water bottles or other device. The patient must be monitored carefully to prevent overheating or burns. Once the bird has regained its body temperature, as evidenced by increased activity, further treatment and diagnostic efforts can be attempted.

Fluid therapy

Most sick birds can be assumed to be at least 5% dehydrated, if not more. Signs of marked dehydration in birds include decreased skin turgor, sunken or closed eyes, dry oral mucous membranes and thick mucus in the pharynx. The clinician should aim to correct the calculated fluid deficit over 2–3 days, while at the same time providing maintenance fluids (50 ml/kg/day). A simplified calculation is to give 10% of the bird's bodyweight daily for three days, and then reduce this volume to 5%. The calculated volume should be divided into two or three doses, given over a 24-hour period.



Figure 4.6 **A heat lamp is advised to keep a hospital patient warm following surgery.**

In mildly dehydrated patients with a functional gastrointestinal system and the ability to withstand the necessary handling, replacement fluids can be given orally. Tap water, oral rehydration formulae or lactated Ringer's solution can be delivered by a crop needle or plastic tubing directly into the crop. Hypertonic liquids should be avoided, as these may exacerbate dehydration. A crop needle (a stainless steel needle with a stainless steel ball on the end) or plastic tubing is passed over the tongue and gently guided down the cervical oesophagus and into the crop (**Figs 4.7a** and **b**). Care must be taken not to inadvertently place the needle or tube into the trachea or to force it through the oesophageal/crop wall. It is important that the crop needle be placed into the crop rather than just at the back of the pharynx. Careful palpation of the crop can confirm the placement of the implement before giving the fluids. Failure to do so may result in the fluids flooding the pharynx and oral cavity, possibly leading to aspiration. Care must be taken not to press on the crop while restraining the bird, to overfill the crop, or to fill it too quickly. If fluid appears in the pharynx or comes from the mouth while gavaging, the tube or needle should be immediately withdrawn and the bird placed back in its cage. Continued handling of the bird is more likely to result in it panicking and aspirating the fluid. As a general rule, the amount given by crop gavage should start initially at 5% of the bird's bodyweight. It can then be built up to 8–10%. It will typically take about 2–4 hours for the crop to empty completely (thicker mixes, such as hand-rearing formula, will take longer).

Severely dehydrated patients, or those that cannot handle either oral fluids or physical restraint, can be rehydrated parenterally through subcutaneous, intravenous or intra-osseous routes.

Subcutaneous fluids are not recommended for hypothermic patients or those in shock due to

Downloaded From Vet-eBooks.com

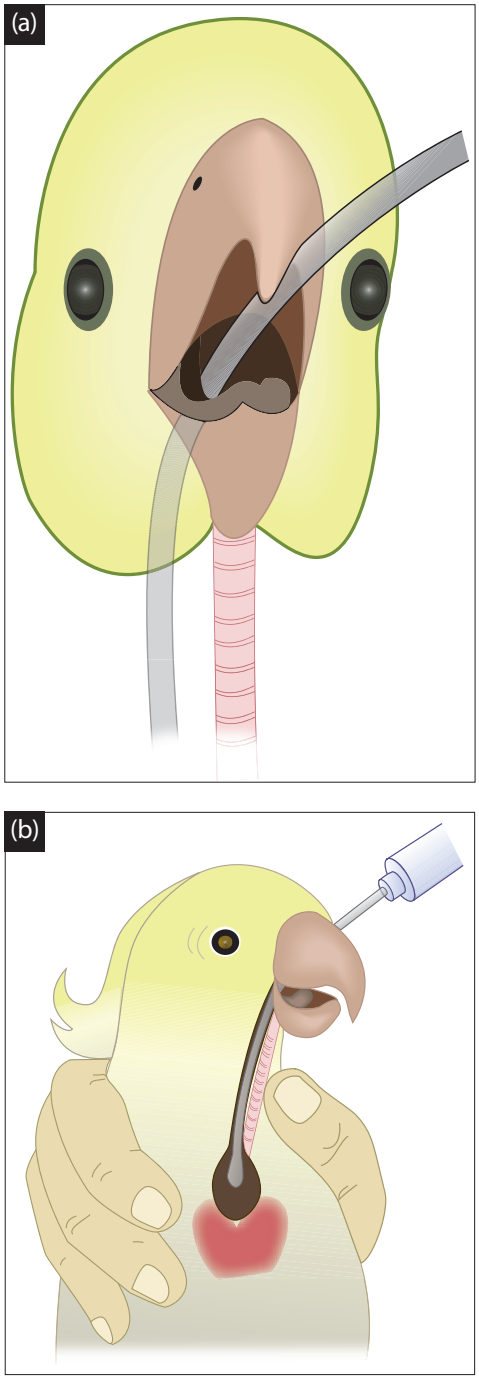


Figure 4.7 Procedure for the insertion and correct placement of a crop tube for fluid therapy. With the patient facing you and its head restrained, the crop needle is introduced into the bird's mouth on the left side, passed over the top of the tongue and directed to the right side of the oropharynx (a). The tube is gently passed down the oesophagus, into the crop (b).

concerns over absorption (peripheral vasoconstriction may lead to poor absorption). They are, however, very useful for most ill patients. They may be given in the inguinal region or the inter-scapular region; the inguinal region allows for greater volumes to be given with less patient discomfort (**Figs 4.8, 4.9a** and **b**). Volumes up to 10 ml/kg can be given two to three times daily in each site. If oedema is noted prior to subsequent administration, the fluid is not being absorbed (due to poor circulation, excessive volume administered or hypoproteinaemia) and additional fluid should not be administered by this route.

Fluids may be administered by constant infusion or slow bolus through an aseptically placed intravenous or intra-osseous catheter. The jugular, basilic and medial metatarsal veins are suitable for intravenous fluid administration. Depending on the size of the patient, 22–26 g intravenous catheters can be placed with little difficulty in birds weighing more than 200 g. Secured in place with adhesive tape, tissue glue or suturing, they can be maintained for several days.

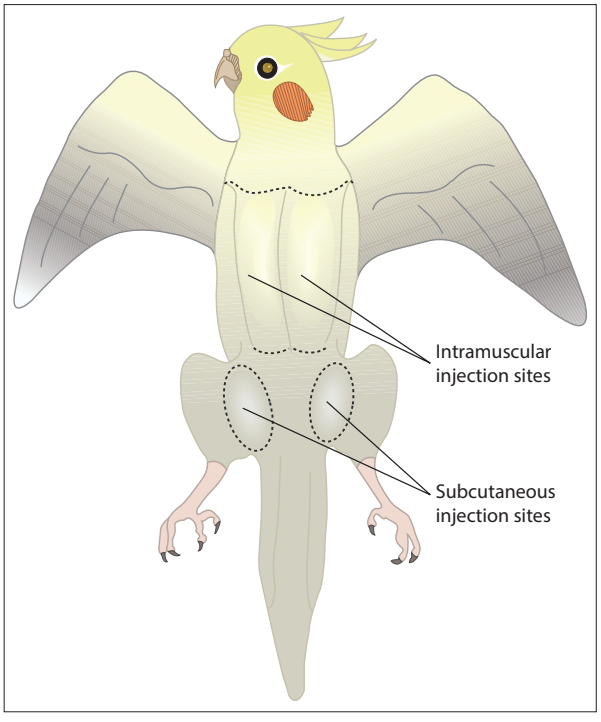


Figure 4.8 Diagram illustrating suitable sites for the administration of intramuscular and subcutaneous injections.

Downloaded From Vet-eBooks.com

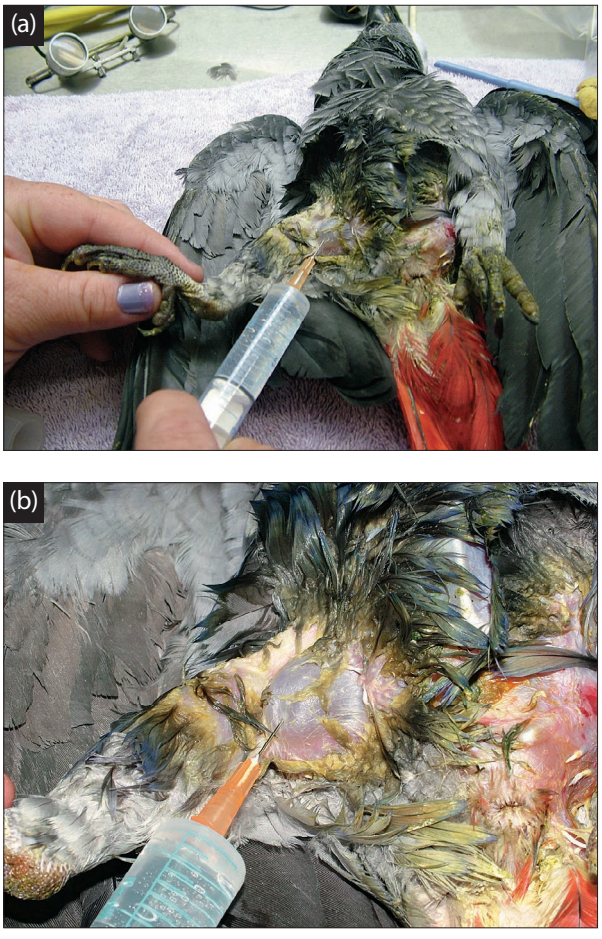


Figure 4.9 **An African grey parrot administered subcutaneous fluids in the inguinal area (a). Note that once the fluids are introduced the skin has bulged slightly (b).**

For smaller patients, intra-osseous fluids are considered equivalent to intravenous administration, with 50% of the fluid given by this route appearing in the circulation within 30 seconds. Intra-osseous catheters are most commonly placed in the proximal tibiotarsus and the distal ulna. A spinal needle is used to prevent the lumen plugging with bone and tissue, rendering the catheter useless. Care must be taken to insert the catheter correctly the first time, as subsequent attempts usually result in either a large hole or multiple holes in the bone at the entry site. Leakage will occur through each hole, resulting in discomfort from the deposition of the fluid or drugs into the soft tissues around the bone.

The needle is placed into the tibiotarsus in a manner similar to the normograde insertion of an intramedullary pin. The tibiotarsus is grasped, a small stab incision is made in the skin over the proximal cranial tibiotarsus, lateral or medial to the cnemial crest, and the needle is inserted into the marrow cavity with gentle pressure and a twisting motion of the needle. Following placement of the catheter, the stylet is removed and the catheter's position is evaluated to ensure it is within the medullary canal. In larger birds, aspiration will result in blood or bone marrow flash back. (If bone marrow is needed, it can be collected in this manner.) Alternatively, a small amount of saline can be injected and then re-aspirated. If the needle is in the medullary canal, this will usually yield blood-tinged fluid. A larger volume of fluid can then be injected; if the needle is outside the bone, the soft tissues will swell. Once placement is confirmed, the catheter should be secured.

For placement in the ulna, the dorsal crest of the distal ulna is palpated with the carpus flexed. The needle is inserted between the distal ulna and the ulnar carpal bone and advanced into the medullary canal (**Fig. 4.10**). The ulna is held with one hand during insertion to ensure that the needle

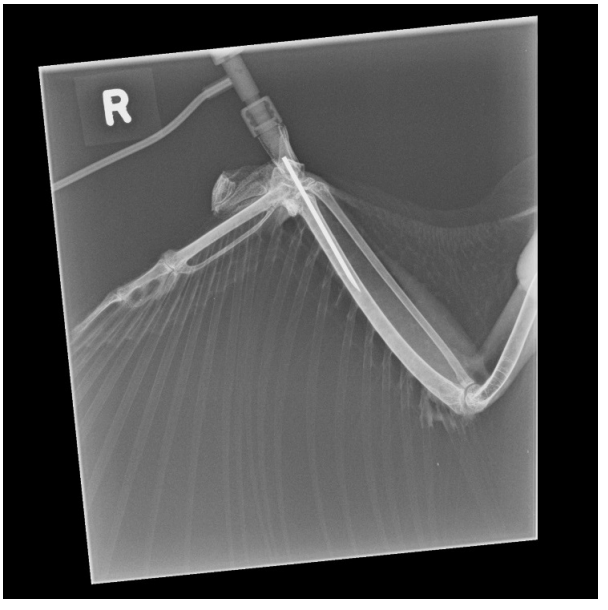


Figure 4.10 **Radiograph showing correct needle position following an intra-osseous catheterization of the ulna.**

Downloaded From Vet-eBooks.com

is inserted at the proper angle. Again, aspiration, flushing and re-aspiration can be used to confirm correct placement. As the ulna in some birds is pneumatic, if air is aspirated the catheter should be removed and placed in the tibiotarsus. Infusion of a larger volume of fluids into a correctly placed ulnar catheter should result in a visible 'blanching' of the basilic vein as the fluid enters the circulation. Once correct positioning is assured, the catheter should be secured with a bandage.

As the bone is not distensible, and the rate that the fluid enters general circulation is a limiting factor, care must be taken to give intra-osseous injections slowly in order to allow the fluid to enter the circulation without being forced. If the injection is forced, it will exit the insertion hole in the bone, causing discomfort.

Infusion pumps and syringe drivers are most appropriate for constant rate infusion. While birds may be given up to 100 ml/kg/hour intravenously without adverse effects, rates of between 3 and 10 ml/kg/hour are usually more appropriate. Fluid boluses of 20–30 ml/kg may safely be administered over a 2–3-minute period. If using this technique, the use of a butterfly catheter on the syringe and a luer injection port in the catheter can minimise handling of the bird.

Guidelines on the selection of the type of fluid to be given are similar to those for mammalian medicine. Crystalloids, colloids and whole blood are all used successfully in avian patients (See *Table 4.1*).

Fluid therapy is an important tool in the resuscitation of a critical patient. A guide to its use is in *Table 4.2*.

Nutritional support

Sick birds often do not eat; birds that do not eat, die. It is therefore vital that anorexic or emaciated birds with a functional gastrointestinal tract should receive nutritional support. The procedure is identical to crop gavaging fluids; if it is anticipated that the patient will require long-term feeding or if crop

Table 4.1 **Guidelines for fluid therapy selection**

FLUID TYPE	INDICATIONS	EXAMPLES
Replacement crystalloids	Rapid replacement of intravascular volume and electrolytes as seen with shock, haemorrhage or severe volume depletion (secondary to the losses associated with vomiting, diarrhoea, third body spacing, or excessive diuresis).	Normal saline (0.9% NaCl) Lactate Ringer's Solution (lactate or acetate) Normosol R Plasmalyte A
Maintenance crystalloids	Meet the electrolyte requirements of patients with normal daily electrolyte losses that are unable to maintain adequate fluid or electrolyte intake. They are rarely infused at rates greater that that necessary to meet the patient's maintenance needs.	0.45% saline 0.45% saline + 2.5% dextrose Lactated Ringer's + dextrose Plasmalyte 56
Hypertonic crystalloids	Rapidly increase intravascular volume when severe hypovolaemia is present that may lead to impending death. It is contraindicated in severely dehydrated patients, or those with uncontrolled haemorrhage.	7% saline
Dextrose in water	Used as a vehicle for infusion of other medications and to provide free water in severely hypernatraemic states. Infusion of large volumes of this fluid can lead to dilution of serum electrolytes and/or the development of oedema.	5% Dextrose
Colloids	Expand intravascular volume by holding and potentially drawing water into the vasculature, therefore indicated for the treatment of hypovolaemia, sepsis, inflammatory conditions such as pancreatitis, and to improve colloid oncotic pressure in patients with hypoproteinaemia secondary to renal or gastro-enteric disease, vasculitis, or burns.	Hetastarch Dextrans Modified gelatin solutions
Whole blood	Severe blood loss or anaemia.	See Chapter 8, Supportive Therapy.

Downloaded From Vet-eBooks.com

Table 4.2 **Guidelines for fluid therapy during the treatment of a critical patient**

STEP	ACTION
Assess degree of dehydration or hypovolaemia	Physical assessment, blood pressure if possible, PCV (if <20%, consider blood transfusion).
Administer crystalloids	1–2 boluses of 10 ml/kg IV/IO.
Administer colloids	1–2 slow boluses (over 10 minutes) of 3–5 ml/kg.
If patient is not improving	Give third bolus of crystalloids and colloids.
If patient still not improving	Give hypertonic saline (7.5%) as a slow bolus (10 minutes) at 5 ml/kg.
If no improvement	Maintain IV fluids while waiting for other therapies to take effect.

gavage cannot be done for any reason, oesophageal feeding tubes can be used. Under anaesthesia, curved haemostats are placed in the oropharynx and directed into the oesophagus so the tip is at a point half way along the cranial oesophagus on either the right or left side of the neck. The tip is pushed up against the skin (taking care to avoid entrapping the jugular vein) and the jaws slightly opened. A 3-5 mm scalpel incision is made between the open jaws of the haemostat; they are then closed and pushed through the incision. The tip of the oesophagostomy tube is then grasped with the forceps and drawn rostrally out of the mouth (**Fig. 4.11**), reversed, and guided into the crop or, if desired, into the distal oesophagus. The exposed end of the tube is then secured to the skin with a Chinese finger trap suture, capped, and the neck lightly bandaged.



Figure 4.11 Superb parrot receiving nutritional support by introducing an oesophagostomy tube. Note that the catheter coming out of the mouth will be reversed and subsequently passed into the crop.

Foods that will pass easily through a syringe and feeding tube and are high in energy, easily digestible and have minimal residue are ideal. For these reasons, parrot hand-rearing formulae are often used for short-term maintenance feeds. If not readily available, pureed vegetable and fruit baby food can be used until a more appropriate replacement is obtained. If the tube becomes blocked with hand-rearing formula it can often be unblocked by flushing the tube with a pancreatic enzyme extract dissolved in water.

Respiratory support

Birds, with their high metabolic rates, have a higher oxygen demand than dogs and cats. Conversely, their gas exchange is more efficient; indeed, oxygen levels of 70–100% for 3–8 days have been shown to be toxic in birds. Birds with respiratory distress appear clinically to benefit from a 40–50% oxygen environment. Oxygen may be delivered by mask in the short term or in an oxygen cage.

In cases of tracheal obstruction (e.g. inhaled foreign body, tracheal stricture, *Aspergillus* granuloma, or diphtheritic plaques) it may be necessary to provide an alternative airway by way of an air sac catheter. This is a 2 mm or larger plastic tube (e.g. a non-cuffed endotracheal tube) placed into the caudal thoracic air sac. With the patient in right lateral recumbency, the left leg is drawn cranially, exposing the left flank. The following landmarks are identified: the caudal rib, the flexor crura medialis muscle and the pubic bone. A 4–5 mm vertical incision is made in the skin where the flexor crura medialis muscle crosses the caudal rib.

Downloaded From Vet-eBooks.com

Curved haemostat forceps are then used to reflect the flexor muscle dorsally and then dissect bluntly through the coelomic wall. If done correctly, the operator will feel a 'pop' as the haemostats enter the caudal thoracic air sac. The endotracheal tube or other catheter is then advanced through this incision into the caudal thoracic air sac, taking care not to advance it as far as the caudal border of the lung. Patency is determined by observing expired air moving a feather placed over the free end of the catheter. Once satisfied with the placement, the clinician can then use a Chinese finger-trap suture to anchor the catheter to the skin. The free end of the catheter should be long enough so that powder down and feather dust are not freely inhaled; the author prefers to leave the catheter as long as the bird's tail, securing it to the tail feathers as shown in **Fig. 4.12**.

Administering medications

Once a patient has been stabilised and diagnostic samples collected, medication can then be administered based on either a confirmed or suspected diagnosis. The route of administration is determined by a number of factors: the ease with which the patient can be handled; the patient's clinical condition; the pharmacodynamics and pharmacokinetics of the drug itself; and the clinician's experience and preferences. Commonly used administration routes include injection (intramuscular, intravenous or subcutaneous), oral dosing, topical application and nebulization. Some of these routes are discussed above and will not be repeated here.



Figure 4.12 **An air sac catheter inserted and correctly positioned in a Sun conure during surgery. Note that the free end of the catheter has been left as long as the bird's tail and securely attached to it.**

The pectoral muscles are most commonly used for intramuscular injections because of their large size (**Fig. 4.8**). Other muscles such as the quadriceps, caudal thigh muscles and gluteal muscles can be used in larger birds. Clinicians need to be aware that many drugs are, at best, tissue irritant when given intra-muscularly; caution needs to be taken to avoid giving repeated injections into one site. Care should also be taken with the volume to be injected: a 0.1 ml injection for a 100 g bird is equivalent to a 10 ml injection for a 10 kg dog. If necessary, divide the sternum into quadrants and divide the dose between the quadrants.

Oral medications are often surprisingly easy to administer. In large birds (e.g. poultry, waterfowl), tablets can be placed directly into the oropharynx; for smaller birds, tablets can be crushed and suspended in either water or methylcellulose for administration with a syringe. When using a syringe, it should be placed over the tongue and the medication delivered in small quantities into the oropharynx. Care must be taken with screaming birds to ensure that the medication is not aspirated. In these birds, and with larger volumes of medication, crop gavage may be more appropriate.

While water-based or other drugs can sometimes be used topically in birds, topical application of oil-based medications is contraindicated. Their grooming behaviour usually sees the medication dispersed through the plumage, destroying the insulation and waterproofing capacity of the feathers. The thinness of the avian skin makes transdermal absorption of medication very easy. This can result in systemic absorption of toxic levels of a drug (e.g. use of topical corticosteroids invariably results in steroidal hepatopathy and other adverse effects).

Nebulization is often used in the treatment of respiratory disease, both to deliver medications directly to site where it is needed and to humidify the respiratory tract. The unique anatomy of the avian respiratory tract means that the nebuliser used should create droplets between 1 and 3 µm

Downloaded From Vet-eBooks.com



Figure 4.13 **An African grey parrot receiving medication via a nebuliser attached to a plastic storage box. Note the air sac catheter by the bird's tail.**

in size. Hypertonic saline or diluted acetylcysteine (2% solution in saline) can be used to reduce the viscosity of airway secretions. Antibiotic and antifungal medications can be added to the nebulising solution to deliver the drug directly into the respiratory tract (**Fig. 4.13**).

Immobilising limb fractures using external coaptation

External coaptation (bandaging) can be used for many limb fractures, either as a temporary splint before or after surgery, or as the sole means of immobilisation for repair of a fracture. There are benefits and disadvantages to using external coaptation. While bandaging can be quick and inexpensive, and provide almost immediate pain relief by immobilising the ends of the fracture, it can lead to joint ankylosis, tendon contracture and delayed or non-union of a fracture. Not all fractures can be immobilised by external coaptation, (e.g. femoral fractures and many proximal tibiotarsal fractures cannot be completely immobilised by splinting), nor is splinting always a good choice as the sole means of fracture repair (e.g. the distortion produced by the powerful flight muscles makes external coaptation a poor choice for humeral fractures if a complete return to flight is desired).

The figure-of-eight bandage is used for immobilising fractures of the antebrachium and carpus. It should not be used for humeral or pectoral girdle fractures. Care must be taken not to make the bandage too tight as it may compromise circulation, see **Fig. 4.14**.

The figure-Y bandage is used to immobilise humeral and pectoral girdle fractures. It will also stabilise antebrachial and carpal fractures. If necessary, additional support can be provided by taping the tip of the primary feathers to the tail. Care must be taken to ensure that, while the bandage is tight enough to prevent slipping or allowing the bird to get a foot between the skin and the bandage, it is not so tight that it compromises the bird's respiration (**Fig. 4.15**).

Modified Robert Jones bandages and 'Altman' splints can be used to stabilise tibiotarsal, tarsometatarsal and metatarsal fractures. The limb should be flexed into a normal perching position and the feathers plucked from the area that will be covered by the splint. Where possible, adhesive tape 'stirrups' should be used to both hold the joint in flexion and to prevent the bandage slipping (**Figs 4.16–4.19**).

As a general rule, splints and bandages should be removed every two weeks, the stability of the fracture assessed, and the joints of the affected limb flexed and extended to prevent ankylosis.

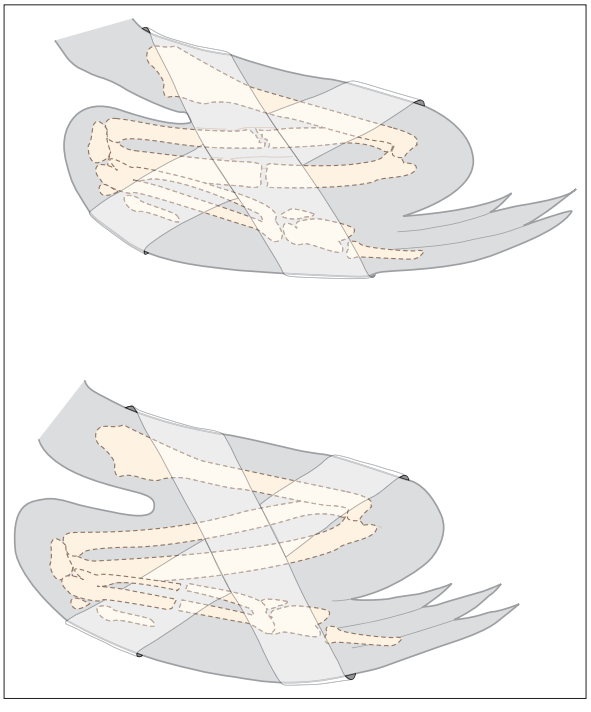


Figure 4.14 **Illustration of a figure-of-eight bandage application for metacarpal/radius–ulna fractures.**

Downloaded From Vet-eBooks.com

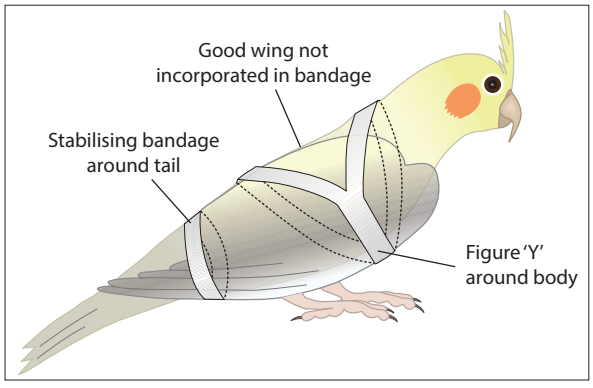


Figure 4.15 Demonstration of the correct positioning for a figure-Y or full wing bandage.

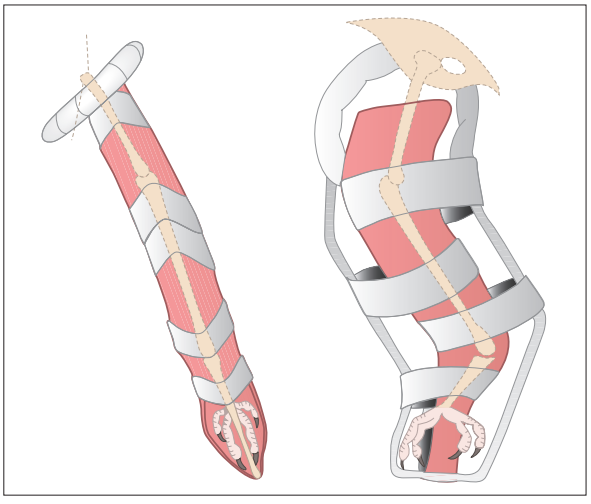


Figure 4.16 Diagrams showing the correct application of a Thomas splint.

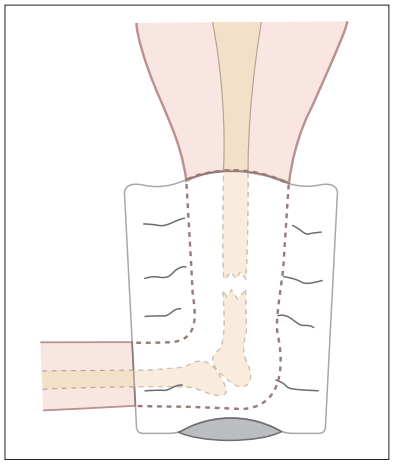


Figure 4.17 Illustration showing correct tape splint positioning.



Figure 4.18 Illustration of positioning and technique for a Robert Jones bandage to stabilise tibiotarsal, tarsometatarsal and metatarsal fractures.

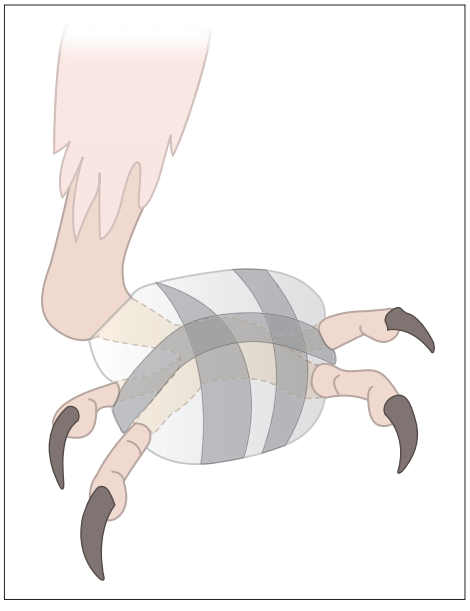


Figure 4.19 Illustration showing correct bandage application for bird feet.

Downloaded From Vet-eBooks.com

FURTHER READING

Campbell TW, Ellis CK (2007) *Avian and Exotic Animal Hematology and Cytology*, 3rd edn. Blackwell Publishing, Ames.

Divers SJ (2003) Modern endoscopy equipment and advanced endoscopy techniques in birds and reptiles. *Exotic DVM* **5(3):**61–63.

Divers SJ, Hernandez-Divers SM (2004) Avian diagnostic endoscopy. *Compendium on Continuing Education for the Practicing Veterinarian* **26(11):**839–852.

Fudge AM (2000) *Laboratory Medicine: Avian and Exotic Pets*. WB Saunders, Philadelphia.

Jaensch SM, Cullen L, Raidal SR (2001) The pathology of normobaric oxygen toxicity in budgerigars (*Melopsittacus undulatus*). *Avian Pathology* **30(2):**135–142.

Krautwald-Junghanns ME, *et al*. (2004) Research on the anatomy and pathology of the psittacine heart. *Journal of Avian Medicine and Surgery* **18(1):**2–11.

Krautwald-Junghanns ME, Pees M (2005) Ultrasonographic diagnosis of avian urogenital disorders. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 39–44.

Krautwald-Junghanns ME, Pees M (2006) Ultrasonography of the avian liver and gall bladder. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 53–58.

Nemetz L. (2006) Application of high definition digital radiology for the avian patient. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 263–268.

Nemetz L. (2006) Principles of high definition digital radiology for the avian patient. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 39–46.

Smith BJ, Smith SA (1997) Radiology. In: *Avian Medicine and Surgery*. RB Altman, *et al*. (eds). WB Saunders, Philadelphia, pp. 170–199.

Straub J, Pees M, Krautwald-Junghanns ME (2002) Measurement of the cardiac silhouette in psittacines. *Journal of the American Veterinary Medical Association* **221(1):**76–79.

Downloaded From Vet-eBooks.com

CHAPTER 5

DIAGNOSTIC IMAGING

RADIOLOGY

Radiology is a valuable diagnostic tool in avian medicine, but is surprisingly under-utilised. This may reflect the clinician's 'comfort level' with avian radiology; a lack of training and experience; the apparent difficulty in obtaining good quality images, or a reluctance to use radiography in such a small patient. The advent of digital radiography may go a long way towards overcoming these problems but the basic principle – do it right the first time – is as true in birds as it is in other species.

Equipment

Good avian radiography requires short exposure times (to minimise the effect of 'blurring' due to the patient's relatively rapid respiratory rate) and a combination of high detail screens and films. These requirements mean that a relatively powerful X-ray machine is needed, with the ability to produce a high mA with a short exposure time. Most X-ray machines used in veterinary practices will fulfill these requirements. Non-screen film can be used but requires a higher exposure time. The screens that are used for radiographs of the extremities in man, cats and dogs are also excellent for birds. Mammography cassettes, screens and film are also very useful and give excellent detail but processing the film is more critical than with other films. (Mammography film/screen combination gives higher line pair per mm resolution, therefore excellent detail.)

Digital radiography (DR) is becoming more obtainable in many private practices (**Fig. 5.1**). With the ability to obtain and manipulate high definition images as well as storing them electronically, high definition digital radiography offers major advancements in avian radiology. The author finds that, for small patients (<50 g), a combination of digital plates and a dental X-ray machine achieves excellent images that were previously difficult to achieve using film, or even using DR or computed radiography (CR) equipment with a conventional X-ray tube or machine. For birds that weigh over 100 grams, DR or CR equipment with conventional X-ray tube or machine produces high quality images. Having a Picture Archiving and Communication System (PACS) which allows the clinician to more closely examine images, measure parts of them, annotate them and then communicate



Figure 5.1 **Example of a commercially available digital radiology unit enabling the easy manipulation and storage of high definition images.**

Downloaded From Vet-eBooks.com

these images with colleagues and clients via email or CD has dramatically improved the quality of avian diagnostic imaging.

Contrast

Contrast studies can add greatly to the diagnostic value of radiology. Contrast media such as barium, iohexol and diatrizoate have all been used in avian radiology.

Gastrointestinal

Gastrointestinal contrast studies (**Fig. 5.2**) utilising iohexol or diatrizoate are often comparable to those using the more routinely recommended barium sulphate. Barium and iohexol can be used undiluted, or diluted 1:1 with water. (Higher dilutions may result in poor contrast.) Diatrizoate has a high osmolality; this hypertonicity may cause dehydration in small birds. Care should therefore be taken when using diatrizoate, but it may be safer than barium if a gastrointestinal perforation is suspected.

The dose is based on the estimated size of crop volume (approximately 25–30 ml/kg for parrots). In addition to time-zero survey radiographs, additional orthogonal images are typically taken at 30 minutes, 1-, 2-, 4-, 8- and 24-hour intervals. The timing of these images will vary with species and age. In small species, images may be made at 15-, 30-, 60- and 90-minutes, with additional images taken on an 'as needed' basis. It is recommended to fast a parrot for 3–4 hours before a gastrointestinal contrast study, but transit time does not appear to be affected by anaesthesia. Transit time for barium (crop to cloaca) in a healthy bird is approximately three hours; using iohexol it may be as short as one hour.

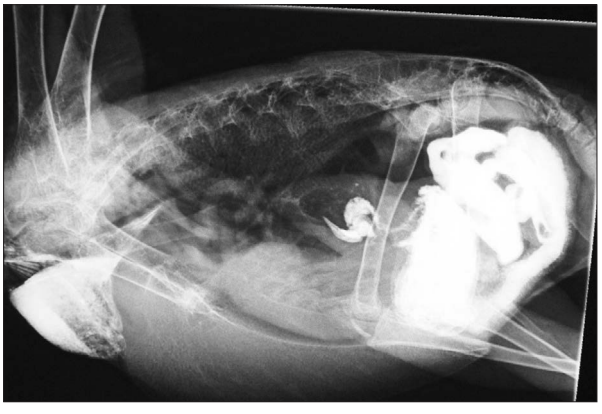


Figure 5.2 **Radiograph following administration of a contrast media, part of a gastrointestinal contrast study.**

Urography

The patient should be fasted for about two hours before the contrast medium is given. It may be necessary to aid emptying the gut by introducing liquid paraffin into the cloaca. For urography, the organic iodine compounds are warmed to body temperature and injected intravenously slowly under anaesthesia and usually into the basilic vein. The dose is 2 ml/kg body weight, as a 70–80% solution of organic iodine compound or containing 300–400 mg of iodine per ml (800 mg/kg) intravenously. The quality of the contrast obtained depends on the concentrating capacity of the kidneys, on the preparation used, and on the iodine concentration of the medium. The aorta, heart, and pulmonary arteries are demonstrated ten seconds after the injection, kidneys and ureters are shown 30–60 seconds after injections, and the cloaca and terminal gut are visualised 2–5 minutes after the injection. Urography must not be performed in dehydrated or severely renal compromised birds.

Sinography

To visualise the infraorbital sinuses and nasal cavity, iodinated compounds are instilled directly into the sinuses to demonstrate the flow of contrast medium into the nasal cavity. From 0.1 to 1.0 ml of 15–20% iodine agent is installed directly in the nares or in the sinus. After taking the radiographs, the contrast medium should be flushed out with sterile saline to minimise local reaction after radiography has been performed. (Side-effects have been described in some birds, including oedema and periorbital swelling.)

Restraint and positioning

Whether using conventional or digital radiography, positioning of the patient is absolutely vital for the accurate interpretation of an exposed radiograph. Correct positioning is best obtained under anaesthesia; only very tame (or very sick) patients may be positioned conscious in a plexiglass restraint device or similar restraints. Often, however, anaesthesia is less stressful and therefore safer than manual restraint.

Downloaded From Vet-eBooks.com

The time of anaesthesia is generally brief (less than ten minutes). Ideally, birds should be fasted for several hours before performing radiographs to prevent regurgitation and aspiration, whether or not anaesthesia is used. Small birds, such as cockatiels and budgies, should be fasted for 1–2 hours, while larger birds should be fasted for 2–6 hours. If fasting is not possible, or regurgitation is a concern, the head should be elevated above the level of the crop during the procedure. If possible, the patient should be intubated.

The lateral view (**Fig. 5.3**) is taken with the patient in lateral recumbency, with the wings extended dorsally and the legs pulled caudally. The limbs can be held in position with adhesive tape. When examining the resultant image (compare **Figs 5.4a** and **b**), the clinician should first assess that the acetabulae are superimposed, as should the coracoids. The ventral dorsal (VD) view is taken with the patient in dorsal recumbency with the wings extended laterally and the legs caudally alongside the tail, with the femurs ideally parallel to the spine and each other (**Fig. 5.5**). The keel should be superimposed over the vertebrae – a thin air space in the carina (seen as a thin black line) is used to check this (compare **Figs 5.6a** and **b**).

Normal anatomy

As with any species, it is important to become familiar with the normal gross and radiographic anatomy of birds. Reviewing all systems with a systematic approach in all studies will increase the diagnostic

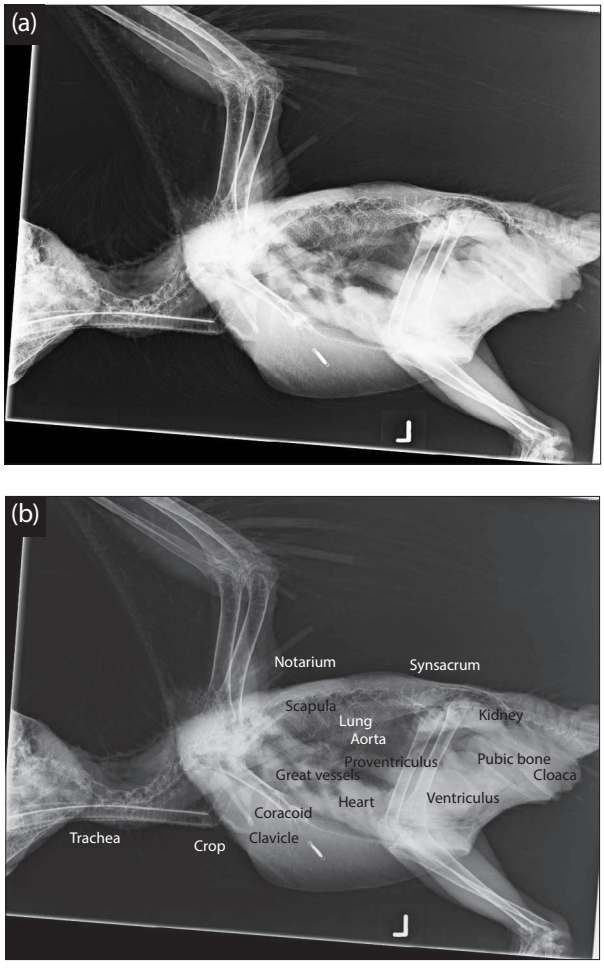


Figure 5.4 **Radiographs showing the normal lateral view of a bird (a) and with all parts labelled (b). Note that the acetabulae are level and the coracoids are superimposed.**



Figure 5.3 **Eclectus hen anaesthetised and correctly positioned for a lateral X-ray exposure.**

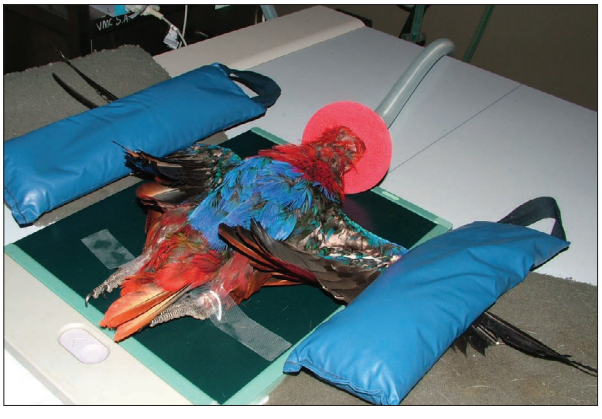


Figure 5.5 **Same bird as in Figure 5.3 correctly positioned for a ventrodorsal X-ray exposure.**

Downloaded From Vet-eBooks.com

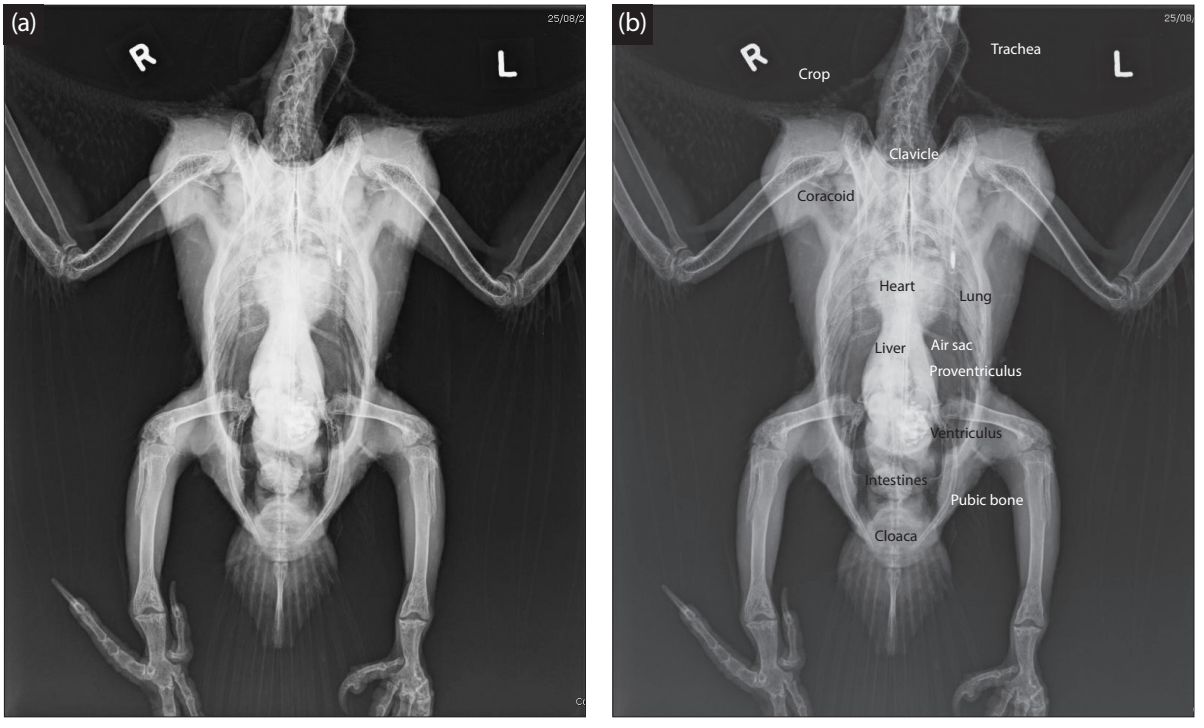


Figure 5.6 **Radiographs showing the normal ventrodorsal view of a macaw (a) and with all parts labelled (b). Note the carina of the sternum is superimposed on the spine.**

yield of each study, while improving the clinician's comfort and familiarity in interpreting avian radiographic images.

Musculoskeletal system

The anatomy of the musculoskeletal system is illustrated in Chapter 1, Clinical Anatomy and Physiology (see **Figs 1.9–1.14** on pages 5–9). Reviewing the radiograph should include the following: the conformation of the skeleton; the density and thickness of the bone cortices; the radio-opacity of the medullary cavities, including that of the pneumatic bones (humerus and femur); and the presence of internal struts in the humerus and other long bones.

Cardiovascular system

The size and shape of the heart is often best assessed in a VD view. The maximum width of the cardiac silhouette should be equal to 51–61% of the maximal width of the rib cage. On the lateral view the length of the heart should be 35–40% the length of the sternum. The shape of the heart on the VD view should be symmetrical, broader at the base than the apex, and there should be no clear distinction between the heart and the liver (the cardio-hepatic shadow). On a lateral view the aorta can usually be visualised leaving the base of the heart and coursing caudally, below and approximately parallel to the spine.

Respiratory system

The proximal trachea can be readily visualised. The distal trachea, within the rib cage, and the syrinx can be difficult to evaluate due to the overlying ribs, soft tissues and great vessels. Thorough interpretation of the lungs requires orthogonal views because a significant proportion of the lungs is covered by the cardiac silhouette on the VD view. On the lateral view, the right and left lung fields are superimposed rendering the localisation of unilateral lesions impossible. Careful consideration of both views may allow for the detection and localisation of pulmonary lesions. The normal radiographic appearance of the avian lung is somewhat honeycomb-like (due to the appearance of end-on parabronchi). The air sacs can be visualised around the pectoral joints

Downloaded From Vet-eBooks.com

(clavicular) and caudal to lungs, lateral to the liver (thoracic and abdominal). Healthy air sacs have little or no radio-opacity, with no lines visible between adjacent air sacs.

Digestive tract

The crop is usually empty, although small amounts of ingesta are sometimes present. Gas or fluid should not be present. The oesophagus passes over the dorsal aspect of the heart and proximal liver and becomes the proventriculus about one-quarter of the way along the dorsal hepatic surface. In health, a narrow air sac space can often be identified between the proximal portion of the dorsal hepatic silhouette and the oesophagus/proventriculus. The oesophagus and proventriculus typically course on an approximately 40–45 degree angle from the notarial vertebrae before entering the ventriculus (or gizzard). The ventriculus is seen as a spherical organ, often filled with slightly radio-opaque grit. On a VD view it lies on the left side of the spine, between the coxofemoral joints. On a lateral view it lies on the ventral coelomic floor between the stifle joints of the extended legs. On the dorsal side of the proventricular-ventricular junction lies the spleen. The intestinal tract lies caudal to the ventriculus, with individual loops usually indistinguishable. It terminates in the cloaca, usually visible as a small dome-shaped soft tissue density at the base of the tail.

Liver

The heart and liver are contrasted against the thoracic and abdominal air sacs. In general, the silhouette is of an hour-glass shape on the VD view with the hepatic waist being marginally wider than that of the heart. The lateral margins of the liver should not extend beyond a line joining the pectoral and coxofemoral joints on each side. The proventriculus lies on the left side and sometimes lateral to the liver; contrast media may be required to differentiate it from the liver. On the lateral view, the liver is caudal to the heart and fills approximately one-third of the ventral coelom, tapering caudally and ventrally at about 15–20° from the heart base. There is often a small space between the dorsal liver and the ventral proventriculus.

Urogenital system

The kidneys are best assessed on the lateral view, despite superimposition. They are difficult to assess on the VD view because of overlying bone and soft tissues (although the cranial margin of the cranial division of the kidneys can occasionally be visualised). The kidneys are surrounded by the abdominal air sac, including dorsal diverticula of the abdominal airs sacs which lie between the kidneys and the synsacrum. On the lateral view, with both acetabulae superimposed, the kidneys should extend below a line drawn through the ventral acetabular rim, parallel to the spine. The ovary or testes may be visible immediately cranial and slight ventral to the cranial division of the kidney. The oviduct is generally not distinguishable from the intestinal tract, unless it contains an egg.

Radiographic abnormalities

Musculoskeletal system

Abnormalities seen include:

- Congenital abnormalities such as bifid sternum are occasionally seen. This develops as a failure of the two halves of the sternum to close during embryonic development.
- Developmental abnormalities such as kyphosis (**Fig. 5.7**), scoliosis, bowing of the long bones and tibiotarsal rotation are often seen in juvenile birds on a poor diet, or whose parents were on a poor diet. Metabolic bone disease (usually nutritional secondary hyperparathyroidism)



Figure 5.7 **Radiograph of a cockatoo indicates the developmental abnormality kyphosis.**

Downloaded From Vet-eBooks.com

is characterised by these changes, as well as decreased cortical thickness and the presence of large numbers of internal struts in the metaphyseal regions of the long bones.

- Fractures and dislocations (**Fig. 5.8**). These are commonly seen in older birds, malnourished birds, or those that have been subject to trauma. Care must be taken to ensure that subtle changes are not overlooked when obvious fractures or dislocations are seen. Coracoid fractures, in particular, can be easily overlooked.
- Arthritis (**Fig. 5.9**) is common in older birds although septic arthritis can occur at any age.



Figure 5.8 **Radiograph of a cockatoo with a femoral fracture.**



Figure 5.9 **Radiograph of a 36 year-old female galah shows signs of arthritis in this femero-tibiotarsal joint.**

The amount of bony proliferation with degenerative joint disease in birds is less obvious radiographically than in mammals. Septic arthritis in birds often presents radiographically as an enlarged joint space and requires arthrocentesis to confirm.

- Osteomyelitis typically presents as osteolysis and periosteal reaction. It is distinguished from osteosarcoma by its diffuse nature, often affecting several adjacent bones.
- Neoplastic changes in the bones usually appear lytic, although periosteal reactions and remodelling of the bone can be frequently seen. Osteosarcomas in bird do not appear to metastasise as quickly as they do in mammals.
- Polyostotic hyperostosis. Under the influence of oestrogen, calcium is laid down in the medulla of the long bones as a reservoir for egg formation. This gives the bones a patchy or solid radio-opaque appearance. This condition is also seen in oestrogen-secreting Sertoli cell tumours (**Fig. 5.10**).
- Osteopaenia is seen as a reduced radio-opacity of the bones, often with a patchy appearance in the medullary cavity.

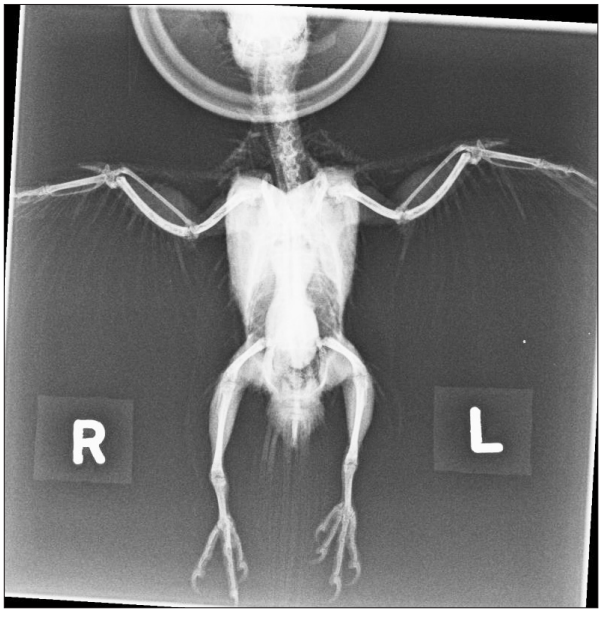


Figure 5.10 **Radiograph of a male budgerigar reveals polyostotic hyperostosis. This bird has an oestrogen-secreting Sertoli cell tumour.**

Downloaded From Vet-eBooks.com

Cardiovascular system

Abnormalities seen include:

- Cardiomegaly is occasionally seen, but it requires determination of the heart width:rib cage width ratio (see above) to distinguish absolute cardiomegaly from a relative cardiomegaly seen with microhepatica (see below) (**Fig. 5.11**).
- Pericardial effusions can be seen as a globoid cardiac silhouette, but ultrasound is needed to definitively visualise fluid in the pericardial sac.
- Atherosclerosis can be seen as increased radiopacity of the great vessels, especially the aorta. In more advanced cases these vessels can demonstrate a patchy mineralised appearance.

Respiratory system

Abnormalities seen include:

- Pneumonia and pulmonary contusions can result in a generalised decrease in the prominence of the parabronchi.
- Air saculitis has a spectrum of severity, and can present with variably radio-opaque lines highlighting thickened air sacs, through to near complete loss of coelomic detail.
- Tracheal foreign bodies can occasionally be visualised, although care must be taken not to confuse ingesta in the crop with a tracheal foreign body.
- Hyperinflation of the air sacs occurs with chronic respiratory disease and is seen as relatively over-inflated air sacs, extending down around the cloaca and caudal intestinal tract.
- Subcutaneous emphysema occurs with air sac rupture associated with either trauma or chronic infection. Air can be seen under the skin anywhere on the body.

Digestive system

Abnormalities seen include:

- Crop stasis. Although it is common to have a small amount of ingesta in the crop, large quantities of fluid and ingesta distending the crop are considered abnormal.
- Proventricular dilation. The proventriculus is usually empty and compact when examined radiographically, with a clearly defined isthmus where it enters the ventriculus. The presence of gas, ingesta or grit in the proventriculus is abnormal, especially if the proventriculus is greatly distended (**Fig. 5.12**).



Figure 5.11 Radiograph of a budgerigar showing cardiomegaly.

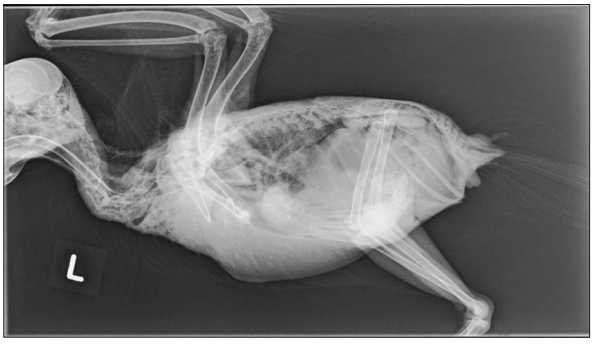


Figure 5.12 Radiograph of an eclectus parrot showing proventricular dilation.

Downloaded From Vet-eBooks.com

- Ventricular impaction. While it is common to see a small amount of radio-opaque material in the ventriculus, it should not be tightly compacted or extending cranially into the proventriculus or caudally into the duodenum.
- Filling defects in the proventriculus and ventriculus, suggestive of a foreign body. (**Fig. 5.13**)
- Intestinal ileus. Large, gas-filled loops of intestine are suggestive of intestinal ileus.
- Cloacal impaction. The cloaca should be visible as a soft tissue density dome-shaped termination of the rectum. Distension or the presence of radio-opaque material is abnormal.
- Coelomic effusion. Loss of detail in the coelom is suggestive of fluid. On a lateral view the ventral coelomic wall between the end of the sternum and the pubic bones should be slightly concave; a convex ventral wall is suggestive of coelomic distension die to effusion, organomegaly or excessive coelomic fat.

Liver

Abnormalities seen include:

- Hepatomegaly. An enlarged liver extends laterally past the lines drawn between the shoulder and acetabulum (**Fig. 5.14**). On a lateral view the space between the dorsal liver and the ventral proventriculus disappears, the proventriculus may be pushed dorsally and the ventriculus caudally.
- Microhepatica. It is normal in some species (e.g. macaws) to see a liver that is much smaller in appearance than expected. This is usually not a clinical condition.
- Traumatic injuries. Blunt force trauma (e.g. collisions with windows and cars) can lead to rupture of the air sacs and air leaking out into the coelom. This is seen as a separation of the liver ventrally from the sternum, cranially from the liver, and caudally from the ventriculus.

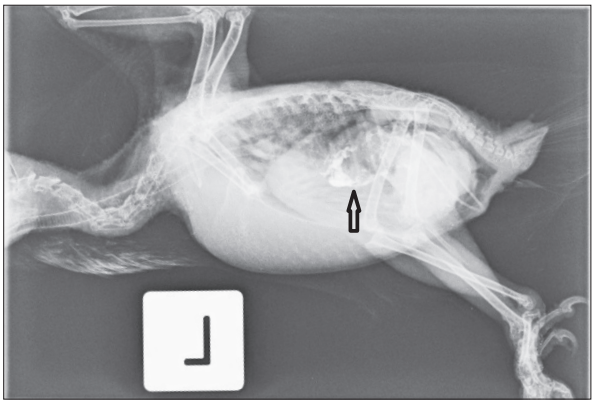


Figure 5.13 **Radiograph examination of a lorikeet following administration of contrast media reveals a proventricular obstruction, seen as a filling defect in the proventriculus.**



Figure 5.14 **Radiograph of a galah reveals hepatomegaly.**

Spleen

Splenomegaly is readily apparent on the lateral view (**Fig. 5.15**) and occasionally on the VD view. While it usually suggests an infectious process, splenomegaly can also be seen with leukaemic diseases such as lymphoma or lymphosarcoma.

Downloaded From Vet-eBooks.com

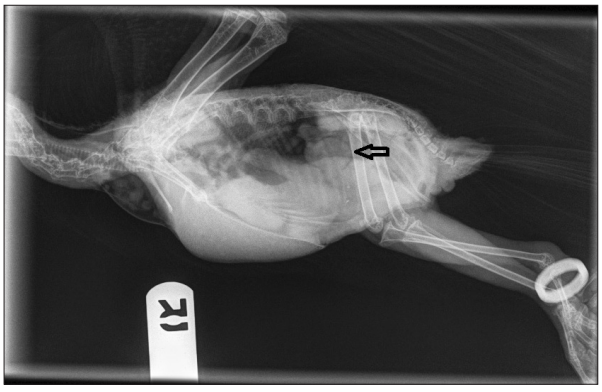


Figure 5.15 **Radiograph of a parrot indicates splenomegaly (spleen location shown).**

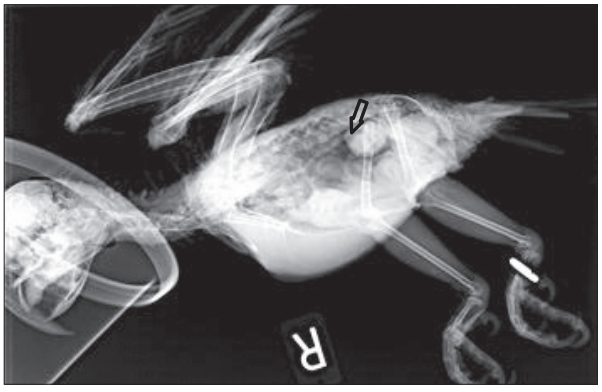


Figure 5.16 **Radiograph of a parrot indicates nephromegaly (arrow).**

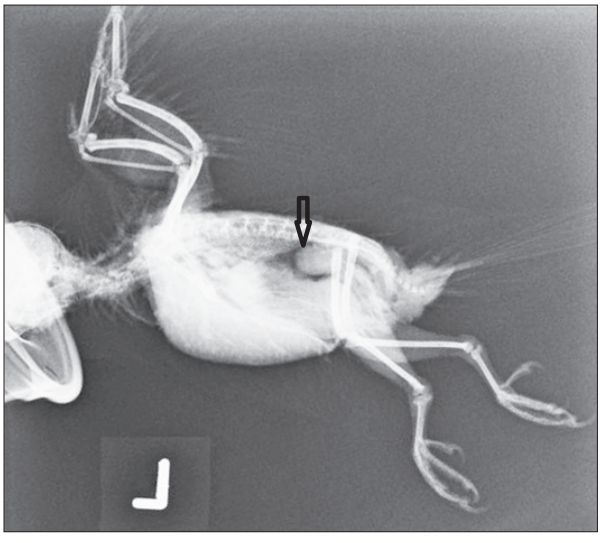


Figure 5.17 **Radiograph of a budgerigar reveals a Sertoli cell tumour at the cranial pole of the kidney. Note the increased radio-opacity of the bones (polyostotic hyperostosis).**

Urogenital system

Abnormalities seen include:

- Nephromegaly. The kidneys are normally surrounded by air and lie above a line through the ventral acetabular rim and parallel to the spine. Extension ventral to this line and loss of the surrounding air space is suggestive of nephromegaly (**Fig. 5.16**).
- Renal mineralisation, seen as increased radio-opacity of the kidneys, is uncommon.
- Urolithiasis is rarely seen. Radio-opaque densities lying between the caudal division of the kidney and the cloaca are very suggestive of uroliths. Other findings include an atrophic ipsilateral kidney with a normal to hypertrophic contralateral kidney. In some cases a dilated ureter obstructed with one or more urate stones may be visible radiographically.
- Testicular neoplasia may present as increased mass-effect in the area of the cranial division of the kidney. It must be distinguished from enlargement associated with normal seasonal hormonal activity. The degree of this enlargement is somewhat species-specific (e.g. in ducks there is a marked enlargement). If a Sertoli cell tumour is present, the resultant hyperoestrogenism may produce polyostotic hyperostosis (**Fig. 5.17**).
- Ovarian enlargement will also produce an increased mass-effect in the area of the cranial division of the kidney. Large cysts may cause a loss of coelomic detail and must be distinguished from a coelomic effusion by ultrasound.
- Egg binding is usually readily apparent as an egg in the caudal coelom (**Fig. 5.18**). If it has been present for more than a few days the shell may become thickened and roughened in appearance. Soft-shelled eggs may be more difficult to visualise, requiring ultrasound to detect.
- Retained eggs present radiographically as either very thick-shelled eggs or collapsed radio-opaque shapes that are not readily recognisable as an egg (**Fig. 5.19**).

Downloaded From Vet-eBooks.com

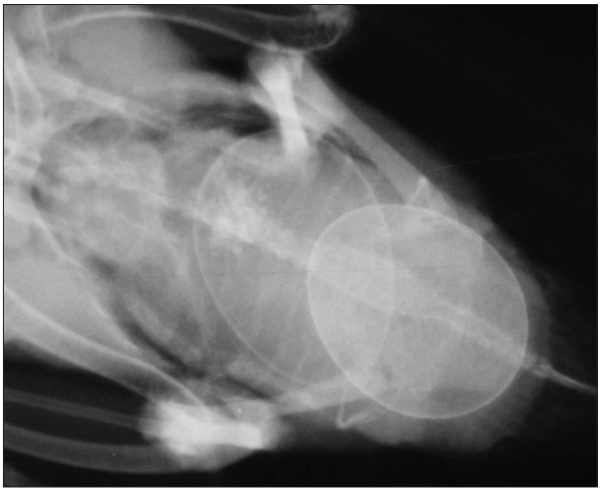


Figure 5.18 Radiograph of a cockatiel with egg binding.



Figure 5.19 Radiograph of this cockatiel reveals a retained and collapsed egg.

ULTRASOUND

Ultrasonography is not commonly used in avian medicine. The sonogram produced by the reflection of high frequency sound waves generated by a transducer is based on the unique echoes produced by different tissues. Bone completely absorbs the sound waves, while air reflects them (preventing passage through to deeper structures). The presence of air sacs therefore limits the usefulness of ultrasound in birds. However, it can be used to give some definition of the internal structure of tissues and organs, including examination of small organs such as gall bladder, spleen and pancreas; examination of fluid-filled lesions; and to measure cardiac function. It is a valuable tool in assessing coelomic distension in avian patients (fluid vs organomegaly) (**Fig. 5.20**).

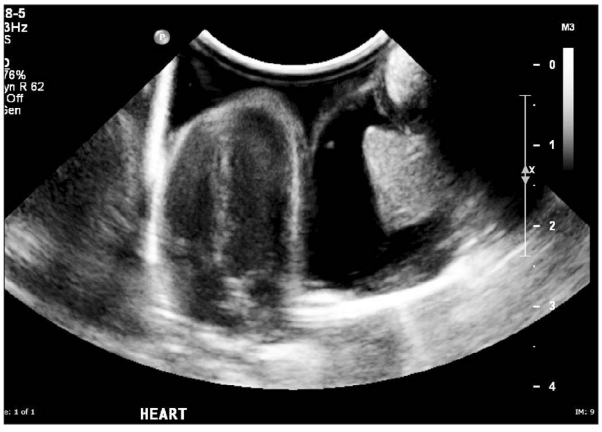


Figure 5.20 Ultrasound image from an Alexandrine parrot with dilated cardiomyopathy and free coelomic fluid.

The patient should be fasted, if possible or safe, prior to the procedure. A 7.5–18 MHz transducer with a small contact area (e.g. a paediatric scanner) is most commonly employed. The most common 'windows' used are the ventromedial coelom (caudal to the sternum) and right lateral coelom, behind the last rib. (The right side is used to avoid interference from grit in the ventriculus.) With the patient restrained or anaesthetised in an upright, dorsal, or left lateral position, the feathers are parted (to reveal apterylae) or plucked and ultrasound gel applied to the skin. (Be aware that ultrasound gel can affect radiographic images – if both ultrasound and radiology are planned, radiology should be performed first.) Stand-offs may be required for small patients.

Using the ventromedial window, the liver and heart are first examined in two planes (vertical and horizontal). The transducer is then directed to the left of the bird's body to examine the gastrointestinal tract from the proventriculus to the cloaca. Using a high frequency transducer the wall of the tract and peristaltic movement can be assessed. The pancreas, lying against the ventral coelomic wall, is often difficult to see even using a spacer. The kidneys and gonads, surrounded by air sacs, are difficult to visualise unless enlarged. Mature active gonads, especially ovarian follicles, may be seen; immature

Downloaded From Vet-eBooks.com

Table 5.1 **Effective uses for ultrasonography in diagnostic imaging of birds**

ORGAN	NORMAL	PATHOLOGY
Liver	The parenchyma has a homogenous, finely granular appearance with sharp edges. The gall bladder, if present, lies to the right of midline.	Blood vessels appear enlarged, with increased wall density, with severe hepatomegaly. Hepatic lipidosis produces hepatomegaly with a diffuse increase in echogenicity. Neoplasia causes focal or diffuse changes in echogenicity. Abscesses, granulomas or necrosis also produce focal or diffuse changes in echogenicity. Haematomas produce focal, hypoechoic lesions.
Spleen	Can be seen on the lateral approach. Its size and shape varies between species. It has a slightly more granular and echodense appearance than the liver.	
Kidneys	Normal kidneys are difficult to visualise	Enlargement is readily seen in some patients. Neoplasia causes a non-homogenous parenchyma. Renal cysts are clearly defined anechoic areas.
Reproductive tract	Both approaches can be used. Inactive gonads are difficult to detect, while active follicles appear as anechoic areas. The oviduct can be difficult to distinguish from the intestines unless an egg is present. It lacks peristalsis, a feature which may assist in differentiating the two tracts.	Eggs are easy to detect if shelled. Prior to this they can be seen as the hyperechoic yolk surrounded by the hypoechoic albumen. Ovarian cysts and gonadal neoplasia are also detectable. Metritis and pyometra can be detected readily. Free fluid in the coelomic cavity associated with yolk peritonitis is usually obvious.
Gastrointestinal tract	Structure and motility can be visualised. The proventriculus is often only visible if enlarged. The ventriculus is identifiable by the presence of shadowing arising from insoluble grit in the lumen. Layers within the intestinal walls can be recognised. The duodenal loop is easy to identify, as it lies close to the body wall. A spacer may be required. The cloaca has a narrow, hypoechoic oval form with a regular edge. The presence of faecal material can make visualisation difficult, but retrograde flushing from the vent can help to remove this material.	Proventricular dilation can be seen e.g. PDD, lead toxicosis. Thickening of wall (>2 mm) can be seen with enteritis. Cloacal concretions, papillomas and dilation can be seen.
Pancreas	The pancreas is observed as a hyperechoic structure lying between the loops of the duodenum, with a blood vessel through the centre of its structure.	
Heart	Only B mode and Doppler flow have been used successfully in birds.	Cardiomegaly and pericardial effusion are readily differentiated. Mitral valvular disease has been reported.

gonads are difficult to identify. *Table 5.1* provides a summary by body organ of what can be usefully detected in terms of pathology when compared with 'normal' tissues using ultrasound techniques.

COMPUTERISED TOMOGRAPHY (CT)

Avian anatomy, with an often complex overlay of bone and soft tissue structures, can sometimes make radiographic assessment quite difficult. The use of new technologies such as computerised tomography (CT) (**Fig. 5.21**), fluoroscopy and magnetic resonance imaging (MRI) can assist with non-invasive assessment of internal organ structure and function.

CT makes use of computer-processed X-rays to produce tomographic images ('slices') of the scanned object (**Fig. 5.22**). The X-ray slice data is generated using an X-ray source that rotates around the object as the object passes through the beam; X-ray sensors are positioned on the opposite side of the circle

Downloaded From Vet-eBooks.com



Figure 5.21 **A cockatoo anaesthetised and positioned during a computed tomography (CT) scan.**

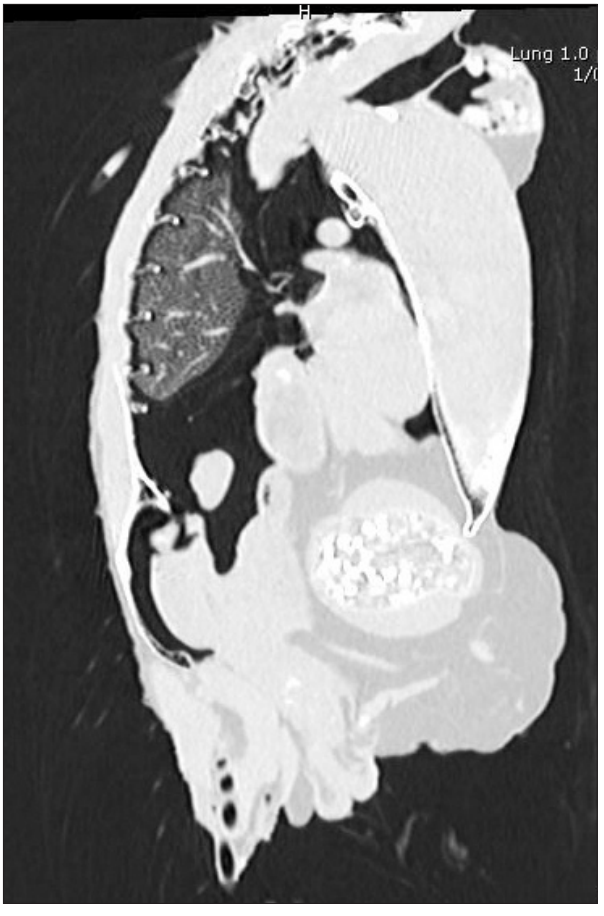


Figure 5.22 **A saggital section taken from the CT scan of the cockatoo, revealing the extent of a ventral hernia.**

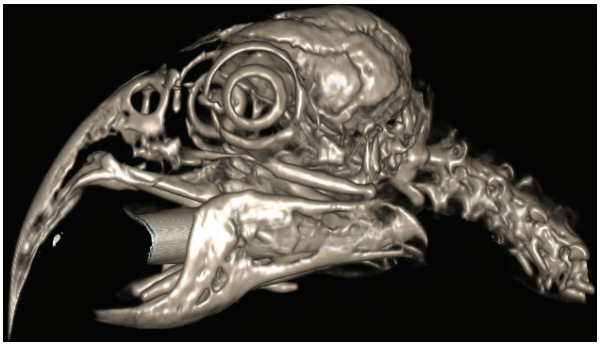


Figure 5.23 **A 3D reconstruction of the head of a long-billed corella following CT scanning.**

from the X-ray source. Increasing the number of sensors increases the resolution (e.g. a 16-slice scanner has 16 sensors). The data is then processed by a computer to generate images in a number of planes (i.e. transverse, sagittal and coronal) and even three-dimensional reconstructions.

A new development is the use of micro-CT to produce even higher resolution images and 3D reconstructions of small patients such as birds (**Fig. 5.23**). This technology is not readily available to most clinicians, but is being used to gain a more detailed understanding of avian anatomy and disease processes.

Although only short scanning times are required with modern equipment, it is vital the patient does not move during the procedure. General anaesthesia is therefore usually required. Although scanning times are short, it does take some time to format the images and more time to interpret them.

Exposure to radiation must be minimised by limiting the area to be examined and the number of studies performed in a short period of time. Intravenous contrast media can be used to help distinguish between soft tissue structures in the coelom, and to highlight areas of inflammation.

FLUOROSCOPY

Fluoroscopy uses an image intensifier that amplifies low level ionising radiation to allow capture of real time motion using a closed charge-coupling video imaging device. Anaesthesia is often not required; the patient can often be restrained by

Downloaded From Vet-eBooks.com

placing it in a closed cardboard box fitted with a wooden perch.

This technique is particularly useful for studying gastrointestinal motility, especially in diseases such as proventricular dilatation disease (see a detailed description in Chapter 16, Diseases of the Gastrointestinal Tract, p. 235). By gavaging the patient with a contrast media such as barium and then placing it in a cardboard box, the passage of the contrast media through the gastrointestinal tract can be watched in 'real time'. Care must be taken with the length of time utilised for a study, as radiation burns can occur with prolonged exposure (**Fig. 5.24**).

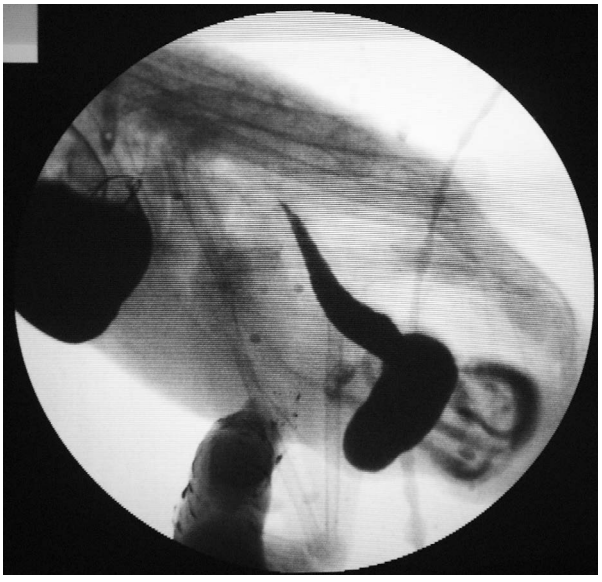


Figure 5.24 **An image taken during a fluoroscopic examination of an Eclectus parrot following administration of a contrast media as part of a gastrointestinal motility study.**

MAGNETIC RESONANCE IMAGING (MRI)

Magnetic resonance imaging (MRI) scanners use strong magnetic fields and radiowaves to form images of the body. MRI allows subtle differentiation between soft tissues, but CT is more useful when evaluating bony structures. Because MRI does not use ionising radiation it is considered to be safer for the patient than CT. Drawbacks to its use clinically in birds include the cost, the long scan times with associated prolonged anaesthesia, and the limited cases in which information can lead to a successful treatment. MRI appears to be most useful in screening for brain or spinal injuries or disease such as neoplasia, cerebrovascular accidents, hydrocephalus or spinal cord injuries.

FURTHER READING

Echols S An online resource. The grey parrot anatomy project: http://www.avianstudios.com/the-grey-parrot-anatomy-project (accessed December 2014).

Helmer P (2006) Advances in diagnostic imaging. In: *Clinical Avian Medicine Vol 2*. Harrison GJ, Lightfoot TL (eds). Spix Publishing, Florida, pp. 653–659.

Silverman S, Tell LA (2010) *Radiology of Birds: An Atlas of Normal Anatomy and Positioning*. Saunders/Elsevier, St Louis, Missouri.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 6

ENDOSCOPY

Endoscopy, which is the internal examination of body organs, joints and cavities using an endoscope, first began in the early 1900s when medical doctors developed telescopic systems that could illuminate the internal cavities of human patients. In avian medicine, rigid endoscopes were first used regularly in the 1970s to determine the sex of parrots, many of which are sexually monomorphic (i.e. there are no distinguishing external physical differences between the sexes). Endoscopic examination enabled the identification of gonads, located between the lung and kidney, leading to a rapid and accurate identification of sex which revolutionised the breeding of many species. It was quickly realised that the unique avian anatomy, with its air-filled body cavities, made birds an ideal candidate for endoscopy. Insufflation is not required, reducing the costs and technical difficulty involved. New techniques and instruments have since been developed to take advantage of this, and endoscopy is now a widely utilised diagnostic tool in avian medicine around the world.

EQUIPMENT

Modern rigid endoscopes utilise a series of glass rods separated by small air spaces contained within a rigid telescope. This rod lens system, often referred to as the Hopkins' system after its designer (**Fig. 6.1**), is considered to give better light transmission, image resolution, and magnification and a wider field of view than the older convex glass lens systems. Most practitioners use a 2.7 mm rigid endoscope with a 30° oblique view. This allows a much wider field of view than the more common 0° scopes. 1.9 mm endoscopes are available for smaller patients (<100 g), but the loss of optical detail limits their use.

Despite its rigidity, the endoscope is susceptible to damage if excessively flexed or torqued. For this reason it is recommended to use either an examination and protective sheath or an operating sheath. The sheath slides over the telescope and locks into place. The operating sheath provides two stopcocks (to allow irrigation or insufflation) and an operating channel for the use of endoscopic instruments.

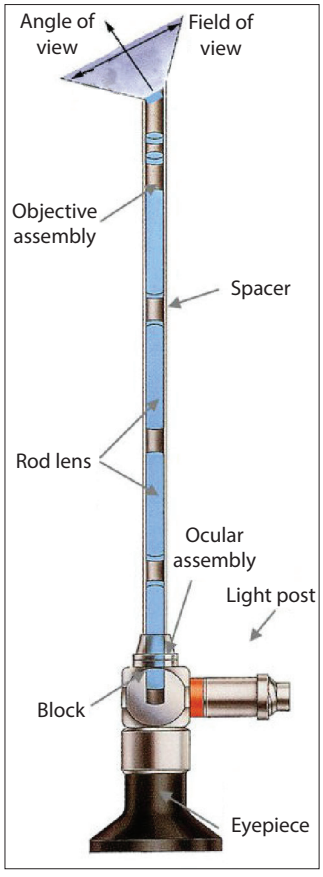


Figure 6.1 **Illustration of the principle components to the Hopkins' rod lens system.**

Connecting the telescope to the light source is a cable (also known as a fibre optic light guide). This is composed of bundled fibre optic cables that transmit light from the source to the telescope.

Downloaded From Vet-eBooks.com

The fibre optic cables are flexible, but do not readily withstand twisting forces.

The use of cold light sources (i.e. light generated without the use of incandescence) minimises the generation of heat at the tip of the telescope. Most commonly used light sources are either tungsten-halogen or xenon. Xenon light sources provide a more intense, whiter and cooler light than tungsten-halogen. Not only does this give better illumination while viewing inside the body, it is essential if cameras and video documentation are required. Smaller LED light sources are now also available, but do not provide the degree of illumination provided by the xenon light source.

Although the endoscope can be used with the operator viewing the image through the telescope's eyepiece, this can lead to both eye and back strain. The use of an endoscopic camera and monitor provides better magnification of the operating field while being more ergonomic for the operator. A three-chip digital camera generates a high-quality picture and is light and easy to use whilst attached to the endoscope. Viewing the image on a high-quality monitor improves both the image seen and the operator's ergonomics (**Fig. 6.2**). Video recordings are desirable as they document procedures which can be a valuable tool for educating both veterinarians and clients.

A variety of endoscopic instruments are available for use. These include: biopsy forceps; grasping forceps; scissors; aspiration/injection needles; radiosurgical probes Two sizes of radiosurgical probes are used most frequently: 5 Fr (for use with the 2.7 mm endoscope) and 3 Fr (for use with the 1.9 mm endoscope). The biopsy forceps are used for collecting tissue biopsies. The cutting edge is sharp, and excessive force to close the forceps is usually not warranted and may result in crushing artifacts. The grasping forceps are used to manipulate tissue and remove small foreign bodies. The scissors are most frequently used for incising fibrous capsules (e.g. over the liver) or air sacs. The fine aspiration/injection needle is used to inject drugs into lesions, irrigate a small area, or to aspirate material. The use of radiosurgery probes allows the use of radiosurgery during endosurgical procedures (**Figs 6.3a–d**).



Figure 6.2 **Example of a portable unit composed of a light source, camera, monitor, recording equipment and light cable (©2015 Photo courtesy of KARL STORZ GmbH & Co. KG).**

Downloaded From Vet-eBooks.com

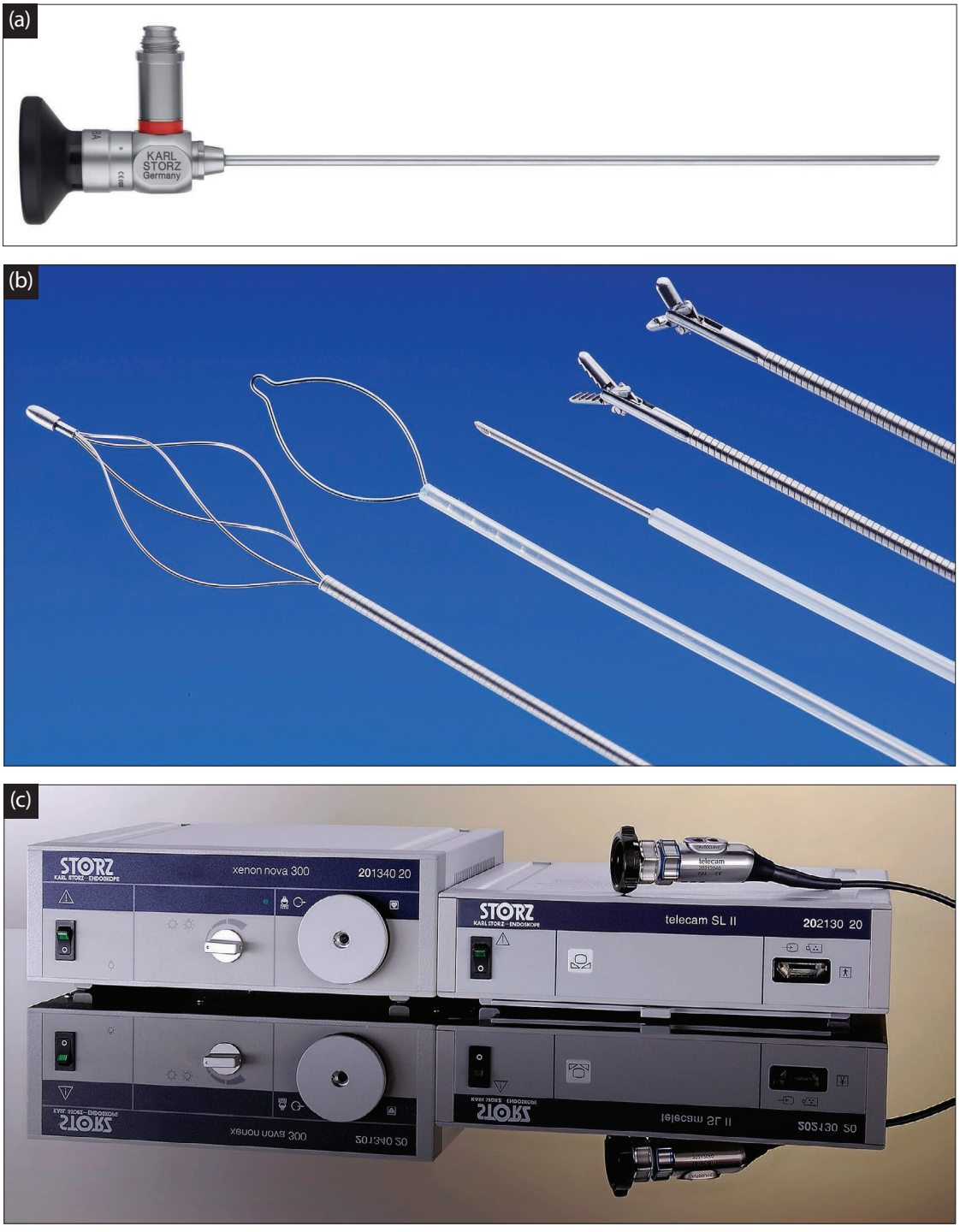


Figure 6.3 (a) Endoscopic telescope with a 30 degree oblique tip. (b) Endoscopic instruments (left to right): a stone basket; polypectomy snare; flexible needle; grasping forceps; biopsy force. (c) An endoscopy light source and camera. *(Continued)*

Downloaded From Vet-eBooks.com

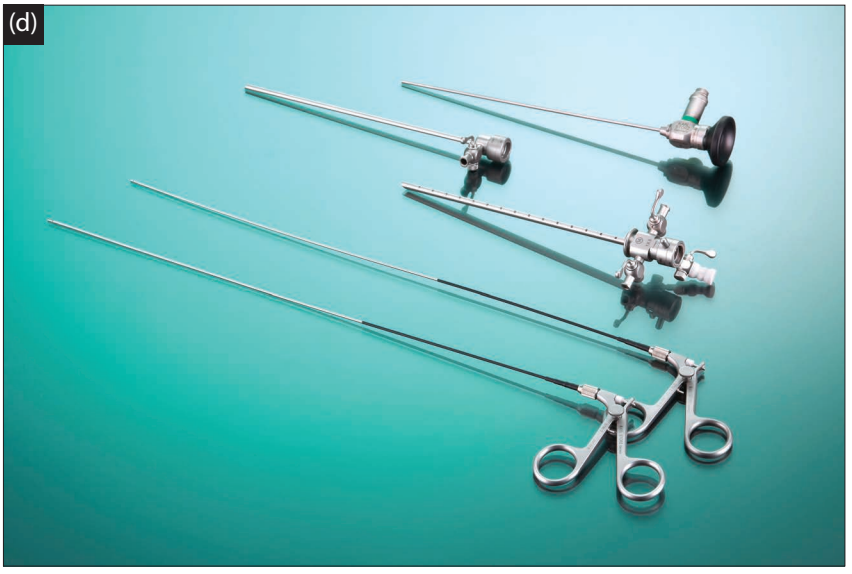


Figure 6.3 (Continued) **(d) The basic endoscopy kit: telescope; examination sheath; biopsy forceps; grasping forceps (©2015 Photo courtesy of KARL STORZ GmbH & Co. KG).**

CARING FOR THE EQUIPMENT

Handling

Rigid endoscopes are fragile and must be handled with care during transport, use and cleaning. Each telescope contains glass fibre optic bundles and a series of rod lenses; excessive forces applied to these fragile components can damage the telescope to the point of been unusable. When not in use, the telescope should be stored in the padded telescope case provided by the manufacturer. When out of the case the plastic telescope guard should be placed over the telescope until ready to use and set down on a flat surface to avoid accidental flexion and possible fracture of the optics.

Rigid endoscopes should always be picked up by the eyepiece; never by the shaft. Be aware of the torsional forces applied to the shaft of the telescope when in use. Avoid overflexion by partially supporting the long axis of the telescope with one hand while in use. The appearance of a crescent on part of the image indicates that the shaft is in imminent danger of being over-flexed.

The light cable consists of fibre optic bundles; although flexible, these bundles are susceptible to damage if twisted or kinked. Careful handling and coiling of the cable when not in use is essential to prevent broken fibre optics. Broken fibre optic cables will appear as black dots in the viewed image.

Endoscopic instruments must be carefully cleaned after use, and tested to ensure they are opening and closing smoothly.

Cleaning

Disconnect the light cable from the telescope, and remove any light cable adaptors. Remove any residual blood, protein material and contaminants with a sponge, soft cloth, or a cotton cloth applicator using a neutral pH enzymatic cleaning solution and then place the telescope in a flat plastic container and soak with the same cleaning solution for no more than 45 minutes. Never use an ultrasonic cleaner. Rinse thoroughly in distilled water to remove any residual cleaning solution.

Clean the lenses (tip and eyepiece) and the fibre optic inlet post with alcohol wipes or cotton tip applicators soaked in 70% alcohol to remove any residue or film. When satisfied they are clean, wipe these optical elements clean with a lint-free soft cloth and then dry the entire telescope with a lint-free soft cloth or filtered, compressed air.

Downloaded From Vet-eBooks.com

Sterilisation/disinfection

Rigid telescopes may be sterilised or disinfected following the manufacturer's recommendation. If cold sterilising, do not soak for more than 45 minutes. Some telescopes can be autoclaved although this may affect their longevity. Only use an autoclave after confirming with the manufacturer that the telescope is suitable for this process. Ensure that the autoclave is placed in a suitable container, and is not in contact with other metal items (including the container). Do not wrap the telescope in a towel, as this may leave residue and stains. After autoclaving, allow the telescope to slowly cool to room temperature – abrupt temperature changes may cause the glass rods to fracture.

OPERATING THE ENDOSCOPE

Endoscopy equipment should be stored in a mobile vertical tower with the monitor on top at the operator's eye level. This allows the equipment to be positioned so that the operator, sitting or standing facing the patient, is also squarely facing the monitor. Such a position reduces operator fatigue and avoids twisting the operator's back or neck during lengthy procedures.

Wherever possible, the endoscope should not be used without the protective or operating sheath over the shaft. After making a suitable entry incision the shaft is held and supported by the inferior hand, while the dominant hand holds and controls the body of the telescope and camera in a 'pistol-grip' as demonstrated in **Fig. 6.4**. This position is maintained as the endoscope is introduced and the body cavity is examined. If it is necessary to use instrumentation, the inferior hand is closed around the shaft, with the thumb running along the shaft towards the operator. This hand is now taking the full weight of the telescope and camera. The dominant hand is free to introduce and use instruments.



Figure 6.4 **Demonstration of the 'pistol grip' recommended for holding an endoscope.**

BIOPSY COLLECTION

Endoscopy is often used to collect tissue biopsies for histopathology or culture. Manipulating the endoscope as described above, the biopsy forceps are introduced into the operating channel until they are just visible in the operating field. They are then advanced slightly to ensure that the spring-loaded cups can open and close without interference from the operating sheath. The operator should avoid the temptation to advance them any further; instead, the entire system is advanced as one unit to the organ of interest. A suitable area is selected and the biopsy cups are opened and then closed firmly on the selected site. If the cups do not cut through the tissue cleanly a short sharp tug is used to collect the sample, rather than dragging the organ away from its normal position.

With the cups closed, the biopsy forceps are then drawn back into the sheath and either the forceps or the entire unit are withdrawn from the patient. The tissue biopsy is then removed gently from the cups; the author prefers to irrigate with a fine stream of sterile saline so that the sample is 'washed' onto a piece of gauze swab in a tissue cassette. The sample can then be placed in formalin or culture medium.

ENDOSCOPIC APPROACHES

Several approaches are used in birds; the more commonly used are described below:

Tracheal approach

This is used for examination of the trachea and syrinx for foreign bodies, diphtheritic plaques and tracheal strictures. It is important to note that for anything other than a quick examination of the trachea and syrinx that the patient should have an air sac catheter placed to provide anaesthesia and respiratory

Downloaded From Vet-eBooks.com

support during the procedure. Under anaesthesia the patient is placed in either dorsal or ventral recumbency, and the head and neck are extended. The endoscope is introduced over the tongue and through the glottis into the trachea (**Fig. 6.5**). It is important that the patient is in a surgical plane of anaesthesia and the trachea is kept extended and straight to avoid iatrogenic trauma. It is also important to keep in mind that the tracheal cartilage in birds is a complete cartilage ring – and therefore the trachea cannot be stretched from the inside – and that the lumen of the trachea narrows as it passes caudally to the syrinx (**Fig. 6.6**). If the syrinx cannot be visualised, it is sometimes helpful to withdraw the endoscope, reverse the patient's positioning (i.e. dorsal to ventral and *vice versa*), and then reintroduce the endoscope. It is not uncommon to see some iatrogenic trauma and bleeding during tracheoscopy, but this rarely causes significant complications.



Figure 6.5 Use of the tracheal approach with an African grey parrot.

Coelomic approach

The standard position is to place the patient in right lateral recumbency with the wings extended and restrained. The left foot is pulled forward and secured to the neck. The left flank behind the leg is then aseptically prepared – it is rare to have to remove more than a few feathers (**Fig. 6.7**). The following landmarks are identified: the caudal rib; the *flexor crura medialis* muscle; and the pubic bone. A 4 mm vertical incision is made in the skin where the *flexor crura medialis* muscle crosses the caudal rib. Curved haemostat forceps are then used to reflect the flexor muscle dorsally and then blunt dissect through the coelomic wall. If done correctly, the operator will feel a 'pop' as the haemostats enter the caudal thoracic air sac.

Once the coelom has been entered the haemostats can be withdrawn and the endoscope introduced (**Fig. 6.8**). If entry has been performed correctly, the endoscope will now be in the caudal thoracic air sac; this can be confirmed by a clear view of the lung and ostium, with air sac membranes visible on the left and right of the view. The endoscope



Figure 6.6 Tracheoscopy procedure in an African Grey parrot reveals a syringeal aspergilloma.



Figure 6.7 An African grey parrot prepared and in position for a left flank coelomic approach.

Downloaded From Vet-eBooks.com

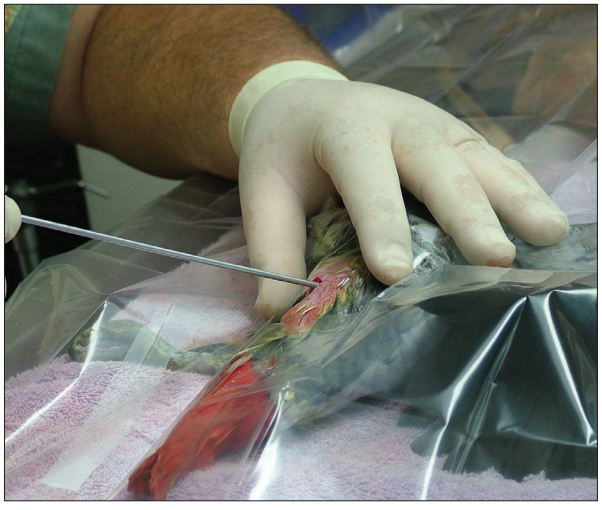


Figure 6.8 **Endoscope introduced in a left flank coelomic approach.**

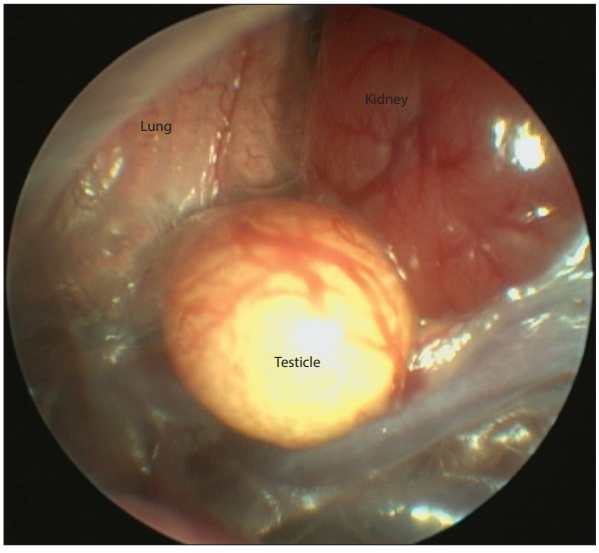


Figure 6.9 **Endoscopic landmarks: sex identification.**

can be advanced to the lung and, in some patients, can enter the lung through the ostium. The air sac membranes on either side of the caudal thoracic air sac can be penetrated either with endoscopic scissors or by placing the tip of the endoscope against the membrane and twisting sharply. Going to the left allows entry into the cranial thoracic air sac; to the right lies the abdominal air sac. The kidney, adrenal gland, gonad and oviduct/ductus deferens are visible within the abdominal air sac, against the dorsal wall (**Figs 6.9** and **6.10**); the spleen and proventriculus are located ventrally, below the gonad-lung-adrenal triad. Within the cranial thoracic air sac lie the liver, proventriculus and, further cranially, the heart.

Once the procedure is completed the endoscope is withdrawn. Tears in the air sac membranes heal well without any requirement for surgical closure. The muscle and skin incisions can be closed if the operator feels it is advisable, but are often left to heal by secondary intention (which occurs rapidly).

Ventral coelomic approach

This approach is used to access the liver for examination and biopsy. With the bird in dorsal recumbency and elevated cranially, the area over the caudal sternum and ventral coelomic wall is plucked and prepared aseptically. A small midline skin incision is made 2–3 mm caudal to the sternum; the linea alba

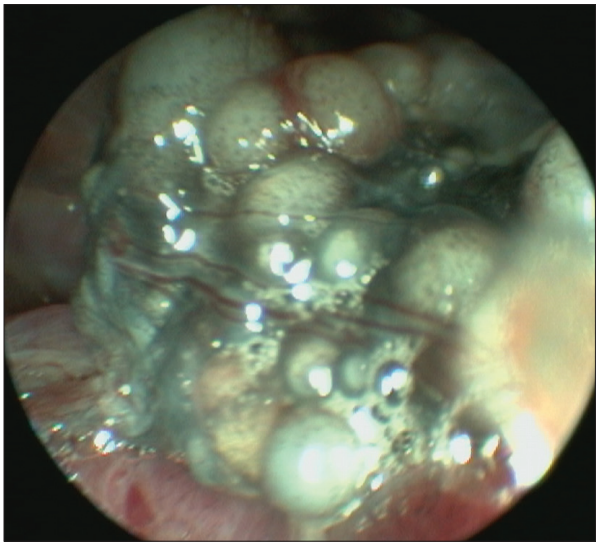


Figure 6.10 **A melanistic ovary observed in a Port Lincoln parrot during endoscopic explorations.**

is identified, lifted and carefully incised. The view may be obstructed by coelomic fat. The endoscope is introduced and directed cranially, to the left and right of the midline. The left and right ventral hepatic peritoneal cavities, formed by peritoneal partitions, are identified and entered after either sharp or blunt dissection. The right liver lobe is larger than the left and is often the easiest to visualise. Once the procedure is completed the linea alba and skin are closed in separate layers.

Downloaded From Vet-eBooks.com

Choanal approach

Introducing the endoscope through the choanal slit allows visualization of the left and right internal nares, the nasal cavities and nasal turbinates. Saline can be flushed through the external nares to provide a better examination and to flush any foreign bodies out – or at least into the field of view. The patient should be intubated before this is performed.

Cloacal approach

Introducing the endoscope into the cloaca while insufflating with saline allows excellent visualisation of the three chambers of the cloaca, the ureteral openings, the opening into the oviduct or the ductus deferens, and – in young birds – the cloacal bursa. After the endoscope is inserted into the cloaca, the vent is occluded while warmed saline is flushed into the cloaca, 'ballooning' the chambers for examination. Care should be taken to release this saline periodically to prevent retropulsion of excessive amounts of saline into the rectum and the rest of the gastrointestinal tract.

Approach to the upper gastro-intestinal tract

The oesophagus can be examined by passing the endoscope past the glottis in an anaesthetised patient. The crop can be examined in birds with crops (parrots, pigeons, doves, gallinaceous birds, and some passerine birds) by passing the tip of the endoscope through the cervical portion of the oesophagus. The bird should be fasted for several hours before the procedure to increase visualisation when possible. Insufflation of the crop with air or water will increase visualisation. An ingluviotomy is usually needed to examine the proventriculus and ventriculus. The bird is anaesthetised, intubated, and placed in dorsal recumbency. The cervical oesophagus or pharynx should be packed off with gauze swabs if possible. A small incision is made into the crop and the endoscope is passed through the distal oesophagus into the proventriculus. The ventriculus may be entered by passing the endoscope through the proventricular-ventricular junction. Insufflation with saline will be necessary; again, care should be taken to remove some of this saline to minimise the amount passing down into the duodenum. When the procedure is completed the crop is closed with a two-layer closure.

FURTHER READING

Divers SJ (2010) Avian diagnostic endoscopy. *Veterinary Clinics of North America: Exotic Animal Practice* **13**(**2**): 187–202.

Divers SJ (2010) Endoscopy equipment and instrumentation for use in exotic animal medicine. *Veterinary Clinics of North America: Exotic Animal Practice* **13**(**2**):171–185.

Downloaded From Vet-eBooks.com

CHAPTER 7

INTERPRETING DIAGNOSTIC TESTS

CHOOSING WHICH TESTS TO USE

Veterinarians treating birds are faced with challenges often not encountered by their counterparts treating dogs and cats. Many birds are presented to veterinarians only when near terminally ill, and a quick answer is often the difference between life and death. Birds are often limited in their range of expression of clinical signs and many clinicians, through no fault of their own, lack the experience to conduct a thorough physical examination. The combination of these factors has led to an increasing tendency in avian medicine to conduct exhaustive diagnostic tests on patients, often with scant attention being paid to a complete history and a careful physical examination, and with little attempt to refine or focus the diagnostic efforts. The selection of diagnostic tests should be based on a solid understanding of the species in question, the results of a thorough history taking and physical examination, and a shortened list of probable differential diagnoses based on this work up.

Before proceeding with diagnostic tests, the clinician should first ask:

- Are the test(s) appropriate to the patient (e.g. species, age, sex) and its clinical signs?
- Has the test been validated to ensure that the result obtained is likely to be both accurate and meaningful?
- Are the physical risks to the patient and cost of the test(s) to the client justified by the likely clinical value of the results?

If the answers to these questions are 'yes', then diagnostic testing should proceed.

Diagnostic testing should be done in tiers, with each level focusing on the diagnostic effort of the next level. Where appropriate, the clinician should endeavour to start with low-cost, minimally invasive tests (e.g. faecal wet smears, faecal flotation and Gram staining) before moving on to more expensive and often more invasive tests. As each tier is passed, the information gained should allow the clinician to focus in on an ever-shortening list of differentials and concentrate efforts towards a final answer.

Clinicians also need to be familiar with the advantages, disadvantages and accuracy of the test(s) they are considering using. They need to be aware of the issues surrounding them. There is controversy and healthy debate within the avian medicine community on many of the tests currently in use. Examples include:

- Are faecal Gram stains an appropriate diagnostic test to use on healthy patients?
- Should the client's money be spent on assessing zinc levels in patients not showing clinical signs consistent with zinc toxicosis?
- Can a diagnosis of zinc toxicosis be made from a single blood level evaluation?
- Can Aspergillosis be diagnosed in a patient based solely on serological tests?
- Can any disease be diagnosed solely on the evidence of a single positive PCR test result or serology?

Available diagnostic tests include:

- Haematology.
- Protein electrophoresis.
- Clinical biochemistries.
- Serology.
- PCR.
- Cultures and Gram stains.
- Cytology.

Downloaded From Vet-eBooks.com

HAEMATOLOGY

The complete blood count (CBC) is an important test in determining many disease states in birds. A CBC involves assessing:

- The erythrocytes, through the determination and assessment of:
  - The haematocrit or packed cell volume (PCV).
  - Total erythrocyte count.
  - Haemoglobin concentration.
  - Erythrocyte morphology.
  - Reticulocytes.
- The leucocytes, through the determination and assessment of:
  - The total white cell count.
  - The leucocyte differential count.
  - The morphology of the leucocytes.
  - Thrombocyte numbers.

Cellular development and morphology is described in Chapter 1, Clinical Anatomy and Physiology.

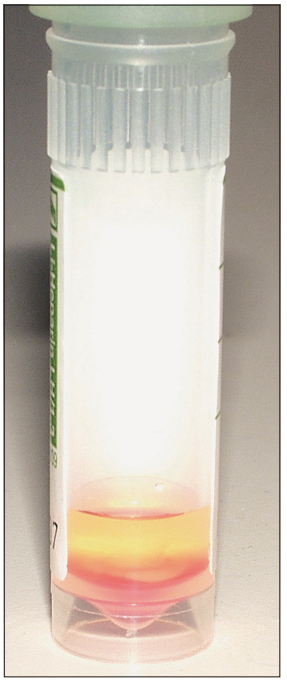


Figure 7.1 **Centrifuged blood sample in lithium heparin, demonstrating a marked anaemia.**

Erythrocytes

The PCV of most parrots lies between 0.4 l/l and 0.55 l/l. A low PCV (<0.35 l/l) can indicate blood loss, anaemia, shock or haemodilution following fluid therapy (**Fig. 7.1**). A high PCV (>0.55 l/l) indicates dehydration or polycythaemia (primary or secondary). Primary polycythaemia may be seen in birds with bone marrow dysplasias. Birds kept at high altitudes, or those with chronic respiratory disease, may be polycythaemic secondary to low oxygenation of the blood.

Total red blood cell (RBC) numbers in birds are affected by age, sex, environment, hormonal influences and hypoxia. Because the erythrocytes are larger, red cell numbers are lower in birds than in mammals. RBC numbers tend to be lower in young birds and females. The normal RBC count in birds ranges from 1.5–4.5 × 10^{12} cells/l.

Morphological abnormalities seen in avian erythrocytes include:

- Excessive polychromasia. Polychromasia is an indicator of the patient's erythrocyte regenerative abilities. Although some polychromasia (1–5%) is normal, excessive polychromasia indicates a regenerative response to blood loss or anaemia.
- Reticulocytosis, especially when combined with increased polychromasia, is seen as a regenerative response to blood loss or anaemia.
- Anisocytosis. Variation in the size of avian erythrocytes is occasionally seen in peripheral blood smears as a normal finding. However, the number increases in response to anaemia.
- Poikilocytosis, or variable cell shapes, may represent artefactual error, but is also seen when severe systemic infections affect the bone marrow. Erythrocytes may appear round, elongated or irregular. The nucleus may vary in appearance, location and number. Erythrocytes that appear round with oval nuclei are indicative of accelerated erythropoiesis. Binucleated erythrocytes may also indicate abnormal erythropoiesis in association with severe, chronic inflammatory processes and neoplasia. Poikilocytes are susceptible to damage, and therefore have a shorter life.
- Erythrocytic ballooning has been reported to be commonly associated with, although not

Downloaded From Vet-eBooks.com

pathognomonic for, lead toxicosis. It is also seen with 'conure bleeding syndrome'. There are bulges in the normal ellipsoid shape of the erythrocyte, often accompanied by areas of hypochromasia.
- Haemoparasites are occasionally seen in the erythrocyte cytoplasm of wild-caught birds or those exposed to biting insects.

Anaemia

Anaemia is usually the result of increased loss, increased destruction and/or decreased production of erythrocytes. It is classified as regenerative, non-regenerative, haemolytic or haemorrhagic.

Regenerative anaemia

This is characterised by the presence of polychromasia, reticulocytosis, macrocytosis and anisocytosis (**Figs 7.2, 7.3**). It is an indication of an appropriate bone marrow response. In order to distinguish between polychromasia and reticulocytosis the blood smear must be vitally stained with new methylene blue.

Non-regenerative anaemia

This is the most common type of anaemia described in birds. It arises due to a lack of appropriate bone marrow response combined with the relatively short life of avian erythrocytes. It is characterised by a lack of a regenerative response (i.e. little or no reticulocytosis, polychromasia or anisocytosis). Possible aetiologies include:

- Acute, overwhelming infections: bacterial, viral (e.g. PBFD) or fungal.
- Chronic disease including chronic inflammatory conditions, chronic infectious diseases (tuberculosis, colibacillosis, salmonellosis, Aspergillosis, chlamydiosis), some viral diseases, hypothyroidism and neoplasia.
- Toxicosis.
- Nutritional deficiencies.

Haemolytic anaemia

This is often regenerative in nature and is indicated by increased polychromasia, macrocytosis, anisocytosis and reticulocytosis. Possible aetiologies include:

- Haemoparasites (*Plasmodium* spp. and *Aegyptianella* spp.).
- Bacterial septicaemia.
- Acute toxicosis (oil ingestion, lead, aflatoxicosis).
- Immune-mediated conditions.

Haemorrhagic anaemia

This results from blood loss due to trauma, gastrointestinal parasitism, coagulation disorders (as in conure

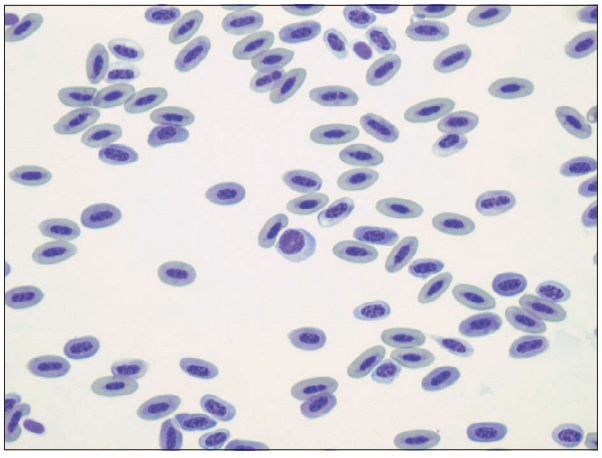


Figure 7.2 **Blood cell sample with polychromasia (variability in erythrocyte cytoplasmic colour) and anisocytosis (variability in erythrocyte size) indicating *mild* regenerative anaemia.**

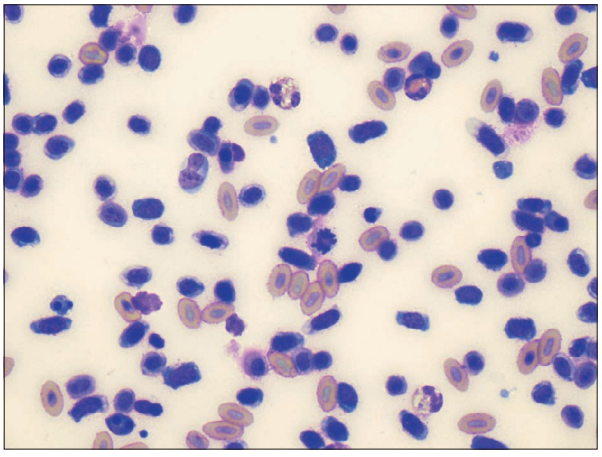


Figure 7.3 **Blood cell sample with mitotic figures, poikilocytosis (variability in erythrocyte shape) and binucleate erythrocytes indicating *pronounced* regenerative anaemia.**

Downloaded From Vet-eBooks.com

bleeding syndrome), organ rupture or ulceration, aneurysms and some viral diseases. Following acute haemorrhage there is often a rapid response during which erythropoiesis increases dramatically. Healthy birds are remarkably adept at dealing with acute blood loss and haemorrhagic shock rarely occurs. Studies have shown that after the loss of even 50% of the blood volume, the PCV returns to normal within 3–7 days.

Leucocytes

The white blood cell (WBC) count and differential are important tools in assessing a patient's response to disease or injury. The WBC count in birds can be determined using three testing methods:

- An automated count, which has recently become available in some laboratories.
- An estimated WBC count determined from a blood smear by counting all leucocytes in 10 high-power (40×) microscopic fields, dividing by the number of fields, and then multiplying this average by 2,000, giving a total WBC/µl.
- The Unopette method using phloxine B stain and a haemocytometer to count eosinophils and heterophils. This count is compared with the percentages of these cells in the differential and the WBC count is calculated using the formula: WBC = (Total Het + Eos)/(% Hets + % Eos) × 100. (Note: In late 2007 the Unopette system was discontinued by the manufacturer. An alternative test is the Avian Leukopet®, Vetlab)

Leucocytosis can be normal in young birds, but it can also be due to:

- Stress.
- Inflammation, often associated with bacterial and fungal infections.

Leucopenia can be due to:

- Chronic inflammation or disease, often with an acute decompensatory episode at the time of presentation.
- Overwhelming bacterial and viral infections (e.g. PBFD in juvenile parrots, especially young African grey parrots).
- Artefacts resulting from poor sample handling (blood clotting) or technique.

White cell differential counts are best obtained from fresh blood smears, as cellular morphology can be affected by anticoagulants in blood collection tubes. A differential count is typically obtained by examination of stained smears under high magnification. Both the type and morphology of the white cells seen are recorded. In many cases the differential count and cellular morphology give more indication of a bird's health status than the total white cell count.

Heterophils

The heterophil is the avian equivalent of the mammalian neutrophil. While it has a similar function to the neutrophil, morphologically it appears quite different. The nucleus contains coarsely clumped chromatin and usually has two to three lobes. The cytoplasm contains eosinophilic, spherical, oval or spindle-shaped granules. It is, in most species, the predominant white cell. Heterophils lack lysozyme which is why birds form caseated, rather than liquid, pus. Abnormal changes seen with heterophils include:

- Heterophilia. Seen with inflammatory disease or stress and is considered normal in many young birds.
- Heteropaenia. Seen with overwhelming septicaemia and certain viral diseases (e.g. PBFD in young African grey parrots). Poor smear preparation technique can 'smudge' many heterophils, causing an artefactual heteropaenia.
- Toxic heterophils (increased cytoplasmic basophilia, vacuolization, nuclear degeneration, degranulation or abnormal granules) are seen with severe diseases that affect production and release from the bone marrow. As the severity of the disease increases, the toxicity of the heterophils may also increase as well.
- Immature (band) heterophils have a less distinctively lobulated nucleus and fewer granules, which are round and deeply basophilic. (Care must be taken not to confuse basophils

Downloaded From Vet-eBooks.com

with immature heterophils.) They are produced in response to bacterial or fungal infections, and indicate a depletion of mature bone marrow storage pool and therefore a poor prognosis.

Eosinophils

The eosinophil is a round cell with a slightly basophilic cytoplasm (in contrast to the colourless cytoplasm of the heterophil). The granules are usually rounded, although shape and colour may vary greatly between species. The granules are distinctly eosinophilic and brighter in colour when compared with the heterophil granules. The function of eosinophils is still largely unknown; eosinophilia in pet birds is rare, sometimes associated with parasitic infections or marked tissue damage.

Lymphocytes

Lymphocytes are second only to the heterophil in frequency in most species except Amazon parrots and some passerines (canaries) in which the lymphocytes appear to be the predominant leucocyte. Size and shape varies, with small, medium and large cells that may be round or moulded around neighbouring cells being seen in the same smear. They have a large, round, centrally located nucleus with densely clumped or reticulate nuclear chromatin. The cytoplasm is clear or slightly basophilic. Reactive lymphocytes (i.e. those responding to antigenic stimulation) usually have a more deeply basophilic cytoplasm, vacuoles in the cytoplasm, a clear perinuclear ring and scalloping of the cell edges. Occasional reactive lymphocytes are a normal finding in avian blood, but large numbers indicate marked antigenic stimulation as seen in severe infections (e.g. severe viral infections, chlamydiosis, Aspergillosis, salmonellosis and tuberculosis).

Lymphocytosis is seen in:

- Chronic infectious or inflammatory conditions.
- Lymphoid leukaemia.
- Normal finding in Amazons and canaries.

Lymphopaenia is seen in:

- Viral infections and diseases that cause bursal damage or bone marrow suppression.
- Relative to a marked increase in heterophils.

Monocytes

Monocytes are the largest of the mononuclear leucocytes, but are rarely seen in peripheral blood smears. They spend only a short time in circulation before passing into tissues and becoming macrophages. The eccentric nuclei are either round, elongated or indented, and the cytoplasm typically stains a blue–grey colour with a reticular or finely granular appearance, with occasional vacuoles. Care must be taken not to confuse them with large lymphocytes.

Monocytosis is most commonly associated with chronic granulomatous infections such as chlamydiosis, tuberculosis, mycoses and bacterial granulomatous diseases (e.g. mycobacterial infections). Because circulating numbers are usually so low, monocytopaenia is not reported.

Basophils

Basophils are uncommon in peripheral blood smears of birds. They appear as small cells with clear cytoplasm and spherical basophilic granules. The nucleus stains a light blue colour. Care must be taken not to confuse them with immature heterophils. Basophilia has been reported in respiratory disease (e.g. air sac mite in canaries), chlamydiosis and tissue trauma more than 48 hours old. In birds basophils appear to play an important role in early inflammatory and immediate hypersensitivity reactions, but differ from those in mammals by not contributing to delayed hypersensitivity.

Thrombocytes

Thrombocytes are small, oval, nucleated cells that can be differentiated from erythrocytes by their size (they are smaller than erythrocytes) and their nucleus, which is larger, more rounded and darkly basophilic-staining. The cytoplasm is colourless or a faint blue colour with one to two small basophilic inclusions at the poles. Total counts are difficult and not routinely performed as the thrombocytes tend to clump. However, there are typically 1–2 cells seen per high-power field. Their function is unclear; they contain little thromboplastin, so it is unlikely that they initiate clotting. With bacterial infections they tend to increase in numbers and become activated (pseudopodial formation and vacuolation) and tend to aggregate in clumps. They appear to have some phagocytic activity.

Downloaded From Vet-eBooks.com

Thrombocytosis is rarely reported and may arise as in response to thrombocytopenia. Thrombocytopenia may occur due to bone marrow suppression or disease processes causing an excessive demand (e.g. viral diseases such as circovirus, reovirus or polyomavirus).

Assessing blood coagulation

Vascular injury results in three events:

- The exposure of blood to tissue thromboplastin (factor III), which activates the extrinsic clotting pathway.
- The exposure of blood to subendothelium, which both activates the intrinsic pathway (which is weak or absent) and stimulates the adhesion and aggregation of thrombocytes to form a plug in the injury.
- Vasoconstriction, through various humoral and local mechanisms, which slows blood flow, reduces blood loss and allows time for thrombocytes to adhere to the injury.

Therefore, the extrinsic pathway is the major means of blood clotting in birds. Following vasoconstriction and thrombocyte adherence, the extrinsic pathway forms a fibrin clot over the thrombocytes and allows the vascular endothelium to begin repairs. This entire process can occur in as little as ten seconds.

Unfortunately there are no commercially available tests to evaluate the avian extrinsic pathway. Clinical tests for the assessment of coagulation in birds include:

- Thrombocyte evaluation. There should be 1–2 thrombocytes per oil immersion field. An estimated thrombocyte count/µl can be performed by counting the average number of thrombocytes in five high-power fields and then using the formula: estimated thrombocyte count per µl = average number of thrombocytes/1,000 × 3,500,000. The normal count is 20,000–30,000 per µl (20–30 × 10^9/l).
- Bleeding time has been assessed in poultry by pricking the comb with a needle. The normal bleeding time averages eight minutes.
- Whole blood clotting time, where whole blood is placed in a plain tube, incubated and then inverted until clot forms. This assesses the intrinsic and common pathway and blood will normally clot in 2–10 minutes. This time will be accelerated by contamination with tissue thromboplastin. An alternative technique is to place blood in capillary tubes and break the tubes until a clot forms, normally in less than five minutes.
- Prothrombin time (PT), which evaluates the extrinsic and common pathway, can be used but requires avian thromboplastin. Mammalian thromboplastin significantly prolongs PT.

PROTEIN ELECTROPHORESIS

Protein electrophoresis – the measurement of different fractions of plasma protein – can take the clinician beyond the white cell count in differentiating a bird's response to disease and stress. Used in conjunction with haematology, it is a valuable tool in the assessment of a bird's health status.

Plasma protein is composed of two major fractions - albumin and globulins. Albumin is a reserve protein and carrier of other molecules, while globulins include the inflammatory proteins, clotting proteins and immunoglobulins. A range of physiological and disease factors affect the levels of protein in the blood, including the bird's age, sex, diet, hydration status, haemorrhage, inflammation, and tissue repair. Albumin and globulin are further broken down into smaller fractions:

1. **Pre-albumin** is often included in measurements of albumin. Its occurrence is variable, with different researchers reporting its absence or presence in birds of different ages and sex. It has a role in thyroxine binding and transport.
2. **Albumin** is produced in the liver and serves as the major storage reservoir of proteins. It contributes 75% of osmotic activity of plasma. Its role is that of a carrier of minerals, vitamins, fatty acids and thyroid hormones.

Downloaded From Vet-eBooks.com

3. **Globulins** are composed of five fractions:
   I. Alpha 1 and 2 globulins are produced in the liver. They include:
      - Macroglobulins - inhibit proteolytic enzymes.
      - Lipoproteins (lipid transport).
      - Haptoglobin - haemoglobin transport.
      - Ceruloplasmin - copper transport.
   II. Amyloid A – acute inflammatory protein.
   III. Beta 1 and 2 globulins are also produced in the liver. They include:
      - Complement: enzymatic proteins involved in antigen-antibody interactions.
      - Haemopexin: haem-binding protein.
      - Ferritin: iron storage protein.
      - Fibrinogen: clotting factor – non-specific indication of inflammation.
      - C-reactive protein: produced in response to tissue injury, inflammation or infection. Immunosuppressive, promotes phagocytosis, inhibits platelets, activates complement.
      - Lipoproteins.
      - Some immunoglobulins (IgM, IgA).
   IV. Transferrin is a gamma globulin in chickens (beta globulin in mammals).
   V. Gamma globulins are the immunoglobulins, produced by B lymphocytes. They include:
      - IgA: found in the intestinal tract.
      - IgM: first immunoglobulin produced.
      - IgG: supersedes IgM after 10–14 days; also known as IgY.
      - IgN: found in some waterfowl.

Protein Electrophoresis (EPH) uses electrophoresis, the movement of charged particles suspended in a fluid on various media (e.g. paper, gel), under the influence of an applied electric field. It is widely used in the separation of plasma protein fractions and is therefore valuable in the study of diseases in which the serum and plasma proteins are altered.

Submission of samples for Protein Electrophoresis

Plasma must be submitted for EPH, as serum is produced by the effect of clotting activation, which reduces fibrinogen and other globulin levels in the sample. Collect the blood into lithium heparin, centrifuge, remove the plasma and submit.

Interpretation of results

As with all diagnostic tests, EPH must be interpreted in conjunction with a good history, clinical examination and other diagnostic tests. It is not an indicator of specific disease, as earlier work had suggested. Rather, it should be used as an indicator of debility and inflammation. Not all diseases will result in changes in an EPH (e.g. neoplasia rarely causes any changes).

Reference intervals vary between laboratories, often significantly. However, a generic 'normal' set of values for parrots is:

Albumin:	1.2–3.2 g/dl
Alpha-1 globulins:	0.8 g/dl
Alpha-2 globulins:	0.8 g/dl
Beta globulins:	0.57 g/dl
Gamma globulins:	0.57 g/dl

Techniques typically employed by laboratories working on mammalian blood invariably report falsely decreased albumin levels. As globulin is calculated by subtracting this albumin level from the total protein, the report received by the clinician will also have incorrect globulin and A:G ratios reported. EPH is considered to be the gold standard for the measurement of avian albumin.

Elevated albumin

- Plasma albumin will increase with dehydration.

Decreased albumin

Decreased synthesis:

- Liver disease.
- Poor diet.

Increased loss:

- Protein losing enteropathies and nephropathies.
- Intestinal parasites.

Increased usage:

- Chronic inflammatory disease.

Downloaded From Vet-eBooks.com

Elevated beta globulins
- Acute inflammation or infection.

Elevated alpha and beta globulins
- Acute infection/inflammation.
- Reproductively active female.

Elevated gamma globulins
- Chronic inflammation or infection.

Elevated beta and gamma globulins
- Chronic-active inflammation.
- Acute infection in a chronically ill bird (e.g. chlamydiosis).

Elevated alpha, beta and gamma globulins, decreased albumin
Severe, chronic-active inflammation or infection in a bird with chronic disease and debility. This is an uncommon pattern, as if a disease is severe enough to elevate all globulins, the bird usually dies before albumin decreases.

Elevated gamma globulins; decreased albumin
Chronic, debilitating disease.

CLINICAL BIOCHEMISTRY

Clinical biochemistry involves the measurement of specific groups of chemicals within the body and the interpretation of the results obtained. In-house diagnostic equipment (**Fig. 7.4**) assists in the rapid diagnosis of critically ill patients. The chemicals measured include:

- Metabolites. Those chemicals that are produced as the end-products of various metabolic processes within the body.
- Tissue enzymes, which catalyse chemical reactions within the body without being altered themselves.
- Electrolytes, including sodium, potassium and chloride.
- Minerals, such as calcium, phosphorus and magnesium.
- Bile acids, produced in the liver from cholesterol and used in the emulsification of dietary fats.



Figure 7.4 **In-house laboratory equipment such as this bench top diagnostic blood analyser enables the rapid diagnosis of critically ill patients.**

The levels of these chemicals in the blood can be influenced by either physiological or pathological processes. Physiological variations can be due to age, sex, body fat to muscle ratio, nutritional status, reproductive status and species. However, pathological processes, including cellular damage or abnormal function of an organ system (or systems), often produce significant changes in blood levels.

There are three major causes of abnormal clinical biochemistries:

- Normal variation between species and individuals.
- Artefacts.
- Pathology.

Normal variation between species and individuals
There are over 9,000 species of birds, with major differences in anatomy, physiology, form and function. Some are carnivorous, some are nectivorous, some are granivorous and some are omnivorous. It is unrealistic to expect that they would all conform to a relatively narrow range of biochemical values.

Downloaded From Vet-eBooks.com

Other variations arise between individuals of the same species. These variations occur because of differences in age, sex, diet, husbandry, etc. For this reason some clinicians recommend establishing a set of normal values for individual birds during annual health examinations, and then using these values as a comparison should the bird become ill.

Artefacts

When interpreting a biochemistry analysis, care must be taken to distinguish between abnormal results due to disease, and abnormal results due to other factors. These other factors, referred to as artefacts, can occur for a variety of reasons, including:

- Physiological changes.
- Previous therapy.
- The clinical condition of the patient.
- The collection method.
- Storage and transport of the sample.

Physiological changes

Stress due to transport and handling of the patient can lead to a release of endogenous corticosteroids, resulting in changes in the haemogram and in blood glucose.

Lipaemia, while occasionally seen in diseases of the liver and reproductive system, can also occur naturally in the reproductively active female (**Fig. 7.5**). Regardless of the cause, lipaemia can cause false elevations in bile acids, protein, calcium, phosphorus and uric acid. It may also falsely decrease amylase. Postprandial lipaemia is uncommon in pet birds, so fasting will not help; the clinician needs to check with the laboratory if the sample submitted was lipaemic before interpreting these biochemistries.

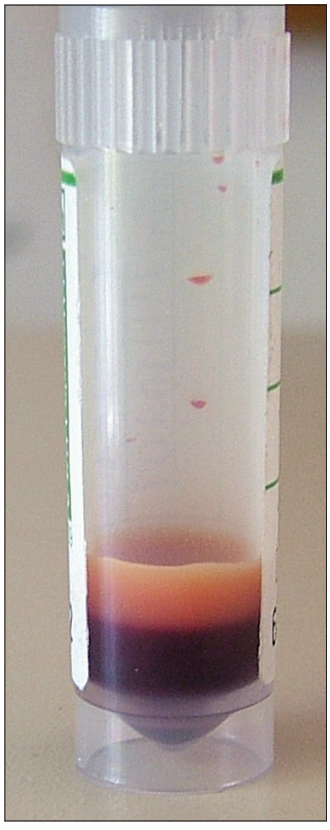


Figure 7.5 Centrifuged blood sample demonstrating lipaemia. This condition in birds is usually associated with high-fat diets, liver disease, diabetes mellitus or reproductive activity.

Previous therapy

Before interpreting biochemistries, the clinician should consider if any treatment given prior to the sample collection could have had an effect on the results. Therapy given by another veterinarian or in an attempt to stabilise a crashing patient can have marked effects. Parenteral fluids can dilute biochemistries; exogenous corticosteroids can markedly elevate aspartate aminotransferase (AST), creatine kinase (CK) and lactate dehydrogenase (LDH); intramuscular injections, particularly of irritant drugs, can do the same.

The clinical condition of the patient

Trauma, starvation and dehydration can all have marked effects on biochemistries, and need to be considered when interpreting results. Trauma can cause elevations in AST and CK and possibly glucose; starvation can lower glucose and also elevate AST and CK if protein catabolism has begun; dehydration can elevate uric acid.

The collection method

Ideally, sample collection should be performed in such a manner that it has minimal impact on the patient while providing an artefact-free sample suitable for analysis. This usually requires venepuncture to be performed on a minimally stressed patient.

Downloaded From Vet-eBooks.com

Inexperienced clinicians may need to consider gaseous anaesthesia in order to collect a good sample without the bird struggling.

Venepuncture should be performed on a large vein (e.g. the jugular) using a needle that is large enough to minimise haemolysis while being small enough to minimise trauma to the blood vessel wall. Haemolysis can cause elevations in bile acids, LDH, CK, alkaline phosphatase (ALP), potassium and phosphorus. Glucose and albumin may be decreased. Calcium may be elevated or decreased, according to the methodology used.

Toenail clipping should be discouraged. Not only is it unduly painful, but crush artefacts and contamination with uric acid and bacteria from droppings on the perch can cause elevations in uric acid and decreased glucose if testing is delayed long enough for bacterial growth to occur in the sample.

Storage and transport of the sample

Blood collected for biochemistry analysis should be placed immediately into a lithium heparin tube. Ideally, miniature tubes as used in medical paediatrics should be used. The sample should be gently rolled or rocked; clotting must be avoided, but haemolysis must be as well. If the analysis is to be performed in-house, it should be processed immediately. If a delay is likely, or if the sample is to be shipped to an outside laboratory, the sample should be centrifuged and the plasma harvested. Sending whole blood to an outside laboratory can result in decreased glucose (as cell metabolism continues) and haemolysis.

EDTA tubes are unsuitable for biochemistry analysis, but can be used for haematology, lead analysis and fibrinogen determination.

Biochemical analysis by organ system

Liver

The detection of liver disease through biochemistry is complicated by the fact that there are no specific 'liver enzymes' that can be evaluated conclusively in each and every case. Liver disease can be broadly classified into three conditions: hepatocellular rupture, decreased hepatic function and cholestasis. These conditions can occur either separately or concurrently.

Hepatocellular rupture

This releases intracellular enzymes, which then reach elevated levels in the blood. These so-called 'leakage enzymes' include:

- Aspartate aminotransferase. This cytosolic enzyme is found in many tissues in the body, but the highest concentrations are found in skeletal muscle and liver. Significant elevations usually represent either muscular or hepatocellular damage. AST, therefore, must be interpreted alongside CK (released from damaged muscle) to distinguish between the two. In general, an elevated AST with a normal CK indicates hepatocellular rupture. However, CK has a much shorter half-life than AST; a single-point muscle injury (e.g. an injection) 4–7 hours before sample collection could duplicate this biochemistry pattern. Although AST is considered to be the most useful liver enzyme, it cannot be considered in isolation as an indicator of liver disease.
- Glutamate dehydrogenase (GLDH), a mitochondrial enzyme, is the most specific enzyme for the detection of liver disease, but its sensitivity is low. Because it is bound to mitochondria, extensive and severe liver damage is required before elevations are detectable.
- Lactate dehydrogenase is not specific to any tissue; its main advantage lies with a half-life shorter than CK. Persistent elevation in the presence of normal CK is strongly suggestive of liver disease.
- Alanine aminotransferase (ALT) and alkaline phosphatase are not considered useful in detecting liver disease in birds. ALT in birds is very non-specific for the liver, and normal levels have been shown in cases with severe liver damage. ALP elevations are more commonly associated with bone disease in birds.

Decreased liver function

Decreased liver function can occur with any number of liver diseases, not all of which involve hepatocellular rupture. Chronic cirrhosis, amyloidosis and hepatic lipidosis can all have an adverse effect on liver function without causing any cellular damage.

Downloaded From Vet-eBooks.com

In these cases a 'liver function test' is necessary to detect the problem. Bile acids serve this purpose well. Produced in the liver, they are excreted in bile into the small intestine where they act to emulsify fat. Most of the bile acids are then resorbed in the small intestine, enter the portal system and are taken up by the liver to be recycled. Elevated levels occur when there is impairment of the liver's ability to remove bile acids from the portal circulation. A two- to fourfold increase in bile acids indicates a significant decrease in liver function. It needs to be noted though that a severely dysfunctional liver (e.g. end-stage cirrhosis) may not be able to produce normal levels of bile acids, leading to low to normal results. Total protein, especially albumin, may also be decreased with decreased liver function.

Cholestasis

Cholestasis occurs when the biliary system is partially or totally obstructed. This can be seen with biliary neoplasia, pancreatic disease or diffuse swelling of the entire liver. Gamma glutamyl transferase (GGT) is an enzyme found in the cell membranes of the bile ducts. Elevations can be seen in cholestatic disease (e.g. bile duct carcinoma), but it is considered to be a relatively insensitive test for liver disease in parrots. Bilirubin is not produced in birds; they utilise biliverdin instead. There are no commercial assays for biliverdin.

Kidney

The end-product of protein metabolism in birds is uric acid. It is produced in the liver, enters the circulation and is then secreted by renal tubules (>90%) or filtered in the glomerulus (<10%). Significant loss of renal tubules will therefore result in elevations of uric acid. Dehydration is less likely to cause hyperuricaemia because glomerular filtration is relatively unimportant.

At first glance it would appear that uric acid offers a sensitive and specific test for renal disease. There are, however, several confounding factors. Firstly, species differences: carnivorous birds have higher normal uric acid levels than granivorous birds. Secondly, age: juvenile birds may have lower levels than adults. Thirdly, although significant elevations usually indicate renal disease, normal levels do not mean the kidneys are normal: mild increases could indicate early renal disease or dehydration (or both). There must be severe renal damage before uric acid levels begin to rise.

Because of this relative insensitivity of uric acid in detecting renal disease, levels are best interpreted alongside a determination of the bird's water intake and loss and a physical examination. To distinguish renal disease from dehydration, the patient's haematocrit, total protein and blood urea nitrogen (BUN) should be evaluated concurrently. Dehydration can lead to decreased glomerular filtration rates (GFRs), in turn leading to elevated levels of BUN; this same decrease in GFR can lead to elevations of uric acid without primary renal disease being present. It is therefore prudent, in cases of an elevated uric acid level, to rehydrate the patient over 2–3 days before definitively diagnosing renal disease. Persistent hyperuricaemia after fluid therapy, and with haematocrit, total protein and BUN returning to normal, confirms a diagnosis of renal disease.

Creatinine is generally accepted as being of little or no value in evaluating renal function in birds. Phosphorus elevations are usually not seen in birds with renal disease.

Reproductive system

Clinical biochemistries can tell the clinician little about the male reproductive tract; they can, however, reveal something about the activity of the female reproductive tract. Oestrogen, produced by developing follicles, induces the production of calcium-binding protein and vitellogenesis in the liver. The net result of this activity is an increase in circulating total protein, calcium, triglycerides and cholesterol. The serum may appear lipaemic.

Radiographic evidence of hepatomegaly and increased long bone density can confirm reproductive activity. It should be noted, though, that normal calcium and protein do not reflect a lack of reproductive activity. Elevated triglyceride levels can be due to dietary factors, liver disease, ovarian activity, pancreatic disease, or causes not yet understood in parrots.

Gastrointestinal system

Gastrointestinal disease typically only gives non-specific results with clinical biochemistry. Elevations of CK, AST and LDH are not uncommon, and

Downloaded From Vet-eBooks.com

are not specific to the intestinal tract. Electrolytes may give more information and should be evaluated with an understanding of the patient's appetite and thirst, hydration status, previous or current therapy and pathologic processes (i.e., gastrointestinal or renal disease) which may alter electrolyte concentrations.

- Sodium must be interpreted with the knowledge of the patient's hydration status. It may be elevated with decreased water intake or dehydration through renal disease, vomiting or diarrhoea. Sodium may also be lost through the gastrointestinal tract or the kidneys. Other causes of hyponatraemia include over-hydration, end-stage liver disease and congestive heart failure.
- Chloride is interpreted alongside sodium. It may be elevated with vomiting or regurgitation, although this is uncommon; low levels are usually associated with regurgitation or vomiting, renal disease congestive heart failure and other conditions which cause water retention.
- Potassium may be decreased with vomiting/diarrhoea and elevated with dehydration, haemolysis, tissue damage or poor sample handling.

There are many other possible causes of electrolyte disturbance, and our understanding of avian electrolyte balance is still in the very early stages.

Amylase and lipase have been proposed as useful parameters in the detection of pancreatic disease. There is still considerable discussion of the incidence of pancreatic disease and the specificity of these enzymes. Significant elevations of these enzymes, when accompanied by clinical signs of gastrointestinal dysfunction (vomiting, ileus, diarrhoea, coelomic pain) should lead the clinician to consider pancreatic disease as a differential diagnosis. However, normal levels do not preclude a diagnosis of pancreatic disease, nor do abnormal levels confirm such a diagnosis.

Blood glucose

Glucose is an essential energy source for nearly every cell in the body. Blood levels are governed by its intake, absorption, the interactions of hormones controlling carbohydrate metabolism (insulin, glucagon and somatostatin), the body's metabolism, its ability to store glucose and its excretion. As disorders of glucose metabolism involve so many organ systems, it is treated here as a separate entity.

Hyperglycaemia may be a normal physiological process, (e.g. in juvenile birds). However, elevated levels are usually related to increased production or release (e.g. stress) or failure of tissues to take it up out of the blood (diabetes mellitus). Iatrogenic hyperglycaemia occurs when corticosteroids are administered or intravenous dextrose is given. Female reproductive disease may also elevate blood glucose, but this may be an indirect result due to inflammation affecting the endocrine pancreas.

Hypoglycaemia may result from poor handling of blood samples (i.e. artefactual rather than factual), or with decreased food intake (starvation, anorexia), increased glucose usage (septicaemias, neoplasia and multi-organ failure) or decreased production (liver disease).

LIPIDS

Cholesterol and triglycerides are lipids synthesised in the body and used for a variety of metabolic processes. As they are insoluble, they are transported through the body bound to lipoproteins - high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL).

Cholesterol, synthesised in the liver, plays a major role in the production of bile acids, steroid hormones, and Vitamin D. It also provides stability and flexibility to cellular plasma membranes. Triglycerides, fatty acids attached to glycerol, are synthesised in the liver and intestinal mucosa and stored in fat. They are used as an energy source for metabolic processes.

VLDLs transport cholesterol, triglycerides and protein around the body. Once in circulation triglycerides are released to cells for energy; as the triglycerides are removed the cholesterol component increases until it is higher than the triglyceride component, forming low-density lipoprotein (LDL). This is then taken up by cells where the contents are either stored, used for cell membrane structure or converted into other products such as steroid hormones or bile acids. Excess cholesterol in the cells and blood vessels is taken up by high-density lipoproteins (HDL), transporting it back to the liver for subsequent conversion to bile acids.

Downloaded From Vet-eBooks.com

So, while cholesterol and triglycerides are necessary for normal body functions, high levels (associated with high fat diets, lack of exercise, obesity, and reproductive activity) may predispose birds to disease.

Elevations in cholesterol concentrations have been reported to be associated with a variety of disease conditions in birds, including hepatic lipidosis, atherosclerosis, hypothyroidism, bile duct obstruction, and diabetes mellitus. Decreased plasma cholesterol concentrations have been associated with some cases of hepatic disease (such as aflatoxicosis), decreased dietary fat, *E. coli* endotoxaemia and spirochaetosis.

Elevated triglycerides are frequently associated with reproductive activity in female birds. Very high levels that are persistently elevated have been associated with ovarian pathology.

Elevated VLDL, LDL and HDL concentrations, especially when combined with elevated cholesterol and triglycerides, are considered to be likely markers for atherosclerosis and hepatic lipidosis. More research needs to be done in this field. Most birds transport cholesterol mainly in the form of HDL (unlike humans, where LDL is predominant). Therefore, higher HDL values may not be associated with decreased risks of developing atherosclerosis as it is in humans – in fact, the converse may be true.

SEROLOGY

Serological tests are designed to detect antibodies (usually IgG or IgM) in serum or plasma. A number of easy-to-use commercially available tests are now available (**Fig. 7.6**). The results can be affected by a range of factors:

- Host factors:
  - Persistence of antibodies post-infection. Serial tests, weeks apart, are needed to assess what is happening in the patient.
  - Lack of antibody post-infection. If a test only assesses IgM, chronic cases with only IgG will be missed. Similarly, some animals that fail to seroconvert due to immunosuppression will test negative.
  - Changes in antibody titre. A single sample only indicates prior infection. Detection of active infections requires a measurement of rising titres (usually fourfold) over a 4–6-week period.
  - Species-specific reaction to certain testing.
- Antigen factors:
  - Prepatent period. Usually 1–2 weeks. If testing during the prepatent period, a false negative may be obtained.
  - Antigen-specific reaction to certain testing methods.
- Assay factors:
  - Laboratory error.
  - Poor test selection.
  - Lack of reagents.
  - Cross-reaction between antibodies may cause false positives.
  - Sensitivity and specificity of test.

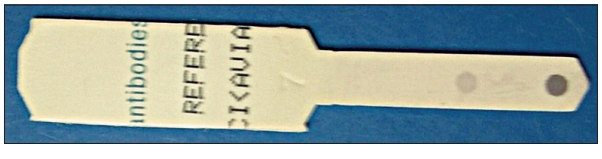


Figure 7.6 **The ImmunoComb® *Chlamydia psittaci* test is available as an in-house serological test.**

A variety of serological tests are available today (see *Table 7.1*). The selection of the best test requires a clinician to have an understanding of the principles of immunology and the interaction between host and pathogen. He/she also needs to have an understanding of the types of tests available and the advantages and disadvantages of each.

Using serology

Serology has several advantages that make it unlikely its use will ever disappear:

- PCR and other antigen-detection tests can sometimes be too accurate, detecting extremely low levels of antigen (or portions of antigen) that may not be significant. At other times the antigen may be extremely difficult to detect: it may shed only intermittently or at very low concentrations. In both these cases, serological evaluation of the bird (or birds) may better detect the presence of significant levels of antigen. Serological assays of a flock may be more

Downloaded From Vet-eBooks.com

Table 7.1 **Summary of the serological tests currently available**

TEST	TECHNIQUE	DISEASE/PATHOGEN	ADVANTAGES	DISADVANTAGES
Enzyme-linked immunosorbent assay (ELISA)	Detection of antibody–antigen complex by use of a second enzyme-conjugated antibody that produces a colour change	Adenovirus in poultry, mycobacteria, *Chlamydophila*, paramyxoviruses, aspergillosis	Simple to operate, rapid, automated	Requires species-specific second antibody
Haemagglutination inhibition	Proteins present on the surface of some viruses cause agglutination of erythrocytes. By adding antibodies against these viral proteins, this agglutination is inhibited. Therefore, if agglutination occurs, no virus-specific antibodies are present, and *vice versa*. Titres are determined by serial dilution of the sample	Circovirus, paramyxoviruses, eastern equine encephalitis, avian influenza	Secondary antibodies not required, inexpensive, good sensitivity	Serum must be treated to clear non-specific agglutination. Not all avian viruses cause agglutination
Complement fixation	Complement is needed to bind antibody and antigen. Detection of complement fixation demonstrates the presence of the antigen	Polyomavirus, psittacine herpesvirus, mycobacteria, aspergillosis, *Chlamydophila*	Quick, simple test; moderate sensitivity and specificity	Can have false positives. Does not work in all species. Usually only detects IgM
Virus neutralization	Serum is mixed with an antigen. If antigen-specific antibody is present in the serum, the antigen will be neutralised and will be incapable of causing cytopathic changes in a cell culture	Polyomavirus, psittacine herpesvirus	Very specific and sensitive. Detects both IgM and IgG	Takes up to 7 days to run; requires both cell culture and virus propagation. Non-specific substances in the serum can give a positive result
Immunodiffusion assay	Antibody and antigen placed in agar gel diffuse towards each other. If an antibody–antigen complex forms, a precipitate develops that is visible as a white line	Adenovirus in poultry, reovirus in psittacines	Simple and cheap. Secondary antibodies not required. Moderate specificity	Low sensitivity. Difficult to quantify
Immunofluorescent antibody	Multiple dilutions of serum are incubated with cells infected with antigen fixed to plastic. A fluorescent-labelled second antibody is added. Positive results can be viewed with a fluorescent microscope	Avian influenza, *Chlamydophila*	Quick and simple. Moderate sensitivity and specificity	Requires species-specific second antibody. Requires a fluorescent microscope

sensitive in detecting a pathogen than more direct antigen-detection tests.

- Its relatively low invasiness. Direct testing for an antigen that is only intermittently shed (if at all) often requires the collection of a tissue sample, either by autopsy or biopsy. Serology, on the other hand, simply requires a blood sample. This is obviously much less invasive.
- Serological tests, especially when performed in large numbers, can be significantly cheaper than more direct pathogen-detection tests.

Although at first glance these appear to be almost overwhelming advantages, other factors play key roles in the selection of an appropriate serological test. These factors can be categorised into three groups, discussed below.

The characteristics of the test

The selection of the test is of paramount importance. Is this test appropriate for this species? Has it been thoroughly validated for this disease? Antibodies are not necessarily consistent across

Downloaded From Vet-eBooks.com

species; a test that works well in poultry may not work well, or at all, in parrots.

The sensitivity and specificity of a test will often determine its usefulness. Sensitivity is a measure of a test's ability to accurately detect antibodies in an infected bird. A highly sensitive test will hopefully detect most of the antibody-positive birds in a population. As such, it is useful as a screening test during the early days of a disease outbreak, where it is important to identify quickly potentially infected birds. Once these birds have been removed from a population, it becomes more important to be sure that the remaining birds that have tested negative are, in fact, not infected. At this time a highly specific test is desirable. Specificity is the proportion of truly non-infected birds that test negative. A clinician using a serological test should therefore be aware of the published sensitivity and specificity of a test.

Another test characteristic to be considered is its complexity to perform. The more complex a test, the more susceptible it is to operator error leading to erroneous results. Errors can occur in the collection, storing and transport of the sample to the laboratory, and then again in carrying out the test; equipment can be faulty, the operator may be inexperienced or reagents may be out of date. There may also be error in the interpretation of the results, or even dissension about the significance of the results.

In some cases a test requires specific reagents, which may not be readily available (e.g. the haemagglutination inhibition test for PBFDV requires specially prepared galah erythrocytes, readily available in Australia, but harder to obtain elsewhere. However, goose erythrocytes can sometimes be used.) This limits its availability and usefulness elsewhere in the world.

The characteristics of the antigen and the disease

Some antigens/diseases are not readily detectable by some tests due to the behaviour of that antigen. Some viruses, for example, do not agglutinate erythrocytes, making haemagglutination inhibition tests unsuitable.

Some pathogens, such as viruses like the influenza A virus, undergo antigenic variance wherein the surface proteins of the virus change and prevent immediate detection. Other pathogens, such as *Mycobacterium* spp. and *Aspergillus* spp., seem not to provoke an antibody response in some patients for, as yet, undetermined reasons. In these situations, false-negative test results may occur.

From an epidemiological point of view, there will always be false-positive and false-negative results for any test. The clinician must therefore interpret results with care; if a disease is of a low incidence in a species or in a geographical area, is a positive result truly significant or even accurate? And *vice versa*? As with any laboratory test, the results must be interpreted as a part of the diagnostic test, not as a sole entity.

Host factors

Some clinicians seem to fall into the trap of forgetting that there is a patient attached to a laboratory test result. How the host is responding to the challenge of a pathogen has a major impact on the interpretation of a serological test (or any other test). The interaction between the host and the pathogen must be understood by the clinician in order to accurately assess the patient's status.

In the initial stages of infection (the prepatent period) there will be a delay while the immune system firstly recognises the presence of an antigen and then mounts a response to it (usually in the form of IgM). In this prepatent period (usually 1–2 weeks), a serological assay that only detects IgG will give a false-negative result. On the other hand, direct antigen-detection tests may have a prepatent period of only 1–2 days, and are therefore more reliable in detecting early infection.

With appropriate treatment or an effective immune defence (or both) the host will inactivate/kill the pathogen and then clear it from the body. During this process there is a rising antibody titre. After the pathogen has been cleared, these titres should start to decline. The rate of this decline is dependent on many factors (e.g. the strength of the immune system, the persistence of the antigen, the degree of response to a specific pathogen). A single-point serological assay is unable to distinguish between rising and declining antibody titres; serial tests, 2–3 weeks apart, are needed to assess what is happening in the patient. For example,

Downloaded From Vet-eBooks.com

polyomavirus infection in a bird may result in detectable antibody titres for the life of the bird, even though the bird cleared the virus from its body after a few weeks or months. So while a single serological test may indirectly detect that a pathogen was/is present in the patient, it cannot (by itself) detect the ongoing presence of disease.

The serological detection of pathogens that are ubiquitous in the environment is fraught with error. Most birds, for example, are exposed to *Aspergillus* spp. at some time; relatively few develop Aspergillosis. The detection of antibodies to *Aspergillus* does not, therefore, constitute a diagnosis of Aspergillosis; rather, it indicates the patient has been exposed to the pathogen and has mounted an immune response to it.

Despite the presence of a pathogen actively causing disease, some birds fail to produce antibodies. This immunosuppression can be due to a number of factors. Concurrent disease frequently causes immunosuppression: viruses such as PBFD, polyomavirus, pox and herpes, bacterial and parasitic infections and aflatoxicosis have all been implicated in immunosuppressed patients. Certain drugs such as tetracycline, tylosin and gentamicin are known to decrease antibody production. Environmental stress (inadequate temperatures, humidity, noise, poor nutrition) is known to suppress both the bursa and the thymus, possibly through corticosteroid production by the adrenal gland. Immunosuppressed birds, for whatever reason, may give a false-negative result.

A final consideration is that antibodies produced in response to a particular pathogen may be difficult to distinguish from antibodies produced against another pathogen. Antibodies produced by vaccination may be indistinguishable from those produced by natural infection. In either case, false-positive results can be produced.

Scenarios involving the use of serology

Disease outbreaks

Serology is often employed in the face of an outbreak of an infectious disease. Serology has the advantages of being relatively simple (most labs can perform it), quick and cost-effective, particularly when compared with other (more direct) tests such as DNA/PCR and histopathology. Large numbers of birds can be tested quickly, giving an indication of the incidence of the disease in a given population. In the early stages of an outbreak, high sensitivity is needed; all infected (positive result) birds need to be identified as soon as possible. Sensitivity can be increased by using known high-sensitivity tests, increasing the sample size of the population being tested and parallel testing, using several different tests to increase the likelihood of an infected bird being detected.

Once the incidence of a disease has been determined, steps can be taken to control it. This will usually require the removal of birds that have tested positive and further testing of those that have tested negative. This introduces the problem of false-negative results (seronegative birds that are latent carriers of a disease). At this time highly specific tests are needed, and may require the employment of a different serological test or tests. As the incidence of a disease approaches zero, the detection of these latent carriers becomes even more difficult, and complete eradication may not be possible without complete culling.

Specific pathogen-free flocks

One of the cornerstones of successful farming is maintaining a disease-free flock. This is no different with birds, whether it is an intensive poultry flock, a zoological collection or an avicultural facility. The development of a specific pathogen-free (SPF) flock requires the testing of all members of that flock, preferably before they join it.

Veterinarians advising clients on a SPF protocol encounter similar problems to those faced when dealing with an outbreak of disease. No test can hope to be 100% sensitive and 100% specific. Therefore, parallel testing (several different tests, not necessarily all serological) and serial testing (repeated testing over a period of time) may help to detect those birds that are latent carriers and test negative the first time. While this can add considerably to the cost of new birds, it must be weighed up against the costs of remedying an outbreak of disease if it escapes detection and is introduced.

The individual bird

While serological tests are extremely useful in screening flocks, they often lack the sensitivity

Downloaded From Vet-eBooks.com

and specificity required for screening an individual bird. While relatively inexpensive, they may lack the accuracy of a more direct antigen-detection test. However, these more direct tests only detect the presence of a pathogen, not necessarily the presence of a disease. Combining the two tests together can lead to a better screening of an individual. By looking for the pathogen, and the body's immune response to it, a better understanding of the bird's status can be achieved. When combined with ancillary tests such as haematology and biochemistry (which can reflect the effect the pathogen is having on the bird), a more complete picture of the host–pathogen interaction becomes apparent.

PCR TESTING

PCR is the amplification of a specific fragment of nucleic acid (DNA) from minute quantities of source DNA material. It is rapidly becoming one of the most widely used techniques in molecular biology because it is quick, inexpensive and simple.

Samples are first heated, which causes the DNA double helix strand to separate into single strands. Primers are then added to bind to these single strands (annealing) and the sample is allowed to cool. Each time this heating and cooling cycle is repeated, the amount of DNA present in the sample effectively doubles (amplification). Once the process has been completed the sample now contains numerous copies of specific DNA sequences. The reaction products are separated by gel electrophoresis. Depending on the quantity produced and the size of the amplified fragment, the reaction products can be visualised directly by staining with ethidium bromide or a silver-staining protocol or by means of radioisotopes and autoradiography.

The specificity of PCR depends on the primers used. As a general rule most PCR assays are highly specific, but a quantitative PCR assay may have variable degrees of sensitivity and may fail to detect very low quantities of DNA. In an attempt to increase the sensitivity of PCR, 'real-time PCR' has been developed. This assay allows the laboratory to view the increase in the amount of DNA as it is amplified by characterizing (through the use of fluorescent markers) the point in time during cycling when amplification of the target DNA is first detected, rather than the amount of target produced at the end of the assay. The higher the starting copy number of the nucleic acid target, the sooner a significant increase in fluorescence is observed.

While PCR is an accurate test for the detection of target DNA, results must be interpreted with caution. Firstly, the sensitivity and specificity of the test means that false positives can occur easily if the sample is contaminated during collection, processing or testing. Secondly, a problem sometimes arises with an over-diagnosis of a disease solely through the detection of a pathogen, without necessarily diagnosing the presence of a disease process. Clinicians must therefore exercise caution in the use of PCR, using it as a tool for obtaining a diagnosis, rather than as a diagnosis in its own right.

CULTURES

Over the last several decades there has been a quantum shift in thought about the significance of bacterial and fungal cultures taken from birds. The concept of bacterial and fungal infections being primary diseases has largely been replaced by a better understanding of how pathogens can take advantage of an immunosuppressed, malnourished patient. In other words, clinicians, instead of asking 'What infection does this bird have?' are now asking 'Why does this bird have an infection?' This change in thought has led to better diagnoses, better treatments and better survival rates.

As part of this evolving thought process, clinicians have reviewed many of the tests that were commonly used to diagnose disease, and are questioning the correct use and interpretation of tests such as Gram stains and cultures. It is not appropriate to base a diagnosis of health or disease in a bird solely on a Gram stain and/or culture. Questions that need to be asked when selecting this test include:

- Does the clinical database (history, physical examination, haematology and biochemistries) collected on this bird suggest a bacterial or fungal infection?

Downloaded From Vet-eBooks.com

- Is the site the sample has been collected from likely to give significant information about the patient's problem?
- What do the culture or stain results mean?

As veterinary skills and diagnostic equipment have improved, clinicians are now recognizing that many diseases in birds are not caused by infectious agents. Nutritional, metabolic, endocrine and traumatic conditions, amongst others, can all give rise to the classic 'sick bird look'. Neither bacterial cultures nor antibiotics are likely to assist in the diagnosis and treatment of these patients. A sound clinical database provides the clinician with the best information from which to direct further diagnostic and therapeutic steps.

While a Gram stain or culture from the faeces of a bird with gastroenteritis is likely to provide valuable information, it does not constitute a diagnosis in its own right. Nor will it assist in the diagnosis of a bird with a respiratory problem or a traumatic injury, or even an assessment on a bird that is otherwise normal and healthy. But there still persists a school of thought that insists a faecal Gram stain should be performed on all birds, regardless of why they have been presented. Cultures and Gram stains are useful diagnostic tools, but the clinician must evaluate if their use is justified for each patient (**Fig. 7.7**).

Normal faecal bacteria cultured from healthy birds include gram-positive bacilli (*Lactobacillus* spp., *Bacillus* spp., *Corynebacterium* spp., and *Streptomyces*) and gram-positive cocci (*Staphylococcus epidermidis*, *Streptococcus* spp., *Aerococcus* spp. and *Micrococcus* spp.). Older birds tend to have more *Corynebacterium* and less *Lactobacillus* than juvenile birds. *Escherichia coli* is commonly recovered from cockatoos, but less commonly in other species. Other gram-negative bacteria occasionally found in clinically normal birds include *Enterobacter*, *Klebsiella*, *Citrobacter*, *Pasteurella* and *Moraxella* spp. While some *Pseudomonas* spp. have been recovered from healthy birds, *P. aeruginosa* is rarely recovered from healthy birds. Isolation of *Proteus*, *Salmonella*, *Pseudomonas*, *Klebsiella*, *Listeria*, *Erysipelothrix* and haemolytic *Staphylococcus aureus* is clinically significant in sick birds.

Cultures from other sites (eyes, ears, choana) usually show similar results.

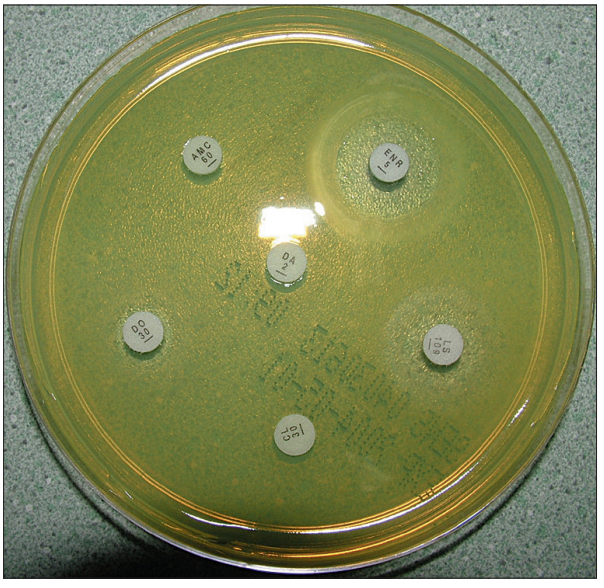


Figure 7.7 **A culture plate used to investigate antibiotic sensitivity. It should be remembered that although this test indicates the most appropriate antibiotic to use, it does *not* answer the clinician's primary question, 'Why does this bird have an infection?'**

A heavy growth of a pure culture of any bacteria can reflect bacterial overgrowth and warrants further investigation.

Candida albicans is often detected by Gram stain in the droppings of birds eating yeast-containing feedstuffs (e.g. bread, biscuits). Unless the yeast are numerous and budding, or can be cultured in heavy numbers, they have little or no clinical significance. Development of pseudohyphae, seen on Gram staining, is significant and may indicate invasion of the intestinal mucosa by the yeast.

Culture of *Aspergillus* spp. or *Cryptococcus* spp. from a healthy bird is likely to reflect environmental contamination rather than infection.

CYTOLOGY

Cytological samples (**Figs 7.8, 7.9**) can be obtained from:

- Fine needle aspirates of masses or organs.
- Coeliocentesis.
- Sinus aspirates.

Downloaded From Vet-eBooks.com

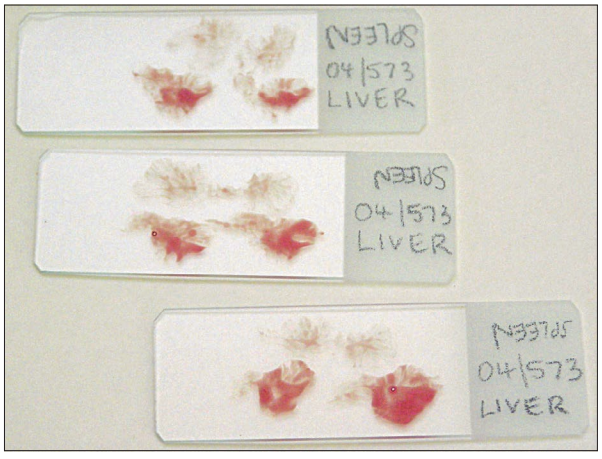


Figure 7.8 **Smears of liver and spleen tissue will be air-dried and then fixed in methanol ahead of staining. Note: It is always advisable to prepare more than one smear so that different stains can be applied (Photo courtesy S Raidal).**

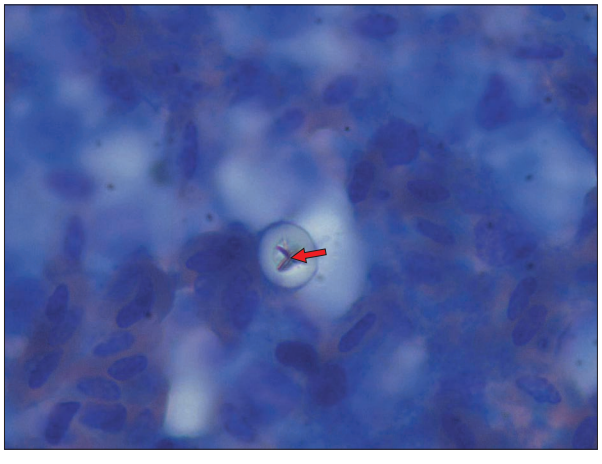


Figure 7.9 **Stained liver tissue smear demonstrating how artefacts can be confused with pathology. In this case a talc crystal (identified) is from a disposable glove (Diff Quik, 100×) (Photo courtesy S Echols).**

- Crop washes.
- Arthrocentesis.
- Tracheal and air sac washes.
- Contact smears from skin and internal organs.
- Scrapings from the palpebral conjunctiva, cornea, oral cavity or tissues that normally yield poorly cellular samples.

Assessment of a cytological sample requires the clinician to firstly identify both the cell types and the cellular response that is occurring in the sample.

Classification of cell types

- Haemic cells are those cells found in the blood and the haematopoietic tissues.
- Epithelial cells typically exfoliate easily and are found in clusters or sheets. They vary in shape depending upon their origin and can be oval, cuboidal, columnar or polygonal. They typically have an abundant cytoplasm, small round to oval nuclei and distinct cytoplasmic margins. Cells from secretory epithelium may contain cytoplasmic granules or vacuoles.
- Mesenchymal cells tend to exfoliate poorly and normally occur as single cells. They have indistinct cytoplasmic margins. Fibroblasts are the most frequently encountered cell of this group.
- Nervous tissue cells are rare in cytological specimens. They may be seen as deeply basophilic, stellate cells with cytoplasmic projections.

Classification of cellular responses

The goal of cytology is to classify the cell response into one of the basic cytodiagnostic groups. These groups include inflammation, tissue hyperplasia, benign neoplasia, malignant neoplasia and normal cellularity.

Inflammation

A diagnosis of inflammation is made when an increased number of inflammatory cells is detected in the sample. The inflammatory cells of birds are heterophils, lymphocytes, plasma cells and macrophages. Eosinophils may be included in the list of inflammatory cells; however, eosinophilic inflammation is either extremely rare or difficult to detect based on routine cytological methods. Heterophils and eosinophils may be difficult to differentiate based on routine cytological methods. Avian inflammatory responses are classified as heterophilic, mixed cell or macrophagic:

- Heterophilic inflammation is present when heterophils make up >70% of the inflammatory cells and indicates an acute inflammatory response.

Downloaded From Vet-eBooks.com

Degenerate heterophils indicate a toxic microenvironment, usually caused by microbial toxins. If bacterial phagocytosis can be demonstrated, the cytodiagnosis of septic heterophilic inflammation can be made. If only extracellular bacteria are found, they may represent either normal flora or contaminants of the sample.

- Mixed-cell inflammation is represented by the presence of heterophils and mononuclear leucocytes. Heterophils usually make up at least 50% of the inflammatory cells. Mixed-cell inflammation usually represents an established, active inflammation.
- Macrophagic inflammation is indicated by the predominance of macrophages (>50%) in the inflammatory response. This type of inflammation does not necessarily imply chronicity, but may be suggestive of a number of aetiologies (e.g. intracellular pathogens). Macrophagic inflammation is common to certain avian diseases including tuberculosis, chlamydiosis, foreign body reaction, mycotic infections and cutaneous xanthomatosis. Multinucleate giant cell formation is often associated with macrophagic inflammation. Giant cells can appear within hours of the onset of some inflammatory responses and, unlike in mammals, their presence does not imply chronic inflammation.

Tissue hyperplasia or benign neoplasia

Tissue hyperplasia resulting from cellular injury or chronic stimulation is difficult to differentiate from benign neoplasia on cytology alone. Cells from hyperplastic tissue appear mature and do not exhibit much pleomorphism.

Malignant neoplasia

Malignant neoplastic cells show varying degrees of nuclear pleomorphism. There is an increase in nuclear size, which is reflected by an increased nucleus to cytoplasm ratio. Multinucleation may be present. The nuclei often have coarse, hyperchromatic chromatin and large or multiple (more than five) nucleoli. The cytoplasm shows increased basophilia, decreased volume and variability in the staining and it may have abnormal vacuolation or inclusions. There is often variation in cell margins. The cell types seen can be grouped into four basic classifications:

- Carcinomas: malignancies of the epithelial cells. Adenocarcinomas are frequently seen in birds. Cytological evidence of adenocarcinomas includes epithelial cells that tend to form giant cells, have cytoplasmic secretory vacuoles and tend to occur in aggregates.
- Sarcomas: malignancies of mesenchymal cells. Fibrosarcomas are the most frequently encountered sarcomas of birds. The cells tend to exfoliate poorly. They are abnormal appearing fibroblasts, which are spindle-shaped cells that typically exfoliate as single cells. Abnormal fibroblasts show increased cellular size and nuclear:cellular ratios and nuclear and cellular pleomorphism. Other mesenchymal cell neoplasms such as chondromas, chondrosarcomas and osteogenic sarcomas may produce a heavy eosinophilic background material that can be seen on the microscopic sample.
- Discrete or round cell neoplasms: the only common neoplasm of this type is lymphoid neoplasia. Cellular features include a marked increase in the number of lymphoblasts, nuclear and cellular pleomorphism, an increase in cytoplasmic basophilia and mitotic figures and abnormal or multiple nucleoli.
- Poorly differentiated neoplasms produce cells having features of malignancy, but the cells are difficult to classify as carcinomas or sarcomas.

Cytology of commonly sampled fluids and tissues

Coelomic fluids

Coelomic fluids can be classified as transudates, modified transudates, exudates, haemorrhagic or malignant effusions.

Transudates: Transudates are odourless, transparent fluids characterised by total cell count <1 × 10^9/l (<1,000 cells/mm^3), a specific gravity <1.020 and a total protein <30 g/l. Transudates do not clot. The cell types are primarily macrophages and occasional mes-

Downloaded From Vet-eBooks.com

othelial cells. Transudates occur as a result of oncotic pressure changes or other circulatory disturbances. The common causes of coelomic transudates in birds include hepatic cirrhosis, cardiac insufficiency and hypoproteinaemia.

Modified transudates: Modified transudates have total cell counts of 1–5 × 10^9/l (1,000–5,000 cells/mm^3). The mononuclear leucocytes predominate, with occasional mesothelial cells and rare heterophils. The mesothelial cells tend to be round or oval with increased cytoplasmic basophilia. Multinucleation, cytoplasmic vacuolation and mitotic activity are often associated with reactive mesothelial cells. Care should be taken not to mistake these cells for malignant neoplasia. Transudates and modified transudates are commonly found in the coelomic cavity of mynah birds suffering from haemochromatosis.

Exudates: Exudative effusions are characterised by high cell counts, >5 × 10^9/l (>5,000 cells/mm^3), a specific gravity >1.020 and protein content >30 g/l. The majority of cells are inflammatory cells. Acute exudative effusions demonstrate primarily a heterophilic inflammatory response; however, macrophages quickly move into the fluid, creating a mixed-cell inflammatory response within a few hours of onset. Lymphocyte and plasma cells are often seen in long-standing exudative effusions. Lesions often associated with exudates include septic peritonitis, egg-related peritonitis and coelomic malignancies (**Fig. 7.10**).

Haemorrhagic effusions: Haemorrhagic effusions are identified by the presence of erythrocytic phagocytosis. Thrombocytes usually disappear rapidly in haemorrhagic effusions. Iron pigment or haemosiderin crystals found in macrophages are also indicators of haemorrhagic effusions. Iron pigment appears grey to blue–black using Wright's stains. Haemosiderin appears as diamond-shaped, golden crystals within the macrophage cytoplasm.

Malignant effusions: Malignant effusions have features of either exudative or haemorrhagic effusions, but contain cells compatible with malignant neoplasia. Cystadenocarcinomas of the ovary are a common cause of malignant effusions in older females. These cells often form cellular aggregates of balls or rosettes and have cytoplasmic secretory vacuolation.

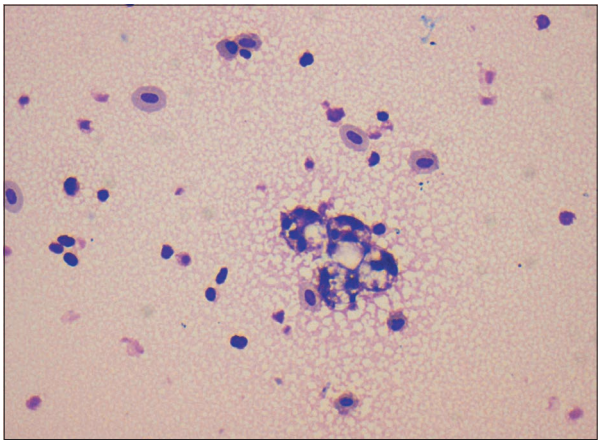


Figure 7.10 **Stained sample of egg yolk from a budgerigar reveals coelomitis (Diff Quik, 40×) (Photo courtesy S Echols).**

Urate peritonitis is a rare effusion that can occur when urinary fluids leak into the coelomic cavity. The acute lesion is poorly cellular, but it contains a marked number of sodium and potassium urate crystals. Urate crystals are spherical and have a spoke-wheel appearance. If the bird survives long enough, inflammatory cells will migrate into the fluid.

Cytology of the alimentary tract

Oral cavity

The oral cavity is easily sampled when lesions are visible. Normal cytology of the oral cavity shows occasional squamous epithelial cells, varying amounts of background debris and extracellular bacteria represented by a variety of morphological types. The differential diagnoses for common oral lesions include septic stomatitis, candidiasis, Trichomoniasis and squamous cell hyperplasia. *Alysiella filiformis*, a gram-negative small paired coccobacillus that forms ribbon-like chains, is normally associated with squamous epithelial cells.

- Smears made from a bacterial abscess reveal either a heterophilic or mixed-cell inflammation with bacterial phagocytosis.

Downloaded From Vet-eBooks.com

- Candidiasis is evidenced by large numbers of typical organisms. *Candida* can be a normal inhabitant of the upper alimentary tract, so low numbers of organisms do not produce inflammation. An inflammatory response often occurs when the infection has involved the mucosa. The presence of hyphae formation suggests a potential systemic invasion by the yeast.
- Trichomoniasis is best diagnosed by observing the movement of the piriform flagellate protozoa in a wet mount preparation. It is important to recognise these organisms in a stained cytological sample if wet mount preparations are not part of the routine examination or if Trichomoniasis is not suspected. Trichomonads appear as basophilic, piriform cells with flagella on Wright's stained smears (**Fig. 7.11**). The cell nucleus usually stains more eosinophilic than most cell nuclei. An eosinophilic axostyle can often be seen as a straight line running from the nucleus to the opposite pole of the cell. An inflammatory response is usually found associated with Trichomoniasis.
- Squamous cell hyperplasia and metaplasia lesions may grossly resemble bacterial, yeast or protozoal infections, but the cytological appearance is very different (**Fig. 7.12**). Normally, squamous epithelial cells exfoliate as single cells or small sheets. With squamous hyperplasia associated with vitamin A deficiency, smears with large numbers of cornified squamous epithelial cells that exfoliate in large sheets or aggregates are common. The cytology resembles that of the vaginal cytology of a dog in oestrus. Inflammatory cells are not seen unless there are secondary infections with bacteria, yeast or protozoa.

Oesophagus and crop

Cytological evaluation of the oesophagus and crop is indicated in birds with clinical signs of regurgitation, vomiting, delayed crop emptying or other suspected oesophageal and crop disorders. Normal cytology reveals occasional squamous epithelial cells and a variable amount of background debris and extracellular bacteria. An occasional yeast is accepted as normal. Also, some foods contain yeast as a source of supplemental B vitamins, so this source of large numbers of non-budding yeast must be ruled out. The presence of many bacteria represented by one morphological type, even without inflammatory cells, may indicate a problem. This may be a common finding in peracute ingluvitis. A pH >7 is also suggestive of acute or peracute ingluvitis. *Capillaria* ova may be detected in cytological samples from the oesophagus or crop of some birds with Capillariasis. These ova are double operculated and may not stain.

Cloaca

Cloacal cytology is indicated whenever a disorder of the lower intestinal tract, reproductive tract, urinary



Figure 7.11 **Staining reveals a *Trichomonas* spp. within a tissue smear from a bird with bacterial pharyngitis (Photo courtesy R Schmidt).**

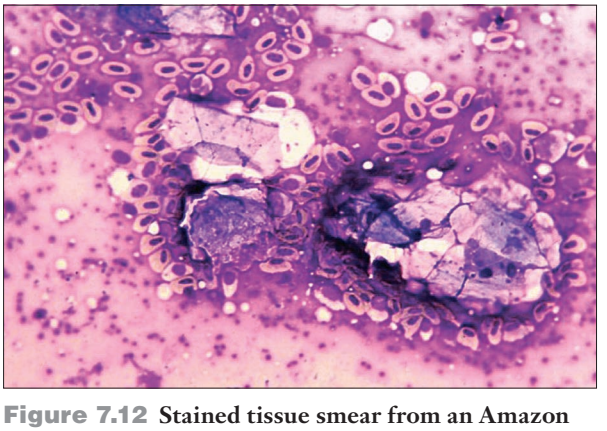


Figure 7.12 **Stained tissue smear from an Amazon parrot reveals squamous metaplasia with secondary bacterial invasion due to vitamin A deficiency (Photo courtesy R Schmidt).**

Downloaded From Vet-eBooks.com

tract or cloaca is suspected. Normal cytology reveals a few non-cornified epithelial cells, extracellular bacteria, background debris and urate crystals. Abnormal findings would include the presence of inflammatory cells, large numbers of yeast or a uniform population of bacteria.

Cytology of the respiratory tract

Nasal and infraorbital sinuses

Normal cytology reveals occasional non-cornified squamous epithelial cells and low numbers of extracellular bacteria with little background debris. Evidence for periorbital sinusitis is provided by the presence of inflammatory cells in the aspirate. Lesions with a bacterial aetiology are indicated by a septic, heterophilic or mixed-cell inflammation. Mycotic lesions often reveal either a mixed-cell or macrophagic inflammation, with the presence of fungal elements such as yeast, hyphae or spores (**Fig. 7.13**). Chlamydial sinusitis often reveals a mixed-cell or macrophagic inflammation. Chlamydial inclusions appear as small, blue-to-purple spherules, often in dense clusters, within the cytoplasm of macrophages when stained with Wright's stain.

Trachea

Normal cytology from a tracheal wash consists of a few ciliated respiratory epithelial cells and goblet cells.

Septic tracheobronchitis reveals inflammatory cells showing bacterial phagocytosis (**Fig. 7.14**). Degenerative respiratory epithelial cells show a loss of cilia, cytoplasmic vacuolation and karyolysis. There is often increased mucin formation, which causes an increased thickness to the non-cellular background. Mycotic lesions involving the trachea, syrinx and bronchi may reveal fungal elements on the tracheal wash. Aspergillosis is characterised by thick, septate hyphae that branch at 45 degree angles. Mycotic lesions usually reveal a mixed-cell or macrophagic inflammation (**Fig. 7.15**).

Air sacs

Normal air sac samples are poorly cellular with the presence of a few non-cornified epithelial cells. Bacterial infections show the typical septic

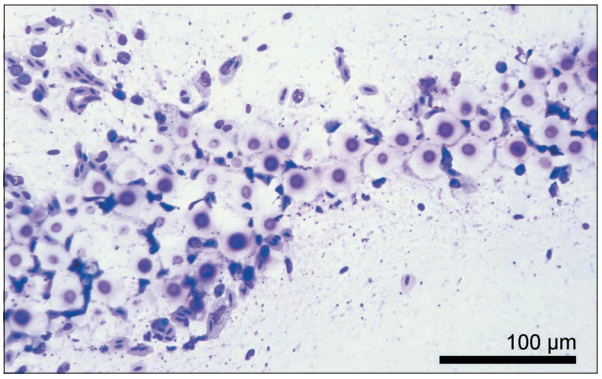


Figure 7.13 **Fungal spores revealed in a stained sample from a bird who has nasal cryptococcosis (Photo courtesy S Raidal).**

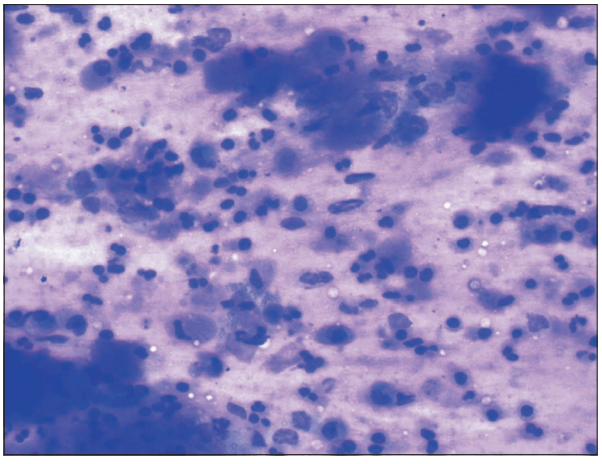


Figure 7.14 **Tissue smear from an Umbrella cockatoo once stained reveals evidence of purulent tracheitis (Diff Quik, 40×) (Photo courtesy S Echols).**

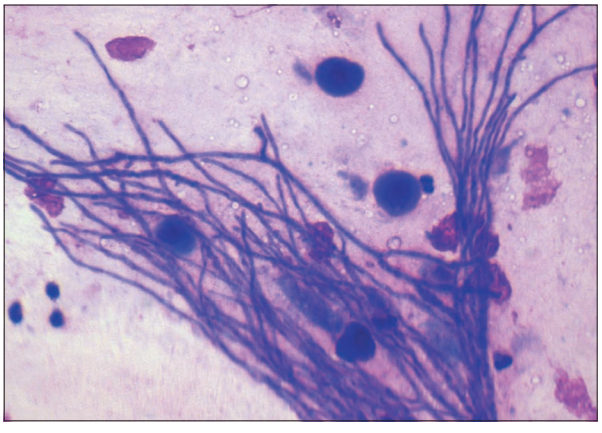


Figure 7.15 **Fungal hyphae clearly visible after staining in a sample from an Amazon parrot with fungal tracheitis (Diff Quik, 40×) (Photo courtesy S Echols).**

Downloaded From Vet-eBooks.com

inflammatory patterns. Chlamydial and mycotic lesions demonstrate mixed-cell or macrophagic inflammation with the presence of chlamydial inclusions or fungal elements, respectively. Neoplastic lesions of the respiratory tract of birds are rare.

Skin

Normal skin samples typically contain squamous epithelial cells, debris and extracellular bacteria.

- Bacterial infections involving the skin are usually associated with a heterophilic or mixed-cell inflammation (**Fig. 7.16**) (bacterial phagocytosis must be demonstrated to detect a septic inflammatory lesion). Care must be taken not to confuse basophilic-staining powder down for bacteria or yeast.
- Cutaneous xanthomatosis is a unique condition of birds caused by an excessive accumulation of lipids in the skin. It is a macrophagic inflammatory response, with multinucleated giant cells and cholesterol crystals observed on the cytological specimen. (Cholesterol crystals appear as angular, translucent crystals that vary in size and shape.)
- Subcutaneous lipomas produce a cytological specimen that appears 'greasy' on the unstained slide. The cytology reveals numerous lipocytes, which vary in size. Fat droplets usually partially dissolve in the alcohol-based stains, but are easily seen in water-soluble stains such as new methylene blue. Special fat stains such as Sudan IV can be used to demonstrate the fat droplets.
- Feather cyst cytology may reveal RBCs and erythrocytosis in early lesions. More chronic lesions develop a caseous exudate with a mixed-cell inflammation.
- Cutaneous and subcutaneous malignancies are rare in birds. Lymphoid neoplasia produces a highly cellular sample of immature lymphocytes. Cutaneous melanosarcomas have also been found in birds.
- Avian poxvirus lesions reveal clusters of squamous epithelial cells that contain large eosinophilic cytoplasmic vacuoles. The large cytoplasmic vacuoles found in the affected squamous cell push the cell nucleus to the cell margin.

Cornea and conjunctiva

Normal conjunctival scrapings provide poorly cellular samples with little background material. The cells may contain intracytoplasmic pigment granules. Normal cytology of the cornea is also poorly cellular and consists of occasional non-cornified squamous epithelial cells.

Inflammatory lesions involving the cornea and conjunctiva reveal inflammatory cells (**Fig. 7.17**) and increased numbers of exfoliated epithelial cells. Chronic lesions may also reveal the presence of cornified squamous epithelial cells that are not normally found in the conjunctiva or cornea.

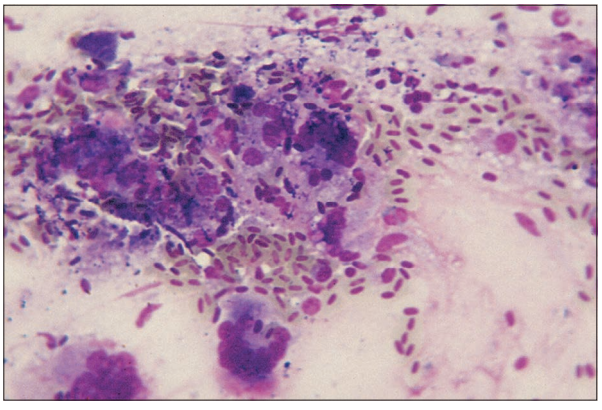


Figure 7.16 **Stained tissue smear shows presence of erythrocytes, leucocytes and epithelial cells indicative of pododermatitis (Photo courtesy R Schmidt).**

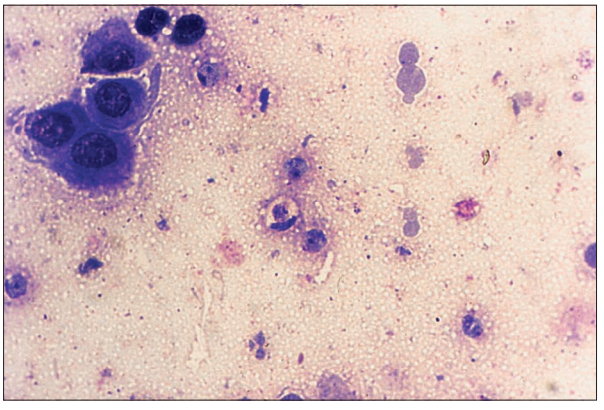


Figure 7.17 **Stained conjunctival smear reveals epithelial cells, leucocytes and encapsulated yeast cells (*Cryptococcus* spp.) (identified) in the centre of the image (Photo courtesy R Schmidt).**

Downloaded From Vet-eBooks.com

Synovial fluid

Normal synovial fluid is poorly cellular. The cells are mononuclear cells, representing either synovial lining cells or mononuclear leucocytes. The background of normal synovial fluid consists of a heavy, granular, eosinophilic substance representing the mucin in the fluid.

An increase in the inflammatory cells and change in the colour, clarity and viscosity of the fluid is indicative of inflammatory joint lesions. There may be a decrease in the granular eosinophilic background material, suggesting a decrease in mucin content. Erosion of the articular cartilage may result in the presence of multinucleated osteoclasts in the synovial fluid. Spindle-shaped fibroblasts suggest erosion into the fibrous layer of the articular capsule. Septic joint lesions may demonstrate bacterial phagocytosis by leucocytes. Traumatic arthritis also results in increased numbers of inflammatory cells.

Articular gout produces a cream–yellow-coloured deposit in affected joints. Cytology reveals numerous, needle-shaped crystals. Inflammatory cells are often present and the mucin content is often reduced, as reflected in the reduction in the amount of eosinophilic granular background.

Cytology of internal organs

Liver

Cytological samples are usually highly cellular, with a predominance of hepatocytes, erythrocytes and free nuclei. Hepatocytes are large epithelial cells that occur in sheets or clusters or as single cells. Normal haematopoiesis is occasionally found because the liver is a common location for ectopic haematopoiesis. Macrophages containing haemosiderin are occasionally seen.

Inflammatory lesions of the liver reveal numerous mature heterophils and an increase in the number of macrophages and plasma cells (**Figs 7.18, 7.19**). It is important not to confuse normal ectopic granulopoiesis with heterophilic inflammation. (If developing stages of the heterophils can be found, the cytology is representative of granulocytopoiesis. If the heterophils are mature cells, the cytology indicates inflammation.)

Avian tuberculosis produces a macrophagic inflammatory response in the liver. The cytology reveals numerous macrophages and multinucleated giant cells (**Fig. 7.20**). The background of the smear and the macrophages may contain numerous bacterial rods that do not stain the waxy cell wall produced by mycobacteria.

Chlamydiosis often results in a mixed-cell or macrophagic inflammation in the liver, with a marked increase in the number of plasma cells. Small, blue–purple, intracytoplasmic inclusions suggestive of chlamydial elementary and initial bodies may be seen in macrophages.

Hepatic lipidosis reveals enlarged hepatocytes that contain round, cytoplasmic vacuoles (**Figs 7.21a** and **b, 7.22**).

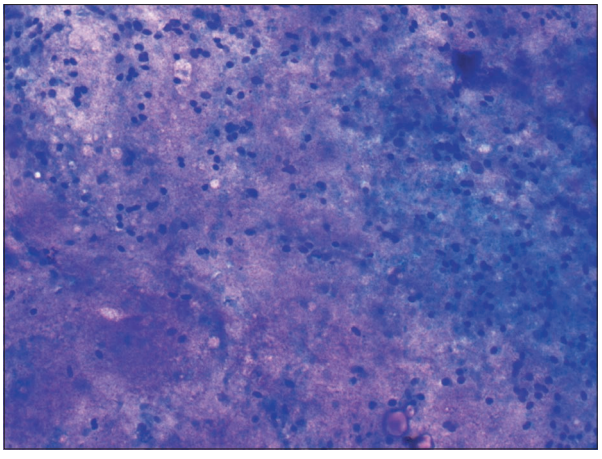


Figure 7.18 **Stained liver tissue smear from an African grey parrot indicates hepatic necrosis (Diff Quik, 20×) (Photo courtesy S Echols).**

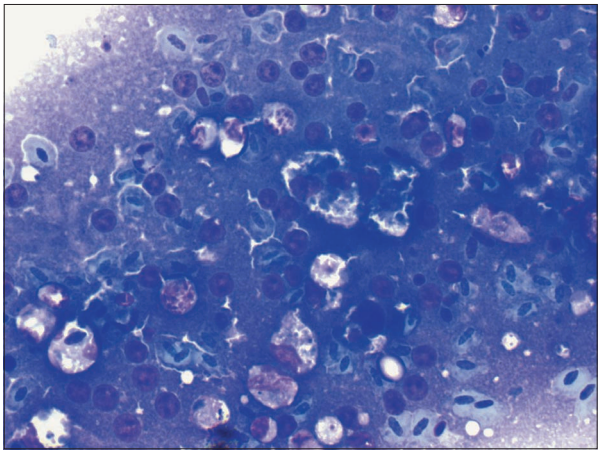


Figure 7.19 **Stained liver tissue smear from an African grey parrot indicates granulomatous hepatitis (Diff Quik, 400×) (Photo courtesy S Echols).**

Downloaded From Vet-eBooks.com

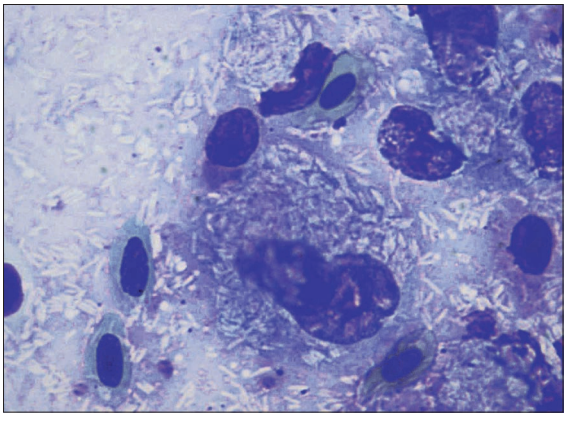
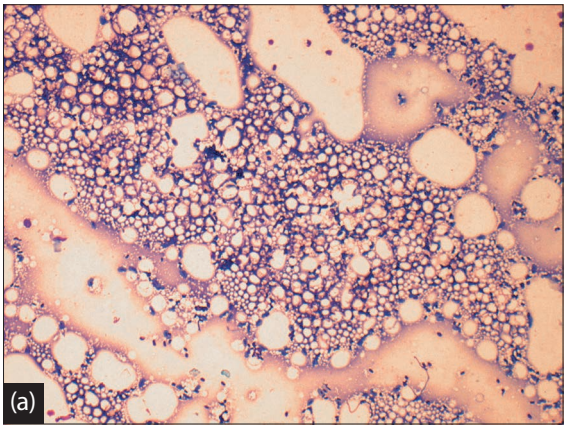


Figure 7.20 Stained liver tissue smear from a bronze-winged pionus indicates granulomatous hepatitis due to a mycobacterial infection (Diff Quik, 100×) (Photo courtesy S Echols).



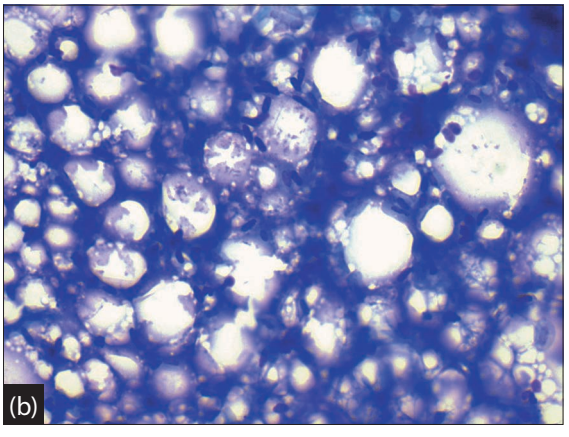


Figure 7.21 Stained liver tissue smear from a cockatiel indicate hepatic lipidosis (a: Diff Quik, 10×; b: Diff Quik, 40×) (Photos courtesy S Echols). Note the enlarged hepatocytes that contain round, cytoplasmic vacuoles.

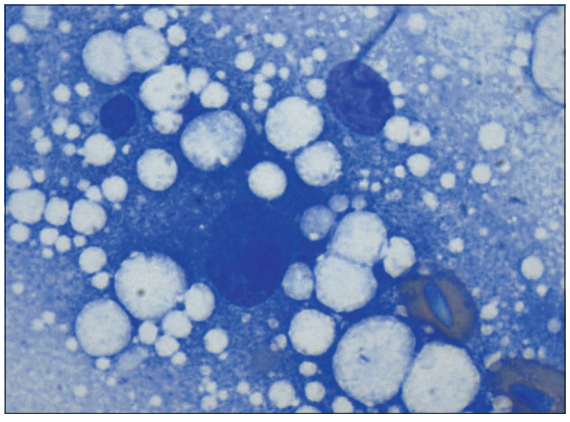


Figure 7.22 Stained liver tissue smear from an Umbrella cockatoo showing hepatic lipidosis (Diff Quik, 100×) (Photo courtesy S Echols).

Occasionally, parasites may be found on hepatic imprints. Those commonly seen are schizogony of *Haemoproteus* and *Leukocytozoon* and sporozoites of *Atoxoplasma* and microfilaria.

Spleen

Normal cytology shows a marked number of erythrocytes and lymphocytes, reflecting the cytology of a lymphoid tissue. Chlamydial infections often cause a marked increase in the number of splenic plasma cells. Macrophages often demonstrate intracytoplasmic chlamydial inclusions. See **Figs 7.23, 7.24**.

Kidney

Normal kidney produces a highly cellular sample that contains numerous epithelial cells with an abundant, slightly basophilic cytoplasm and slightly eccentric, round-to-oval nuclei (**Fig. 7.25a**). Abnormal cytology (**Fig. 7.25b**) may include inflammatory cells or the presence of neoplastic cells:

- Epithelial cells from renal adenomas show increased cytoplasmic basophilia, slight pleomorphism and occasional mitotic figures.
- Renal adenocarcinomas produce epithelial cells having features of malignant neoplasia.
- Nephroblastomas produce poorly differentiated epithelial and mesenchymal cells.

Downloaded From Vet-eBooks.com

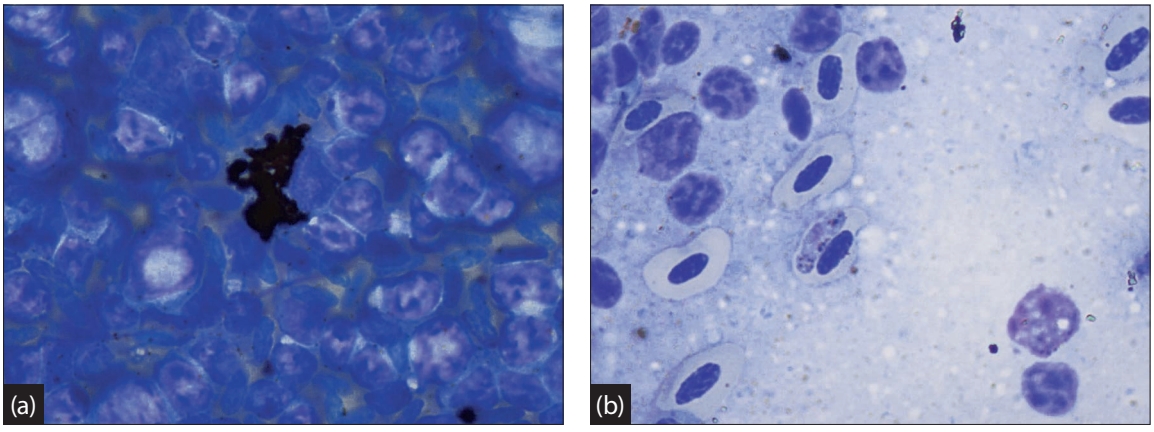


Figure 7.23 Stained splenic tissue smear from a pigeon with haemoparasitism revealing haemosiderosis (a) and reactive splenitis (b). Note the haemoparasite located within the erythrocyte (b) (Diff Quik, 100×) (Photos courtesy S Echols).

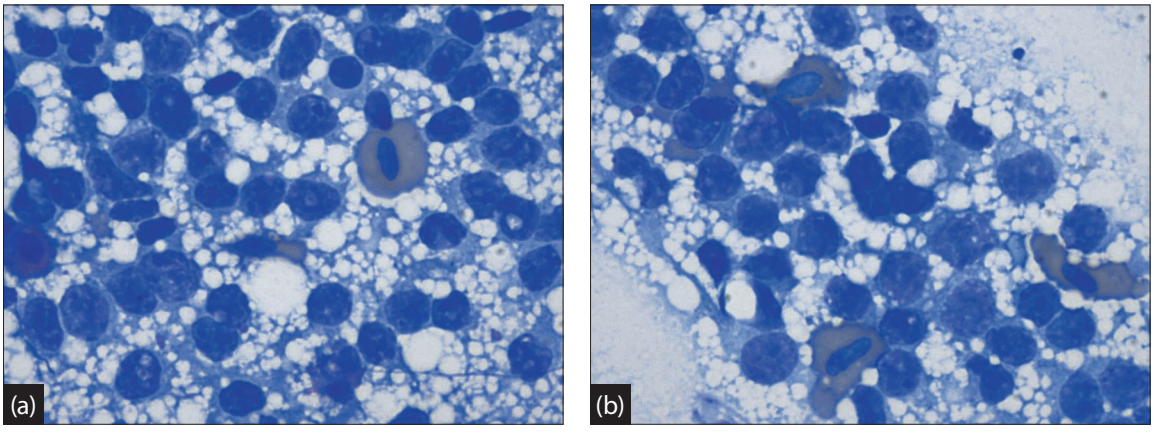


Figure 7.24 Stained splenic tissue smear from an Umbrella cockatoo indicate hepatic lipidosis (Diff Quik, 100×) (Photos courtesy S Echols).

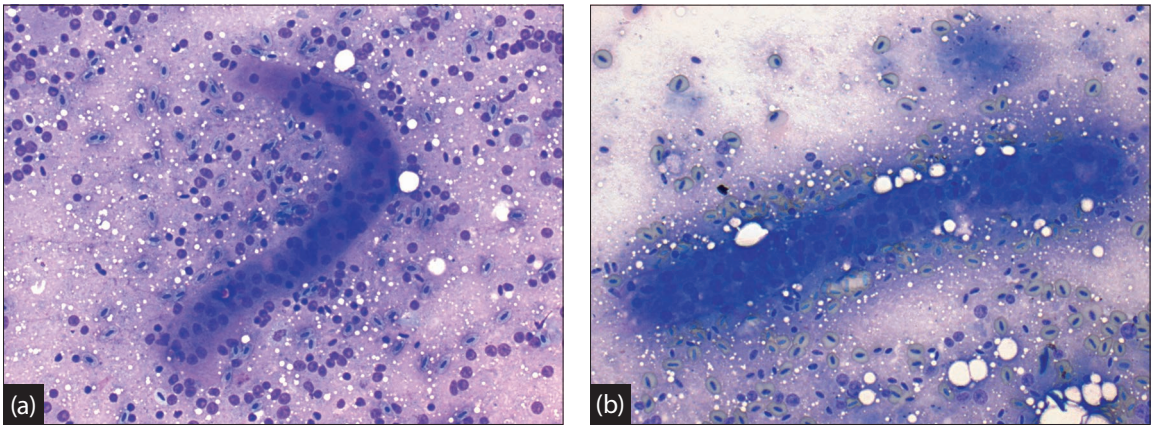


Figure 7.25 Stained tissue smears from an Umbrella cockatoo show a normal renal tubule (a) and an abnormal renal tubule due to hepatic lipidosis (Diff Quik, 20×) (Photos courtesy S Echols).

Downloaded From Vet-eBooks.com

FURTHER READING

Beaufrère H, Cray C, Tully T (2014) Association of plasma lipid levels with atherosclerosis prevalence in Psittaciformes. *Journal of Avian Medicine and Surgery*, 28(3):225–231

Campbell TW, Ellis CK (2007) *Avian and Exotic Animal Hematology and Cytology*, 3rd edn. Blackwell Publishing, Ames.

Fudge AM (1997) Avian clinical pathology. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadephia, pp. 142–157.

Fudge AM (2000) *Laboratory Medicine: Avian and Exotic Pets*. WB Saunders, Philadelphia.

Gerlach H (1994) Bacteria. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 949–983.

Harr KE (2006) Diagnostic value of biochemistry. In: *Clinical Avian Medicine, Vol 2*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 611–630.

Hochleithner M (1994) Biochemistries. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 223–245.

Ritchie BW (1995) Diagnosing viral infections. In: *Avian Viruses: Function and Control*. Wingers Publishing, Lake Worth, pp. 83–104.

Samour J (2006) Diagnostic value of hematology. In: *Clinical Avian Medicine, Vol 2*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 587–610.

Downloaded From Vet-eBooks.com

CHAPTER 8

SUPPORTIVE THERAPY

Although obtaining an accurate diagnosis and then applying a specific treatment are essential components of avian medicine, the clinician must not overlook the importance of supportive care. It is usually far better to have a tentative diagnosis in a live patient than a confirmed diagnosis in a dead one! When the masking phenomenon (hiding signs of illness until the bird is decompensating) is combined with an owner's approach of 'waiting a few days to see if he gets better', the result is often a patient that is presented badly dehydrated, in hypothermic shock and in a catabolic state due to anorexia. Occasionally, some birds will present with severe respiratory compromise, either acute or chronic in onset. In some cases, especially trauma, the patient may be in pain or have experienced significant blood loss. The clinician must recognise the clinical signs of these conditions and deal with them aggressively, often prior to making any diagnostic attempts.

DEHYDRATION

Clinical presentation

Signs include sunken eyes, strands of mucoid saliva seen in the mouth when the beak is opened, and decreased capillary refill (seen by pressing on the basilic vein on the medial side of the elbow and assessing refill time). There may be wrinkling and/or tenting of the skin on the toes and torso, decreased urinary output; and the urates may appear thick and pasty.

Management

Fluid therapy may be given via the following routes:

- Orally, if the bird is not moribund or vomiting.
- Subcutaneously, if the bird is not in shock, hypothermic, hypoproteinaemic or has poor circulation.
- Intravenously or intra-osseously. These routes can be difficult to maintain for extended periods in an alert, active patient.

The volume of maintenance fluid to be given can be simply calculated as follows:

- 10% of the bird's bodyweight in grams = the volume (in ml) to be given daily for three days.
- Then reduce to 5–7.5% daily.
- Increase this amount if there are ongoing fluid losses (e.g. diarrhoea, polyuria).
- Divide total daily requirement into two or three doses.

The types of fluid to use and their appropriate administration are discussed in Chapter 4, Clinical Techniques pp. 86–89.

HYPOTHERMIC SHOCK

Clinical presentation

The bird's feathers are fluffed to trap body heat and the bird is lethargic and sleeping a lot to conserve energy. It may be unable to remain on the perch, and is often found on the floor of the cage.

Management

Exogenous heat should be provided by placing the bird in a heated cage (**Fig. 8.1**) or by placing a heat lamp beside the cage, preferably next to a perch. Ideally, birds should be hospitalised in a heated room. The ambient temperature around the bird should be raised to 30–32°C. The bird should be monitored for signs of heat stress: panting, wings held away from the body. Care should be taken to ensure that adequate humidity is provided to prevent the bird dehydrating in a hot, dry environment.

Downloaded From Vet-eBooks.com



Figure 8.1 **A simple hospital cage which is a modified aquarium, meets many of the requirements of a hospital cage (i.e. heat, humidity, security and ease of cleaning and disinfection).**

Covering a cage with a towel or blanket does not help to keep a bird warm. This technique relies on trapping the bird's body heat and increasing the ambient temperature. A sick bird cannot generate enough warmth to heat a cage covered in a blanket.

CATABOLISM

Clinical presentation

Food may be untouched in dishes and droppings reduced in size and quantity; faeces are often dark (or even black) and urates are small and sometimes yellow. There is wasting of the pectoral muscles and weight loss (all patients should be weighed on every visit so that a record of their normal weight is maintained in their records).

Management

Placing food and water bowls in easily accessible places and offering favourite foods can encourage an ill patient to start eating. Placing food in a dish on the floor of a cage for a patient that is perching (and reluctant to leave the perch) will not encourage eating. The food bowls may need to be placed adjacent to the perch where the bird is easily able to reach them.

Alternatively, spreading the food over the floor of the cage may encourage ground-feeding birds (e.g. many finches and Australian parrots) to browse and pick at the food.

If the patient is not eating, but is not vomiting or moribund, crop gavage with a hand-rearing formula or other appropriate semi-liquid food can be instituted. An oesophagostomy feeding tube, described in Chapter 4, Clinical Techniques, p. 90 can be used to bypass the patient's head and used in cases such as head trauma where eating or passing a stomach tube is not feasible. Duodenal catheters have been used with a degree of success by some clinicians. This is a reasonably complex surgical procedure.

Total parenteral nutrition via an intravenous catheter is not routinely practised in avian medicine at this time.

RESPIRATORY COMPROMISE

Clinical presentation

Signs include open-mouth breathing, increased respiratory effort (seen as tail bobbing and inspiratory sternal lift), audible respiratory noise and collapse.

Management

If the dyspnoea is of an acute nature, the clinician must consider the possibility of tracheal obstruction. If this is the case, an air sac catheter placed in the left caudal thoracic air sac can be life-saving (see **Fig. 4.12** in Chapter 4, Clinical Techniques pp. 90–91).

Oxygen therapy supplied either through a face mask or in an oxygen chamber can help patients with respiratory compromise. Clinicians must be aware that prolonged exposure to 100% oxygen can cause perivascular oedema and increase the degree of respiratory compromise.

ANALGESIA

Clinical signs of pain

Birds, unlike domestic mammals, indicate pain in a less obvious manner than many clinicians are accustomed to. They will respond to painful stimuli in one of two ways:

- 'Fight-or-flight' responses:
  - Excessive vocalisation.
  - Wing flapping.
  - Decreased head movement.

Downloaded From Vet-eBooks.com

- Conservation–withdrawal responses:
  - Immobility.
  - Closure of eyes.
  - Inappetence.
  - Fluffing of feathers.

It is thought that the 'fight-or-flight' response is more common with acute pain from which the bird attempts to escape. In contrast, with chronic or overwhelming pain, from which perhaps the bird feels it cannot escape, the bird may adopt the 'conservation–withdrawal' responses, perhaps in an attempt to minimise the further pain that struggling would induce. Care must be taken not to misinterpret lack of movement or vocalisation as an indication that the bird is not in pain.

It is wise, therefore, to make the assumption that what would be painful to another species would be painful to humans, and adequate analgesia should therefore be provided.

Management

- Butorphanol appears to be more effective in many birds than buprenorphine. Dose: 1–4 mg/kg q6h IM, orally.
- Tramadol 15–30 mg/kg q8–12h IM, orally.
- Morphine 1 mg/kg q6–12h IM.
- Meloxicam 1 mg/kg q12h IM, or 1.5 mg/kg q12h, orally.
- Carprofen 2–4 mg/kg q24h IM, orally.

The combination of an opioid (e.g. butorphanol) and a non-steroidal anti-inflammatory drug (NSAID) (e.g. meloxicam) may achieve better analgesia than either alone. Other analgesic protocols can be found in the formulary (see Appendix 1).

BLOOD LOSS

Birds are able to withstand comparatively greater blood loss than mammals. This is thought to be the result of:

- An increased capillary surface area within skeletal muscle allowing for rapid extravascular fluid resorption to maintain vascular volume.
- The ability to mobilise large numbers of immature erythrocytes.
- The absence of the autonomic response to haemorrhage that contributes to haemorrhagic shock.

Clinical presentation

There may be a history or physical evidence of recent blood loss. Mucous membranes are pale and the respiratory rate and effort are increased. The bird may be weak and lethargic. The PCV is usually less than 20%.

Management

Many cases of blood loss do not require a transfusion. In mild cases, or when a blood donor is not available, intravenous or intra-osseous colloids or crystalloids, alone or in combination, may be sufficient. When given together, crystalloids are administered at doses of 30–40 ml/kg while the colloid is administered at 5 ml/kg. With the bird's ability to mobilise immature erythrocytes, it is not uncommon for the PCV to return to normal within seven days with this therapy alone.

In more severe cases or where a significant blood loss is anticipated (e.g. surgery), a blood transfusion may be necessary (**Fig. 8.2**). Homologous transfusions are ideal, preferably between the same species, but the same genus will give similar results. The half-life of a homologous transfusion is believed to be 6–11 days. Heterologous transfusions, on the other hand, have a half-life of three days or less. Transfusion reactions can occur with repeated transfusions, particularly heterologous transfusions.

Blood may be collected into syringes containing an anticoagulant such as sodium citrate (0.1 ml/0.9 ml blood), heparin (0.25 ml/10 ml blood), acid citrate dextrose (0.1 ml/0.9 ml blood) or citrate phosphate dextrose (0.1 ml/0.9 ml blood). Up to 10% of the donor's blood volume (1% of its bodyweight) can be collected. Once collected the blood is used within 12–24 hours. Nucleated avian erythrocytes are very metabolically active, metabolising fat and protein and consuming 7–10 times more oxygen than mammalian erythrocytes. Consequently, they do not store well.

Transfusions can be given via an indwelling intravenous catheter (**Fig. 8.2a** and **b**) or intra-osseous catheter. They can be administered as a constant

Downloaded From Vet-eBooks.com

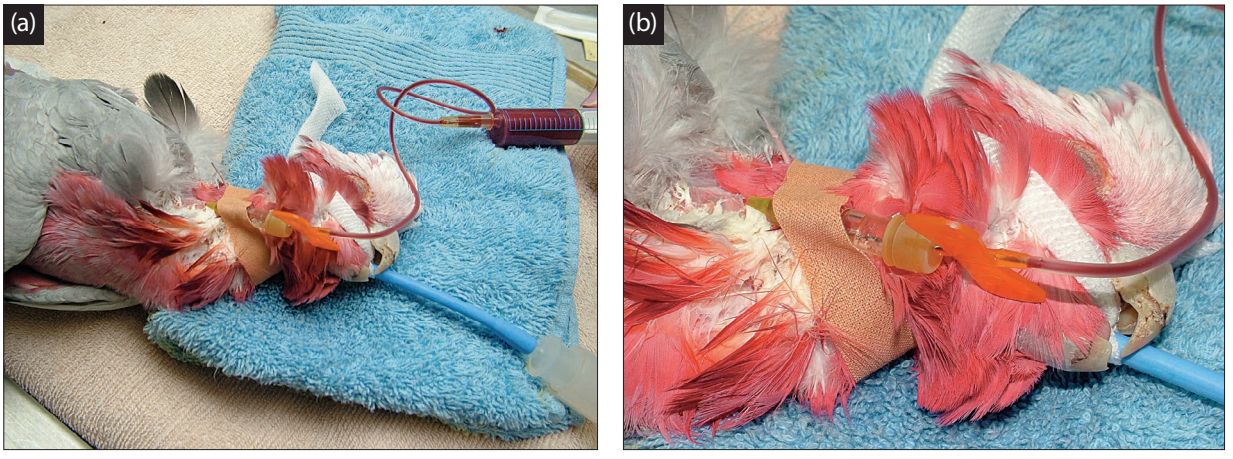


Figure 8.2 **A galah receiving a blood transfusion prior to a surgical procedure where blood loss is anticipated (a). Note that an indwelling intravenous catheter has been placed in the right jugular vein and sutured to the skin (b). Freshly collected blood is being administered via a luer plug and extension set.**

rate infusion over 1–2 hours, or a slow bolus over 1–5 minutes. If using a bolus approach, care must be taken to avoid fluid overload.

HOSPITAL CARE

Hospital care for companion birds requires a veterinary clinic to make accommodation for their differing requirements, often far removed from those needed by dogs and cats.

Security

Most companion birds are 'prey' species, and they do not feel safe or secure when housed near predators (i.e. dogs, cats, birds of prey and reptiles). Loud noises and constant movement around a cage can be stressful to a sick bird. Ideally a separate bird ward should be provided, but cage covers can provide some security to a stressed bird (**Fig. 8.3**).



Figure 8.3 **An example cage cover for a hospital cage.**

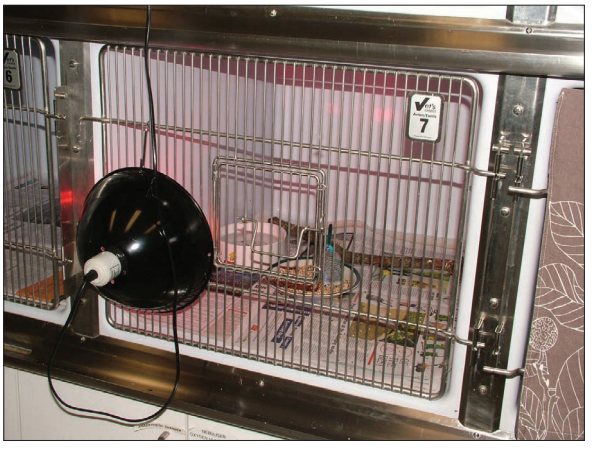


Figure 8.4 **Cages can be heated by using vivarium heating lamps.**

Warmth

As mentioned earlier, hypothermic birds need a focal heat source. Most companion birds, whether sick or healthy, are more comfortable in warmer temperatures. Air conditioning, for example, adds to a bird's energy requirements to maintain its body temperature. If possible, the bird ward should be kept slightly warmer than the rest of the hospital. Care must be taken when heating a room that humidity levels do not decrease excessively, predisposing a bird to dehydration (**Fig. 8.4**).

Downloaded From Vet-eBooks.com



Figure 8.5 **Placing food on a flat surface or the cage floor as seen in here may encourage ground-feeding birds to browse or pick at the food.**

Biosecurity

Many of the diseases for which birds require hospitalisation are infectious. All hospitalised birds are stressed to some degree, and therefore likely to be immunocompromised. Attention must therefore be taken to prevent aerosol or mechanical transmission of disease within an avian hospital facility.

Feeding

Most companion birds cannot withstand long periods of food deprivation. Therefore, every effort must be made to ensure that hospitalised birds are eating. Techniques for ensuring patients are receiving nutrition are discussed in the Catabolism section of this chapter (**Fig. 8.5**).

Psychological care

Many companion birds are closely bonded to their owner. Hospitalisation of these birds can result in separation anxiety evidenced by anorexia, lethargy or hyperexcitability. In some cases feather picking may develop. In these cases consideration must be given to discharging a patient to home care, on the proviso that the bird is returned to hospital if it fails to improve when back in familiar surroundings.

FURTHER READING

De Matos R, Morrisey JK (2005) Emergency and critical care of small psittacines and passerines. In: *Seminars in Avian and Exotic Pet Medicine: Emergency and Critical Care*. TN Tully, MA Mitchell, JJ Heatley (eds). **14**(**2**):90–105.

Graham JE (2004) Approach to the dyspneic avian patient. In: *Seminars in Avian and Exotic Pet Medicine: Emergency Medicine*. AM Fudge, MS Johnston (eds). **13**(**3**):154–159.

Harrison GJ, Lightfoot TL, Flinchum GB (2006) Emergency and critical care. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 213–232.

Jaensch SM, Cullen L, Raidal SR (2001) The pathology of normobaric oxygen toxicity in budgerigars (*Melopsittacus undulatus*). *Avian Pathology* **30**(**2**):135–142.

Jenkins JR (1997) Hospital techniques and supportive care. In: *Avian Medicine and Surgery*. RB Altman, *et al*. (eds). WB Saunders, Philadelphia, pp. 232–252.

Jenkins JR (1997) Avian critical care and emergency medicine. In: *Avian Medicine and Surgery*. RB Altman, *et al*. (eds). WB Saunders, Philadelphia, pp. 839–863.

Lichtenberger M (2004) Principles of shock and fluid therapy in special species. *Seminars in Avian Exotic Pet Medicine* **13**(**3**):142–153.

Lichtenberger M (2005) Determination of indirect blood pressure in the companion bird. *Seminars in Avian Exotic Pet Medicine* **14**(**2**):149–152.

Lichtenberger M (2006) Emergency case approach to hypotension, hypertension and acute respiratory distress. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 281–290.

Lichtenberger M, Chavez W, Thamm DH, *et al*. (2007) Use of hetastarch and crystalloids for resuscitation of acute blood loss shock. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 103–106.

Paul-Murphy J (2006) Pain management. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 233–240.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 9

DIFFERENTIAL DIAGNOSES

Listed below, by clinical signs, are the differential diagnoses that the clinician should be considering when examining a patient. Although every effort has been made to make this list as exhaustive as possible, it would be difficult, if not impossible, to guarantee that every possible differential diagnosis was included in this list.

In many cases the list goes only as far as describing the organs likely to be involved. Clinicians are referred to the chapters dealing with those organs for a more comprehensive discussion of diseases that could affect them.

CHANGE IN DROPPINGS

Diarrhoea (Fig. 9.1)

- Enteritis: bacterial; fungal; viral or toxic.
- Intestinal parasites (helminths, protozoa).
- Proventricular dilatation disease (PDD).
- Avian bornavirus (ABV).
- Chlamydiosis.
- *Macrorhabdus* (previously known as megabacteria or avian gastric yeast).

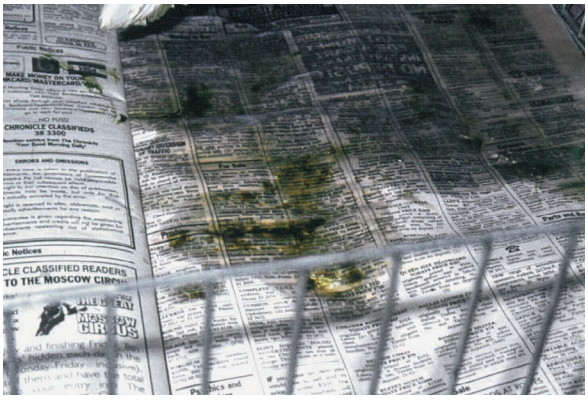


Figure 9.1 **Soiled newspaper, evidence of a bird with diarrhoea.**

- Liver disease.
- Zinc and lead poisoning.
- Intestinal foreign body.
- Change in diet.
- Normal in lorikeets.

Change in colour of the faecal portion

- Black: anorexia; melena; foreign body (see **Fig. 3.13,** Chapter 3, The Physical Examination p. 70).
- Pale: exocrine pancreatic insufficiency; maldigestion due to *Macrorhabdus* or other gastrointestinal diseases.
- Brown: pelleted diet.
- Dietary change (e.g. purple with berries).

Enlarged faecal portion (see Fig. 3.12, Chapter 3, The Physical Examination p. 70)

- Egg laying; broody.
- Space-occupying lesion in coelom.
- Exocrine pancreatic insufficiency (see **Fig. 18.1**, Chapter 18, Diseases of the Pancreas p. 265).
- Formulated diets.
- Malabsorptive diseases.
- Hernia.

Frank blood in the droppings

- Oviductal disease:
  - Egg binding.
  - Salpingitis.
  - Metritis.
  - Neoplasia.
- Cloacal disease:
  - Internal papilloma disease.
  - Cloacoliths.
  - Cloacitis.

Downloaded From Vet-eBooks.com

  - Cloacal neoplasia.
  - Cloacal prolapse.
- Warfarin toxicosis.
- Severe enteritis.

Whole seed in the droppings

- Increased intestinal motility.
- Any condition affecting proventricular and ventricular function:
  - PDD (**Fig. 9.2**).
  - Candidiasis.
  - *Acuaria* (gizzard worm) infection.
  - *Macrorhabdus*.

Change in the colour of the urates

- Green: hepatic disease; post-trauma (metabolism of haemoglobin).
- Pink/crimson: renal disease; lead poisoning.
- Yellow: anorexia; early or resolving hepatic disease.
- Orange: vitamin B injection; doxycycline injection.

Malodorous droppings

- Clostridial overgrowth.
- Cloacoliths.
- Yeast enteritis.
- Faecal retention (e.g. hernia or broody behaviour).

Polyuria

- Stress or fear, especially on initial presentation ('stress polyuria').



Figure 9.2 **Undigested seed in droppings from a bird with PDD.**

- Renal disease.
- Diabetes mellitus.
- Diabetes insipidus.
- Hepatic disease.
- Pancreatic disease.
- Hyperadrenocorticism.
- Heavy metal toxicosis.
- 'Phosphate flush' due to an all-seed diet.
- Psychogenic polydypsia, usually seen in juvenile birds.
- Pituitary adenoma in budgerigars.
- Normal in lories and lorikeets.
- Normal in birds being hand-fed or crop-fed.
- Hens during broody behaviour.

APPETITE AND THIRST

Increased appetite

- Diabetes mellitus.
- Disorders of digestion, including *Macrorhabdus* and endoparasitism.
- Jaw injuries (the bird is not actually eating, but is attempting to do so).
- Exocrine pancreatic insufficiency.
- Starvation (check there is food, and not just husks, in the food dish).

Decreased appetite

- Non-specific sign of illness.
- Mouth or jaw injuries.

Polydypsia

- Usually secondary to polyuria (see above).
- Formulated diets may cause a moderate increase in thirst in some birds.
- Psychogenic polydypsia is occasionally seen in weaning cockatoo chicks.

Decreased thirst

- Non-specific sign of illness.
- Sufficient water in green foods and vegetables.

VOMITING

- Ingluvitis (e.g. Trichomoniasis); bacterial and yeast infections.
- Generalised illness (e.g. renal disease).

Downloaded From Vet-eBooks.com

- Ileus, associated with heavy metal toxicosis or other gastrointestinal disease.
- Foreign body obstruction (e.g. from chewing on substrate or cotton fibre toys).
- *Macrorhabus.*
- Motion sickness.
- Behavioural regurgitation (e.g. after crop feeding or handling).
- Extramural obstruction (e.g. neoplasia pressing on the distal oesophagus).

WEIGHT LOSS

- Non-specific sign of illness.
- Response to controlled diet.
- Increasing fitness due to increasing exercise.
- Normal weaning response (if less than 10% weight loss).
- Malabsorption/maldigestion.

POSTURE

Fluffed, immobile, eyes closed, both legs on perch

- Non-specific sign of illness. (Note: Some sick birds will make an effort to appear normal when being examined, but can rarely maintain this for more than a minute or two before reverting to the 'sick bird look') (**Fig. 9.3**).

Fluffed, immobile, eyes closed, head tucked under one wing, only one leg on perch

- Sleeping.

Tail pointing down, perpendicular to cage floor, while wings remain at a normal angle

- May be increased respiratory effort as evidenced by tail bobbing.
- Spinal kyphosis (**Fig. 9.4**).
- Caudal coelomic mass (e.g. neoplasia, pyometra, egg binding).

Tail bobbing up and down in an exaggerated movement

- Respiratory disease: tracheal obstruction; pulmonary disease; severe air sac disease.
- External pressure on air sacs: internal organ enlargement; egg binding; ascites; obesity; kyphosis compressing coelomic space.

Head held down but bird is looking up, wings spread out, tail spread

- Reproductively active hen: courting posture.
- Heat stress.
- Egg binding.



Figure 9.3 Galah presenting with 'sick bird look'. Note the feathers are fluffed and the eyes are sunken (see also Figure 3.2, Chapter 3, The Physical Examination).



Figure 9.4 African grey parrot with spinal kyphosis.

Downloaded From Vet-eBooks.com

Wing droop
- Bilateral: generalised weakness, heat stress, bilateral wing injuries.
- Unilateral: wing injury. As a general rule, the lower the droop, the more distal the injury.

Sitting on the floor of the cage, body upright, perhaps panting
- Leg injuries.
- Egg binding.
- Renal disease causing paresis.
- Spinal disease causing paresis.
- Heavy metal intoxication.
- Clenched leg paralysis (lorikeet paralysis syndrome, barraband paralysis syndrome).

Neurological signs (ataxia; fitting; paralysis/paresis; tremor)
- Weakness due to illness.
- Agonal signs.
- Lead poisoning.
- Head trauma.
- Hypocalcaemia.
- Hypoglycaemia.
- CNS lesions (e.g. proventricular dilatation disease; encephalitis neoplasia).
- Cardiovascular disease.

Bilateral leg paresis/paralysis
- Spinal disease: trauma, neoplasia.
- 'Obturator' paralysis in hens that are egg-bound or have recently laid eggs.
- Hypocalcaemia.
- Barraband paralysis syndrome.
- Lorikeet paralysis syndrome.
- Bilateral leg trauma.
- Renal swelling.
- Heavy metal poisonings.

Unilateral leg paresis/paralysis
- Fractured leg.
- Soft tissue trauma.
- Renal enlargement (e.g. neoplasia) pressing on the sciatic nerve, predominantly seen in budgerigars.
- Neoplasia.

One wing held out to the side, resting on the floor or perch
- Check for leg trauma or unilateral leg paralysis/paresis on that side.

FEATHERS AND SKIN

Generalised feather loss
- Psittacine beak and feather disease (PBFD).
- Polyomavirus.
- Feather damaging behaviour. (Note: If this is self-inflicted, the head will appear normal).
- Extreme age.
- Obesity.
- Normal lack of feathers along apterylae.
- Excessive mutual grooming by cage mate (**Fig. 9.5**).
- Bald patch on crown of head, behind the crest, is normal in lutino cockatiels.

Feathering gradually darkening or becoming 'greasy-looking'
- Liver disease.
- Malnutrition.
- Hypothyroidism
- Age.

Broken primary feathers on wings and/or tail
- Heavy falls, often associated with poorly done wing clips.

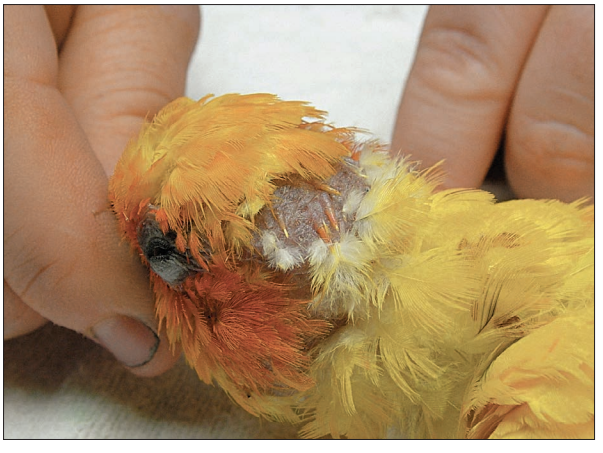


Figure 9.5 **Sun conure showing evidence of social feather picking.**

Downloaded From Vet-eBooks.com

- Small caging combined with wing flapping or inability to perch steadily.
- Cage mate aggression.
- Malnutrition.
- Feather damaging behaviour.

'Stress lines' (horizontal breaks in the feather vane)

- Physiological stress or illness at the time the feather was emerging through the skin. If severe, it could indicate generalised disease in the bird.
- Fenbendazole if administered while feathers were growing.

Long straw-like feathers over the thighs or other feathers still encased in keratin sheaths

- 'Straw feathers' in canaries, a lethal genetic disorder.
- Inability to groom properly:
  - Illness.
  - Obesity.
  - Spinal abnormalities.
  - Elizabethan collar to prevent feather-picking.
  - Beak malformations.

Abnormally coloured feathers

- Red feathers on African grey parrots:
  - PBFD.
  - Chronic feather plucking.
  - Malnutrition.
  - Liver disease.
  - Normal genetic variation.
- Abnormally coloured eclectus parrots:
  - PBFD.
  - Polyomavirus.
  - Nutritional issues, especially with hand-rearing formulae.
  - Liver disease.
  - Thyroxine medication.
- Green feathers turning black: saprophytic fungal growth resulting in grossly visible opaque black discoloration. This fungal growth is usually due to the oils from human hands left on the feathers after petting or holding the bird. This is not seen in birds with powder down (e.g. cockatoos and African greys) because the powder keeps the feathers clean.
- Galahs (Rose-breasted cockatoos) with dark grey and pink feathers:
  - Chronic liver disease.
  - Hypothyroidism.
- Lutino cockatiels becoming a deep 'buttercup' yellow; feathers have a 'greasy' appearance:
  - Chronic liver disease.
  - Hypothyroidism
- Green feathers turning yellow, blue feathers turning white (see **Fig. 10.1**, Chapter 10, Diseases of the Skin and Feathers p. 163):
  - PBFD.
  - Malnutrition.
- Young white cockatoos with dirty feathering:
  - PBFD.
  - Feeding (e.g. charcoal blocks).

Ragged looking plumage without pruritus

- Delayed moult:
  - Malnutrition.
  - Endocrine disorders.
  - Abnormal diurnal rhythm.
  - Excessive egg production.
- Incompatibility of cage mates (e.g. aggressive plucking is common in zebra finches, red-eared waxbills, golden-breasted waxbills and orange-cheeked waxbills).
- Improper housing.
- Dermatophytosis.

Continued growth of flight, tail and contour feathers in budgerigars unable to fly

- 'Feather duster'syndrome:
  - Associated with a lethal recessive gene.
  - Some reports of association with a herpesvirus.

Pruritis

- Lice and mites.
- Malnutrition.
- Dermatitis.
- Polyfolliculosis.
- Feather inclusion cysts.
- Allergic dermatitis.

Downloaded From Vet-eBooks.com

Feathers missing on the head
- Cage mate aggression.
- Dermatitis.
- Folliculitis.
- Cutaneous neoplasia.
- Dermatophytosis.
- Cutaneous candidiasis.
- Trauma.

Greenish discoloration of skin
- Bruising.

Flaky, dry skin (see Fig. 10.2, Chapter 10, Diseases of the Skin and Feathers p. 156)
- Malnutrition.
- Lack of bathing opportunities.
- Dermatophytosis.
- Cutaneous candidiasis.

Feather cysts
- Retained feathers: often pruritic. Usually seen on wings or over sternum:
  - Traumatic, often associated with incorrect housing.
  - Incomplete removal of a broken 'blood feather'.
  - Overpreening by bird.
  - Secondary to folliculitis.
- Benign follicular tumours, commonly seen in canaries, but can be seen in other species. Usually not pruritic and can occur anywhere on the body.
- Mycobacterial granulomas in the skin can be confused with feather cysts.

Polyfolliculosis
- Chronic condition most commonly seen in budgerigars and lovebirds.
- Newly emerging feathers have short, stout quills with retained sheaths.
- Usually intensely pruritic.
- Unclear whether it is a primary problem or secondary to folliculitis.

Enlarged uropygial (preen) gland
- Hyperplasia.
- Neoplasia.
- Blockage with keratin plug (sometimes associated with hypovitaminosis A).
- Infection.

Yellow subcutaneous deposits
- Fat.
- Lipoma.
- Xanthoma.

Self-mutilation of African lovebirds (*agapornis* spp.)
- Seen on the shoulder region and prepatagial membrane or, less commonly, on the inguinal region, chest, back, base of tail and around the cloaca.
- Can be unilateral or bilateral.
- Intensely pruritic.
- Cause still undetermined.

Feather damaging behaviour
- Physical problems:
  - Dermatitis/folliculitis.
  - Underlying painful lesions (e.g. arthritis, neoplasia, internal organ disease or enlargement [if painful or uncomfortable]).
  - Malnutrition.
  - Nicotine sensitivity.
  - Reproductive-associated feather picking.
  - Giardiasis in cockatiels.
- Psychological causes:
  - Fear.
  - Boredom.
  - Insecurity.
  - Anxiety.
  - Attention-seeking behaviour.
  - Sexual frustration.
  - Combination of any of the above.

WINGS

Blood on feathers
- Wing tip trauma, often associated with excessively severe wing trimming and improper housing (see **Fig. 3.18**, Chapter 3, The Physical Examination p. 74).
- Broken 'blood feather'.
- Trauma to bone, muscle or skin.

Downloaded From Vet-eBooks.com

Wing drooping
- Broken bones.
- Muscle damage.
- Weakness.
- Respiratory disease.

Swellings
- Neoplasia.
- Healing/healed broken bone.
- Soft tissue trauma.
- Feather cyst.
- Granuloma.
- Air sac rupture with subcutaneous emphysema.

Wings held away from the body
- Heat stress.
- Behavioural:
  - Courtship.
  - Fear.
  - Aggression.

Green discoloration
- Bruising.

FEET AND LEGS

Limping
- Injury to bone, muscle or joints.
- Pododermatitis (bumble foot).
- Hypocalcaemia.
- Pre-existing deformity (e.g. developmental varus/valgus deformity).

Swollen joints
- Articular gout.
- Arthritis:
  - Degenerative.
  - Infectious.
- Neoplasia.
- Trauma.

Missing nails or toes
- Aggressive cage mates.
- Unsafe caging or cage furniture.
- Ergotism (**Fig. 9.6**).



Figure 9.6 **Parrot with avascular necrosis of the digits, possibly associated ergotism.**

- Toe constriction due to fibrous band formation (constricted toe syndrome) or foreign bodies (e.g. cotton thread).
- Frost damage.

Overgrown nails
- Chronic liver disease.
- Inadequate/unsuitable perches.
- Malnutrition.
- PBFD.
- Abnormal toe anatomy preventing normal perching (e.g. articular gout), chronic trauma.

Abnormal shape or direction of the legs
- Coxofemoral subluxation: splay leg.
- Nutritional secondary hyperparathyroidism.
- Incorrectly aligned healed fractures.
- Joint luxation.
- Slipped tendon (the gastrocnemius tendon 'slips' out of the groove on the back of the hock). Associated with trauma, poor diet, poor conformation and perhaps a genetic influence.
- Angular limb deformity (rotated femur and/or tibiotarsus).

Hyperkeratosis of the scaled part of the leg
- *Cnemidocoptes*.
- Nutritional deficiency, especially zinc and/or biotin.
- Dermatophytosis.

Downloaded From Vet-eBooks.com

Self-mutilation of foot and toes
- Osteomyelitis.
- Myositis/tendonitis.
- Pododermatitis.
- Neuralgia.
- Necrosis of the extremities (see above: missing nails or toes).
- Leg ring construction.

BEAK

Overgrown beak
Sometimes with bruising present in the keratin of the beak:
- Liver disease.
- *Cnemidocoptes*.
- Lack of occlusal wear.
- Lack of chewing opportunities.
- Hypothyroidism.

Beak twisted to the left or right
- Scissor beak:
  - Congenital.
  - Acquired.

Upper beak inside the lower beak
- Prognathism.

Inability to close beak properly
- Subluxation of the palatine bone due to hyperextension of the maxilla in macaws.
- Hyperextension of the mandible.
- Fractured jaw.

Flakes of keratin on the beak
- Malnutrition.
- Lack of an abrasive surface in the cage to groom beak on.

White, crusty, honeycombed lesions on the beak
Sometimes also on feet and vent:
- Scaly face mite.

Nares unequal in size
- Chronic respiratory disease.
- Cere abscess.
- Neoplasia.
- Rhinolith.

Thickening and hypertrophy of cere of budgerigar hens
- Cere hypertrophy associated with hyper-oestrogenism.

Nares blocked or staining/matting of feathers above nares (Fig. 9.7)
- Chronic respiratory disease.
- Choanal atresia (African grey parrots).

EYES

Feather loss around eyes
- Rubbing of the face against a perch or sides of cage
  - Conjunctivitis (**Fig. 9.8**).
  - Sinusitis.
  - Ocular and periocular pain due to other causes (e.g. neoplasia, avian poxvirus).
  - Blocked nasolacrimal duct.
  - Overgrooming by companion bird.



Figure 9.7 **Cockatiel presents with nasal exudate due to sinusitis.**

Downloaded From Vet-eBooks.com



Figure 9.8 **Cockatiel with periocular feather loss and chlamydial conjunctivitis.**

Eyelid abnormalities

- Congenital:
  - Cryptophthalmos (failure of eyelid formation, resulting in fusion of the eyelid margins) is occasionally seen in cockatiels.
  - Blepharophimosis (narrowing of the palpebral fissures without fusion of the eyelid margins) is occasionally seen in all species.
- Acquired:
  - Symblepharon (adhesion of the eyelid(s) to the globe) can be seen as a sequela to severe conjunctivitis.
  - Acquired blepharophimosis is occasionally seen after conjunctivitis or other ocular inflammatory conditions.
  - Scarring and deformity is occasionally seen after avian poxvirus infections.

Thickening and hyperaemia of the conjunctiva

Often associated with epiphora or ocular discharge:

- Conjunctivitis:
  - Chlamydiosis.
  - Mycoplasma.
  - Mycobacteria.
  - Parasites ('eye' worms): *Oxyspirura mansoni*, *Ceratospira* spp. and *Thelazia* spp.
  - Other chronic infections.
  - Allergic, especially secondary to nicotine.
- Sinusitis.
- Avian poxvirus.
- Neoplasia.

Exophthalmos

- Sinusitis.
- In chicks it is often associated with stunting or nutritional secondary hyperparathyroidism.
- Retrobulbar neoplasia, especially lymphoma.
- Pituitary adenomas in budgerigars.

Enophthalmos

- Dehydration.
- Sinusitis in macaws.
- Microphthalmia.

Corneal changes

- Usually multifocal, white, glistening, raised, non-inflammatory corneal lesions: lipid or cholesterol deposits associated with high-fat diets and obesity.
- Keratitis or corneal ulceration:
  - Primary: bacterial, fungal or viral (avian poxvirus).
  - Secondary: trauma or exposure due to eyelid deformity or malformation.
- Mass on cornea:
  - Dermoid.
  - Staphyloma: uveal herniation into a weakened, distorted area of the cornea.
  - Descemetocoele.

Hyphema

- Trauma.
- Warfarin toxicosis.
- Neoplasia.

Cataracts

Similar aetiology to mammals; can be inherited or acquired.

FACE

Swellings on the face

- Sinusitis.
- Neoplasia.
- Insect bite.
- Trauma.

Matting/staining of feathers below and caudal to eye

- Otitis externa.

Downloaded From Vet-eBooks.com

Matting of the feathers over face and head
- Vomiting.

Small nodules on the facial skin of macaws
- Macaw acne: ingrown feather follicles that produce a reactive nodule.

BODY

Prominent keel bone ('going light')
- Chronic illness, resulting in catabolism of the muscle.
- Inability to fly, causing the muscles to atrophy due to disuse. (Note: This degree of muscle wastage is usually not as severe as seen with illness.)

Twisted keel bone
- Nutritional secondary hyperparathyroidism.
- Genetic abnormality.

Split keel bone (non-traumatic)
- Bifid sternum - genetic abnormality where the two halves of the sternum have failed to fuse correctly.

Ulcerative lesion on the cranial end of the keel
- Chronic self-mutilation and trauma resulting from falling heavily to the ground. Most commonly seen in birds with overly severe wing trims (**Fig. 9.9**).
- Ulcerating lipoma or 'fat abscesses'.

Overabundance of pectoral muscle mass (i.e. 'cleavage') along keel
- Obesity.

Enlarged coelom
Increased space between the sternum and the pubic bones.
- Solid enlargement:
  - Obesity.
  - Hepatomegaly.
  - Internal neoplasia (e.g. renal, gonadal).
  - Egg binding.
  - Oviductal enlargement in breeding season.
  - Normal in young birds still being fed.
  - Intestinal parasitism (severe).
- Fluid enlargement:
  - Yolk-related peritonitis.
  - Ovarian cyst.
  - Neoplastic cyst.
  - Ascites associated with heart and/or liver disease.
  - Neoplastic effusion.
- Hernia.



Figure 9.9 **Cockatiel with sternal trauma associated with heavy falls due to excessive wing trimming.**

Subcutaneous emphysema
- Trauma.
- Post-endoscopy air leakage.
- Rupture of cervicocephalic air sac due to trauma or air saculitis.

Split in skin between vent and tail
- Heavy landing associated with overly severe wing trim.

Subcutaneous masses
- Lipomas (**Fig. 9.10**).
- Xanthomas.
- Fat deposits.
- Hernias.
- Abscesses.
- Feather cyst.

Downloaded From Vet-eBooks.com

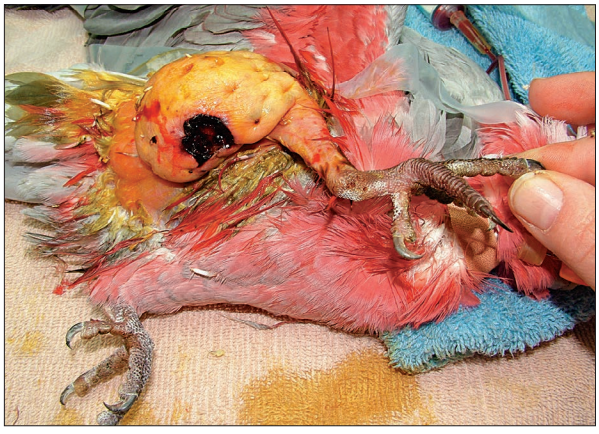


Figure 9.10 Galah with a lipoma.

PAEDIATRICS

Crop not emptying, or slow to empty
- Generalised illness with ileus.
- Bacterial or yeast ingluvitis.
- Hand-rearing formula mixed incorrectly: incorrect temperature or consistency.
- Foreign body obstruction.

Cervical emphysema
- Ruptured cervicocephalic air sac, often due to rough handling while being fed.
- Gulping air while feeding (in this case the air is in the crop, not under the skin).

Erythematous skin
- Dehydration.
- Heat stress.
- Generalised illness.

Pallor
- Cold stress.
- Illness.
- Anaemia.

Overly large head
- Stunting: reduced rate of growth due to any cause.

Bruising on the skin
- Severe bacterial or viral infection, especially polyomavirus.
- Injections (e.g. enrofloxacin).
- Trauma from parents or siblings.

Feathers not growing normally
- Stunting: reduced rate of growth due to any cause.
- Polyomavirus.

Swollen toes
- Constricted toe syndrome.
- Bedding or thread wrapped around toe, acting as a tourniquet.

Thin toes
- Stunting: reduced rate of growth due to any cause.

Vomiting
- Ingluvitis.
- Generalised illness (e.g. renal disease).
- Ileus, associated with heavy metal toxicosis or other gastrointestinal disease.
- Foreign body obstruction (e.g. from chewing on substrate or cotton fibre toys).
- Weaning, particularly South American species.

Refusing to eat
- Weaning.
- Non-specific signs of illness.
- Inappropriate husnandry.

Reddened skin or scab over the crop
- Crop burn.
- Trauma from crop needle.

FURTHER READING

Doneley B, Harrison GJ, Lightfoot TL. (2006) Maximizing information from the physical examination. In: *Clinical Avian Medicine, Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 153–212.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

DISEASES OF THE SKIN AND FEATHERS

CONGENITAL DISORDERS

'Feather duster' or 'chrysanthemum' syndrome in budgerigars

Definition/overview

Juvenile budgerigars exhibit continued growth of flight, tail and contour feathers. As the feathers are soft and curled, it gives the bird the appearance of a feather duster (**Fig. 10.1**).

Aetiology

This is believed to be a lethal recessive genetic disorder. There is thought to be an association with budgerigar herpesvirus, but a 'cause and effect' has yet to be established.

Clinical presentation

Affected birds have excessively long and curled feathers; they are unable to fly and make a barely audible noise. Most die within the first few years of life.



Figure 10.1 **Budgerigar with 'feather duster' syndrome.**

'Straw feather' in canaries

Definition/overview

Feathers fail to emerge from the feather sheath, giving them a straw-like appearance.

Aetiology

This is a lethal disorder, believed to be hereditary in canaries.

Differential diagnosis

It must be differentiated from any condition that prevents the bird from grooming properly.

Feather cysts in canaries

Definition/overview

These appear as in-grown feathers forming hard, yellow nodules on the skin of canaries with complex feathering such as the Norwich, Border and Gloucester strains.

Aetiology

They may actually be benign neoplasms of the feather follicle. Several dermal papillae form in each follicle, resulting in a tangle of feathers that fail to erupt from the skin. It is believed to be a hereditary condition. Affected canary strains have been genetically selected to produce an extra down-type feather (double buff) that may predispose them to cyst formation. The mode of inheritance is not clear and other factors (e.g. infections; trauma) may play a role in the development of the condition.

Clinical presentation

The dorsal thoracic area is a frequent site of multiple cysts, but they can occur anywhere on the body and wings. They often occur in several sites along a feather tract. The cysts appear as hard, yellow nodules containing the trapped curling feathers.

Downloaded From Vet-eBooks.com

Badly affected birds have irregularly directed feathers all over their bodies. Actively growing feather cysts will have vascular walls and contain blood and gelatinous material. Mature cysts will contain drier keratinous material, and the cyst wall may be more expansive, thickened and reduced in vascularity.

Management

Treatment of individual cysts can be conservative, simply lancing and expressing the contents of the cyst. Recurrence is common, but surgical excision of the affected follicle is generally curative. When multiple cysts are present, surgical excision of the affected tract may be required. Cysts on the wings may not be amenable to surgery without partial wing amputation. Therefore, conservative therapy may be warranted, so long as the owner is aware of the probability of an ongoing problem.

'Porcupine feathers' in homer and fantail pigeons

A similar condition to 'straw feather' in canaries.

Baldness in lutino cockatiels

A bald patch is present behind the crest feathers on the crown of the head in lutino cockatiels. No treatment is required or available.

NUTRITIONAL DISORDERS

Malnutrition may affect the skin and feathers in several ways.

Feather quality

- Brittle feathers.
- Abnormal moulting resulting in frayed or damaged feathers.
- Inability to preen, resulting in retained feather sheaths.
- Stress bars. Breaks in the feather vane due to brief episodes of dysfunction of epidermal collar associated with release of corticosteroid hormones during some stress. It can be associated with periods of malnutrition, especially during the weaning process.

Feather colour

Colour in birds is the result of a combination of feather structure (affecting the passage or reflection of light), melanin pigments (black, grey and brown) and carotenoid pigments (yellow and red). Nutrition will affect both the feather structure and the amount and type of carotenoids in the feather. Malnutrition can therefore produce some of the following effects on feather colour:

- Fading and dullness of plumage (**Fig. 10.2**).
- Red feathering in African grey parrots.
- Abnormal colours in eclectus parrots.
- Colour changes (e.g. green feathers turning yellow, blue feathers turning white).

It must be remembered that other agents can also affect feather colour, notably genetics and viral diseases such as PBFD.

Skin changes

- Increased scaliness of the skin (**Fig. 10.3**).
- Subcutaneous fat deposits giving the skin a yellowish hue.
- Increased skin fragility such that the skin tears easily. A common example of this is 'tail split' injuries in cockatiels with badly done wing clips. The combination of a heavy fall and fragile skin leads to a split in the skin between the vent and the tail (**Fig. 10.4**).



Figure 10.2 **King parrot fed an all-seed diet for many years. Note the general dullness of the plumage, the red feathers turning orange and the green feathers turning yellow.**

Downloaded From Vet-eBooks.com



Figure 10.3 Black cockatoo with nutritional dermatitis.



Figure 10.4 Cockatiel with 'tail split' injury associated with a heavy fall.

Management

A dietary assessment to identify and correct nutritional deficiencies is an essential component of the evaluation of any dermatological problem in birds (see Chapter 2, Husbandry, Grooming and Nutrition pp. 56–61).

ENDOCRINE DISORDERS

Hypothyroidism

Clinical presentation

Poultry with thyroiditis demonstrate changes in feather quality and colour. Black, brown and yellow feathers become red, longer and more pointed



Figure 10.5 Cockatiel with suspected hypothyroidism, note the darkening of the feathers and a 'greasy' appearance.

and have fewer pennaceous barbules than normal. A similar syndrome has been seen in galahs with suspected hypothyroidism. These birds develop long, narrow primary flight feathers and develop a pink–red discoloration of the grey plumage. At the same time, the normally pink feathers deepen in colour intensity.

An obese scarlet macaw with confirmed hypothyroidism had non-pruritic feather loss, mild non-regenerative anaemia, mild leucocytosis, heterophilia, hypercholesterolaemia and sparse feathers, and it had not moulted in over a year. It responded well to thyroxine therapy.

Lutino cockatiels with suspected hypothyroidism may show deepening colour intensity and a loss of barbs and barbules in the feathers, giving the bird a 'greasy' deep yellow colour (**Fig. 10.5**). The rhinotheca is often overgrown. Results of liver function tests in these birds are often normal, although cholesterol levels are often elevated.

Diagnosis

Hypothyroidism is much over-diagnosed. Diagnosis is difficult because normal resting T_4 levels in birds (5–15 ng/ml) are much lower than in mammals and are often below the detectable limits of many laboratory techniques and equipment. Additionally, T_3 levels (0.5–4 ng/ml) may be as important as T_4 in overall thyroid function. Measuring only T_4 may not present an accurate picture of the health of the

Downloaded From Vet-eBooks.com

thyroid gland therefore. The half-life of both hormones is shorter in birds than mammals, resulting in significant diurnal variation in a normal bird. There is also seasonal variation, with higher baseline levels in spring. Correct diagnosis of hypothyroidism therefore requires demonstration of a failure to respond to thyroid stimulating hormone (TSH). (Note: Avian TSH is not commercially available, so mammalian TSH has to be used.) After obtaining a baseline T_4 measurement, a bird should be given 1 IU/kg of TSH and T_4 levels determined 4–6 hours later. A positive response in a normal bird will see a 2.5 fold or greater increase of the resting T_4 level.

Supporting evidence for a diagnosis of hypothyroidism includes hypercholesterolaemia, elevated triglycerides and liver enzymes, and a mild non-regenerative anaemia. It has been suggested that, in larger birds, endoscopic biopsy of the thyroid gland may lead to a diagnosis of hypothyroidism.

Management

Despite the lack of laboratory confirmation, some obese birds who demonstrate a lack of weight loss after following a rigid diet, accompanied by poor quality feathers and infrequent moults, have been seen to respond favourably to thyroxine therapy. Thyroid supplementation can be given orally at a starting dose of 0.01–0.02 mg/kg PO q12h. However, please note that no pharmacological data is currently available to indicate the ideal administration of thyroid hormone replacement in birds. In addition, a positive response to thyroid supplementation does not confirm a diagnosis of hypothyroidism.

Delayed moulting

Aetiology

Delayed moulting is usually caused by a combination of malnutrition, abnormal diurnal rhythm (especially for companion birds kept indoors), concurrent illness and endocrine disorders (e.g. hypothyroidism). Excessive egg production may cause abnormal or delayed moulting due to endocrinal effects on feather growth.

Clinical presentation

Birds present with untidy plumage, bald spots and damaged, brittle feathers. The ends of the feathers often become frayed and lose their colour. Other (non-dermatological) signs may include decreased vocalisation in male canaries, excessive or decreased egg production and general lethargy.

Management

Normal diurnal rhythms should be re-established by ensuring the bird gets at least 12–14 hours of 'sleep time' in a darkened, quiet environment. Simply covering a cage in a busy family area is insufficient to meet the sleep requirements for most birds. Nutritional problems must also be corrected.

Egg production should be controlled through nutritional, environmental, social and hormonal manipulation (see Chapter 23, Disorders Affecting the Reproductive Tract p. 326).

Trauma

Aetiology

A variety of conditions can result in traumatic skin damage, including self-trauma, sharp objects and attacks by other animals.

Clinical presentation

Care must be taken to differentiate between a laceration and a normal apteryla. Avian skin sometimes appears transparent, giving the appearance of a wound with underlying structures visible. Most skin wounds will be obvious with blood matted into the surrounding feathers.

Treatment

Some of these injuries are simple lacerations; others are more complex injuries associated with extensive soft tissue and even bone damage (**Fig. 10.6a**). Considerations when planning repair include:

- Avian skin is closely attached to underlying bony structures, but less closely attached to muscle. Loose skin can be found on the neck and the inguinal area, leaving most of the skin on the torso relatively immobile. This makes undermining and mobilising skin to close wounds on the torso and limbs more difficult than similar surgery in many mammalian species.
- Avian skin has a fatty subcutaneous layer but sutures in this layer often do not hold well.

Downloaded From Vet-eBooks.com

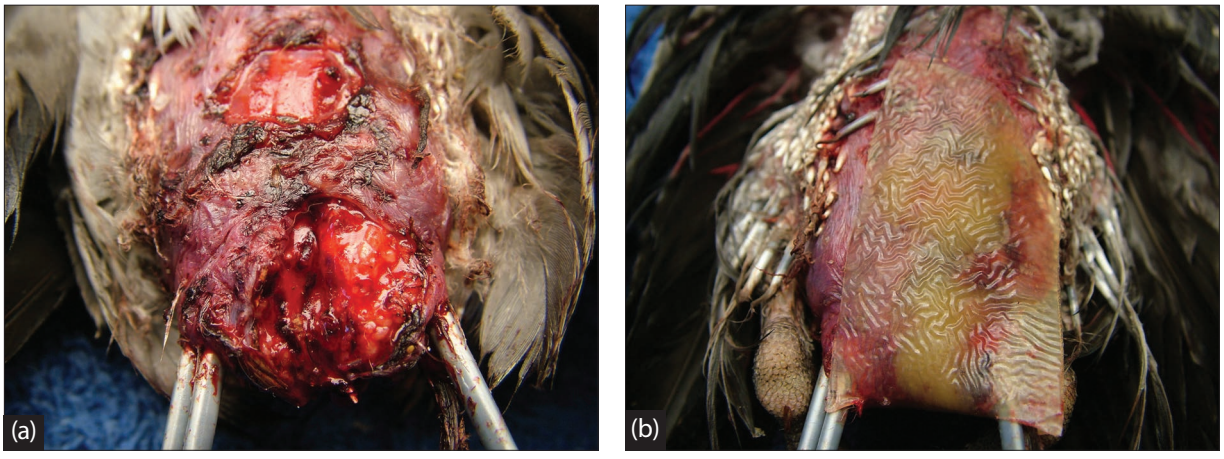


Figure 10.6 **This galah was attacked by another bird and has traumatic lesions on the dorsal aspect of its tail (a). The wound was covered with a hydrocolloid dressing (Duoderm®) (b). The wound has healed well over a 6-week period.**

- Post-operative swelling is not as severe in birds as it is in mammals, and sutures can therefore be tied tighter and closer together.
- Avian heterophils lack lysozymes, meaning that avian pus is usually caseous and therefore does not drain from a contaminated wound readily. The use of drains in these wounds may not be of significant benefit.
- If primary closure is achieved, sutures can be left in place for 10–14 days. However, the thin epidermis of birds allows dehiscence of primary skin closure if primary healing has not occurred. If a significant scab occurs over the sutures, it is best not to remove them until the scab is ready to slough; this may take several weeks.

Repairs of skin lacerations in birds require that the surgeon:

- Thoroughly debrides and flushes the wound. In most cases, the use of sterile saline is satisfactory as a flushing solution. Chlorhexidine and povidone iodine, even diluted, can be toxic to granulation tissue and should be avoided if possible.
- Mobilises available skin where possible to achieve primary closure without tension on the wound edges.
- Avoids the use of drains unless combined with a flushing or irrigation system.
- Uses minimally reactive absorbable sutures, placed closely together.
- Removes the sutures only when healing is completed.

Where primary closure cannot be achieved, skin grafts/flaps or healing by second intention is required. Using the same surgical techniques employed in mammals such as dogs and horses, skin grafting and flaps are feasible alternatives in birds. If skin grafting is not employed, good healing can often be achieved by secondary intention. In this situation, avoid the underlying subcutis and muscle drying out by using wet or hydrocolloid dressings (e.g. Duoderm®) (**Fig. 10.6b**).

Aggressive post-operative antibiosis and analgesia will significantly improve morbidity and mortality rates in these patients. In most cases, if the area is less painful after the surgery, the bird will usually not bother the surgery site and an Elizabethan collar is unnecessary. These collars are stressful to the bird and reduce its ability to move, eat and drink normally; they should be used only when absolutely necessary.

BACTERIAL INFECTIONS

Definition/overview

Staphylococcus spp. are suspected of being the most common bacterial skin pathogens. They can

Downloaded From Vet-eBooks.com

result in generalised skin infections, which may appear as a folliculitis or a dermatitis (**Fig. 10.7**). Localised or multifocal swellings may be abscesses from infected wounds, damaged feather follicles or foreign bodies (**Fig. 10.8**), or *Mycobacterium* infection.

Clinical presentation

In generalised *Staphylococcus* infections the skin is usually very pruritic, and often erythematous. *Mycobacterium* infection causes localised or multifocal lesions that may be wart-like, dry flaky swellings of the skin, granulomatous skin lesions or raised ulcers.



Figure 10.7 **Cockatoo with dermatitis triggered by a staphylococcal infection following a dog 'mouthing' the bird.**



Figure 10.8 **Sun conure with a feather cyst.**

Diagnosis

Diagnosis requires skin biopsy (including follicles) and culture. Meticillin-resistant *Staphylococcus aureus* (MRSA) is becoming a more recognised entity in these cases.

Management

Systemic antibiotic therapy is given as indicated by culture. Washing the bird two to three times weekly with benzyl peroxide or chlorhexidine shampoos can be beneficial by removing a lot of the scale and debris on the skin and reducing the bacterial load. Surgical excision of localised lesions may be curative.

The zoonotic potential of *Mycobacterium* and MRSA must be discussed with the owner before attempting treatment (see Chapter 16, Diseases of the Gastrointestinal Tract p. 243).

FUNGAL INFECTIONS

Aetiology

Candida albicans, *Malassezia pachydermatis*, dermatophytes (*Microsporum gallinae*, *M. gypseum*, *Trichophyton verrucosum*, other *Trichophyton* spp.), *Cryptococcus bacillisporus* (formerly *C. neoformans* var. *gattii*) and *C. neoformans* var. *grubii* (formerly *C. neoformans* var. *neoformans* serotype A), *Aspergillus* spp.

Clinical presentation

Candida causes lesions around the commissures of the mouth and nares and occasionally around feather follicles on the head, back and ventral abdomen.

Malassezia results in generalised pruritus, sometimes with crusting and folliculitis.

Dermatophytes cause a slow-spreading infection leading to scabs, crusts and alopecia on the body and thin-skinned areas of head and upper beak, and a rough, porous appearance of the podotheca. In gallinaceous birds the characteristic scaly, crusty lesions of the wattle, comb and legs are known as 'favus'.

Cryptococcus has been reported to cause subcutaneous nodules on the head and body and raised nodular lesions on the face and beak.

Cutaneous aspergillosis has been associated with focal ulcerative pruritic lesions.

Downloaded From Vet-eBooks.com

Diagnosis
Cytology must be used with caution; yeast bodies stain similarly to feather dust and can be nearly impossible to distinguish from one another. Biopsy and fungal culture may therefore be required to confirm a diagnosis.

Management
Systemic antifungals (itraconazole, fluconazole, ketoconazole, terbinafine) are given orally for 1–3 months. Topical treatment may be carried out using enilconazole wash, clotrimazole wash or cream, or miconazole shampoos.

VIRAL INFECTIONS

Viral infections can cause dystrophic feathers (PBFD virus, polyomavirus, adenovirus and parvovirus [waterfowl]) or skin lesions (poxvirus, papillomavirus and herpesvirus).

Psittacine beak and feather disease

Aetiology
PBFD, a circovirus, is a non-enveloped single-strand DNA virus measuring 14–17 nm.

Pathogenesis
The virus has a minimum incubation period of 21–25 days, but it could be as long as several years. It is shed in faeces, crop secretions and feather dust (vertical transmission is suspected but not confirmed). Following infection the virus is disseminated haematogenously, localising in the feather follicle epithelium, the thymus and the bursa. In cockatoos it also localises in the growth areas of the beak. The result is feather dystrophy, immunosuppression and, in cockatoos, overgrowth and underrunning of the rhamphotheca.

Clinical presentation
There are two forms of PBFD: acute and chronic. PBFD is more common in juveniles than in adults, but naïve adults are also susceptible. Although all psittacines are susceptible, New World parrots and cockatiels appear to be rarely affected.

Acute PBFD is seen in juveniles around weaning age (especially African grey parrots). They are lethargic, fluffed and anorexic. Haematology may show pancytopenia and non-regenerative anaemia. Affected birds often die (within a few days) with severe hepatic necrosis before feather abnormalities develop. Feather lesions have been noted in fledgling birds at 28–32 days old.

Chronic PBFD causes progressive replacement of normal feathers with dystrophic feathers (retained sheaths and blood supply, clubbed appearance, stress lines, constrictions and abnormal shapes). The degree and location of the feather loss may depend on the state of moult when the bird was initially infected. Typically lesions develop in order of powder down, contour, primaries, secondaries, tail and then the crest. Poicephalus species and lories may only lose tail feathers and primary flight feathers (**Fig. 10.9**). These feathers may then regrow. Neophemas may develop untidy plumage and lose feathers easily when handled. Many parrots develop feather colour changes; blue feathers become white, green feathers become yellow. Beak lesions are only seen in cockatoos, and include palatine necrosis, ulceration, elongation and easily fractured beaks (**Fig. 10.10**). Immunosuppression is common in all species.

Diagnosis
Histopathology: Basophilic intracytoplasmic inclusion bodies found in feather follicles and the cloacal bursa are considered diagnostic (**Fig. 10.11**). Care must be taken not to confuse them with herpesvirus



Figure 10.9 **Lorikeet with PBFD showing loss of distal primary feathers.**

Downloaded From Vet-eBooks.com



Figure 10.10 **Wild cockatoos with PBFD.**

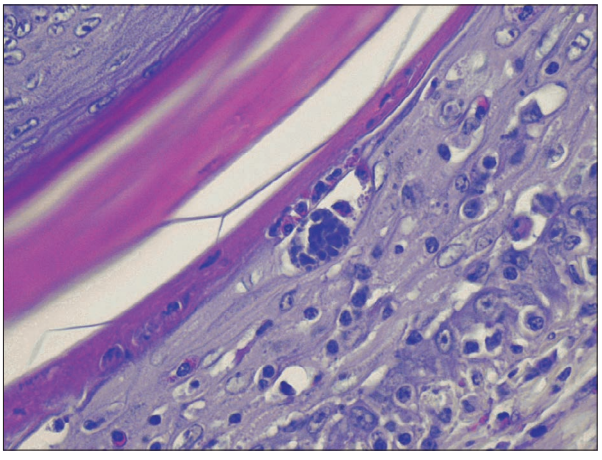


Figure 10.11 **Stained tissue smear reveals a botryoid inclusion follicle in a case of PBFD. (Magnification × 400).**

or adenovirus. Feathers and skin show multifocal necrosis of epidermal cells, epidermal hyperplasia and epidermal hyperkeratosis. Diffuse necrosis of epidermal cells is seen throughout the epidermal collar and in basal and intermediate layers of developing feathers. Beak histopathology shows hyperkeratosis and separation of the cornified outer layer from underlying tissues and bones. Atrophy and focal aggregation of necrotic cells are seen in the thymus and bursa.

Haemagglutination (HA) and Haemagglutination Inhibition (HI): HA is the agglutination of washed erythrocytes induced by the presence of virus in the sample. The result, expressed as a titre, is the highest dilution of the erythrocytes that causes agglutination. The higher the titre the higher the virus excretion in the patient.

HI is the inhibition of this agglutination due to the presence of antibody. The titre is the highest dilution that still inhibits agglutination i.e. the higher the titre, the higher the level of antibodies. A positive HI titre confirms previous exposure to BFDV infection.

- Birds with chronic PBFD typically have a high HA titre and a low HI titre.
- Birds with high HI titres are generally free of the disease.

PCR: Viral-specific PCR probes are the most sensitive way to detect BFDV infection, but they give no indication of whether the bird is latently infected or transiently viraemic. Non-replicating viral DNA may take up to three months to clear from the blood. PCR should therefore be used alongside HA/HI measurements, rather than replacing them. PCR can be also be used alongside viral DNA sequencing and other genotyping methods to determine the source of infection.

If HA/HI measurements are not possible, birds who present as clinically normal but have a positive PCR result should be re-tested after three months.

Management

Avian interferon may be of value if given before the bird shows clinical signs. Otherwise, supportive care (e.g. treating secondary infections and providing a good diet) is all that can be done. Although research has been conducted, there is no commercially available vaccine at this time.

Prognosis

Some birds (e.g. lorikeets) appear to mount an effective immune response and apparently recover. However many of these birds become inapparent carriers. Some birds, despite showing clinical signs, may live for 10–30 years, but most infected birds die within two years of secondary diseases related to immunosuppression.

Downloaded From Vet-eBooks.com

Avian Polyomavirus

Aetiology

Polyomavirus is a non-enveloped virus that is relatively environmentally stable. All parrots can be affected, but it is most common in macaws, conures, eclectus parrots and caiques. It is rare in African grey parrots, cockatoos and cockatiels. Passeriformes, including grass finches, canaries, goldfinches and greenfinches can also be infected.

Clinical presentation

Budgerigars are the primary reservoir of polyomavirus and the signs of infection include decreased hatchability and embryonic death, abdominal distension, subcutaneous haemorrhages and feather dysplasia which may resolve after several months. Primary and secondary feathers on the wings and tail are lost or fail to erupt. The budgerigar fancy call these birds 'runners' or 'creepers' and the condition is known as 'French moult' (**Figs 10.12a** and **10.12b**). There may be signs of neurological damage if the cerebellum is affected.

In other parrots the virus typically causes sudden death 10–14 days after exposure (especially those less than 15 days old at the time of exposure). In larger parrots, most deaths occur between 20 and 140 days old.

In estrilid finches (grass finches) the virus causes acute mortality in two- to three-day-old fledglings, young adults and mature finches. Survivors may demonstrate poor feather development and misshapen lower beaks.

More detail on this virus can be found in Chapter 27, Paediatrics (p. 366).

Avian poxvirus

Aetiology

Avian poxvirus is a large (up to 400 nm) enveloped DNA virus. All avipoxviruses are morphologically similar, but they have differing host specificity.

Pathogenesis

The virus is environmentally stable and can survive for years in dried organic debris. The virus must enter the body through mucous membranes or abraded skin. It is unable to penetrate intact epithelium. It can be transmitted directly through fighting, feather damaging behaviour or preening, or it can be indirectly transmitted by blood-sucking insects (e.g. mosquitoes). The virus will either remain at the point of entry, causing localised infection, or spread haematogenously to the liver and bone marrow, producing a systemic infection. The incubation period is 7–9 days in pigeons and four days to three weeks in canaries.

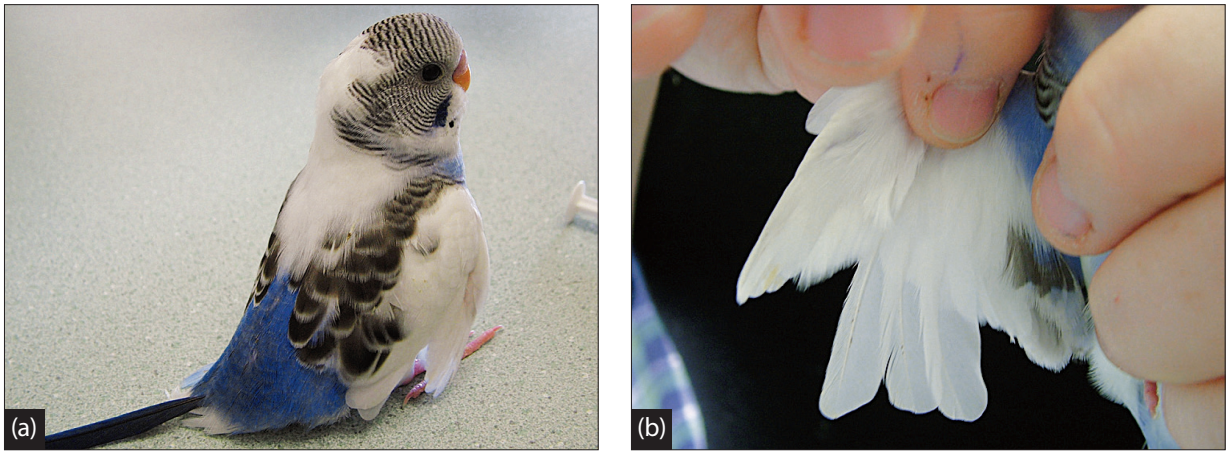


Figure 10.12 **Budgerigar with shortened tail (a) and wing feathers (b) due to polyomavirus infection. The budgerigar fancy call this disease 'French moult' and the birds are known as 'runners' or 'creepers' because of their inability to fly.**

Downloaded From Vet-eBooks.com

Clinical presentation

There are four different syndromes:

- Dry pox: discrete scabby lesions on non-feathered parts of the body (e.g. combs; wattles; beaks; eyelids).
- Wet pox: fibronecrotic diphtheritic lesions in the oropharynx.
- Septicaemic form in canaries.
- Viral-induced neoplastic lesions in the skin and lungs.

The presenting syndrome is determined by the strain of the virus, the mode of transmission and the age, species and health of the infected bird. Lesions may become infected and painful, and can interfere with eating, respiration and vision.

Diagnosis

Intracytoplasmic inclusion bodies, known as Bollinger bodies, are considered pathognomonic. Virus isolation and PCR can be utilised.

Management

Most localised lesions are self-limiting, healing in 3–4 weeks (although diphtheritic lesions may persist for several months) and producing immunity for 6–12 months or longer. If lesions are infected, antibiotics and gentle cleansing are indicated. Forceful removal of scabs may result in scarring and deformity.

Control revolves around isolation of affected birds, minimizing fighting and preventing access of biting insects. Sodium hypochlorite is an effective disinfectant. Attenuated live vaccines are available for pigeons and poultry. The fowl pox vaccine is effective in ostriches. There is also a canary pox vaccine available.

Papillomavirus

Aetiology

Papillomavirus is a double-stranded non-enveloped DNA virus.

Clinical presentation

Cutaneous papillomas (small fleshy pedunculated masses originating primarily from featherless areas such as the feet and face) are seen in African grey parrots, canaries and finches. Their effects are usually simply mechanical, and so the clinical signs are determined by their location.

This is a different disease syndrome from internal papilloma disease (IPD) seen in many parrots (see Chapter 16, Diseases of the Gastrointestinal Tract p. 247).

Diagnosis

Koilocytosis (a large, dense, cytoplasmic perinuclear space with an irregular edge) is the hallmark of papillomaviruses. Virus isolation is the gold standard, but papillomavirus viral particles may be rare or absent. Western blot immunoassay and immunohistology are ideal along with standard histology.

Management

Radiosurgery, cryosurgery and laser surgery have all been used to remove the papillomas and, possibly, stimulate an immune response to delay or prevent recurrence.

PARASITIC INFECTIONS

Mites

Mites are the most important and commonly encountered external parasite of birds. Adult mites have eight legs; the larval stage has six legs.

Cnemidocoptes pilae

See **Fig. 10.13**. Further details of this condition are in Chapter 11, Diseases of the Beak and Cere p. 192.

Dermanyssus gallinae (red or roost mites)

This mite lives in the environment, where it can survive long periods without feeding. It takes blood meals at night and leaves the bird in the day. It causes skin irritation, scaliness and anaemia (especially in juveniles or small birds). Heavy infestations may kill chicks in the nest box.

Ornithonyssus spp. (fowl mite)

These host-dependent, blood-sucking mites remain on the bird, where they congregate at the eyelids and vent. They can cause anaemia, pruritis and

Downloaded From Vet-eBooks.com



Figure 10.13 **Budgerigar with a *Cnemidocoptes* infestation.**

damaged plumage. They may also transmit *Borrelia* and *Lankesterella*.

Epidermoptic mites

This group includes *Myialges* spp., *Epidermotes* spp. and *Microlichus* spp. They may be carried mechanically by the hippoboscid fly. They cause depluming dermatitis with scale formation in passerines.

Quill mites

This group includes *Syringophilus* spp., *Dermoglyphus* spp., *Pterolichus* spp., *Analges* spp. and *Harporhynchus* spp. (**Fig. 10.14**). Their entire life-cycle is spent on the host. They can be found by examining the pulp material within a developing or damaged feather. They cause partial or complete loss of the feather.

Lice

Definition/overview

Lice are six-legged, wingless, crawling external parasites with chewing mouthparts. Sucking lice do not



Figure 10.14 **In lorikeets and other species, the mite *Harporynchus* causes nodular skin lesions.**

infect birds. They are species specific. They may serve as a vector for some contagious diseases (e.g. eastern equine encephalitis).

Species

There are many hundreds of species of lice found on birds. Just a few examples include:

- Biting lice of parrots include *Neopsittaconirmus*, *Psittaconirmus*, *Eomenopon* and *Pacifimenopon* species.
- Biting lice of pigeons include the Philopteridae and Menoponidae families.
- Biting lice of raptors include *Mallophaga* spp.

Life cycle

Lice usually live on just one host. Eggs are glued to feather shafts and hatch in 4–7 days. Nymphs go through three moults before maturing.

Clinical presentation

Lice feed on skin scales, feather debris and hair, causing pruritus and poor feather quality.

Fleas

The stickfast flea, *Echidnophaga gallinacea*, is a blood-sucking ectoparasite. It occasionally attaches to the skin of the head of non-poultry birds and causes irritation and blood loss, resulting in depression, anaemia and pruritis on the skin of the head.

Downloaded From Vet-eBooks.com

Flies

Flies that cause problems with aviary and companion birds include *Pseudolynchia canariensis* (the pigeon fly) and the hippoboscid fly (louse or parrot fly). Blood-sucking flies are found on many species of birds. They seldom leave the host, but are not host specific. They lay their eggs off the host, in a secluded spot. They also act as vectors of *Haemoproteus*.

Ticks

Argas persicus, the fowl tick, is a soft-bodied tick that feeds for 3–7 days on the bird before dropping off. It normally lives in the environment. *Ixodes holocyclus and I. cornuatus*, are hard-bodied ticks that cause ascending paralysis.

Management

Sprays and washes can be effective. Natural and synthetic pyrethrins are effective and generally safe. Organophosphate washes are not recommended because of the high incidence of adverse effects. Ivermectin and moxidectin are effective against blood-sucking and keratin-feeding parasites.

NEOPLASTIC AND PSEUDONEOPLASTIC CONDITIONS

Lipomas

Incidence

Lipomas can be seen in any bird, but are most common in Amazon parrots, budgerigars and galahs. They usually occur in older birds and there does not appear to be a sex predilection. Lipomas are associated with an exclusively, or at least predominantly, all-seed diet, combined with a sedentary life style.

Clinical presentation

Masses are usually located along the main fat deposits on the body (around the crop, the ventral abdomen, thighs and down the legs). They are occasionally found around the cloaca. They are rarely seen on the back or the wings. They are firm masses, often embedded in subcutaneous fat, with the overlying skin often yellow in colour (**Fig. 10.15**). The masses may be mobile. Birds are occasionally presented for bleeding due to the lipoma ulcerating.



Figure 10.15 **Galah with an ulcerated lipoma in the inguinal area.**

Diagnosis

Diagnosis is based on the history and clinical appearance. Lipomas must be distinguished from hernias and xanthomas. Radiography can be useful for differentiating lipomas and hernias.

Management

Lipomas are often an indication of extensive underlying disease processes including cardiovascular disease, hepatic lipidosis, hypothyroidism and, occasionally, diabetes mellitus. Before starting any treatment it is important that this information is communicated to the owner and the patient's health (as a whole) is determined.

Surgical excision, while curative, is often not the first treatment of choice. The author's preference is to only excise ulcerating lipomas or those that are mechanically interfering with the bird's lifestyle (e.g. interfering with the normal range of motion of the leg). If surgery is decided upon, great care must be taken to monitor for coagulopathies, often not apparent until the post-operative period. In the case of ulcerating lipomas, where surgery is required urgently, fresh whole blood transfusions may be required during and after the procedure (see Chapter 8, Supportive Therapy).

If the lipoma is not ulcerating, the bird should be converted to a formulated diet and have its weight monitored. In many cases the lipoma will shrink to a size where it is either no longer clinically significant

Downloaded From Vet-eBooks.com

(and therefore no further treatment is required) or, if surgery is indicated, it becomes a more simple procedure with fewer post-operative complications.

Some clinicians report that treatment with L-levothyroxine is beneficial in shrinking lipomas. If the decision is made to use this therapy, care must be taken to monitor for adverse side-effects such as tachycardia and a hyper-metabolic state.

Neoplasia of the uropygial gland

Incidence

These tumours are seen in those parrots that possess an uropygial gland (see Chapter 1, Clinical Anatomy and Physiology p. 1). They are most commonly seen in budgerigars. Most tumours are adenomas, carcinomas and squamous cell carcinomas.

Clinical presentation

There is abnormal swelling of the uropygial gland. It will occasionally present as an ulcerated mass with bleeding from the dorsal tail area.

Diagnosis

Tumours must be distinguished from adenitis, hypovitaminosis A with glandular metaplasia and hyperkeratosis, abscessation and impactions. Biopsy is the best means of achieving a diagnosis.

Management

Surgical excision can be achieved in the early stages, but these masses are often very vascular and extensive by the time they are noticed by the owner. Chemotherapy appears to have little success in treating this condition. Irradiation with Strontium-90 has shown promise as an effective therapy. Surgical debulking may be necessary in some cases. Response time after therapy is directly related to the mitotic activity of the neoplasm (the higher the mitotic rate, the faster the response time).

Other neoplasms

Fibromas/fibrosarcomas can occur anywhere on the body. They may be subcutaneous or proliferative with a nodular, red appearance. They tend to be locally invasive and recurrent (**Fig. 10.16**). Surgical excision has been followed by both radiation and chemotherapy with some success.

Squamous cell carcinomas also may occur anywhere on the body, being most prevalent at mucocutaneous junctions of the head, on the distal wing and on the phalanges. They also tend to be locally invasive, and radiation therapy has been attempted with some success.

Soft tissue sarcomas, including haemangiomas and haemangiosarcomas, have been reported in several locations in birds.

Lymphosarcoma (**Fig. 10.17**) will occasionally present as hyperkeratotic, alopecic areas of skin, often on the head, or nodular dermal masses. Chemotherapy has been used successfully in some cases.



Figure 10.16 **Budgerigar with a fibroma/fibrosarcoma on the wing.**



Figure 10.17 **Budgerigar with an epitheliotropic lymphosarcoma on the crown of the head.**

Downloaded From Vet-eBooks.com

Xanthomas

Incidence

Xanthomas are locally invasive benign masses consisting of foamy macrophages, multinucleated giant cells, and cholesterol crystals. They can be seen in any bird at any age. They are considered by some authors to be a metabolic problem (hypercholesterolaemia) combined with an inflammatory response following localised trauma and haemorrhage with resultant deposition of cholesterol, triggering an inflammatory response.

Clinical presentation

Xanthomas are generally friable, yellow-coloured, fatty-appearing masses that may be located anywhere on the body, but are often seen on the distal wing, on the ventral abdomen and on the sternum. Rarely, they can be seen in internal organs such as the bones, brain and liver. Lesions may be pruritic and are often associated with other pathology such as lipomas, hernias, and sites of chronic irritation (e.g. wing tip trauma). Occasionally they will bleed due to repeated self-trauma.

Diagnosis

Diagnosis is made on the physical appearance of the mass and confirmed by biopsy. Histopathology reveals masses of foamy macrophages and multinucleated giant cells associated with cholesterol clefts. Many birds will have a concurrent hypercholesterolaemia, although this is not consistent.

Management

Surgical excision is often difficult or not feasible due to the widespread nature of the lesion, the friability of the tissue and overlying skin and the vascularity of the lesion, which requires good haemostasis. Severe painful xanthomatosis on the distal wings may require amputation.

Medical therapy involves treating the underlying causes of the hypercholesterolaemia (e.g. diet, hepatopathy, hypothyroidism), NSAIDs (e.g. meloxicam) to control the inflammatory response and prevention of further trauma to the area. Some may respond to thyroxine supplementation.

TOXIC CONDITIONS

Fenbendazole can damage feathers if given during new feather growth. The feathers that develop during this period appear dysplastic and brittle, breaking easily.

Silver sulphadiazine, used as a topical antibacterial cream, has been reported to cause agyria (e.g. silver poisoning) after excessively prolonged application. It causes a bluish discoloration of the skin, feathers and mucous membranes.

CONDITIONS AFFECTING THE UROPYGIAL GLAND

Impaction

This is associated with hypovitaminosis A, infection, neoplasia or trauma. The gland becomes distended with a caseous-like secretion. Hot compresses and gentle expression of the gland can temporarily relieve the impaction, but underlying causes need to be addressed.

Infection and abscesses

Untreated impactions can become infected and go on to form abscesses. These usually are painful and erythematous. Surgical ablation of the gland may be required if the abscess fails to respond to debridement, flushing, antibiotics and NSAIDs.

Neoplasia

See above under Neoplastic and pseudoneoplastic conditions.

IATROGENIC TRAUMA

The most common iatrogenic trauma seen in companion birds is that resulting from an excessive wing trim. One-winged trims and/or trimming secondary and tertiary flight feathers reduce the bird's ability to slow while in flight and control its descent. The result is a heavy fall. Repeated (or even single) heavy falls can result in broken 'blood feathers' (newly erupted feathers with a rich blood supply in the shaft) (**Fig. 10.18**), bruising to the cranial sternum that leads the bird to mutilate the area, often resulting in an ulceration in the skin and muscle, sometimes

Downloaded From Vet-eBooks.com



Figure 10.18 **Princess parrot with severely traumatised wing tip resulting from the bird suffering repeated heavy falls.**



Figure 10.19 **Princess parrot with sternal and wing tip trauma resulting from heavy falls.**

with an underlying osteomyelitis (**Fig. 10.19**), and tail splits (splits in the skin between the vent and the tail [see **Fig. 10.4**]).

Treatment must be focused on the existing trauma whilst preventing further trauma. Badly broken feathers may need to be removed. (If more than one feather needs to be removed, general anaesthesia and analgesia are appropriate.) If the feather(s) are not badly broken, it may be possible to leave them *in situ* if the haemorrhage has stopped. Sternal trauma, if mild, may respond to antibiotic and analgesic therapy while preventing the bird from further damaging the area (Elizabethan collar ± a neck brace). If osteomyelitis is present or the ulceration is deep, surgical debridement may be necessary. Tail splits can be debrided and sutured.

Further trauma can be reduced by restoring flight. This can be achieved by 'imping' new primary feathers onto the stumps of those that have been trimmed and/or removing the stumps of trimmed feathers (under anaesthesia) to encourage the regrowth of new feathers. The bird should be confined in a large cage and not allowed free flight until it is able to fly safely. The perches in the cage should be lowered and the floor padded with soft towels to prevent heavy falls and hard landings within the cage.

FEATHER DAMAGING BEHAVIOUR AND OTHER SELF-MUTILATING CONDITIONS

Polyfolliculosis/polyfolliculitis

Definition/overview

This is a chronic pruritic condition that can cause multiple short feathers to erupt from one follicle. It is most common in budgerigars and lovebirds (*Agapornis* spp.).

Aetiology

The aetiology remains unclear, but some feel it may be viral or genetic in origin. There is some discussion as to whether this condition is the cause of the bird's intense pruritus or is the result of damage done by the bird in response to the pruritus.

Clinical presentation

Affected birds are presented for pruritus predominantly over the back and neck. Newly emerging feathers have short, stout quills with retained sheaths. There appear to be several feathers emerging from a single, enlarged follicle. Histologically there is the appearance of multiple short feather shafts growing from the same follicle, with a thin layer of epidermis separating the shafts. Chronic inflammation occurs beneath the pulp cap and the feather sheath is thickened.

Downloaded From Vet-eBooks.com

Management
Surgical excision of affected follicles or feather tracts may be of benefit. NSAIDs such as Meloxicam may be of benefit.

Self-mutilation in *Agapornis* species
Clinical presentation
This is an intensely pruritic self-mutilation problem affecting the shoulder region and prepatagial membrane. Less commonly it can involve the inguinal region, chest, back, base of tail and around the cloaca. It can be unilateral or bilateral. Polyfolliculosis is found in some cases.

Long-term scarring of the prepatagial membrane often restricts the bird's ability to fly, and often the affected area cracks and bleeds when the bird stretches its wings.

Management
Treatment is often unrewarding. Placing an Elizabethan collar until the skin wounds have healed can be of benefit, but recurrence once the collar is removed is common. Antibiotics and anti-inflammatory therapies should be given.

Cockatiel feather mutilation syndrome
Definition/overview
This is associated with intestinal giardiasis and has been reported in cockatiels only. It appears to be less common now than when first reported.

Aetiology
It is postulated that giardiasis causes an intestinal malabsorption syndrome, leading to a vitamin E/selenium deficiency which in turn leads to dry, flaky skin that can progress to episodes of feather pulling, alopecia and pruritus.

Clinical presentation
Dry, flaky skin that can progress to episodes of feather pulling, alopecia and pruritus. Other clinical signs include weight loss, depression, ruffled feathers, chronic diarrhoea, neonatal mortality, cachexia and weakness.

Diagnosis
Eosinophilia and hypoproteinaemia are commonly reported; the eosinophilia is most likely to be due to the tissue damage associated with feather damaging behaviour. Detection of the trophozoites (on very fresh faecal smears suspended in normal saline) and cysts (using centrifuged zinc sulphate flotation) is very suggestive that this syndrome is present.

Management
Treatment with nitroimidazoles is usually effective; a rapid response is usually observed.

Quaker mutilation syndrome
Aetiology
The aetiology is unclear. There is no histopathologic evidence of a pathogen. Insufficient cases have been fully worked up to determine a pattern.

Clinical presentation
There is acute onset of severe self-induced skin trauma, often directed at the neck and sternum and usually unrelated to prior episodes of feather-damaging behaviour (**Fig. 10.20**).

Management
Treatment is directed at preventing further trauma through the use of neck braces and/or Elizabethan collars. Analgesia is given through the use of NSAIDs and antibiotic coverage is provided.



Figure 10.20 **Quaker parrot suffering from Quaker self-mutilation syndrome. Note the severe self-inflicted trauma to the inguinal region.**

Downloaded From Vet-eBooks.com

Euthanasia is often requested because of the severity and recurrence of the trauma.

Feather damaging behaviour directed towards other birds

Towards nestlings

Some parents will start to pluck feathers from their nestlings while they are still in the nest box. Mild cases involve removal of feathers, usually from the back, dorsal wings and head (**Fig. 10.21**). Severe cases will see the parent(s) remove wing tips or toes, or even kill the nestling.

Two theories exist as to why this behaviour occurs:

- A learnt behaviour: feather pluckers were often feather plucked themselves as chicks.
- A desire to remove the nestlings so as to prepare the nest box for another clutch of eggs.

Affected chicks are best removed for hand rearing. They should not be used for breeding if possible.

Towards a bonded mate

Some individuals will pluck feathers from their mate, often at the beginning of the feather season. Both hens and cocks may do the plucking. Usually it is the feathers on the head and face that are plucked (**Fig. 10.22**). It is unusual to see this plucking extend to traumatic mutilation. Many of these pairs will breed successfully. They should be separated if mutilation is likely to result in injury.

Towards cage mates

In overcrowded or otherwise stressful living conditions some birds (particularly finches, budgerigars, *Agapornis* spp., *Neophema* spp. and gallinaceous birds) will pluck the head and back of their cage mates. Occasionally this behaviour can lead to cannibalism and death. Some birds will also chew and pluck flight and tail feathers of cage mates, even in good living conditions.

Affected birds and, if possible, the offending bird(s) should be removed. Stocking density and stress factors need to be addressed and corrected. Foraging activities and toys should be provided for birds that appear to be indulging in this behaviour due to a lack of environmental enrichment.



Figure 10.21 Cockatiel chick which has been feather-picked by its parents.



Figure 10.22 Sun conure feather-picked by her mate. Note that not all feather-picking is self-inflicted.

Downloaded From Vet-eBooks.com

Feather damaging behaviour directed towards the bird itself

Definition/overview

This is one of the most common and most frustrating conditions that veterinarians can be presented with. The causes for this condition are often multifactorial and cascading in their effects. By the time the bird is presented to the clinician the original inciting cause may have disappeared or been obscured by other complicating or reinforcing factors.

It must be stressed to the bird's owner that a successful outcome may simply be a reduction, rather than elimination, of the activity. In other words, a feather picker will nearly always remain a feather picker, but may pick less.

Aetiology

Feather damaging behaviour may be due to either physical or behavioural problems. It is simplistic and inaccurate to diagnose 'boredom' or 'fear/anxiety' without a thorough investigation to rule out physical problems first (compare **Figs 10.23** and **10.24**). It is also simplistic to suggest that a single aetiology exists for each individual case. In many cases several factors, both physical and behavioural, have combined to produce the clinical sign of feather damaging behaviour.

Physical causes of feather damaging behaviour:

- Dermatitis: infectious (bacterial, fungal, viral); chemical (e.g. nicotine absorbed from the owner's fingers) or allergic (still unproven, but strong evidence exists for allergic dermatitis to be a factor in some feather-pickers). Allergies are suspected to occur in birds, but a reliable diagnostic test has yet to be developed.
- Folliculitis: bacterial; fungal; viral.
- Malnutrition: an all-seed diet often results in dry, flaky skin that is predisposed to superficial infections, with resultant pruritus.
- Environmental conditions: extremes of humidity, aerosol contamination and cigarette smoke have been associated with feather damaging behaviour.
- Heavy metal toxicoses are often implicated as a cause of feather damaging behaviour. However, all reported cases are anecdotal and there is no reliable evidence that heavy metal toxicosis causes feather damaging behaviour.
- Underlying painful lesions: hepatopathy (e.g. chlamydiosis); osteomyelitis; pancreatic disease; renal disease; neoplasia; underlying abscesses.
- Reproductive activity, perhaps through ovarian and oviductal enlargement and liver changes (vitellogenesis), is believed to cause abdominal discomfort that may trigger feather damaging



Figure 10.23 **Cockatiel with pruritus. Care is needed to differentiate pruritus from psychological feather damaging behaviour.**



Figure 10.24 **Cockatiel who has been feather-picking. Note the untouched head and neck feathers, evidence that the feather picking on the torso is self-inflicted.**

Downloaded From Vet-eBooks.com

behaviour of the thighs and ventral abdomen of some reproductively active hens.
- Parasites: external (mites, lice) and internal (*Giardia*) are often over-diagnosed as a cause of feather damaging behaviour (**Fig. 10.25**). Pet shop employees, in particular, often advise bird owners to treat feather damaging behaviour birds for lice. *Giardia* has **only** been associated with feather damaging behaviour in cockatiels, yet many case studies report *Giardia* testing in non-cockatiel species.

Psychological causes of feather damaging behaviour:
- Attention seeking behaviour: this occurs when the bird uses feather damaging behaviour to obtain attention from an owner when that owner is not engaged in an activity with the bird.
- Anxiety: a state of apprehension, uncertainty and fear resulting from the anticipation of a realistic or fantasised threatening event or situation, often impairing physical and psychological functioning. Anxiety disorders may have biological and/or environmental causes. Improper socialisation of companion parrots during the hand-rearing process may be reflected in an inability of such birds to cope with otherwise normal situations due to fear, real or imagined, of a person or situation that the bird is exposed to combined with a 'lack of self-confidence' due to a history of undermining events. An example of this is severe wing clips in juvenile birds that are learning to fly. The resultant heavy falls to the ground may trigger feather damaging behaviour. Separation anxiety is another disorder characterised by behavioural signs of distress (screaming, feather disruptive behaviour) that occur when the bird is left alone. These are a distress response to separation from the person or persons to whom the bird is attached. This form of anxiety often results in birds with inadequate social skills due to a failure to teach the bird coping behaviours whilst being hand reared or weaned.
- Boredom: parrots are intelligent animals who need to be kept occupied. As a general rule, wild birds spend up to 80% of daylight hours foraging for, and consuming, food. The other 20% of the day is spent socialising and grooming, and occasionally sleeping. For captive birds, with food provided in a dish every day, this situation can be reversed (i.e. only 20% of the day is spent foraging and eating food, the other 80% is spent socialising and grooming). When socialising opportunities are limited (e.g. the lone bird in a household where everyone is at work during the day), over-grooming may occur and feather damaging behaviour results.
- Compulsive disorders are characterised by repetitive, stereotypic motor, locomotor, grooming, ingestive or hallucinogenic behaviours that occur out of context, or at a frequency or for a duration that is in excess to that required to achieve a goal. They interfere with the bird's ability to function normally in its social environment. An example is compulsive grooming (i.e. grooming in excess of that required for its purpose and which interferes with normal behaviour). It cannot be interrupted. (This must be distinguished from excessive grooming, which is unrelated to hygienic or maintenance requirements and is more frequent or intensive than previously exhibited. However, unlike compulsive grooming, it can be interrupted.)



Figure 10.25 **Feather lice as seen here are often erroneously stated as the cause of feather damaging behaviour.**

Downloaded From Vet-eBooks.com

- Displacement behaviours secondary to stressors: examples include unwanted exposure or contact with people may lead to feather picking. Such behaviour may be accompanied by aggression and fearfulness of humans.

Diagnosis

A thorough history of both the bird and the problem is required. In particular, this history needs to focus heavily on the bird's interaction with its environment and the people (and other animals) around it. A detailed physical examination should be performed and a minimum database on the bird's health status gathered (haematology and clinical biochemistries, whole body radiographs). Based on this physical examination and clinical database, the following additional diagnostic tests may be warranted:

- Skin biopsy.
- Pathogen detection through culture or PCR.
- Endoscopy.

If a physical cause is unable to be determined and the patient's history supports it, a diagnosis of behavioural feather damaging can then be made (see *Table 10.1*). At this stage every effort should be made to incorporate the services of a veterinary behaviourist or a suitably qualified parrot-behaviour consultant.

Management

Elizabethan collars are rarely indicated unless self-mutilation and/or physical trauma is occurring. In many cases Elizabethan collars and other restraints may worsen the patient's anxiety state and aggravate the original problem.

Psychotropic drugs may be of benefit as a short-term therapy to break a feather damaging behaviour 'cycle', but are rarely, if ever, warranted as a long-term solution. They can be used to suppress, modify or change unwanted behaviour, but should be used in conjunction with behavioural modification. Most exert their effect by enhancing or inhibiting the effects of excitatory or inhibitory neurotransmitters. Examples of drugs that may have a role in short-term therapy include:

- Benzodiazepines (e.g. diazepam) inhibit dopamine and potentiate gamma-amino butyric acid (GABA). They are also muscle relaxants, and can interfere with learning.

Table 10.1 **Diagnosis of feather damaging behaviour**

TIMING AND NATURE OF FEATHER DAMAGING BEHAVIOUR	DIFFERENTIAL DIAGNOSES	REINFORCING FACTORS
Occurs when owner is not present	Separation anxiety; boredom	Habit; lack of environmental enrichment/foraging activities
Occurs when owner is present but not paying attention to the bird	Attention seeking behaviour	Owner's behaviour (drama, attention); habit
Bird interrupts other behaviour to damage feathers	Obsessive/compulsive disorder; true pruritus	Owner's behaviour (drama, attention); habit
As well as the feather damaging behaviour, the bird exhibits signs of unwarranted fear, anxiety or stress	Generalised anxiety disorders	Owner's behaviour (avoidance); habit; major changes in household
Problem starts at an extremely young age; handfed bird	Improper preening; poor early socialisation	Owner's behaviour (drama, attention); habit
Involves primarily remiges and retrices. The feathers are frayed and splintered	Anxiety disorders. Iatrogenic: improper wing trim, feather trauma due to small cage	Owner's behaviour (drama, attention); habit
Overly bonded, sexually mature bird that displays sexual behaviours out of context	Reproductively related	Owner's behaviour (drama, attention); habit

Downloaded From Vet-eBooks.com

- Phenothiazines (e.g. acepromazine) are dopamine antagonists. They are rarely used in birds.
- Butyrophenones (e.g. haloperidol) inhibit dopamine and are used for self-mutilation and feather damaging behaviour.
- Antihistamines (e.g. diphenhydramine) inhibit histamine receptors, producing sedation.
- Progestins (e.g. medroxyprogesterone acetate) potentiate GABA; they have a calming and an anti-inflammatory effect. However, they have multiple side-effects including obesity, hepatopathy and diabetes mellitus.
- Tricyclic antidepressants (e.g. clomipramine, doxepin and amitriptyline) potentiate serotonin, producing sedation and anticholinergic activity. They can be used to alleviate anxiety and depression. They must be used with care because of side-effects including constipation and arrhythmias.
- Serotonin specific re-uptake inhibitors (e.g. fluoxetine) have serotonergic effects.
- Narcotic antagonists and agonist/antagonists (e.g. naltrexone) act at the opiate centres in the brain, blocking endorphin response to self-injurious behaviour. This may be useful in some birds.

As mentioned earlier, psychotropic drugs, if used at all, should be employed as a short-term solution. Behavioural feather damaging is a behavioural problem and the real solutions lie in behavioural modification. It must be ensured that stability and security are present in the bird's lifestyle at home. Anxiety disorders arising from, for example, fear of human interaction or fear of falling due to an inappropriate wing trim can lead to feather damaging behaviours. These types of factors should be identified and eliminated if present. Triggering events that may have had a role in the development of feather damaging behaviour, such as changes in the household or a perceived lack of attention, should be identified. Techniques can be used to teach the bird to accept these triggering environmental events if, indeed, it is appropriate for the bird to accept them.

Basic training is implemented and strengthened using a system of positive reinforcement. Once this has been achieved, training can be extended to guide more normal behaviours. The '80–20 rule' is important: 80% of the bird's day should be spent foraging for food, the remaining 20% on grooming and socialising activities. In practical terms, for a companion parrot, this can be done by:

- Providing foraging activities both with and without the owner being present. The bird may need to be taught how to participate in these activities, as at first it might be afraid of new objects or activities.
- Enhancing 'normal' feather care through gentle misting with water and by providing other items that can be groomed in addition to the bird's feathers.
- Developing more normal social interactions between the bird and its owners. The aim is to provide social interaction that is engaging, stimulating and changing for the bird. If there is a 'one-person bond' present between the bird and an owner this must be modified into a more normal flock and social interaction. It must be remembered, though, that it is essential to replace unwanted behaviours with more desirable behaviours; at no time should a behavioural void develop through removing an activity or interaction without replacing it with others.

The goal is to decrease the feather damaging behaviour; it is unlikely that a permanent cure will be achieved. Regular communication and follow-up evaluations are essential to monitor changes in the bird's behaviour and environment. If the result is a healthy, well socialised bird that actively engages its environment and its human companions, the state of its plumage becomes less significant.

FURTHER READING

Briscoe JA, Reisner IR, Rosenthal KL (2004) Incorporating the veterinary behaviourist: a new model for the diagnosis and treatment of a feather damaging pet parrot. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 293–296.

Downloaded From Vet-eBooks.com

Chitty J (2005) Feather pulpitis in plucking parrots. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 259–265.

Clubb SL (2006) Clinical management of feather damaging behaviour associated with inflammatory skin disease in parrots. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 73–78.

Clubb SL, Buerkle M, Crosta L, Ciembor PG, Latima KS, Garner MM, Ritchie BW (2004) Feather damaging behaviour in Lories and its association with psittacine circovirus. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 319–320.

Clubb SL, Elmo N, Buerkle M, Crosta L, Enders F (2004) Incidence and characterisation of feather damaging behaviour in a large parrot collection. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 321–332.

Clubb SL, Garner MM, Cray C, Goodman M (2004) Diagnostic assessment of feather picking behaviour in African Grey Parrots (*Psittacus erithacus*). In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 313–318.

Danylyk I, Oldfield T, Raidal SR. (2010) Evaluation and Assessment of Hypothyroidism in Psittacine Birds. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians and Unusual and Exotic Pet Veterinary special interest group*, pp. 111–118.

Ferrell ST (2002) Avian integumentary surgery. *Seminars in Avian and Exotic Pet Medicine* **11**(**3**):125–135.

Garner MM (2006) Inflammatory skin disease in feather picking birds: histopathology and species predispositions. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 17–20.

Gartrell BD, Rogers L, Alley MR (2005) Eosinophilic dermatitis associated with *Trichophyton asahii* in a cockatiel (*Nymphicus hollandicus*). *Journal of Avian Medicine and Surgery* **19**(**1**):25–29.

Gill JH (2002) Avian skin diseases. *Veterinary Clinics of North America: Exotic Animal Practice* **4**(**2**):463–492.

Greenacre CB (2005) Viral diseases of companion birds. *Veterinary Clinics of North America: Exotic Animal Practice* **8**(**1**):85–106.

Hochleithner M, Hochleithner C (1996) Surgical treatment of ulcerative lesions caused by automutilation of the sternum in psittacine birds. *Journal of Avian Medicine and Surgery* **10**(**2**):84–88.

Johnston MS, Preziosi DE, Morris DO, Rosenthal KL, Rankin S (2004) The role of *Malassezia* in feather destructive behaviour in psittacines. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 89–90.

Koski MA (2001) Dermatological diseases in psittacine birds: an investigational approach. *Seminars in Avian and Exotic Pet Medicine* **11**(**3**):104–125.

Lightfoot TL, Nacewiz CL (2006) Psittacine behaviour. In: *Exotic Pet Behaviour*. TB Bays, TL Lightfoot, J Mayer (eds). Saunders Elsevier, St Louis, pp. 51–102.

Martin KM (2004) Behavioural approach to psittacine feather-picking. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 307–312.

Merryman JI, Buckles EL (1998) The avian thyroid gland. Part One: a review of the anatomy and physiology. *Journal of Avian Medicine and Surgery* **12**(**4**):234–237.

Merryman JI, Buckles EL (1998) The avian thyroid gland. Part Two: a review of function and pathophysiology. *Journal of Avian Medicine and Surgery* **12**(**4**):238–242.

Nemetz LP (2004) Strontium-90 therapy for uropygial neoplasia. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 15–20.

Phalen DN (2006) Implications of viruses in clinical disorders. In: *Clinical Avian Medicine*, *Vol 2*. GH Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 721–746.

Powers LV, Van Sant F (2006) Axillary and patagial dermatitis in African Grey Parrots (*Psittacus erithicus*). In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 101–105.

Raidal SR (1995) Viral skin diseases of birds. *Seminars in Avian and Exotic Pet Medicine* (**4**):77–82.

Ritchie BW (1995) Papoviridae. In: *Avian Viruses: Function and Control*. BW Ritchie (ed). Wingers Publishing, Lake Worth, pp. 127–170.

Ritchie BW (1995) Circoviridae. In: *Avian Viruses: Function and Control*. BW Ritchie (ed). Wingers Publishing, Lake Worth, pp. 223–252.

Ritchie BW (1995) Poxviridae. In: *Avian Viruses: Function and Control*. BW Ritchie (ed). Wingers Publishing, Lake Worth, pp. 285–312.

Rosenthal KL, Morris DO, Mauldin ES, Ivey ES, Peikes H (2004) Cytologic, histologic and microbiologic characterization of the feather pulp and follicles of feather-picking psittacine birds. *Journal of Avian Medicine and Surgery* **18**(**3**):137–143.

Schmidt RE (2002) Avian thyroid metabolism and diseases. *Seminars in Avian and Exotic Pet Medicine* **11**(**2**):80–83.

Schmidt RE, Lightfoot TL (2006) Integument. In: *Clinical Avian Medicine*, *Vol 2*. GH Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 395–410.

Downloaded From Vet-eBooks.com

Schmidt RE, Reavill DR, Phalen DN (2003) Integument. In: *Pathology of Pet and Aviary Birds*. RE Schmidt, DR Reavill, DN Phalen (eds). Iowa State Press, Ames, pp. 177–196.

Seibert LM (2006) Feather-picking disorder in pet birds. In: *Manual of Parrot Behaviour*. AU Luescher (ed). Blackwell Publishing, Oxford, pp. 255–266.

Tully TN, Foil CS, Nett-Mettler C, Columbini-Osborn S, Heatley JJ, Hosgood G (2006) Status of intradermal skin testing in avian species. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 33–37.

Welle K (2005) Clinical approach to feather picking disorders in pet birds. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 83–87.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 11

DISORDERS OF THE BEAK AND CERE

MALFORMATION

Definition/overview

Malformation of the beak may be congenital or acquired. Congenital conditions include lateral deviations of the maxilla (wry/scissor beak) (**Fig. 11.1**) and mandibular prognathism (**Figs 11.2, 11.3**). Acquired conditions include lateral deviations of the maxilla (wry/scissor beak), compression deformities of the mandible or maxilla (**Fig. 11.4**) and other malformations, often associated with a 'softening' of the beak due to malnutrition.

Aetiology

Congenital malformations are thought to be associated with incorrect incubation parameters (temperature, humidity, ventilation or turning of the egg) or parental nutrition. Acquired malformations are thought to be due to damage to all, or part of, the germinal epithelium of the beak or to the poorly calcified beaks of neonates.

A variant of scissor beak is sometimes seen when the pre-frontal bone of the skull has collapsed on one side, turning the whole beak to that side (**Fig. 11.5**).

Clinical presentation

In lateral deviations of the maxilla (wry/scissor beak), the maxillary beak is deviated laterally from the level of the cere or beak tip. The side of the mandible no longer in wear and the maxilla often overgrows. This condition is most common in macaws up to two months of age, but acquired scissor beak can occur in any species at any age.

Mandibular prognathism is most common in cockatoos aged 1–2 weeks. The maxilla sits inside the mandible.

Compression deformities of the mandible are most commonly seen in macaws up to five weeks of age. Other malformations are often associated with sinusitis, *Cnemidocoptes* infection, trauma and PBFD.



Figure 11.1 Juvenile blue and gold macaw with congenital scissor beak.



Figure 11.2 Eastern rosella with congenital prognathism.

Downloaded From Vet-eBooks.com



Figure 11.3 Adult quaker parrot with acquired prognathism.



Figure 11.4 Juvenile blue and gold macaw with lateral beak compression due to excessive holding pressure whilst hand-feeding.

Management

Conservative treatment may be effective in very young chicks. Applying gentle digital pressure for ten minutes two to three times daily may straighten a deviated maxilla or lift the maxilla up and forward in prognathic chicks. Cases that do not respond to this treatment, or older birds with calcified beaks, will need more aggressive therapy.



Figure 11.5 Juvenile Amazon parrot with congenital scissor beak due to collapse of the frontal bone on one side.

Deviated maxilla: Beak trimming, involving grinding the overgrown gnathotheca on the side contralateral to the maxillary deviation and the overgrown tomia on the maxilla, allows the maxilla to move back into a normal position. At the same time the occlusal ledge inside the maxilla has to be reshaped so that it is perpendicular to the lateral walls of the rhinotheca. The bird should then be encouraged to chew hard objects (e.g. branches) frequently and the beak reshaped every two weeks until normal.

Acrylic ramps or prostheses can be used to augment the trimming described above. An acrylic ramp is built up on the gnathotheca on the same side as the maxillary deviation, forcing the maxilla into a normal position. The ramp needs to be high enough so that the bird cannot open its mouth wide enough to get its maxilla over the top of the ramp. This technique frequently fails due to damage to the gnathotheca during the attachment of the ramp.

Trans-sinus pinning is an 'orthodontic' procedure, designed to provide constant lateral tension on the deviated maxilla to guide it into a more anatomically normal position (**Fig. 11.6**). A K-wire or small Steinmann pin is placed perpendicular to the skull, through the frontal sinuses just caudal to the craniofacial hinge joint on a line between the lateral canthus of the eye and the cere. Care must be taken not to position this pin too caudally or too low. In macaws, a small bony protuberance marks the point where the pin is introduced on one side and exits

Downloaded From Vet-eBooks.com



Figure 11.6 **Blue and gold macaw with a deviated maxilla undergoing a trans-sinus pinning procedure.**

on the other. On the side of beak contralateral to the direction of the deviation, the pin is bent 90° as it leaves the skin. The pin should run the length of the maxilla, and the end (level with the beak tip) is curled over on itself. The other end of the pin is trimmed and curled over on itself so that it is flush against the skin and cannot be pulled through the skull.

The tip of the beak is then placed under tension by means of a rubber band around the beak and the distal end of the wire. In older birds this rubber band is replaced by a piece of wire running through a hole in the distal rhinotheca. The tension is maintained until the beak has straightened. In young birds this can be as soon as 10–14 days; older birds may require 10–14 months. The tension can be adjusted by loosening or tightening the wire/rubber band as required.

Once the beak has straightened the wire/rubber band should be removed. If, after several days, the beak has remained in a normal position, the pin can be removed.

Scissor beak associated with pre-frontal bone collapse is not, at this time, amenable to treatment. These birds usually adapt to their disability and do well. Regular beak trimming is often required due to the lack of occlusal wear.

Prognathism: An acrylic prosthesis can be placed over the rhinotheca. One or two small pins can be placed through the rhinotheca to provide 'anchor points' to prevent the prosthesis falling off. The goal is to force hyperextension of the craniofacial hinge joint into a more normal range of motion, allowing for a 'firming' up of the joint so that the upper mandible cannot be over-flexed to the point of mandibular prognathism. There is no correction of 'growth' of the beak, but rather guidance and reduction of the abnormal range of motion of the craniofacial hinge joint. The prosthesis must be long enough to prevent the bird opening its mouth wide enough to place the prosthesis inside the gnathotheca.

A trans-sinus pinning technique can be used in older birds. It requires a pin placed through the frontal sinuses as described above. The ends of the pin are then bent forwards to meet just rostral to the beak tip and are linked to form a triangle. A second pin is placed through the rhinotheca midway along its length, with the triangular trans-sinus pin resting on it. Cerclage wire is then used to link the tip of the beak with the apex of the triangle. This arrangement also extends the craniofacial hinge joint into a more normal position.

Prognosis

Many birds with untreated malformations will adapt to the deformity and learn to eat well. However, they will usually do better if the malformation can be corrected. With appropriate techniques, follow-up and care, the prognosis for most beak malformations is good.

TRAUMA

Definition/overview

Common injuries include puncture wounds of the rhinotheca or gnathotheca (**Fig. 11.7**), avulsion of the rhinotheca or gnathotheca or both (**Figs 11.8a** and **b**), mandibular or maxillary fracture and mandibular symphyseal fracture (**Fig. 11.9**).

Aetiology

Causes include bite wounds from other birds and occasionally from other animals, entrapment in wire or metal objects, particularly in cages, and blunt force trauma resulting from striking objects while in flight (e.g. ceiling fans, walls, windows).

Management

Initial treatment should be aimed at relieving pain, stopping bleeding and controlling infection. Once this

Downloaded From Vet-eBooks.com

initial assessment and treatment are finished, attention needs to be given to providing nutritional and fluid support to the patient. Tube feeding or placing an oesophagostomy tube may be required until the patient is able to eat for itself.

Once the patient is stable, consideration must be given to how to repair the injury, if indeed repair is feasible.

The dermis that produces the rhamphotheca is present at the base of both upper and lower beaks, and also entirely covers the dorsal and lateral surfaces of the maxilla and mandible. The rhamphotheca therefore grows from deep to superficial as well as from caudal to rostral. Any injury to the beak that damages this dermis can result in a loss of keratin. This will usually grow out with time. With severe injuries, where the bone underlying the dermis is crushed or fractured, the loss of bone and dermis usually results in very limited regrowth.

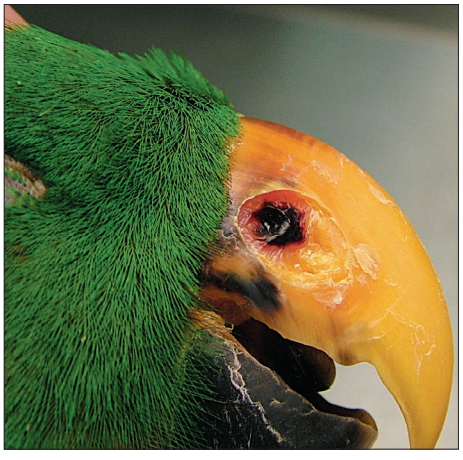


Figure 11.7 **Eclectus parrot with a bite wound (from another bird) on the maxilla.**

Puncture wounds, if superficial, can be treated conservatively by keeping them clean and giving antibiotics and analgesics as appropriate. Deeper wounds that may affect the structural integrity of the beak should be carefully debrided, dried and then 'plugged' with dental acrylic. As granulation tissue fills in the wound, the acrylic plug will be forced out. This may be a matter of days or perhaps weeks. The plug can be replaced until the clinician is assured the beak's integrity is no longer at risk.

Avulsion of all or part of the mandible or maxilla is a common injury. As a general rule, avulsions of less than the rostral third of the maxilla will usually result in regrowth of the avulsed portion. Loss of more than this amount will usually result in permanent deformity (**Fig. 11.10**). Surprisingly, many birds will adapt to this deformity and do well, so long as infection and pain are controlled and the bird is nutritionally supported until the wounds have healed. Overgrowth of the opposing rhinotheca or gnathotheca is common, requiring regular trimming. Beak prostheses rarely work in birds, as the kinetic forces transmitted into the prosthesis by normal beak actions (e.g. eating, gripping objects) rapidly lead to implant failure.

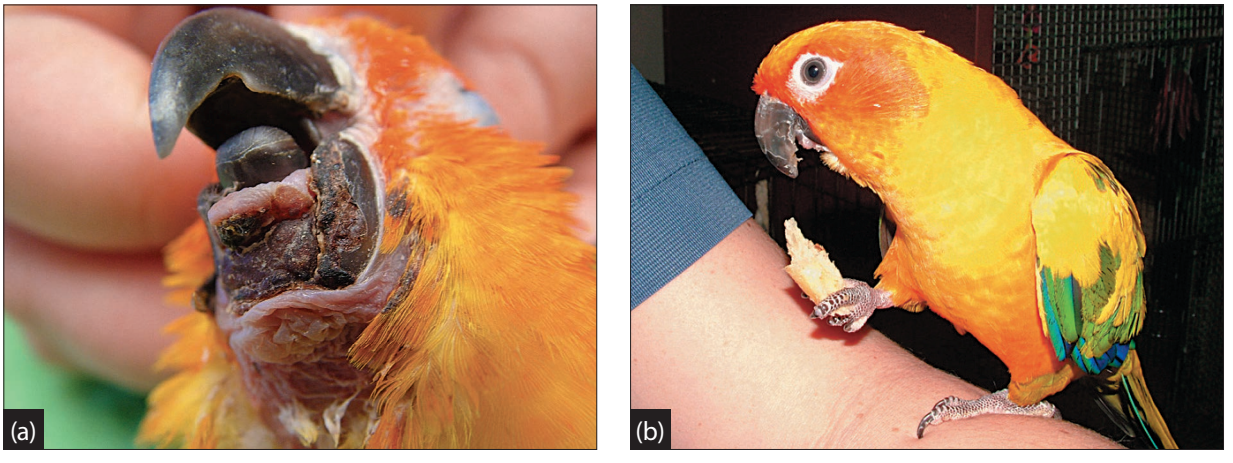


Figure 11.8 **Sun conure with a traumatic avulsion of the mandible (a). Despite loss of the entire lower beak, the bird is still able to eat normally (b).**

Downloaded From Vet-eBooks.com



Figure 11.9 **Sulphur-crested cockatoo with a traumatic split in the gnathotheca. Note that this is a common injury in breeding pairs who fight.**



Figure 11.10 **Superb parrot with avulsion of the majority of the rhinotheca (upper beak). Note that this degree of loss will result in permanent deformity.**

Fractures of the mandible or maxilla are more difficult to handle. With fresh injuries, repair with cerclage wire may be effective in some cases. Splinting the fractured area by taping the beak shut may also be of benefit. Nutritional support via an oesophagostomy tube is obviously essential.

Mandibular symphyseal fractures, if fresh, can be treated by wiring with cerclage wire and sealing the fracture line with tissue glue. Care must be taken not to allow the tissue glue to seep into the fracture line. If non-union results, or if the injury is already several days old when first presented, many of these birds will adapt and be able to eat well. Again, nutritional support may be required via tube feeding or an oesophagostomy tube until the bird is able to eat by itself. Long term regular beak-trimming is usually required.

Prognosis

Most beak injuries have a good prognosis. Despite severe injuries, many birds will adapt and survive quite well.

HYPEREXTENSION OF THE MAXILLA

Definition/overview

This is predominantly a problem in macaws. The maxilla is hyperextended, leaving the beak slightly open. Simple reduction, even under anaesthetic, is usually unsuccessful.

Aetiology

Trauma, particularly biting hard on to a solid object and forcefully hyperextending the maxilla, leads to the palatine bone moving rostrally and then luxating dorsally beyond its normal end-point on the mesethmoidal bony ridge at the ventral base of the braincase. This then engages the infraorbital septum, locking the maxilla into a hyperextended position.

Management

Under anaesthesia a Steinmann pin or closed artery forceps is introduced through a lateral approach through the infraorbital sinus just caudal to the beak commissure, dorsal to the palatine bones. It is inserted to a depth half the width of the skull. This can be done blindly or with the guidance of an endoscope. Several large blood vessels are in this area.

The beak is hyperextended further to 'disengage' the palatine bone from the septum. With the beak still hyperextended, the pin or forceps are used to lever the palatine bone ventrally, disengaging it from the septum and allowing the beak to close. Additional stabilisation can be provided by passing absorbable suture around the suborbital arch and jugal bones bilaterally, or around a transmaxillary pin placed in the proximal central maxilla. The bird is treated with analgesics for 5–7 days and prevented from climbing or biting hard foods for two weeks.

Downloaded From Vet-eBooks.com

Prognosis
The prognosis is usually good.

OVERGROWN MAXILLA

Aetiology
An overgrown maxilla is often associated with chronic liver disease, although the exact pathogenesis is unclear (**Fig. 11.11**). Other possible causes include damage to the germinal epithelial layer, malnutrition, lack of occlusal wear (e.g. scissor beak), lack of opportunity to chew (**Fig. 11.12**), *Cnemidocoptes* and neoplasia. See also **Fig. 11.13**.

Diagnosis
A thorough review of the patient's history, including diet and history of head trauma, is required.

Haematology and biochemistries are carried out to determine health status, with a particular emphasis on assessment of the liver. Radiography or CT imaging of the head may be necessary in some cases.

Treatment
The diet should be corrected and appropriate treatment given for liver disease if present (see Chapter 17, Disorders of the Liver). Corrective trimming is carried out as required and the opportunity for increased chewing behaviour should be provided. Any other causative factors should be identified and remedied.

CNEMIDOCOPTES

Aetiology
The species of mite are *Cnemidocoptes pilae* (parrots and passerine birds) and *Cnemidocoptes mutans*



Figure 11.12 **Eastern rosella with an overgrown rhinotheca. Note the cause was not determined, but may be due to the lack of opportunity to chew on branches, etc.**

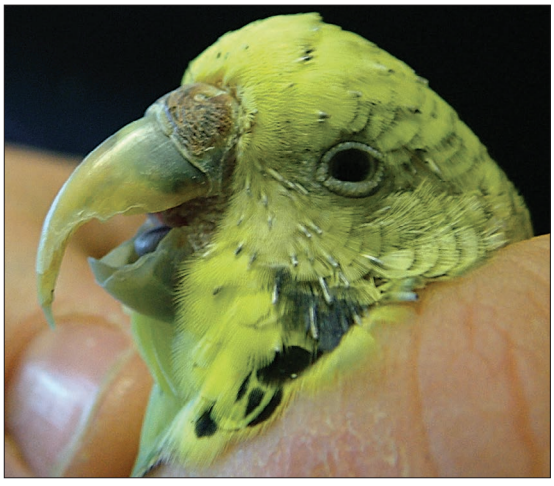


Figure 11.11 **Budgerigar with an overgrown beak, due to chronic hepatopathy after ten years on an all-seed diet.**



Figure 11.13 **Long-billed corella with a normal beak. Note that this should not be confused with an abnormality!**

Downloaded From Vet-eBooks.com

(poultry), also known as scaly-face (or leg) mite. It appears that the mite spends its entire life cycle on its host, although it may be transmitted between birds via dead skin and scales. It is probable that immunosuppression and genetic factors play a role in the development of clinical signs.

Clinical presentation

There is slight variation between affected species:

- Budgerigars, kakarikis, neophemas and *Polytelis* spp. develop proliferative crusty, exuberant growths the beak, cere, face, legs, the margin of the vent and on the wing tips. A characteristic 'honey-combed' appearance is due to the mite tunnelling through the keratin (**Fig. 11.14**). With severe infection, marked beak deformities can occur through damage to the dermis under the rhamphotheca. These include thickening of the beak, overgrowth of all of the beak or overgrowth of part of the beak. Weakening of the keratin can cause part of the beak to crumble and break off.
- Canaries develop 'tassel foot', keratin tags arising from the caudal edge of the podotheca along the tarsometatarsus, giving the appearance of 'tassels'. The scales on the podotheca may also become hyperkeratotic.



Figure 11.14 **Budgerigar with cnemidocoptic mange leading to advanced beak lesions.**

- Poultry also develop hyperkeratosis on their legs, which can become swollen and painful, causing the bird to limp.
- Owls are rarely affected, but if so they develop bilateral, proliferative papillary hyperkeratosis on the feet and legs.

Diagnosis

The classical 'honeycomb' appearance is typical. Scraping the lesion is usually rewarding. The mite can be identified by its dorsal shield and scapular setae.

Management

Ivermectin or moxidectin is used at 0.2 mg/kg topically, orally or parenterally, repeated every two weeks until the crusts disappear (usually three treatments are required). Topical creams and liquids (e.g. mineral oil) are often not effective, as the whole bird must be treated.

Prognosis

The prognosis is good, although beak deformities may become permanent if treatment is left too late.

INFECTION

Bacterial and fungal infections

Aetiology

Bacterial and fungal infections may result from extension of nasal and sinus infections into the bone and keratin of the beak, or they may be the result of trauma (see above). Causative organisms include *Candida*, *Aspergillus*, *Cryptococcus*, *Mycobacterium* and *Pseudomonas* spp. Other bacteria can also be isolated.

Clinical presentation

The beak and nares become distorted. Underrunning of the keratin occurs, which could lead (in severe cases) to sloughing of all, or part of, the rhamphotheca (**Fig. 11.15**).

Diagnosis

Diagnosis is based on clinical signs, transillumination of the beak to determine the depth and extent of the lesion, and biopsy and culture.

Downloaded From Vet-eBooks.com



Figure 11.15 **Alexandrine parrot with keratin loss on the rhinotheca which was a consequence of a fungal infection.**



Figure 11.16 **Sulphur-crested cockatoo with damaged beak as a result of PBFD.**

Management

Surgical debridement is carried out as required and antimicrobial therapy administered as indicated by culture.

Viral infections

Aetiology

Viruses affecting the beak include PBFDV in cockatoo species (**Fig. 11.16**), Avipox virus in all species and Polyomavirus in finches.

Clinical presentation, diagnosis, management

See Chapter 10, Disorders of the Skin and Feathers.

NEOPLASIA

Definition/overview

Tumour types involved in the beak include fibrosarcoma (**Fig. 11.17**), squamous cell carcinoma, liposarcoma and keratoacanthoma (**Fig. 11.18**).

Diagnosis

Diagnosis is by biopsy.

Management

The use of chemotherapy and radiation in the treatment of avian neoplasia continues to be explored.



Figure 11.17 **Budgerigar with a beak fibrosarcoma.**

Prognosis

The prognosis is guarded, although newer treatment protocols, including radiation, offer more hope.

KERATIN FLAKES ON THE BEAK

The continued growth of keratin culminates in flakes or sheets of keratin on the surface of the beak that are normally exfoliated by the bird rubbing

Downloaded From Vet-eBooks.com



Figure 11.18 Budgerigar with keratoacanthoma.



Figure 11.19 Budgerigar with cere hypertrophy.

its beak on abrasive substrates (e.g. branches, grooming perches). If these abrasive surfaces are not available, the beak may take on a flaky appearance or become thickened (particularly rostrally). Often these flakes can be easily removed by simply peeling them off with a fingernail. Occasionally, trimming with a rotary hobby grinder (e.g. Dremel®) is necessary to reshape the beak into a more normal shape.

DISORDERS OF THE CERE

The cere is the naked skin situated at the base of the maxilla found in parrots, pigeons and owls. The nares are located within the cere.

Hypertrophy

This may be caused by *Cnemidocoptes* (see above) or, more commonly, hyperoestrogenism in budgerigar hens. The latter usually presents as a smooth (or slightly roughened) hypertrophic growth on the cere (**Fig. 11.19**). It is easily peeled off with a fingernail, with no underlying haemorrhage. Malnutrition, especially hypovitaminosis A, may result in cere hypertrophy.

Colour change

Increasing brown coloration of the cere in female budgerigars is indicative of oestrogenic activity associated with the onset of the breeding season.

Sertoli cell tumours in male budgerigars may present as a blue cere turning brown. Treatment with surgery may be successful in selected cases. Some birds will respond (in the short-term) to GnRH agonists such as leuprolide or deslorelin.

Pallor of the cere is often seen in budgerigars with chronic illness of any type.

Distortion

Chronic respiratory infections may present as 'cere abscesses', swelling and distortion of the cere due to an underlying accumulation of caseated pus in the frontal sinus and nasal cavity. Rhinoliths, large plugs of caseated debris in the nasal cavity under the nares, are commonly seen in African Grey parrots, but can be seen in any species. Affected birds commonly present for sneezing, swelling of the cere and occlusion of the nare (usually unilateral). Rhinoliths must be differentiated from tumours and fungal granulomas. Many can be gently debrided under general anaesthesia, with the area thoroughly flushed after completion. In some cases surgical debridement may be necessary. Samples should be submitted for histopathology and culture to identify the underlying cause.

Permanent asymmetry of the size and shape of the nares often results after cere abscesses or untreated chronic infections erode the supporting bone and soft tissues (**Fig. 11.20**).

Downloaded From Vet-eBooks.com



Figure 11.20 **Budgerigar with chronic rhinitis leading to asymmetrical nares.**

FURTHER READING

Altman B (1997) Beak repair, acrylics. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 787–799.

Clipsham R (1997) Beak repair, rhamphorthotics. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 773–786.

Gelis S (2006) Evaluating and treating the gastrointestinal system. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 411–440.

Olsen GH (2003) Oral biology and beak disorders of birds. *Veterinary Clinics of North America: Exotic Animal Practice* **6(3)**:505–521.

Schmidt RE, Lightfoot TL (2006) Integument. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 395–410.

Speer BL (2002) Trans-sinus pinning to address scissor-beak deformities in psittacine species. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 283–290.

Speer BL (2012) Surgical procedures of the psittacine skull. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*. Accessed on-line through the Veterinary Information Network http://www.vin.com/doc/?id=6347756

Downloaded From Vet-eBooks.com

DISORDERS OF THE EYE

ASSESSING THE EYE

Distant examination

Initially the bird should be watched at a distance (flying, moving around the cage or room). If there are eye problems, the bird may have difficulty finding food or show reluctance to fly or move around. There may be abnormal head posture; many birds with unilateral blindness will tilt their head so that the 'good' eye is uppermost. The bird may have an exaggerated startle reaction.

Physical examination

Symmetry, position and mobility of globes should be assessed. The eyes of a normal bird are clear, symmetrical and centred in the socket. The normal conjunctiva is pale pink and moist.

Hydration can be evaluated by tenting the skin over the eyelids.

The periorbital area is assessed for swelling, discoloration, pustules, scars, scabs or abnormal growths. Conjunctivitis, sunken eyes and ocular discharges are abnormal. Pale or white coloration of the conjunctiva can indicate anaemia. Periorbital feather loss can indicate the bird is rubbing its eyes against perches and the cage sides, suggestive of pain or irritation.

The iris colour should be noted. The colour is an indication of age or sex in many species of birds. Young Amazon parrots have brown irises that become red–orange as they age. Young African grey parrots have brown irises that change to grey, then white, by 15 months of age (**Figs 12.1** and **12.2**). Young macaws have brown irises that change to grey within the first year, and then between one and three years of age the colour changes from grey to yellow. Young cockatoos have brown irises. In some species the adult female cockatoo has red–brown irises (**Fig. 12.3**) and adult males have dark brown to



Figure 12.1 Adult African grey parrots have a pale iris.



Figure 12.2 Juvenile African grey parrots have a dark iris.

Downloaded From Vet-eBooks.com



Figure 12.3 **Mature galah hens often (but not always) have a reddish iris.**

black irises. (Note that not all hens have red–brown irises.)

Examination of the globe

The cornea and anterior chamber should be examined with focal illumination and transillumination.

Fundic examination can be difficult, as the striated muscle in the iris makes pupillary dilation difficult; atropine and tropicamide are ineffectual. Techniques that have been used include:

- Intracameral injection of d-tubocurarine. Topical application of this drug does not work well, as it does not penetrate the cornea sufficiently. The drug therefore has to be administered directly into the anterior chamber using a 27–30 gauge needle, carrying with it substantial risk for injuries of intraocular structures causing hyphaema, increasing intraocular pressure (IOP), transmission of conjunctival flora with consecutive uveitis, and systemic side-effects if larger doses than recommended are used. This technique may best be used therapeutically for the prevention of posterior of anterior synechia.
- Topical vecuronium bromide (4 mg/ml). Vecuronium is a synthetic curariform non-depolarizing neuromuscular blocking agent. It competes for nicotinic acetylcholine receptors at the motor end plate. It does not work topically in South African black-footed penguins, but has worked in citron-crested and sulphur-crested cockatoos and African grey and blue-fronted Amazon parrots with minimal systemic effects.
- General anaesthesia, especially with ketamine or air sac perfusion. Air sac perfusion utilises an air sac catheter, placed in the caudal thoracic air sac, to administer and maintain general anaesthesia utilising isoflurane and oxygen (0.3 L/kg/min). This technique frees the head for examination and treatment but, while it has been reported to be successful in inducing mydriasis in pigeons, it does not appear to work as well in cockatoos.

Once the pupil is dilated, the fundus can be examined using a focused light beam (e.g. Finoff Transilluminator, Welch Allyn®) and a double aspherical ophthalmoscopic lens (at 30, 40, 60, 78 and 90 dioptres (D) refractive power). A 30 D lens is used in birds with larger pupil diameters (nocturnal and diurnal raptors); a 78 D lens is used for pigeons and larger parrots; and a 90 D lens is used in smaller parrots. Alternatively, monocular indirect ophthalmoscopy can be performed in all birds, even the smaller species. The retina, fovea and pecten can all be visualised. All birds with traumatic head injuries should have an ocular examination, as the incidence of retinal haemorrhage and detachment is relatively high in these patients.

Responses and reflexes

- Menace reflex (assesses cranial nerves II and VII). Eye-blink, pulling away of the head, or aggressive action of the beak can be provoked by bringing the hand towards each eye.
- Pupillary light response (assesses cranial nerves II and III). The avian pupil's movements are poor in response to light, but rapid in response to accommodation or voluntary control. Although there is no consensual pupillary light response (as there is complete decussation of the optic nerves at the chiasm), the orbital bone is so thin that a response can often be detected in one eye after a light is shone in the other.

Downloaded From Vet-eBooks.com

- Corneal reflex. A symmetrical eye-blink normally occurs when the cornea is gently touched with a moist cotton swab. Failure to do so indicates a lesion in cranial nerves V and VII.
- Palpebral reflex and facial sensation. A symmetrical eye-blink should be elicited by touching each side of face or the lateral canthus of the eye. Failure to do so suggests a lesion in cranial nerves V and VII.
- There should be symmetrical deviations of the eyes when the head is moved in different positions, with the eyes always returning to the centre. Failure to do so suggests either a bilateral lesion in cranial nerve VIII or a brainstem lesion (especially in the pons and midbrain).

Ancillary testing

- Cytology and culture of the conjunctival flora should be permed in cases of conjunctivitis or ocular discharge.
- A Schirmer tear test is performed as with other species; in clinically normal birds the result is 3–12 mm, but there is a wide variation between species. Local anaesthesia in the cornea significantly reduces tear movement along the strip.
- The Phenol Red Thread Test (Zone-Quick, San Mateo) is a new test available in the evaluation of tear production in animals. It is a yellow cotton thread, 75 mm long, impregnated with the pH indicator phenol red (phenolsulphonephthalein). One end of the thread is folded to a length of 3 mm and, at the time of use, opened to an appropriate angle and placed within the ventral conjunctival fornix. The thread remains in this position for 15 seconds. The colour of the thread changes to red when wet and, once the thread is removed, the length of this change is measured in millimetres similar to the Schirmer tear test. The Phenol Red Thread Test is faster and causes less reflexive tearing than the Schirmer tear test due to the significantly less irritating contact with the cornea in species where it has been evaluated. Normal results appear to lie in the range of 8–23 mm, with no effect from local anaesthesia discerned.
- Fluorescent staining of the cornea can detect small ulcerations and lacerations.
- The Tonopen® is ideal for larger birds, as it can be used with a minimal corneal diameter of 9 mm. Readings of eyes with corneal diameters of 5 mm or less (budgerigar range) are not reliable unless the newer Tonovet® is used.
- Ultrasonography allows detection of retinal detachment or sequestration of the pecten bridge.
- More advanced tests include:
  - Electroretinography; the recording of electrical potentials after light stimulation. It gives no information about vision, but does assess retinal function.
  - Fluorescein angiography; the observation of the distribution of fluorescein within ocular blood vessels following intravenous injection. This enables diagnosis of subtle haemorrhages of the pecten and choroid, atrophy of vessels and retinal disease.

DISORDERS OF THE EYE

Lids and periorbital region

Poxvirus (in mild cases) may cause a unilateral blepharitis with eyelid oedema, starting about 10–14 days after infection. It may also cause keratitis and, less commonly, anterior uveitis. Prophylactic vitamin A injections and antibiotic therapy may decrease the severity of the infection. Most cases are self-limiting and resolve over a few weeks. In more severe cases, scabby sores can develop on the eyelids, resulting in distortion and scarring of the eyelids.

Chlamydial infections and other causes of sinusitis may lead to blepharitis, thickening of the eyelids and matting or loss of periorbital feathering (due to lacrimation and rubbing).

Proliferative lesions on the eyelids and periorbital region may be due to *Cnemidocoptes pilae*, papillomas or vitamin A deficiencies.

Feather loss around the eye without any evidence of inflammation or conjunctivitis can be due to excessive grooming by a cage mate. This is often seen in finches when they are overcrowded, or in parrots during the breeding season.

Downloaded From Vet-eBooks.com

Eyelid abnormalities include:

- Cryptophthalmos (failure of eyelid formation, resulting in fusion of the eyelid margins) is occasionally seen in cockatiels (**Figs 12.4** and **12.5**). Surgical correction is usually unrewarding, but affected birds appear to have some degree of vision through their eyelids.
- Blepharophimosis (narrowing of the palpebral fissures without fusion of the eyelid margins) is occasionally seen in all species. Again, surgical correction is usually unrewarding.



Figure 12.4 **Cockatiel with acquired cryptophthalmos following a severe chlamydial infection.**



Figure 12.5 **Indian ring-neck parrot with congenital cryptophthalmos.**

- Symblepharon (adhesion of the eyelid to the globe) can be seen as a sequel to severe conjunctivitis. Surgical correction is possible in some cases.
- Scarring and deformity is occasionally seen after avian poxvirus infections and traumatic injuries.

Globe and orbit

Microphthalmia, a congenitally abnormally small eye, is rarely seen. In some cases the eye is structurally and functionally normal, but in other cases cataracts, retinal dysplasia, or retinal detachment result in complete blindness. Chronic conjunctivitis, due to conjunctival exposure, should be treated with good hygiene and topical antibiotics as indicated.

Anophthalmia (the actual total absence of a globe) is rare. In many cases a rudiment of ocular tissue can remain, which may result in a weeping sinus.

Exophthalmia (the protrusion of the globe from the orbit) may be due to malformation of the orbit associated with metabolic bone disease, or it may be secondary to infection (bacterial, fungal or trichomonad in origin) in the infraorbital sinuses. Other reported causes include trauma, inflammation of the Harderian gland, or neoplasia of the orbit or globe. Sinusitis is the most common cause and may require surgical removal of caseated material to achieve resolution. Retrobulbar sinusitis is difficult and frustrating to treat because of the caseous nature of the exudate and the lack of surgical access to the sinus. Surgical trephination of the sinus followed by flushing and mechanical debridement may assist in some cases.

Panophthalmitis (inflammation of the globe) may be caused by infection or trauma. Early clinical signs are referable to extension of the inflammatory process throughout the eye. These include: pain; blepharospasm; hypopyon; corneal oedema; ulceration; secondary glaucoma. Chronically the eye may develop phthisis bulbi, a shrunken, fibrotic globe. Treatment requires aggressive antimicrobial therapy, NSAIDs and temporary tarsorrhaphy in early cases, but in more chronic or non-responsive cases, enucleation is often the preferred treatment.

Glaucoma (increasing pressure within the globe) may be primary, or secondary to trauma. The globe is enlarged and painful. Glaucoma must be differentiated from exophthalmos. Diagnosis is based on the clinical signs and intraocular pressure, as measured

Downloaded From Vet-eBooks.com

by a Tonopen® or Tonovet® (see Ancillary testing section above). Intraocular pressures are between 9.2 and 22 mmHg in normal bird eyes. One suggested treatment is topical dorzolamide applied three times daily initially, then titrated to effect.

Conjunctiva

Overview

Thickening and hyperaemia of the conjunctiva, often associated with epiphora or ocular discharge, may be seen with conjunctivitis, sinusitis (see Chapter 19, Diseases of the Respiratory System) or neoplasia.

Aetiology

Conjunctivitis (**Fig. 12.6**) may be due to:

- Bacterial infections.
- Chlamydiosis.
- *Mycoplasma*.
- Mycobacteria.
- Viral infections (e.g. adenovirus, poxvirus, cytomegalovirus [in Gouldian finches]).
- Fungal infections (*Aspergillus*, *Candida*, *Cryptococcus*).
- Parasites: 'eye' worms (*Oxyspirura mansoni*, *Ceratospira* spp. and *Thelazia* spp.); cryptosporidial infections; *Plasmodium* spp. can cause eyelid swelling in canaries and domestic poultry; microsporidiosis has been reported to cause conjunctivitis. Trichomoniasis is an uncommon cause of conjunctivitis.
- Foreign bodies and physical irritants such as smoke or chemical fumes.
- Allergies, especially secondary to nicotine.

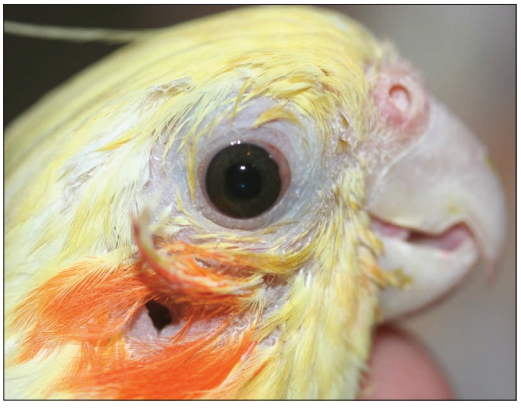


Figure 12.6 **Cockatiel with conjunctivitis.**



Figure 12.7 **Cockatoo with a conjunctival tumour (haemangioma).**

Neoplasia involving the eyelids includes conjunctival papillomas, haemangiomas, squamous cell carcinomas and melanomas (**Fig. 12.7**).

Diagnosis

Diagnosis involves cytology and culture of the conjunctiva. Conjunctival biopsy or PCR of a conjunctival swab are also useful diagnostic tools.

Management

This is determined by the aetiological agent. Topical therapy, if indicated, should be in drop form. Eye ointments should be avoided, or at least used very sparingly, because of the likelihood that grooming will see the ointment spread throughout the feathering. Ointments containing cortisone should not be used at all, as systemic absorption and subsequent immunosuppression and hepatopathies can be significant.

In many cases, especially chlamydiosis, systemic as well as topical therapy is indicated.

Cornea

Overview

Xerophthalmia (a lack of tear production) is occasionally seen in birds who have vitamin A deficiency. A long-term deficiency may result in an irreversible keratoconjunctivitis sicca.

Cholesterol deposits are seen frequently in parrots, especially Amazons, usually on a high-fat diet or with other metabolic issues. They appear as painless,

Downloaded From Vet-eBooks.com

multifocal, white, non-inflammatory corneal lesions. Even with resolution of the dietary problems, these corneal precipitates do not resolve, but rarely seem to cause a problem.

Infectious keratitis may be bacterial or fungal or be caused by microsporidia. Non-infectious keratitis may be caused by trauma or a foreign body. Corneal ulceration is often associated with foreign bodies such as dust or seed husks.

Management

Antimicrobial therapy, topically and/or systemically, is given as indicated. Flubiprofen topically or meloxicam systemically may be used for analgesia and anti-inflammatory therapy.

The cornea can be protected by a temporary tarsorrhaphy. Third eyelid flaps have been used, but they are difficult to perform and experience in other species indicates they are not as useful as originally thought.

In severe cases, enucleation may be required. If possible, a specialist opinion should be sought before taking this step.

UVEA

Acute uveitis

Aetiology

Acute uveitis (**Fig. 12.8**) may be caused by trauma, infection (e.g. reovirus, West Nile virus, and toxoplasmosis), septicaemia or lens rupture.

Clinical presentation

Signs include: photophobia; blepharospasm; corneal oedema; aqueous flare; hyphaema; hypopyon; miosis; dyscoria; iris thickening or discoloration; anterior/posterior synechiae.

Management

In general, birds respond to treatment for uveitis better than mammals. Anti-inflammatory therapy is required using topical (e.g. flubiprofen) and systemic (e.g. meloxicam) NSAIDs.

Chronic uveitis

Aetiology

This is a sequela to acute uveitis.



Figure 12.8 **Hawk with acute uveitis.**

Clinical presentation

Signs include: diffuse corneal oedema; posterior synechiae causing pupillary seclusion and iris bombé; anterior synechiae; secondary glaucoma; cataract; retinal atrophy and chronic detachment; blindness.

Management

Anti-inflammatory therapy is given as for acute uveitis: topical (e.g. flubiprofen) and systemic (e.g. meloxicam) NSAIDs.

LENS

Cataracts

Aetiology

Familial cataracts are reported in Yorkshire and Norwich canaries. A fully penetrant autosomal recessive gene is responsible. Acquired cataracts (**Fig. 12.9**) can be due to uveitis toxins or ageing (see above). Idiopathic cataracts have been noted in a number of species.

Downloaded From Vet-eBooks.com



Figure 12.9 **Mature kakariki hen with acquired cataracts due to ageing.**

Treatment

In many cases no treatment is required or feasible, due to other disease processes in the eye affecting vision. Phacoemulsification has been used successfully in many species and may be used if the retina is believed to be intact.

Luxation

This may occur secondary to trauma or uveitis. Lens luxation also leads to further uveitis. If this lens-induced uveitis does not respond to NSAID therapy, phacoemulsification is necessary to remove the lens. If this is not available, enucleation should be considered.

Retina

Retinitis may occur due to infectious agents (e.g. *Toxoplasma*), trauma or diseases of the CNS. Retinal detachment may occur following trauma or retinitis. Retinal dysplasia has been noted sporadically. Retinal degeneration may be due to malnutrition, toxins or hypoxia, but no specific cause has yet been identified.

FURTHER READING

Gancz AY, Malka S, Sandmeyer L, Cannon M, Smith DA, Taylor M. (2005) Horner's syndrome in a red-bellied parrot (*Poicephalus rufiventris*). *Journal of Avian Medicine and Surgery* **19**(**1**):30–34.

Kern TJ (1997) Disorders of the special senses. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (Eds). WB Saunders, Philadelphia, pp. 563–589.

Korbel RT (2000) Avian ophthalmology: a clinically oriented approach. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 439–456.

Korbel RT (2007) Avian ophthalmology: principles and application. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 191–200.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

DISORDERS OF THE EAR

OTITIS EXTERNA

Aetiology

Otitis externa is an uncommon disorder. It may result from infection with bacteria (*Escherichia coli*, *Staphylococcus aureus*, *Pasteurella multocida*, *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Klebsiella oxytoca*, *Enterococcus* spp.), fungi (*Candida* spp., *Microsporum gallinae*) or mycobacteria, or infestation with arthropod parasites (*Cnemidocoptes*). It may occur as an extension of generalised skin disease or may be due to neoplasia or trauma.

Clinical presentation

Affected birds often demonstrate head shaking, yawning, head tilt and ataxia. Closer examination may reveal an aural exudate (serous, purulent or haemorrhagic) (**Fig. 13.1**). There may be caseated material in the ear canal (**Fig. 13.2**). Swelling and erythema, auricular feather loss and self-inflicted excoriation are often seen. There may be hyperkeratosis of the ear canal and surrounding skin. In some chronic cases, the ear canal may become so stenotic as to close over.

Diagnosis

Diagnosis is based on clinical signs, cytology and culture. The narrow ear canal makes visualization with anything larger than a 1.9 mm rigid endoscope difficult, if not impossible.

In cases that fail to respond to therapy, judicious biopsy may be required to better evaluate the disease process.

Management

Systemic and topical antibiotics are given as indicated by culture. Meloxicam or another NSAID can be given if the ear appears to be particularly painful or swollen. Careful flushing with saline may assist in the removal of exudate; if this is unsuccessful debridement with a loop curette may be required.

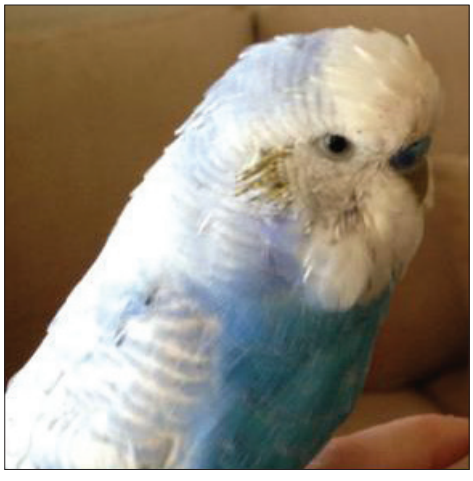


Figure 13.1 **Budgerigar with otitis externa. Note the aural exudate.**



Figure 13.2 **Cockatoo with otitis externa. Note the caseated material in the ear canal.**

Downloaded From Vet-eBooks.com

OTITIS MEDIA AND OTITIS INTERNA

Aetiology

Otitis media and otitis interna are uncommon. They can occur as a result of developmental abnormalities seen in some canaries. Other causes include an extension of otitis externa or sinusitis; PMV-3 infection (especially in Australian *Neophema* spp., canaries and finches); poxvirus; iatrogenic damage following treatment with aminoglycoside antibiotics (e.g. gentamicin); and neoplasia.

Clinical presentation

Signs include head tilt, 'star gazing', torticollis and vestibular disorders (loss of proprioception).

Diagnosis

Diagnosis is based on clinical signs, PMV-3 serology or virus isolation (from tissue samples), if appropriate. In larger birds CT or MRI may be of value in confirming a diagnosis.

Management

Antimicrobial and anti-inflammatory therapy may be of some value, although the prognosis is guarded.

In cases of otitis media a myringotomy, perhaps combined with an ear canal ablation, may allow gentle irrigation and removal of caseated pus in the middle ear. The skin is incised around the external meatus and careful dissection is used to mobilise and excise the external ear canal. Care must be taken in the dorsomedial region, where the external ear connects to the inner ear and vestibular system. Complications of this surgery include recurrence of the otitis, draining tracts, and facial palsy.

FURTHER READING

Kern TJ (1997) Disorders of the special senses. In: *Avian Medicine and Surgery*. RB Altman, *et al*. (eds). WB Saunders, Philadelphia, pp. 563–589.

Schmidt RE, Reavill DR, Phalen DN (2003) Special sense organs. In: *Pathology of Pet and Aviary Birds*. RE Schmidt, *et al*. (eds). Iowa State Press, Ames, pp. 197–212.

Shivaprasad HL (2007) The avian ear: anatomy and diseases. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 127–133.

Downloaded From Vet-eBooks.com

CHAPTER 14

DISEASES OF THE LEGS, FEET AND TOES

MALFORMATIONS

Introduction

It can be difficult to distinguish between congenital and acquired malformations, especially in neonates and juveniles. Factors that can have an effect on normal skeletal conformation include:

- The bird's parents:
  - Genetics: hereditary malformations have been identified in some birds.
  - Health: in particular, subclinical heavy metal toxicosis has been associated with congenital limb malformation in the offspring of affected birds.
  - Diet: poor quality diets fed to parent birds are often reflected in the health and conformation of the offspring. In particular, calcium–phosphorus imbalances in the parental diet will often result in chicks with limb malformations.
- Incubation procedures and parameters. Artificial incubation is more likely to produce chicks with limb malformations than natural incubation. Incorrect incubator temperature, humidity, ventilation and rate and degree of egg turning can have deleterious results on the development of the embryo and result in malformations.
- The nesting environment. Nest box design can have an effect of neonates in several ways: slippery floors are believed to contribute to coxofemoral subluxation ('splay leg') in budgerigars; inexperienced parents who can enter the nest box quickly and land heavily on the chicks can cause injuries resulting in malformations.
- Hand rearing:
  - Although many hand rearers now use good quality commercial hand-rearing diets, there are still occasions where: home-made diets are used; commercial diets are not used according to the manufacturer's directions; or commercial diets are supplemented with other ingredients. These actions may result in skeletal malformations.
  - Birds that are hand reared in a brightly lit, roomy container are often encouraged to move around more than they would in a dark and relatively cramped nest box. This can lead to premature and excessive weight bearing on the legs, with subsequent bowing and malformations.
  - Many chicks reared on a good quality diet are then weaned onto a seed-based diet, often prematurely. This decrease in the nutritional value of a diet fed to a still-growing chick frequently results in acquired malformations.

Nutritional secondary hyperparathyroidism and rickets (collectively referred to as metabolic bone disease) are underlying factors behind many of these problems. Rickets is a metabolic bone disease in growing animals caused by a vitamin D3 deficiency. (This can be due to inadequate nutrition, lack of exposure to sunlight, defective vitamin D activation, defective vitamin D receptors, hypoparathyroidism, renal failure, renal phosphate loss or gastrointestinal malabsorption.) The result is impaired mineralization of osteoid tissue or epiphyseal cartilage, leading to thinning and weakening of the bones and excessive growth of cartilaginous structures. This, in turn, causes deformity at the ends of the

Downloaded From Vet-eBooks.com

bones, particularly in the proximal tibiotarsus, the beak (rubber beak), the head of the ribs and, sometimes, the costochondral junction (rachitic rosary). Radiographically there may be widening and distortion of the growth plates.

Nutritional secondary hyperparathyroidism is commonly seen in both juvenile and adult birds fed a diet that is either calcium deficient or has an excess of phosphorus (or both). For example, seed-only diets may have a calcium: phosphorus ratio as low as 1:10. Fruits, nuts and most vegetables are also calcium deficient and/or an excess of phosphorous. When these diets are fed to birds, especially parents rearing chicks or to recently fledged juveniles, the parathyroid gland releases parathyroid hormone which, amongst other effects, withdraws calcium from the bone to maintain normal serum calcium levels. The result is thin bones which bend or break easily, leading to malformations.

Coxofemoral subluxation

Aetiology

Coxofemoral subluxation or splay leg (see **Fig. 14.1**) may be the result of a lack of nonslip substrate in the nest box. This allows the legs to splay out laterally away from the body, subluxating the coxofemoral joint and leading to laxity or damage of the medial collateral ligaments of the stifle, angular limb deformities of the femur, tibiotarsus and tarsometatarsus, or slipped tendons. Sometimes, if the chicks have metabolic bone disease as well, folding fractures of the tibiotarsus will result in secondary 'splay' deformities.

Management

Early recognition and correction are important; once skeletal ossification occurs conservative techniques such as splinting or hobbling are unlikely to be effective. Very young birds can have their legs hobbled into a normal position in order to guide the leg into a normal position during growth. Various techniques have been used to achieve this goal, including tying the feet together with bandages or placing the chick's legs into a foam block. It is important to adjust the hobbles every 1–2 days (to allow for the chick's growth) until the problem is corrected. However, if the chick is not presented until after ossification is complete, surgical derotational osteotomies may be required.

ANGULAR LIMB DEFORMITIES

Leg rotation

Aetiology

Rotation of the femur or tibiotarsus occurs when more weight is borne on one side of the growth plate than the other (**Figs 14.2, 14.3**). Growth on this side of the physis is inhibited while, on the other side, growth continues normally. Common causes include incubation problems, nutritional imbalances and trauma. In precocious chicks (e.g. ratites), leg rotation may occur for a range of reasons including

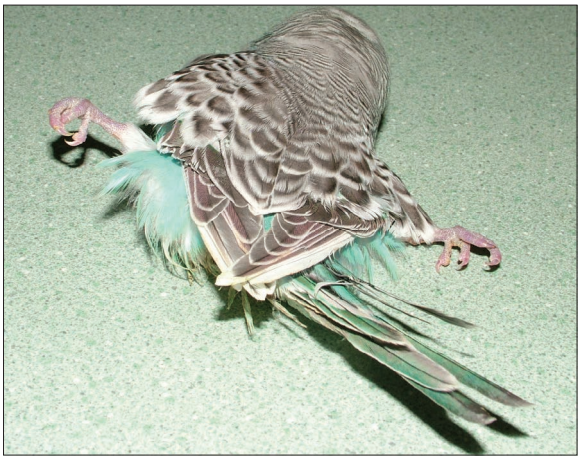


Figure 14.1 Budgerigar with a coxofemoral subluxation or splay leg.



Figure 14.2 Black cockatoo chick with femoral and tibiotarsal rotation. This chick was artificially incubated and hand reared, but its parents had a calcium-deficient, all-seed diet.

Downloaded From Vet-eBooks.com



Figure 14.3 **Indian Ringneck with a tibiotarsal rotation. Note the outward rotation of the right leg compared with the left.**

insufficient exercise, high-energy diets, trauma and heated flooring. In altricial chicks, such as parrots, the inciting cause is rarely determined.

Clinical presentation

The result of a leg rotation is a deviation of the growth of the limb from the midline: if the growth is directed laterally, it is termed a valgus deformity; if medial to the midline, it is termed a varus deformity. The deformity may be a bowing or rotation of the affected bone. This then has a cascading effect on the muscles, tendons and joints of the affected leg. As the chick grows this rotation often worsens as tendons contract and even mineralise.

The direction and severity of the rotation can be assessed by flexing the tibiotarsal-metatarsal joint so that the tarsus meets the cranial proximal tibiotarsus. In the normal chick the centre of the tarsus should be in alignment with the cnemial crest. Deviation medial or lateral to this midline indicate an ALD.

Management

Corrective surgery, in the form of derotational osteotomies, is the treatment of choice, but it must be done sooner rather than later. Timing is essential when addressing affected joints; waiting until the bird has finished growing may result in irreversible tendon and joint contracture.

Bowed legs

Aetiology

Malnutrition, especially when combined with excessive and premature weight-bearing, causes one or both tibiotarsus to bow outwards, cranially or caudally.

Clinical presentation

The bowing of the tibiotarsii shortens the legs and changes the placement of the phalanges. The result is that the chick weight-bears on the lateral phalanges (P3 and P4), giving the chick a 'pigeon-toed' appearance. The gait may be shortened. Radiology can determine the severity of the malformation.

Management

If a severe deformity is present a derotational osteotomy may be required to correct this condition.

PATHOLOGICAL FRACTURES

Aetiology

Pathological fractures commonly occur in chicks with nutritional secondary hyperparathyroidism or rickets. The fractures most commonly occur in the long bones—the humerus, the radius and ulna, the femur and the tibiotarsus. They are often first noticed when the bird fledges and attempts to fly. Flapping of the wings may be enough to fracture the humerus, radius or ulna while a heavy landing can break the tibiotarsus or femur. It is not uncommon for multiple fractures to be present in the one bird. Other birds will suffer fractures while still in the nest; these fractures have healed by the time the bird fledges but deformities become obvious when the bird is on the perch or tries to fly.

Diagnosis

Physical examination and radiographic assessment are usually sufficient to make the diagnosis. Measuring total and ionised calcium can assist in diagnosing hypocalcaemia and assessing its severity.

Downloaded From Vet-eBooks.com

Management

Some of these fractures will heal with minimal intervention, but this invariably results in some degree of bone deformity. Attempts to repair these fractures surgically can be frustrated when the contralateral leg is required to support the bird's weight and lacks the cortical strength to do so, resulting in another fracture or bowing of that leg. Slinging the chick to minimise this problem can cause other problems such as compression of the sternum and ribs. These factors need to be considered before undertaking such a procedure, making the prognosis guarded in many cases.

TOE ANTEROFLEXION

Definition/overview

Anteroflexion of P4 (and occasionally P1) is a developmental issue in psittacine paediatrics, more often encountered where there has been subclinical stunting or substrate mismanagement. Once the first phalangeal bone has ossified normally, it is fused to the tarsometatarsus and becomes less flexible. If the anteroflexion has not been corrected (by splinting) by that time, the anteroflexion may be permanent (**Fig. 14.4**).

Management

The affected digit can be splinted out into a normal position, but the abnormal angulation of the joint means that once the splint has been removed, the digit will return to an abnormal position. This can mean the nail is not in wear on a perch and will overgrow, requiring regular trimming (or even amputation) of the last phalanx and nail bed.



Figure 14.4 **Black cockatoo with an anteroflexion of P4.**

SLIPPED TENDON (PEROSIS)

Definition/overview

Medial or lateral luxation of the gastrocnemius tendon occurs over the tibiotarsal condyles. It is most commonly seen in juvenile poultry, waterfowl and ratites (where it is associated with nutritional deficiencies), but has been seen in parrots with leg deformities.

Diagnosis

Diagnosis is made on clinical signs and palpation of the joint and tendon.

Management

Surgical replacement of the tendon and repair of the tendon retinaculum is invariably unsuccessful, the repair often breaking down within hours or days when the bird attempts to use the affected leg.

Another technique involves placing a K-wire into the tarsometarsus on the medial side of the ligament, with the proximal end of the pin protruding from the bone and effectively preventing the ligament from slipping medially. This technique appears promising, although a degree of joint stiffness often results. Any bone deformities predisposing to the tendon luxation must be corrected.

This condition often requires arthrodesis of the hock joint.

SWOLLEN JOINTS

Swollen joints, often associated with pain and lameness, is a common presentation at any age. The swelling can be associated with articular gout, arthritis or luxation of the joint.

Articular gout

Definition/overview

Chronic renal insufficiency, with a gradual elevation of plasma uric acid levels, results in the deposition of uric acid crystals in the synovial capsules and tendon sheaths of the joints. These uric acid crystals

Downloaded From Vet-eBooks.com

provoke an inflammatory response that causes the swelling and pain associated with articular gout.

Clinical presentation

Gross lesions typically consist of soft painful swellings on the feet at the metatarsophalangeal and interphalangeal joints. White urate tophi may be visible within these swellings. The bird may have a shifting lameness and the toes are often held in an extended position rather than curled around the perch.

The diagnosis and management of this condition is discussed in more detail in Chapter 24, Disorders of the Urinary System p. 338.

Luxation of the joint

See Chapter 15, Disorders of the Musculoskeletal System p. 211.

Arthritis

Aetiology

Swollen painful joints (arthritis) can be due to traumatic injuries, septic inflammation (*Streptococcus* spp., *Pseudomonas* spp., *Chlamydia psittaci*, *Mycoplasma* spp., *Reovirus*), microfilaria, especially in wild-caught Indonesian cockatoo species, articular gout (see above), degenerative changes or neoplasia (see **Fig. 5.9**, Chapter 5, Diagnostic Imaging p. 100).

Diagnosis

Diagnosis is based on the patient's history, physical examination, haematology and biochemistries, radiography and fine needle aspirate with cytology and culture where appropriate.

Management

The cause should be identified and removed if possible (e.g. weight reduction in obese birds); septic joints may need to be opened and lavaged; caseated debris should be removed if possible. Long-term antibiotic therapy (as dictated by culture and sensitivity testing) will be required in cases of bacterial infection.

Pain relief may be achieved with NSAIDs or oral tramadol.

Attempts have been made to improve the joint physiology using pentosan polysulphate and glucosamine. These are unproven therapies in birds at this time. Anecdotal reports are encouraging but adverse effects to pentosan polysulphate (including death) have been recorded at high doses, so these therapies should be used with caution.

Husbandry should be adapted to cater for disabilities. Arthritic birds may have mobility problems and may have difficulties grasping small diameter perches. Wide flat perches and providing easier access to food and water may be beneficial.

PODODERMATITIS

Definition/overview

Pododermatitis is also known as bumblefoot. It refers to an inflamed and often infected lesion on the plantar surface of the foot. It is most commonly seen in raptors, but can be seen in other species as well, especially gallinaceous birds, passerines and parrots. It is a disease of captivity, being rare or non-existent in wild birds.

Aetiology

The plantar surface of the foot is protected by a thick layer of stratified squamous epithelium, which in turn is covered by a layer of keratin. Over the surface of this keratin is a layer of papillae, which are thought to spread evenly the weight-bearing requirement of the foot.

When this barrier is eroded or breached, an inflammatory response is often provoked and infection can be introduced. These infections are usually associated with *Staphylococcus aureus*, but other bacteria including *E. coli*, *Pasteurella* spp., *Klebsiella* spp., *Clostridium* spp., *Corynebacterium* spp., *Bacillus* spp., *Diplococcus* spp., *Nocardia* spp., *Actinobacillus* spp., *Actinomyces* spp., *Aeromonas* spp., *Proteus* spp. and *Pseudomonas* spp. have been implicated. *Candida* spp. and *Aspergillus* spp. may also be involved in some cases.

The inflammation and/or infection may extend into the joints, tendons and bones of the foot.

Contributing factors to the erosion or breaching of the keratin barrier include trauma, hypovitaminosis A, obesity, perches (smooth, regular surfaces or fine sandpaper-covered perches), long periods of inactivity leading to excessive weight bearing without relief, and excessive weight bearing on one leg due to a problem with the other leg

Downloaded From Vet-eBooks.com

(e.g. a unilateral lameness may lead to pododermatitis in the contralateral leg). In many cases, pododermatitis in one foot will lead to some degree of pododermatitis in the other foot.

Clinical presentation

Raptor veterinarians have developed a classification scheme for pododermatitis that provides clinicians with a treatment plan and guide to prognosis. This classification scheme can be extended to other species with good results.

Class I: There is early devitalisation of a prominent plantar area without disruption of the epithelial barrier. It is subdivided into:

- Hyperaemia (bruise) or early ischaemia (a blanched area with compromised capillary perfusion) (**Fig. 14.5a**).
- Hyperkeratotic reaction (an early callus) (**Fig. 14.5b**).

Class II: There is localised inflammation/infection of underlying tissues in direct contact with devitalised area, with no gross swelling. It is subdivided into:

- Puncture wound.
- Ischaemic necrosis of epithelium (a penetrating callus or scab).

Class III: More generalised infection; gross inflammatory swelling of underlying tissues is present. The origin may be puncture wounds or ischaemic necrosis; however, by this stage the initial cause is of minor significance in comparison with the ongoing pathology. It is subdivided into:

- Serous (acute): oedema and hyperaemia of the tissues.
- Fibrotic (chronic): attempt at encapsulation and confinement.
- Caseous: accumulation of necrotic debris.

Class IV: There is established infection with gross swelling and involvement of deeper vital structures. Radiology and surgical exploration will often be required to differentiate class III from class IV. Class IV is a chronic condition causing tenosynovitis and occasionally arthritis and osteomyelitis.

Class V: This is an extension of class IV, characterised by crippling deformities.

Diagnosis

A thorough history is taken to investigate the bird's husbandry, diet and previous medical problems. A thorough physical examination is carried out to classify the pododermatitis and identify any concurrent or predisposing problems (e.g. leg or spinal injuries). Ancillary diagnostic steps include culture

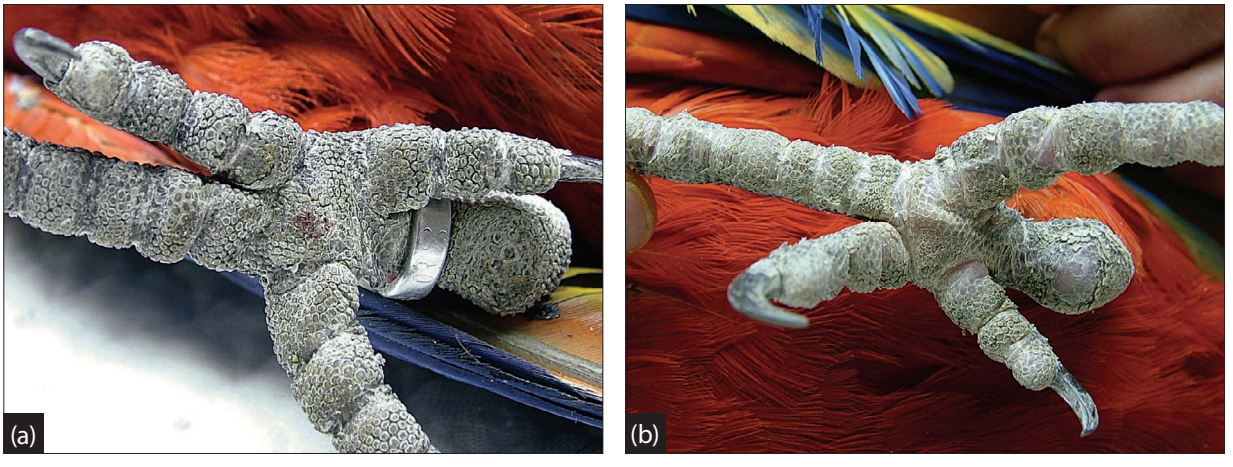


Figure 14.5 **Scarlet macaw with early signs (Class 1) of pododermatitis on its first foot (a) (note bruising on the plantar surface), and an early callus on the ventral hock of its second foot (b).**

Downloaded From Vet-eBooks.com

and sensitivity testing of infected lesions and radiography of any case worse than class II.

Management

This is usually determined by the classification of the condition, as outlined above:

- Class I carries a favourable prognosis, as there is no evidence of infection. The changes generally respond to conservative husbandry changes including changing perching surfaces and application of topical emollients.
- Class II carries a good prognosis, as infection is localised. Such lesions respond well to surgery, as the total affected area is easily resected and epidermal defects are characteristically small, hence the architecture of the weight-bearing structures of the plantar aspect are maintained intact. This class will generally not respond to conservative treatment.
- Class III traditionally carries a good to guarded prognosis, as infection is well established and structural changes have affected the foot. Some can be treated as for class II; however, the majority should be treated by complete surgical removal of all affected tissue, followed by first-intention healing.
- Class IV carries a guarded to poor prognosis, as infection is harboured in and affects deeper vital structures, making surgical debridement difficult or impossible. In view of the chronicity, pockets of encapsulated infective tissues are often present, which if not cleared will result in later recurrence.
- Class V carries a poor to hopeless prognosis and may require euthanasia.

Treatment regimens include improving the bird's health and nutritional status, usually by conversion to a formulated diet, and improving the bird's husbandry by supplying natural branches for perches. Some cases of pododermatitis may benefit from padded perches until the condition has resolved.

If surgery is contemplated, culture and sensitivity testing should be performed in advance so that the results are available at the time of surgery. This allows an appropriate antibiotic to be used at the time of surgery rather than several days later when postsurgical fibrotic encapsulation of pathogenic bacteria is underway. While waiting for the results, broad-spectrum antibiotic coverage and analgesia can be commenced.

Surgery is aimed at debulking the infection, removing all caseous debris and infected and fibrotic tissue, and closing the site so that primary-intention healing can occur. Placing antibiotic-impregnated methylmethacrylate beads into the area before closure can assist with long-term delivery of antibiotics into the affected area.

After surgery it is important that pressure is relieved from the surgery site. This is achieved by padded bandages or gauze/rubber 'doughnuts' to distribute pressure evenly while keeping the surgery site clean and away from potentially contaminated surfaces.

Close attention must be given to the contralateral foot to ensure it does not develop pododermatitis as well.

CONSTRICTED TOE SYNDROME

Definition/overview

This is a circumferential constriction caused by foreign material or fibrous bands, which may result in avascular necrosis of the digit distal to the constriction (**Fig. 14.6**).



Figure 14.6 **Scarlet macaw chick with constricted toe syndrome.**

Downloaded From Vet-eBooks.com

Aetiology

Two forms are seen:

- The formation of constricting fibrous bands on the toes of neonatal chicks, possibly due to low humidity in the nest box, incubation issues or ergot-like intoxication.
- Entanglement of the toes with artificial or natural fibres in birds of any age.

Clinical presentation

In the early stages the toe distal to the constriction may appear swollen and oedematous. If the bird is perching, lameness may be noted. As the condition progresses, circulation to the toe is lost. Cyanosis of the toe is followed by a desiccated appearance; finally, the affected area drops off, sometimes leaving a nub of necrotic bone protruding.

Management

Early recognition and prompt treatment increase the chances of a successful outcome. After 24 hours the prognosis for saving the toe becomes poor. If the patient is presented after circulation loss is severe and necrosis is apparent, amputation is usually required.

Artificial or natural fibres need to be carefully removed. The use of general anaesthesia and magnification is strongly recommended.

Mild or early neonatal cases may respond to increasing the humidity around the toe (by using moisturising creams) and massage.

More advanced or severe cases will require surgery. Two techniques have been advocated:

- A deep longitudinal incision on either side of the toe to sever the band and relieve the constriction.
- Complete excision of the band followed by a circumferential skin anastomosis using only two or three sutures to establish skin apposition without tension. Following the anastomosis a release incision should be made on the medial and lateral aspects of the digit longitudinally across the anastomosis to allow swelling to occur without compromising circulation.

After surgery a hydroactive dressing should be used to keep the area moist and prevent scar formation while the incisions heal. The toe must be monitored during healing for any reformation of the constricting band.

BILATERAL PARESIS OR PARALYSIS

Spinal trauma or neoplasia

Definition/overview

If trauma or neoplasia severs or compresses the spinal cord, it can result in paresis or paralysis. Depending on the level and extent of the injury or neoplasm, cloacal and vent tone may also be lost. Most spinal trauma will occur at the notarial–synsacral junction, where there is some spinal flexibility.

Management

Conservative or surgical therapy is rarely successful, and a guarded prognosis for return to normal function should be given. Non-steroidal anti-inflammatory drugs and, if indicated, tramadol may give symptomatic therapy while for any response or recovery.

Obturator paralysis

Definition/overview

An 'obturator paralysis' syndrome is sometimes seen in hens that are egg-bound or have had difficulty passing an egg. Calcium deficiencies, pelvic fractures and bruising of the sciatic and other nerves, acting in concert or individually, can lead to leg paresis.

Clinical presentation

Affected birds are otherwise bright and alert. Cloacal and vent tone may be affected.

Management

Supportive care, NSAIDs and calcium supplementation are indicated. Prognosis is fair to good, with most birds recovering within a week.

Lorikeet paralysis syndrome

Clinical presentation

Lorikeet paralysis syndrome (clenched foot syndrome) (**Fig. 14.7**) is seen in both wild and captive lorikeets. Affected birds, which can be of any age and either sex, are presented throughout the year with

Downloaded From Vet-eBooks.com



Figure 14.7 **Swainson's (Rainbow) lorikeet with lorikeet paralysis syndrome (clenched foot syndrome).**

leg paralysis and clenched feet. They are otherwise bright and alert and usually have a good appetite. Occasional individuals will become progressively more extensively paralysed.

Diagnosis

Histopathology of the cerebellum, brain stem, spinal cord and brain shows non-suppurative leptomeningitis, perhaps consistent with a viral encephalomyelitis. However, no virus has yet been identified. Lead toxicosis has been implicated in some cases and so blood lead levels should be assessed.

Management

Other than lead toxicosis, there is no specific therapy but some birds that are given supportive care may recover over a period of weeks or months.

Barraband (or *Polytelis*) paralysis syndrome

Barraband (or *Polytelis*) paralysis syndrome, seen most commonly in the Barraband or Superb parrot (*Polytelis swainsonii*), is a now uncommon condition similar to lorikeet paralysis syndrome. No known cause has been identified. With supportive care some birds will recover, but recurrence is seen occasionally.

Bilateral leg trauma

Definition/overview

Fledgling parrots with nutritional secondary hyperparathyroidism may present with bilateral pathological fractures of the legs.

Management

Surgical therapy may be warranted, but the prognosis is guarded.

Lead toxicosis

This may present as a bilateral paresis.

UNILATERAL PARESIS OR PARALYSIS

Unilateral trauma

Unilateral trauma, either skeletal or soft tissue, may result in a unilateral paresis. Affected birds often lie in sternal recumbency with the wing on the affected side extended as a support to that side of the body. This is sometimes mistaken for wing trauma.

Sciatic nerve compression

Definition/overview

Sciatic nerve compression by a renal tumour (nephroblastoma or adenocarcinoma) is seen occasionally in budgerigars, but rarely in other species. The sciatic nerve, as it comes off the spinal cord and passes between the middle and caudal divisions of the kidney, is compressed by the tumour.

Clinical presentation

Affected birds show unilateral paresis and may also have polyuria, polydypsia, a distended coelom and hyperuricaemia, depending on the extent of renal involvement.

Management

An anecdotal report of treatment with carboplatin indicated a short-term response only. Radiation therapy has yet to be evaluated for efficacy with this disease. At the moment, the prognosis remains poor for patients diagnosed with renal tumour.

Spinal neoplasia

Spinal neoplasia may produce a unilateral paresis/paralysis, although bilateral effects are more common.

HYPERKERATOSIS

Definition/overview

This is seen in most species, but canaries and poultry are the most commonly reported.

Downloaded From Vet-eBooks.com

Aetiology
Usually associated with *Cnemidocoptes* infection (see Chapter 11, Disorders of the Beak and Cere p. 192), but there is some suspicion that a nutritional deficiency (possibly zinc and/or biotin) associated with all-seed diets may be involved in some cases. Secondary bacterial and fungal infections may contribute to the pain sometimes seen with this condition.

Clinical presentation
The scales and skin on the podotheca become hyperkeratotic and may be painful.

Management
Treatment with ivermectin or moxidectin, combined with correction of nutritional deficiencies, is usually curative. In refractory cases, biopsies and/or culture may be needed to identify secondary pathogens.

SELF-MUTILATION OF THE FEET AND TOES

Definition/overview
This is a complex problem, with no simple answers or solutions. Affected birds will bite and chew at their feet and toes, sometimes to the point of amputating their own toes.

Aetiology
Some of the suggested/proven causes include: dermatitis; allergic dermatitis; underlying pain due to conditions such as osteomyelitis, healing fractures, soft tissue infection or inflammation, tendonitis, arthritis; chemical irritants (e.g. nicotine); vasculitis (e.g. immune-mediated, frost bite); obsessive-compulsive disorders; or neuritis and neuralgia.

Neuritis is the inflammation of a nerve or group of nerves characterised by pain, loss of reflexes and atrophy of the affected muscles. It may be viral (e.g. PDD), traumatic (e.g. entrapment of the superficial nerves by a fracture or external skeletal fixator) or toxic (e.g. lead). Other aetiologies undoubtedly exist but are, at this time, obscure. In humans the causes of neuralgia are varied and can include chemical irritation, inflammation, trauma (including surgery), compression of nerves by nearby structures (e.g. tumours) and infections. In many cases, however, the cause is unknown.

Neuralgia is pain that follows the path of a specific nerve. It is suspected, but not yet proven, in some birds that self-mutilate their feet and toes.

Management
Wherever possible, causative agents and secondary infections should be identified and eliminated. The bird should be prevented from mutilating the area through the use of bandages or Elizabethan collars. NSAIDs can be given to reduce inflammation and provide analgesia. Gabapentin has been used to treat neuralgia in humans and small mammals. Limited use in birds suggests that it may be a useful medication for self-mutilation disorders.

TOE TAPPING IN ECLECTUS PARROTS

Definition/overview
Toe tapping is the bilaterally symmetrical rhythmic extension and contraction of the digits of both feet, manifested when the bird is at full rest. Wing tip 'flipping' may also be seen in some individuals – an apparently involuntary flicking movement of the wing tips. When stimulated, these 'toe tappers' are able to stop the behaviour. Self-inflicted trauma is not a feature of this condition.

Aetiology
Numerous aetiologies have been suggested including post-polyomavirus neuritis, nutritional deficiencies and lead toxicosis. Over-supplementation with vitamins appears to be emerging as the 'favourite' aetiology. A dietary history usually reveals the excessive use of vitamin supplements, often in conjunction with a formulated diet. This theory is not yet proven.

Management
Discontinuing the supplementation often resolves the problem. In some cases, reducing the amount of formulated diet or simply changing brands is also required. As more research is done into this condition, it is likely that more appropriate therapies will be developed.

Downloaded From Vet-eBooks.com

LEG BAND CONSTRICTION

Definition/overview

Swelling and vascular necrosis of the distal foot occurs due to the tourniquet effect of a constricted leg band (**Fig. 14.8**).

Aetiology

This constriction develops as the result of applying too small a band to a growing bird; applying a soft 'split band' (usually made of aluminium) that is chewed on by the bird, causing it to overlap and constrict (**Fig. 14.9**); accumulation of scale and debris under the band (this is sometimes seen in budgerigars with *Cnemidocoptes* infection); or trauma and resultant swelling of the leg, with the (once loose) band becoming too tight (see **Fig. 3.20**, Chapter 3, The Physical Examination p. 77).

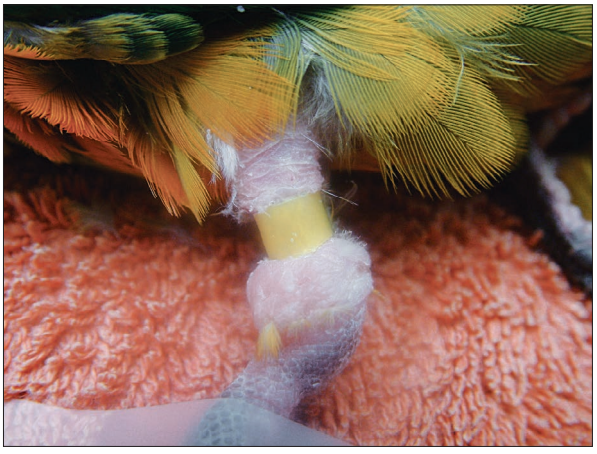


Figure 14.8 **Sun conure with a leg band constriction due to a plastic band which had moved upwards to the tibiotarsus.**



Figure 14.9 **A damaged 'split band' aluminium leg band after removal. Note the overlapping that had led to leg constriction.**

Clinical presentation

Initially there is pain (shifting lameness, chewing at foot), followed by swelling and more pain. Eventually the foot may necrose and 'fall off'.

Management

Treatment in the early stages requires removal of the band, followed by a supportive bandage and NSAIDs (e.g. meloxicam). However, owners need to be advised that constriction of the periosteum of the tarsometatarsus often leads to avascular necrosis of the bone below the band. In these cases, and where there is significant superficial necrosis, amputation of the leg may be required. In some cases the ring is all that is holding the foot on to the leg; removing the band results in the foot coming away with it. In rare cases, removal of the ring and reperfusion of the distal limb may result in acute hyperkalaemia, with subsequent cardiac arrhythmia and death.

TOE NECROSIS

Aetiology

Any condition that compromises the circulation within the toe can result in necrosis and loss of the toe. These conditions include ergotism, fibre constriction, deep infections of the skin, tendons or bone (bacterial or fungal), frost bite (see below), trauma, neuritis leading to self-trauma, vasculitis, and contact dermatitis.

Clinical presentation

Initially affected toes will often be swollen and appear discoloured. The bird may exhibit lameness, 'foot-shaking' behaviour, or chew at its foot. As the condition progresses the toe will become dark and dry, eventually falling off if not chewed off by the bird or amputated (**Fig. 14.10**).

Diagnosis

In the early stages the bird should be +anaesthetised to allow detailed examination of the foot for fibrous

Downloaded From Vet-eBooks.com



Figure 14.10 **Cockatoo with toe necrosis leading to complete loss of a digit.**

constrictions. Radiology may demonstrate underlying bone lesions. Biopsy (sometimes via amputation of an affected toe is multiple toes are affected) is often rewarding. If circulation is thought to have been lost, cutting the nail through the nail bed or pricking to distal toe with a needle can establish of circulation is intact or lost.

Treatment

In the early stages the restoration of circulation is vital if the toe is to be saved. If the circulation appears to be intact isoxsuprine, a beta-adrenergic agonist, can be used as a vasodilator. It appears to be more functional on tissue that is relatively healthy than tissue that is significantly damaged. Another drug that may be of benefit is pentoxifylline; this drug improves blood supply through inflamed vessels by reducing leukocyte adhesion to vascular endothelium and by making red cells more 'pliable', allowing them to pass through damaged vessels. It is more effective in damaged tissue, probably doing relatively little for the blood supply to healthy tissue.

Other treatments are determined by the result of other diagnostic testing.

If the circulation is lost, amputation is the best option.

FROST BITE

Aetiology and pathogenesis

Frost bite is a localised tissue injury following the freezing and thawing of soft tissue. It can be a common problem in birds in colder climates, often when the onset of cold weather is unexpected, when the birds are on metal surfaces (e.g. wire), or when there is pre-existing circulatory compromise to the toes and feet. Non-native birds housed in cold climates may also be at greater risk.

The pathophysiology involves both the initial freezing injury and secondary vascular impairment. The initial cooling of the tissue results initially in vasodilation (often pulsatile in nature) followed by vasoconstriction as the cooling develops into freezing. Damage to the affected area therefore results from both direct cellular damage and from ischaemia; the latter is often more significant than the first.

The damaged tissue releases inflammatory mediators, which trigger further vasoconstriction, platelet aggregation, and thrombosis, leading to a cycle of further microvascular damage, hypoxia, tissue damage, and more inflammatory mediator release. This cascade may not be just limited to the damaged extremity; death associated with aseptic vegetative valvular endocarditis is well recorded in birds with frost bite.

Diagnosis

A history of exposure to cold, combined with evidence of pain (lameness, self-mutilation) and a proprioceptive deficit in the affected limbs, can lead to a presumptive diagnosis before physical changes become obvious. After 24 hours oedema and swelling of the toes may become obvious, but the line of demarcation of necrosis may take several weeks to become evident.

Treatment

It is essential that treatment be initiated as soon as possible, before necrosis is evident. There are three goals in treatment: maintaining optimal blood supply to affected tissue; avoiding secondary infections; and providing analgesia.

If the tissue is still frozen, rapid re-warming in a warm water bath is recommended over gradual re-warming. Avoid massaging the affected area, as this is likely to cause further tissue damage. Non-steroidal anti-inflammatory drugs may reduce the inflammation and inhibit the release of inflammatory mediators. Pentoxifylline improves blood

Downloaded From Vet-eBooks.com

supply through inflamed vessels by reducing leukocyte adhesion to vascular endothelium and by making red cells more 'pliable', allowing them to pass through damaged vessels. Isoxsuprine, a beta-adrenergic agonist, can be used as a vasodilator but it appears to be more functional on tissue that is relatively healthy than tissue that is significantly damaged. As such, its use may be limited to the first few days after injury. Topical aloe vera cream may be of benefit, although its mode of action remains obscure.

Secondary infections with either *Clostridia* spp. or normal skin flora can be quite common. The use of antibiotics directed at these bacteria, such as amoxicillin-clavulanic acid, for several weeks is recommended.

Frost bite can be an extremely painful condition and, as such, it is appropriate to provide analgesia through the use of opioids and tramadol in addition to the use of non-steroidal anti-inflammatories.

Surgical debridement or amputation should be delayed until the line of demarcation becomes obvious. This may take 3–6 weeks.

OVERGROWN NAILS

Aetiology

Nails may overgrow when they fail to wear normally (inappropriate perch size or failing to grip properly due to toe or foot abnormalities), when the bird is malnourished or has chronic liver disease, or with diseases such as PBFD.

Clinical signs

The nails become overgrown to an extent that the bird is unable to grip a perch or walk normally. In some cases the nails may become twisted and curled.

Treatment

Affected nails should be trimmed regularly using nail clippers (small birds) or a rotary hobby grinder (e.g. Dremel®). Predisposing factors should be identified and corrected or removed.

MISSING NAILS AND TOES

Aetiology

It can be due to aggressive cage mates; unsafe caging or cage furniture; ergotism; toe constriction due to fibrous band formation (constricted toe syndrome) or foreign bodies (e.g. cotton thread); or frost damage.

Management

This is usually diagnosed after the event and treatment is rarely required. Birds that are missing toes or nails rarely appear to be handicapped.

FURTHER READING

Echols MS (2007) Avian kidney disease, Part I: types of renal disease. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 101–116.

Echols MS (2007) Avian kidney disease, Part II: diagnosis of renal disease. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 117–128.

Echols MS (2007) Avian kidney disease, Part III: treatment of renal disease. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 129–137.

Quesenbery K (1997) Disorders of the musculoskeletal system. In: *Avian Medicine and Surgery*. RB Altman, *et al*. (eds). WB Saunders, Philadelphia, pp. 523–539.

Remple JD, Forbes NA (2000) Antibiotic-impregnated polymethyl methacrylate beads in the treatment of bumblefoot in raptors. In: *Raptor Biomedicine III*. JT Lumeij, *et al*. (eds). Zoological Education Network, Lake Worth, pp. 255–266.

Stanford M (2006) Calcium metabolism. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 141–151.

Wellehan JFX (2003) Frost bite in birds: pathophysiology and treatment. *Compendium on Continuing Education for the Practicing Veterinarian* **25**(**10**):776–778.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 15

DISORDERS OF THE MUSCULOSKELETAL SYSTEM

SKELETAL DISORDERS

HEREDITARY, CONGENITAL AND DEVELOPMENTAL

These may be caused by genetics; teratogens (e.g. organophosphates); malnutrition (either parental or the juveniles); malpositioning within the egg; or artificial incubation (problems with temperature, humidity, ventilation, and turning frequency).

Spinal bifida

Spinal bifida occurs sporadically in pet birds. Incomplete closure of the embryonic neural tube results in an incompletely formed spinal cord. In addition, the vertebrae overlying the open portion of the spinal cord do not fully form and remain unfused and open. This allows the abnormal portion of the spinal cord to protrude through the opening in the bones. There may, or may not, be a fluid-filled sac surrounding the open spinal cord.

Kyphosis

Flexion of the spine occurs due to collapse or malformation of a vertebra, usually a notarial vertebra. The condition can be congenital or acquired (as a result of metabolic bone disease or osteomyelitis). The result is a 'hunchback'; the spine is bent in a dorsal direction such that the tail may point almost perpendicular to the floor (see **Fig. 5.7**, Chapter 5, Diagnositic Imaging p. 99). There may be dyspnoea evident due to compression of the abdominal and thoracic air sacs

Scoliosis

Lateral deviation and curvature of the spine occurs due to apical malformations of vertebrae. In poultry it has been associated with genetics, melatonin deficiency through pinealectomy or continuous light exposure. The indication is that there is a threshold level of serum melatonin below which scoliosis may develop, probably in conjunction with other factors that have yet to be identified. Deficiencies of copper, manganese or vitamin B6 have been shown to aggravate the condition.

How this correlates with the occasional scoliosis seen in companion birds remains unclear, but it raises issues about parental nutrition and hand-rearing practices.

NUTRITIONAL/METABOLIC

See Chapter 14, Diseases of the Legs, Feet and Toes pp. 207–209.

Osteomalacia

Osteomalacia, or osteoporosis, is a similar condition to rickets, occurring in adult birds housed indoors and suffering from a vitamin D3 deficiency. There is a generalised thinning of the bone, with pathological micro- or gross-fractures and resultant pain (**Figs 15.1** and **15.2**).

Nutritional secondary hyperparathyroidism

See Malformations section of Chapter 14, Diseases of the Legs, Feet and Toes p. 207.

Osteopetrosis or polyostotic hyperostosis

Definition/overview

Osteopetrosis is the development of bone in the medullary cavity of the femur, ulna, radius, pectoral girdle and vertebrae. It is seen as increased radiopacity of these bones (**Figs 15.3a** and **b**). It should be

Downloaded From Vet-eBooks.com



Figure 15.1 Radiograph of an adult galah indicates osteomalacia. Note the bowing of the radius and ulna; a consequence of an all-seed diet.

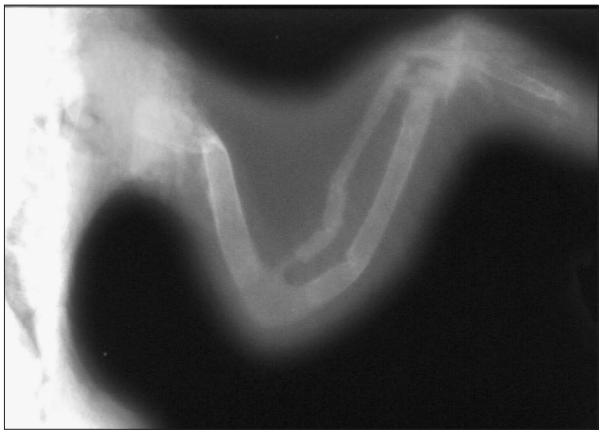


Figure 15.2 Radiograph of a juvenile cockatoo indicating folding fractures; when this severe, a guarded prognosis is necessary.

noted that that this is solid bone, not a loose matrix of calcium.

Aetiology

The deposition of this bone is stimulated by oestrogen. It may be non-pathologic (a normal change in the reproductively active hen, as this bone is the primary source of calcium for the formation of the egg shell) or pathological (associated with hyperoestrogenic conditions such as ovarian neoplasia and cystic ovarian disease).

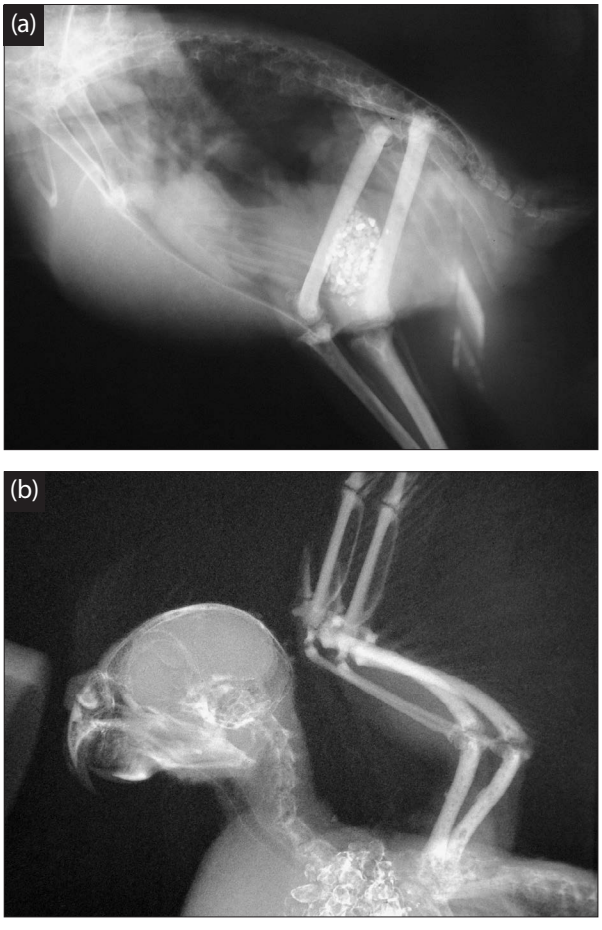


Figure 15.3 Radiographs (a) main body and (b) head and wings of a reproductively active budgerigar hen indicate osteopetrosis (polyostotic hyperostosis).

Management

Once the oestrogen levels return to normal, the bone density also returns to normal. In the normal reproductively active hen, no treatment is required. Pathological cases may require treatment for the inciting cause (see Chapter 23, Disorders of the Reproductive Tract p. 326).

Complications

Fractures of long bones that have a solid medullary cavity can be difficult to repair as, without a medullary and endosteal blood supply, there may be a poor blood supply to the fracture site. This frequently results in a non-union of the fracture. Repair of these fractures should include drilling out part of the medullary bone

Downloaded From Vet-eBooks.com

and utilising bone grafts. In medical and small animal orthopaedics the use of bone morphogenetic protein (BMP) shows great promise is stimulating healing of non-union fractures; it is possible that may be an avenue to be explored in avian medicine.

Osteomyelitis

Aetiology

Osteomyelitis may be a result of infection (localised or systemic disease) by bacteria (aerobic and anaerobic both implicated), fungi (*Aspergillus*, see **Fig. 15.4,** and *Candida*) or mycobacteria. It may also be the result of trauma or neoplasia.

Clinical presentation

There is soft tissue swelling around the lesion. The lesion is painful, sometimes shown as lameness or an inability to fly. There may be a fracture (and subsequent instability) associated with the infection.

Diagnosis

Radiographically there is usually clearly defined lysis of bone, as opposed to the more diffuse changes associated with neoplasia (**Fig. 15.5**). Note that mycobacterial infections can also be diffuse. The pus is caseous and does not drain; if an abscess forms, it enlarges to form an expanded outline within the bone, especially if it has formed within the medullary cavity. There is often a marked proliferative periosteal reaction.

Haematologically there may be a leucocytosis, with heterophilia, monocytosis and basophilia.

Management

Surgical debridement of the lesion is usually required to remove the caseated pus and necrotic bone. Antibiotic-impregnated polymethylmethacrylate beads, placed into the cavity left by the abscess after debridement and lavage, allow antimicrobials to be delivered locally in high concentrations. Antibiotics used include gentamicin, amikacin, tobramycin, lincomycin, clindamycin, neomycin, ceftiofur and cephalothin. Bacterial and fungal culture and antibiotic sensitivity should be performed, ideally before beginning treatment, to guide the choice of antibiotic therapy. With the exception of intra-articular placement, implanted beads can be left in place indefinitely.

Systemic antibiotic therapy should be directed by culture and continued long term (2–3 months).

Neoplasia

Aetiology

Neoplasia of skeletal structures can be either primary (osteomas, osteosarcomas, see **Fig. 15.6a**; chondromas; chondrosarcomas; osteochondroma; fibrosarcomas, haemangiomas) or metastatic (air sac carcinomas; other carcinomas).

Clinical presentation

The tumour usually presents as a firm swelling.

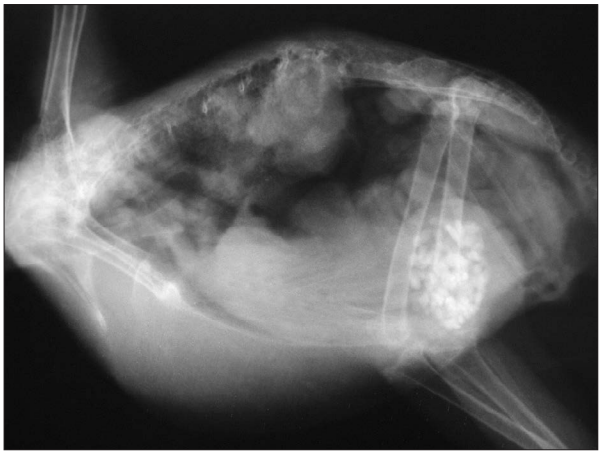


Figure 15.4 **Radiograph of an African grey parrot indicates spinal osteomyelitis and acquired kyphosis due to aspergillosis.**



Figure 15.5 **Radiograph of a pelican reveals osteomyelitis following a dog bite.**

Downloaded From Vet-eBooks.com

Diagnosis

Radiographically there are varying degrees of bone changes including proliferative lesions, osteolysis and pathological fractures (**Fig. 15.6b**).

Neoplasia must be distinguished from osteomyelitis. This may require deep bone biopsy using a fine needle aspirate or a bone biopsy needle (e.g. Jamshidi bone biopsy needle). In small birds an 18- to 22-gauge needle can be used to obtain a core sample: the needle is guided through the centre of the lesion from one side to the other, and then a stylet of sterile stainless steel wire is pushed through the needle to extrude the biopsy specimen. Care must be taken to sample the centre of the lesion, as proliferative bony reactions on the periphery of the lesion may mask the origin of the lesion.

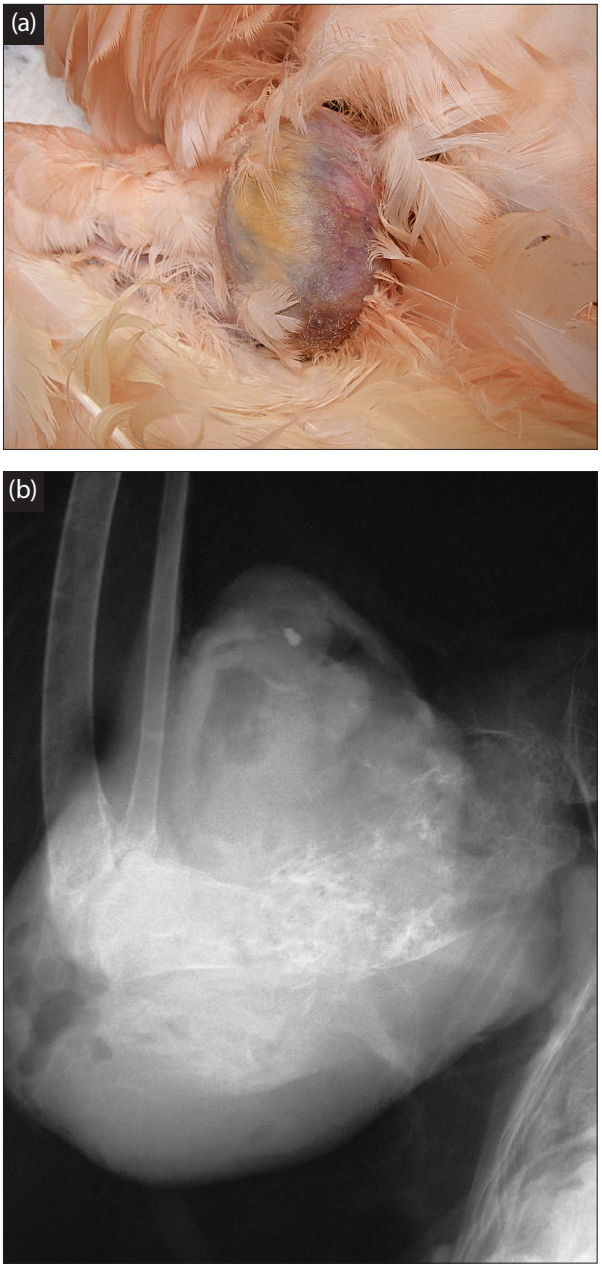


Figure 15.6 **An aged cockatoo with a humeral osteosarcoma (a) which, once imaged (b), suggests a poor prognosis due to the likelihood of metastatic disease.**

Management

Treatment usually requires amputation, followed by adjuvant chemotherapy and/or radiation.

JOINTS

Luxation

Definition/overview

All joints can be affected, but the femoro-tibiotarsal joint and elbow joints are most commonly affected.

Aetiology

Luxation may be the result of trauma or a congenital condition.

Clinical presentation

Localised swelling, pain and reluctance to use the affected limb are seen.

Diagnosis

Diagnosis is based on the clinical signs referable to luxation, and radiography.

Management

Conservative therapy is often unsuccessful because of the joint anatomy and powerful contraction of flight or leg muscles.

Surgical repair is often required. This is further described in Chapter 29, Surgery p. 417.

Prognosis

The prognosis for full return to normal use of the joint is fair to guarded; however, a pain-free leg can usually be achieved even if it is not fully functional.

Downloaded From Vet-eBooks.com

Swollen joints

See Chapter 14, Disorders of the Legs, Feet and Toes p. 210.

MUSCLE DISORDERS

CONGENITAL

Muscular dystrophy

This is seen in chickens and turkeys, but not in companion birds. Muscle fibres are lost and are replaced by fat.

Arthrogryposis

Congenital flexure or contracture of joints occurs secondary to atrophy of skeletal muscle and subsequent fibrosis of the muscle. This atrophy is due to congenital neurological defects, either anatomical deficits or following parental alkaloid plant toxicosis (e.g. lupines).

NON-INFLAMMATORY

Muscle atrophy

Aetiology

Atrophy may be caused by disuse (e.g. painful conditions of a limb, inability to fly, joint contraction following excessively long splinting of an injured limb or pressure on the sciatic nerve from a renal tumour). It may also result from ageing, and denervation caused by trauma or neoplasia.

Generalised chronic disease can cause muscle atrophy. Acute illness in birds sees a demand for energy greater than dietary intake. This triggers glycogenolysis, which quickly exhausts body stores of glycogen. Fat oxidation cannot meet the demands for energy and, after a few days, the process of gluconeogenesis using muscle protein (catabolism) begins. This results in muscle atrophy. The pectoral muscles are most noticeably affected, with the keel bone becoming more prominent. This condition is colloquially known as 'going light'. It is not a disease syndrome in itself, it is simply an indication of a chronic disease process.

Management

Disuse atrophy and denervation require identification and correction of the underlying problem, if possible.

Muscle atrophy due to chronic disease requires correction of the underlying disease problem, supplemental feeding (e.g. tube feeding for debilitated birds that cannot eat normally) and good-quality food (e.g. formulated diets to assist in the recovery and place the bird into an anabolic state). Recovery of the lost muscle mass is invariably slow, taking several days or weeks to return to normal.

Vitamin E and selenium deficiency

Aetiology

This deficiency is seen in piscivorous species fed a diet of improperly frozen and thawed fish. Any species fed a diet containing rancid polyunsaturated fat can develop the same condition.

Clinical presentation

Vitamin E and selenium deficiency can result in cardiomyopathy and muscle lesions, showing as generalised weakness.

Management

Recovery is usually rapid once nutritional deficiencies are corrected.

Ionophore toxicosis (monensin, lasalocid, salinomycin, narasin)

Aetiology

This condition is seen in non-gallinaceous birds fed ionophore-medicated poultry feeds. Ionophores are used in poultry as a coccidiostat.

Clinical presentation

Toxicosis is seen as generalised weakness and paresis due to damage to skeletal muscle.

Management

No specific treatment is available. The drug should be removed from the animal's diet and supportive care provided.

INFLAMMATORY: NON-INFECTIOUS

Trauma

Aetiology

Muscle inflammation may be caused by blunt trauma (e.g. flying into objects such as ceiling fans or aviary

Downloaded From Vet-eBooks.com



Figure 15.7 **Adult quaker parrot with severe necrosis and mutilation of the pectoral muscles. This bird had quaker mutilation syndrome but the cause of this problem remains unclear.**

wire) or iatrogenic trauma (e.g. intramuscular injections, especially enrofloxacin, doxycycline and oxytetracycline).

Clinical presentation
Bruising and swelling can be seen and this may develop into necrosis or fibrosis with chronicity.

Management
Severe or painful muscle injuries (**Fig. 15.7**) should be treated with an analgesic (e.g. meloxicam, tramadol or butorphanol).

Exertional/capture myopathy
Definition/overview
This is usually seen in flamingos, crowned cranes, Canada geese, Sandhill cranes and wild turkeys (USA) that have been recently captured or handled.

Clinical presentation
Muscle necrosis and intramuscular haemorrhage are commonly noted, followed by fibrosis with chronicity.

Diagnosis
Diagnosis of exertional myopathy is based on history of recent capture or trauma, clinical signs and elevation of AST, ALT, CK, LDH and serum potassium.

Management
Treatment includes fluid therapy, vitamin E and physiotherapy. The use of corticosteroids has been advocated by some authors; its benefits must be weighed against possible adverse effects on the liver and immune system.

Prognosis
Prognosis in severe cases is poor.

INFLAMMATORY: INFECTIOUS

Viral
Viral myositis is uncommon. Acute polyomavirus infections may cause muscle pallor and haemorrhage.

Bacterial
Bacterial myositis is usually an extension of an infection in the skin or underlying bone; however, foreign bodies (e.g. microchips implanted without attention to sterility) can introduce an infection. The infection may be aerobic or anaerobic.

Fungal
Fungal myositis usually occurs as an extension of infection from air sacs or by haematogenous spread. It is similar in appearance to bacterial myositis and granulomas and can only be distinguished histologically.

PARASITES

Sarcocystis
Aetiology
Caused by *Sarcocystis* spp., a parasite of many species of birds, with a worldwide distribution. There are believed to be six species of *Sarcocystis* that infect birds. They have an obligate two-host life cycle that alternates between a sexual intestinal phase in the definitive host and an asexual multi-organ (especially the lungs) and ultimately muscular cyst phase in the intermediate host. The definitive host is usually a carnivore that eats an animal (the intermediate host) with mature cysts in its muscles. The intermediate host is usually an herbivorous or insectivorous bird. Transmission to the intermediate host is usually mechanical, in the form of insects such as flies and cockroaches. There is no direct spread from bird to bird, although several birds in one collection can be infected simultaneously.

Downloaded From Vet-eBooks.com

Clinical presentation

While some intermediate hosts have evolved with both the parasite and the definitive host (and therefore do not develop clinical illness) naïve species exposed to *Sarcocystis* often develop clinical signs.

Acute pulmonary disease, due to asexual reproduction in the lung, causes severe dyspnoea and often acute death. Encephalitis can develop in parrots and raptors (posterior or unilateral paresis, intention tremors and head tilt). Cardiac disease can develop in some birds if cysts localise in the myocardium. Severe myositis can occur in some birds; other birds may have the cysts present, but display no clinical signs.

Diagnosis

Diagnosis in the live bird is difficult. Blood tests show elevated CK and AST, and leucocytosis. An immunofluorescent antibody (IFA) test is available in some countries. Muscle biopsy of the quadriceps muscles gives better results than the pectoral muscles because the muscular cysts there are less likely to degenerate before reaching maturity.

Management

For the surviving mate, pyrimethamine is given at 0.5–1 mg/kg orally q12h for 2–4 days then 0.25 mg/kg for 30 days. Treatment for affected birds includes the triazinone antiprotozoal drugs (toltrazuril, diclazuril and ponazuril), amprolium, primaquine phosphate and sulfadoxine/pyrimethamine,

Control involves keeping definitive hosts out of the aviary or immediate surroundings and utilizing effective insect control.

Leukocytozoon

Aetiology

Leukocytozoon is a protozoan parasite of erythrocytes and leucocytes, but with schizogony occurring in tissues other than the blood. Usually the parasite is non-pathogenic, but in some cases it can cause disruption of skeletal and cardiac muscles.

Clinical presentation

Clinical signs can include anorexia, weakness, haemoglobinuria, depression and dehydration.

Management

Chloroquine, primaquine or pyrimethamine may be used for treatment. Exposure to biting insects should be prevented.

NEOPLASTIC

Muscle neoplasia is uncommon in companion birds. It can be primary (rhabdomyoma; rhabdomyosarcoma) or secondary (lymphosarcoma; melanoma). Metastatic disease is rare.

TENDONS AND LIGAMENTS

Tendon contracture ('joint disease')

Aetiology

Enforced immobility of a joint in a flexed condition (e.g. when a wing or leg is splinted) can lead to contracture of the tendons controlling that joint. This can result in a permanent reduction in the joint's range of movement.

Management

Treatment requires a programme of gradual extension and extension of the joint, performed under general anaesthesia, to break down the fibrous adhesions causing the contracture. The prognosis for return to normal joint flexibility is guarded.

To prevent this problem, joints should be immobilised for no more than two weeks, if possible. If this is not possible, weekly physiotherapy should be performed (e.g. passive flexion and extension of the joint through its normal range of movement). This usually requires a short general anaesthetic to remove the splint, perform the physiotherapy and then replace the splint.

Slipped tendon (perosis)

See Chapter 14, Disorders of the Legs, Feet and Toes p. 210.

Tendonitis

Aetiology

Tendonitis may be infectious (may start in a joint and extend into the surrounding tendons) or non-infectious. Infectious causes include bacteria, *Mycoplasma* spp. and reovirus. Non-infectious causes

Downloaded From Vet-eBooks.com

include trauma, articular gout and tendon contracture (see above).

Clinical presentation

There is lameness or reluctance to use the limb, and localised swelling. Radiology may be required to rule out fractures or luxations.

Management

Inflammation is reduced with NSAIDs such as meloxicam. Antimicrobial therapy is given if appropriate.

FURTHER READING

Quesenbery K (1997) Disorders of the musculoskeletal system. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 523–539.

Stanford M (2006) Calcium metabolism. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 141–151.

Villar D, Kramer M, Howard L, Hammond E, Cray C, Latimer K (2008) Clinical presentation and pathology of sarcocystosis in psittaciform birds: 11 cases. *Avian Diseases* **52(1)**:187–194.

Downloaded From Vet-eBooks.com

DISEASES OF THE GASTROINTESTINAL TRACT

THE OROPHARYNX AND CROP

Candidiasis

Definition/overview

Candidiasis, also known as thrush, moniliasis, sour crop and crop mycosis, is most commonly due to an overgrowth of the yeast *Candida albicans*, which can be part of the normal intestinal flora and is frequently isolated from droppings of normal birds. It is primarily a disease of the upper gastrointestinal tract (oropharynx, oesophagus and crop), but can be found lower in the tract on occasion.

Predisposing factors to this overgrowth include: young birds that are not fully immunocompetent; prolonged antibiotic use; concurrent immunosuppressive conditions (e.g. debilitation, PBFD, malnutrition); poor hygiene in the bird's environment and food preparation; failure to clean excess formula from the skin or mouth of hand-reared chicks; high concentrations of sugar in fruit and hand-rearing formulae providing an optimal medium for the growth of yeast; and alkaline crop contents, seen when crop stasis occurs for other reasons, encouraging yeast overgrowth.

Clinical presentation

Affected birds show general malaise, weight loss and reduced growth rates. There may be regurgitation/vomiting. Crop emptying times are increased, the crop may be distended with fluid and mucus, and the crop wall becomes thickened. Diphtheritic membranes may be present in the oropharynx and crusty lesions can be seen on the commissures of the mouth. Infection can extend through the choana into the sinuses, causing signs of upper respiratory tract infection. It can also present as a dermatitis (see Chapter 10, Disorders of the Skin and Feathers, p. 168).

Diagnosis

Diagnosis is based on history, clinical signs and lesions, as culture alone may not distinguish between normal flora, dietary yeast and pathogenic overgrowth. Cytology (Gram stain) of throat swabs, crop washes and faeces may reveal the yeast in three different forms: oval non-budding yeast, budding yeast and pseudohyphae formation:

- Oval non-budding yeast are frequently of dietary origin.
- Large numbers (>5 per high powered field) of budding yeast are significant.
- The presence of pseudohyphae may indicate tissue invasion.

Gross lesions (**Fig. 16.1**) must be distinguished from pox virus, Trichomoniasis, hypovitaminosis A and internal papilloma disease (IPD).



Figure 16.1 **Lorikeet with candidiasis. Note the thickening of the ingluvial wall.**

Downloaded From Vet-eBooks.com

Management

Underlying factors must be addressed (e.g. environment, hygiene, husbandry, food handling, and concomitant antibiotic therapy). Antifungal therapy includes nystatin, ketoconazole, fluconazole or itraconazole. Nystatin is not absorbed systemically and must therefore be given into the mouth (rather than by crop needle) as it requires direct contact with the yeast to be effective. Acidification of crop contents by adding apple cider vinegar to drinking water or hand-rearing formula (10 ml/litre of water) may decrease crop pH and discourage growth of yeast.

Trichomoniasis

Definition/overview

Trichomoniasis is also known as canker (pigeons and budgerigars) or frounce (raptors). It is most commonly seen in budgerigars, lorikeets, pigeons and raptors, but has been reported in most species. It has a worldwide distribution.

Aetiology

The causative organism is *Trichomonas* spp. (*T gallinae* and *T columbae*), a protozoan parasite with an undulating membrane and four anterior flagella (see **Fig. 7.11**, Chapter 7, Interpreting Diagnostic Tests p. 138). It has a direct lifecycle, with the protozoa being passed from bird to bird via feeding (courtship behaviour, feeding young) or via contaminated drinking water (faeces, crop secretions). The organism dies quickly outside the host.

Pathophysiology

Following ingestion of the protozoa they remain free-swimming in the bird's saliva and crop fluids. Although they do not attach to the mucosa they will cause inflammation, followed by a necrotic stomatitis and ingluvitis, often with diphtheritic membranes. This is often complicated by secondary bacterial infections.

Clinical presentation

Signs include unthriftiness and high mortality in young birds; gagging, regurgitation, vomiting; diarrhoea; ptyalism; weight loss; and diphtheritic membranes in the oropharynx, oesophagus and crop (**Fig. 16.2**).



Figure 16.2 **Budgerigar with ingluvial ulceration due to trichomoniasis. Note that seed is present in the tract from mouth to the lesion but not beyond that point. Starvation is a common cause of death.**

In raptors it may extend into the sinuses (causing a caseous sinusitis) or into the Eustachian tubes (causing a head tilt). Young pigeon squabs develop a visceral form of the disease involving the liver, gastrointestinal tract and navel (often referred to as 'wet canker').

Diagnosis

A crop wash or throat swab usually (but not always) shows motile protozoa. Rarely, there is a single lesion in the thoracic oesophagus causing clinical signs, but the protozoa are not readily detectable. These cases are difficult to diagnose antemortem. Motile protozoa are occasionally found in the faeces.

Management

Treatment with a nitroimidazole (metronidazole, dimetridazole, ronidazole, carnidazole) is usually effective. Treatment with antibiotics for secondary bacterial infection is often warranted. Some cases with severe ulceration and diphtheritic membranes will die despite treatment. In some raptors, surgical debridement of lesions may become necessary.

In pigeons, strategic treatment of the flock may be necessary (i.e. during the breeding season, at weaning, immediately prior to and after the racing season).

Downloaded From Vet-eBooks.com

The extent of treatment required in pigeons is the subject of some debate, as there is evidence that infection with lentogenic strains of *Trichomonas* spp. provides some protective immunity against velogenic strains.

Hypovitaminosis A

Definition/overview

Vitamin A is formed in the liver through the conversion of beta-carotenes (from vegetables and fruit) and retinol (from liver and fish oils). Deficiencies in vitamin A (or its precursors) result in keratinization of epithelial cells, causing squamous metaplasia of the mucous membranes of the oropharynx. The metaplasia blocks the ducts of the salivary and mucus glands, causing abscesses in these glands.

The author's experience is that birds that have evolved with high levels of beta-carotenes in their natural diet (i.e. fruit and vegetables) appear to be more susceptible to hypovitaminosis A than birds that have evolved a much poorer quality diet (e.g. seeding grasses). Birds such as eclectus parrots, Amazon parrots, African grey parrots and lorikeets are more likely to show clinical signs of deficiency than cockatiels and budgerigars.

Aetiology

Hypovitaminosis A is caused by all-seed diets.

Clinical presentation

White or pale swellings are seen in the inter-ramular region, beneath the tongue or elsewhere in the oropharynx. There is blunting of the choanal papillae. Ptyalism occurs and sinusitis is common.

Diagnosis

History and clinical signs are very suggestive. Differential diagnoses include Trichomoniasis, pox virus, candidiasis and IPD.

An aspirate of the swellings shows keratinised epithelial cells rather than inflammatory cells.

Management

Treatment includes parenteral vitamin A and diet correction. Surgical removal of masses may be required in extreme cases, but severe haemorrhage is likely to ensue.

Poxvirus infection

Birds with avian poxvirus infection may have diphtheritic membranes in the oropharynx. See Chapter 10, Disorders of the Skin and Feathers p. 171 for more details.

Internal papilloma disease

Birds with IPD may have papillomas in the oropharynx. This condition will be discussed in more detail later in this chapter.

Crop stasis

Definition/overview

Crop stasis, where the crop fails to empty, is commonly seen in pre-weaning chicks, but it can also be seen in adult birds.

Aetiology

There are several possible causes. Crop stasis may occur as a result of generalised ileus caused by systemic illness (e.g. severe renal disease), stunting, foreign bodies below the level of the crop, chilling, heavy metal toxicosis or dehydration. Conditions affecting the crop such as foreign bodies, an overstretched/atonic crop, infectious ingluvitis (e.g. *Candida albicans*, bacteria), fibrous food impaction or crop burns may also cause stasis. Dietary factors may also be involved (e.g. cold food, watery food, food that settles out, chronic overfeeding or overly dry food).

Clinical presentation

The crop fails to empty within six hours. It is palpably distended, often filled with fermenting food and water ('sour crop'). Regurgitation may occur, often on palpation of the crop. There are clinical signs of dehydration (sunken eyes, weight loss, wrinkled skin, thick saliva).

Management

If possible, the aetiological agent should be identified and eliminated. The crop should be emptied (by surgery if necessary) and lavaged with warm saline. Parental fluids are given (to correct dehydration) until crop motility is restored, and antimicrobials are given as appropriate. Motility modifiers (metoclopramide, cisapride, and fennel or cumin tea) may assist, but their efficacy is not universal.

Downloaded From Vet-eBooks.com

Once the bird is rehydrated, small, watery meals should be given often. Predigesting hand-rearing formulae with pancreatic enzymes can liquefy the diet without diluting it.

Chronically distended, non-responsive crops may require support with an elastic sling ('crop bra') or, as a last resort, crop reduction surgery.

Thermal injuries

Definition/overview

Thermal injuries are less common than in past years, as aviculturists have become aware of the problem. Novice hand rearers, however, still present chicks with this problem.

Aetiology

The most common aetiology is a hand-rearing formula that has been overheated in a microwave oven and then fed before it has cooled sufficiently. 'Hot spots' (small foci of super-heated food) may be present in a mix and be overlooked when (if) the food temperature is checked. Some cases may be due to chicks coming into contact with incandescent light bulbs or heating pads, particularly while the crop is distended after a recent feed.

Clinical presentation

In the early stages (1–2 days) affected chicks may be lethargic and refuse feeding. Physical examination may reveal mild to moderate crop stasis and erythema of the most prominent part of the ventral crop. After the initial stage of erythema, blanching of the affected tissue develops. A crust then forms over the area; when it lifts off a fistula is usually revealed, often with food leaking from it (**Fig. 16.3**).

Diagnosis

Thermal injuries must be differentiated from a crop perforation (see below).

Management

Surgical resection of the burn and repair of the crop and skin are necessary to effect a cure. However, surgery must be delayed until the fistula has formed and all devitalised tissue has become obvious (usually 4–7 days after the incident, see Chapter 29, Surgery p. 390 for more details). The crop has an incredible ability to stretch and even large crop resections seem to be well tolerated by most young birds. Subsequent feedings will obviously need to be reduced in volume depending on the post-operative size of the crop.



Figure 16.3 **Juvenile parrot whose outer crop wall has sloughed off following a crop burn.**

While waiting for the burn to become clearly demarcated, the chick must be given supportive care, including analgesia and antibiotic coverage, and small feeds given frequently so as to avoid distending the crop. Placement of an oesophagostomy tube may be necessary in some cases (see Chapter 4, Clinical Techniques p. 90).

Crop perforations

Aetiology

Crop perforation usually occurs when using a metal feeding tube. The tube perforates the crop because either the chick has an unrestrained feeding response and thrusts up against the tube or the person feeding the chick does so roughly or impatiently. Food can be deposited outside the crop and under the skin, in some cases without being noticed.

Clinical presentation

Early cases may be presented because of blood on the feeding tube when it is withdrawn. These birds are usually asymptomatic. More advanced cases may be presented because of apparent crop stasis, often with severe systemic illness. Distension is palpable in the crop region, but food cannot be aspirated with a feeding tube. The skin overlying the crop is often discoloured and gas may be palpated under the skin.

Downloaded From Vet-eBooks.com

Management
Surgical removal of the food deposited under the skin, followed by debridement and flushing of the subcutaneous tissues and repair of the crop injury, is essential. Prognosis is determined by the time lapse since the initial injury and the degree of sepsis present in the patient.

Ingluvoliths and other foreign bodies in the crop
Aetiology
Ingluvoliths are chronic concretions of starch and other materials that form in the crop. Other foreign bodies can be ingested by the bird or fed to it by its parents. They can include fibrous material, seed and nut husks, large pieces of food and pieces of wood.

Clinical presentation
Signs include regurgitation, gagging, vomiting, weight loss, crop stasis and ptyalism. Some birds are presented extremely emaciated and dehydrated; these birds have a guarded prognosis.

Diagnosis
Diagnosis is based on palpation of the crop and plain and contrast radiography.

Management
Endoscopic retrieval through the mouth is feasible in some cases. Ingluviotomy may be required to remove some objects (see Chapter 29, Surgery p. 389). Small ingluvoliths can be lubricated with liquid paraffin (mineral oil) and massaged until they break down into sludge or small particles that can pass through the digestive tract or be aspirated via a large-bore feeding tube.

PARASITES
Capillaria contorta (hair worm, thread worm)
Definition/overview
Capillariasis occurs in quail and pheasants. The worms burrow into the mucosa of the oesophagus and crop, creating tracts that fill with blood, producing hyperaemic streaks and some diphtheritic lesions. The lifecycle can be direct or indirect.

Clinical presentation
Clinical signs include anorexia, dysphagia, diarrhoea and weight loss.

Diagnosis
Diagnosis is by finding double-operculated eggs on faecal examination.

Management
Treatment is with an appropriate anthelminthic. Levamisole, ivermectin and moxidectin have all been used. It should be noted that resistance is common, and it is important that follow-up screening is performed to assess the efficacy of the treatment.

Spiruroid worms (Spiruroidea)
Definition/overview
Spiruroid worms occur in wild corvids and strigiformes, and are also reported in passerines and parrots. They produce granulomatous lesions in the oropharynx, crop and proventriculus, with a worm protruding. The lifecycle requires an arthropod vector.

Management
Treatment is with ivermectin or moxidectin, followed by manual removal of the worms if possible.

PROVENTRICULUS AND VENTRICULUS

FUNGAL INFECTIONS
Macrorhabdus
Definition/overview
Macrorhabdus ornithogaster, also known as avian gastric yeast or Megabacteria, is an anamorphic ascomycetous yeast, a large, Gram-positive, periodic acid–Schiff (PAS)-positive, highly pleomorphic yeast. It colonises the gastric isthmus of many species of birds; in large numbers it can cause a maldigestion disorder and proventricular ulceration. It is probably transmitted by the ingestion of faecal material, although parent birds may pass it to their chicks when feeding them.

Clinical presentation
Macrorhabdus is most commonly recognised in budgerigars, but it has been diagnosed in most species

Downloaded From Vet-eBooks.com

including parrots, canaries, ostriches and poultry. Clinical signs include weight loss and weakness, and polyphagia, although often the bird is grinding its food and then letting it fall from its mouth. In acute cases, especially in parrotlets, the birds may regurgitate food and there may be blood staining around the beak. Undigested seed may be passed in the droppings and melena may be seen in some cases.

Diagnosis

Faecal examination is used to diagnose *M. ornithogaster*. As it often washes off a slide during staining, it is important that a wet mount be examined under 40× magnification with the stage condenser nearly closed. Gram staining and other cytology stains shows variably staining large 'cigar-shaped' Gram-positive organisms. It is a long, straight, narrow rod with rounded ends, 2 µm to 4 µm wide and 20 µm to 80 µm long. On occasion, a single Y-shaped branch is found at one end. Most sick birds will pass large amounts of the organism, but asymptomatic birds may only shed the organism inconsistently. If doubt remains, either a Calcofluor-white stain, or PCR can be used to confirm the presence of the organism.

Necropsy findings include a dilated proventriculus (**Fig. 16.4a**), ulcerations of the proventricular–ventricular isthmus (**Fig. 16.4b**) and large numbers of the organism in mucosal scrapings (**Fig. 16.4c**).

Management

The recommended treatment is Amphotericin B, given at 100 mg/kg twice daily by crop gavage for 30 days. Resistance may develop, and some birds will relapse after treatment. Iodine preparations, luphenuron, ketoconazole, terbinafine, and itraconazole have been shown to be ineffective; fluconazole is toxic to budgerigars at the doses needed to be effective against Macrorhabdus; nystatin may be effective in some birds, but resistance is likely.

Flock treatment is problematic; a water-based formulation of Amphotericin B (Megabac-S, Vetafarm, Wagga Wagga, Australia) may not clear a flock, and may lead to the development of resistant strains. Sodium benzoate has been used in some flocks with variable results – palatability is poor, and it appears to be toxic in finches and canaries.



Figure 16.4 **Dissection of a gastrointestinal tract from a cockatiel reveals a dilated proventriculus (a), and ulcerations in the proventricular–ventricular isthmus (b) both suggestive of *Macrorhabdus* infection. A stained mucosal scrape confirms *Macrorhabdus* presence (c). Note the 'cigar-shaped' organisms present on the right-hand edge of this image.**

Downloaded From Vet-eBooks.com

As *Macrorhabdus* does not appear to be egg-transmitted, artificial incubation and rearing of chicks in isolation can produce 'clear' birds as a basis for a *Macrorhabdus*-free flock.

Candidiasis

Candidiasis occasionally causes ventriculitis in small passerines (see above).

Zygomycetes and *Aspergillus* spp.

Definition/overview

These fungi (*Rhizopus*, *Mucor*, and *Absidia* spp., and *Aspergillus* spp.) are ubiquitous, usually fast-growing fungi. They will occasionally invade the mucosa and muscular layers of the proventriculus and ventriculus (**Fig. 16.5**). They may be confined to the gastrointestinal tract or disseminated widely through the body, possibly haematogenously.

Clinical signs

Affected birds are usually presented for vomiting/regurgitation and weight loss.

Diagnosis

Antemortem diagnosis is difficult, although endoscopic biopsy and brush cytology may be rewarding. Radiology using contrast may demonstrate thickening of the proventricular wall, and fungal granulomas elsewhere in the body.

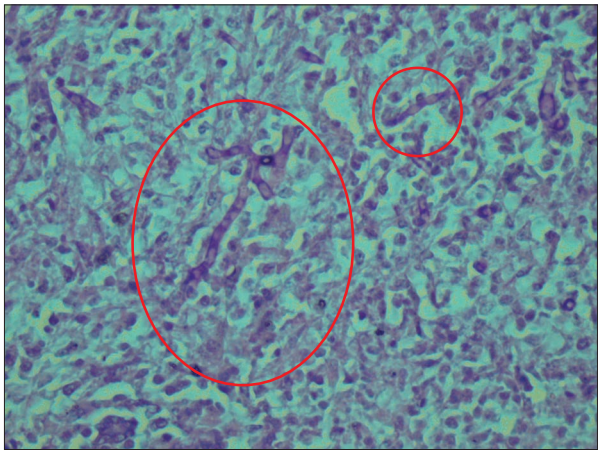


Figure 16.5 **Stained tissue sample of proventricular tissue from a green-cheek conure shows fungal hyphae (circled) invading mucosal cells.**

Management

Long term treatment (1–2 months) with systemic antifungal drugs (fluconazole, itraconazole, ketoconazole) is required.

Viral infections

Proventricular dilatation disease

Definition/overview

Proventricular dilatation disease (PDD), also known as macaw wasting disease, macaw fading syndrome, myenteric ganglioneuritis, infiltrative splanchnic neuropathy and neuropathic gastric dilatation, is a progressive fatal neuropathy. It has a worldwide distribution, and has been reported in most psittacine species and suggestive lesions have been described in toucans, honey-creepers, canaries, weaver finches, Canada geese and roseate spoonbills. It usually affects adult birds but it has been reported in chicks as young as two weeks.

Aetiology

Research since 2008 has confirmed that the aetiological agent is a previously unknown avian bornavirus (ABV), a negative-encoded, single-stranded, non-segmented RNA virus of the order Mononegavirales. There are nine genotypes of which two, ABV4 and ABV2, are the most common in parrots around the world. ABV-C, seen in canaries, has recently been described, as has ABV-CG in Canada geese in North America.

It must be noted that not all birds infected with ABV go on to develop PDD, as many apparently healthy birds carry ABV for prolonged periods of time.

The virus appears to be shed predominantly in urine, and transmission is likely to be via ingestion of faeces and urine. Aerosol transmission is suspected, but not proven. Vertical transmission appears to be possible, but has not yet been conclusively proven. Cockatiels may be asymptomatic carriers of ABV.

Pathogenesis

PDD is characterised by the presence of lymphoplasmocytic inflammation of the brain and nerves, especially in the enteric ganglia and nerve plexuses. Although the anterior gastrointestinal tract is most commonly affected, these inflammatory infiltrates

Downloaded From Vet-eBooks.com

can be found in other nerves including the brachial, sciatic and vagus nerves. It is believed that the lesions seen are the result of the action of cytopathic T-cells rather than a direct cytopathic effect of the virus. The role of antiganglioside antibodies has not yet been fully determined.

Clinical presentation

The incubation period of ABV is extremely variable, with periods as short as 11–14 days in unweaned chicks and months to years for older birds having been reported. It appears that, in most adult birds, the incubation period is 1–3 months.

PDD is a segmental neuropathy, with clinical signs dependent on the nerves (and therefore the innervated organs) affected. Most commonly clinical signs are related to gastrointestinal dysfunction: passing whole food in droppings (see **Fig. 3.11**, Chapter 3, The Physical Examination p. 70); regurgitation; weight loss; anorexia, lethargy and depression.

Occasionally CNS and peripheral nervous system signs are seen including ataxia, abnormal head movements, progressive paresis leading to paralysis, seizures, and proprioceptive or motor deficits. Lesions in the eye, when present, are characterised by moderate to severe perivascular cuffing in the optic nerves and in the choroid, ciliary body, and occasionally in the iris and pecten. This can result in severe retinal lesions and blindness. On rare occasions PDD causes sudden death due to an effect on the conduction pathways of the heart.

Secondary infections in the dysfunctional gastrointestinal tract are common.

Diagnosis

Although, in classical cases, the clinical signs of PDD are highly suggestive of the disease, clinicians should strive to obtain a definitive diagnosis in order to advise their client on prognosis and risk to other birds.

Haematology and biochemistry are often unrewarding, with no consistent changes noted in either. Some birds may have a non-regenerative anaemia of chronic disease.

In classical cases radiology will demonstrate a dilated, thin-walled proventriculus. This can be better defined through the use of contrast media. This finding is, however, not pathognomonic for PDD and can also be seen with heavy metal toxicosis and gastrointestinal foreign bodies.

Contrast fluoroscopy is a useful tool to demonstrate impaired movement of contrast through the tract. In the normal parrot, boluses of ingesta leave the crop and travel unidirectionally along the distal oesophagus to the proventriculus at an approximate rate of one bolus per minute. Little or no barium remains in the oesophagus between boluses. The proventriculus is relatively immobile, but a large contraction followed by partial emptying into the ventriculus should be seen every few minutes. Ventricular movement, often described as 'washing machine-like', is the constant grinding and mixing of ingesta prior to emptying into the duodenum. Normal intestinal peristalsis is bi-directional and wave-like. Birds with PDD, by contrast, show little (if any) movement of the proventriculus and the normal movement of the ventriculus is replaced by a shallow and irregular flutter of the ventricular wall. Peristalsis of the small intestine may become fast and erratic or slower than usual.

A reverse transcriptase PCR (rt-PCR) has been developed to demonstrate the presence of ABV RNA. The selection of samples for PCR testing is critical to obtaining a meaningful result. A key consideration in collecting samples is the intermittent shedding of the virus. This necessitates the collection of pooled samples (cloacal swabs or faeces and urates) over a period of at least one week. Samples should be collected fresh and kept frozen till dispatched to the laboratory. Blood is not recommended as a diagnostic sample as viraemia is not a feature of this disease. Similarly, although some success has been reported testing feathers, this has yet to be fully evaluated.

Necropsy can provide reliable samples for testing. The vitreous of the eye, the brain, spinal cord, peripheral nerves, the adrenal glands and the major organs are suitable samples to submit. Immunohistochemistry and viral isolation are also alternate means of diagnosis. Serological tests, especially if run in conjunction with rt-PCR, may prove to be a sensitive test for ABV.

Downloaded From Vet-eBooks.com

If PCR or serology are not available, an alternate means of diagnosis is via biopsy. The organ sampled will determine the degree of sensitivity:

- Crop: the least accurate, but the easiest and safest to access (reports of its sensitivity vary from 66–72% to as low as 17%).
- Proventriculus: the most accurate, but sampling a dilated, thin-walled proventriculus is hazardous.
- Adrenal gland: accurate in cases affecting that area.

The biopsy specimen must include a blood vessel and associated nerves. The myenteric plexus in the tunica muscularis of the proventriculus and ventriculus contains more lesions. The segmental nature of the disease means that the pathologist must examine stepped sections of the specimen to ensure lesions are not overlooked. Histopathology shows a non-suppurative lymphoplasmocytic ganglioneuritis of central and peripheral nerve tissue, with associated myositis and atrophy of the gastrointestinal tract, leading to dilatation and impaction (**Fig. 16.6**) Adrenal glands show a lymphoplasmocytic infiltrate.

Necropsy usually reveals the characteristic dilated, thin-walled proventriculus, often distended with ingesta (**Figs 16.7a** and **b**). Multiple tissues, including the brain and adrenal glands, should be submitted for histopathology and, as mentioned above, for rt-PCR.

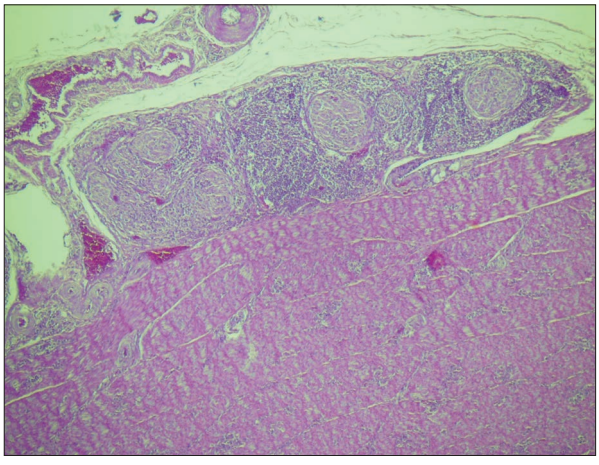


Figure 16.6 **Stained tissue sample of proventricular tissue from a bird with PDD showing lymphoplasmocytic infiltrate ganglioneuritis.**

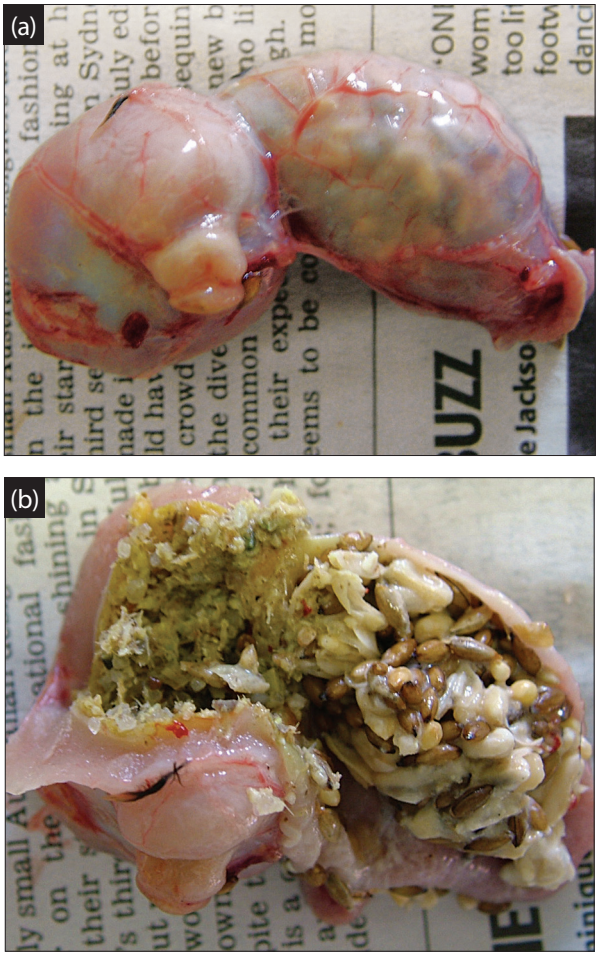


Figure 16.7 **An excised proventriculus from an Eclectus parrot who had PDD is clearly enlarged and dilated (a) and when cut open has a thin wall stretched over the contents (b).**

Treatment

There is 100% mortality in untreated birds diagnosed with PDD. Most birds that are treated also die, albeit months or years after their diagnosis.

Based on the inflammatory nature of this disease, it makes sense to seek to reduce this inflammation. The first treatment that was reported to be successful involved the use of the non-steroidal anti-inflammatory drug, celecoxib, a cyclooxygenase (COX)-2 inhibitor. Complete recovery in some birds, with return to normal function and biopsy showing resolution of the neuritis, was reported. Birds are given 10 mg/kg once daily for 6–24 weeks,

Downloaded From Vet-eBooks.com

with treatment cessation based on the bird regaining weight and resolution of lesions on biopsy.

Meloxicam, by contrast, may worsen the onset and severity of PDD and should not be used at this time. The use of other drugs such as cyclosporine and ribavirin is still in the developmental stage.

Cisapride, metoclopramide and high-fibre diets have all been used to assist gastrointestinal motility, with varying degree of success.

Control

As discussed previously, the most likely mode of transmission is the faecal–oral route, however aerosol and vertical transmission should not be discounted. Transmission appears to be more likely in indoor aviaries with poor ventilation and insufficient attention paid to traffic control. Control, therefore, includes:

- Testing of all birds in the flock.
- Quarantine and testing of all new and sick birds.
- Isolation and removal of birds that test PCR positive.
- Control of traffic.
- Good sanitation and thorough cleaning of all areas using detergents and suitable disinfectants.

It needs to be made clear to the client that the intermittent shedding of this virus may make detection difficult and re-testing may be required.

Parasitic infections

Finches, pheasants, quail and poultry are most commonly affected.

Acuaria spp. (gizzard worm)

Aetiology

Acuaria spp. (gizzard worm) are the most common worms involved. They include *A. skrjabini*, *A. humulosa* and *Dispharynx nasuata*. In canaries and finches these worms inhabit the proventriculus (causing swelling of the proventriculus mucosa) and under the koilin lining of ventriculus where they cause thickening of the ventricular wall. The intermediate hosts include weevils, grasshoppers, slaters and other insects.

Clinical presentation

Clinical signs are usually ill-thrift and death.

Diagnosis

Diagnosis can be made on necropsy, or by demonstrating the typical double-operculated eggs in the faeces.

Management

Levamisole, moxidectin and ivermectin can be used, but resistance is common, so treatment efficacy must be monitored. Control of intermediate hosts is important.

Foreign bodies

Definition/overview

A bird's curiosity and exploring nature will often lead it to ingest foreign bodies. The normal functioning of the ventriculus—the tougher koilin lining and the presence of grit, when combined with the grinding effect of the powerful ventricular muscles—effectively grinds up most of these foreign bodies within a few days. While the great majority of these foreign bodies cause little or no harm, others can cause gastrointestinal blockages, damage the gastrointestinal mucosa and release toxins such as lead and zinc, which are then absorbed and become a systemic problem.

Unless the bird is displaying clinical signs consistent with the foreign bodies causing a problem, most can be left and monitored to see if the body is removing them unaided. The addition of a small amount of grit to the bird's diet may assist this process. The administration of paraffin oil or psyllium may help to move smaller particles through the tract.

Clinical presentation

Clinical signs include anorexia, weight loss, regurgitation, decreased faecal output, melena and signs consistent with heavy metal toxicosis.

Diagnosis

Radiography, both plain and contrast, is useful. (The entire tract must be evaluated; don't identify a foreign body in the crop and overlook the one in the proventriculus!)

Haematology and biochemistry can be used to evaluate systemic changes.

Downloaded From Vet-eBooks.com

Management

If a foreign body needs to be removed, it is easier to remove it from the crop or proventriculus rather than waiting until it is in the ventriculus or intestinal tract. Endoscopy, either through the mouth or via an ingluviotomy, can be used to remove foreign bodies in the crop or proventriculus.

A proventriculotomy or ventriculotomy can be used to remove foreign bodies in the proventriculus and ventriculus. These surgeries have a high morbidity associated with them, and should not be undertaken lightly.

Smooth, non-penetrating foreign bodies in the crop, proventriculus or ventriculus may be removed by flushing:

- Anaesthetise the patient, intubate and pack the choanal slit with gauze. Place the patient in either ventral or dorsal recumbency.
- Pass a flexible, large-bore feeding tube into the thoracic oesophagus through the exit from the crop in the right lateral dorsal corner of the thoracic inlet. This can be done blindly or with guidance from endoscopy.
- Tilt the bird head down at an angle of 30°.
- Flush the proventriculus gently and steadily with warmed saline. The flush can be allowed to run out or it can be aspirated with gentle suction.
- Collect the flush to identify and count the number of foreign bodies retrieved.
- Repeat the flush until all the foreign bodies have been retrieved.

Neoplasia

Definition/overview

The most common gastrointestinal neoplasia are papillomas associated with IPD (see section, Disorders of the Cloaca) and gastric carcinomas, found at the proventricular–ventricular junction. Death from gastric neoplasia may be due to haemorrhage, gastric perforation and sepsis or endotoxic shock, or anorexia and subsequent catabolism.

Diagnosis

Diagnosis is by radiography, endoscopic biopsy or necropsy.

DISORDERS OF THE INTESTINAL TRACT

Ileus

Definition/overview

Ileus is decreased motility or complete stasis of the intestinal tract.

Aetiology

Causes include foreign body obstruction, decreased or absent neurological function (e.g. lead toxicosis, PDD), enteritis, peritonitis, pancreatic disease (e.g. zinc toxicosis) and intussusception or torsion of the intestine.

Clinical presentation

Clinical signs include regurgitation, dehydration, lethargy and decreased faecal output.

Diagnosis

Radiography shows fluid or gas-filled loops of intestine, often with proventricular dilation.

Management

Dehydration should be corrected, and causative factors identified and rectified. Motility enhancers such as ranitidine, cisapride or metoclopramide may be of assistance.

Parasites

Ascaridia (roundworms)

Definition/overview

Species identified include *Ascaridia hermaphrodita*, *A. columbae* (shared between pigeons and parrots), *A. galli* (shared between gallinaceous birds and parrots) and *A. platycercii* (restricted to parrots). They are most commonly seen in birds with access to the ground, and are particularly common in budgerigars, cockatiels, quaker parrots and princess parrots.

Pathophysiology

The worms are found in the small intestine of the affected bird, particularly in the duodenal loop (**Fig. 16.8**). They have a direct lifecycle.

Clinical presentation

Clinical signs include lethargy, poor condition, diarrhoea and death.

Downloaded From Vet-eBooks.com

Diagnosis
Diagnosis is by faecal examination (**Fig. 16.9**).

Management
Most anthelmintics are effective. Ascarids are resistant to disinfectants but susceptible to desiccation, steam and flames.



Figure 16.8 **An excised duodenal loop from a Princess parrot is found to contain large numbers of roundworms (*Ascaridia* sp.).**

Capillaria (hair worm, thread worm)
Definition/overview
Species identified include *Capillaria annulata*, *C. contorta* (which causes pseudomembrane formation in the oesophagus and crop [see above]) and *C. obsignata* (found in small intestine).

Pathophysiology
Capillaria imbed in mucosa, causing a marked thickening and inflammation of either the crop or the intestinal mucosa. The worms have a direct or indirect lifecycle, using insect vectors. Eggs are infective in two weeks. They can remain infectious in the environment for several months.

Clinical presentation
Signs include anorexia, dysphagia, diarrhoea, and weight loss.

Diagnosis
Faecal examination reveals a double-operculated egg (**Fig. 16.10**).

Management
Ivermectin, moxidectin or levamisole can be used. Anthelmintic response needs to be monitored, as resistance is common. Access to insect vectors should be controlled.

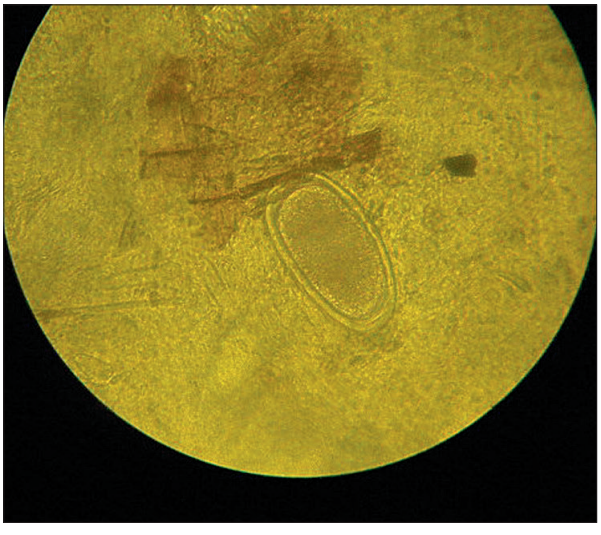


Figure 16.9 **Avian faecal smear containing a roundworm (*Ascaridia* sp.) egg.**

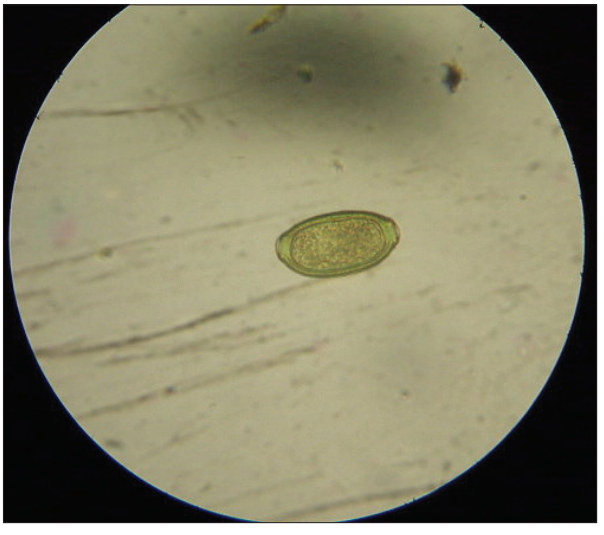


Figure 16.10 **Avian faecal smear containing a threadworm (*Capillaria* sp.) egg. Note the eggs are double-operculated.**

Downloaded From Vet-eBooks.com

Heterakis (caecal worm)

Definition/overview

Species identified include *Heterakis isolonche* in quail and *H. gallinarum*, which affects gallinaceous birds.

Pathophysiology

The lifecycle is indirect, through earthworms.

Clinical presentation

There are usually no clinical signs, but the worms are significant because they transmit *Histomonas* spp. (see below).

Management

Most anthelmintics can be used. If possible, access to intermediate hosts should be controlled.

Cestodes (tapeworms)

Definition/overview

Species identified include *Raillietaenia*, *Choanotaenia*, *Gastronemia*, *Idiogenes* and *Amoebotaenia*. Tapeworms are commonly found in finches and Old World parrots (African grey parrots, cockatoos, lorikeets and eclectus parrots). The lifecycle is indirect and may be complete in 3–4 weeks. Intermediate hosts include grasshoppers, beetles, ants, horseflies, earthworms, slugs, snails and crayfish.

Clinical presentation

Signs include catarrhal enteritis, emaciation and anorexia.

Diagnosis

Diagnosis is based on the finding, on faecal examination, of oncospheres (tapeworm embryos with six hooks).

Management

Praziquantel and fenbendazole may be used. Exposure to intermediate hosts should be controlled.

Coccidia

Definition/overview

Species identified are numerous, but include *Eimeria*, *Isospora* and *Caryospora* spp. This parasite tends to be host specific.

- *Isospora* oocysts have two sporocysts, each with four sporozoites. They are most common in parrots, passerines and Piciformes.
- *Eimeria* oocysts have four sporocysts, each with two sporozoites. They are most common in Galliformes and Columbiformes.
- *Caryospora* oocytes have one sporocyst and eight sporozoites. They are most common in raptors.

Pathophysiology

The lifecycle is direct. The bird ingests the sporulated oocysts lying in its environment. Oocysts undergo schizogony in the intestine (asexual reproduction, producing multiple parts). If the schizonts produced are deep in the intestinal mucosa, they may damage the mucosa when they divide, causing enteritis. After schizogony, they differentiate into male (microgametocytes) and female (macrogametocytes) forms. They then undergo sexual reproduction (gametocytic), which is non-pathogenic. This produces non-sporulated oocysts, which are shed in faeces. Oocysts sporulate in a warm, moist environment to become infective.

Coccidia require 6–8 days to complete the lifecycle, with clinical signs seen 4–6 days after infection.

Clinical presentation

Affected birds may not show clinical signs until stressed. Signs include lethargy, weight loss, diarrhoea (sometimes with blood and mucus), dirty vents and death.

Diagnosis

Oocysts can be found in faeces on smear and/or flotation. Note that clinical signs can be present before oocysts are detectable in the faeces.

Management

Anticoccidial treatments include amprolium, toltrazuril, ponzuril and sulphadimethoxine. Birds are treated daily for 2–3 days, then treatment is repeated after five days to treat those organisms that were in the prepatent period when the first treatment was given.

Access to infected faecal material is prevented by using concrete floors that are cleaned regularly, or having suspended wire floors. Food and water dishes should be kept off the ground.

Downloaded From Vet-eBooks.com

Cryptosporidia

Definition/overview

Cryptosporidia are enteric coccidians that usually lie just inside the limiting membrane of the enterocytes. They can also attach to proventricular epithelium, the respiratory epithelium, the conjunctival sac, the urinary tract and the bursa. The oocysts are the smallest of any coccidia, with four naked sporozoites and no sporocysts in oocytes.

Pathophysiology

Cryptosporidia develop intracellularly at an extracytoplasmic location on the apical surface of the epithelial cells. They are often secondary pathogens. Although not very host specific, they appear to be infective only to other birds, not to mammals. The lifecycle is direct. The oocysts are sporulated when passed and, as such, are fully infective immediately.

Clinical presentation

Cryptosporidiosis is often asymptomatic. Clinical signs may include depression, dehydration, anorexia, persistent diarrhoea, malabsorption problems, coelomic pain, vomiting, coughing, sneezing and nasal discharge.

Diagnosis

Diagnosis is based on histopathology using modified (Kinyoun's) acid-fast stains. Infective cysts may be seen on faecal flotation.

Management

Treatment is with paromomycin sulphate.

Giardia

Definition/overview

Giardia has a wide geographic and host distribution. Its zoonotic potential is unclear. It has been reported in most avian species, although it has not been reported in finches and canaries. It has a direct lifecycle, although cysts are shed only intermittently.

Clinical presentation

Signs include weight loss, depression, ruffled feathers and chronic diarrhoea. Persistent feather picking and pruritus may be seen in cockatiels (see Chapter 10, Disorders of the Skin and Feathers p. 180). Infected chicks may have poor growth rates; many die.

Diagnosis

Cysts or trophozoites can be detected in the faeces. As the trophozoites are not stable outside the bird, faecal examination should be performed on droppings within ten minutes of defecation.

Management

Nitroimidazoles are generally effective.

Treatment

Good hygiene is essential to break the lifecycle. *Giardia* cysts may survive in chlorinated water, but are inactivated by quaternary ammonium compounds.

Cochlosoma

Definition/overview

Cochlosoma is a motile protozoan that is common in waterfowl and finches. In Australia it is common in cockatiels. Bengalese (Society) finches can carry the parasite without clinical disease but when used as foster parents for Gouldian finch chicks, there is often severe loss of the chicks. It has a direct lifecycle, with transmission due to the ingestion of infective trophozoites in faecal material and contaminated food and water.

Clinical presentation

Clinical signs include diarrhoea, weight loss and death.

Diagnosis

Diagnosis is made on examination of a fresh faecal smear.

Management

Drug resistance is common. Nitroimidazoles are used, but the treatment must be monitored for efficacy. Care must be taken when fostering Gouldian finches with Bengalese finches, as Gouldian are very susceptible to this parasite.

Hexamita (*Spironucleus* spp.)

Definition/overview

This is a motile protozoa with eight flagella (six anterior and two trailing). It has been reported in pigeons (*Hexamita columbae*), game birds (*Hexamita meleagridis*), cockatiels, lorikeets, grass parrots, galahs

Downloaded From Vet-eBooks.com

and Australian king parrots. It has a direct lifecycle. Concurrent disease and parasitism are common.

Clinical presentation
In pheasants and pigeons it is most commonly seen at 6–12 weeks of age. It causes decreased appetite, lethargy, emaciation and intractable diarrhoea. Similar signs are seen in parrots.

Diagnosis
Diagnosis is made on examination of a fresh faecal smear; the parasite swims in a smooth linear fashion.

Management
Nitroimidazoles are used, but the treatment must be monitored for efficacy. Supportive therapy is essential, as many birds are emaciated, dehydrated and hypoglycaemic on presentation.

Histomonas meleagridis
Definition/overview
Histomonas meleagridis is a motile protozoan that affects turkeys, quail, peacocks and pheasants. Chickens and guinea fowl may harbour latent infections. The disease in turkeys and peacocks is known as 'blackhead'. The lifecycle is indirect, with transmission occurring through the ingestion of infected eggs of the caecal worm *Heterakis gallinarum*.

Clinical presentation
Clinical signs include weight loss, increased thirst, decreased appetite and brown–black to mustard-coloured diarrhoea. In chickens there may be blood in the droppings. There can be a high mortality rate, especially amongst juveniles.

Diagnosis
Diagnosis is based on clinical signs, identification of *Heterakis* eggs in droppings and, on necropsy, caecal cores (caecal necrosis) and hepatomegaly with multifocal yellow necrotic lesions).

Management
Treatment is with nitroimidazoles. Control of *Heterakis* is required (see above).

Microsporidiosis (encephalitozoonosis)
Definition/overview
Microsporidia spp. is an obligate intracellular protozoan, measuring only 1–2 µm that is Gram-positive and acid fast. It appears to have a direct lifecycle, probably through oral transmission. The aerosol route is also thought to be feasible. *Encephalitozoon hellum* has been isolated from eclectus parrots, Amazons, lovebirds, budgerigars and finches. It has also been isolated from humans, so there is possibly a zoonotic potential.

Athophysiology
There may be some element of immunosuppression involved in outbreaks (stress, overcrowding, or concurrent disease e.g. Pasteurellosis, PBFD). Latent infections may exist.

Clinical presentation
Signs include anorexia, lethargy, weakness, diarrhoea, stunting, ruffled feathers, weight loss and neonatal mortality. Chronic conjunctivitis and sinusitis, as well as exophthalmos, corneal oedema and blepharospasm, were reported in a double yellow-headed Amazon parrot.

Diagnosis
PCR may be available. Histopathology using silver stain or acid-fast staining is necessary, as standard stains may not reveal the organism.

Management
Although not proven, microsporidiosis should be considered zoonotic.

Treatment is with albendazole (50 mg/kg orally q24h for five days).

Mycobacteria
Definition/overview
Most avian infections are caused by *Mycobactrium avium* and *M. genevense*. *M. tuberculosis* and *M. bovis* are occasionally reported, but are considered rare.

Mycobacteriosis is most commonly an enteric infection, but it may be respiratory or disseminated. It is considered to be a leading infectious cause of avian deaths in many zoo parks with mixed species aviaries, where up to 14% of annual mortality can be attributed to mycobacteria.

Downloaded From Vet-eBooks.com

Although not conclusively proven, it should be considered a potentially zoonotic disease.

Pathophysiology

Transmission is by ingestion of infected faeces, either directly or via contaminated food and water. Insects may act as mechanical vectors. Pulmonary disease by the aerogenous route is rare. The incubation period following infection can be months or years. Following ingestion, the mycobacteria penetrates the mucosa and colonises under the serosa. It enters the blood supply and is removed from circulation by the liver, spleen and bone marrow. The lack of well-developed regional lymph nodes in birds may be one reason for the ease with which this disease becomes systemic. Lesions therefore develop in the intestinal wall, liver, spleen and bone.

Clinical presentation

Weight loss and severe muscle atrophy are the most consistent clinical signs. Coelomic distension, hepatomegaly and dilated, fluid-filled, thickened intestines may be palpable. Lameness, due to arthritis or osteomyelitis, may be present. Nodular or diffuse keratinous skin lesions at the mucocutaneous junctions of the eyes and beak have been reported in parrots.

Diagnosis

Antemortem diagnosis in birds is difficult and often inconclusive, particularly in the early stages. It can be based on clinical signs, marked leucocytosis with monocytosis and elevations in AST and bile acids. Radiographs may show an enlarged liver, kidney and spleen, as well as focal, cloudy densities in the medullary cavities of long bones with osteolysis. Endoscopy and biopsy are used to confirm the diagnosis.

Faecal acid-fast stains may reveal the organism but, as it is only shed intermittently, this is not an effective screening tool. An intradermal test can indicate the presence of mycobacteria in a flock, but it is unreliable in an individual. It is probably unreliable in non-poultry species. PCR performed on cloacal swabs may have value, but the results must be interpreted with caution. False positives and negatives are not uncommon.

Necropsy usually reveals granulomas in the liver (**Fig. 16.11a**), spleen and subserosa of the intestinal tract. There may be hepatomegaly, splenomegaly and enlarged, thickened intestines.

Histopathology shows a spectrum of lesions from nodules of large foamy histiocytes packed with acid-fast bacilli (**Fig. 16.11b**) to giant cell-containing granulomas that are often caseous but not cavitated or calcified. Amyloidosis is seen in approximately 20% of cases.

Management

Consideration must be given to whether to treat because of the possible zoonotic implications.

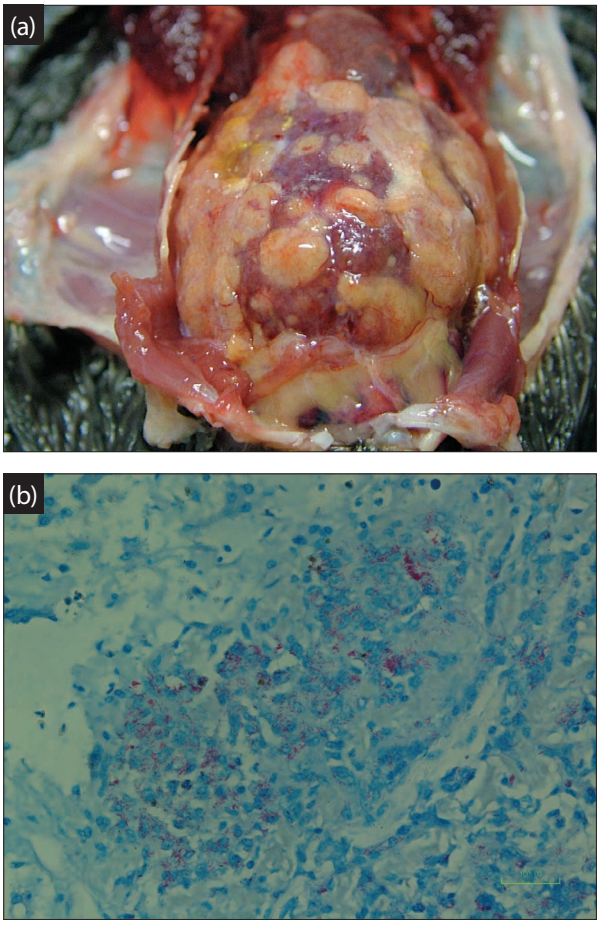


Figure 16.11 **Post-mortem examination of a Red Cap parrot with a mycobacterial infection is found to have disseminated granulomas throughout its liver (a) and a stained liver tissue smear confirms presence of acid-fast bacteria (Mycobacteria) (b).**

Downloaded From Vet-eBooks.com

Resistance develops rapidly, therefore combinations of drugs must be used. Treatment must be given for nine months or more, as the organism is only susceptible when replicating, which occurs every 16–20 hours, and even less in caseated lesions. One should aim to achieve the highest possible blood levels, not consistent levels, therefore once daily dosing is used. Commonly used combinations include isoniazid, ethambutol, rifampin and enrofloxacin.

Mycobacteria are resistant to typical physical and chemical destruction. They can survive for months to years in the environment.

Bacterial enteritis

Several bacteria are commonly implicated in bacterial enteritis in birds. Most are secondary pathogens, although some can be primary. They include *E. coli*, *Clostridium* spp., *Salmonella* spp. and *Chlamydia psittaci* (see Chapter 17, Disorders of the Liver p. 259). Many other species of bacteria can be involved, usually as secondary invaders.

Treatment is based on bacterial culture and sensitivity, and identification and elimination of predisposing factors and concurrent disease.

Escherichia coli

Definition/overview

E. coli is a Gram-negative, non-spore forming bacillus. It has a worldwide distribution and is a normal inhabitant of mammalian gastrointestinal tracts. Most *E. coli* strains produce endotoxins.

E. coli can be isolated from clinically normal parrots. It is debatable whether the isolation of *E. coli* from the cloaca of clinically normal parrots indicates a definitive pathogen, a potential pathogen or a commensal organism, or is merely transient. It is more common in cockatoos than in other parrots.

Aetiology

Infection with *E. coli* is primarily the result of inadequate hygiene and faecal contamination of water sources, food, perches, floors and the general environment.

Clinical presentation

Signs vary from chronic diarrhoea to severe generalised septicaemia (lethargy, anorexia, weakness, ruffled feathers, diarrhoea and death). *E. coli* can also cause respiratory infections, infertility, ingluvitis and arthritis.

Clostridium spp.

Definition/overview

The Clostridial organisms found in birds include *Clostridium perfringens* types A and C, *C. colinum* and *C. tertium*. They are anaerobic Gram-positive bacilli.

Pathophysiology

An abrupt change in diet may disrupt the intestinal microflora, allowing Clostridia to proliferate in the upper intestine. They can cause necrotic enteritis, 'gangrenous dermatitis' and malignant oedema.

Clinical presentation

Clostridial infection is often seen in juvenile cockatoos with concurrent enteric problems; they produce a characteristic foul-smelling diarrhoea.

In lorikeets it may present as an acute haemorrhagic diarrhoea, followed by death.

Diagnosis

Spore-forming bacteria are seen on faecal Gram stains. Severe necrosis and petechial haemorrhages are found in the mucosa of the duodenum and jejunum.

Management

Addition of apple cider vinegar (10 ml/litre of water) has been effective in treating juvenile cockatoos with a simple enteritis. Penicillin derivatives and metronidazole are usually effective in more severe cases.

Salmonella spp.

Definition/overview

Salmonella spp. are a (usually) motile, Gram-negative bacteria. *Salmonella* spp. are members of the family Enterobacteriaceae. There are over 2,100 serotypes, including the Arizona group. There are five subgenera; subgenus I is the most important to birds. Subgenus III (*S. arizonae*, *S. hinshawii*) is occasionally reported in birds, especially those in contact with reptiles. Two organisms are host specific and non-motile: *S. pullorum* (pullorum disease) and *S. gallinarum* (fowl typhoid). *S. typhimurium* is

Downloaded From Vet-eBooks.com

the most common parrot isolate. It is also the most common isolate in human beings with salmonellosis, making it important as a zoonosis.

Pathophysiology

Faeces of chronically infected carrier birds (often asymptomatic) are one of the most common sources of infection. *Salmonella* spp. can survive for extended periods in organic matter, faeces and dirt (up to two years). Ingestion of contaminated food or water or direct contact by aerosolisation of faecal or feather dust is the most common mode of transmission.

Improper husbandry and sanitation can spread the infection through the contamination of seed, fruits, vegetables, drinking water and food containers.

Salmonella spp. can be a primary pathogen; some serotypes can penetrate the mucosal barrier. Virulent strains can penetrate intact intestinal mucosa, while non-virulent strains require a mucosal lesion to enter the host. The incubation period can be as short as 3–5 days in acute cases.

Clinical presentation

The disease can be peracute, acute, chronic or subclinical depending on the number of organisms, their serotype and the age, species and condition of the host. Clinical signs include depression, lethargy, anorexia, weight loss, diarrhoea, a soiled vent, dehydration and crop stasis. Lameness and wing droop are associated with septic arthritis (swollen joints). In breeding birds with subclinical infections, there is often poor hatching or excessive fledgling mortality. Neurological signs may be seen, including convulsions.

Diagnosis

Diagnosis is based on necropsy findings: hepatomegaly, splenomegaly, air saculitis and pulmonary congestion, caecal cores (caecal necrosis) in game birds, ulcerative enteritis, orchitis and oophoritis.

Bacteria are present on histological sections and there is focal necrosis in the affected organ, with organisms found within the necrotic areas. Culture of the digestive tract or liver sections, and faecal culture can be used for organism identification.

Antemortem diagnosis can be difficult because of intermittent shedding. A serological test is available for poultry.

Management

Antibiotics are given based on culture and sensitivity. Treatment must be given for 3–8 weeks.

Control of vectors (wild birds, rodents) and prevention of contamination of food and water are required. Good hygiene practices must be implemented. A vaccine is available for pigeons, but it may not be fully protective. Birds should be vaccinated four weeks prior to a show or race season. Autogenous vaccines may be produced from faecal cultures.

DISORDERS OF THE CLOACA

Prolapse

Aetiology

Cloacal prolapse can be caused by excessive straining: masturbatory behaviour in male cockatoos, sexual overwork in waterfowl and ratites, oviductal disease (egg binding, salpingitis), increased intra-coelomic pressure (fluid accumulation or organomegaly), cloacal disease (cloacoliths, cloacitis, IPD), or constipation or diarrhoea.

It may also be related to loss of cloacal tone, as a result of spinal cord disease or chronic trauma.

Clinical presentation

There is protrusion of the cloacal mucosa, with or without rectum or oviduct, through the lips of the vent (**Fig. 16.12**). Phallic prolapse may be seen in waterfowl and ratites.



Figure 16.12 **King parrot hen with a prolapsed cloaca.**

Downloaded From Vet-eBooks.com

Cloacal prolapse is usually accompanied by straining and grunting while defecating, and blood around the vent or in the droppings. If the prolapse has been long-standing, the prolapsed tissue may be oedematous and superficially necrotic.

Diagnosis

Small prolapses need to be differentiated from papillomas (see below).

Management

Initial treatment is to stabilise the patient (if required), reduce the prolapse, hold it in place and treat any swelling and damage to the cloacal mucosa until the aetiology can be determined.

Under general anaesthesia, the prolapsed tissue is gently cleaned and, if possible, the prolapsed tissue is identified (cloaca, rectum or oviduct). Endoscopy may be of value, following the centre of the prolapse back into the body. If the prolapse is oedematous, the swelling can be reduced with the osmotic effect of sugar or glucose poured on to the mucosa. Once the cloaca is clean and normal sized, the prolapse should be gently reduced. Moistened cotton buds can be used to return the tissue to its normal anatomical position and it can be flushed with saline to inflate and clean it. Instilling 1–2 ml of silver sulphadiazine cream has both a soothing and an antibacterial effect. As the vent is elongated rather than rounded, a purse-string suture is not recommended. Instead, one to two vertical mattress or simple interrupted sutures should be placed on both sides of the vent in order to reduce the size of the vent opening. This opening must be sufficiently large to allow the bird to defecate and urinate (see **Fig. 16.13**).

The cause of the prolapse is identified through a thorough history and physical examination, haematology and biochemistry, cloacal culture (caution must be taken interpreting this, as the possibility of contamination is high), whole body radiographs or cloacal endoscopy with/without biopsy.

Follow-up treatment is aimed at minimizing the possibility of recurrent prolapses by eliminating or minimizing the impact of the inciting cause and performing surgery to maintain the cloaca in place (cloacopexy or ventoplasty) (see Chapter 29, Surgery).

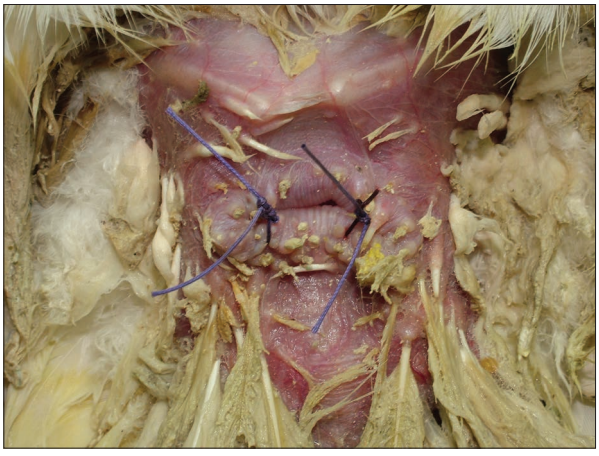


Figure 16.13 **Cockatoo with a prolapsed cloaca managed using a temporary ventoplasty.**

Long-term follow-up on cloacal prolapses indicates a reasonably high degree of recurrence, regardless of the techniques used. This must be communicated to the client before commencing any treatment.

Internal papilloma disease (IPD)

Aetiology

The causative virus is psittacid herpesvirus 1 (PsHV-1) genotypes 1, 2 and 3. This is the same virus that causes Pacheco's disease (see Chapter 17, Disorders of the Liver p. 251). It is thought that birds with internal papilloma disease (IPD) have survived an initial episode of Pacheco's disease. Recently a novel herpesvirus, PsHV-2 was identified in grey parrots with papillomas.

Pathophysiology

IPD has been reported mainly in macaws (green-winged, blue and gold, great green, scarlet, severe and military), Amazon parrots, conures and hawk head parrots. It has also been reported in cockatiels, budgerigars and African grey parrots. Lesions are rarely seen in eclectus parrots and cockatoos.

The mode of transmission is through ingestion of material contaminated by oral and faecal secretions from infected birds. There is no evidence of egg transmission, although parent-reared chicks with IPD-positive parents have developed the disease. Infected birds may develop a necrotising hepatitis

Downloaded From Vet-eBooks.com

(Pacheco's disease); untreated, these birds will die. Birds that survive, or those that remain sub-clinical, often go on to develop IPD with 12 months.

Clinical presentation

The clinical signs depend on the site of the location of the papillomas:

- Cloacal. Most birds with papillomas will have lesions in their cloaca. Often the first signs noticed are straining to defecate, pasting of the vent and blood on the droppings. The bird may develop enteritis and cloacoliths. There may be a red mass protruding from the cloaca (**Fig. 16.14**). Cloacal examination reveals papillomatous lesions ('cobblestone' appearance of the mucosa). These may blanch with the application of 5% acetic acid (vinegar), but this test is neither specific nor sensitive. The appearance of the lesions is characteristic; if the lesions appear inflamed a deep biopsy may be necessary to identify lesions underlying the inflammation.
- Oropharynx. Lesions of the choanal slit and glottis are more common in macaws (**Fig. 16.15**). Lesions may also be seen in the crop, proventriculus and ventriculus in some of the larger macaws. Clinical signs of papillomas are rare, but may include dysphagia, dyspnoea, wheezing, anorexia, and weight loss.



Figure 16.14 **Green Wing macaw presents with a red mass protruding from its cloaca suggestive of internal papilloma disease.**

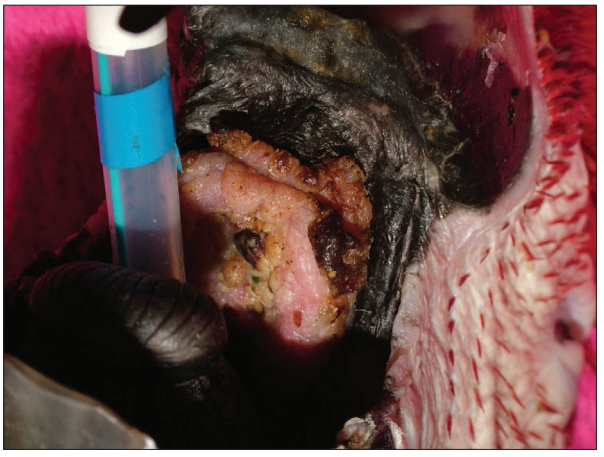


Figure 16.15 **Scarlet macaw with oropharyngeal lesions as a consequence of internal papilloma disease.**

- Extension into the intestinal tract, nasal mucosa and nasolacrimal duct is extremely rare.
- Some birds may ultimately develop bile duct or pancreatic carcinomas.

Diagnosis

Gross findings are usually sufficient for diagnosis. Blanching the lesions with acetic acid (vinegar) is often suggested as a means of gross diagnosis, but this recommendation is falling out of favour because of its low specificity and sensitivity.

Cloacal or choanal biopsy shows a characteristic histological picture. There is proliferation of undifferentiated columnar epithelial cells arranged in a pseudo-stratified fashion, forming fronds of villi and papillae on a thin fibrovascular stalk.

PCR is now available to confirm the diagnosis when screening asymptomatic or in-contact birds.

Elevations in GGT may indicate development of bile duct carcinomas. (This may not be reliable in Amazon parrots.) AST and ALT remain unchanged. Ultrasonography may detect bile duct carcinomas before endoscopy.

Treatment

Lesions may wax and wane over a period of time, and there is anecdotal evidence that they may be worse when the affected bird is reproductively active.

A variety of techniques have been tried for the removal of papillomas: cauterization with silver

Downloaded From Vet-eBooks.com

nitrate; cryosurgery; surgical debridement with radiosurgery or laser; cloacal mucosal stripping; imiquimod (Aldara) application (an interferon-immune response modifier). Mixed results have been obtained at this time.

Most birds initially respond to treatment, but later regress. Aggressive surgical debridement may result in strictures and should be reserved for severe cases only. Debridement of a small area may stimulate a (temporary) immune response and regression of the lesions.

Cloacoliths

Definition/overview

These are dried concretions of faecal material and urates in the urodeum or proctodeum. They are often adhered to the cloacal wall and, in extreme cases, can cause obstipation and intermittent ureter obstruction.

Clinical presentation

Signs include straining to defecate, decreased faecal output and fresh blood in droppings and around the vent. A flatulent sound is occasionally heard.

Diagnosis

Diagnosis is based on clinical signs and palpation of the cloaca. Radiography often reveals the cloacolith. Cloacal endoscopy may be used to confirm the presence of the cloacolith and the severity of the associated cloacitis.

Management

Mild cases may respond to lubrication and gentle pressure to break the cloacolith into small pieces. Severe cases may require a cloacotomy to relieve the obstruction (see Chapter 29, Surgery p. 396). Supportive care, especially fluid therapy and anti-inflammatory treatment, is essential.

Cloacal atony

Aetiology

Cloacal atony can be due to loss of neurological innervation to the cloaca (spinal cord dysfunction or PDD) or loss of muscular strength of the cloacal sphincter. The latter may be a result of soft tissue trauma and swelling associated with egg laying, systemic illness and weakness or chronic cloacal disease (e.g. recurrent prolapses or cloacitis).

Clinical presentation

Signs include a flaccid appearing vent, faecal accumulation around the vent and loss of cloacal tone (when a normal cloaca is stimulated lightly with a cotton bud, the sphincter should close in a manner similar to anal tone in a mammal). Faecal material, usually in large amounts, is easily expressed from the cloaca on palpation.

Management

Treatment includes NSAIDs and regular expression of the cloaca. Ventplasty may be necessary to reduce the size of the vent and prevent a prolapse.

Cloacitis

Aetiology

Inflammation of the cloaca can be due to cloacal atony (see above), cloacoliths, a retained egg in the distal oviduct, bacterial infection or trauma.

Clinical presentation

Signs include tenesmus, fresh blood in the droppings and faecal pasting around the vent.

Diagnosis

Cloacitis must be differentiated from IPD, cloacoliths and cloacal prolapse. Endoscopy, with or without biopsy, can help to determine the cause and nature of the cloacitis.

Management

Treatment includes NSAIDs and topical application of silver sulphadiazine cream into the cloaca. Topical flurbiprofen eye drops may assist when the peri-cloacal area is very inflamed.

Neoplasia

Definition/overview

Carcinomas, smooth muscle tumours and haemangiomas have been reported in the cloaca of birds.

Clinical presentation

Neoplasms produce similar signs to IPD and cloacitis.

Downloaded From Vet-eBooks.com

Diagnosis

Biopsy is necessary to achieve an accurate diagnosis.

Management

A cloacotomy can be performed to resect a mass if necessary. Many carcinomas will metastasise, and staging via radiology, ultrasound or CT may be indicated before undertaking any therapy.

FURTHER READING

Doneley RJT, Miller RI, Fanning TE (2007) Proventricular dilatation disease: an emerging exotic disease of parrots in Australia. *Australian Veterinary Journal* **85(3)**:119–123.

Gancz AY; Clubb S, Shivaprasad HL (2010) Advanced diagnostic approaches and current management of proventricular dilatation disease. In: *Veterinary Clinics of North America: Exotic Animal Practice* **13(3)**:471–494.

Gelis S (2006) Evaluating and treating the gastrointestinal system. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 411–440.

Hadley TL (2005) Disorders of the psittacine gastrointestinal tract. *Gastroenterology*. TK Ritzman (ed.) *Veterinary Clinics of North America: Exotic Animal Practice* **8(2)**:329–350.

Hoefer HL (1997) Diseases of the gastrointestinal tract. In: *Avian Medicine and Surgery*. RB Altman, *et al*. (eds). WB Saunders, Philadelphia, pp. 419–453.

Hoppes ST, Tizard I, Shivaprasad HL (2013) Avian bornavirus and proventricular dilatation disease: diagnostics, pathology, prevalence, and control. In: *Veterinary Clinics of North America: Exotic Animal Practice* **16(2)**:339–355.

Hoppes ST, Heatley JJ, Guo J, Turner MS, Shivaprasad HL, Tizard I (2013) Meloxicam treatment in cockatiels (*Nymphicus hollandicus*) infected with avian bornavirus. *Journal of Exotic Pet Medicine* **22(3)**:275–279.

Phalen D (2005) Diagnosis and management of Macrorhabdus ornithogaster (formerly Megabacteria). In: *Veterinary Clinics of North America: Exotic Animal Practice* **8(2)**:299–306.

Phalen D (2014) Update on the diagnosis and management of *Macrorhabdus ornithogaster* (formerly Megabacteria) in avian patients. In: *Veterinary Clinics of North America: Exotic Animal Practice* **17(2)**:203–210.

Reavill D (2007) Lesions of the proventriculus/ventriculus of pet birds: 1640 cases. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 89–94.

Downloaded From Vet-eBooks.com

CHAPTER 17

DISORDERS OF THE LIVER

OVERVIEW OF LIVER DISEASE

Because of its unique metabolism and relationship to the gastrointestinal tract, the liver is an important target for the damaging effects of infectious organisms, endotoxins, drugs, and metabolic disease. Initial damage from any of these aetiologic agents stimulates the secretion of pro-inflammatory cytokines and chemokines by the Kupffer cells and heterophils, leading to the accumulation and activation of heterophils in the hepatic vasculature. Ongoing damage and further release of cytokines and chemokines from the hepatic parenchyma results in extravasation of these heterophils and an attack on the hepatocytes, resulting in cellular death or damage.

Fortunately, the liver has enormous regenerative capacity and, so long as the acinar architecture of the liver is maintained, regeneration is frequently rapid. If, however, the acinar structure collapses, regeneration either does not occur or is very haphazard; fibrosis usually results. The same cytokines and chemokines that activate heterophils will also activate the stellate cells. Normally quiescent, these stellate cells (also known as Ito cells) are found in the peri-sinusoidal space (a small area between the sinusoids and hepatocytes), making up 5–8% of the total number of liver cells. When activated these stellate cells increase in number and contractility and secrete collagen, leading to the fibrosis of damaged areas of the liver. Early fibrosis may respond to treatment or removal of the source of injury, but more advanced fibrosis (seen in chronic liver disease) is generally irreversible.

It is obviously important that the clinician understands this disease process and the aetiological agents that provoke this damage, in order to diagnose, treat and manage liver disease in their patients.

AETIOLOGY OF LIVER DISEASE

Liver disease can result from congenital abnormalities, trauma, metabolic and nutritional disorders, toxins, parasites, infectious agents (bacterial, fungal, viral, chlamydial, mycobacterial and rickettsial), and neoplasia. Often the exact aetiology cannot be determined, lost in the fibrotic changes accompanying the disease.

Congenital

Extra-hepatic biliary cysts have been reported in an African grey parrot. A congenital coelomic hernia involving the liver has been reported in Japanese quail.

Trauma

Trauma can cause tearing of the liver parenchyma and/or the capsule. This occurs when the bird is subjected to massive force (e.g. a moving motor vehicle) or when the liver is friable due to other problems (e.g. hepatic lipidosis). The severity of the liver rupture and resultant haemorrhage will determine whether the bird survives the initial trauma.

Metabolic/nutritional

Visceral gout

This is the deposition of uric acid crystals on and in organs. It is typically associated with renal disease, although dehydration and high-protein diets may be involved. In the liver, most of this deposition occurs on the capsule, but it can occur within the parenchyma where it is associated with necrosis and heterophilic inflammation.

Amyloidosis

Amyloidosis is most commonly seen in raptors, waterfowl and passerine birds. Amyloid A is a degradation product of inflammatory proteins and its deposition

Downloaded From Vet-eBooks.com

is commonly seen in birds with chronic disease. Affected livers are usually enlarged and friable and can be confused with hepatic lipidosis. Histologically, the amyloid is seen as a pale eosinophilic or amphophilic deposit between the cells, compressing them.

Hepatic lipidosis

Hepatic lipidosis or fatty liver disease occurs when excessive fatty acids are consumed (in the form of dietary fat), when there is increased lipolysis (e.g. diabetes mellitus or egg laying activity), decreased fatty acid oxidation in the liver, or when there is a decreased ability of the liver to secrete processed fatty acids back into the circulation (due to dietary deficiencies of lipotropic factors such as choline, biotin and methionine). Affected livers are enlarged, pale yellow and friable (**Fig. 17.1**). Histologically, there is vacuolation of the hepatocytes.

Iron storage disease (haemosiderosis)

Haemosiderosis occurs when there is more iron in the circulation than is needed for erythrogenesis, allowing the iron to accumulate in the liver. It results from excess iron intake, either dietary or via excessive blood transfusion. This condition is commonly seen in Sturnidae (mynahs, starlings), Paradisaeidae (birds of paradise), Ptyonorhynchidae (bowerbirds), Bucerotidae (hornbills) and Ramphastidae (toucans and toucanettes). It has also been reported in parrots, especially lories and lorikeets. Affected livers are enlarged and are usually golden-brown in colour, often with scattered dark foci. Iron can be seen histologically in the hepatocytes and Kupffer cells. There may be an associated inflammatory process with lymphocytes and occasional heterophils.

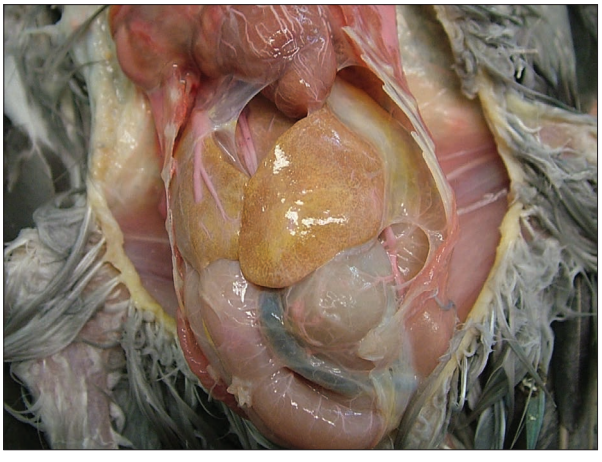


Figure 17.1 **Necropsy of an African grey parrot reveals hepatic lipidosis. Note the lipaemic blood in the vasculature.**

Lipofuscinosis

Lipofuscin pigment accumulates in hepatocytes secondary to a range of diseases. It is due to excessive biological oxidation at the cellular level. Vitamin E deficiency has been suggested as one possible cause.

Toxic

Toxic causes of liver disease include:

- Drugs: alcohol, dimetridazole, medroxyprogesterone.
- Plants: pyrrolizidine alkaloids, oleander, gossypol, avocado fruit.
- Aflatoxins, ergot.
- Heavy metals: lead, copper, iron.
- Pesticides: metaldehyde, phosphorus, vitamin D3 analogues.

Parasitic

Protozoa

Apicomplexa: Cryptosporidia may occasionally be found attached to biliary epithelium, causing proliferation of the epithelium and a mild chronic mononuclear reaction.

Atoxoplasma is primarily reported in passerines, especially canaries. It undergoes schizogony in the liver, causing a generalised inflammatory reaction composed of macrophages, plasma cells and lymphocytes. The organism may be found in macrophages and lymphocytes.

Birds infected with *Sarcocystis* and *Toxoplasma* are usually intermediate hosts. They produce a similar inflammatory reaction to *Atoxoplasma*. Organisms can be difficult to locate.

Haemoprotozoa: *Plasmodium* (avian malaria) is a widespread blood parasite. Some species, especially penguins, appear to be more susceptible. Schizogony occurs in the reticuloendothelial cells of many organs, with merozoites released to infect erythrocytes.

Downloaded From Vet-eBooks.com

Affected livers are enlarged, and in falcons often appear grey–black. There is infiltration of the liver with macrophages, plasma cells and lymphocytes, with the organism found in some of the inflammatory cells.

Haemoproteus: is usually non-pathogenic, with schizonts occasionally found in the endothelial cells of the liver.

Leukocytozoon: affects many species, with mortalities reported in ducks and geese. Where liver damage does occur it is usually acute and severe, with haemorrhage and necrosis, but minimal inflammation. Gametocytes can be found in peripheral blood smears.

Flagellates: *Histomonas* causes the disease known as blackhead in domestic poultry, especially peafowl and turkeys. It produces classical white–yellow granulomas throughout the liver parenchyma. The organism can be found in the lesions, although it may resemble macrophages and be difficult to detect.

Trichomonas is usually a gastrointestinal parasite, but heavy infections can spread to the liver. Necrotic lesions with the parasite found at the periphery are diagnostic.

Trematodes

Flukes of the family Dicrocoelidae inhabit the bile duct of many avian species, including parrots, Anseriformes, Ramphastidae and ratites. Wandering flukes may be found in dilated bile ducts, and schistosomes can be found in dilated sinusoids. There is usually minimal inflammation unless there are degenerating eggs present. *Fasciola* flukes in emus provoke an eosinophilic response, with macrophages and lymphocytes present. Granulomas may form with giant cells and fibrosis.

Nematodes

Intestinal nematode larvae can migrate through the liver causing extensive fibrosis, bile duct hyperplasia and inflammatory cell infiltrates.

Infectious

Bacteria

Both Gram-positive and Gram-negative bacteria can cause liver disease, usually secondary to septicaemia or enteritis. *Salmonella* spp., *E. coli*, *Pseudomonas* spp., *Yersinia* spp. and *Campylobacter* spp. are common isolates. Affected livers are usually swollen, with grey–white foci throughout the parenchyma. Multifocal hepatocyte necrosis with a heterophilic inflammatory response is usually seen. Bacteria are usually seen in macrophages and Kupffer cells. Additionally, endotoxins arising from bacterial enteritis can enter the portal circulation, damaging the periportal hepatocytes.

Viruses

Herpesvirus has been isolated from nearly all species of birds. Serotypes involved include Pacheco's disease virus (see below), pigeon herpesvirus, owl herpesvirus, falcon herpesvirus, crane herpesvirus, stork herpesvirus, quail herpesvirus and finch herpesvirus. Other strains of herpesvirus, while not specifically attacking the liver, may produce some degree of hepatic damage. Affected livers are enlarged, with variable yellow–grey mottling and haemorrhage. Histologically there is acute necrosis with variable inflammation, syncytial cell formation and intranuclear inclusion bodies.

Polyomavirus is primarily recovered from parrots and finches, but may affect a range of birds. Affected livers are enlarged and friable, with the degree of severity often varying according to the species. There is multifocal or mid-zonal necrosis and haemorrhage, with characteristic inclusion bodies in the Kupffer cells.

Adenovirus rarely causes clinical disease unless there are other immunosuppressive factors at work (e.g. PBFD, juvenile birds). Many species of birds are affected including parrots, poultry, pigeons and ostriches. Affected livers are discoloured, with scattered yellow–grey areas present (**Fig. 17.2**). There is multifocal necrosis and haemorrhage, non-suppurative cholangitis and large basophilic intranuclear inclusion bodies in the hepatocytes.

Paramyxovirus causes hepatomegaly in isolated cases, but its primary effects are neurological, gastrointestinal and respiratory. Histologically there is a lymphoplasmocytic infiltrate in the periportal area.

Circovirus is occasionally seen as an acute disease in young birds. In these birds, and in some adults with severe feather changes, there may be a mild

Downloaded From Vet-eBooks.com



Figure 17.2 **Necropsy of a juvenile malabar parrot indicates adenoviral hepatitis.**

necrosis and a lymphohistiocytic inflammatory reaction in the portal areas (**Fig. 17.3**). Inclusion bodies can be seen in the Kupffer cells.

Reovirus has been recovered from parrots, Galliformes, Anseriformes, raptors, pigeons and chickens. Affected livers are enlarged with scattered grey–white or yellow foci. Histologically there is hepatocellular necrosis with minimal inflammation. Hepadnavirus is the cause of duck viral hepatitis, resulting in hepatic necrosis and periportal inflammation. Togavirus (eastern equine encephalitis) causes enlarged livers with some necrosis in many species of birds.

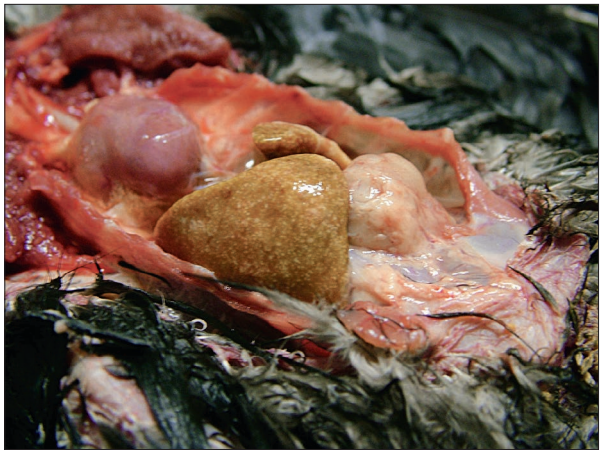


Figure 17.3 **Necropsy of a juvenile black cockatoo reveals circoviral hepatitis.**

Fungi

Fungal infections involving the liver are usually opportunistic spread from other sites within the body. *Aspergillus* spp. affecting nearby air sacs have been reported to invade the liver locally, causing hepatic necrosis.

Chlamydia

The obligate intracellular bacterium *Chlamydia psittaci* is a common cause of liver disease in all species of birds. Affected livers are enlarged, discoloured and may show grey–yellow foci of necrosis. There is multifocal to confluent necrosis with a mononuclear inflammatory reaction. Organisms may be found in macrophages and hepatocytes.

Mycobacteria

Mycobacterial infection in birds is primarily a gastrointestinal infection, with caseated tubercles occurring in the intestinal mucosa and the liver (**Fig. 17.4**). Histologically, early lesions are comprised primarily of heterophils and macrophages, with only a few microorganisms. As the lesions progress, large epithelioid macrophages containing mycobacteria appear. These are best seen with an acid-fast stain (see **Fig. 16.11b**, Chapter 16, Disorders of the Gastrointestinal Tract p. 244).

Rickettsia

Aegyptianella pullorum causes hepatitis in many species of birds, especially in the Mediterranean region.

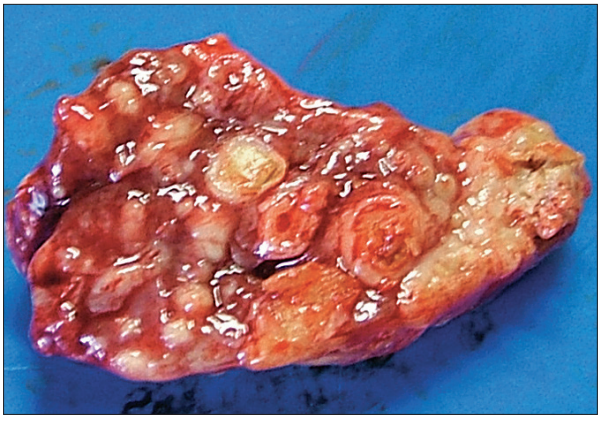


Figure 17.4 **Section through an excised avian liver showing mycobacterial granulomas.**

Downloaded From Vet-eBooks.com

Intra-erythrocytic inclusion bodies can be seen with Giemsa stain.

Neoplastic

Hepatic neoplasia is usually primary, although metastatic neoplasia can occur. Primary bile duct tumours are more common than hepatocellular tumours. In parrots these may be associated with IPD.

Idiopathic

Sometimes referred to as 'hepatopathy', 'chronic active hepatitis' or 'hepatic cirrhosis', this is a common condition in many species of birds. No one single cause has been identified, and it is probably multifactorial, possibly involving an immune-mediated component. Affected livers are often shrunken, pale and fibrotic. In the early stages of the disease there is hepatic vacuolization, a pleocellular inflammatory infiltrate in the periportal areas, bile duct proliferation and mild fibrosis. As the condition progresses, the fibrosis worsens and diffuse biliary hyperplasia develops (**Fig. 17.5a**).

CLINICAL SIGNS

Clinical signs seen in birds with liver disease can reflect any or all of the following malfunctions.

Cholestasis

Swelling of the hepatocytes and inflammatory infiltrates can cause occlusion of the biliary system. This results in the increased retention of bile and subsequent rise of serum bile pigment levels. Because of the lack of biliverdin reductase, it is uncommon for birds to produce bilirubin. Icterus is therefore uncommon in birds. However, increased levels of biliverdin in the blood due to cholestasis will result in biliverdin-stained urates and urine, giving these waste products a green discoloration (see **Fig. 3.14**, Chapter 3, The Physical Examination, p. 71) Decreased bile secretion into the intestine can result in maldigestion (weight loss, diarrhoea).

Inadequate conversion of ammonia to uric acid and urea

Failure to convert ammonia to uric acid and, to a lesser extent, urea leads to increased serum levels of ammonia and the onset of hepatic encephalopathy. Signs include weakness, depression, personality changes, behavioural disturbances, seizures and paresis. This is uncommon in granivorous birds such as parrots because of the lack of dietary encephalopathic precursors.

Protein synthesis deficits

Inadequate production of clotting factors can lead to coagulopathies. This can be as subtle as mild haemorrhages in the rhinotheca or as dramatic as sudden death due to internal haemorrhaging. Decreased synthesis of ceruloplasmin can result in decreased iron mobilization and subsequent anaemia. Decreased albumin can be one factor in the development of ascites.

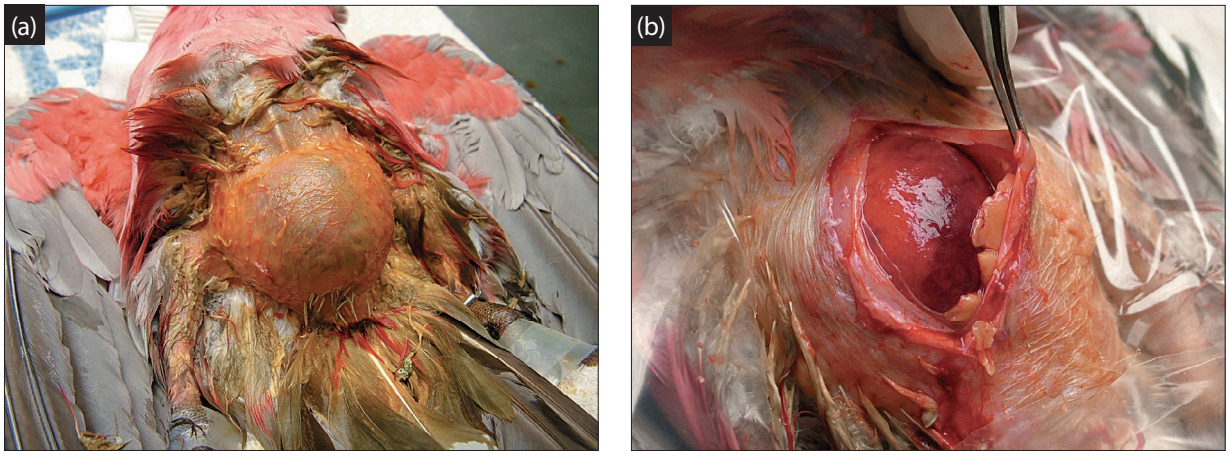


Figure 17.5 **Galah with significant abdominal distension (a) due to hepatomegaly (biliary hyperplasia) subsequently visualised during an exploratory laparotomy (b).**

Downloaded From Vet-eBooks.com

Abnormal carbohydrate and fat metabolism

Hypoglycaemia, weight loss and debility are frequently seen in birds with severe liver disease. Nutritional deficiencies resulting from liver disease may be the cause of the dermatological signs seen in affected birds. These include feather and skin changes, and overgrown beaks and nails (see **Fig. 11.11**, Chapter 11, Disorders of the Beak and Cere, p. 192).

Failure of Kupffer cell activity

Endotoxaemia and bacteraemia may develop when the Kupffer cells are unable to perform their function correctly.

Portal hypertension

Increased blood pressure within the sinusoids can result in the development of ascites, as oedema in the liver results in the movement of a modified transudate out of the liver into the peritoneal cavity.

Inadequate or inappropriate metabolism of drugs and chemicals

Decreased ability of the hepatocytes to modify or metabolise drugs and chemicals can lead to unexpected aberrant responses to administered drugs.

Other clinical signs

These result from other, less obvious, processes:

- Bile salts deposited in the skin may be the cause of some of the apparent pruritus seen in birds with liver disease.
- Pain from the stretching of the capsule over an enlarged liver can cause feather picking over the torso.
- Occlusion of the space normally occupied by the air sac by either the liver or ascites can result in dyspnoea.

DIAGNOSIS

Clinical pathology

Haematology will usually reflect the nature of the aetiological agent or the chronicity of the disease.

Biochemistry testing indicates either hepatocellular damage (AST, CK, LDH, GLDH), function (plasma protein, bile acids), or cholestasis (GGT). Cholesterol, triglycerides, VLDL, LDL and HDL can be markers of fat metabolism e.g. hepatic lipidosis. If indicated coeliocentesis and fluid analysis can be performed (ascites arising from liver disease is usually a modified transudate). See Chapter 7, Interpreting Diagnostic Tests, for further details.

Diagnostic imaging

Radiography

Two well-positioned views are required: lateral and ventrodorsal. Both the size of the liver and the displacement of nearby organs should be examined. See Chapter 5, Diagnostic Imaging, p. 102.

Lateral view: On the lateral view the liver should not extend past the end of the sternum, and the proventriculus should slope down towards the ventriculus at an angle of 30–45°. There should be little or no space between the heart and the liver.

Ventrodorsal view: On the ventrodorsal view the liver shadow should not extend past a line joining the shoulder and acetabulum. Care must be taken in interpreting this view that the cardiac shadow or the proventriculus is not confused with liver.

Ultrasonography

This offers several advantages over radiography, in that it allows examination of the internal structure of the liver as well as examination of the nearby heart and pericardium, spleen and gastrointestinal tract. The avian liver is homogeneous, finely granular and contains transverse and longitudinal blood vessels throughout the parenchyma. The gall bladder, when present, lies to the right of the midline. Changes in echogenicity and size of the liver should be noted.

Endoscopy/coeliotomy and biopsy

After using clinical pathology and diagnostic imaging to focus on the liver as the source of the patient's problem, the next step is to assess accurately the aetiology and pathophysiology of the disease process. To do this requires invasive technology; endoscopy or

Downloaded From Vet-eBooks.com

an exploratory coeliotomy to visually assess the liver and a biopsy to examine the histological basis of the problem. Several approaches have been described: through an incision behind the caudal ribs on either side; through the caudal thoracic air sac; and through a ventral midline incision (**Fig. 17.5b**).

TREATMENT

Treatment of an avian patient with confirmed liver disease should have three objectives: support of the patient and correction of the abnormalities caused by the liver disease; treatment of the specific condition; and creation of an environment within the liver most favourable to regeneration of normal liver tissue.

Supportive care

Problems include dehydration, anorexia, weight loss, hypoglycaemia, hepatic encephalopathy (in raptors and other carnivorous birds), anaemia, septicaemia, dyspnoea (enlarged liver and/or ascites), bleeding disorders and diarrhoea.

Supportive care therefore revolves around addressing these problems. See Chapter 8, Supportive Therapy, for more detail on supportive care including fluid therapy, assisted feeding, blood transfusions, oxygen therapy, and warmth.

Retrospective studies in both people and animals have shown that protein restriction is contraindicated in liver disease, and should only be used in cases where severe hepatic encephalopathy is present. Patients with liver disease are often in a catabolic state and protein restriction may exacerbate both the catabolic state and the liver disease, making the development of hepatic encephalopathy more likely. Protein levels in the diet should therefore be increased to allow for anabolic regeneration of the liver. (In carnivorous birds there may be the need to increase protein tolerance by feeding high-quality protein and the use of lactulose.) Diets high in simple and complex carbohydrates (such as vegetables, rice and pasta) offer many advantages to patients with liver disease. Vegetable protein appears to lack many encephalopathic components; the higher fibre alters the intestinal flora to minimise ammonia production; carbohydrate metabolism is useful for hypoglycaemic patients; and insoluble fibre (e.g. psyllium) binds many endotoxins and noxious bile acids. Fat levels in the diet can be left untouched unless cholestasis causes a maldigestion problem with subsequent diarrhoea, or in cases of hepatic lipidosis. Conversion to a formulated diet achieves many of these requirements and is the cornerstone of treatment for liver disease.

Vitamin supplementation may be given, but caution is needed with vitamins A and D to prevent toxicosis. B group vitamins are needed in many animal species to improve healing, promote normal metabolism and improve energy utilization by cells. They are stored in the liver as co-enzymes and may become depleted with liver damage and anorexia. Supplementation with a multivitamin B complex is often indicated.

Treatment of the specific condition

Amyloidosis

Therapy for amyloidosis is aimed at correcting the underlying problem. Colchicine has been used in dogs to minimise further deposition and may be of use in birds. Other treatments used in humans and dogs have included immunosuppressive therapy, chemotherapy and dimethyl sulfoxide (DMSO) (oral or subcutaneous). No reliable treatment has been shown to work in all cases, and success is limited.

Hepatic lipidosis

Hepatic lipidosis in birds is most commonly due to the feeding of a high-fat, low-protein diet, where the fat becomes the major source of calories. All-seed diets are a typical example of this sort of diet, but hepatic lipidosis occurs in most avian species. Treatment requires a reduction of dietary fat, an increase in dietary protein, the provision of carbohydrates to replace fat as the main source of calories, and supplementation with Vitamins E and B-complex.

Iron storage disease

Most cases of iron storage disease are believed to be associated with a high-iron diet. Treatment consists of feeding a low-iron diet, minimizing the absorption of iron from the intestinal tract and reducing the level of iron in the body. As the ascorbic acid found

Downloaded From Vet-eBooks.com

in citrus fruit reduces the ferric ion (Fe^{3+}) to the more easily absorbed ferrous ion (Fe^{2+}), citrus fruit should be eliminated from the diet. Commercial diets for susceptible species (Ramphastidae and mynahs) are now made with low iron levels (<100 ppm). Tannin added to the diet also binds iron in the intestine, reducing absorption. Iron levels in the body are reduced by weekly phlebotomy (1% bodyweight) and chelation therapy with deferoxamine.

Parasitic

Drugs used to treat hepatic protozoan infections are the same as those used for intestinal or systemic infections. Trematode infections are often difficult to treat. Praziquantel has been shown to reduce egg laying, but may not eliminate the parasite itself. Albendazole has been shown to eliminate *Fasciola hepatica* infections in emus.

Infections

Bacterial, fungal, viral, mycobacterial and chlamydial infections all have different treatments, according to the isolate and its sensitivity.

Neoplastic

Up until recently, hepatic neoplasia has been considered untreatable. Recent work suggests that some neoplasms may well respond to chemotherapy, and drugs such as carboplatin and cisplatin are been trialled for their efficacy.

Creation of an environment favourable to regeneration

Given the liver's ability to regenerate, treatment to stabilise hepatocyte cell membranes, maintain the acinar structure and minimise fibrosis will have a positive effect in patients with liver disease. Several drugs have been trialled in mammals to achieve these aims, and they show potential in avian medicine.

Ursodeoxycholic acid

Birds produce cholic, allocholic and chenodeoxycholic acids. These bile acids are hydrophobic and may act as biologic detergents causing solubilisation of cell membranes and hepatocellular lysis when cholestasis leads to elevated liver levels. Ursodeoxycholic acid (UDCA) is a hydrophilic bile acid that decreases the detergent effects of the hydrophobic bile acids, thereby reducing their hepatotoxic effects. It may also increase the flow of bile from the liver, reducing the concentration of hydrophobic bile acids and therefore their toxic effects. UDCA is used as an adjunct in the treatment of cholestatic and necro-inflammatory liver disorders. Its use as a single agent therapy in mammals is relatively ineffective.

Colchicine

Colchicine is used as an antifibrotic drug. It inhibits the synthesis and secretion of collagen by stellate cells and fibroblasts, and may induce the production of collagenase. It may also play a role in stabilising hepatocellular membranes and facilitate the excretion of hepatic copper. It may also block the synthesis and secretion of serum amyloid A (SAA) by hepatocytes, preventing the formation of amyloid-enhancing factor and subsequent amyloid disposition. Anecdotally it has been used as an antifibrotic agent in birds with some success.

Silibinin

Silibinin, the extract from the milk thistle *Silybum marianum*, is frequently recommended as a therapy for avian disease. It is widely used in medical and veterinary medicine for the treatment of liver disease, even though its mechanisms of action are not fully understood. It is thought to have antioxidant and free radical scavenger effects as well as cell membrane stabilising effects that prevent toxins entering hepatocytes. It is believed to stimulate hepatocyte regeneration while at the same time inhibiting stellate cells from depositing collagen fibres, reducing hepatic fibrosis. It may also inhibits the cytotoxic, inflammatory, and apoptotic effects of tumour necrosis factor.

However, in its original state, its water solubility and bioavailability are poor. Recent developments have improved both solubility and bioavailability but not all available commercial products utilise these advances. Care must be exercised in the selection of a suitable formulation. If these problems can be overcome, silymarin shows great promise as a hepatoprotective agent in the treatment of liver disease.

Downloaded From Vet-eBooks.com

S-adenosyl-methionine (SAMe)

Methionine is an essential amino acid requiring conversion to S-adenosyl-methionine (SAMe) to be active. SAMe is part of three major biochemical pathways in the body that serve to activate or excrete drugs; to maintain functional cell membranes; and for anti-inflammatory effects and cellular regeneration. The conversion of methionine to SAMe requires SAMe synthetase, an enzyme produced in the liver and decreased or deficient in the presence of liver disease. A lack of SAMe results in a deficiency of glutathione, important in many metabolic processes and cell detoxification. Exogenous SAMe (rather than methionine) requires supplementation in animals with liver disease, and has been shown to increase liver and red cell glutathione levels and/or prevent its depletion. It may also have a hepatoprotective effect. Its use in birds has not been trialled extensively, but may prove to be a valuable adjunct to the treatment of liver disease.

CHLAMYDIOSIS

Aetiology

As of 2009, the causative organism has been classified as a Gram-negative, non-motile, obligate intracellular bacteria of the order Chlamydiales, family Chlamydiaceae, genus *Chlamydia*. There are nine recognised species of which eight are of veterinary interest:

- *C. psittaci*: birds, humans.
- *C. abortus*: sheep, goats, cattle.
- *C. pneumoniae*: humans, reptiles, amphibians, horses, koalas.
- *C. pecorum*: ruminants, marsupials.
- *C. felis*: cats.
- *C. caviae*: guinea pigs.
- *C. suis*: pigs.
- *C. muridarum*: mice.

Chlamydia psittaci: There are five serovars (A–E); different serovars infect specific species (e.g. A infects parrots, E infects Columbiformes). Parrots are most frequently infected with serovar A (genotype A) strains, but can also be infected with serovar B strains. Serovar A strains are considered to be highly pathogenic. Chlamydiosis, also known as psittacosis and ornithosis has been a known zoonotic infection for about 100 years.

Clinical presentation

The virulence of the strain affects the clinical course, with clinical signs ranging from inapparent to severe septicaemia and acute death. Typically though, the disease affects either the respiratory tract, the gastrointestinal tract and liver, or both (**Figs 17.6a** and **b**).

Respiratory signs: These include conjunctivitis, often with loss or matting of periocular feathers, dyspnoea (tail bobbing, mouth-breathing) and sneezing with a purulent nasal discharge and sinus distension.

Gastrointestinal/hepatic signs: These include diarrhoea, biliverdinuria (green urates and urine), and a 'sick bird look' (fluffed, lethargic, anorexia and weight loss).

Other signs: There may be poor feathering and neurological signs (torticollis, tremors, and convulsions). Polyuria is frequent due to the nephrotoxic effects of elementary bodies. Affected birds may be infertile.

Pathophysiology

This can be a highly contagious disease, with transmission occurring through ingestion (faecal material, mutual feeding and feeding chicks) or inhalation (respiratory secretions, aerosolised feather or faecal dust). Egg transmission may occur. Shedding of the organism can start 72 hours after infection, and birds may be shedding for up to 10 days before they start to show clinical signs. The incubation period can be as short as four days, and up to 1–2 years.

The infection can be latent and activated during stress. There are indications that shedding in pigeons is greater in hot weather, possibly as a stress response to heat. A carrier state may exist. Immunity to infection is short lived and birds are susceptible to reinfection shortly after treatment ends.

Diagnosis

Diagnosis can be difficult, as there is no single 'best' test. Testing can rely on antigen detection or antibody

Downloaded From Vet-eBooks.com

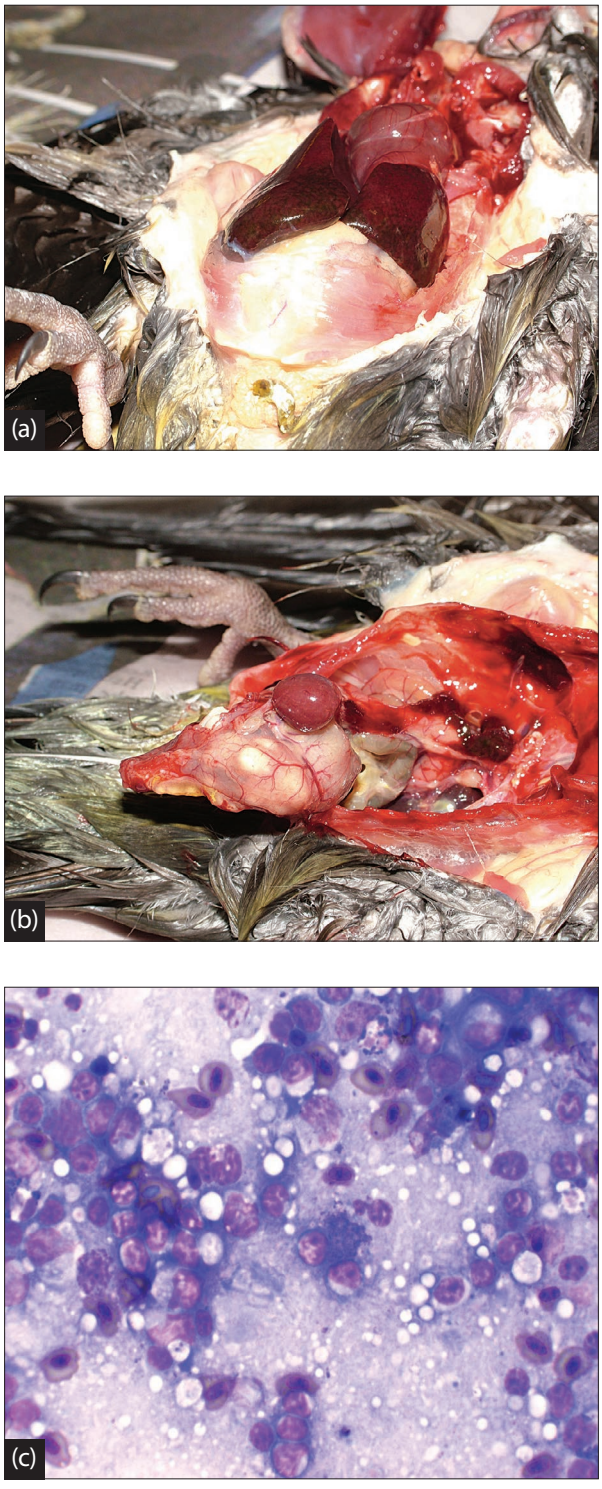


Figure 17.6 **Necropsy of a cockatiel reveals hepatomegaly (a) and splenomegaly (b) due to chlamydiosis. Subsequent cytology confirms presence of chlamydial inclusion bodies within hepatic tissue (a).**

detection, while ancillary testing can be used to support the presence of disease, rather than just the organism. The Centre for Disease Control (2010) classifies the level of diagnosis as confirmed, probable or suspect:

- A confirmed case of avian chlamydial infection is defined on the basis of one of the following:
  - Isolation of *C. psittaci* from a clinical specimen.
  - Identification of chlamydial antigen by use of immunofluorescence (fluorescent antibody) in the bird's tissues.
  - A fourfold or greater change in serologic titre in two specimens from the bird obtained at least two weeks apart and assayed simultaneously at the same laboratory.
  - Identification of *Chlamydia* within macrophages in smears or tissues (e.g. liver, conjunctival, spleen, respiratory secretions) stained with Gimenez or Macchiavello stain.
- A probable case of avian chlamydial infection is defined as compatible illness and one of the following:
  - A single high serologic titre in a specimen obtained after onset of clinical signs.
  - *Chlamydia* antigen (identified by use of enzyme-linked immunosorbent assay [ELISA], PCR or fluorescent antibody) in faeces, a cloacal swab specimen, or respiratory tract or ocular exudates.
- A suspected case of avian chlamydial infection is defined as one of the following:
  - A compatible illness that is not laboratory confirmed but is epidemiologically linked to a confirmed case in a human or bird.
  - A bird with no clinical signs and a single high serologic titre or detection of chlamydial antigen.
  - Compatible illness with positive results from a non-standardised test or a new investigational test.
  - Compatible illness that is responsive to appropriate therapy.

This classification highlights the complexity of confirming a diagnosis of chlamydial infection, and it also demonstrates the potential difficulty in stating that a bird is free of *Chlamydia*.

Downloaded From Vet-eBooks.com

Antigen detection: The respiratory epithelium (choanal/oropharynx) and the cloaca yield the most reliable samples for antigen detection, and a single combined swab is often submitted for testing:

- Isolation. Cell culture is the gold standard.
- Cytology. *Chlamydia* inclusions are most easily demonstrated in impression smears from serosal membranes, liver, spleen and affected air sacs. Smears are stained with Giemsa or Macchiavello stain to show intracytoplasmic inclusions (**Fig. 17.6c**). Negative staining does not rule out *Chlamydia*.
- Immunofluorescence antibody testing. Commercial FA conjugate for detection of *Chlamydia* spp. may cross-react with *M. avium* giving false-positive results.
- ELISA tests are available; however, be aware of false positives from *S. aureus* in the sample. ELISAs that detect the *Chlamydia* lipopolysaccharide (LPS) should be avoided because it is serologically related to the LPS of Enterobacteriaceae. False positives result from the presence of cross-reactive antibodies in the serum; false negatives occur when *Chlamydia* is shed intermittently.
- Latex agglutination tests (e.g. Clearview® *Chlamydia* MF, Unipath). A high number of false positives are seen due to bacteria.
- PCR tests. Be aware of false negatives: caused by presence of inhibitors of PCR reaction such as blood, serum, urine, faeces, sputum, hair shafts and lab reagents specifically haematin, DMSO, sodium chloride, phosphate-buffered saline, melanin, heparin, detergents and glove powder. DNA cannot distinguish between live organisms and dead organisms (which may be contaminants).

Antibody detection (serology): A positive serologic test result is evidence that the bird has been infected by *Chlamydia*, but it does not indicate whether it has an active infection or a persistent titre following exposure. Most serological tests detect IgG, which does not appear until at least two weeks after infection. False-negative results can therefore occur in birds that have acute infection and have not yet seroconverted. IgG titers may also persist following successful treatment and should not be used to monitor treatment. In the individual bird, a serology result should be interpreted in light of the bird's clinical signs, the history of the bird (or flock), ancillary testing (see below), and antigen detection. Available tests include:

- Elementary Body Agglutination detects IgM, but may give false positives to *S. aureus*, *P. multocida* and *Sarcina* spp.
- Direct complement fixation (CF) can be used to measure IgG, bit is best used with paired serum samples are tested.
- ELISA (e.g. Immunocomb® Avian *Chlamydia psittaci* antibody test kit, Biogal, Galed Labs) detects IgG.
- BELISA (blocking ELISA). This inhibitory ELISA is a very sensitive test; marketed in Germany.

Ancillary testing: Haematology may demonstrate moderate anaemia, leucocytosis, absolute or relative heterophilia, and monocytosis. Blood chemistry may show elevations in CPK, AST, LDH, total protein and bile acids if the liver is involved. Uric acid may be elevated if the kidneys are involved. Electrophoresis shows elevations in total globulins, beta and gamma globulins and a decrease in albumin.

Radiology may demonstrate hepato- and/or splenomegaly, air saculitis and pneumonia. Biopsy of liver, spleen or air sac may also be helpful.

Acute lesions found at autopsy include fibrinous peritoneal exudate, air saculitis, splenomegaly, hepatomegaly, perihepatitis, pericarditis, myocarditis, bronchopneumonia, catarrhal enteritis, nephrosis, orchitis, epididymitis and oophoritis. Chronic lesions include liver and kidney cirrhosis, pancreatic necrosis (budgerigar and pigeon) and non-purulent meningitis.

Differential diagnosis

Differential diagnoses include adenovirus, acute PBFD, herpesvirus, paramyxovirus, Influenza A, and bacterial infections, especially Enterobacteriaceae.

Treatment

Tetracyclines: These inhibit *Chlamydia* protein synthesis and are active only when intracellular reticulate bodies are actively replicating. Disadvantages

Downloaded From Vet-eBooks.com

include low intracellular concentration, immunosuppression, chelation with calcium (dietary calcium should be reduced to 0.7% or less) and inhibition of autochthonous flora. Duration of therapy is empirically set at 45 days, the average lifespan of a macrophage. Oxytetracycline is poorly absorbed from the digestive tract of birds and should not be used. This leave two members of the tetracycline group available for therapy:

- Chlortetracycline. This is excreted renally and should be used cautiously in birds with renal disease. Therapeutic tissue levels are difficult to maintain when administered through drinking water.
- Doxycycline. This is excreted extra-renally (faeces, bile). Adequate plasma levels are often achievable in medicated drinking water and seeds. It may cause hepatic necrosis with elevation of AST, which resolves with cessation of therapy.

Enrofloxacin: Clinical improvement and cessation of faecal shedding have been reported for five weeks post treatment. This is not fully documented, and enrofloxacin may not be effective clinically. Field trials indicate that it may not be as effective as doxycycline.

Azithromycin: This is a macrolide antibiotic, able to penetrate macrophages. There are anecdotal reports of effectiveness, but no clinical trials have been done as yet to confirm this.

Disinfection

Free elementary bodies are unstable and can be inactivated in the environment within days. They are sensitive to heat, quaternary ammonium products, 70% ethanol and 3% hydrogen peroxide.

Clinical signs in man

Chlamydia infection is a zoonosis, causing fever, chills, pneumonia, headache, weakness, fatigue, myalgia, chest pain, anorexia, nausea, vomiting and diaphoresis. It is important to discuss the zoonotic implications of this disease with clients when *Chlamydia* is diagnosed in a bird.

PACHECO'S DISEASE

Aetiology

Pacheco's disease is caused by psittacine herpesvirus 1 (PsHV-1). There are four genotypes of PsHV-1 recognised, with different genotypes affecting different species of birds in different ways.

- All four PsHV-1 genotypes have been shown to cause Pacheco's disease in Amazon parrots
- Only PsHV-1 genotypes 2, 3, and 4 have been identified in African grey parrots with Pacheco's disease
- Only genotype 4 causes mortality in macaws and conures.
- Parrots from Australia, Indonesia, the Philippines and Southeast Asia appear to be affected by any of the four PsHV-1 genotypes.
- PsHV-1 genotype 2 is the second leading cause of Pacheco's disease outbreaks in Europe, but has not been detected in parrots with Pacheco's disease in the USA.

These genotypes appear to have co-evolved with several species of Neotropical (Central and South American) parrots. In their host species, they do not appear to cause disease. However, disease may result when other parrots are infected with a PsHV-1 genotype to which they are not evolutionarily adapted.

Infected birds may:

- Become acutely sick and die (all genotypes) – this is known as Pacheco's disease
- Become acutely sick, respond to therapy and then become lifelong carriers
- Develop Internal Papilloma Disease (IPD; genotypes 1, 2, or 3) See Chapter 16, Disorders of the Gastrointestinal Tract. p. 247.
- Develop biliary adenocarcinomas (genotype 3)

Pathophysiology

Asymptomatic carriers (especially conures, Amazon parrots and macaws) may serve as virus reservoirs, shedding the virus for life in faeces and respiratory/pharyngeal secretions. Shedding may be triggered by periods of stress. Birds dying of Pacheco's disease will shed large amounts of virus into their environment.

Downloaded From Vet-eBooks.com

Most infections occur when contaminated material is ingested. The incubation period may last five days to several weeks. Severe hepatic and splenic necrosis usually ensues, with occasional pancreatic and intestinal necrosis occurring in some birds.

Birds that survive the initial disease may develop IPD within a year of the initial infection.

Clinical presentation

Sudden death is a common presentation (**Fig. 17.7**). Birds that are exposed to virulent strains of PsHV-1 and survive long enough to develop clinical signs will show evidence of lethargy, regurgitation, diarrhoea, biliverdinuria, and neurological signs, often dying within 3–10 days of exposure to the virus (**Fig. 17.8**).

Birds that are infected with PsHV-1 genotype 3 and survive the initial infection may develop bile duct and pancreatic duct carcinomas. Birds with bile duct carcinomas will often exhibit signs of chronic liver disease such as weight loss, an overgrown beak, and poor feather quality, eventually dying of chronic liver failure. Pancreatic duct carcinomas are much less common and clinical signs are vague and not specific. Bile duct carcinomas develop in the months and years following the onset of mucosal papillomas.

Diagnosis

Autopsy may show no lesions or hepatomegaly and splenomegaly, with a mottled tan discoloration of the liver (**Fig. 17.9**). Histopathology shows multifocal necrosis in the liver and spleen with intranuclear eosinophilic inclusion bodies.

Viral-specific PCR probes using oral and cloacal swabs have been developed to detect infected birds.

Treatment

Without treatment, birds that develop Pacheco's disease die. However, virus replication is rapidly inhibited by acyclovir, an acyclic purine nucleoside, and birds in the early stage of infection can be saved with treatment.



Figure 17.7 A group of *Neophema* spp. parrots who died acutely from Pacheco's disease. Such outbreaks can cause devastating loss in a collection (Photo courtesy B. Speer).



Figure 17.8 Green and gold macaw with visible haemorrhaging from the nares and oral cavity indicative of Pacheco's disease from which this bird died (Photo courtesy B Dalhausen).

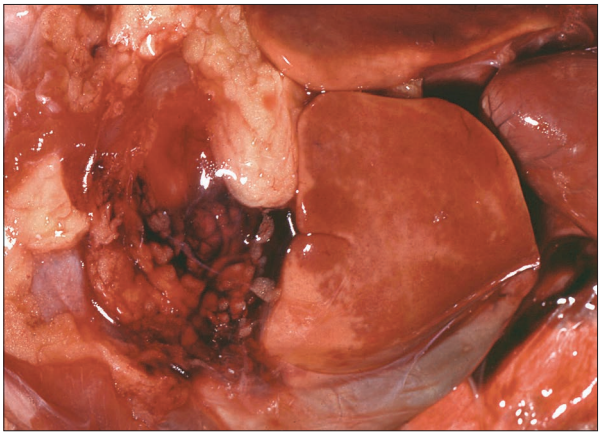


Figure 17.9 Examination reveals haemorrhagic hepatitis in a bird that died from Pacheco's disease (Photo courtesy R Schmidt).

Downloaded From Vet-eBooks.com

It works best in in-contact birds not showing signs of illness.All infected birds that survive the initial disease (or never develop illness) become carriers.

Management

Sub-clinically infected birds are readily detected by PCR, so all new birds should be screened for this disease in quarantine. Birds that have survived an outbreak should be regarded as carriers. All positive birds should be held in isolation. Although there is strong evidence of parent-to-offspring transmission after hatch, vertical transmission through the egg does not appear to occur. Therefore positive birds can be used as breeders, as long as incubation and hand rearing is used to isolate the chicks from their parents.

An inactivated vaccine was available in the United States but appears to have been discontinued.

FURTHER READING

Smith KA, Campbell CT, Murphy J, Stobierski MG, Tengelsen LA (2010) Compendium of measures to control *Chlamydia psittaci* infection among humans (psittacosis) and pet birds (avian chlamydiosis). Accessed online at www.nasphv.org. http://www.nasphv.org/Documents/Psittacosis.pdf (accessed November 2015).

Doneley B (2004) Treating liver disease in the avian patient. *Seminars in Avian and Exotic Pet Medicine* **13(1)**:8–15.

Hochleithner M, Hochleithner C, Harrison LD (2006) Evaluating and treating the liver. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 441–449.

Styles DK, Tomaszewski EK, Phalen DN (2004) Psittacid herpesviruses associated with mucosal papillomas of neotropical parrots. *Virology* **325(1)**:24–35.

Phalen D, E. Tomaszewski E, Styles D (2009) Epizootiology, diversity and pathogenicity of psittacid herpesviruses. In: *Proceedings of the Annual Conference August 10–13, 2009*. Association of Avian Veterinarians, Milwaukee, Wisconsin, pp. 47–51.

Tully TN (2006) Update on *Chlamydia psittaci*: a short comment. In: *Clinical Avian Medicine*, *Vol 2*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 679–680.

Downloaded From Vet-eBooks.com

DISORDERS OF THE PANCREAS

Diseases of the pancreas can affect the exocrine pancreas, the endocrine pancreas, or both. Pancreatic disease is infrequently described in birds but as our ability to diagnose disease improves, the incidence of diagnosed disease is rising.

EXOCRINE

Pancreatic insufficiency

Aetiology

Pancreatic insufficiency (an inability to digest food properly) may be caused by congenital pancreatic atrophy, prolonged calorific deficiency, or end-stage chronic pancreatic disease (see below) with loss of exocrine tissue.

Clinical presentation

The loss of exocrine pancreatic tissue results in maldigestion, particularly of fatty foods and starch. Undigested fats and starch account for steatorrhoea (seen as pale, bulky and often malodourous faeces) and weight loss. In mammals, depletion of body stores of cobalamin occurs rapidly, but this has not been looked at in birds at this time. Clinical signs seen in affected birds include lethargy, generalised malaise, weight loss and steatorrhoea, often described as 'popcorn' droppings (**Figs 18.1a** and **b**). Polyphagia is variable.

Diagnosis

Diagnosis is based in clinical signs and pancreatic biopsy. A ventral midline coeliotomy is performed and the duodenal loop exteriorised. The dorsal and ventral lobes of the pancreas can be seen within the duodenal lobe. Overall, the degree of pancreatic atrophy can vary; typically an affected pancreas appears small and irregular in shape. Lifting the tip

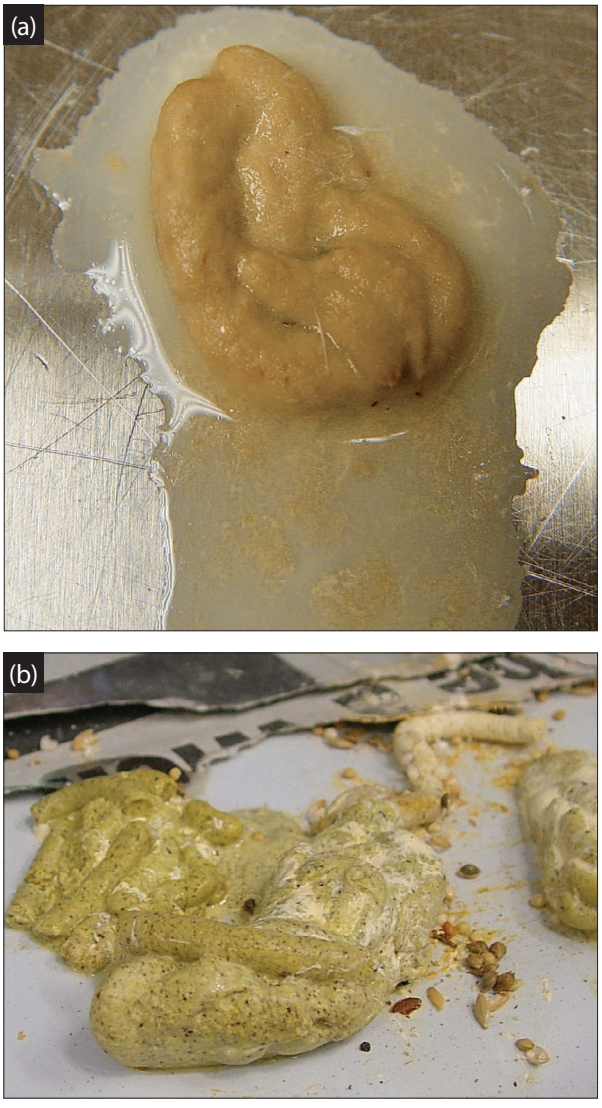


Figure 18.1 **Birds with exocrine pancreatic insufficiency have pale voluminous droppings referred to as 'popcorn' droppings as seen here from a macaw (a) (Image courtesy of B Speer) and from a cockatoo (b).**

Downloaded From Vet-eBooks.com

of the ventral lobe reveals the pancreatic blood vessels. These vessels are avoided while the tip of the pancreas is biopsied. Other areas of the pancreas can be biopsied if necessary, again avoiding the pancreatic vasculature. The splenic lobe of the pancreas is difficult to visualise and care must be taken not to place excessive tension on the duodenal loop trying to access the splenic lobe (**Fig. 18.2**).

An alternative technique is endoscopic biopsy through the right abdominal air sac.

Histologically there is a variable loss of zymogen granules and shrinkage of acinar cells.

Management

If possible, the causative agents should be identified and eliminated. The affected bird should be converted to a formulated diet and vegetables, which is supplemented with pancreatic enzymes in a powdered form, Palatability can be an issue in some birds, and intake must be monitored. Response to therapy is based on weight gain and a return to normal faeces.

Pancreatitis

Definition/overview

The incidence of primary pancreatitis in birds is a matter for some discussion. However, pancreatitis secondary to other disease processes is probably underdiagnosed in avian medicine. This undoubtedly reflects on the lack of specific and sensitive tools to diagnose this disease. Our understanding of avian pancreatic disease is still in its formative stages.



Figure 18.2 **Eclectus undergoing a pancreatic biopsy via a ventral midline coeliotomy.**

Aetiology

Causes of pancreatitis include obesity (often when combined with high-fat diets or fatty meals); toxicosis particularly zinc, mycotoxins and selenium; trauma; viral infection (including paramyxovirus-3, adenovirus, polyomavirus, avian influenza type A, infectious bronchitis and herpesvirus); chlamydiosis; bacterial infection; egg yolk peritonitis; and neoplasia. Of particular note is an acute pancreatic necrosis seen in quaker parrots, possibly caused by a high-fat diet but the exact cause has not been determined.

Pathophysiology

Activation of the digestive enzymes (trypsin, protease and phospholipase) within the gland results in pancreatic autodigestion. Damage to the pancreatic cell walls allows the release of these enzymes into the intracellular space and ducts, and this in turn causes the production of unopposed free radicals, which cause even more damage. This again releases more enzymes, and the cycle continues. The result is inflammation, cellular destruction, and finally fibrosis.

Clinical presentation

Gastrointestinal dysfunction causes vomiting, ileus, weight loss, polyuria, polydypsia and coelomic distension. The condition is painful and signs of this include anorexia, lethargy, feather picking, aggression and obsessive chewing on the cage and other items.

Diagnosis

Clinical signs suggest a diagnosis. Significant elevations may be found in serum amylase and lipase concentrations. However, these biochemistries are neither specific nor sensitive for pancreatitis, and a diagnosis of pancreatitis based solely on hyperamylasaemia or elevated lipase cannot be justified. Ultrasonography can be utilised, but the pancreas is often difficult to visualise. Pancreatic biopsy can be used, either endoscopically or through a ventral midline coeliotomy (see above).

Downloaded From Vet-eBooks.com

Management

If possible, the causative agent should be identified and eliminated (e.g. chelating zinc toxicosis, conversion from a seed diet to a lower fat formulated diet, surgical treatment of yolk peritonitis). Analgesia and fluid support are required and antibiotic therapy is given if indicated.

Pancreatic neoplasia

Definition/overview

Primary pancreatic neoplasia is usually either carcinomas or adenocarcinomas. Metastatic spread of other neoplasias to the pancreas is uncommon. Bile and pancreatic duct hyperplasia and papilloma formation is seen in some birds with internal papilloma disease associated with PsHV 1 genotype 3.

Clinical presentation

Signs are similar to those seen with pancreatitis or pancreatic insufficiency. Coelomic distension may be appreciable if the mass is large enough.

Diagnosis

Antemortem diagnosis is similar to that of other pancreatic diseases and requires ultrasonography and biopsy.

Management

Chemotherapy (e.g. carboplatin) has been used in a few cases with variable degrees of success.

ENDOCRINE

Diabetes mellitus

Definition/overview

Diabetes mellitus is most commonly diagnosed in budgerigars, cockatiels and galahs. It has also been diagnosed in larger parrots, toucans, mynahs and other species.

Aetiology

Diabetes mellitus in mammals, characterised by chronic hyperglycaemia and other metabolic abnormalities, is due to a lack of effect by insulin on glucose metabolism. This can be due to either an absolute lack of insulin or an inability of the insulin to lower blood glucose levels.

Understanding the pathogenesis of diabetes mellitus in birds is complicated by the role of glucagon and somatostatin in glucose metabolism in birds. In granivorous birds the pancreatic islets have a substantially higher percentage of glucagon-secreting cells compared with the mammalian pancreas. Correspondingly, the plasma glucagon:insulin ratio in these birds appears to be two to five times higher than that of mammals; it therefore appears that glucagon is the dominant hormone in glucose metabolism.

Glucagon is a powerful catabolic hormone that stimulates gluconeogenesis, lipolysis and glycogenolysis, thus increasing blood glucose. This process begins within hours of a meal, allowing continual production of glucose to meet the body's demands for energy. Insulin, on the other hand, is a powerful anabolic hormone, increasing the availability of glucose transport carriers, which allows easier transfer of glucose into the cell. It is antigluconeogenic. The effect of these hormones is modulated by somatostatin, pancreatic polypeptide, prolactin, thyroxine and growth hormone. It is therefore unclear whether diabetes mellitus in granivorous birds is due to an excess of glucagon or to a lack of insulin effect. The insulin:glucagon ratio and its regulation by somatostatin may be the critical factor in regulating glucose concentration rather than the actual concentration of the individual hormones.

Diabetes mellitus in mammals is classified as:

- Type I: characterised by destructive lesions of the pancreatic beta cells either by an autoimmune mechanism or by an unknown cause.
- Type II: characterised by combinations of decreased insulin secretion and decreased insulin sensitivity (insulin resistance), without any overt pancreatic disease.
- Other specific types of diabetes, characterised by non-specific destruction of pancreatic tissue associated with other pathological conditions or diseases, or diseases or drugs that cause insulin resistance.

No similar classification system has been applied to diabetes mellitus in birds and it may be that attempts to do so will be thwarted by the differences

Downloaded From Vet-eBooks.com

in glucose metabolism in birds compared with mammals. However, it would appear that the equivalents to these diabetes classifications do occur in birds:

- Type I: selective destruction of pancreatic islets; has been seen in toucans and parrots.
- Type II: commonly associated with obesity. A link between iron storage disease and diabetes mellitus is being increasingly recognised.
- Other specific types of diabetes are commonly associated with non-specific pancreatic disease such as neoplasia and pancreatitis (see above), and the iatrogenic administration of megestrol acetate, medroxyprogesterone acetate or corticosteroids, hormones that inhibit the effect of insulin and lead to insulin resistance.

Clinical presentation

The principal signs are polyuria, polydypsia, polyphagia and weight loss. In some cases, especially those with concurrent disease, non-specific signs such as depression, lethargy, vomiting or anorexia may be seen.

Diagnosis

Diagnosis is based on the finding of persistent hyperglycaemia and glucosuria. Normal avian blood glucose levels (12–25 mmol/l) are 150–300% above that of mammals, and this can go even higher when the bird is stressed (e.g. with capture and restraint). As the renal threshold for glucose in birds is 33 mmol/l, stressed birds, as well as diabetic birds, can demonstrate glucosuria; making it a non-specific indicator of diabetes. Blood glucose levels persistently above 38–44 mmol/l are consistent with a diagnosis of diabetes mellitus. Fructosamine levels have not been fully evaluated in birds, as yet; it therefore remains an unproven but potentially useful tool for monitoring diabetic patients. Ketonuria is present in some avian patients, but is not consistently reported.

It is worth repeating that a single-point finding of hyperglycaemia and/or glucosuria does not equate to a diagnosis of diabetes mellitus.

Management

The goal of treatment is to lower the patient's blood glucose levels and restore a more normal glucose metabolism. This is achieved by determining and correcting the primary disease process that is inducing the diabetes mellitus, stabilising the unwell patient and correcting the hyperglycaemia. The latter may be achieved through the use of insulin or oral hypoglycaemic agents.

If insulin is used, the patient should be hospitalised until an optimal dose of insulin is determined (via blood glucose curves) and the bird is stable. The immediate use of short-acting insulin (0.1–0.2 U/kg) can stabilise the patient initially, but long-term control at home usually requires longer-acting insulin (NPH or ultralente). Again, a glucose curve should be performed and dose rates adjusted accordingly. The dose rates vary considerably and should be based on the observed effects (reduction in PU/PD). They range from 0.067–3.3 U/kg q12–24h. If necessary, insulin can be diluted with sterile water and kept refrigerated for 30 days.

Oral hypoglycaemic agents (sulphonylureas, e.g. glipizide) may assist some patients, although reports of their efficacy vary. Sulphonylureas lower blood glucose concentrations in both diabetics and non-diabetics. The exact mechanism of action is not known, but these agents are thought to exert their effect primarily by stimulating the beta cells in the pancreas to secrete additional endogenous insulin. Extra-pancreatic effects include enhanced tissue sensitivity to circulating insulin and reduction of the production of hepatic basal glucose. The mechanisms causing these effects are yet to be fully explained. For these medications to be effective, pancreatic beta cells need to be functional.

Monitoring the effects of either treatment can be difficult, as a measured glucosuria could reflect faecal contamination. Observing water intake and urinary output may be more practical measures for owners to take at home.

Conversion to a formulated diet is an important part of treatment. Not only does it correct many of the causative factors (e.g. obesity), it also provides a high-fibre diet that assists with regulating blood glucose. In many (but not all) cases, dietary conversion and treatment of primary disease processes can result in clinical resolution of the signs of diabetes mellitus, removing the need for ongoing insulin therapy.

Downloaded From Vet-eBooks.com

FURTHER READING

Doneley B (2000) Acute pancreatitis in psittacine birds. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 261–267.

Gancz AY, *et al*. (2005) Diabetes mellitus in large psittacines: a possible relationship with excessive iron storage. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 267–269.

Gelis S (2006) Evaluating and treating the gastrointestinal system. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 411–440.

Hudelson KS, Hudelson PM (2006) Endocrine considerations. In: *Clinical Avian Medicine*, *Vol 2*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 541–557.

Pilny AA (2008) The avian pancreas in health and disease. *Veterinary Clinics of North America: Exotic Animal Practice* **11**(**1**): 25–34.

Schmidt RE, Reavill DR (2014) Lesions of the avian pancreas. *Veterinary Clinics of North America: Exotic Animal Practice* **17**(**1**):1–11.

Speer B (1998) A clinical look at the avian pancreas in health and disease. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 57–64.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

DISEASES OF THE RESPIRATORY SYSTEM

UPPER RESPIRATORY TRACT

Sinusitis

Aetiology

The complex anatomy of the infraorbital sinuses and diverticula lends itself to the establishment and persistence of upper respiratory tract infections. When combined with factors such as hypovitaminosis A (causing squamous metaplasia of the epithelial lining of the sinuses and decreased normal function and resistance to infection), irritation of the sinus linings (e.g. ammonia toxicosis in poorly ventilated, unhygienic cages and aviaries, cigarette smoke, or other aerosol pollutants), extremes of humidity (too dry, too moist) or choanal atresia (see below), it is little wonder that such infections are common in birds. Anatomic abnormalities such as choanal atresia, collapsed frontal bones, and traumatic injuries can also predispose to infection, as can inhaled foreign objects (e.g. millet seed, hand-rearing formula) and neoplasia (carcinoma, adenocarcinoma, fibrosarcoma, lymphosarcoma, and malignant melanoma).

Infectious agents are usually secondary invaders taking advantage of conditions described above. They include bacteria (*E. coli*, *Haemophilus* spp., *Klebsiella* spp., *Pasteurella* spp., *Pseudomonas* spp., *Mycobacterium* spp., and spirochaetes in cockatiels), viruses (herpesvirus, poxvirus, reovirus and avian influenza), fungi (*Aspergillus* spp., *Candida albicans*, *Cryptococcus* spp. and *Zygomycetes* spp.), *Chlamydia*, *Mycoplasma* and even parasites (*Trichomonas* spp. and *Cryptosporidia* spp.).

Clinical presentation

Clinical signs include oculonasal discharge, occlusion of the nares, exophthalmos (or enophthalmos in macaws — sunken eye syndrome) and distension of the sinuses (**Fig. 19.1**). This sinus distension can be quite marked as the lateral wall of the sinus is skin, rather than bone as in mammals. There may be matting or loss of the periocular feathering, and conjunctival hyperaemia or thickening (see **Fig. 9.8**, Chapter 9, Differential Diagnosis p. 159). Affected birds may sneeze, shake their head and scratch at their face with their feet, or rub their face excessively on objects such as perches. There may be hyperinflation of the cervicocephalic air sac.



Figure 19.1 **Cockatiel with sinusitis. Note the yellow caseated pus distending the sinus, dorsal to the eye, and the periocular feather loss.**

Downloaded From Vet-eBooks.com

Lockjaw syndrome may be seen in cockatiels aged 3–10 weeks. After an initial course of oculonasal discharge and sinus swelling, they are unable to open their beak and starve to death.

Diagnosis

Cytology and culture are helpful (see Chapter 4, Clinical Techniques pp. 83–84, and Chapter 7, Interpreting Diagnostic Tests p. 139). Diagnostic imaging, including contrast radiographs (using barium or other contrast agents), CT and MRI, can be used to examine the anatomy and patency of the infraorbital sinuses and to localise a problem (e.g. a pocket of caseated debris). Endoscopy, via the choana, can be useful when a foreign body is suspected. Transillumination with an intense focal light can be used to examine the sinuses and rhinal cavity.

Management

The underlying cause must be identified and corrected. Nutritional modification and the use of parenteral vitamin A are particularly important in many cases.

If the nidus of infection is not found and removed, recurrence is common. Therefore, it is important to remove caseous exudate and other debris within the sinuses. Nasal flushing using normal saline or diluted acetylcysteine may remove some caseated material, but the complex anatomy of the sinuses makes it difficult to flush the infraorbital sinuses successfully. Infraorbital flushing and aspiration, using the same approaches as used for sampling, can achieve more than nasal flushing in some cases. Surgical debridement and drainage via trephination into the affected sinus is an aggressive, but often successful, therapy. Before attempting this surgery it is important to review the anatomy and consult surgical texts. It is extremely easy to damage underlying structures such as the eye or nerves.

Oral and/or parenteral antimicrobial therapy are given, based on cytology and culture. Topical therapy may also be helpful, using nebulization or application of drops into the nares, if caseous pus has not accumulated in the sinuses.

Rhinitis

Aetiology

Causes of rhinitis are similar to those of sinusitis (see above). Occlusion of the nares can occur due to cere hypertrophy, *Cnemidocoptes* spp. infection or rhinoliths. See Chapter 11, Disorders of the Beak and Cere p. 195.

Clinical presentation

Clinical signs include sneezing, rubbing at the face, nasal discharge and asymmetry in the size of the nares (see **Fig. 11.20**, Chapter 11, Diseases of the Beak and Cere p. 196). In more chronic cases the external nare may be occluded with a dried plug; when this is removed a large hole may be left where the nare once was, the surrounding bone and keratin having been eroded by chronic infection (**Figs 19.2a** and **b**).

Diagnosis

Diagnosis of the causative organism may require cytology and culture.

Management

Rhinoliths can be removed with a small curette. Caseated debris is removed by nasal flushing. Topical or parenteral therapy is given based on culture and sensitivity. Parenteral Vitamin A and nutritional management is often indicated.

Ruptured cervicocephalic air sac

Aetiology

As the cervicocephalic air sac communicates with the infraorbital sinuses rather than the lungs, this condition may be the result of chronic sinusitis (see above), trauma or localised air saculitis.

Clinical presentation

The neck becomes distended with air (**Fig. 19.3**).

Diagnosis

Physical examination and aspiration of air and radiography of the neck will indicate the diagnosis. Endoscopy of the air sac may help to localise the site of rupture, and biopsy of the air sac may better determine the aetiological agent. This condition must be differentiated from aerophagia, where the

Downloaded From Vet-eBooks.com



Figure 19.2 **Princess parrot with a rhinolith before (a) and after removal (b).**



Figure 19.3 **Sulphur-crested cockatoo with a ruptured cervicocephalic air sac which has led to subcutaneous emphysema.**

crop is filled with air. This is done by introducing a feeding tube or crop needle into the crop to see if it can be deflated with a syringe.

Management

The underlying cause should be identified and treated. Lancing the distension with a scalpel or the bevelled edge of a needle, tearing through both skin and air sac, will deflate the air sac. This may have to be done several times until the originating rupture has healed. Refractory cases may require the placement of an acrylic stent in the most prominent area of the distension. Purpose-made stents are available in the USA and are implanted surgically under general anaesthesia. An alternative technique has been developed that creates a shunt between the cervicocephalic air sac and the clavicular air sac, avoiding the maintenance of an external stent.

Choanal atresia

Definition/overview

Failure of the choana to properly form during development results in a persistent membrane or bony plate at the palate of the nasal cavity, occluding one or both of the internal nares and blocking the normal drainage of nasal secretions into the oral cavity. The condition is predominantly reported in African grey parrots and occasionally in cockatoos and Amazon parrots. The author has seen it in several Alexandrine parrots and Indian ringneck parrots; there is no reason why it could not occur in other species. It is present from an early age.

Downloaded From Vet-eBooks.com

Clinical presentation

Clinical signs include unilateral or bilateral mucoid discharge and sinus swellings around the eyes (**Fig. 19.4**). A choanal slit may be present, although the internal nares are not patent.

Diagnosis

Saline flushed into the nares fails to enter the mouth. Instead, the infraorbital sinuses swell. Rhinography using contrast media can be used to demonstrate the lack of communication between the sinuses and the choana. CT can be used to demonstrate the presence of mucus in the sinus and nasal cavity, and the lack of patency of the internal nares. Rigid endoscopy can be used to visualise the choanal slit and assessment of the internal nares.

Management

Surgical correction is required. An appropriately sized Steinman pin is used to bore a hole through the nare on the occluded side(s) to the choanal slit. The pin is positioned so that it is perpendicular to the dorsal surface of the beak and is directed from the external nare towards the midline. This positioning should have the pin exiting through the location of the internal nare. There may be some bone to pass through and, while haemorrhage is usually minimal, in some cases it may be significant and potentially fatal. An 8 Fr red rubber catheter or nasogastric tube is passed through each hole, leaving the ends long. Openings are cut into the sides of the tube at the nares to allow mucus to drain. The ends of the tube are tied behind the head and a chin strap is made of tape to prevent the tubes from slipping off the top of the head. The tubes are left in place for 4–6 weeks to allow the tracts to epithelialise, creating permanent openings. Once the tubes are removed, the nares are flushed twice daily for two weeks to help keep the openings free of debris. It must be emphasised that if the tract does not epithelialise, the procedure will fail.



Figure 19.4 **Indian Ringneck presents with choanal atresia. Note the bilateral mucoid discharge and sinus swelling around the eyes.**

LOWER RESPIRATORY TRACT

Tracheal obstruction

Aetiology

The complete rings of the avian trachea make a functional obstruction (e.g. a collapsing trachea as seen in dogs) unlikely. The same anatomical feature makes external compression equally unlikely, although a fractured coracoid or thyroid enlargement have been known to compress the trachea. This leaves a physical obstruction of the trachea the most likely problem to be seen in practice. It is the author's experience that these physical obstructions can result from three aetiologies: an inhaled foreign body; a diphtheritic, granulomatous or neoplastic lesion within the trachea or syrinx; or a stricture following an insult to the tracheal mucosa.

Inhalation of millet seeds causing a complete, or near-complete, obstruction is frequently seen in cockatiels (*Nymphicus hollandicus*) (**Fig. 19.5a** and **b**). Other inhaled foreign bodies have included hand-rearing formula or even insects. If the foreign body is large enough to occlude the diameter of the trachea, acute collapse and death frequently results. If the object is smaller than the tracheal diameter, acute respiratory distress is more likely to be the presenting sign.

Diphtheritic and granulomatous lesions are often slower to develop, making clinical signs of dyspnoea usually evident before acute collapse occurs. Diphtheritic lesions, in the author's experience, are more common in cockatoos, mid-tracheal in location, and are associated with a bacterial infection often secondary to the lodgement of inhaled objects

Downloaded From Vet-eBooks.com

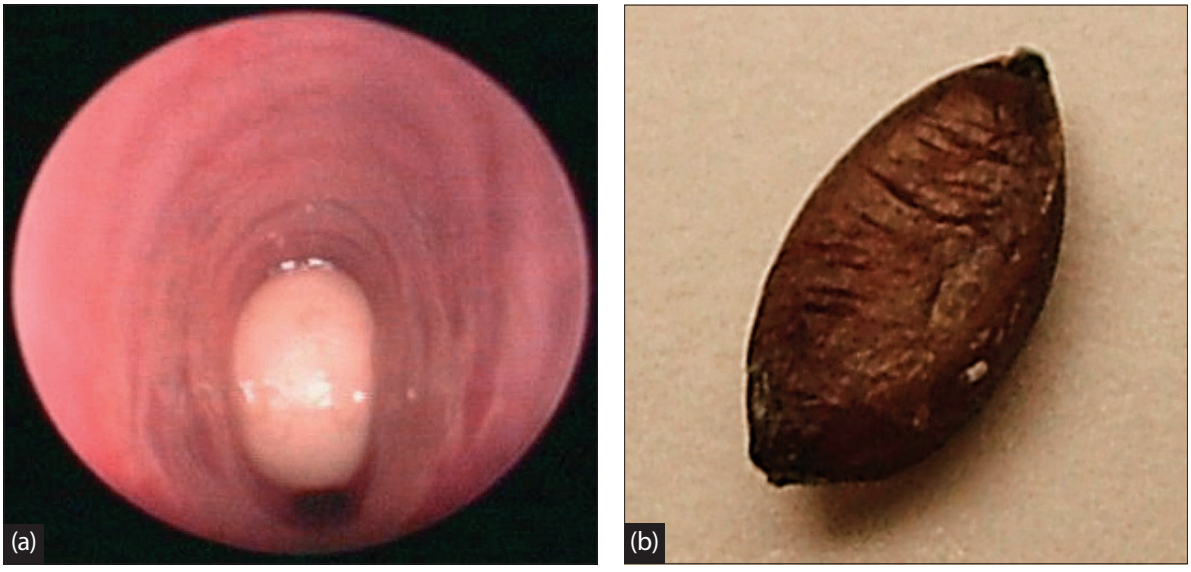


Figure 19.5 **Endoscopy of the trachea of a dyspnoeic cockatiel revealed this millet seed obstructing the trachea (a) (Photo courtesy A Lennox). After removal (b), it can be seen that the millet seed is perfectly shaped for accidental inhalation and subsequent tracheal obstruction.**

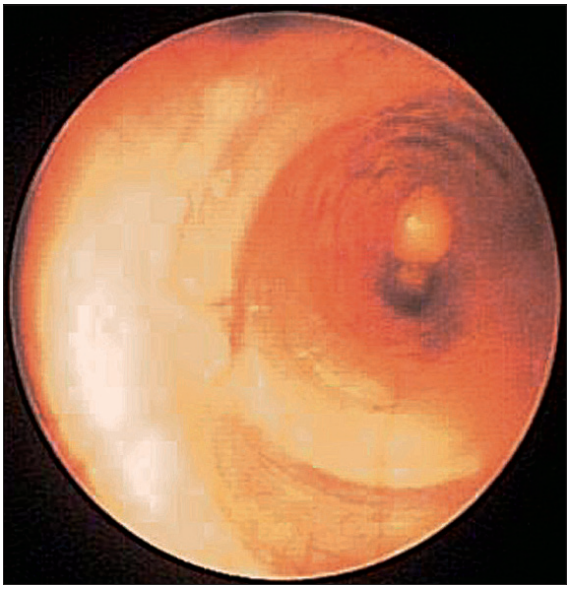


Figure 19.6 **Endoscopic examination of the syrinx of a dyspnoeic African grey parrot revealed a glistening white obstruction; an *Aspergillus* granuloma.**

such as plant material. Granulomatous lesions are more often seen in the syrinx and are often associated with aspergillosis (**Fig. 19.6**).

Strictures are usually associated with trauma, either due to external wounds (such as those resulting from bites from another bird) or internal trauma following endotracheal intubation or other mucosal insults (e.g. inhaled foreign material). The development of a stricture is usually more insidious, with obvious clinical signs often only seen when the bird's respiration is exerted by stress, exercise or handling. Careful observation of the bird at rest may reveal subtle mouth breathing and tail bobbing.

Clinical presentation

The clinical signs seen in a bird with a tracheal obstruction depend on the length of the time the obstruction has been present, and the degree of obstruction that is present. Birds with an acute obstruction may present weak and collapsed with the wings partially extended, the neck extended, obvious mouth-breathing and sternal lift, and audible inspiratory noises. Occasionally, the neck will be stretched out.

However, birds with slowly developing obstructions may show milder signs that become more obvious only when the bird is exerted by stress or handling. These birds compensate for the obstruction by keeping still and have a slightly increased respiratory effort. Vocalisation is decreased and there may be a change in voice. As the degree of obstruction increases, the clinical signs become more obvious.

Downloaded From Vet-eBooks.com

Diagnosis

While the clinical signs described above are suggestive of an obstruction, they are not always pathognomonic. Lower respiratory tract disease i.e. pulmonary or air sac disease, can appear very similar even to experienced clinicians.

Endoscopy usually offers the most rapid and accurate tool for diagnosing tracheal obstruction. The author's approach is to mask induce the patient with isoflurane, and then perform a left flank laparoscopy to evaluate the lungs and air sacs; an air sac catheter is then placed unless contraindicated (e.g. by the presence of fluid, severe airsaculitis or pulmonary disease) and anaesthesia is maintained via this route (see Chapter 4, Clinical Techniques pp. 90–91). With practice and preparation, this can be performed in 1–2 minutes. With an alternate airway secured, tracheoscopy can then be performed to evaluate the trachea and syrinx.

Radiography can be used to identify extramural compression of the trachea. Contrast radiography may be necessary in some birds to obtain a definitive diagnosis. A small amount of barium (0.5 ml/kg) is placed in the trachea using a catheter. Barium is inert and will not induce irritation or inflammation of the respiratory mucosa.

Management

The treatment of tracheal obstruction revolves around two key principles: the obstruction must be removed, and an alternate airway must be maintained until this is done.

Once an alternate airway (the air sac catheter) has been secured, the next step is to relieve the obstruction. The approach taken here will be determined by the location of the obstruction, the type of obstruction, and the size of the obstruction.

Small, non-adherent obstructions in the mid- to caudal trachea have been removed in a variety of ways. These have included removal with grasping forceps, suction, tracheotomy, or the introduction of an IV catheter caudal to the obstruction, directed rostrally. The tip of the catheter is used to dislodge the foreign body and it can then be 'blown out' using air from a syringe attached to the catheter.

Similar obstructions located in the syrinx may be approached through a distal tracheotomy and removed surgically. (Another technique, once recommended but now out of favour, was to use a small bone pin to push the obstruction through the syrinx into the supporting tissues, where it could be left to form a granuloma outside the airways.) The patient is positioned in dorsal recumbency with the shoulders elevated 45° to provide the surgeon with visual exposure to the thoracic inlet. A skin incision is made along the ventral midline, and the crop retracted if necessary (this may require some dissection of subcutaneous tissue). If the obstruction is located in the syrinx, the interclavicular air sac is entered to expose the sternotracheal muscles, which are transected with radiosurgery. This mobilises the syrinx and allows it to be brought rostrally. Great care must be taken not to tear the bronchi off the syrinx. The tracheotomy is made through the ventral half of the annular ligament between the tracheal rings, rather than through them. If approaching the syrinx, the incision is made three to five rings rostral to the syrinx. Closure of the trachea is achieved using a fine, synthetic, monofilament, absorbable material (e.g. 5-0 to 8-0 PDS) in a simple interrupted pattern. The sutures should be preplaced and positioned at 30° circumferentially so that a 50% tracheotomy is closed with two to three sutures. They should encompass at least two rings on each side of the tracheotomy and the knots are tied external to the tracheal lumen.

Diphtheritic or granulomatous lesions present a different problem. Although they can be endoscopically debulked, care must be taken to avoid iatrogenic trauma to the tracheal mucosa. In the short term this can lead to bleeding worsening the degree of obstruction, and in the long term may contribute to stricture formation. Nebulisation with hypertonic saline and/or acetylcysteine can help to 'dissolve' these lesions. Great care, however, must be taken to ensure the patency of the air sac catheter, as the lesions may swell prior to dissipation and completely obstruct the airway. If the lesion is an *Aspergillus* granuloma, injections of diluted amphotericin-B trans-tracheally may be of assistance as well, although it must be remembered that this drug can be irritant to mucosal surfaces (see the end of the chapter).

Tracheal strictures can be removed by resection and anastomosis. The flexibility of the avian neck

Downloaded From Vet-eBooks.com

confers a similar flexibility on the avian trachea, which is relatively longer than its mammalian counterpart. This inherent flexibility makes the resection of even long pieces of trachea feasible in birds; up to 10%. A skin incision is made over the stricture and careful sharp dissection performed to mobilise the trachea from the surrounding connective tissues. The stricture is then excised by transecting the trachea on either side of the affected area (**Fig. 19.7**). The incised ends of the trachea are apposed with four single interrupted 5-0 to 8-0 PDS placed equidistant around the tracheal circumference, each suture enclosing two tracheal rings on each side of the anastomosis and entering the tracheal lumen. The loose connective tissue around the trachea and the skin is closed routinely. Stricture at the site of an anastomosis is considered the major post-operative complication, generally developing 3–6 weeks after surgery. This however, is an uncommon occurrence in birds.

Tracheitis

Aetiology

Tracheitis is usually seen in conjunction with upper respiratory disease or other lower respiratory disease, although primary tracheal disease can be seen. Vitamin A deficiencies can play a role in the development of tracheal disease. Aetiological agents include bacteria, fungi (*Aspergillus*, *Mucor* and *Candida* spp.) viruses (poxvirus, herpesvirus) *Chlamydia*, parasites (*Syngamus trachea*, Gapeworm; *Sternostoma tracheacolum*, air sac mite; *Cryptosporidia*; *Trichomonas*), inhaled irritants (cigarette smoke, other pollutants), and trauma.



Figure 19.7 **Black cockatoo undergoing surgery to remove a tracheal stricture by resection and anastomosis. The stricture is excised by transecting the trachea on either side of the affected area.**

Clinical signs

Birds with tracheitis often present for coughing and gagging. If the syrinx is affected, there may be a loss or change of voice. In some cases there may be audible clicking noises on both inspiration and expiration. As the disease advances there may be increased respiratory effort and open-mouth breathing.

Diagnosis

Clinical signs are somewhat similar to those seen in a bird with tracheal obstruction (see above). Differentiation between the two is usually made endoscopically. Once an obstruction has been ruled out, diagnostic efforts move towards identifying the cause of the tracheitis. Diagnostic testing includes tracheal wash (combined with cytology and culture), transillumination of the trachea, radiography, and faecal parasite testing.

A tracheal wash is performed in birds to obtain samples of the trachea, bronchi, lungs, and potentially air sacs. The patient is anaesthetised or sedated, and a sterile tube, for example a nasogastric tube or an IV catheter, is inserted through the glottis (being careful not to contaminate it with the oral mucosa). Sterile normal saline (0.5–2.0 ml/kg body weight) is quickly infused into the trachea and immediately re-aspirated. Recovery should be 25–50% of the volume infused; this is submitted for cytology and culture.

Transillumination of the trachea is most often indicated in small birds with evidence of tracheal disease. Tracheal mites, strictures and exudates can be visualised in small birds such as finches and canaries. The skin over the trachea is moistened with alcohol and a focal intense light is passed through the skin and trachea. If present, mites will be seen as small, moving black dots.

Treatment

Specific therapy is directed on the basis of the diagnosis or laboratory testing.

Downloaded From Vet-eBooks.com

If the patient is severely dyspnoeic, consideration should be given to placing and maintaining an air sac catheter until the bird is stabilised.

Supportive care is important. While nebulising is frequently recommended, care must be taken not to exacerbate bacterial or fungal infections through increasing humidity in the trachea. Various medications have been used with oxygen and sterile saline as a delivery vehicle. If used, nebulisation should be done for 15–30 minutes every 6–8 hours for a minimum of three days beyond resolution of clinical signs. Mucolytic drugs should not be used if air sac involvement is suspected, as birds lack the ability to clear the exudates from the air sacs.

Pulmonary parenchyma disease

Aetiology

Infectious: Infections may be bacterial (*E. coli*, *Klebsiella pneumoniae*, *Pasteurella multocida* and *Pseudomonas aeruginosa* [**Fig. 19.8**]), fungal (*Aspergillus*, *Cryptococcus*), chlamydial, mycobacterial, mycoplasmal, viral (paramyxovirus, polyomavirus, herpesvirus, avian influenza, canary pox, adenovirus), or parasitic (*Sternostoma* spp., *Sarcocystis* spp., *Cryptosporidium* spp., *Toxoplasma* spp., *Atoxoplasma* spp.)

Pulmonary hypersensitivity: Also known as chronic obstructive pulmonary disease, this is most commonly seen in Blue and Gold macaws housed with birds producing feather dander (e.g. cockatoos, Grey parrots).

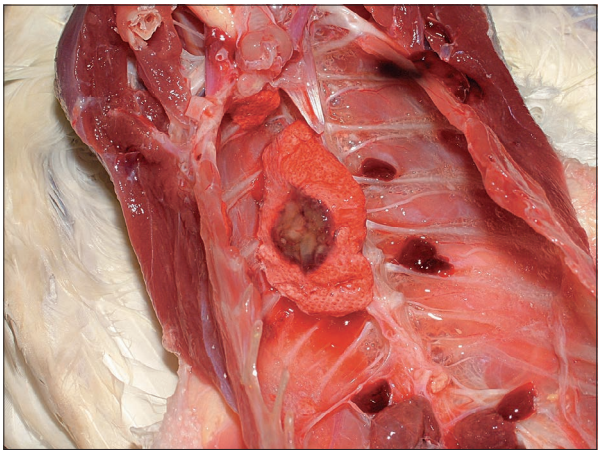


Figure 19.8 **Necropsy of a cockatoo reveals pulmonary parenchymatous abscesses. These can be bacterial, fungal or mycobacterial in origin. Note that abscesses are often found on the dorsal aspect of the lung, making surgical access difficult.**

Inhaled toxins: These include smoke, polytetrafluoroethylene (PTFE—Teflon®), cigarette smoke, aerosol sprays, carbon monoxide, burning cooking oils, foreign bodies, aspirated food material, yolk embolism and fat embolism from bone marrow.

Pneumoconiosis: This is accumulation of dust-laden macrophages. It may arise from an incident that occurred months or years previously.

Pulmonary congestion or oedema: Pulmonary oedema secondary to congestive heart failure occurs occasionally in pet birds.

Neoplasia: Pulmonary tumours include fibrosarcoma, carcinoma and metastatic disease.

Clinical presentation

Signs include tachypnoea and/or dyspnoea, mouth breathing, voice change, exercise intolerance and general signs of illness.

Diagnosis

Auscultation may detect audible respiratory noises. If these are heard on inspiration, upper respiratory tract disease should be suspected. Expiratory wheezes, rales and rasps are more suggestive of lower respiratory tract diseases. It must be noted, however, that even severe pathology may not cause audible detectable changes. Mild exercise in the form of wing flapping will often aid in the detection or localisation of abnormal respiratory sounds. Following mild exercise, the respiratory effort of a normal bird should return to a steady state within minutes. Prolonged respiratory recover time is an indication that further diagnostic testing is necessary.

Haematology and biochemistries: Polycythaemia (PCV 60–80%) may be present in birds with chronic hypoxia due to pulmonary parenchymatous disease (e.g. pulmonary hypersensitivity).

Radiography: Birds with pneumonia can have a variety of radiographic changes ranging from a diffuse to a

Downloaded From Vet-eBooks.com

uniform abnormal/thickened parabronchial pattern. Macaws with pulmonary hypersensitivity may demonstrate a more accentuated reticular pattern due to thickening of the interatrial septa.

Endoscopy: This is carried out via the cranial and caudal thoracic air sacs. See Chapter 6, Endoscopy p. 114.

Tracheal/lung wash: This is performed using 0.5–2.0 ml/kg of 0.9% saline (see above).

Lung aspirates and/or biopsy: These can be utilised for cultures, cytology or histopathologic evaluation. Lung parenchyma biopsies can be obtained by two endoscopic approaches: the cranial thoracic air sac (caudal lung) or the intercostal approach, through the third intercostal space (dorsolateral portion of the lung). The clinician needs to be aware of the potential for haemorrhage; it should be considered only when less invasive diagnostic techniques are ineffective or when a biopsy is necessary to determine and initiate life-preserving therapy.

Management

Treatment of parenchymatous disease can be frustrating if not diagnosed early, since most birds do not show clinical signs until the disease is advanced. Treatment for 4–6 weeks is generally necessary.

Supportive care: Oxygen therapy can be beneficial, but prolonged exposure to 100% oxygen can be dangerous. Oxygen concentrations should not exceed 35–50%, and be given for no more than two hours before going back to room air for a short period. Low flow intra-nasal oxygen via a nasal line can be used in suitable patients (e.g. waterfowl).

Nebulisation using 0.9% normal saline with or without antibiotics is administered for 15–30 minutes three to four times daily for a minimum of three days beyond the resolution of clinical signs (see **Fig. 4.13**, Chapter 4, Clinical Techniques, p. 92). Steam particles must be smaller than 3 µm to penetrate into the lung parenchyma.

Care should be taken in the use of mucolytic drugs in birds because they lack mechanisms to clear the exudates from their air sacs.

Medications: Systemic antimicrobial therapy is given as indicated by the diagnosis and/or culture. Topical antimicrobials, delivered by nebulisation (see above) can be used in conjunction with systemic therapy.

Birds with acute onset of respiratory distress following exposure to feather dust, smoke or PTFE may benefit from the administration of rapidly acting, short-duration corticosteroids (methylprednisolone sodium succinate, 15–30 mg/kg IM or IV) and oxygen supplementation. Bronchodilation with terbutaline (0.05–0.1 mg/kg IM q6–8h) may assist in some cases. Albuterol via an inhaler is also helpful.

Surgery: Surgery (partial pneumonectomy) may be required to remove granulomas.

Ongoing care and prevention: The use of air filters may be necessary in poorly ventilated areas. Macaws should not be housed with cockatoos or African grey parrots. Birds should not be housed near kitchens, especially if non-stick cookware is in use.

Air sac disease

Aetiology

Infectious causes include bacteria, fungi (*Aspergillus*, *Cryptococcus*), *Chlamydia*, mycobacteria and parasites (*Serratospiculum* spp., *Splendidofilaria* spp.).

Aspiration pneumonia – associated with overzealous eating, vomiting/regurgitation, oral disease, hand-feeding, or in sedated/anesthetised birds – frequently causes disease in the caudal lungs and air sacs because the first inspiration brings air directly to these areas.

Air sac carcinomas are uncommon, but may be seen occasionally.

Extramural pressure on air sacs may be the result of obesity, organomegaly (liver, oviduct, ovary [physiological enlargement, ovarian cysts]), neoplasia or fluid effusions (yolk-related peritonitis, ascites, and heart failure).

Clinical signs

Signs include increased respiratory effort, decreased exercise tolerance and weight loss. Auscultation over the bird's back may detect friction rubs.

Downloaded From Vet-eBooks.com

Diagnosis

Radiographically, severe air saculitis can often be documented by the appearance of 'air sac lines' – thin lines of increased radio-opacity where two air sacs meet. In chronic cases, increased respiratory effort can result in hyper-inflation of the air sacs, giving the appearance of extra intra-coelomic air.

Size and position of internal organs can also be assessed to detect extramural pressure on the air sacs. If the air sac space is reduced, ultrasound and/or contrast radiology can be used to better delineate internal structures.

Endoscopy can be used to both evaluate and biopsy the air sacs, as long as they are not occluded by internal organ enlargement or ascites.

A blind air sac wash can be done by inserting an intravenous catheter between the last two ribs. The needle is inserted and the catheter advanced into the air sac. This will place the catheter into the caudal thoracic (shallow) or abdominal (deep) air sac. Sterile saline (1–2 ml/kg) is instilled and collected for evaluation.

Management

The causative agent should be identified and treated. Treatment is similar to that for lung diseases (see above).

The air sacs are only a few cell layers thick and normally have a minimal blood supply. However, with most disease processes, neovascularisation of the air sac develops, and therapeutic concentrations can often be achieved using systemic antibacterial therapy. Humidification and topical treatment of the air sacs via nebulization are usually necessary in addition to systemic antibiotic treatment.

Aspergillosis

Aetiology

Aspergillosis is a non-contagious infectious disease caused by caused by ubiquitous soil saprophytes belonging to the genus *Aspergillus*, a filamentous fungus. The most common species involved is *Aspergillus fumigatus*, but *A. flavus*, *A. niger*, *A. glaucus*, *A. nidulans* are occasionally isolated as well.

These fungi are opportunistic pathogens, causing clinical infections when high concentrations of spores are inhaled or when the host is immunosuppressed. Risk factors for infection include species predilection (e.g. African grey parrots appear to be more represented than other species in the number of reported cases), exposure to spores (e.g. in nest boxes or poorly ventilated, dirty enclosures), immunosuppression (e.g. young birds, or those with concurrent diseases such as chlamydiosis), hypovitaminosis A affecting the resilience and resistance of the respiratory mucosa, and aspiration pneumonia (see above). Other predisposing factors include contact with organic litter and mouldy feed, stress (e.g. hospitalisation) and the use of corticosteroids.

Pathogenesis

The type of disease induced is dependent on the source and number of spores contacted and the general condition of the bird involved. Healthy birds can generally withstand exposure to a high concentration of spores. However, in conditions as described above, disease can result.

The pathogenesis of avian aspergillosis is still not known exactly. *A. fumigatus* is a thermotolerant fungus that grows within a temperature range of 20 to 50°C. It grows well at temperatures over 40°C – the temperature normally found within the avian air sac. The anatomy and function of the avian respiratory system allows direct inhalation of fungal spores into the caudal air sacs while precluding a mechanism for their removal.

In other species, phagocytic cells (macrophages) and respiratory clearance mechanisms are normally the first-line defence against inhaled spores. However, the local immune defences of the avian respiratory tract are not sufficient to resist fungal colonisation. As mentioned, there is no effective mechanism for the removal of inhaled material. The low numbers of resident macrophages, with only a limited capacity to kill fungal spores, combined with heterophils that lack the ability to effectively phagocytise fungal spores, allows fungal colonisation. After initial inhalation and dissemination of spores, fungal plaques and necrotic debris builds up in the airways and air sacs (**Fig. 19.9**), and may eventually obstruct the airways (leading to an acute presentation of a of a chronic problem). Haematogenous spread, or spread into adjacent organs and body

Downloaded From Vet-eBooks.com

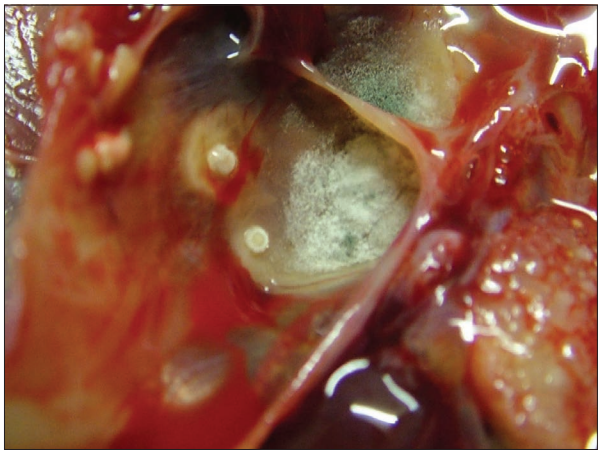


Figure 19.9 **Necropsy of an Amazon parrot reveals fungal plaques due to aspergillosis.**

systems, may result in disseminated lesions (generally focal granulomata) involving pneumatic bone, peritoneum, internal organs or the CNS.

Clinical signs

Clinical signs depend on the location and the severity of the disease. Lesions can become established in any area of the avian respiratory tract but are especially common in the caudal thoracic and abdominal air sacs, where granulomatous plaques may develop, or as a focal granulomatous lesion in the trachea especially at the syrinx. Less commonly, the infraorbital sinuses can be infected. As mentioned earlier, invasion of adjacent organs and body systems can cause disease outside the respiratory tract.

If the infection is localised in the sinuses, the clinical signs will be as described earlier for any sinus infection, i.e. oculonasal discharge, sneezing, sinus swelling, head shaking, etc. Tracheal infections are usually localised in the syrinx and often present acutely. Initial signs may include a voice change and decreased activity. As the trachea and bronchi become progressively blocked the bird will exhibit open mouth breathing, tachypnoea, increased respiratory effort and audible respiratory noises.

Lung and air sac disease usually presents as a chronic disease, but acute fulminating disease ('brooder pneumonia') can also occur when multiple lesions in the lung germinate at the same time. This is most common in young chicks exposed to high levels of *Aspergillus* spores during incubation, hatching or immediately after hatching, especially where there is poor hygiene in incubators, hatchers or nest boxes. Affected chicks present in acute respiratory distress and invariably die quickly.

More commonly the presentation is chronic, characterised by lethargy, weight loss, mild to moderate dyspnoea and prolonged recovery time after exercise. If disseminated spread has occurred it may lead to confusing clinical signs such as ataxia, kyphosis, seizuring, lameness, biliverdinuria etc.

Diagnosis

Antemortem diagnosis of aspergillosis is often difficult; either the disease presents so acutely that there is little time for diagnostic testing, or the clinical signs are so non-descript as to be of little assistance in the diagnosis.

As always, a thorough physical examination is indicated if the patient is stable. In chronic cases, auscultation of the lungs and air sacs may detect inspiratory rasps, rales and whistles. This is, however, not definitive for aspergillosis, and a confirmed diagnosis will rely on further testing such as haematology, radiography, endoscopy, cytology, fungal culture, and serology.

Haematology often, but not always, demonstrates a marked leucocytosis, usually characterised by a monocytosis and heterophilia. This is not reliable as some birds may have a poor immune response with total white blood cell counts in the normal range or just above.

Radiographic signs consistent with aspergillosis include focal densities in air sacs or lungs, loss of definition of the air sacs, asymmetry in their size and shape, or hyperinflation. Air sac lines may be visible. If the lungs are involved there may be an exaggerated parabronchial pattern and some consolidation of the lung pattern.

Endoscopy usually offers the best chance of confirming a diagnosis. See Chapter 6, for equipment and techniques. Tracheal Endoscopy, often done after air sac cannulation has stabilised the patient by providing an alternate airway, can detect syringeal granulomas as smooth, white, glistening masses in the syrinx. Endoscopy of the air sacs may demonstrate diffuse opacity and/or white and yellow fungal plaques that may have a superficial layer of blue, grey or aqua mould. These lesions can be sampled

Downloaded From Vet-eBooks.com

for cytology, PCR or culture, debrided from the air sac surface, excised or treated directly with an antifungal medication.

A positive PCR result or culture of *Aspergillus* spp. from a tracheal or lung wash can be strongly suggestive of the disease. However care should be taken in the interpretation of a positive result on its own. Because the organism is ubiquitous in the environment, can be isolated from the avian respiratory tract of non-infected birds, and is a common laboratory contaminant a positive result without supporting clinical signs and other diagnostic tests can be misleading.

Serological testing using an indirect ELISA test for antibodies for aspergillosis can be used to both detect disease and monitor response to treatment. Although the indirect ELISA can detect antibodies as early as 7–10 days post-infection, in sub-acute infections or in patients with a poor humoral response (sequestered granulomatous infections on air sac membranes or immunocompromised patients), testing for antigen may provide more information than testing for antibody response alone. On the other hand, in more chronic cases in which antigen levels may be low, ELISA for the detection of antibodies may be more helpful.

Another line of serological testing is the detection of *Aspergillus* galactomannan, a component of the fungal cell wall released during growth. This is often combined with a serum protein electrophoresis panel to detect inflammatory proteins. This test is far from receiving universal acceptance as false positive results appear to be not uncommon. These may represent exposure to *Aspergillus* spores without disease or cross-reactive antigens. Care must be taken not over-interpret these tests in a bird not displaying clinical signs consistent with the disease.

Treatment

The treatment of patients diagnosed with aspergillosis revolves around the twin concepts of supportive care and specific therapy directed against the fungus. Supportive care measures such as oxygen therapy, air sac catheterisation, nebulisation and antibiotic therapy (directed against secondary bacterial infections) have already been discussed in this chapter and in Chapter 4, Clinical Techniques pp. 90–92. Treatment should also be directed against the elimination of predisposing causes. In particular, the chronic form of aspergillosis is usually associated with immunosuppression, making immune support critical. Many birds become inappetant (compromising between eating and breathing) leading to further reduction in muscle mass and weight as a consequence of inadequate energy and protein intake. This often requires tube feeding to provide sufficient protein and energy, and supplemental vitamin A is often beneficial.

Syringeal granulomas: Syringeal granulomas may extend into both bronchi, making resolution difficult if not impossible. However, single granulomas not involving the bronchi may respond well to treatment as long an air sac catheter is in place and patent during the treatment (see Chapter 4, Clinical Techniques pp. 90–91). Nebulisation and trans-tracheal injections of amphotericin B (see below) can 'dissolve' the granuloma, clearing the airway. After a few days of therapy the patient will often become dyspnoeic as the granuloma swells and completely blocks the syrinx – hence the vital necessity for a patent air sac catheter. Success is achieved when the patient coughs the granuloma up, or it dissipates and passes down into the lung or air sac. Once the initial crisis is past, long term systemic and topical therapy is required to ensure the infection has been cleared (see below).

Lower respiratory tract infections: Lesions in the lungs and air sacs are more difficult to diagnose and are often well advanced when first detected. Occasionally granulomas can be surgically removed but there can be no guarantee that all the lesions have been resected, necessitating medical therapy. Antifungal therapy must be appropriate for *Aspergillus* spp., must be delivered topically and systemically, and must be given for several months if it is to be effective. Suitable antifungal drugs include:

- Amphotericin B: a polyene fungicidal drug, binds ergosterol and alters fungal cell membrane permeability. It must be given intravenously, by trans-tracheal injection, or by nebulisation. It is tissue irritant and poorly absorbed from the intestinal tract, making intramuscular or oral administration contraindicated.

Downloaded From Vet-eBooks.com

- Ketoconazole, itraconazole and voriconazole: azole antifungal drugs. They inhibit cytochrome P450-dependent 14α-sterol demethylase, an enzyme essential for fungal cell wall synthesis of ergosterol, an essential component of the fungal cell membrane. All are suitable for oral medication, although the effects and safety of ketoconazole is less predictable than the others. The adverse effects of itraconazole have been related to hepatic toxicity, varying between species, but are particularly common in African grey parrots, even at lower dose rates.
- Clotrimazole and enilconazole: azoles, but poorly absorbed. This makes them most useful when nebulised or delivered directly to a lesion endoscopically.
- Terbinafine: an allylamine, inhibits squalene epioxidase, an enzyme that is critical for fungal sterol and cell wall synthesis. It is administered orally either by itself or in combination with itraconazole.

Response to therapy for more chronic disease is rarely rapid, and the patient must be monitored carefully for evidence of clinical deterioration or drug toxicosis. Therapy for all forms of aspergillosis should be continued for 1–3 months after the resolution of clinical signs.

Prognosis

The prognosis for any patient with aspergillosis is guarded to poor. Drug resistance is rare, but not unheard of, but many patients are in an advanced disease state when first presented.

FURTHER READING

Antinoff, N (2001) Understanding and treating the infraorbital sinus and respiratory system. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 245–260.

Hillyer EV (1997) Clinical manifestations of respiratory disorders. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 394–411.

Lichtenberger M, Orosz SE (2007) Acute respiratory distress (ARD) – from anatomy through treatment. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 151–159.

Simone-Freilicher (2008) Avian sinusitis: the sniffles, the sneezes, and the silent. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 251–260.

Tell LA (2005) Aspergillosis in mammals and birds: impact on veterinary medicine. *Medical Mycology* **43** (Suppl **1**):S71–3.

Tully TN, Harrison GJ (1994) Pneumonology. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 556–606.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

DISORDERS OF THE CARDIOVASCULAR SYSTEM

CARDIAC DISEASE

Overview

It is thought that somewhere between 10 and 40 percent of companion birds have some form of cardiovascular disease, based on retrospective pathology studies. It is relatively rarely diagnosed antemortem, but as diagnostic techniques improve, an increase in the incidence of diagnosed cardiovascular disease can be expected.

Predisposing factors for cardiac disease include restricted exercise, poor nutrition and keeping birds in a climate in which they did not evolve (e.g. keeping South American parrot species in Europe).

Congenital heart disease

The diagnosis of intraventricular septal defects is becoming increasingly common, especially in umbrella cockatoos. Most are haemodynamically insignificant, but 2% will be associated with congestive heart failure. Blood is shunted from left to right, which leads to right ventricular failure and ascites secondary to valvular insufficiency. This is usually seen in birds aged 1–3 years. Duplicitas cordis, multiplicatis cordis and ectopia cordis have all also been reported.

Endocardial disease

Vegetative endocarditis of the aortic and mitral valves may cause vascular insufficiency, lethargy and dyspnoea. It is most common in birds with chronic infections (e.g. salpingitis, hepatitis and bumblefoot). Commonly implicated bacteria include *Streptococcus* spp., *Staphylococcus* spp., *E. coli*, *Pasteurella* spp., *Pseudomonas aeruginosa* and *Erysipelothrix rhusiopathiae*. Lesions consist of yellow irregular masses on any of the heart valves.

Myocardial disease

Myocarditis can occur secondary to viral, bacterial, mycotic and protozoan infections (**Fig. 20.1**). Myocarditis has been reported with haemosiderosis, polyomavirus infection, chlamydiosis, PDD, *Sarcocystis* infections, Pasteurellosis, and mycobacterial and *E coli* infections.

Cardiomyopathies arise from similar mechanisms to those that occur in mammals. Myocardial ventricular hypertrophy can occur with any condition that results in an elevated preload on the heart, including pulmonary hypertension and atherosclerosis. As an example, it has been noted in young broilers (4–6 weeks old) selected for high meat yield. The oxygen demand by the large muscle exceeds what the heart/lungs can provide. This results in polycythaemia, right ventricular dilatation/hypertrophy, valvular hypertrophy/insufficiency and right-sided failure. Vitamin E and selenium deficiencies cause cardiomyopathy in gallinaceous birds. It may also

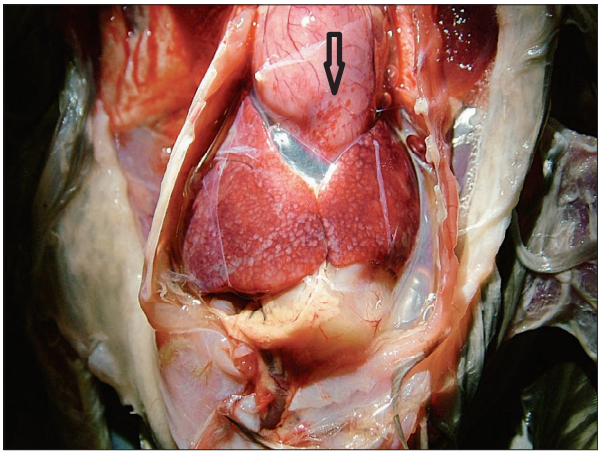


Figure 20.1 **Necroscopy of an African grey parrot reveals myocardial haemorrhages (as indicated) due to septicaemia following a bacterial infection.**

Downloaded From Vet-eBooks.com

cause myocardial and skeletal muscle degeneration in ratites less than six months old and myocardial degeneration in cockatiels.

Ruptures of the myocardium may occur secondary to degenerative, inflammatory or neoplastic conditions of the myocardium or aneurysms of the myocardial vessels.

Myocardial infarctions are occasionally seen when an embolism originating from damaged muscle or valvular endocarditis lodges in a coronary blood vessel.

Myocardial damage has been reported with avocado poisoning, and with visceral gout.

Pericardial disease

Pericardial effusion is a common finding in birds. It may result from cardiac or systemic disease and may be inflammatory (due to pericarditis or myocarditis) or non-inflammatory. Transudative effusions may be due to congestive heart failure or systemic conditions causing hypoalbuminaemia.

Fibrinous pericarditis may lead to adhesions of the epicardium to the pericardium and subsequently to constrictive pericarditis. Serofibrinous pericarditis is most common with bacterial (*E. coli*, *Streptococcus* spp., *Listeria*, *Salmonella*, *Chlamydophila*, *Mycoplasma*, *Mycobacterium*) and viral (reovirus in Galliformes and polyomavirus in parrots) infections (**Fig. 20.2**).

Visceral gout may lead to a non-infectious pericarditis (**Fig. 20.3**).

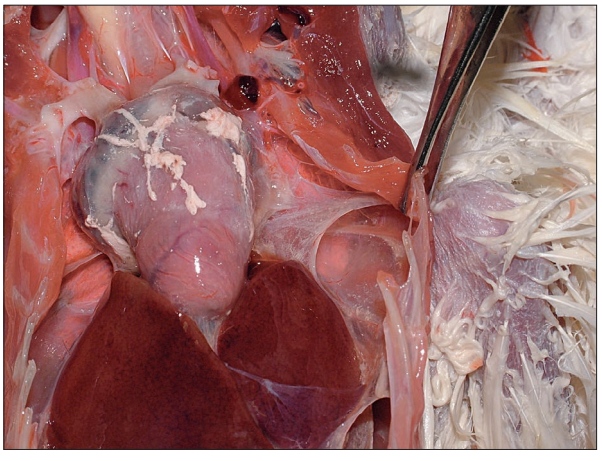


Figure 20.2 **Necroscopy of a cockatoo reveals pericarditis and air sacculitis.**



Figure 20.3 **Necroscopy of a scarlet macaw reveals visceral gout. Note that uric acid crystals have precipitated out over the pericardium giving it a white appearance.**

Arrhythmias

Arrhythmias can be normal (see below).

Sinus bradycardia can be induced by vagal stimulation and can be converted by the administration of atropine. Various anaesthetics and sedatives (e.g. halothane, methoxyflurane, xylazine and acepromazine) have been reported to cause sinus bradycardia when atropine is not given simultaneously. Hypothermia, which may accompany long-term anaesthesia, may potentiate this arrhythmia. Pathological conditions that may induce sinus bradycardia and sinus arrest include hypokalaemia, hyperkalaemia, thiamine deficiency and vitamin E deficiency. Several toxins have been reported to induce bradycardia, including organophosphorus compounds and polychlorinated biphenyls. Reflex vagal bradycardia may occur when pressure is exerted on the vagus nerve by neoplasms, and space-occupying lesions impinging on the vagal nerve should be considered when unexplained atropine-responsive bradycardia is seen. Atrioventricular (AV) nodal escape rhythm has been noted in ducks with sinus bradycardia from hyperkalaemia.

Atrial arrhythmias

- Sinus tachycardia is considered non-pathologic. The most common cause of sinus tachycardia is nervousness, but stress and pain may also precipitate the condition as well.

Downloaded From Vet-eBooks.com

- Atrial tachycardias may be seen as a result of pathological conditions of the atrium. Sinus tachycardia has been reported in chickens infected with avian influenza virus. When the heart rate is rapid, the P-wave may be superimposed on the T-wave (P on T phenomenon). This has been recorded in 16% of normal Amazon parrots and in 6% of African grey parrots.
- Atrial fibrillation occurs when electrical impulses are generated in the atrium in a rapid and irregular way, and the atrium is in a state of permanent diastole. Impulses reach the AV node in a high frequency and at irregular intervals, and hence the ventricular rhythm is irregular. The ECG is characterised by the absence of P-waves, normal QRS complexes (which may have an increased amplitude and duration because of ventricular hypertrophy) and irregular SS intervals.
- Supraventricular tachycardias may originate from the sinoatrial node (sinus tachycardia) or junctional area (junctional tachycardia). Differentiation between the two may be accomplished by measuring the PP interval. This interval is perfectly equidistant in atrial tachycardia, but may be irregular in sinus tachycardia due to vagal effects. Junctional tachycardias can be diagnosed by the presence of inverted P-waves in lead II.

Ventricular arrhythmias

- Ventricular premature contractions (VPCs) are characterised by QRS complexes that are unrelated to the P-waves. Bigeminy is a rhythm characterised by alternating normal beats and VPCs, while in trigeminy two normal beats are followed by one VPC. VPCs in birds have been associated with hypokalaemia, thiamine deficiency, vitamin E deficiency, Newcastle disease and avian influenza viruses, myocardial infarction due to lead poisoning, and digoxin toxicity.
- Capture beats (normal P-QRS complexes in between VPCs) and fusion beats (a QRS complex intermediate between a normal P-QRS complex and a bizarre QRS complex that is formed by the simultaneous discharge of the ectopic ventricular focus and the normal sinoatrial node) are common with ventricular tachycardia.
- AV dissociation is a special form of ventricular tachycardia. The atrial and ventricular rhythms are independent of each other, whereby the atrial rate is lower than the junctional or idioventricular rate.
- VPCs, ventricular tachycardia and ventricular fibrillation may occur during periods of hypoxia and with the use of halothane. Changes in the configuration of the T-wave and ST segment should alert the clinician that myocardial hypoxia is present and more severe ECG abnormalities are imminent.

AV node arrhythmias

- First-degree heart block occurs when the impulse through the AV node is delayed, resulting in an increased PR interval. This may be caused by halothane and xylazine, and the PR interval may increase to three to four times its normal value. This causes severe bradycardia that is reversible with atropine.
- Second-degree heart block occurs when some impulses do not reach the ventricles, but the majority of P-waves are followed by a QRS complex. Second-degree heart block Mobitz type 1 (Wenckebach phenomenon) has been reported as a physiological phenomenon in 5% of trained racing pigeons and is seen occasionally in asymptomatic parrots and raptors. In this form of AV block the PR interval lengthens progressively until a ventricular beat is dropped.
- Third-degree AV block is characterised by independent activity of atria and ventricles, where the frequency of the atrial depolarizations is higher than that of the ventricular depolarizations. The ventricular complexes may have a normal configuration or may be wide and bizarre, depending on the site of ventricular impulse formation. Complete AV block has been seen in chickens with hypokalaemia.

Congestive heart failure

This is the compensated condition associated with fluid retention that results from a sustained

Downloaded From Vet-eBooks.com

inadequacy of the cardiac output to meet the demands of the body. The causes are numerous and include endocardial, epicardial, myocardial and combined diseases.

The pathophysiology of congestive heart failure involves both backward failure and forward failure. Backward failure involves increased atrial and venous pressure due to a failing ventricle, while forward failure involves decreased renal blood flow resulting in sodium and fluid retention. In response to low blood volume, renin is released from the juxtaglomerular cells of the kidney. Renin acts on circulating angiotensinogen to form angiotensin I, which is converted to angiotensin II. Angiotensin II stimulates aldosterone synthesis. Aldosterone causes sodium and fluid retention. Both mechanisms ultimately result in increased venous and capillary pressure, so that more fluid escapes by transudation into the interstitial spaces.

Pulmonary oedema predominates in isolated left ventricular disease. Systemic oedema with hepatomegaly and ascites will predominate in isolated right ventricular disease or when both ventricles are affected (see **Fig. 5.20**, Chapter 5, Diagnostic Imaging, p. 104).

In birds, the right AV valve (muscular flap) thickens along with the right ventricle in response to an increased workload, and it has been postulated that this predisposes birds to right AV valvular insufficiency and right-sided heart failure.

Clinical presentation

Early stage signs include periodic weakness, syncope, lethargy and exercise intolerance. As the disease progresses, dyspnoea, coughing and coelomic distension due to ascites and hepatomegaly are seen. Neurological signs, both central and peripheral, can be seen with poor circulation and perfusion.

Diagnosis

Physical examination: Auscultation of the heart is best performed over the left and right ventral sternum. Pleural or pulmonary fluid accumulation may cause muffled lung sounds or rales when a bird is auscultated over the back between the shoulder blades. Unique to birds, hepatomegaly can cause muffled heart sounds as the enlarged liver partly envelops the heart. Subtle murmurs may best be heard when a bird is anaesthetised. Arrhythmias may be detected.

Peripheral pulse palpation can be performed over the femoral and brachial arteries, while capillary refill can be assessed by pressing the basilic vein on the medial aspect of the elbow. The presence of jugular vein distension or pulsation is suggestive of poor venous return. Coelomic palpation should be performed to detect ascites and/or hepatomegaly. Finally, poor peripheral perfusion is suggested by cyanosis or pallor of the feet, mucous membranes and facial skin.

Cardiac Biomarkers: Cardiac assessment in human and small animal medicine now frequently uses biomarkers for diagnostic and prognostic purposes.

- Troponins are regulatory proteins that are part of the contractile apparatus of skeletal and cardiac muscle tissue. They are not present in smooth muscle tissue. The troponin complex consists of three interacting and functionally distinct proteins (troponin I, T, and C). Troponins are considered leakage markers. Damage to cardiac myocytes resulting in loss of membrane integrity causes the release of Cardiac troponin I (cTnI) into the circulation. The severity of cardiac disease has been correlated with cTnI levels, while persistent elevations suggest irreversible myocardial damage. In dogs cTnI levels begin to rise after two hours and peak in 12–24 hours.
- Brain natriuretic peptide (BNP) is a neuroendocrine hormone with a primary role in fluid homeostasis by increasing the glomerular filtration rate and inhibition of the renin-angiotensin-aldosterone system. It is found in myocardial tissue and released in response to atrial muscle stretch or injury. NT-proBNP is the metabolically inactive precursor to BNP and appears to be more suitable for analysis due to a longer half-life in plasma. NT-proBNP relies on renal excretion and may be elevated in non-cardiac disease, such as renal failure.

The use of these cardiac biomarkers is an area of avian medicine waiting for further evaluation. Cardiac troponin I measurements appear to be

Downloaded From Vet-eBooks.com

potentially useful in birds, but until reference intervals in different species have been determined caution is warranted in their interpretation.

Radiography: Radiographic detection of cardiovascular abnormalities may be difficult, although an enlarged cardiac silhouette or microcardia can often be visualised (see **Fig. 5.11**, Chapter 5, Diagnostic Imaging p. 101). The maximum width of the cardiac silhouette in a ventrodorsal radiograph should be 35–45% of the length of the sternum, 51–61% of the width of the rib cage and 545–672% of the width of the coracoid.

Radiographic detection of an enlarged cardiac silhouette with muffled heart sounds is suggestive of pericardial effusion. An increased cardiac silhouette with normal heart sounds is suggestive of an enlarged heart. Microcardia is indicative of severe dehydration or blood loss that has resulted in hypovolaemia.

Other radiographic changes that suggest cardiac disease include congestion of pulmonary vessels, pulmonary oedema, pleural effusion, hepatomegaly and ascites.

Echocardiography: Echocardiography is the imaging technique that generally provides the most diagnostic information. Probes with small coupling surfaces and frequencies of at least 7.5 MHz are necessary, and the ultrasound devices should be able to produce at least 100 frames/second. Bipolar echocardiography is used to examine the heart in end-systolic and end-diastolic stage.

In small birds the echocardiographic image of the heart is best obtained by sweeping through the liver. B mode provides slices of the heart and allows anatomical evaluation of cardiac structures. M mode makes perpendicular slices of the B mode, allowing for quantitation of cardiac dimensions and timing of cardiac events. Colour Doppler facilitates identification of turbulence associated with valvular insufficiencies, stenosis and shunts.

Electrocardiography: This provides a graphic record of sequential electrical depolarization–repolarization patterns of the heart, detected on the surface of the body by changes in electrical fields (**Fig. 20.4**). Electrocardiography may be useful for detection of cardiac enlargement from hypertrophy of any of the four cardiac chambers. It is indispensable for the diagnosis and treatment of cardiac arrhythmias and is also useful in monitoring changes in electrolyte concentrations during the treatment of metabolic diseases that alter electrolyte balance. Because the myocardium is very sensitive to hypoxia, electrocardiography can be a reliable indicator of oxygenation.

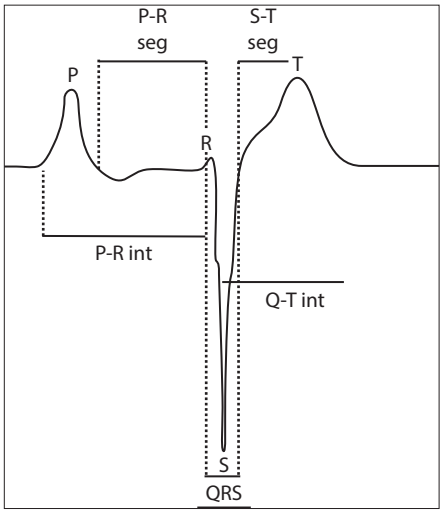


Figure 20.4 This lead II electrocardiogram provides a graphic record of sequential electrical depolarization–repolarization patterns of the heart, detected on the surface of the body by changes in electrical fields:

- **The P-wave signifies that the atria have depolarised, causing contraction and ejection of their complement of blood into the ventricles.**
- **The PR segment indicates the short delay in the atrioventricular node that occurs after the atria contract, which allows complete filling of the ventricles before ventricular contraction occurs.**
- **The QRS complex represents ventricular depolarization and contraction with the ejection of blood into the aorta and pulmonary artery. The Q-wave is the first negative deflection, the R-wave is the first positive deflection and the S-wave is the first negative deflection following the R-wave.**
- **The ST segment and T-wave depict the repolarization of the ventricles. In clinically asymptomatic parrots, the ST segment may be very short or even absent, the S rising directly into the T-wave ('ST-slurring').**

Downloaded From Vet-eBooks.com

Leads are attached to both wings and the left leg. In large or well-muscled birds one may need the V electrode to the right or left of the sternum for greater amplitudes of all complexes. Needle electrodes placed subcutaneously are superior to alligator clips for use in avian patients. The Biolog® hand-held ECG machine has been used with one foot (lead) on the chest, one on the right wing and one on the right thigh. QRS complexes were larger than with standard machines, but they were comparable.

Different lead systems are used to record differences between different limbs:

- Standard lead system: leads I, II, III.
- Unipolar or augmented lead system: avR, avL, avF.
- Unipolar chest lead: V1–10.

The sequence of depolarization and repolarization is recorded:

- Atrial depolarization: P-wave (the P-wave is positive on I, II, III and avF, and is negative on avR).
- Ventricular depolarization: QRS complex. This is negative in I, II, III and avF; there is no Q-wave in I, II and III (the negative mean electrical axis of ventricular depolarization in birds occurs because the depolarization wave begins sub-epicardially and then spreads through the myocardium towards the endocardium. Ventricular depolarization of the canine ventricles starts sub-endocardially).
- Ventricular repolarization: T-wave (positive on II, III and avF; negative on avR and avL; +/– or absent on I).
- T–P frequently overlap.

The paper needs to run at 50 mm/second or greater, as heart rates can vary from 100–1,000 beats per minute (bpm) (e.g. budgerigars: 600–750 bpm; cockatiels: 496–604 bpm). Some authors state paper speed needs to be at least 100 mm/second. For smaller species, paper speed may need to be set at 200 mm/second.

Determination of heart rate:
The top of the ECG paper has a series of marks. The marks are 75 mm apart. The number of complexes that occur in three seconds are counted and multiplied by 20 to get beats/minute.

Determination of heart rhythm:

- Is the heart rate normal or abnormal for the species?
- Is the heart rhythm regular or irregular?
- Is there a P-wave for every QRS complex, and is there a QRS complex for every P-wave?
- Are the P-waves related to the QRS complexes?
- Do all the P-waves and all the QRS complexes look alike?

Determination of mean electrical axis:

- Find an isoelectric lead.
- Use the six-axis reference system chart and find which lead is perpendicular to the isoelectric lead.
- Determine if the perpendicular lead is positive or negative on the tracing and examine the angle value on the six-axis reference system. Compare these values with reference values.
- The average mean electrical cardiac axis is between 83 and –162°.

Measurements are all made on the lead II rhythm strip:

- P-wave. This signifies that the atria have depolarised, causing contraction and ejection of their complement of blood into the ventricles. With right atrial hypertrophy the P-wave becomes tall and peaked (P pulmonale), and with left atrial hypertrophy the P-wave becomes too wide (P mitrale). P pulmonale has been associated with dyspnoea induced by aspergillosis or tracheal obstruction. A tall, wide P-wave is suggestive of biatrial enlargement and is common with influenza virus in gallinaceous birds.
- PR interval. This indicates the short delay in the AV node that occurs after the atria contract, which allows complete filling of the ventricles before ventricular contraction occurs. In the normal pigeon ECG, a small T_a-wave can be seen in the PR segment, indicating

Downloaded From Vet-eBooks.com

repolarization of the atria. This may also occur in some asymptomatic parrots and gallinaceous birds. This finding is considered normal.

- QRS complex. This represents ventricular depolarization and contraction with the ejection of blood into the aorta and pulmonary artery. The Q-wave is the first negative deflection, the R-wave is the first positive deflection and the S-wave is the first negative deflection following the R-wave. When there is no R-wave, the negative deflection is called a QS-wave. The largest wave in the QRS complex is depicted with a capital letter (i.e. Rs or rS). Two measurements are made on the QRS complex. The duration is measured from the beginning of the R-wave to the end of the S-wave. The second is the amplitude of the S-wave, measured from the baseline downwards. Low voltage ECGs often occur in birds with pericardial effusion. A QRS complex that is too wide or too tall indicates left ventricular hypertrophy or a bundle branch block. Prominent R-waves are suggestive of right ventricular hypertrophy.
- ST segment. Along with the T-wave this depicts the repolarization of the ventricles. In clinically asymptomatic racing pigeons and parrots, the ST segment is often very short or even absent. When present, it may be elevated above the baseline, which should not be interpreted as a sign of myocardial hypoxia, as it is in the dog.
- T-wave. In the normal avian ECG, this is always in the opposite direction to the main vector of the ventricular depolarization complex, and always positive in lead II. All measurements are made in lead II. When the T-wave changes its polarity, it suggests that myocardial hypoxia is occurring. The same is true for a T-wave that progressively increases in size (e.g. during anaesthesia). T-wave changes may also occur in association with electrolyte changes (e.g. increased T-wave amplitude with hyperkalaemia).
- QT interval. Prolongation might be associated with electrolyte disturbances (hypokalaemia, hypocalcaemia) and sedation, possibly due to a decreased heart rate. The QT interval may be prolonged in African grey and Amazon parrots.

Normal arrhythmias: Sinus arrhythmia, sinus arrest, sinoatrial block and wandering pacemaker have been reported in association with normal respiratory cycles and are considered physiological in birds.

The normal rhythm of the heart is established by the sinoatrial node. A normal sinus rhythm does not vary in rate from beat to beat. An increase in vagal activity may decrease the heart rate, while a decrease in vagal activity may increase the heart rate. Heart rate may increase during inspiration and decrease during expiration and hence the SS interval may not be equidistant. The associated rhythm is called sinus arrhythmia.

Sinus arrest is an exaggerated form of sinus arrhythmia and can be diagnosed if the pause is greater than twice the normal SS interval. Sinoatrial block occurs when an electrical impulse from the sinoatrial node fails to activate the atria. The pauses are exactly twice the SS interval. Wandering pacemaker refers to a continuous shifting of the pacemaker site in the sinoatrial node or the atrium, and an alteration in P wave morphology.

Blood pressure: The determination and monitoring of blood pressure has recently been recognised as a valuable diagnostic tool in the avian patient. Systolic blood pressure is the pressure exerted against the blood vessel wall during systole.

Direct arterial pressure measurement requires specific skills and is both invasive and expensive. Consequently, indirect blood pressure measurement techniques, based on the detection of blood flow beneath an inflated cuff, are more commonly used and have been found to correlate well with direct blood pressure measurements. The ultrasonic Doppler flow detector (Parks Medical Electronics Inc.) uses ultrasonic waves to detect arterial blood flow distal to a blood pressure cuff and produce an audible signal.

An appropriately sized cuff (depending on the size of the bird) is placed around the distal humerus or femur and taped in place. The Doppler probe is then placed above the radial carpal bone or tibiotarsal bone in order to detect arterial blood flow, and secured in place. The cuff is inflated until the Doppler signal is lost, and then slowly deflated. The pressure on the cuff when the first sound is heard

Downloaded From Vet-eBooks.com

is marked and recorded as the systolic pressure (see Chapter 28, Analgesia and Anaesthesia p. 380).

Management

Congestive heart failure: As mentioned earlier, congestive heart failure is a combination of inadequate cardiac output and an increased preload. In left heart failure, an increase of the pressure in the left atrium and the pulmonary veins leads to pulmonary oedema. In birds with right heart failure, ascites, liver congestion and hydropericardium are commonly seen. Treatment therefore revolves around improving contractility of the heart, reducing afterload, and removing excessive retained fluid in the lungs or liver/coelom.

Contractility is improved by the use of inotropes. The most commonly used drug is digoxin (0.02–0.05 mg/kg q24h). This drug is indicated for myocardial dysfunction, chronic mitral insufficiency and chronic volume overloads. However, it is contraindicated for hypertrophic cardiomyopathy, ventricular tachycardia and sinus or AV node disease. Adverse effects relate to myocardial toxicity, therefore patients should be monitored for clinical improvement and via ECG for prolonged PR time. Serum concentrations should be measured after one week of therapy, or one week after the dose is changed. The dose can be increased if the serum concentration is <0.8 ng/l 8–10 hours after dosing.

Another drug that is showing promise in the treatment of congestive heart failure in birds is pimobendan. This drugs acts as a positive inotrope (increased cardiac contractility), a positive lusitrope (improved ventricular relaxation), and a peripheral vasodilator. In small animal medicine it has a wider safety margin than digoxin, but has yet to be extensively trialled in birds. Recent research has suggested that the dose rates used in small animals (0.25 mg/kg q24h) may not be adequate, with suggested doses for bird of 6–10 mg/kg q12–24hrs been recommended.

Excessive neurohormonal activation is blocked by angiotensin converting enzyme (ACE) inhibitors (e.g. enalapril 0.5 mg/kg q12h). ACE inhibitors block the formation of angiotensin II, thereby blocking the renin–angiotensin–aldosterone system. They also have a diuretic effect. If the bird is on concurrent diuretic therapy, it may be best to start the ACE inhibitor at a low dose, while reducing the other diuretic therapy and then gradually increase the dose of the ACE inhibitor. Side-effects include hypotension, reflex tachycardia, gastrointestinal disorders, renal dysfunction and hyperkalaemia.

Signs of fluid retention are treated with diuretics such as furosemide 0.15–2 mg/kg q12–24h. Overuse or high doses of diuretics can reduce the blood volume and trigger the renin–angiotensin–aldosterone system. Therefore, it is necessary to use the lowest effective dose.

Cardiac arrhythmias: Treatment is not required unless the patient is clinically affected by the arrhythmia. The underlying cause should be treated.

- Supraventricular tachycardia:
  - Digoxin.
  - Beta blockers (e.g. propranolol 0.2 mg/kg q24h).
- Ventricular tachycardia:
  - Beta blockers.
- Bradycardia and AV blocks (note that limited therapy is available):
  - Atropine may be of some benefit
  - Ephedrine 0.5 mg/kg q8h.

Pericardial effusions: Diuretics are usually contraindicated because of the severe hypotension seen in many patients with reduced cardiac preload. If used, the bird should be monitored carefully for signs of hypotension (including weakness, ataxia and collapse). Endoscopic pericardial fenestration may give temporary relief, but the effusion may return when the fenestration heals.

ATHEROSCLEROSIS

Definition/overview

Atherosclerosis is a diffuse or local degenerative condition of the internal and medial tunics of the wall of arteries, causing chronic inflammatory fibroproliferative vascular disease. The degenerative changes include proliferation of smooth muscle cells and the deposition of collagen, proteoglycans and cholesterol (**Fig. 20.5a** and **b**). As tissue damage progresses, dystrophic mineralization occurs in the wall of the blood vessels. The resultant plaque protrudes into the vessel lumen and retards blood flow (**Fig. 20.5b**),

Downloaded From Vet-eBooks.com

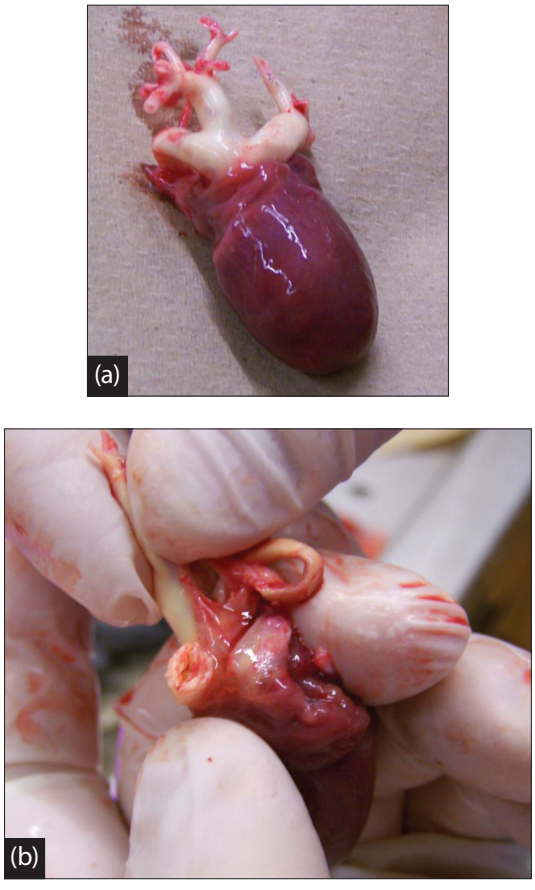


Figure 20.5 An excised atherosclerotic heart from an African Grey parrot shows a proliferation of smooth muscle cells and the deposition of collagen, proteoglycans and cholesterol of the aorta, and brachiocephalic trunks (a) and plaques protruding into the vessel lumen retarding blood flow (b) (Photos courtesy Chris Collis).

increases surface tension and may result in embolism formation (although rare in birds), aneurism and possible vessel rupture. These changes are most commonly seen in the aorta, the brachiocephalic trunks and the pectoral and carotid arteries, but any blood vessel can be affected. The resulting clinical signs are caused by ischaemia, arterial aneurysm, stenosis, and cardiac consequences (e.g. congestive heart failure and arrhythmias).

Risk factors are an individual's measurable characteristics that predict the probability of developing clinical disease. With atherosclerosis the risk factors appear to be species, age, sex, diet, activity and plasma cholesterol. Many avian orders may be affected, but Psittaciformes and Anseriformes appear to be particularly susceptible. Within the parrot family Amazon parrots, macaws, African grey parrots and cockatiels seem to be particularly prone, whereas cockatoos and macaws appear to be uncommonly affected. Other risk factors appear to include age (most affected birds are more than 8–15 years old; birds aged 20–30 years have a dramatically higher incidence), sex (females are more prone to severe lesions than males, possibly because of vitellogenesis and the mobilisation of lipids from the liver under the influence of oestrogen), high plasma cholesterol levels, lack of exercise and high-fat diets (i.e. seed). It is unclear if chlamydial infections create an additional risk factor.

Aetiology

The accumulation of pathogenic material in the arterial wall has been explained by the insudative theory. Normally a transfer of plasma proteins occurs through the arterial wall, with subsequent removal from the outer coats by lymphatic vessels. During this process of permeation, fibrinogen and very low-density lipoproteins are selectively entrapped in the connective tissue of the arterial wall. Their presence stimulates reactive changes that give rise to the production of atherosclerotic lesions. Variations in vascular permeability and arterial blood pressure can explain the preference of atherosclerotic lesions for certain areas.

What triggers these changes is still the subject of debate, but it may include genetic predisposition in some birds, hyperlipaemia, endothelial inflammation, toxins, immune complexes and hypertension. The role of cholesterol and lipoproteins remains unclear.

Clinical presentation

Clinical signs associated with atherosclerosis in avian species can vary depending on severity of lesions, location of lesions, and concurrent disease. Atherosclerosis is rarely diagnosed antemortem in birds, and the condition is often associated with sudden death. Subtle and intermittent signs include dyspnoea, weakness, signs of congestive heart failure (see earlier) and neurological signs (e.g. ataxia, seizures, tremors, and paresis).

Downloaded From Vet-eBooks.com

Diagnosis

Atherosclerosis is difficult to diagnose antemortem in birds. Clinical signs, knowledge of known risk factors, and the application of currently available diagnostic testing may be of some value.

There may be elevated plasma cholesterol, triglycerides and low density lipoprotein levels but this finding is not consistent and may be confounded by the effects of female reproductive physiology. High total blood cholesterol and triglyceride levels are considered major risk factors for atherosclerosis. Lipoproteins – low-density lipoprotein (LDL) and high-density lipoprotein (HDL) – may not be as useful in birds as they are in mammals. Most birds transport cholesterol mainly in the form of HDL (unlike humans, where LDL is predominant). Therefore, higher HDL values may not be associated with decreased risks of developing atherosclerosis as it is in humans – in fact, the converse may be true.

Cardiac biomarkers, discussed earlier, may assist in detecting cardiac disease associated with atherosclerosis, but this may be already advanced by the time this methodology detects a problem.

Radiography offers a useful but somewhat insensitive screen for atherosclerosis. Atherosclerosis targets the major arteries (brachiocephalic arteries, pulmonary arteries, and ascending and abdominal aorta) and these arteries are visible on plain radiographs. Although insensitive, radiographic detection of arterial calcification is fairly specific to advanced atherosclerotic lesions (**Fig. 20.6**).

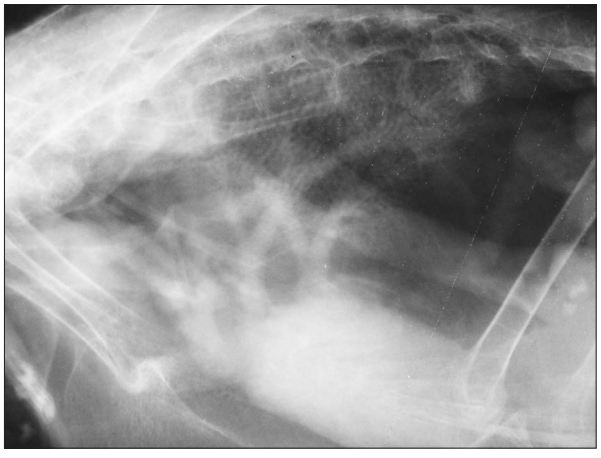


Figure 20.6 **Radiograph of an elderly cockatoo showing signs of atherosclerosis. Note the evidence of arterial calcification which is fairly specific to advanced atherosclerotic lesions.**

Angiography, using fluoroscopy or CT, may detect narrowing of arterial lumens and may be the preferred means of testing for atherosclerosis.

Management – treatment and prevention

Managing risk factors: Identifying and minimising the known risk factors may assist in the prevention of atherosclerosis. Controlling and treating reproductive problems such as chronic egg laying, oophoritis, etc. as well as dietary management and increasing physical activity may have beneficial effects on a bird before atherosclerosis develops or becomes advanced.

Statins: A variety of statins (e.g. atorvastatin, rosuvastatin) have been advocated to lower cholesterol levels, but none appear to be consistent in their efficacy, nor have their pharmacodynamics been determined. Target levels of blood cholesterol and LDL that would reduce atherosclerotic risks have not been determined.

Omega-3 and Omega-6 fatty acids: Omega-3 and -6 fatty acids show promise as a means of decreasing cholesterol levels in both mammals and birds. The ratio of Omega-3 to Omega-6 (3:1) is more important than absolute levels. Flax seed oil is a suitable source of Omega-3 fatty acids for birds.

Treating cardiovascular disease: Clinical signs of peripheral arterial disease have been anecdotally treated with pentoxifylline or isoxsuprine. Medical management of congestive heart failure, as discussed earlier, may also be appropriate if clinical signs are present.

Beta blockers (e.g. propranolol, oxprenolol) may have a protective effect against the development of atherosclerotic plaques. Verapamil, nifedipine and diltiazem (calcium channel blockers) may decrease the extent and distribution of atherosclerotic lesions. However, the lack of accurate means of measuring the arterial blood pressure in clinical cases to both diagnose and monitor the response to treatment makes these therapies difficult to justify at this time.

Downloaded From Vet-eBooks.com

HYPERTENSION

Definition/overview

Systolic blood pressures (measured with a Doppler) >200 mmHg (in conscious birds) or >160 mmHg (in anaesthetised birds) are considered to indicate hypertension in psittacine birds.

Blood pressure is determined by cardiac output and systemic vascular resistance. Cardiac output is determined by heart rate, preload and myocardial contractility. Tachycardia and increased myocardial contractility usually do not result in hypertension, as there is a corresponding decrease in systemic vascular resistance. Acute fluid overload can trigger a hypertensive episode.

The common denominator in the majority of hypertensive patients is an increase in systemic vascular resistance. This increase is mediated by increased levels of circulating catecholamines, increased alpha-adrenergic activity and activation of the renin–angiotensin–aldosterone system.

Aetiology

Primary or essential hypertension, although common in people, has been only occasionally documented in dogs. Its occurrence in other species, including birds, is unknown. It is due to excessive catecholamine release due to chronic stress.

Renal failure is relatively commonly associated with severe hypertension (through activation of the renin–angiotensin–aldosterone system) in dogs and cats, particularly in patients with protein-losing nephropathy (glomerular nephritis). The association between protein-losing nephropathy and hypertension in birds has not yet been documented, although glomerular nephritis is commonly found. Renal tumours may induce hypertension (through activation of the renin–angiotensin–aldosterone system) and are common in parrots. An association between renal tumours and hypertension has been documented in parrots.

Atherosclerosis, as discussed above, is another risk factor for the development of hypertension.

Clinical presentation

Signs include ocular lesions (retinal haemorrhages, detachments, and/or papilloedema), neurological signs (depression, confusion, and seizures), cardiovascular signs (ventricular hypertrophy and congestive heart failure) and renal system abnormalities (glomerulonephritis).

Diagnosis

Diagnosis is based on clinical signs and indirect (Doppler) blood pressure measurement. Although this measurement is not reliable in parrots, direct arterial blood pressure measurement is invasive and rarely clinically practical.

Treatment

In patients without hypertension-related disease, treatment should be initiated cautiously with the goal of reducing blood pressure by 25% over several weeks. In patients with acute, severe hypertension-related disease, rapid reduction in blood pressure may be necessary.

Drugs used include ACE inhibitors (benazepril, enalapril). Calcium channel blockers (e.g. amlodipine) offer promise, but have not yet been trialled in birds.

FURTHER READING

Beaufrère H (2013) Atherosclerosis and vascular medicine. *Journal of Exotic Pet Medicine* **22**(**4**):315–420.

Beaufrère H, Cray C, Tully T (2014) Association of plasma lipid levels with atherosclerosis prevalence in Psittaciformes. *Journal of Avian Medicine and Surgery* **28**(**3**):225–231.

Hanley SH, Helen GM, Torrey S (1997) Establishing cardiac measurement standards in three avian species. *Journal of Avian Medicine and Surgery* **11**(**1**):15–19.

Johnson HJ, Phalen DN, Kondik VH, Tippit T, Graham DL (1992) Atherosclerosis in psittacine birds. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 87–93.

Lichtenberger ML, Orosz S (2008) Cardiology: from anatomy to treatment. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 237–249.

Pees M, Krautwald-Junghanns ME, Straub J (2006) Evaluating and treating the cardiovascular system. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 379–394.

Wells SM, Sleeper M. (2008) Cardiac troponins. *Journal of Veterinary Emergency and Critical Care* **18**(**3**): 235–245.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 21

DISORDERS OF THE LYMPHATIC AND HAEMATOPOIETIC SYSTEMS

OVERVIEW

The lymphatic organs are classified as primary (thymus and bursa of Fabricius) or secondary tissue (spleen; Harderian gland; pineal gland; bone marrow; scattered aggregates of lymphoid tissue). The anatomy and function of these organs and tissues are described in Chapter 1, Clinical Anatomy and Physiology pp. 42–43. An important feature to note is the lack of lymph nodes in birds; rather, lymphoid tissue is diffusely disseminated in many tissues including the liver, spleen, kidney, and gastrointestinal tract.

Thymic cysts

Aetiology

The aetiology of thymic cysts is unknown, but they may be congenital abnormalities associated with persistent thymopharyngeal ducts, or acquired during thymic involution. They are similar in appearance and location to ultimobranchial cysts.

Clinical presentation

The cysts may fill with a colloidal fluid or gel and present as swellings on the neck. If large enough they may cause dysphagia and dyspnoea.

Management

Surgical removal of the cysts is recommended, but is often technically difficult because of extensive attachments to surrounding tissues.

Premature thymic and/or bursal atrophy

As a bird approaches maturity, the thymus normally involutes. The bursa normally involutes in birds at 18–20 months of age. Premature thymic and bursal atrophy results in the loss of the lymphocyte population (T cells) and humoral immunity (B cells). Such an event is particularly important in young birds as it makes them more susceptible to many bacterial diseases and some fungal infections.

Causes include acute or chronic disease: viral infection (e.g. circovirus; polyomavirus; avian pox virus; adenovirus; herpesvirus); bacterial, yeast or parasitic infections (e.g *Cryptosporidium*); toxins (e.g. oil; mycotoxins; pesticides); malnutrition (e.g. hypervitaminosis D3; hypovitaminosis A; calorie-deficient diets) and poor management leading to environmental stress (e.g. excessive temperatures; humidity; noise).

Splenic atrophy

Hypoplasia or atrophy of the spleen may occur secondary to bursal disease. Lymphoid necrosis can be due to chronic disease or other severe stress. Atrophy also occurs naturally with age.

Splenomegaly

Unlike in mammals, the bird spleen is a lymphoid organ not a blood reservoir. Splenic enlargement therefore reflects lymphoid stimulation or lymphoid hyperplasia/neoplasia.

Infectious causes include:

- Acute viral disease: (e.g. polyomavirus; adenovirus; herpesvirus).
- Bacterial infections: (especially *Salmonella* spp., and *Yersinia pseudotuberuclosis*) or septicaemia from gram-negative bacterial infections.
- Chlamydiosis: (see **Fig. 17.6b,** Chapter 17, Disorders of the Liver, p. 260).
- Mycobacterial infections.
- Mycotic infections.
- Parasites: *Atoxoplasma*; haemoparasites (e.g. *Plasmodium* spp.).

Downloaded From Vet-eBooks.com

Non-infectious causes include amyloidosis or neoplasia (lymphosarcoma; fibrosarcoma; haemangiomas; haemangiosarcomas; metastatic carcinomas).

Diagnosis

Diagnostic imaging (radiology, CT and ultrasound) can indicate splenomegaly but not determine the cause. Haematology, biochemistries and other diagnostic modalities for infectious disease (e.g. serology, PCR) should be pursued.

Endoscopic biopsy of the spleen via left flank coelioscopy has proven to be safe (mild to moderate haemorrhage can be observed but does not require intervention) and effective; good quality biopsy samples can be obtained.

Treatment

Treatment is directed by clinical pathology and biopsy results.

Lymphosarcoma

Lymphosarcomas (**Figs 21.1a** and **b**) arise from lymphocytes, either of thymic origin (T cells) or bursal origin (B cells). It is the most common lymphoid neoplasia reported in parrots and passerine birds, and is most commonly multicentric with tumours developing anywhere in the body where lymphoid tissue can be found, in particular the liver, spleen and kidney. They may be solid, cystic or haemorrhagic. Less common forms of lymphosarcoma include:

- Epitheliotropic lymphosarcoma, where neoplastic lymphocytes with an affinity for the epidermis and adnexal structures diffusely infiltrate the skin. This presents as a pruritic hyperkeratotic dermatitis, associated with alopecia (see **Fig. 10.17**, Chapter 10, Disorders of the Skin and Feathers, p. 175).
- Non-epitheliotropic cutaneous lymphosarcoma has been reported in several species of birds.
- Several cases of exophthalmos in parrots have been diagnosed as retrobulbar lymphosarcoma.
- Intestinal lymphosarcoma has been reported in individual cases involving several species where the presenting complaint was weight loss, accompanied by inappetance and lethargy.

Diagnosis

Haematology may demonstrate a leukaemic profile, although the combination of leukaemia and lymphosarcoma is uncommon in birds. Hypercalcaemia, commonly seen in dogs with lymphosarcoma, is rare in birds. Other biochemical changes may reflect hepatic or renal dysfunction.

Monoclonal hyperglobulinaemia is suggestive of a possible lymphoproliferative disorder and has been reported in several birds with lymphosarcoma. The monoclonal bands or spikes found using protein electrophoresis are considered to be representative of a single clone producing an abnormal amount of a single immunoglobulin, and may reflect the production of acute phase proteins.

Diagnostic imaging (radiology, CT and ultrasound) may reveal enlargement of the liver, spleen and kidney.

Although fine needle aspiration of palpable masses is technically simple, the results may not be diagnostic for lymphosarcoma. Surgical biopsy (incisional or excisional) allows the pathologist to examine not only the cell type but also the tissue architecture, giving a more accurate diagnosis. Immunohistochemistry staining of the biopsy then allows differentiation of T cell and B cell lymphosarcoma, an important guide to both treatment and prognosis (Note: B cell is a Bad prognosis, while T cell gives a Terrible prognosis).

Management

Chemotherapy protocols for avian lymphosarcoma have been developed and have moderate degrees of success. Most are extrapolated from small animal medicine, as only limited reports of treatment in birds are available, see Chapter 30, Oncology p. 423 for further discussion on the use of chemotherapy in birds. Some protocols that have been suggested include:

1. Oral medications:
   a. Prednisolone and cyclophosphamide.
   b. Lomustine (CCNU).
2. Injectable protocols:
   a. COP-L Protocol.
      i. L-asparaginase injection.
      ii. Oral prednisolone (tapering dose).
      iii. Oral weekly cyclophosphamide.
      iv. Vincristine injections weekly IV.

Downloaded From Vet-eBooks.com

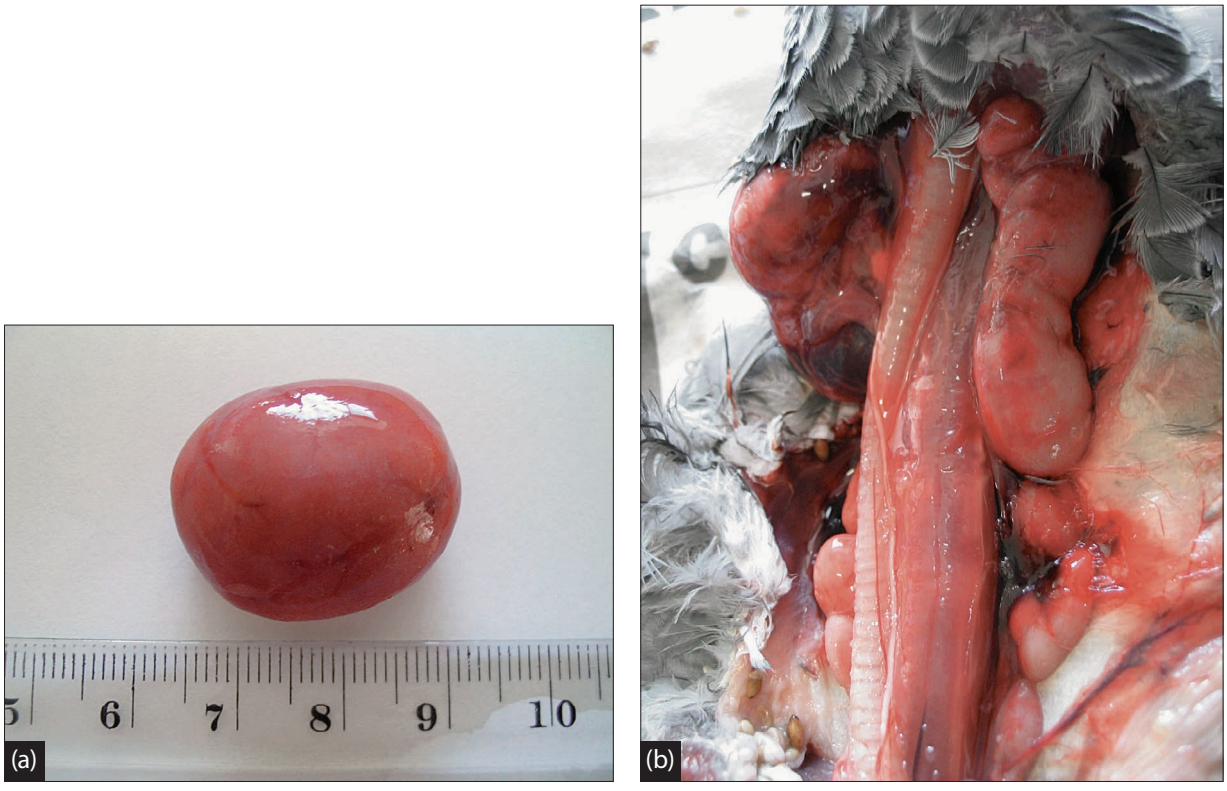


Figure 21.1a,b **Necroscopy of an African grey parrot reveals a massive splenomegaly that was due to neoplastic infiltration (a) and lobulated masses running down the neck; the thymic gland infiltrated with neoplastic cells (b).**

b. Single-agent adriamycin injections q3 weeks IV.
c. UW-Madison Cyclic Combination:
   i. L-asparaginase.
   ii. Vincristine.
   iii. Cyclophosphamide.
   iv. Adriamycin.
   v. Prednisolone.

Dose rates for these protocols have not been fully evaluated in birds, and need to be used with caution. The suggested doses are extrapolated from treatment regimens in small animals. Owners should be made aware of the limitation of current knowledge and the possibility of unexpected side-effects.

The effect of the renal portal system on the pharmacokinetics of chemotherapy drugs is not well known, and it may be advisable to give these drugs in the front half of the body where appropriate. When long-term intravenous administration of tissue irritant drugs is contemplated, the use of surgically implanted vascular access ports should be considered.

Close monitoring of patients for treatment-related side-effects is important. These side-effects include depression, vomiting, and heteropaenia.

Standard precautions for the use of chemotherapy apply, including appropriate personal protective equipment, suitable waste disposal, and separate caging. If treatment is given on an outpatient basis, owners must be cautioned to wear gloves when cleaning the bird's cage for 2–3 days post-therapy, especially as exact excretion times are unknown.

DISEASES OF THE BONE MARROW

The bone marrow is the main site of granulopoiesis (granulocyte production) and erythropoiesis (erythrocyte production). It is also a source of some long-lived lymphocytes. Various disorders can affect the

Downloaded From Vet-eBooks.com

bone marrow. Lesions can be hypocellular or hypercellular; granulocytic or erythroid. Evaluation of bone marrow aspirates requires comparison with peripheral blood.

Infectious diseases caused by bacteria, fungi, *Chlamydia* or mycobacteria, for example, usually trigger a hypercellular granulocytic response leading to a leucocytosis. Some viral infections (e.g. herpesvirus; poxvirus; reovirus; polyomavirus) may have the opposite effect, inducing leucopaenia. All infectious diseases have the capacity to cause a hypocellular erythroid response, leading to a non-regenerative anaemia.

Various toxins can affect the bone marrow, causing a broad hypocellular response. These include ochratoxin A (produced by *Aspergillus ochraceous*), sulphaquinoxaline, cisplatin and fenbendazoles. Lead toxicosis induces a hypocellular erythroid response, leading to anaemia.

Myeloproliferative disorders (neoplastic proliferation of non-lymphoid haematopoietic cells in the bone marrow which subsequently invade the spleen and liver) are uncommon. They can cause anaemia but, histologically, it may be difficult to tell which cell lines are involved, particularly if the cells are immature. Haemangiosarcomas can be primary neoplasms of the bone marrow.

Lymphoid leukaemia is a tumour of the peripheral blood and bone marrow, in contrast to lymphosarcoma which develops as solid tumours in multiple tissues but can also involve the bone marrow. Affected birds may show signs of lethargy and weakness, while haematology demonstrates dramatic lymphocytosis. The advanced stage of lymphoid leukaemia at the time of diagnosis may make it difficult to identify the origin of the neoplasia in birds, but immunohistochemistry can determine if the predominant cell type is a T cell or B cell. Treatment is warranted when the bird shows clinical signs of disease, a dramatic increase in the peripheral lymphocyte count, evidence of organ involvement, associated haematologic abnormalities such as anaemia, and rapid lymphocyte doubling time in less than six months. Complete cure is rare; the aim is to reduce the number of circulating lymphocytes while sparing the bone marrow. Most treatment protocols most are based on an alkylating agent (e.g. chlorambucil or cyclophosphamide) paired with prednisone.

PRIMARY DISORDERS OF THE IMMUNE SYSTEM

Immune suppression

Immune suppression can occur either directly or indirectly.

Direct suppression is the inhibition of plasma cells, lymphocytes and macrophages, preventing antibody production, cell-mediated immunity or antigen processing. It occurs with:

- Drugs: tetracycline, tylosin and gentamicin can decrease antibody production.
- Aflatoxicosis: depresses complement activity and decreases phagocytic activity; also impairs cell-mediated immunity through inhibition of thymic-associated lymphocytes.
- Some viruses (e.g. influenza A virus) undergo antigenic variance wherein the surface proteins of the virus change and prevent immediate detection.
- Bursal and thymic atrophy (see above).

Indirect suppression occurs through the activation of the adrenal gland (by environmental stress) and subsequent production of corticosterone. This, in turn, inhibits antibody-forming cells and macrophage activity.

IMMUNE-MEDIATED DISORDERS

Allergies

Allergic dermatitis has been reported as a cause of feather-picking and dermatitis in parrots. Confirmation can be difficult, as histological lesions are not definitive. Intradermal skin testing in birds is still to be completely validated, but it remains promising.

Allergic pneumonitis (respiratory hypersensitivity) is seen occasionally in macaws exposed to powder down/feather particles from other parrots and organisms such as *Aspergillus* spp. See Chapter 19, Disorders of the Respiratory System p. 278.

Immune-mediated haemolytic anaemia

Definition/overview

Immune-mediated haemolytic anaemia (IMHA) occurs when antibodies produced by the bird's

Downloaded From Vet-eBooks.com

immune system target the bird's own erythrocytes. It is seen infrequently in poultry and is rarely documented in parrots (only two documented cases at the time of writing).

Clinical presentation

Signs include: lethargy; weakness; weight loss; pallor; polyuria; biliverdinuria.

Diagnosis

Haematology demonstrates a strongly regenerative anaemia with a predominance of round, small erythrocytes (spherocytes), leucopaenia and/or leucocytosis, and elevated plasma protein. Serum biochemistries are non-specific.

Saline autoagglutination may be positive. Blood is diluted with sterile saline (3:1 dilution) in an EDTA tube. This is then centrifuged and the supernatant removed. The remaining cells are washed several times with saline solution, and then examined grossly and microscopically. If IMHA is present, autoagglutination (clumping) of the erythrocytes is visible grossly; microscopic examination is used to confirm true autoagglutination and not rouleaux formation.

Radiographic changes include marked splenomegaly and possibly hepatomegaly.

Liver, spleen and bone marrow biopsies can be evaluated, both for evidence of other haemolytic diseases (e.g. malarial parasites) and evidence of regenerative changes (in the liver, spleen and bone marrow) and haemosiderosis (in the liver). Bone marrow cytology may not demonstrate the expected low myeloid-to-erythroid cell ratio as seen in mammals. This may be because, in birds, erythropoiesis occurs in the bone marrow primarily but maturation of the erythroid cell series occurs intravascularly, within bone marrow and in other haematopoietic organs such as the liver and spleen. These cells may therefore be seen in blood smears and liver and splenic biopsies.

Management

Treatment options for parrots with IMHA involve the administration of immunosuppressive agents such as corticosteroids, cyclosporine, and cyclophosphamide. These drugs have not been widely used in birds, and their use must be extrapolated from small animal medicine.

Membranous glomerulonephropathy

Definition/overview

See Chapter 24, Disorders of the Urinary System. Glomerular disease is an important cause of end-stage renal disease in birds.

Aetiology

The cause of glomerulopathies is generally assumed to be immune-mediated, but the inciting aetiology is often unknown. PCR has been used to identify polyomavirus particles in the kidneys of many affected birds.

Diagnosis

Although proteinuria is the hallmark sign of glomerulonephritis in mammals prior to the onset of clinical renal insufficiency, avian leucocytes lack the proteolytic enzymes that would potentially damage the glomerular basement membrane (and allow protein leakage). Therefore, birds may not develop pathological proteinuria with glomerulopathies.

Renal biopsy is the best way to definitively diagnose glomerular (and other) kidney diseases in birds.

Management

Regardless of the possible causes, identifying and correcting underlying inflammation and/or systemic disease(s) are the first steps in managing renal disease. Omega-3 and -6 fatty acids and low dose aspirin have shown promise in treating this condition.

Transfusion reactions

Transfusion reactions occur due to the incompatibility of donor red cells with host plasma. They have been reported in birds given multiple heterologous transfusions. Transfusion reactions may result in haemolysis of the donor red cells. Death may be the only sign associated with a transfusion reaction in a bird.

FURTHER READING

Gerlach H (1994) Defence mechanisms of the avian host. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 109–120.

Downloaded From Vet-eBooks.com

Johnston MS, Son TT, Rosenthal KL. (2007) Immune-mediated hemolytic anemia in an eclectus parrot. *Journal American Veterinary Medical Association*. **230**(**7**):1028–1031.

Jones JS, Thomas JS, Bahr A, Phalen DN (2002) Presumed immune-mediated hemolytic anemia in a blue-crowned conure (*Aratinga acuticaudata*). *Journal Avian Medicine and Surgery* **16**(**3**):223–229.

Lightfoot TM (2006) Clinical avian neoplasia and oncology. In: *Clinical Avian Medicine.* Harrison G, Lightfoot TM (eds). Spix Publishing, Palm Beach, Florida pp. 560–566.

Ritchie BW (1995) Viral attack and avian response. In: *Avian Viruses: Function and Control*. Wingers Publishing, Lake Worth, pp. 47–82.

Schmidt RE (1997) Immune system. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 645–652.

Downloaded From Vet-eBooks.com

CHAPTER 22

DISORDERS OF THE NERVOUS SYSTEM

INTRODUCTION

Birds showing neurological disorders, including loss of consciousness, seizures, abnormal mentation, ataxia, paresis or paralysis of legs and/or wings, cloacal atony, head tilt and nystagmus, are frequently presented to veterinarians.

Diagnosing neurological disorders requires a thorough examination in order to determine if the neuropathy is focal or diffuse. If the examination finds that dysfunction is present at more than one level, the lesion can be assumed to be either located at the highest location or multifocal. If the dysfunction is focal, an attempt is then made to localise the lesion to the head, cervical, notarial or synsacral spinal cord. If the examination fails to localise the lesion, a metabolic or generalised neuromuscular lesion should be considered.

As with any examination, a neurological assessment begins with a detailed history and involves a thorough physical examination before moving on to diagnostic testing to confirm a diagnosis or further localise the lesion.

HISTORY

The bird

The age of the bird is important: hereditary and inflammatory conditions are more common in the young bird, while degenerative and neoplastic conditions are more frequent in the older bird. Questions should be asked about diet, as malnutrition can be a primary cause of some neurological conditions. The bird's lifestyle should be determined with regard to access to toxins and likelihood of trauma. Reproductive activity may indicate the possibility of a yolk embolism. It is also important to ask the owner for details of any previous medical problems.

The problem

If the condition is of acute onset, one should consider toxicosis, trauma, vascular accident, or generalised severe inflammation. If onset is more chronic, degenerative changes, neoplastic disease or low-grade inflammation is more likely. If the disorder is progressive, a clear description of the chronological development of the clinical signs must be obtained.

The owner should be asked about the bird's mental state and demeanour, including changes to personality and behaviour. A precise description of the clinical signs is important (e.g. seizures may be difficult to differentiate from syncope). Generally, seizures are characterised by ataxia, disorientation, and falling off the perch, remaining rigid or having some form of motor activity for varying lengths of time.

DISTANT EXAMINATION

Mental status

If the bird is showing uncharacteristic aggression, a psychomotor-like epilepsy or a space-occupying mass in the forebrain should be considered. A depressed response to stimuli may result from lesions in the brainstem or forebrain. Semi-consciousness may be caused by acute encephalitis, forebrain trauma or neoplasia.

Posture

If there is a head tilt, lesions may be present in cranial nerve VIII. Tremor may be caused by lesions in the cerebellum or vestibular system, and falling or loss of balance may also be because of lesions in the vestibular system.

Flight

Uncoordinated flight or non-rhythmic fanning of wings may be related to a poor wing clip or lesions in

Downloaded From Vet-eBooks.com

the cerebellum, vestibular system or corpora striata. If the bird has poor obstacle avoidance, lesions in the eyes, cranial nerve II or the visual centre in the brain (neoplasia, abscess, granulomas) should be considered.

If the bird has poor take-off or landing, one should consider a poor wing clip or lesions in the cerebellum, vestibular system, visual system, spinal cord or peripheral nerves. Paresis (flight not sustainable for lengthy periods) may be due to systemic disorders or lesions in the spinal cord or peripheral nerves.

Gait

Ataxia may be caused by lesions in the cerebellum or vestibular system. Dysmetria (inability to control or limit movement) may be due to lesions in the cerebellum. Circling can result from lesions in the vestibular system (unilateral) or corpora striata.

Poor obstacle avoidance may be due to lesions in the eyes, cranial nerve II or the visual centre, and poor righting reflex may be a result of lesions in the vestibular system, corpora striata, visual or proprioceptive systems, cerebellum, spinal cord or peripheral nerves.

Other

Difficulties with prehension (abnormal tongue movement, reduced beak strength) may be a result of lesions in cranial nerves V and IX–XII. Reduced perching ability (not able to grasp a perch and support weight) can be due to lesions in the peripheral receptors, corpora striata, vestibular or visual system, cerebellum, spinal cord or peripheral nerves.

PHYSICAL EXAMINATION

A physical examination can be conducted after obtaining a thorough history and observing the patient from a distance. The examination should be performed methodically and logically. However, the order of the neurological examination depends on the clinical condition and cooperation of the patient. It is important to take into account the degree of cooperation of the patient when interpreting responses to neurological tests. Examination of the neurological system is detailed in Chapter 3, The Physical Examination pp. 78–79.

DIAGNOSTIC TESTS

Tests used in the diagnosis of neurological disorders include haematology and biochemistry, toxin analysis (especially blood lead levels), diagnostic imaging (radiography, CT, MRI), PCR and serology for infectious diseases such as avian bornavirus and *Chlamydia*, electroencephalograms (continuous recordings of the electrical activity of the cerebral cortex) and electromyograms (can be used to detect diseases of motor neuron cell bodies, ventral nerve roots, nerve plexuses, peripheral nerves, neuromuscular junctions and muscle fibres).

CENTRAL NERVOUS SYSTEM DISORDERS

Congenital/hereditary conditions

Hydrocephalus has been seen in older parrots, although possibly acquired rather than hereditary. Lafora body neuropathy has been reported, mainly in cockatiels. Glycoprotein-containing cytoplasmic inclusion bodies form within neurones, probably as a result of a defect in intracellular metabolism.

Teratogenic toxins such as dioxins can lead to asymmetrical brain development.

Lysosomal storage disease has been reported in emus, but not in companion birds.

VIRAL DISEASES

Paramyxovirus 1, 2, 3 and 5

Definition/overview

Paramyxovirus 1 (PMV-1, Newcastle disease) is seen in poultry, parrots and pigeons. PMV-2 is most commonly isolated from passerines. PMV-3 is the main paramyxovirus affecting parrots. Transmission of PMV-1 is horizontal through respiratory, faecal and oral secretions. Mechanical vectors (wind, insects, equipment and humans) may also spread the virus. Vertical transmission is possible. The incubation period may be between three and 28 days, depending on the strain. PMV-1 is a zoonotic disease. In humans (the only mammal to have clinical signs) there is mild acute granular conjunctivitis, general malaise and sinusitis, with recovery in 7–20 days.

Downloaded From Vet-eBooks.com

Clinical presentation

PMV-1: PMV-1 may result in sudden death. Clinical signs can involve the CNS, respiratory and gastrointestinal systems, including depression, tremors, paralysis, a twisted neck, ataxia, opisthotonos, torticollis, conjunctivitis, nasal discharge, polyuria and diarrhoea.

PMV-3: PMV-3 may cause pancreatitis, lymphoplasmocytic myocarditis, otitis media and otitis interna. In Neophemas, there is pulmonary oedema/congestion and hepatomegaly (high flock morbidity and low mortality). In nestling cockatiels there may be opisthotonos, tremors, leg paralysis, dyspnoea and a high mortality. In finches there may be initial conjunctivitis, anorexia, yellowish diarrhoea and dyspnoea.

Diagnosis

Diagnosis is made through serological testing (PMV-3 can cross-react with PMV-1) or viral isolation and culture.

Management

Vaccines (live or inactivated) are available, but may not be suitable for all species. The virus is inactivated by high temperatures (>56°C), sunlight, detergents, 1% chloramine, sodium hypochlorite, lysol, phenol and 2% formalin.

Proventricular dilatation disease (Avian Bornavirus; PDD)

PDD can present as a primarily neurological disease (**Fig. 22.1**) (see Chapter 16, Disorders of the Gastrointestinal System pp. 235–238).



Figure 22.1 Moluccan cockatoo with leg paresis due to proventricular dilatation disease.

West Nile virus

Definition/overview

This is a flavivirus that is transmissible to birds, people and horses. Birds act as a reservoir for the virus, while mosquitoes act as vectors. The disease is seen in Africa, the Middle East, Europe, Asia and North America. In birds, death is caused by myocarditis and encephalitis.

Clinical presentation

Clinical signs include sudden death or depression, anorexia, weakness, weight loss and recumbency. Some birds show neurological signs, including abnormal head/neck posture, ataxia, tremors, circling, disorientation, paresis and impaired vision.

Management

Prevention revolves around vaccination and mosquito control.

Avian Polyomavirus

Polyomavirus in budgerigars can cause cerebellar disease with marked intention tremors. It has also been observed in cockatoos with concurrent PBFD.

Herpesvirus

Pigeon herpesvirus can cause encephalomyelitis in pigeons. Parakeet herpesvirus affects *Neophema* and *Psittacula* parrots. They present with stargazing, stumbling, tremors, torticollis and dyspnoea. Marek's disease is seen in poultry, turkeys and quail.

Adenovirus

This rarely acts as a primary pathogen; one case the author saw was secondary to PBFD. Neurological signs have been noted in cockatiels and budgerigars. A non-suppurative encephalitis is seen on pathology.

Togavirus

Togavirus causes eastern equine encephalitis and western equine encephalitis in emus, with clinical signs including ataxia, depression, anorexia, haemorrhagic diarrhoea and emesis, and ending with death. Togavirus is also considered to the cause of avian

Downloaded From Vet-eBooks.com

viral serositis, which can result in non-suppurative meningitis and encephalitis in parrots.

Other viruses

A suspected viral encephalomyelitis is seen in lorikeets and *Polytelis* spp. parrots presenting with progressive bilateral paralysis of the legs, with clenched feet (see Chapter 14, Disorders of the Legs, Feet, and Toes, pp. 214–215).

Avian influenza virus may cause loss of balance, ataxia and torticollis in some birds, although neurological signs are relatively rare.

BACTERIAL INFECTIONS

Bacteria identified as causing meningitis, encephalitis and myelitis include *Salmonella*, *Listeria*, *Staphylococcus* spp., *E. coli*, *Pseudomonas* spp., *Klebsiella* and *Mycobacterium*.

FUNGAL DISEASES

Aspergillus spp. can invade the brain, spinal cord and meninges via haematogenous spread or extension from the nasal cavity, sinuses or air sacs. (See Chapter 19, Disorders of the Respiratory System, pp. 280–283).

CHLAMYDIA

Chlamydia can produce a non-suppurative meningitis (see Chapter 17, Disorders of the Liver pp. 259–262). This can result in seizures, tremors, torticollis and opisthotonos. In cockatiels, weakness or paralysis of the legs is occasionally seen.

PROTOZOAL DISEASES

Sarcocystis falcatula

This can cause an encephalitis in parrots and passerines (see Chapter 15, Disorders of the Musculoskeletal System p. 226).

Toxoplasma gondii

Definition/overview

Toxoplasma gondii infection is noted primarily in Galliformes and passerines, but has been diagnosed in parrots, raptors and owls. Cats shed the infective oocysts, which are then consumed by an intermediate host or by a bird.

Clinical presentation

Clinical signs include anorexia, diarrhoea, blindness, conjunctivitis, head tilt, circling and ataxia.

Diagnosis

Toxoplasmosis is diagnosed by serology or histopathology.

Management

Treatment is with pyrimethamine, toltrazuril, diclazuril, or trimethoprim/sulphadiazine.

Leukocytozoon

This may form megaloschizonts in the brain (see Chapter 15, Disorders of the Musculoskeletal System p. 227).

NEMATODES

For the clinician and pathologist alike, the diagnosis of cerebrospinal nematode infections in birds can be problematic; the clinical signs are non-specific, and there are no suitable antemortem tests specific for these parasites. Diagnosis is often made by necropsy; fortunately, this condition is rare.

Baylisascaris procyonis (the raccoon roundworm) causes ataxia, recumbency and other locomotor abnormalities in birds eating the faeces of infected racoons. It has been reported in cockatoos, cockatiels and emus in the USA.

Chandlerella quiscali is a parasite of grackles in the USA, transmitted by gnats. When emus are infected, adult worms develop in the lateral ventricles of the cerebrum, causing torticollis, ataxia, recumbency and death.

Wild-caught cockatoos from Indonesia are occasionally found to have microfilaria plugging the small vessels in the brain.

Migrating larvae of the rat lungworm (*Angiostrongylus cantonensis*) have been reported in the brain of yellow-tailed black cockatoos displaying neurological signs in Australia.

Downloaded From Vet-eBooks.com

TRAUMA

Aetiology
Concussive head injuries are common in fully flighted birds that fly into ceiling fans, windows or aviary wire. They may also occur when a bird is attacked by another bird, either a cage mate or a wild predatory bird.

Clinical presentation
Clinical signs include depression, head tilt, circling and unilateral or bilateral paresis of the wings or legs. Radiographically, skeletal trauma may be visible. In many birds an air-filled space is visible between the heart and the lung, presumably because of air sac rupture.

Diagnosis
Diagnosis is based on history and neurological examination, or on necropsy. Traumatic injuries usually result in haemorrhage in the meninges, extending into the brain parenchyma. There is often associated bruising and trauma to the skin and skull.

Post-mortem pooling of blood in the venous sinuses of the calvarium is common with any cause of death, and must not be confused with traumatic injuries.

Treatment
Shock, pain and infection should be treated as appropriate. Some birds may require assisted feeding until neurologically stable.

Prognosis
Prognosis improves with neurological improvement. In some cases this may take several weeks; improvement after 3–4 weeks is unlikely.

CEREBROVASCULAR ACCIDENTS

Aetiology
Thrombosis and/or aneurysm rupture can lead to acute ischaemia and/or haemorrhage within the brain. Common causes of these emboli include atherosclerosis in older birds, and yolk emboli in reproductively active females.

Clinical presentation
Affected birds present with neurological signs that can be localised to the CNS (head tilt, nystagmus, seizures, etc.). Their signalment may suggest either cardiovascular disease or reproductive activity.

Diagnosis
Serum biochemistries may indicate either potential cardiovascular disease or reproductive activity.

Advanced diagnostic imaging e.g. MRI may assist in diagnosing and localising a lesion.

Treatment
See Chapter 20, Disorders of the Cardiovascular System, p. 292 for a discussion on treatment of atherosclerosis in birds.

If yolk embolism is suspected, reproductive rest is recommended.

Meloxicam or other NSAIDs may be helpful.

TOXINS

Lead toxicosis
Definition/overview
Lead is a both an acute toxin and a cumulative toxin. If a large amount is ingested quickly, acute toxicosis develops; however, small amounts taken in gradually will accumulate within the body until tissue levels are high enough to cause disease. There are three target organs of toxic lead intake: the nervous system, the gastrointestinal system, and erythropoietic bone marrow. The source is often not determined. It may be lead shot, leaded petrol and oil, galvanised wire, lead-based paints, lead putty, solder, foil from some champagne and wine bottles, some welds on wrought-iron cages, lead weights (curtain and fishing), bells with lead clappers, lead-lighted glass, improperly glazed ceramics, batteries, bird toys with lead weights, costume jewellery or mirror backs.

Pathophysiology
Lead is solubilised in the ventriculus by the combination of its grinding action and the low pH (2–3.5). Once solubilised, lead is absorbed across the intestinal mucosa and is bound to metalloprotein for distribution around the body. Lead competes for calcium at the myoneurological junction, resulting

Downloaded From Vet-eBooks.com

in neuromuscular blockage, and it affects neuronal cell adhesion molecules and glucocorticoid receptors in neurones and glial cells. It also suppresses aminoleuvulinic acid dehydrase and heme synthetase, leading to an accumulation of aminoleuvulinic acid and protoporphyrin IX in erythrocytes, which inhibits the heme synthesis cascade. This leads to decreased erythrocyte production and anaemia.

Clinical presentation

Multifocal neurological signs include hyperexcitability, ataxia, paresis and paralysis (**Fig. 22.2**), convulsions, wing droop and head tilt. Gastrointestinal signs include ileus, vomiting/regurgitation, and abnormally coloured diarrhoea (green to black). There may be renal signs of polyuria and polydypsia, and haematuria in some parrots, particularly Amazons and galahs. Anaemia causes lethargy, depression and weakness.

Diagnosis

Clinical pathology: Haematology reveals a hypochromic, regenerative anaemia. Erythrocytic ballooning is common, but not pathognomonic.

Biochemistry shows elevated LDH, AST, CK and uric acid concentrations, reflecting increased protein catabolism and renal dysfunction. In waterfowl, inhibition of delta-aminoleuvulinic acid dehydrogenase to levels <86 IU/l is suggestive of lead toxicosis.

Blood lead determination is a reliable indicator of toxicosis, as 90% of circulating lead is contained in erythrocytes. There is still debate as to what constitutes a 'normal' blood lead level. It might be better to say that below certain levels, clinical signs are unlikely to be seen. What effect these 'normal' levels are having on the patient is difficult to assess.

- 'Normal' blood level is <0.7 µmol/l (14.5 µg/dl, 0.145 ppm).
- 0.7–1.7 µmol/l (35.2 µg/dl, 0.352 ppm) suggests exposure.
- >1.7 µmol/l confirms lead toxicosis.

Diagnostic imaging: Plain and contrast radiographs often show a dilated proventriculus and other evidence of ileus. Radiodense particles may be visible in the crop, proventriculus and ventriculus, but the absence of these particles does not exclude a diagnosis of lead toxicosis.

Histopathology: Acid-fast intranuclear inclusions are found in epithelial cells of proximal convoluted tubules and hepatocytes. There may also be neuronal degeneration (cerebral cortex, optic lobes, medulla oblongata) and Zenker's degeneration of the myocardium, the ventriculus, and pectoral muscles (hyaline degeneration and loss of muscle striations).



Figure 22.2 **Rainbow lorikeet with leg paresis due to lead toxicosis.**

Management

Chelation: Calcium EDTA is given at a dose of 20–40 mg/kg IM q12h. There are reports of doses up to 100 mg/kg q12h being given without any evidence of nephrotoxicity. Metal ions displace the calcium in CaEDTA to form a water-soluble complex that is excreted in the urine. The author prefers to treat twice daily until the patient is asymptomatic (usually 3–5 days), then twice weekly until blood lead levels are in the 'normal' range (often up to six weeks). Response is usually rapid, with a clinical improvement usually noticeable within 24–36 hours.

D-penicillamine is an effective oral chelator, but it may cause nausea and vomiting in some birds.

Downloaded From Vet-eBooks.com

Removal of particles from the gastrointestinal tract: Most small particles will pass through the gastrointestinal tract in 4–5 days. Larger pieces may have to be retrieved endoscopically or by concurrent flush and suction with warmed water. As a last resort, a proventriculotomy can be performed.

Supportive care: See Chapter 8, Supportive Care. Patients will require fluid support, nutritional support and thermal support. Blood transfusion may be required in very anaemic patients.

Botulism

Definition/overview

Botulism is most commonly a problem with game birds, ducks and pheasants, although it occurs sporadically in other wild or exotic species.

Aetiology

Botulism is due to the ingestion of bacterial exotoxins produced by *Clostridium botulinum*, a widely dispersed saprophytic bacterium found in soil. This toxin (type C predominantly, although types A and E are sometimes identified) develops when *C. botulinum* grows in anaerobic conditions. It is present in vertebrate carcasses, decaying vegetation and dead invertebrates found in stagnant ponds, wetlands and marshes. Fly maggots and aquatic insects accumulate the toxin and are then ingested by birds. It is most often seen in late summer and early autumn when many ponds are drying out and water levels drop, exposing dead animals and rotting vegetation.

Pathophysiology

The toxin interferes with release of acetylcholine (ACH) at motor endplates.

Clinical presentation

Affected birds show ataxia and flaccid paralysis of the legs, wings and neck ('limber neck'). There may be diarrhoea, loose feathers, dyspnoea, gasping, conjunctivitis or sudden death.

Diagnosis

Diagnosis can be difficult, as there are few lesions found on necropsy. Botulinum toxin can be detected by a variety of techniques including ELISA, electrochemiluminescent (ECL) tests and mouse inoculation or feeding trials. The toxins can be typed with neutralization tests in mice.

Management

Treatment involves administration of antitoxin, if it is available, and supportive care (fluids, tube feeding, warmth, etc.). Anecdotally meloxicam may be of some value, presumably by reducing myositis in paretic/paralysed birds. The prognosis is usually guarded to poor.

A vaccine is available in some countries. Prevention revolves around preventing access to decaying vegetation and dead animals by birds.

Organophosphates and carbamates

Definition/overview

Birds are 10–20 times more susceptible to acetylcholinesterase inhibitors than mammals. These chemicals are used as pesticides. Exposure is usually through inhalation of aerial spraying or cutaneous absorption through washing or dipping. Occasionally, wild birds are poisoned by ingesting grain laced in organophosphates or carbamates in an attempt to control wild bird numbers around grain silos.

Pathophysiology

Organophosphates and carbamates are two different classes of pesticides that both inhibit acetylcholinesterase, the enzyme that breaks down the neurotransmitter ACH. This results in ACH accumulating at nerve synapses and subsequent continued stimulation at cholinergic nerve endings and myoneural junctions.

Clinical presentation

Clinical signs include anorexia, diarrhoea, crop stasis, ptyalism, ataxia, tremors, seizures and paralysis. Death is usually due to respiratory failure.

Prolonged exposure to organophosphates may result in 'delayed neuropathy syndrome'. The organophosphate molecules bind to neuropathy target esterase; this binding then initiates degeneration of long axons in the peripheral and central nervous systems. The effects of this are seen 7–10 days after exposure as weakness, ataxia, decreased

Downloaded From Vet-eBooks.com

proprioception and paralysis. This syndrome is not associated with the inhibition of acetylcholinesterase.

Management
Atropine 0.2 mg/kg IM is given as needed until the clinical signs stop. Atropine has little or no effect on nicotinic receptors and used alone will not counteract neuromuscular paralysis. Pralidoxime 10–20 mg/kg IM is administered every 8–12 hours as needed. It is only effective if used within the first 24 hours of exposure. It is contraindicated for carbamate toxicosis.

Chlorinated hydrocarbon (DDT)/organochlorines
Definition/overview
These chemicals have been used widely as pesticides since 1945. Exposure occurs through aerial spraying and ingestion of poisoned insects.

Pathophysiology
The mechanism of action is unclear, but it is believed to interfere with axonic transmission of nerve impulses.

Clinical presentation
Clinical signs include thin-shelled eggs and tremors and convulsions.

Diagnosis
Diagnosis is made by measuring levels of the chemicals in fat, brain and liver.

Management
Treatment is unrewarding.

Dimetridazole
This causes convulsions, wing-flapping and opisthotonos in budgerigars, goslings, pigeons and ducks.

Levamisole
Definition/overview
Levamisole causes toxicity by stimulation of cholinergic receptors in the autonomic ganglia, neuromuscular junctions and CNS. However, in most studies the reported cause of death is asphyxia and respiratory failure. In poultry, toxic reactions only occur with doses in excess of 160 mg/kg and deaths at >480 mg/kg. This is why it has gained wide acceptance as a safe anthelmintic for poultry. It is considered to be contraindicated in finches and other small passerines, and administration should be carefully monitored in other species when water intake is likely to increase (e.g. during hot weather) or when young are in the nest.

Clinical presentation
Levamisole causes regurgitation, ataxia, recumbency, catatonia, dyspnoea and death in parrots and poultry. Pulmonary oedema is a feature of levamisole toxicosis in a range of species whether given by injection or orally.

Nitrofurazone
Nitrofurazone causes screaming, convulsions, aimless running or flying and opisthotonos. There may be depression and growth retardation.

NUTRITIONAL DEFICIENCIES

Vitamins
Hypovitaminosis B1 (thiamine)
This causes anorexia, ascending paralysis and opisthotonos, polyneuritis with myelin degeneration and adrenal hypertrophy and skin oedema. This is most commonly seen in wild birds that are fed meat without supplementation. Cases respond within hours to parenteral vitamin B1.

Hypovitaminosis B2 (riboflavin)
Hypovitaminosis B2 causes curled toe paralysis in poultry and nestling budgerigars, weakness, emaciation (despite a good appetite), diarrhoea, walking on hocks with toes curled inward and leg muscle atrophy; and demyelinating peripheral neuritis with nerve oedema. Chronic cases have irreversible damage.

Hypovitaminosis E and selenium deficiency
Vitamin E deficiency in young birds causes encephalomalacia, exudative diathesis and muscular dystrophy. It is particularly common in hatchling budgerigars.

Downloaded From Vet-eBooks.com

Hypovitaminosis B6 (pyridoxine)

Hypovitaminosis B6 causes jerky, nervous walking progressing to running and flapping the wings. The bird then falls with rapid, clonic tonic head and leg movements, which usually lead to death.

Calcium

Definition/overview

Hypocalcaemic tetany is most common in adult African grey parrots.

Aetiology

It is usually associated with the long-term feeding of a diet deficient in calcium, combined with hypovitaminosis D3 (due to lack of UVB radiation or dietary supplementation). Initially the bird will maintain normal blood calcium concentrations by utilizing the calcium reservoir present in the bones, but eventually the homeostatic system fails and the blood calcium level will fall. In a normal adult grey parrot the blood ionised calcium concentration is kept within a very narrow range (0.96–1.22 mmol/l). Once the homeostatic system fails and the blood ionised calcium concentration falls below the normal level the bird will demonstrate a variety of neurological clinical signs.

Clinical presentation

Initially the bird may just twitch or flick its head. Ataxia develops and progresses to fits or convulsions.

Diagnosis

Blood calcium levels (preferably ionised calcium) should be measured and interpreted as above.

Caution must be taken not to assume that all seizuring African grey parrots are hypocalcaemic.

Treatment

The clinical signs respond rapidly to parenteral calcium and vitamin D3 supplementation. Long-term therapy should involve improvement of the diet with respect to calcium and vitamin D3 content.

UVB radiation should be provided, either from natural sunlight or artificial full spectrum lights, particularly in birds exposed to low light levels kept indoors. Care must be taken when using artificial UVB lights that UVB-induced keratitis does not develop.

NEOPLASIA

Pituitary adenomas

Definition/overview

These are either adenomas or adenocarcinomas arising from chromophobe cells in the pituitary gland. They most commonly occur in young to middle-aged male budgerigars but have been reported in other parrot species including cockatiels and one case in an Amazon. A recent study (Langohr, *et al.*, 2012) concluded that the high incidence of this disease in budgerigars suggests a possible genetic predisposition of budgerigars to develop somatotroph pituitary tumors with a high incidence of local invasion and with metastatic potential.

Clinical presentation

Clinical signs are related to compression of the brain and cranial nerves from the space-occupying mass. These signs include uncoordinated wing flapping, clonic leg twitches, seizures, circling, depression, somnolence, unconsciousness, exophthalmos, mydriasis, blindness and inappropriate behaviour responses.

If the tumour is functional, other clinical signs can include polydypsia and polyuria, feather abnormalities, cere colour changes and obesity.

Primary brain tumours

Primary brain tumours are rare. They include astrocytoma, glioblastoma multiforme, schwannomas, ganglioneuroma, neurofibrosarcomas, pineal body tumours, undifferentiated sarcomas and haemangiomas. Clinical signs are similar to those seen with pituitary tumours.

EPILEPSY/SEIZURES

Overview

A seizure is the clinical manifestation of a sudden surge of excessive electrical activity in the brain. There are several characteristic stages of a seizure incident; the prodromal stage, the seizure itself and the postictal stage. The prodromal stage is characterised by restlessness or anxiety lasting several hours or even days before the seizure. The seizure

Downloaded From Vet-eBooks.com

starts with the bird hiding, seeking the owner, or other behavioural changes minutes to seconds before the seizure itself starts, usually lasting a few minutes. Postictally there are temporary neurologic abnormalities such as disorientation, blindness, and ataxia for a few minutes or hours.

Seizures themselves can be focal (*petit mal*) or generalised (*grand mal*).

A bird exhibiting a focal seizure generally does not lose consciousness, but exhibits localised abnormal movement of a body part (e.g. wing twitching) or unusual behaviour (fear, aggression, etc.). It may progress on to a generalised seizure.

Generalised seizures generally begin with a tonic phase – all of the bird's muscles contract and the patient usually falls to one side with its limbs extended. Defecation is common during this phase. After a few minutes the clonic phase begins, in which there is rhythmic contraction of muscles (i.e. paddling, limb twitching, or chewing). This again lasts for a few minutes before the bird regains consciousness and moves into the postictal stage.

Status epilepticus is defined as a seizure lasting five minutes or more, or two or more seizures without full recovery of consciousness between seizures.

Aetiology

Epilepsy/seizures can be due to a number of causes, either intra- or extra-cranial:

Intracranial

Infectious: See earlier in this chapter.

Non-infectious:

- Toxins (lead; organophosphates; organochlorines; chocolate; caffeine; some mycotoxins).
- Neoplasia.
- Cerebrovascular accidents.
  - Ischaemic:
    - Yolk embolism.
    - Atherosclerosis.
  - Haemorrhagic:
    - Ruptured aneurysm.
    - Trauma.
- Heat stress.
- Storage diseases such as Lafora's disease.
- Idiopathic epilepsy has been reported in peach-faced lovebirds, red-lored Amazons and greater Indian hill mynahs. It is a diagnosis of exclusion, reached after ruling out other causes of seizures.

Extra-cranial

- Metabolic disorders (e.g. hypoglycaemia, hypocalcaemia).
- Hepatopathy/hepatic encephalopathy (rare in parrots).
- Renal disease.
- Respiratory insufficiency leading to hypoxia.
- Cardiac insufficiency.
- Nutritional deficiencies (vitamin E, vitamin B1).
- Iatrogenic (e.g. insulin overdose).

Management

Initial stabilisation of the seizuring bird may require a benzodiazepine (e.g. diazepam, clonazepam, or midazolam). Blood glucose should be assessed and, if low, a dextrose bolus (1 ml/kg of 50% dextrose IV) should be given. Similarly, if ionised calcium is low, calcium gluconate should be given by slow IV injection or IM.

If possible, other causes of the seizures should be identified and eliminated. A detailed history, a thorough physical examination, haematology and biochemistry, blood lead measurement, and radiographs are appropriate starting points.

Longer-term stabilisation requires anticonvulsant therapy such as phenobarbital, levetiracetam, or gabapentin. It is important to realise that there is relatively little data about the use of anticonvulsant therapy in birds; speed of absorption and drug metabolism are unknown and dose rates are largely extrapolated from small animal medicine. Monitoring the response to treatment is usually based on the patient's clinical signs. Serial assessment of liver function should be performed.

Prognosis

Unless the inciting cause can be identified and removed, the long term prognosis of the seizuring bird remains guarded. However, short to mid-term control can often be achieved with good owner compliance.

Downloaded From Vet-eBooks.com

PERIPHERAL NERVOUS SYSTEM DISORDERS

Many of the disorders that are described above may present as peripheral neuropathies. There are, however, some that are confined to the peripheral nervous system.

Trauma

Overview

Traumatic injuries, especially to the long bones, can result in peripheral nerve damage. This damage can range along a spectrum of nerve injury – neurapraxia (temporary loss of motor and sensory function due to blockage of nerve conduction), axontmesis (an interruption of the axons, but a preservation of the surrounding connective tissues around the axon; recovery will take longer than neurapraxia), neurotmesis (both the nerve and the nerve sheath are disrupted; while partial recovery may occur, complete recovery is impossible), or complete transection; the extent of the injury determines the prognosis. In many cases it is transient, but occasionally the damage can be permanent.

Clinical signs

Clinical signs include loss of withdrawal and pain reflexes, muscle atrophy, and paresis/paralysis. It is often difficult to assess the degree of nerve injury associated with a fracture. If the extent of the injury is confined to neurapraxia, recovery should be complete in 2–4 weeks.

Treatment

Supportive care, analgesia and passive range of motion exercises should be provided until the extent of injury can be assessed.

Renal neoplasia

Pathogenesis

The roots of the lumbar plexus, giving rise to the femoral nerve, are in contact with the dorsal surface of the cranial division of the kidney, while the roots of the sacral plexus and the start of the sciatic nerve are embedded in the medial division. Compression of these nerves by renal enlargement will result in paresis or paralysis of the affected leg. This is most common in budgerigars but has been reported in other species. The high incidence of this disease in budgerigars has led to a suspicion of viral aetiology, but this remains unproven.

Clinical presentation

This is nearly always a progressive, unilateral disease with paresis on one side associated with pressure exerted on the sciatic nerve by a renal tumour (carcinoma, adenocarcinoma, and embryonal nephromas). There is normal flexion and extension of all the joints of the affected limb (**Fig. 22.3**). Withdrawal reflexes below the stifle joint typically are absent or reduced when compared to the other leg. Reflexes are normally present proximal to the stifle joint and, quite often, the bird can still move its leg from front to back but will not weight-bear. The affected leg is held loosely on the perch, with the toes partially closed.

Diagnosis

The clinical picture is very suggestive of this disease, but confirmation can be obtained by demonstrating elevated uric acid and evidence of nephromegaly

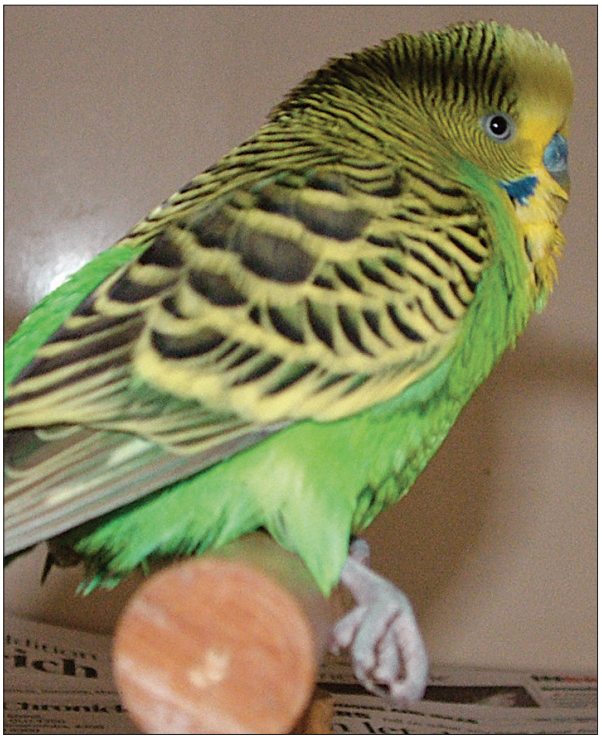


Figure 22.3 **Budgerigar with paresis due to renal neoplasia.**

Downloaded From Vet-eBooks.com

(by either radiology or ultrasound). Contrast radiology may be needed to differentiate renal from gonadal neoplasia.

Endoscopic renal biopsy can be used to identify the type of renal tumour present which may, with more research, indicate the best treatment and likely prognosis.

Management

Chemotherapy (carboplatin) has been tried, but with minimal success at this stage. Prognosis is poor – once a budgerigar starts displaying obvious clinical signs of leg paresis, death usually occurs in 2–8 weeks.

Horner's syndrome

Overview

Horner's syndrome is a loss of sympathetic innervation of the eye, resulting in ptosis and occasionally miosis. The cause is usually trauma, causing interruption of the sympathetic pathway from the cranial cervical spine or the notarial vertebrae. It is rarely reported in birds (only two cases have been documented at the time of writing), and information is therefore lacking on treatment and prognosis. However, the prognosis appears to be favourable.

Obturator paralysis

Clinical presentation

A form of 'obturator paralysis' is sometimes seen in egg-laying hens. After laying an unusually large egg, or after a difficult oviposition, the hen is paretic or paralysed in the legs (**Fig. 22.4**). This is due to compression of the pelvic plexuses causing neurapraxia.



Figure 22.4 **Egg-laying Alexandrine parrot hen with 'obturator' paralysis.**

Treatment

Rest, NSAIDs and calcium supplementation usually result in recovery, although this may take several weeks in severe cases.

Neuralgia

Overview

Foot and toe mutilation is a common presentation of psittacine patients of all species. It is perhaps more frequently observed in cockatoos, although an 'Amazon Foot Mutilation' syndrome has been reported. It has been proposed that this self-mutilation may be the result of neuralgia – pain that follows the path of a specific nerve.

Aetiology

The causes of neuralgia are varied. Chemical irritation, inflammation, trauma (including surgery), compression of nerves by nearby structures (e.g. tumours), and infections may all lead to neuralgia. Diabetes mellitus is another common cause of neuralgia in people. (Diabetes damages the arterial supply to the nerves, resulting in nerve fibre malfunction and sometimes nerve loss.) In many cases, however, the cause is unknown.

Clinical signs

Affected patients present with clinical signs of intense irritation of the lower extremities – foot shaking, shifting lameness, feather damaging behaviour on the lower leg, and self-mutilation (up to, and including, chewing toes off) (see **Fig. 14.10**, Chapter 14, Disorders of the Legs, Feet and Toes, p. 218).

Diagnosis

There are no specific tests for neuralgia, and the diagnosis is usually one of exclusion. Haematology, biochemistry profiles, and skin/toe biopsies are used to rule out other conditions. Nerve conduction studies with electromyography, examining the electrical activity of nerves, may confirm the diagnosis.

Treatment

Non-steroidal anti-inflammatory drugs, such as meloxicam, may be helpful in mild cases.

Downloaded From Vet-eBooks.com

Opioid analgesics may be needed for a short time to control severe pain. However, these analgesics often have disappointing results. Other treatments may include nerve blocks or surgical ablation of the affected nerve using different methods, such as local radiofrequency, heat, balloon compression, and injection of chemicals.

Antiseizure medications such as gabapentin have been found to be helpful for pain associated with neuralgia, sometimes combined with analgesics.

FURTHER READING

Antinoff N (2007) Stop the shakes! Diagnosing and treating neurological disorders in birds. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 201–215.

Delk K. (2012) Clinical management of seizures in avian patients. *Journal of Exotic Pet Medicine* **21**(**2**):132–139.

Gancz AY, Malka S, Sandmeyer L, Cannon M, Smith D, Taylor M (2005) Horner's syndrome in a red-bellied parrot (*Poicephalus rufiventris*). *Journal of Avian Medicine and Surgery* **19**(**1**):30–34.

Langohr IM, Garner MM, Kiupel M (2012) Somatotroph pituitary tumors in budgerigars (*Melopsittacus undulatus*). *Veterinary Pathology* **49**(**3**):503–7.

Platt SR (2006) Evaluating and treating the nervous system. In: *Clinical Avian Medicine*, Vol 2. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 493–518.

Rosenthal K, Orosz S, Dorrestein GM (1997) Nervous system. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds) WB Saunders, Philadelphia, pp. 454–474.

Simova-Curd S, Nitzl D, Mayer J, Hatt JM (2006) Clinical approach to renal neoplasia in budgerigars (*Melopsittacus undulatus*). *Journal of Small Animal Practice*; **47**(**9**):504–511.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

DISORDERS OF THE REPRODUCTIVE TRACT

THE MALE REPRODUCTIVE TRACT

Congenital

Abnormalities include abnormally shaped testes, fusion of the cranial poles, hypoplasia and agenesis. Usually there are no clinical signs, although aviary birds may be presented for infertility investigation. Diagnosis is by endoscopy, and no treatment is required.

Non-inflammatory

Degeneration of the testes is associated with various drugs and toxins (e.g. furazolidone, copper fungicides and mercury). It is diagnosed by endoscopic biopsy or needle aspiration cytology.

Atrophy may occur as the end stage of degenerative changes.

Orchitis

Aetiology

Orchitis may be bacterial (e.g. *E. coli*, *Salmonella* spp., *Pasteurella* spp.) (**Fig. 23.1**), fungal (extension from fungal air saculitis), mycobacterial (extension from systemic disease) or chlamydial (extension from systemic disease) in origin. Infection occurs by haematogenous spread or via an ascending infection from the cloaca/phallus.

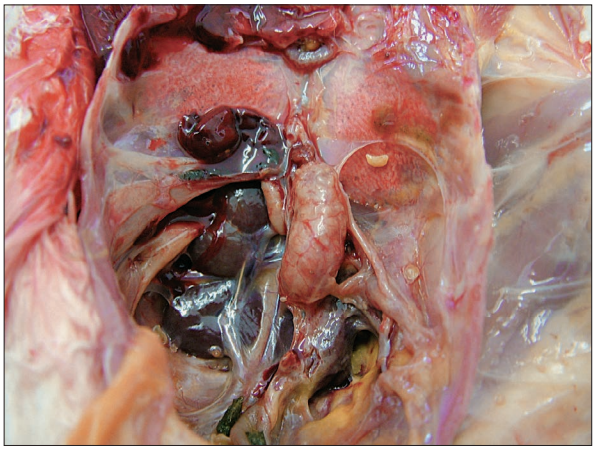


Figure 23.1 **Necropsy of a pigeon reveals orchitis due to *Salmonella* infection.**

Clinical presentation

No clinical signs are seen unless the bird is septicaemic. The presenting complaint may be infertility or, if a flock is infected, there may have been deaths. In the case of salmonellosis, there may be other clinical signs, such as polyarthritis, unrelated to orchitis.

Diagnosis

Haematology may reflect an inflammatory process. Diagnosis is by aspiration cytology or endoscopic biopsy.

Management

Antimicrobial therapy is given as indicated. Surgical orchidectomy may be required.

Neoplasia

Definition/overview

Neoplasia of the male reproductive tract is reported in numerous species. Tumours seen include Sertoli cell tumours (most common), interstitial cell tumours, seminomas, teratomas, lymphosarcoma, teratoma and leiomyosarcoma and carcinoma of the ductus deferens and epididymis.

Clinical presentation

Clinical signs include chronic weight loss, coelomic distension, unilateral paresis of the leg (due to sciatic nerve compression) and cere colour change in budgerigars (blue to brown). (Note: This colour change is not pathognomonic for Sertoli cell tumours.)

Downloaded From Vet-eBooks.com

Diagnosis

Radiology may demonstrate an enlarged testicle and, if a Sertoli cell tumour is present, increased radio-opacity of the bones (polyostotic hyperostosis, in response to hyperoestrogenism) may be seen. (see **Fig. 5.17**, Chapter 5, Diagnostic Imaging, p. 103) On endoscopic examination the testicles may appear cystic and can be confused with an immature/inactive ovary. Diagnosis is by endoscopic biopsy.

Management

Sertoli cell tumours may respond, in the short term, to GnRH agonists such as deslorelin implants or leuprolide injections. Otherwise orchidectomy is indicated. There is a good prognosis if there is no metastatic disease.

Phallic prolapse

Aetiology

Phallic prolapse is seen in ratites and waterfowl and is associated with sexual overwork. Inflammatory changes at the base of the phallus, occasionally associated with chronic bacterial infection, can maintain the prolapse even though the bird is sexually inactive.

Clinical presentation

The prolapsed phallus protrudes from the vent. The end of the phallus may become traumatised and/or necrotic from chronic exposure and contact with the ground.

Management

Sexual rest is required. The bird should be isolated from the hens and daylight hours reduced. A change in diet may be needed and one may consider GnRH agonists such as leuprolide acetate, human chorionic gonadotropin (HCG) and deslorelin. Anti-inflammatory treatment (e.g. meloxicam) and broad-spectrum antibiotics are indicated.

If the phallus is damaged, one may have to consider reducing the prolapse and placing lateral vent sutures to keep the phallus in the cloaca until healed. Phallic amputation can be considered, usually as a last resort.

Aggression

Overview

Aggression in male birds is predominately associated with captive-bred cockatoos, but has been seen in macaws, Amazons, and conures. Aggression may be directed towards a mate, neighbouring birds, or a human companion. It may be triggered by territorial behaviour or sexual frustration. Owners are often surprised by this behaviour, whether it is directed at another bird or at people.

Clinical signs

In the case of territorial aggression intrusion into what the bird perceives as its territory gives rise to early warning signs displayed by the bird. These include outspread wings, increased vocalisations, pupil dilation and constriction ('pinning') and fanning of the tail. If these signs are ignored, an attack may follow.

Aggression related to sexual frustration generally occurs early in the breeding season. Lack of reproductive synchrony between even closely bonded pairs leads to the male's sexual advances being ignored by the female. In the wild the hen simply flies away but this option is rarely present in a captive situation. The cock bird attacks the hen, inflicting physical trauma ranging from feather chewing on the face through to beak avulsion or limb fractures. On occasion, the aggression only ends with the death of the hen or the separation of the pair.

Treatment

Territorial aggression in an aviary situation can often be treated by screening the aviary so as to minimise interaction between neighbouring birds. Where possible, owners should avoid entering the aviary, feeding and watering the birds through a small door. Nest boxes can be designed and positioned so that they can be accessed from outside the aviary for inspection. (A safety walkway must be utilised, as some birds will chew through the nest box and escape.)

In a companion bird aggression can be minimised or eliminated by altering the bird's environment (social and physical) to reduce or eliminate reproductive triggers that drive this behaviour. If this is not sufficient, GnRH agonists (e.g. deslorelin and leuprolide) can be utilised to 'shut down' the bird's reproductive drive. (See Chronic Egg Laying p. 326, for a more detailed description of this process.)

Downloaded From Vet-eBooks.com

Sexual frustration can be more difficult to manage, particularly if reproductive success is the owner's main goal in keeping the birds. In many cases the male's aggression can be re-directed towards nest preparation rather than the hen. This can be achieved by:

1. Closing over the nest entrance with cardboard or thin plywood, leaving a small entrance hole. The cock bird will 'work' this entrance hole to make the nesting site accessible and more suitable.
2. Filling the nest box with small blocks of soft untreated wood; the cock bird will chew these into shavings, again to prepare the nest site.

Hopefully, by the time the cock has prepared the nest box, the hen will have synchronised her reproductive drive with him and will be more receptive to his courtship.

Other avenues include the use of large aviaries (giving the hen the opportunity to escape) or special two-entrance boxes with baffled interiors that allow the hen to avoid been trapped by the cock in the next box.

In some cases, trimming the cock's wings to reduce his ability to fly and chase the hen may be necessary. This can be augmented by elevated escape areas for the flighted hen that are inaccessible to the now-clipped cock.

Temporary pair separation is always necessary once trauma has been inflicted on the hen; this may break the aggression cycle and give the pair time to synchronise their reproductive drive. In these cases, pairs may be slowly and cautiously reintroduced. If the aggression continues, there is little choice other than permanent separation of the pair.

Aggressive beak alteration procedures have been recommended in the past, but are not considered to be ethical by the majority of veterinarians; these included attaching acrylic or rubber balls to the tip of the maxilla (offering limited temporary protection) and surgically splitting the mandible at the symphysis. Birds with split mandibles have numerous complications, including difficulties eating, drinking, playing and mating.

THE FEMALE REPRODUCTIVE TRACT

THE OVARY

Congenital abnormalities

Congenital abnormalities include congenital atresia, persistent right ovary and congenital ovarian cysts.

All early embryos have bilateral ovaries and oviducts but, in most birds, the growth of the right ovary and oviduct is inhibited after the first trimester of incubation. It is normal for kiwis and many raptors to have two ovaries. (Kiwis have two ovaries and one oviduct; raptors have two ovaries and two oviducts.) Persistent right ovaries and oviducts are occasionally seen in species (such as parrots) that would normally have only a left ovary and oviduct.

Congenital ovarian cysts are common in budgerigars and canaries.

Oophoritis

Aetiology

Infectious oophoritis can result from either haematogenous spread of a bacterial (e.g. *Salmonella* spp., mycobacteria), fungal or viral infection (e.g. herpesvirus), or spread from adjacent air saculitis or peritonitis. Non-infectious oophoritis may be caused by rupture of follicles and extrusion of yolk into the ovarian stroma, or by the effect of some toxins (e.g. aflatoxicosis).

Clinical presentation

Infectious oophoritis is often part of a systemic illness and many birds will present for generalised weakness, lethargy and weight loss. In milder cases there may be an infertility problem or increased embryonic deaths.

Diagnosis

Haematology will usually demonstrate a leucocytosis. Free coelomic fluid may be detected by ultrasound and should be submitted for cytology and culture. Endoscopy may reveal abnormally coloured or shaped ovary, often haemorrhagic and with abnormal follicles. The ovary of birds with non-infectious oophoritis may appear enlarged, with multifocal yellow spots of varying sizes, or obvious scarring on the ovarian parenchyma.

Downloaded From Vet-eBooks.com

Treatment

Antimicrobial therapy is given as indicated. If possible, the abscessed follicle(s) should be drained completely, being careful not to contaminate the coelom. Partial or complete ovariectomy may be required for chronically infected and caseated follicles.

Ovarian cysts

Aetiology

Ovarian cysts may be congenital (see above) or acquired i.e. secondary to neoplasia or oophoritis (**Fig. 23.2**). They are rarely functional i.e. secreting hormones.

Clinical presentation

If the cysts are small, there may be no clinical signs. Large cysts (**Fig. 23.3**) may cause coelomic enlargement and dyspnoea (through compression of the air sacs). Some birds will strain, occasionally resulting in a cloacal prolapse.

Diagnosis

This is based on ultrasonography, endoscopy and exploratory coeliotomy. Ultrasound is particularly useful in distinguishing free fluid (as in yolk peritonitis or ascites) from the 'contained' fluid within a cyst or cysts. Aspiration of the cyst usually gives clear, straw-coloured fluid with low cellularity.



Figure 23.2 **Necropsy of a cockatoo hen finds ovarian cysts secondary to an ovarian neoplasia.**



Figure 23.3 **Necropsy of a budgerigar reveals a large ovarian cyst.**

Management

Aspiration of the cyst may be carried out by ultrasound-guided transcoelomic aspiration, endoscopic aspiration or surgical aspiration.

Ovariectomy (partial or complete) is difficult to achieve successfully because of the complex vascularity of the ovary. It should not be attempted without good magnification (e.g. an operating microscope).

Hormonal therapy (e.g. deslorelin, leuprolide acetate, HCG) has been suggested to reduce or resolve ovarian cysts in birds and offer a non-invasive treatment option. This, however, assumes that the cysts are functional and are not secondary to other disease processes (neoplasia, oophoritis). However, these GnRH agonists may play a role in reducing ovarian activity and the formation of new cysts.

As long-term resolution of this condition is difficult to achieve, it is important to regularly monitor patients for recurrence.

Neoplasia

Definition/overview

Tumours of the ovaries include granulosa cell tumours, ovarian carcinomas, carcinomatosis, dysgerminoma, arrhenoblastoma and teratoma.

Clinical presentation

If the tumour is small, there may be no clinical signs. Large tumours may cause coelomic enlargement

Downloaded From Vet-eBooks.com

and dyspnoea (through compression of the air sacs). There may be egg retention and oviductal impaction.

Granulosa cell tumours (and possibly other reproductive tract tumours) may be functional and cause increased plasma hormone levels. This can result in hormonal changes (e.g. polyostotic [medullary] hyperostosis).

Diagnosis

Diagnostic imaging (ultrasonography and radiography) helps to identify the presence of a tumour. Confirmation is by endoscopic or surgical biopsy.

Management

Surgical debulking or complete ovariectomy are an appropriate therapy but technically very challenging, and come with a high likelihood of recurrence if complete removal is not achieved. Adjuvant chemotherapy may assist in controlling this recurrence. The use of GnRH agonists such as deslorelin or leuprolide has been suggested and shows promise in controlling some ovarian neoplasias. The prognosis, however, remains guarded.

THE OVIDUCT

Congenital abnormalities

Congenital abnormalities include atresia, segmental aplasia, congenital cysts and persistent right oviducts. When present, a persistent right oviduct is rarely complete. It originates distally from the cloaca along the right caudal dorsolateral coelomic wall, then extends cranially for a variable distance along the ventral surface of the right kidney lateral to the right ureter. It may develop cysts, endometritis or hyperplasia. The bird is usually presented for infertility investigation, yolk-related peritonitis or salpingitis.

Cystic hyperplasia

Grossly visible cysts occur containing clear or cloudy fluid. They are probably endocrinal in origin, but this is not determined as yet. Birds are usually presented for infertility investigation, yolk-related peritonitis or salpingitis.

Neoplasia

Adenomas, adenocarcinomas (**Fig. 23.4**) and leiomyomas have been reported. Birds are usually presented for coelomic distension, yolk-related peritonitis or salpingitis.

Salpingitis and metritis

Definition/overview

Salpingitis is inflammation of the oviduct and mesosalpinx and metritis is inflammation of the uterus (or shell gland).

Aetiology

These are relatively common conditions in parrots and backyard poultry. Predisposing factors include age, malnutrition, excessive coelomic fat, excessive egg laying, egg-binding and other reproductive disorders.

Primary infections are uncommon in domestic situations; they may be caused by Newcastle disease virus or infectious bronchitis virus.

Secondary infection may follow yolk retention or prolonged/excessive egg laying. Haematogenous or ascending infections may occur. Bacterial species involved include *E. coli*, *Klebsiella* spp. and *Pseudomonas* spp.

Clinical presentation

Affected birds often have a history of extremely good egg production. They are often on a predominantly

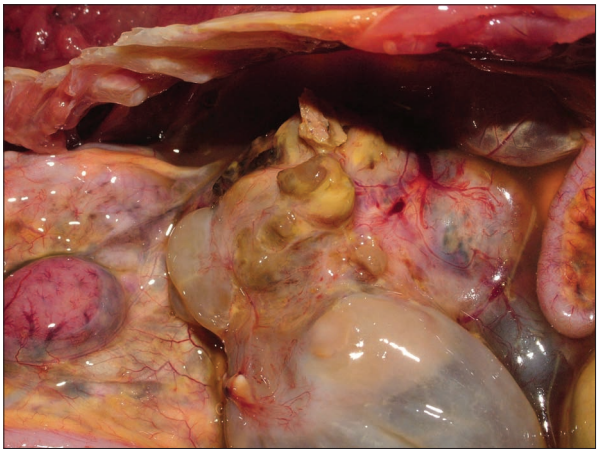


Figure 23.4 **Necropsy of a cockatoo finds a uterine adenocarcinoma.**

Downloaded From Vet-eBooks.com

seed diet with inadequate vitamin and mineral supplementation. There may be a history of infertility or embryonic or neonatal mortality.

Clinical signs include weight loss, ruffled plumage, anorexia and lethargy. If the birds are still laying eggs, these eggs may be malformed (e.g. soft–shelled (**Fig. 23.5**); have stress lines (**Fig. 23.6**); be an abnormal shape) or have streaking of blood on the shell. There may be chronic egg-binding, infertility, a distended coelom, a flaccid vent and cloacal discharge.

Diagnosis

There may be a leucocytosis, either heterophilic or monocytic, depending on the chronicity of the problem. Clinical biochemistries may show a hypercalcaemia (if the bird is still reproductively active) and a hyperamylasaemia (if there is concurrent pancreatic disease associated with a yolk peritonitis).

Radiography may reveal retained eggs (see **Fig. 5.19**, Chapter 5, Diagnostic Imaging, p. 104), an enlarged oviduct or the presence of coelomic fluid. Ultrasonography can distinguish between fluid enlargement and organ enlargement, and may reveal retained eggs or fluid in the oviduct. Endoscopy (if there is not free fluid in the coelom) can demonstrate a swollen and inflamed oviduct. If coelomic fluid is present, coeliocentesis can help to differentiate the inciting causes.



Figure 23.5 **Soft-shelled egg from a Cockatiel.**

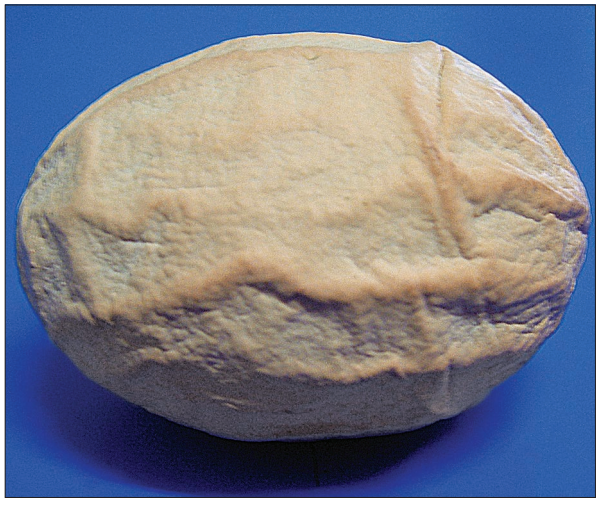


Figure 23.6 **Egg from a chicken with metritis shows visible stress lines.**

Management

Conservative treatment may be attempted, but is usually unsuccessful. Recommendations include reproductive rest through environmental, nutritional and hormonal manipulation, NSAIDs (e.g. meloxicam) and antibiotics. If there is material in the oviduct (e.g. caseous pus, eggs or fluid), the use of prostaglandins may be indicated. Using prostaglandin E2 (PGE2) to relax the uterovaginal sphincter and stimulate oviductal contractility may assist in this regard. Caution must be exercised with this therapy, as chronic cases may have developed adhesions to the oviductal wall and strong contractions may lead to rupture of the oviduct.

In valuable breeding birds an attempt to combine the above therapy with a coeliotomy and retrograde flushing of the oviduct can be made. Definitive treatment may require salpingohysterectomy (see Chapter 29, Surgery, p. 397).

In all probability, a diagnosis of salpingitis or metritis indicates that the bird's reproductive life is almost certainly complete. If the patient is a breeder or egg layer, this must be communicated to the owner before commencing any treatment. Conservative treatment may give some short-term success, but the problem often recurs. Salpingohysterectomy often gives good long-term results, although some birds will continue to ovulate and develop yolk-related peritonitis (see below). These birds require environmental, nutritional and hormonal manipulation to minimise ovulation.

Downloaded From Vet-eBooks.com

Yolk-related peritonitis

Aetiology

Yolk-related peritonitis is the result of an intense inflammatory reaction to ectopic yolk and ova within the coelom. This ectopic ovulation is caused either by a failure of the infundibulum to 'capture' ovulating yolk (because of fat, trauma or disease), or by retropulsion of yolk from the oviduct into the coelom, possibly associated with metritis/salpingitis, oviductal cystic hyperplasia or oviductal impaction, or with exuberant reverse peristalsis.

Clinical presentation

The condition is usually seen in high-producing hens, especially cockatiels. It is usually sterile; if septic, signs are consistent with severe septicaemia.

Clinical signs are related to a fluid-producing inflammatory reaction in the coelom: dyspnoea, coelomic distension (**Fig. 23.7**) and weakness. The bird may stop laying or may lay malformed eggs (often elongated).

Secondary diseases may develop as a result of yolk-related peritonitis including pancreatic disease (including diabetes mellitus), hepatitis, nephritis, splenitis and coelomic adhesions.

Diagnosis

Haematology generally demonstrates a marked leucocytosis. Hypercalcaemia and lipaemia may be seen in reproductively active hens.



Figure 23.7 **Scarlet-Chested parrot with yolk-related peritonitis has coelomic distension due to yolk and fluid.**

Coelomic ultrasonography confirms fluid distension of the coelom rather than organomegaly. Free fluid must be distinguished from ovarian cysts. Caseous material (inspissated yolks) may be detected. Coeliocentesis reveals variably coloured fluid (brown to yellow-pink) and cytology shows mesothelial cells, leucocytes and pink yolk globules.

Management

Short-term therapy includes coelomic drainage, NSAIDs, antibiotics and hormonal manipulation to stop ovarian activity (leuprolide acetate or deslorelin).

Most cases will require surgery to lavage the coelom and perform salpingohysterectomy (see Chapter 29, Surgery). Inspissated yolks should be removed and adhesions broken down if possible. Persistent ovulation post-surgery may occur and incite another bout of peritonitis so the concurrent use of GnRH agonists is often recommended. Ovariectomy may be feasible, but it is fraught with danger due to the nature of the blood supply.

Egg binding (dystocia)

Overview

Egg binding is a slowed passage of the egg through the oviduct. If it is located in the uterine portion it will have a shell deposited on it; proximal to this level it will be encased in shell membranes only. It must be distinguished from egg retention (see below).

The lodged egg may compress the local vessels and nerves, causing lameness, paresis, paralysis, and poor organ perfusion. Pressure necrosis of the oviduct wall can lead to oviductal rupture, and obstruction of the rectum and ureters can cause metabolic disturbances.

Aetiology

Predisposing factors include age (very young and very old birds are more frequently affected), malnutrition and obesity (it is particularly seen in birds on all-seed diets), excessive egg production, especially in cockatiels, budgerigars and backyard poultry, and lack of physical fitness in caged birds.

Causes include oviductal muscle dysfunction (due to a calcium deficiency, myositis due to excessive egg production, or scarring from previous dystocia

Downloaded From Vet-eBooks.com

episodes), concurrent salpingitis or metritis; excessively sized or malformed eggs; and systemic problems (concurrent illness, hypothermia, environmental stress).

Clinical presentation

Signs include depression, excessive straining, persistent tail wagging, a 'penguin-like' wide stance, leg weakness, dyspnoea, collapse and coelomic distension (**Fig. 23.8**). Death may occur suddenly.

Diagnosis

This is based on the history of egg laying and the clinical signs (see above). Coelomic palpation usually reveals an egg, but soft-shelled eggs can be difficult to detect. Coelomic radiography (see **Fig. 5.18**, Chapter 5, Diagnostic Imaging, p. 104) or ultrasonography may be required.

Treatment

If the bird shows no or only mild to moderate signs of discomfort and distress, confirm the time the last egg was laid; eggs are usually laid 23–26 hours apart and the patient may not be ready to lay.



Figure 23.8 **Sun conure with coelomic distension due to egg binding.**

The bird should be placed in a heated hospital cage with adequate humidity and given calcium gluconate by intramuscular injections every 3–6 hours. Oxygen may be required if the bird is dyspnoeic. Tube feeding highly digestible, high-sugar supplements may be given to provide a rapid source of energy. Stress and handling should be minimised, and the bird should be kept in a dark, quiet environment.

If the bird fails to respond to this treatment, oxytocin may be given, but there is controversy over its efficacy in birds, as it is not an avian hormone. (The avian equivalent is arginine vasotocin, not available commercially.) Intra-cloacal PGE2 gel (**Figs 23.9a** and **b**) will usually produce uterovaginal sphincter dilation and straining within 5–10 minutes (**Fig. 23.9c**). PGF2α should be avoided, as it will induce oviductal contractions without relaxing the uterovaginal sphincter. If necessary, PGE2 administration will allow manual manipulation of the egg into the cloaca and its delivery. Caution must be taken not to push the egg up against the kidneys and spine.

If the bird is distressed or dyspnoeic, an emergency situation exists and ovocentesis and egg collapse may be necessary. A large gauge needle is introduced into the egg through the cloaca (preferably) or the coelomic wall and the contents are aspirated, while the egg is simultaneously collapsed with digital pressure. The egg shell is usually passed within 48 hours of this procedure. It must be noted that while this procedure may be life-saving, damage to the oviduct usually requires a salpingohysterectomy at a later date.

In some cases it may be necessary to anaesthetise the bird (mask induction with isoflurane), intubate it and apply intermittent positive pressure ventilation (IPPV) while the egg is being manipulated through the cloaca. Coeliotomy and caesarean section may be necessary in some cases (**Figs 23.10** and **23.11**).

Prognosis

The earlier the case is presented, the better the prognosis. Simple cases have an excellent prognosis, while cases that have reached the stage where the bird is collapsed, dyspnoeic and unable to use its legs properly have a guarded prognosis.

Downloaded From Vet-eBooks.com

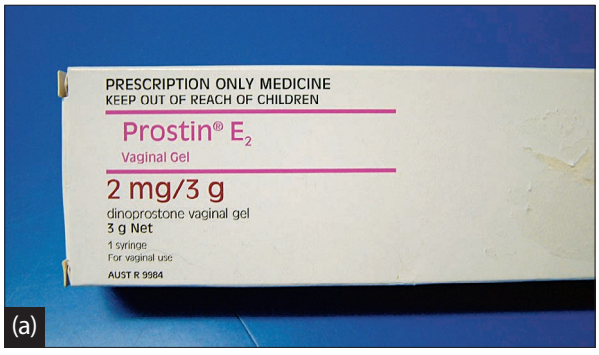






Figure 23.9 Prostaglandin E2, available as Prostin®E_2 vaginal gel (a) is the preferred treatment for egg binding in birds. It is administered directly into the cloaca using a syringe (b) and absorbed rapidly across the cloacal mucosa. Treatment is rapid and effective; two soft-shelled eggs were laid almost simultaneously by this sun conure following prostaglandin E2 treatment.



Figure 23.10 Princess parrot with coelomic distension due to a retained egg.



Figure 23.11 Princess parrot undergoing a caesarean section required to deliver an egg. This procedure should be considered a last resort.

Ectopic eggs

Definition/overview

These cases present clinically very similar to egg-bound birds, with the same aetiology and clinical signs. The hen fails to pass the egg, regardless of the treatment given. This is due to rupture of the oviduct at the level of the shell gland, leaving a fully shelled egg loose in the coelom.

Management

Treatment requires a coeliotomy to remove the egg and repair the oviduct. Prognosis is good and some of these birds return to egg laying uneventfully,

Downloaded From Vet-eBooks.com

provided they are given several months without egg-laying to recuperate.

Retained eggs

Definition/overview

The egg may be retained in the oviduct, but it may have collapsed, leaving only the shell. Others may have the egg still intact, but are not straining to pass it. The egg is often located in the anterior coelom and usually does not cause dyspnoea.

Clinical presentation

These birds may present with coelomic distension (**Fig. 23.10**) or they may be asymptomatic.

Diagnosis

Diagnosis is made radiographically. Eggs may have an excessively thickened shell, or it may have collapsed within the oviduct (see **Fig. 5.19**, Chapter 5, Diagnostic Imaging, p. 104).

Treatment

Conservative treatment (as described for egg-binding) may work in some cases but should not be relied upon. Surgical therapy (caesarean section or salpingohysterectomy) is usually required.

Chronic or excessive egg laying

Definition/overview

This is most commonly seen in cockatiels (**Fig. 23.12**), but any species can be affected. Cockatiels in the wild or in the aviary will usually lay 2–3 clutches, each of 3–5 eggs, over a 12-month period. There is usually a period of several months between clutches. As companion birds, pet cockatiels will often lay this many eggs in a single month, and do it month after month. Left untreated, many of these birds deplete their calcium reserves and develop problems such as egg binding and pathological fractures. Many will develop salpingitis/metritis and subsequent yolk-related peritonitis.

Aetiology

Cockatiels evolved in an arid environment and are consequently opportunistic breeders, laying eggs when environmental conditions are ideal for the survival of their offspring. In captivity these environmental conditions are provided all year round – large amounts of readily available food and water, especially high-fat and sweet foods (e.g. seed and fruit), constant light (these birds are often housed indoors and do not have an appropriate diurnal rhythm), an 'appropriate' mate (this may not be another bird, in many cases it is the owner who has allowed an 'unnatural' bond to develop between themselves and the bird) and a secure nest site, which may be anywhere in the house or cage where the bird has established territoriality. The result is a constant drive to lay eggs.

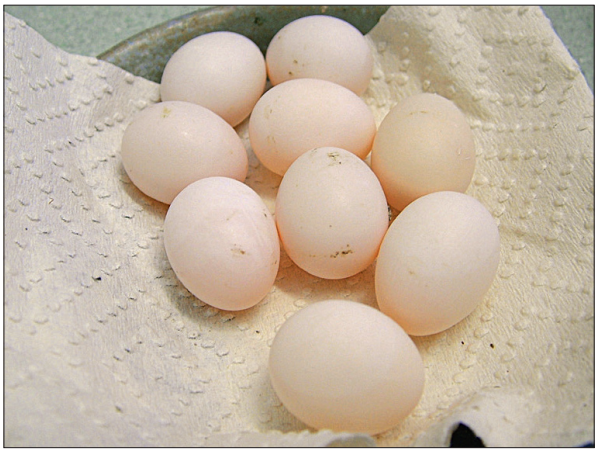


Figure 23.12 **These nine eggs were laid by one cockatiel hen in one month.**

This is often then exacerbated by the owner removing the eggs as they are laid. Cockatiels are indeterminate layers and will simply lay another egg to replace the missing one.

Diagnosis

The history of excessive egg laying makes diagnosis relatively straightforward. The bird's physiological and health status should be evaluated by a physical examination, haematology and biochemistry (including ionised calcium), and diagnostic imaging.

Management

Management of chronic egg laying requires identification and elimination of the environmental, dietary and social factors triggering the bird's reproductive drive and then, if necessary, using hormonal

Downloaded From Vet-eBooks.com

manipulation or surgery to minimise or prevent further egg laying. It should be noted that complete cessation of egg laying is difficult to achieve but, if the bird can physiologically handle the number of eggs laid each year, treatment can be said to have been successful.

Environmental modification: Normal diurnal rhythms need to be re-established. In some cases it may be necessary to drastically reduce daylight hours artificially (to eight hours or less) until the egg laying behaviour has stopped, and then bring the bird up to normal rhythms. Note that in species where daylength has little effect on breeding (e.g. zebra finches and budgerigars), changing daylength will have little effect on reproductive behaviour.

Nest sites should be removed (if possible). If the bird chooses an unconventional nest site (e.g. on the floor of the cage), re-arranging the area may effectively remove its attractiveness as a nest site. The bird's environment can be changed (e.g. new or re-arranged cage furniture, re-positioning the cage on an irregular basis). This induces a certain amount of environmental stress and reduces territoriality.

Social modification: If appropriate (and possible), the companion bird can be removed. A normal relationship between human and bird should be established through the introduction of basic behavioural training and client education. The client must be taught what behaviours sexually stimulate the bird (e.g. stroking the bird's back, kissing the bird or allowing it to take food from the owner's mouth, etc.).

Removing the eggs of an indeterminate layer, such as a cockatiel, will induce further egg laying. Leaving the eggs in the 'nest' or replacing them with artificial eggs will often result in a hen laying a normal sized clutch and then brooding the eggs. This shuts down (temporarily) the production of eggs.

Nutritional modification: Fat and sugar in the diet should be reduced by conversion to a formulated diet and removal of fruit from the diet. Foraging behaviour can be introduced. Typically, wild birds spend 80% of their day foraging for food, leaving 20% of the day to groom, socialise and 'nap'. In captivity, this time allocation is nearly reversed, with birds only having to spend a small amount of time looking for food. By introducing foraging activities, there is less time for pair bonding and other reproductive activities.

Hormonal manipulation: Hormonal therapy does not work in isolation. Unless the modifications in environment, behaviour and diet are instituted beforehand or concurrently, there will be minimal or no response to the use of hormones.

Drugs that have been used include:

- Leuprolide acetate, 100–700 µg/kg every 2–4 weeks.
- Deslorelin, a GnRH agonist available as an implant for dogs, is commonly used in birds
- HCG, 500–1,000 IU/kg every 2–4 weeks. Antibodies to HCG develop quickly, limiting its efficacy. It has been suggested that dexamethasone should be given concurrently to suppress the production of these antibodies, but this raises the spectre of generalised immunosuppression.
- Medroxyprogesterone acetate, 5–25 mg/kg every six weeks. Side-effects (diabetes, obesity and hepatopathies) limit its use.

Surgery: Salpingohysterectomy has been proposed as a means of controlling egg production. Unfortunately it appears that, by itself, it does not stop reproductive behaviour and subsequent ovulation, which then results in yolk peritonitis. Surgery should therefore be reserved as a 'last resort' therapy only.

INVESTIGATING REPRODUCTION PROBLEMS IN THE AVIARY

Introduction

Veterinarians are occasionally asked to investigate an apparent fertility problem in an avicultural collection. The problem is generally one of three types: lack of egg production, infertile eggs or poor hatchability.

Any investigation should start with a comprehensive review of the aviculturist's records including: the aviculturist's objectives and intentions; aviary design and construction; origin, age and species of birds; husbandry practices, traffic flow and nutrition;

Downloaded From Vet-eBooks.com

previous medical histories; production records; and description of the current problem.

In many cases it is appropriate to do this review before visiting the avicultural complex, utilizing written records, a map drawn by the aviculturist of the complex's layout and traffic flow, and photographs.

Lack of egg production

One should determine if the problem is generalised or is limited to a particular pair, a particular species or a particular part of the aviary.

Generalised problem

The following factors should be reviewed:

- Diet. The provision of high-fat foods at the start of the breeding season can stimulate reproductive activity. If these foods are fed all year round, that stimulus can be lost. Alternatively, the provision of a low-fat formulated diet will also often fail to stimulate activity. Feeding a formulated diet as the basis for the aviary nutrition and then using seed and nuts to stimulate reproductive behaviour when desired can be a powerful tool in reproductive management.
- Aviary design should reflect the height and space requirements for the species housed there.
- Privacy provision and stress reduction. Some birds will not feel secure enough to breed if they cannot access 'private' areas in the aviary where they can indulge in courtship and mating behaviours without feeling threatened. Stress from the surrounding environment (e.g. birds of prey, dogs, children, construction activities) must also be minimised.
- Species compatibility. Some birds will not breed if they can see or hear other birds of the same or different species during the breeding season.
- Local weather patterns and if 'artificial weather' in the form of artificial lighting or water sprinkling systems are being used to stimulate reproductive behaviour.

Representative birds should be examined for general body condition and health. Diagnostic tests should be aimed at screening for flock problems (e.g. chlamydiosis, parasites).

Problem is limited to one pair of birds

The veterinarian should determine:

- If they are a true pair. Sexually dimorphic species may require DNA or surgical sexing.
- If they are old enough to breed.
- If they are a compatible pair.
- If the aviary design, nest boxes and perches meet the birds' requirements for privacy, minimal stress, security and natural behaviours.

The health of the birds should be assessed.

Problem is limited to one species of birds

All the factors discussed above should be assessed. The known requirements for captive breeding of that species should be reviewed.

Problem is limited to one part of the aviary

All the factors described above should be assessed, with particular emphasis on aviary design, external stresses and environmental conditions in that area.

Infertile eggs

The first step is to ascertain if eggs are infertile or are failing to develop. This requires several egg necropsies:

- Is the egg infertile or is the problem actually embryonic death?
- If the problem is embryonic death, at what stage of development did the embryo die?

Aetiology

It is not uncommon, with sexually monomorphic species, for two hens to be inadvertently paired together. DNA or surgical sexing should be used to confirm the sex of both birds, or to re-check previous sex determination. (Neither DNA or surgical sexing is not as accurate as some believe.)

A properly formed egg usually, but not always, implies that there is not a medical problem with the hen, but that the cock bird is either not producing spermatozoa or is producing abnormal spermatozoa. One exception is oophoritis or low grade metritis causing embryonic death.

Downloaded From Vet-eBooks.com

If the cock bird is producing healthy semen, it is not reaching the hen's infundibulum due to:

- Lack of successful mating: incompatible pairs, immature cock bird, inexperience on the part of either bird, inappropriate perches preventing normal mating (too small, too large, not fixed in place), or lack of privacy or external stresses (other birds, animals, humans) causing the birds to feel insecure.
- Physical impediments to successful mating (e.g. old leg injuries, excessively heavy feathering around the vent).
- Low-grade cloacitis or metritis inhibiting the passage of semen from the vent to the infundibulum.

Diagnosis

The author's preferred approach is to firstly determine that:

- The birds are, indeed, a true pair, through visual sexing, DNA sexing or surgical sexing.
- The birds are healthy, determined by physical examination, haematology, biochemistries, cloacal cultures and possibly radiography/ultrasonography.
- The cock bird is fertile, determined by semen collection (through cloacal massage) or endoscopic evaluation and biopsy/fine needle aspirate of the testicle.

If the birds' health and fertility can be shown to be normal, it is more likely that the aviculturist will accept that the problem lies with compatibility, immaturity, inexperience, husbandry or nutrition, and make critical evaluation of these areas more acceptable.

Embryonic death

Definition/overview

It is normal for up to 10% of all eggs laid to fail to develop normally. Normal embryonic mortality is as follows:

- First trimester: 3–4%.
- Second trimester: 1–2%.
- Third trimester: 4–5%.

Embryonic mortalities above these levels should be investigated, see *Table 23.1*.

Aetiology

Parental factors include genetics, nutrition (calcium deficiency, excessive vitamin supplementation, especially vitamin D3) and health (oophoritis, metritis).

Table 23.1 **Troubleshooting embryonic mortality**

PROBLEM	POSSIBLE CAUSES
Floating air cell	Rough handling of egg Parental nutritional deficiencies Genetics
Early embryonic mortality	Parental nutritional deficiencies Shaking/jarring of eggs Incubator problem: temperature, turning, ventilation Disinfection of eggs in first three days Formaldehyde fumigation of eggs Delayed egg collection Incorrect storage: temperature incorrect, stored too long
Mid-term embryonic mortality	Parental nutritional deficiencies Infection Inadequate turning Incubator problem: temperature, turning, ventilation Lethal genes
Late-term embryonic mortality	Malposition's Incubator problem: temperature, humidity, turning, ventilation Infection Parental nutritional deficiencies
Air cell pipped, fail to hatch	Hatcher problem: temperature, humidity, ventilation
Malformed chicks	Incubator temperature too high Parental nutritional deficiencies Genetics Teratogens
Oedematous chicks	Humidity too high Shell too thick Inadequate ventilation
Sticky chicks	Humidity too high Shell too thin
External yolk sac	Temperature too high Oedematous chick Inappropriate intervention to assist hatch

Downloaded From Vet-eBooks.com

Incubation factors may be important:

- Natural incubation: nest box conditions (hygiene, humidity, temperature extremes) and parental factors (abandoning the nest, rough handling of eggs).
- Artificial incubation: temperature too high or too low, humidity too high or too low, ventilation inadequate, turning inadequate or poor hygiene.

Diagnosis

An egg necropsy should be carried out.

- The egg is weighed and weight loss (as a percentage of weight when laid) during incubation determined. The normal is 12–17% over the period of normal incubation.
- Shell quality is assessed: surface texture, porosity and cleanliness.
- The egg is candled to identify the air cell, determine its size and mobility, and assess whether an embryo or infection is present. Hairline cracks in the shell can also be identified.
- The egg is placed vertically on to a support (e.g. a plastic ring or cup) with the air cell uppermost. The shell over the air cell is broken with a sharp implement, revealing the air cell. The shell is then removed down to the level of the inner shell membrane forming the base of the air cell. This membrane is then peeled away with fine forceps, revealing the egg contents.
- A swab (for culture of bacteria and fungi) can be taken from the albumen or inside the shell.
- If an embryo is not obviously present, the blastodisc on the yolk is examined. The yolk around this disc is less dense than the rest of the yolk, so it will float uppermost. In an infertile egg the disc will appear as a small white point. A very early embryo will appear as a small white 'doughnut' with a translucent centre. Any blood vessel development at all is confirmation of fertility.
- If an embryo is present, the stage of development and the positioning of the embryo (if it is late-term) are determined.
- Embryonic position is observed before removing the embryo from the egg. The normal late-term embryonic position is: head next to the air cell, right side uppermost and turned to the right, with the beak adjacent to the right foot and shoulder; legs flexed on either side of the body and ventral to the shoulders; and the spine following the long axis of the egg. Any other position is classified as a malposition and may account for embryonic death. Descriptions and possible causes of classical malpositions are described in *Table 23.2*.
- Once the embryo has been removed from the egg, it can be either grossly examined and then fixed in formalin, or necropsied and tissues submitted in formalin.

Table 23.2 **Embryonic malpositions**

MALPOSITION	DESCRIPTION	POSSIBLE CAUSES (IF KNOWN)
I	Head is down between the legs	High incubator temperature
II	Chick is rotated within the egg, with the head at the end opposite to the air cell	Egg position and low temperature during incubation
III	Head is rotated to the left, with the head under the left wing	Egg position, temperature and parental malnutrition
IV	Beak is away from the air cell, rest of the body is normally positioned	Egg position
V	Feet over the head	–
VI	Head over the right wing	Parental nutrition

Downloaded From Vet-eBooks.com

FURTHER READING

Bowles HL (2006) Evaluating and treating the reproductive tract. In: *Clinical Avian Medicine*, *Vol 2*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 519–540.

Joyner KL (1994) Theriogenology. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 748–804.

LaBonde J (2006) Avian reproductive and pediatric disorders. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 229–238.

Orosz S, Dorrestein GM, Speer BL (1997) Urogenital disorders. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 614–644.

Schubot RM, Clubb KJ, Clubb SL (1992) *Psittacine Aviculture: Perspectives, Techniques and Research*. Avicultural Breeding and Research Center, Loxahatchee, Fl.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 24

DISORDERS OF THE URINARY SYSTEM

RENAL DISEASE

Avian renal disease is a frequently diagnosed but often poorly understood problem in birds. It is associated with multiple diseases and has many causes. Understanding the mechanisms of renal disease, the possible aetiologies, and the effects on other parts of the body will assist the clinician with both its diagnosis and treatment.

Mechanism of renal disease

The anatomy and physiology of the kidneys is discussed in Chapter 1, Clinical Anatomy and Physiology.

The mechanisms of renal damage in birds have not been studied as much as in mammals, but there are sufficient similarities to extrapolate and understand how renal disease occurs. Any insult to the kidney (e.g. ischaemic; inflammatory or toxic) results in the release of prostaglandins and thromboxanes. This in turn results in changes in renal vascular resistance, blood flow, recruitment of inflammatory cells and other physiologic effects. Thromboxane, in particular, leads to renal vasoconstriction resulting in decreased glomerular filtration rate (GFR) and delivery of oxygen and nutrients to tubular cells, further adding to renal damage.

The flow-on effect of this renal damage is seen as conditions such as gout (discussed later in this chapter) and fluid loss leading to dehydration.

Renal disease may be classified broadly into two categories: inflammatory and non-inflammatory.

Inflammatory conditions

Nephritis (inflammation of the kidney) may involve the interstitial tissue, the tubules or the glomerulus (although 'glomerulonephritis' is typically reserved for glomerular lesions). The term pyelonephritis is technically incorrect in avian medicine as bird kidneys lack a renal pelvis. Causes of inflammatory renal disease may be infectious or non-infectious (**Fig. 24.1**).

Infectious causes include:

- Viruses: adenovirus; circovirus; coronavirus; herpesvirus; orthomyxovirus; polyomavirus; paramyxovirus; poxvirus and retrovirus.
- Bacteria: most bacterial species, including mycobacteria.
- Chlamydiosis.
- Parasites: coccidia (waterfowl and raptors); Cryptosporidia (rare in all species); microsporidia (lovebirds, budgerigars and finches); *Toxoplasma*, *Sarcocystis* (systemic infection), trematodes (waterfowl), and schistosomiasis (waterfowl).
- Fungal infections: *Aspergillus* infections invading either from neighbouring air sac lesions or by haematogenous spread after fungal invasion of blood vessels.

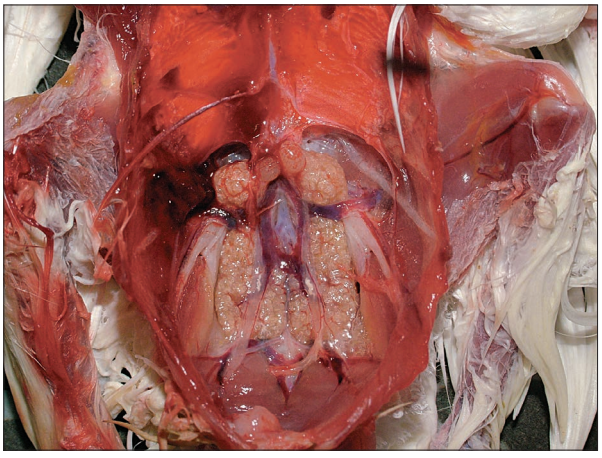


Figure 24.1 **Necropsy of a cockatiel with nephritis. Note that by the time the kidneys have reached this advanced stage of disease, identifying the aetiology is difficult.**

Downloaded From Vet-eBooks.com

- Immune-mediated: membranous glomerulonephritis is an immune-mediated condition following chronic antigenic stimulation (e.g. following a polyomavirus infection).

Non-infectious causes include:

- Egg yolk peritonitis
- Trauma: generalised trauma or following endoscopy or surgery.

Non-inflammatory conditions

Non-inflammatory renal disease (nephrosis) can result from:

- Amyloidosis: most commonly seen in waterfowl, small passerines and raptors. Multiple organs, including the kidneys, are affected.
- Toxins: rodenticides (vitamin D3 analogues), aminoglycosides (gentamicin, amikacin), heavy metals (lead, zinc cadmium, mercury, arsenic), mycotoxins (aflatoxin, oosporein, citrinin, ochratoxin) and others (hypernatraemia, acetone, allopurinol, ethylene glycol, glycine, oxalic acid, selenium).
- Nutritional: hypercalcaemia, hypervitaminosis D3, hypovitaminosis A, hypervitaminosis A. A syndrome is seen in colour mutation varieties of cockatiels, budgerigars and lovebirds fed on a 100% formulated diet. Currently it is thought that these diets may contain excessive levels of vitamin A for these species, although this has yet to be proved.
- Metabolic: disseminated intravascular coagulation (DIC), haemochromatosis, haemoglobin deposits, lipidosis and nephrogenic diabetes insipidus.
- Genetic/congenital: renal cysts, agenesis, and hypoplasia.
- Decreased renal perfusion associated with dehydration or hypovolaemia.
- Degenerative: renal mineralization, tubular nephrosis.
- Obstructive dysuria due to egg binding, cloacoliths, uroliths.
- Neoplasia, including carcinomas, adenocarcinoma, round cell tumours, sarcomas and embryonal nephromas.

Clinical presentation

Most birds with renal disease are presented with advanced disease. Questioning of the owner usually reveals a history of polyuria and polydypsia. Other historical findings may include access to toxins, previous health problems, other birds that have been affected, etc.

Birds with renal disease are often fluffed and lethargic, with variable degrees of muscle wastage and weight loss. Persistent polyuria (distinguished from stress or excitement-induced polyuria) is often noticeable in the cage or during the consultation (**Fig. 24.2**). Many are anorectic, but still have an increased thirst. Despite this increased water intake, the bird will appear dehydrated (sunken eyes, thin toes, wrinkled skin, and thickened saliva). In some acutely affected birds, chalky white deposits in the skin can be seen as visceral gout develops (**Fig. 24.3**). Regurgitation or vomiting is common, especially if the bird is severely polydypsic. Lameness or wing droop associated with articular gout may be seen in chronically affected birds (**Fig. 24.4**).

Differential diagnoses

Other than renal disease, there are a number of conditions associated with similar clinical signs, including:

- Liver disease.
- Pancreatic disease.
- Gastrointestinal disease.
- Septicaemia.



Figure 24.2 **Evidence of polyuria from wet newspaper in association with bird droppings. Note that polyuria must be distinguished from diarrhoea.**

Downloaded From Vet-eBooks.com

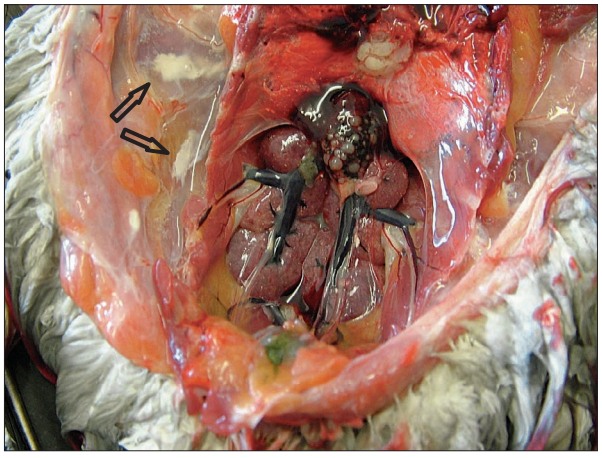


Figure 24.3 **Necropsy of a cockatoo reveals acute nephritis. Note the uric acid crystals deposited in the subcutaneous tissues (arrowed). These are often visible through the skin.**



Figure 24.4 **A *Neophema* spp. parrot presents with articular gout due to the accumulation of urates in the synovial capsules and tendon sheaths of the joints.**

- Pituitary or pineal gland neoplasia is reported as a cause of polyuria/polydypsia, especially in budgerigars.
- Diabetes mellitus is frequently reported in parrots, particularly budgerigars and cockatiels (see Chapter 18, Disorders of the Pancreas).
- Hyperadrenocorticism due to adrenal neoplasia has been reported as a cause of polyuria/polydypsia.
- Renal phosphate flush seen with low-calcium, high-phosphorus diets (i.e. seed diets) is believed to be a cause of polyuria/polydypsia.
- Psychogenic polydypsia is a relatively rare condition, often associated with juvenile hand-reared cockatoos. Stress, excitement and fear may also be contributing factors.

These conditions need to be considered when working up a bird with polyuria and polydypsia.

Diagnosis

Initial tests: Haematology and biochemistry can be helpful (see Chapter 7, Interpreting Diagnostic Tests). Many birds with renal disease have a mild anaemia and an elevated uric acid level. (Persistent hyperuricaemia in a well-hydrated bird is highly suggestive of renal disease.) Blood urea nitrogen may be elevated if the bird is dehydrated but is not a reflection of renal function.

Blood lead and zinc levels can be assessed. While this is useful for lead toxicosis, it is doubtful if measuring zinc levels is of great diagnostic value. See later in this chapter for further discussion on this topic.

Polydipsia should be confirmed quantitatively if possible. Water intake greater than 120 ml/kg/day confirms polydypsia.

Urinalysis: Urinalysis is indicated when there is persistent (not transient) polyuria. Interpretation of the results must be done with caution, as faecal contamination is the norm, and this must be taken into account. Factors to be checked include:

- Colour:
  - Biliverdinuria is suggestive of liver disease (a common cause of polyuria and polydypsia), while haematuria is strongly suggestive of lead-induced nephropathy (see **Fig. 3.15**, Chapter 3, The Physical Examination, p. 71).
- Urine specific gravity (USG):
  - This is difficult to interpret. Birds do not concentrate urine in the kidneys, but rather, in the rectum (see Chapter 1, Clinical Anatomy and Physiology p. 13).
  - Normal birds can have a wide variation in specific gravity.
  - In polyuric birds, the USG has been reported as ranging from 1.005 to 1.020. The main value of USG, therefore, lies in cases with

Downloaded From Vet-eBooks.com

a persistently low USG and those where USG fails to increase with water deprivation.

- Dipstick evaluation:
  - pH: normally 6.0–7.5. Lower readings in parrots may indicate acidosis. Higher values can indicate bacterial metabolism. Care must be taken to ensure that the colour of the urine does not influence the colour readings on the dipstick.
  - Protein: normally only trace amounts are recorded. Higher levels can indicate faecal contamination, renal disease, haemoglobinuria, haematuria, hyperproteinaemia or sepsis. Proteinuria is not usually a feature of renal disease in birds.
  - Glucose: normally zero amounts are registered. Glucosuria indicates faecal contamination, diabetes mellitus or renal damage. Glucosuria without hyperglycaemia, in the author's experience, gives a guarded to poor prognosis.
  - Ketones: ketonuria indicates severe catabolism or complicated diabetes mellitus. It also indicates a poor prognosis.
  - Blood: haematuria/haemoglobinuria can indicate faecal or cloacal contamination, or it can be associated with severe renal disease, and warrants a sediment examination.

Sediment can be examined under the microscope, at first unstained, and then stained with methylene blue. The following can be observed:

- Desquamated epithelial cells from the cloaca.
- Leucocytes and erythrocytes are rare in normal urine. More than two to three per high power field should be considered abnormal.
- Casts (granular or cellular) can be seen with renal disease. Haemoglobin casts may account for positive haemoglobin readings seen on dipsticks and where no erythrocytes are seen.
- Small numbers of Gram-positive bacteria can originate from the faeces. Large numbers of bacteria should be considered abnormal. Comparison with a faecal Gram stain may help to determine their origin.
- Uric acid crystals (small and spherical) are normal. Other crystals are occasionally seen, but their significance is unclear.

Radiography: Plain view radiographs allow visualization of the size and density of the kidneys (see Chapter 5, Diagnostic Imaging). In a lateral view, with both acetabulae on the same plane, the normal kidneys lie in the sacral area and do not extend ventrally past the ventral rim of the acetabulae. Enlarged kidneys can often be seen below these limits (see **Fig. 5.16**, Chapter 5, Diagnostic Imaging, p. 103). Radiodense mineralised kidneys can occasionally be seen. Both metastatic and dystrophic calcification can occur within the kidney as the result of renal disease, excessive dietary calcium or hypervitaminosis D3. Mineralization is not an indication of chronicity, as it can begin within days of the original insult. Radiodense metallic-like particles seen in the gastrointestinal tract can indicate lead or zinc toxicosis as possible causes of renal disease.

Endoscopy: The avian kidney is readily accessible using endoscopy (see Chapter 6, Endoscopy). Renal gout, mineralization or physical anomalies can be visualised. Endoscopy also enables the clinician to biopsy the kidney. Biopsy is perhaps the single most important tool in the accurate diagnosis of renal disease. Haemorrhage is minimal and the resultant histology (and, if appropriate, culture) allows a more accurate diagnosis, a more meaningful prognosis and a more concise treatment plan. It should be noted though that renal histologic lesions are rarely pathognomonic for a specific disease process, as many different diseases cause similar lesions.

Water deprivation test: Once other possible causes of polyuria/polydypsia have been eliminated, a water deprivation test can be used to differentiate between psychogenic polydypsia and diabetes insipidus. This test is potentially very dangerous, and must be performed with great care and caution. The rationale behind a water deprivation test is that a bird with the ability to osmoregulate will concentrate urine in response to increased plasma osmolality, due in turn to water deprivation. A gradual water deprivation test is preferred to an abrupt water dep-

Downloaded From Vet-eBooks.com

rivation test in order to overcome the problem of renal medullary washout and cloacal concentration gradients. Water is gradually restricted by 10% per day over 3–5 days, and then deprived completely. This process gives the kidneys and the cloaca every chance to respond to gradually increasing plasma osmolality. Close monitoring is mandatory, as severe dehydration can result if the patient is unable to concentrate urine. Plasma proteins, PCV, urine specific gravity, and weight should be monitored every 3–4 hours for 12–48 hours. The test should stop when the patient loses 5–7% of its bodyweight, begins to show signs of distress, or is able to concentrate its urine. Birds with psychogenic polydipsia should tolerate this test well and develop more concentrated urine (increased specific gravity) and an increase in PCV and plasma protein, all consistent with dehydration. Failure to concentrate urine in the absence of other possible causes indicates diabetes insipidus. A vasopressin response test can then be performed to distinguish between neurogenic and nephrogenic diabetes insipidus.

Vasopressin response test: Diabetes insipidus (a deficiency of, or failure to respond to, arginine vasotocin [AVT]) has been recorded in birds. Birds that have had other causes of polyuria/polydypsia ruled out and have failed to concentrate their urine on a gradual water deprivation test are likely to have either neurogenic or nephrogenic diabetes insipidus. Desmopressin acetate (an AVT analogue) is given orally at doses of 0.02–0.2 mg/kg. A reduction in polyuria and polydypsia within 30 minutes of administration confirms the diagnosis of neurogenic diabetes insipidus. Neurogenic diabetes insipidus fails to respond to the desmopressin.

Treatment

Initial supportive care includes diuresing the patient and maintaining good hydration. Parenteral fluids given at 100–200 ml/kg/day (divided into 2–3 doses) for three days, followed by 50–100 ml/kg/day until uric acid levels return to normal and remain normal. Fluids can be given IV, IO, or SC. If the patient is severely dehydrated, peripheral circulation is often compromised and SC fluids may not be absorbed (this is seen as subcutaneous oedema hours after fluid administration). These birds should be given IV or IO fluids until better hydrated. If the patient is mildly dehydrated and is still eating, oral administration of fluids is acceptable, so long as vomiting or regurgitation does not occur.

If fluids are not enough to diurese anuric or oliguric birds, furosemide can be given IV. Great care must be taken with this therapy, especially in lorikeets which appear to be exceptionally sensitive to this drug. As this drug will cause a drop in plasma electrolytes, it is important to maintain the bird on balanced electrolyte fluids such as lactated Ringer's solution.

This fluid therapy may be sufficient to lower uric acid levels to normal (<500 µmol/l) in 1–3 days. If it does not come down, or if severe hyperuricaemia (>1200 µmol/l) is present when first seen, medical therapy may be required:

- *Allopurinol* decreases uric acid production by inhibiting xanthine oxidase, which is required to convert hypoxanthine to xanthine and subsequently to uric acid. It should be noted that allopurinol is toxic to the Red-Tailed hawk (*Buteo jamaicensis*) and should not be used in this species or other raptors.
- *Urate oxidase* appears to degrade excessive uric acid to allantoin, more easily cleared by the kidneys. It acts on uric acid alone and does not interfere with the metabolism of purines. There needs to be further evaluation to better understand the use and potential long-term effects of this drug in birds. It is currently only available as a costly injectable medication.
- *Colchicine* reversibly inhibits xanthine dehydrogenase (lowering uric acid levels), blocks the synthesis and secretion of serum amyloid A, and decreases the formation and increases the breakdown of collagen. It is therefore used to lower uric acid levels, treat amyloidosis, and reduce the fibrosis associated with renal disease.

There is still controversy over the role of dietary protein in birds with renal disease. In theory, moderate protein restriction should be instituted, but this in turn may result in malnutrition and a catabolic state.

Downloaded From Vet-eBooks.com

A safe recommendation is that birds with hyperuricaemia and/or gout should not consume diets with protein levels greater that what is considered normal for the given species.

As hypovitaminosis A is suspected to contribute to renal disease, if the patient's dietary history suggests chronic malnutrition a single IM injection of vitamin A can be given at the beginning of the therapy, followed by appropriate dietary modification. Care must be taken to avoid over-supplementing the patient, as hypervitaminosis A has also been associated with renal disease.

More specific therapy can be given when a specific diagnosis is achieved by renal biopsy or other laboratory tests.

Membranous glomerulonephritis: This is commonly diagnosed in older birds by endoscopic biopsy. Therapy involves removal of the antigen responsible for the immune-mediated reaction (if possible), aspirin 1 mg/kg orally q24h and Omega-3 and -6 fatty acid supplementation, mixed in a ratio of Omega-6: Omega-3 of 6:1 and given at a dose of 0.1–0.2 ml/kg PO q24hrs. It is expected that 2–4 weeks of treatment would be needed before changes in the bird's health would be appreciated.

This therapy is not used specifically for the treatment of membranous glomerulopathies and may be used with any inflammatory form of renal disease.

Bacterial nephritis: Treatment of bacterial nephritis with appropriate antibiotics should be based on culture and sensitivity results when available. Otherwise, suspected bacterial-induced nephritis should be treated with broad-spectrum bactericidal antibiotics for a minimum of six weeks. Aminoglycosides, known to be nephrotoxic, should be avoided.

Dietary-induced renal disease: This can often be treated by discontinuing pellets and changing the diet to whole grains, seeds and vegetables. If after 3–6 months all signs of renal disease are gone, pellets (<50% of total diet) can be cautiously added to the diet.

Renal fibrosis: This can be treated with colchicine until the fibrosis resolves (and is confirmed histologically). Otherwise, it can be used for 6–12 months or until laboratory abnormalities normalise.

Zinc or lead toxicosis: This can be treated with chelation therapy (calcium EDTA, d-penicillamine). Zinc toxicosis usually only requires treatment until the metallic particles in the gastrointestinal tract have been removed (approximately 4–5 days if left to pass naturally. This needs to be confirmed radiographically before discontinuing treatment.) Birds diagnosed with lead toxicosis should be chelated twice daily until clinically normal, then twice weekly for 6–8 weeks. At the end of this period blood lead levels should be retested.

UROLITHIASIS

Definition/overview

The formation of ureteroliths in birds is rare. The stones are composed of uric acid crystals and a proteinaceous matrix. They are typically lodged in the ureters and may be unilateral or bilateral. Hypovitaminosis A may be a contributing factor.

Clinical presentation

Signs include non-productive straining, depression and anorexia. Other clinical signs will depend on the extent of renal involvement (see earlier).

Diagnosis

Radiography reveals radiopaque obstructions of the ureter. Intravenous pyelography may help to define the obstruction and ureters. Endoscopic examination of the ureter may reveal a distended ureter proximal to the obstruction. If only one kidney is involved, it is unlikely that serum biochemistries will show elevations in uric acid (assuming the other kidney is normal).

Treatment

Diuretic therapy with fluids may help to 'flush' the stone out. The stone may be able to be milked along the ureter with an endoscope or during a surgical approach. If these techniques fail to relieve the obstruction, an ureterotomy may be required. Care must be taken to minimise postsurgical stricture formation.

Downloaded From Vet-eBooks.com

GOUT

Overview

In birds, protein metabolism sees ammonia initially converted to glutamate and then to glutamine, glycine and aspartate for incorporation into the purine synthetic pathway, which results in xanthine. This in turn is metabolised in the liver by xanthine oxidase to form uric acid. This process occurs primarily in the liver, and to a small extent in the kidney. This uric acid forms soluble salts (urates) with ammonia, sodium, or potassium ions and is then transported via the blood stream to the kidneys where 80% is actively secreted by the proximal tubule (the remainder is filtered by the glomerulus). Glucose, amino acids, a small amount of urea, and electrolytes are found in the glomerular filtrate in the same concentration as the plasma, but most of this (except the urea) is resorbed in the proximal tubule to produce hypotonic urine. The filtrate then entering the collecting ducts and ureters therefore consists of uric acid as a supersaturated colloidal suspension in hypotonic urine, with a small amount of urea. Once this suspension reaches the cloaca the uric acid and ammonia is passed in the bird's droppings (the urates), while the urine is back-flushed into the rectum where it is concentrated across the rectal mucosa.

Anything – renal disease, dehydration, or obstruction of the ureters or cloaca – that disrupts this excretion of uric acid will see the blood levels of uric acid start to rise (hyperuricaemia). Since uric acid is actively secreted by the proximal tubules, hyperuricaemia does not occur until at least 70% of renal function is lost. When the levels are high enough (>600 µmol/l) the solubility of sodium urate in plasma is exceeded, and precipitation of monosodium urate crystal begins. It is the author's clinical impression that acute renal disease, causing a sharp rise in blood uric acid levels, is more likely to result in visceral gout while a slower rise (seen with more chronic renal disease) gives rise to articular gout.

Visceral gout is the deposition of uric acid on the surface of the pericardium, liver and spleen as a white coating (See **Fig. 20.3**, Chapter 20, Disorders of the Cardiovascular System p. 286) Deposits are also found within the lamina propria of the gastrointestinal tract and within the muscles, heart and kidney. Urate crystals cause mechanical damage to tissues, and this may account for the sudden death so often seen in birds affected with visceral gout.

Articular gout, on the other hand, results from the accumulation of urates in the synovial capsules and tendon sheaths of the joints (**Fig. 24.4**). This is the result of the cooler temperature of the extremities encouraging precipitation, combined with a slower rise in blood uric acid levels. The intense inflammatory reaction provoked by the urate crystals causes swelling and pain.

Clinical signs

Many birds with visceral gout are presented dead or dying within hours or days of showing signs of renal disease as described earlier. Occasionally birds will live long enough for deposits of uric acid to become visible in the subcuticular tissues.

Birds with articular gout are frequently presented for lameness. Polyuria, polydypsia and weight loss are common. Close physical examination shows swelling of the joints of the toes, leg and sometimes the wings. These swellings are often associated with grossly visible white deposits under the skin (**Fig. 24.4**). If the swellings are in the joints of the toe, the affected toe(s) is usually held extended, with the bird unable to grip the perch. If both feet are affected, the lameness will shift from foot to foot.

Diagnosis

Visceral gout is frequently a diagnosis made on necropsy (**Fig. 24.5**). Impression swabs made of the white deposits on the visceral organ surfaces will show the typical needle-shaped uric acid crystals. Due to their water-soluble nature, urates will dissolve in formalin and therefore the crystalline form will not be seen on conventionally fixed tissue. However, urates can be seen in alcohol-fixed tissue using Gomori's methenamine silver impregnation technique.

The diagnostic tests described previously for diagnosing renal disease (haematology, biochemistry, heavy metal testing, and radiology) are utilised in both live birds with suspected visceral gout and those likely to have articular gout.

Downloaded From Vet-eBooks.com

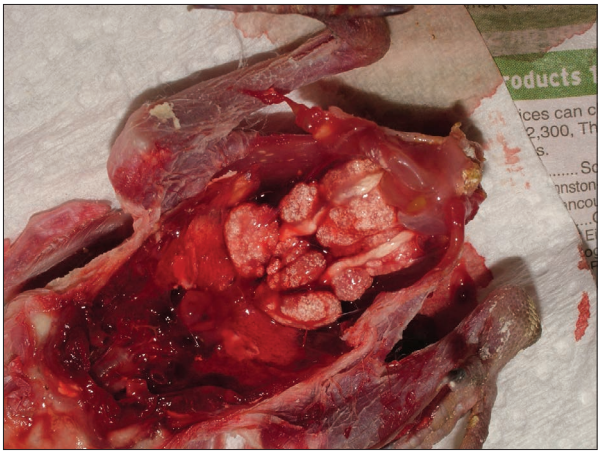


Figure 24.5 **Necropsy of a neonatal Caique chick reveals visceral gout.**

Fine needle aspirates from swollen joints examined as a wet preparation typically reveal uric acid crystals surrounded by a sterile pyogranulomatous infiltrate. Alternatively the murexide test can be performed by placing a small amount of the suspect material on a slide and mixing with nitric acid. Use a flame to evaporate and/or dry the mixture. Once cool, add one drop of concentrated ammonia. If urates are present, a mauve colour will appear.

Treatment

Fluid therapy, Vitamin A, allopurinol, and colchicine as described previously are the first line of treatment for both visceral and articular gout. The allopurinol and colchicine should be continued until the hyperuricaemia and swelling has resolved.

In addition, analgesia should be provided for birds with articular gout. As NSAIDs are generally contradicted in renal disease patients, opioids should be employed (e.g. butorphanol or tramadol).

If necessary gout deposits around joints can be carefully removed by making a small incision over the swelling and expressing the urate material. As this is both a painful and a bloody procedure, the patient should be anaesthetised and the incision bandaged afterwards to maintain some pressure on it and prevent secondary infection.

Prognosis

The prognosis for birds affected with gout, either visceral or articular, is guarded to poor. Birds with advanced articular gout are often in severe pain, and quality of life decisions need to be made by the owner and the veterinarian.

ZINC TOXICOSIS

Overview

Zinc is an essential micronutrient for birds, required for a variety of purposes including many enzyme functions, skin health and fertility. It is absorbed from the gastrointestinal tract bound to metalloproteins and utilised where required. Excess levels are excreted through the kidneys and pancreas. Toxicosis occurs when the amount circulating in the body exceeds the ability of the pancreas and kidneys to maintain homeostasis. The intestinal tract, kidneys and pancreas are the organs affected by zinc toxicosis.

Aetiology

Zinc toxicosis results from the ingestion of large amounts of zinc in the form of galvanised metal particles, wire, some coins, cosmetic jewellery, etc. Although some authors suggest that this can be a chronic intoxication, it is the author's experience that this problem is an acute intoxication as zinc is not stored in the body unless the body's homeostatic mechanisms have been overwhelmed. Clinical signs and death can follow within 1–3 days of ingestion of excessive amounts of zinc.

Clinical signs

Clinical signs relate to the organs damaged by the zinc:

- Direct irritation of the gastrointestinal tract from the presence of zinc-containing particles will result in enteritis, seen as ileus (crop stasis), vomiting and diarrhoea.
- Renal damage will result in polyuria and polydipsia, often very pronounced.
- Pancreatic damage will result in ileus and vomiting.

Consequently, many affected birds will be presented with an acute history of polydipsia, polyuria, and a crop distended with fluid. Passive regurgitation frequently follows palpation of the crop.

Downloaded From Vet-eBooks.com

Often birds are presented dead; necropsy usually shows a well-nourished body with extensive visceral gout and pale, swollen kidneys. This is, however, not pathognomonic for zinc toxicosis.

Other clinical signs that have been attributed to zinc toxicosis, such as feather-damaging behaviour and neurological signs, cannot be correlated with this toxicosis and should not be regarded as clinical signs consistent with this zinc poisoning.

Diagnosis

Clinical signs and a history of access to large amounts of zinc (e.g. a new aviary made with heavily galvanised wire) can lead to a tentative diagnosis of zinc toxicosis. Biochemistry findings typically include elevated uric acid and amylase levels.

As mentioned previously, it is doubtful if measuring zinc levels is of great diagnostic value. Normal plasma zinc values in parrots are <30.6 µmol/l (2.00 ppm), although cockatoos and eclectus parrots may be higher (38.2–45.9 µmol/l; 2.5–3 ppm). There is, however, a natural diurnal variation in blood zinc levels as well as a slight elevation in unwell or stressed birds. There is also the risk of contamination of the sample with zinc (e.g. from rubber stoppers on syringes). While elevations of a magnitude of four or five times the normal level are suggestive of zinc toxicosis, slight elevations are often misinterpreted as zinc toxicosis.

Radiographs demonstrating metallic particles in the crop, proventriculus and ventriculus of a bird displaying consistent clinical signs strengthens the diagnosis of zinc toxicosis.

Histopathologic findings from biopsy or necropsy will often demonstrate pancreatic necrosis and renal nephrosis (degenerative, non-inflammatory lesions of the kidney).

Treatment

Treatment of zinc toxicosis revolves around:

- Removal of absorbed zinc via chelation therapy e.g. CaEDTA
- Removal of zinc in the gastrointestinal tract by flushing the proventriculus and ventriculus (see Chapter 16, Disorders of the Gastrointestinal Tract, p. 239) or by allowing it to pass through. It has been shown that most metallic particles will move through the gastrointestinal tract in 3–7 days. Adding a small amount of insoluble grit to the diet can hasten this process, but tube feeding bulk laxatives such as peanut butter or mineral oil has not shown to be advantageous.
- Correction of dehydration through IV, IO or SC routes.

Removal of the fluid in the crop and denying access to drinking water until the dehydration is resolved will limit the possibility of regurgitation and aspiration of crop contents.

Response to treatment is usually rapid, with most birds showing an improvement 24–36 hours after the start of treatment. Once the metal in the gastrointestinal tract has been removed, chelation therapy can be discontinued.

Prevention

Limiting access to zinc is obviously an important preventative measure. New aviary wire should be examined for metallic tags or flakes, which should be removed. Excessive galvanizing can be removed by washing the wire with diluted acetic acid (e.g. vinegar) and then painting the wire to prevent rust.

FURTHER READING

Echols MS (2006) Evaluating and treating the kidneys. In: *Clinical Avian Medicine*, *Vol 2*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 451–492.

Echols MS (2007) Avian kidney disease, Part I: types of renal disease. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 101–116.

Echols MS (2007) Avian kidney disease, Part II: diagnosis of renal disease. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 117–128.

Echols MS (2007) Avian kidney disease, Part III: treatment of renal disease. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 129–137.

Lumeij JT (1994) Nephrology. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 748–804.

Downloaded From Vet-eBooks.com

Orosz S, Dorrestein GM, Speer BL (1997) Urogenital disorders. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 614–644.

Puschner B, St Leger J Galey F. (1999) Normal and toxic zinc concentrations in serum/plasma and liver of Psittacines with respect to genus differences. *Journal of Veterinary Diagnostic Investigation* **11**(**6**):522–527.

Raidal SR, Echols MS (2007) The advantages and disadvantages of excreting uric acid. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 87–100.

Downloaded From Vet-eBooks.com

CHAPTER 25

BEHAVIOURAL PROBLEMS

INTRODUCTION

All bird behaviour originates from 'wild' behaviour, and can be either instinctive or learned. Companion birds need to effectively acquire learned behaviours that can make them socially acceptable as pets and companions. Common behavioural problems seen in clinical practice include behaviours such as:

- Attention demanding.
- Displacement behaviours.
- Territoriality.
- Biting.
- Feather damaging.
- Screaming.
- Reproductive issues.
- Phobias.
- Psychotic behaviour.

Many of these problems are often chronic by the time the bird is presented to veterinarians. Early recognition and treatment of these problems is more likely to result in successful treatment.

PRINCIPLES

Companion birds face many challenges. These challenges are directly attributable to a lack of knowledge – and sometimes ignorance – of a bird's requirements. The two most common presentations of chronic problems seen by avian veterinarians are malnutrition and behavioural problems. Malnutrition stems from the traditional concept of birds as seed eaters. The behavioural problems, however, stem from a lack of understanding of the unique nature of bird behaviour, and the owner's attempts to impose their own demands and expectations on their bird, expecting them to behave more like a dog or a cat, or even a child.

Understanding bird behaviour is a constantly evolving subject. What was accepted as fact 3–5 years ago has been debunked or disproved or, at best, modified. Pre-existing notions of bird behaviour and its 'modification' or 'therapy' need to be re-examined and, where appropriate, discarded.

UNDERSTANDING BIRD BEHAVIOUR

To understand bird behaviour it is necessary to go back and look at the behaviour of birds in the wild; after all, most pet birds are only a few generations removed from wild birds. However, to generalise and talk about 'bird behaviour' is akin to talking about 'mammalian behaviour'. There are some 9,000 species of birds; approximately 360 of these are parrots. Parrots have evolved in Australia, South America, Africa and Asia. Different environments and environmental pressures have shaped both the physical and psychological development of these birds, and to try and apply 'rules' across all 360 species is fraught with danger. However, there are some generalizations that can be made.

Avian behaviour can be categorised into two functional groups:

- Self-maintenance behaviours designed to accomplish a specific task to maintain the health of the individual. These include feeding, feather care, locomotion and concealment.
- Social behaviours designed to communicate information to another individual. These behaviours include territoriality, concern or fear and courtship.

Downloaded From Vet-eBooks.com

Parrots are altricial; the young are hatched near-naked, blind and perhaps deaf. They live for the first weeks of their life in a quiet, dark hollow, completely dependent on their parents (**Fig. 25.1**). Social interactions at this stage are limited, confined to their siblings and parents. After fledging, many parrots learn to socialise by contact with their parents, siblings and other birds (often their own age) in a flock (**Fig. 25.2**). An example of this is the rose-breasted cockatoo (galah). In the wild, clutches of 2–5 chicks usually survive. Once they fledge, the chicks are taken to a nearby 'crèche', a tree or stand of trees where other galah 'families' are interacting. Here they learn early social skills before joining a nomadic juvenile flock where they remain until reaching sexual maturity. This socialisation period teaches the juvenile birds food recognition (and how to locate it), predator recognition, sentinel duties, grooming behaviour, survival skills and early social behaviours.

As they reach sexual maturity, they learn new skills: how to select a mate, exhibit courtship behaviours and develop a pair bond; how to select, prepare and defend a nest site; and how to reproduce and raise their young.

Some of these behaviours are believed to be innate or instinctive; others are learnt. All are reinforced by the reaction the bird receives. Captive parrots, especially those hand reared as pets, may not have the opportunity to learn these behaviours. Their instinctive behaviours, particularly as they reach maturity, may bring them into conflict with their human flock.

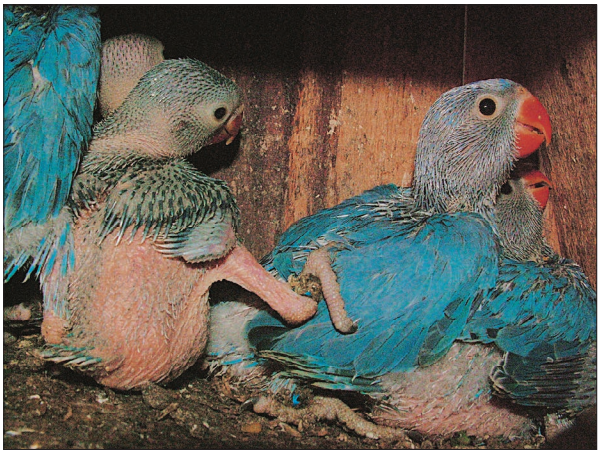


Figure 25.1 **Indian ring-neck chicks (violet colour mutation) in the nest. Note the relatively cramped and dark conditions.**



Figure 25.2 **Flock of galahs feeding on grass seed in Australia. Many parrots learn to socialise by contact with their parents, siblings and other birds (often their own age) in a flock.**

HOW DO BEHAVIOURAL PROBLEMS DEVELOP?

Problems seen in clinical practice can be attributable to one of two causes. The first basic problem is a failure of the socialisation process. This is usually the result of an individual bird being hand reared in isolation and not being taught basic social skills. Once weaned, the bird is often ignored as its novelty value wears off. This process often results in attention-demanding behaviour (begging calls, screaming, feather chewing) and displacement behaviours such as biting, feather damaging behaviour, phobias and sometimes even self-mutilating behaviour.

The second group of problems result as a failure of the human 'flock' to understand normal parrot behaviour, and expecting birds to 'fall into line' with their human expectations (**Fig. 25.3**). It was once said that there are no abnormal behaviours, just normal behaviours expressed inappropriately. Behaviours such as screaming morning and night, displaying territoriality and certain reproductive behaviours are examples of normal behaviour that are inappropriate in a companion bird scenario.

So how do these problem behaviours develop?

The reinforcement of self-maintenance behaviours benefits companion birds, and abnormal

Downloaded From Vet-eBooks.com



Figure 25.3 **Five-month-old Moluccan cockatoo. Many pet birds bond closely with humans at this age.**

self-maintenance behaviours are the most common behavioural disorders seen in these birds. They still have the same self-maintenance behaviours as their wild counterparts. However, they need less time for foraging and feeding behaviours (after all, the food is in a dish in front of them every morning) and therefore feather care, social communication and displays make up more of their daily activities.

Young captive birds need continued mentoring and behavioural moulding and require guidance for the establishment of a normal bird–human flock relationship. This includes a range of normal social behaviours of flock interaction, with appropriate rules of conflict resolution and appropriate maintenance and social behaviours.

Failure to be taught – or learn – these behaviours means that many young birds are not prepared for a life in captivity, and may develop behavioural problems. In the absence of imposed rules, the bird will make its own rules based on immediate gratification and revolving around perceived value; however, these rules may not be socially acceptable. They eventually develop into behavioural problems and the bird becomes unable to interact socially with people without fear or social framework, and therefore a series of displacement or defensive behaviours develops (e.g. aggression, biting). As these behaviours develop, the bird may become even more isolated and therefore become more vocal in trying to re-establish contact with their 'flock'.

This is often reinforced when owners respond: the bird receives a positive response (e.g. talking, feeding) and it may augment the behaviour; if, on the other hand, the bird receives a negative response (e.g. covering cage, time-out, water pistols) that response may augment the feeling of isolation and the problem may worsen.

Unfortunately, these problems are often chronic by the time the bird is presented to a veterinarian. Early recognition and treatment are much more likely to result in successful treatment; prevention through education of bird owners is the most preferable approach. Techniques such incorporating behavioural training into annual wellness examinations are important steps in preventing problems, and they should be pursued vigorously by all those involved in the wellbeing of companion parrots.

HISTORICAL APPROACH TO TREATING BEHAVIOURAL PROBLEMS

Earlier attempts to treat behavioural problems in birds revolved around human concepts of 'discipline' and psychotherapy.

Humans are driven, in many cases, by fear of punishment and negative reinforcement. From early childhood people are made well aware of the consequences of inappropriate behaviour – corporal punishment or even just the disapproval of adults. Given that early thoughts on parrot IQ were along the lines of 'as intelligent as a 3–5-year-old, but with the maturity of a 2–3-year-old', it is not surprising that people expected birds could be motivated by the same fear of negative consequences.

And, if punishment did not work, surely drugs would! The use of hormonal therapies, antidepressants, sedatives and even narcotic antagonists was advocated as the best therapy for 'misbehaving' patients. Conventional medicine sometimes dictates that for every disease or problem there is a drug.

Downloaded From Vet-eBooks.com

So, studies and trials were conducted that resulted in many a companion bird sitting on a perch in a drug-induced stupefaction, and everyone (except the bird) was satisfied.

Eventually, it was realised that neither of these approaches was successful in curing behavioural problems. They might have stopped for a while, but as soon as the threat of punishment or administration of drugs was stopped, the problem recurred.

NEW CONCEPTS

The first change in thinking on bird behavioural therapy came with the realization that drugs and physical restraint devices (e.g. Elizabethan collars) just did not work. Although they may have a limited, short-term role in selected cases, these therapies often worsen a situation and do not provide a long-term benefit.

A second change in thought was in dealing with the concept of dominance. It was long thought that a strict hierarchical structure existed within bird flocks, with dominance being reflected in advantages such as the highest perch, the best food and first choice when selecting a mate. It was further thought that in a human–bird 'flock' situation, a similar dominance had to be maintained, with the human at all times being the dominant member of the 'flock'. However, studies of wild birds has shown that dominance, while it does occur, is not at all strict; rather, it is a fluid arrangement with different birds being dominant at different times. A flock is inconsistent in its structures: members are lost to predators, family groups change between flocks, new members are constantly being added. This makes a straight-line hierarchy difficult to maintain, and too energy demanding to be compatible with survival in the wild. Therefore, the rigid dominance so often seen in human society does not readily transfer over to the bird world, and attempts to impose it usually involve 'flooding' a bird (overwhelming it with an imposed behaviour until it acquiesces) or using negative reinforcements and punishment as means of instilling discipline. The problem with these techniques is that the bird learns to perform a behaviour, or avoid exhibiting one, just enough to escape adverse consequences. Once these consequences are removed, the behaviour returns.

New concepts in behavioural therapy revolve around several principles:

- Birds are not people, or dogs, or cats. They are unique – they are birds.
- The best results are obtained when a bird wants to do the behaviour that the owner desires.
- A successful treatment may be a reduction in an unwanted behaviour, rather than a complete cessation of it.
- Successful behavioural change in companion parrots starts with behavioural change in the parrot owner.

Birds are not people, or dogs, or cats

Imposing human values and emotions on to an animal (anthropomorphism) can hinder the behavioural modifications clinicians are trying to institute. Using terms such as angry, aggressive, jealous and happy implies a state of mind which, realistically, we cannot truly assess. By doing so, a set of expectations is created in the mind of the owners and perhaps in the clinician as well.

Parrots are animals that can:

- Learn to feed from seasonally variable food sources.
- Learn the specific call or 'dialect' of their flock.
- Interpret subtle nuances in body language of other members of their flock.
- Perform elaborate courtship displays.
- Learn to mimic sounds in their environment in order to manipulate an environmental condition or maintain a pair bond.
- Develop monogamous pair bond relationships.
- Rigorously defend breeding territory.
- Develop flight skills advanced enough to avoid/escape predatory attack.
- Successfully rear young and make decisions about resource availability that influence fledgling mortality.

They are not little children with feathers, nor are they similar to mammals such as dogs and cats. They are unique and deserve to be treated as such. It is important to attempt to replicate the environment in which they evolved, providing them with opportunities to

Downloaded From Vet-eBooks.com

exhibit normal behaviours such as foraging for food, flying, interacting socially and just having fun.

The best results are obtained when a bird wants to do the behaviour that the owner desires

Reinforcing behaviour can be either positive or negative. Negative reinforcement involves applying a noxious consequence to a behaviour in the expectation that the bird will cease the behaviour to avoid the consequence. Examples of this include shouting at the bird, giving it time out, covering a cage or squirting the bird with a water pistol. The problem with negative reinforcement is that the bird will do only just enough to avoid the consequence, and no more. In fact, as soon as the consequence is removed, the behaviour usually returns. Undesired consequences of negative reinforcement can include escape/avoidance behaviour, aggression, apathy (a decreased responsiveness in behaviour) and fear, leading to phobic behaviour.

Positive reinforcement, on the other hand, involves providing a favourable consequence (or reward) for a behaviour. This increases the likelihood that the bird will repeat the behaviour in the expectation of the reward. The motivation used to stimulate a desired behaviour must be evaluated from the bird's perspective, and this can be influenced by several factors:

- The relationship between the bird and the person. This relationship must be built on trust, respect and prior positive experiences. Good communication must exist between the two: both must understand the other's body language in order to know what they want (**Fig. 25.4**).
- The bird's confidence and ability to perform the behaviour.
- The bird's past experiences in training and motivation.
- Natural influences such as social interaction, breeding season, comfort and height.
- The bird's hunger state or preference for certain feed items.

Some examples of good motivators include favourite food items, praise, and petting, proximity to people, a familiar object and even out-of-cage time.



Figure 25.4 **Macaws in a large free flight aviary choosing to interact with humans.**

Care must be taken to prevent inadvertently reinforcing a behaviour while trying to use negative reinforcement. The classic example of this is shouting at a screaming parrot. Parrots are naturally noisy animals that seem to thrive on noise and drama; it is, after all, a natural part of their life and normal social interaction. While the owner thinks he/she is 'punishing' an undesired behaviour, they are, in fact, giving the bird positive reinforcement and guaranteeing that the behaviour will be repeated.

Positive reinforcement can be used to increase or decrease the likelihood of a behaviour being repeated. It can also be used to establish and encourage other behaviours. These new behaviours can either be alternative behaviours or behaviours that are incompatible with the undesired behaviour. Either way, it can lead to extinction of an undesired behaviour.

A successful treatment may be a reduction in an unwanted behaviour, rather than a complete cessation of it

Many behavioural problems are often chronic in nature by the time the bird is presented to a veterinarian. Feather picking is a classic example: by the time the owner has waited to see if it goes away, then consults the Internet and tries a variety of pet shop remedies, it may be many months or even years before the bird is presented to a veterinarian. Obviously such behaviours, now deeply ingrained and with their own set of positive motivators (perhaps only known to the bird), are going to be difficult to eradicate. This must be made clear to the

Downloaded From Vet-eBooks.com

owner from the beginning (i.e. that success in these cases may simply be a reduction in the behaviour, rather than its complete elimination).

Successful behavioural change in companion parrots starts with behavioural change in the parrot owner

There is no doubt that many owners of birds are directly attributing to the problems they are experiencing, and directly or indirectly reinforcing them. As mentioned earlier, people grow up in a 'punishing' environment, where transgressions are punished and human behaviours have evolved to avoid such punishments. Birds are not like this, and do not learn well in this manner. People who wish to interact and socialise with birds, preventing or overcoming behavioural problems as they do so, must change their own behaviour in order to better understand and work with their bird.

These principles call for a re-assessment of how humans think of, and interact with, birds. Until we do so, our success in modifying their behaviour will be limited.

BASIC STEPS TO IMPLEMENT IN A BEHAVIOUR-MODIFICATION PROGRAMME

There are some basic steps to implement in a behaviour-modification programme when dealing with problem behaviours. They include:

- Basic training.
- Normalisation of social interaction.
- Avoidance of unwanted behaviours.
- Replacement of unwanted behaviours with acceptable behaviours.

Basic training

Many pet birds are hand reared and seem to recognise and accept people as 'members' of their flock. This recognition, and the interaction that comes with it, is what helps to make parrots such enjoyable companion birds. Normally, other flock members would teach the juvenile bird what social behaviours are appropriate through a system of examples and trial and error. With companion birds, the role of mentor and teacher has to be filled by the owner of the bird. It is essential to have the basic requisites for training in place first in order to provide a solid foundation for acceptable social behaviours. These basics include step-up and step-down, and staying on a perch where placed.

Step-up

Stepping up onto an offered hand is a foundational manoeuvre upon which most training and behavioural guidance relies. In light of what is discussed above, it is important that the bird does this behaviour because it wants to, not because it has to. By using a food reward and the cue words 'step up', a bird can be taught to step up on to the owner's hand and stay there.

Step-down

Stepping down allows the owner to guide the bird's movement. Once again, positive reinforcement (e.g. a food reward) and the cue words 'step down' can be used to teach the bird to step off the owner's hand and on to a suitable perch.

Stay

Staying on a perch is important for a bird to experience 'normal' flock social interaction while outside of its cage with its 'flock members', without getting into mischief or requiring constant supervision. The free-roaming pet parrot is at a much greater risk of traumatic injuries and household poisonings. It is also more likely to develop pair bonded interactions with one person, and less likely to interact with other people. Portable table-top perches are suitable for this training since they can be put anywhere. The bird is stepped down on to the perch and rewarded (verbally or with a food reward) for remaining there. If they climb down and walk around, avoid inadvertently rewarding this behaviour; simply put them back on to the perch without any verbal cue or other reward. If they stay there, offer another reward. It is usually necessary to gradually extend the time between placing the bird on the perch and offering the reward.

Downloaded From Vet-eBooks.com

Normalise social interactions

This has to be done on two levels:

- The interactions between the parrot and the person to whom the bird has bonded have to be modified. Interactions such as picking up the bird, scratching its head and body and playing with it have to be stopped or reduced dramatically while the modification programme is being implemented. The bonded person can clean the cage and put in fresh food and water; however, the less interaction the better.
- Other members of the household need the opportunity to interact positively with the bird. To avoid distracting the bird or triggering protective or territorial aggression, it is best if the bonded person is not around while this is being done and, when 'out of cage' training is being done, the bonded person should bring the bird to a place in which it does not exhibit territorial behaviours. The bird should learn to interact with other people using a combination of positive reinforcement and incremental exposure. For example, the new person can drop a preferred food reward in the bird's food bowl and then walk away. If this is done often enough, eventually the bird will look forward to seeing the new person. This person can then try offering the reinforcement to the bird through the cage bars. Eventually the reward can be offered for stepping up on the hand as well as other cooperative behaviour. Alternatively, working in a neutral territory, the other members of the household can cue the bird to perform simple behaviours the bird already knows how to do. This gives the bird an activity on which to focus, while at the same time receiving rewards from people other than the one the bird has bonded to. This can help build a positive relationship with the rest of the household.

The goal with these two levels of modification is to limit the positive experiences with the bonded person and, at the same time, increase the positive experiences with the new person. However, it may never be possible for the new person to interact at the same level the bonded person can with the bird, and it is unlikely positive interactions can occur while the bonded person is in the room.

Avoid unwanted behaviours

Analysis of a problem behaviour should follow the ABC of behaviour, where:

A = Antecedent: the situation which led to the behaviour.

B = Behaviour: the actual behaviour displayed by the bird.

C = Consequences: what the bird got out of the behaviour. Consequences can be positive or negative.

The consequence will determine whether a behaviour will be repeated or not. If it is not possible to alter the consequence (and therefore the bird's likelihood of repeating the behaviour), it becomes necessary to change the antecedent. For example, if a bird screams loudly each morning until it is fed, the behavioural analysis is as follows:

- The antecedent is that there is no food in the bird's bowl first thing in the morning.
- The behaviour is that the bird screams.
- The consequence is that the owner hurriedly feeds the bird, positively reinforcing its behaviour.

The bird still has to be fed each day; therefore, the consequence cannot be changed. However, if the owner places food in the bowl after the bird has gone to sleep the night before, and therefore food is available to the bird as soon as it wakes up, the antecedent has changed and the behaviour should change (or stop).

Sometimes it is necessary for the owner to change his/her behaviour in order to change the bird's behaviour.

Replacement of unwanted behaviours with acceptable behaviours

Most birds, captive or wild, spend their time during the day performing daily maintenance behaviours that are essential for survival. These behaviours include foraging (**Fig. 25.5**) social interaction and feather care (**Fig. 25.6**). Wild birds spend

Downloaded From Vet-eBooks.com



Figure 25.5 **Wild cockatoo foraging for food, an activity that takes up nearly 80% of their normal day.**



Figure 25.6 **Grooming behaviour in a black-capped lory. Grooming takes up a relatively small amount of time in the wild, but captive conditions leave more time to groom which can lead to over grooming.**

approximately 80% of their waking life foraging for food, with the remaining 20% of their time devoted to socialising and grooming.

If 'normal' social interaction with members of the household is limited, as is often the case when owners are at work or school, the other maintenance behaviours (foraging and grooming) must be increased to fill the time (**Fig. 25.7**). Companion birds, with their food provided in the same dish, in the same place and at the same time each day, do not have to spend so much time foraging in order



Figure 25.7 **A clear example of an undersized cage which doesn't provide sufficient foraging stimulation for this King parrot to alleviate boredom nor sufficient room to move.**

to survive. This deficit in activity may be replaced with abnormal behaviours such as stereotypical behaviours, feather-damaging behaviour through over grooming (**Fig. 25.8**), or screaming. It may also heighten the bird's anticipation of the owner's presence and lead to problems with pair bonding.

By increasing the daily foraging activities available to a bird, its lifestyle can be enhanced significantly. Interestingly, several studies have confirmed that when a bird is given a choice between foraging for food or eating it out of a dish, most birds prefer to forage.

Foraging activities that can be implemented include:

- A piece of non-treated wood (e.g. pine) is drilled with holes into which nuts, seeds or other treats fit tightly. The reward should be visible but not accessible without chewing down through the wood. The wood can be used as a perch in the cage, hung in the cage to increase the challenge, or used as a toy outside the cage (see **Fig. 2.6**, Chapter 2, Husbandry. Grooming and Nutrition, p. 48).
- Wrapping the food bowls with newspaper or cardboard to make the bird chew through it to get at the food. A starter hole may be necessary to encourage the bird to begin chewing.
- Wrapping food items in small pieces of paper, corn husks or other materials. Not all wrappings need to contain a reward.

Downloaded From Vet-eBooks.com



Figure 25.8 **Galah who has been over grooming leading to feather damaging behaviour. Galahs reared in isolation often develop behavioural problems.**

- Mixing food with inedible items (e.g. wood buttons or other items) so that the bird has to dig through to find its food. Some parrot species can be particularly stimulated into new foraging behaviours by having a large box full of different items in which some desired food items or treats can be found. This is particularly useful for ground-feeding birds (such as many Australian parrots); a sandbox full of wooden beads or paper pellets, with food scattered through it, can keep a bird entertained for many hours.
- Puzzle toys that require birds to unscrew parts or manipulate components to get at their reward (**Fig. 25.9**).

Other activities that can be implemented to encourage normal behaviours include:

- Lightly spraying the bird with water will dampen the feathers and encourage the bird to groom normally.



Figure 25.9 **Although a large number of toys are available for birds, as shown here, in some cases birds have to be taught in stages how to use them.**

- Providing items for the bird to chew and destroy (e.g. non-toxic tree branches, cardboard boxes and wooden toys).

Bird behaviour is complex and, as mentioned earlier, our understanding of it is still evolving. There is no simple approach to dealing with individual cases but, by applying the principles and strategies outlined above, clinicians should be able to have a positive impact on the relationship between bird and owner.

FURTHER READING

Briscoe JA, Reisner IR, Rosenthal KL (2004) Incorporating the veterinary behaviourist: a new model for the diagnosis and treatment of a feather damaging pet parrot. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 293–296.

Echols MS (2005) Practical use of foraging as a means of behaviour modification. In: *Proceedings of the Annual*

Downloaded From Vet-eBooks.com

Conference of the Association of Avian Veterinarians Australian Committee, pp. 185–194.

Friedman SG, Edling TM, Cheney CD (2006) Concepts in behaviour. Section I: The natural science of behaviour. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing, Palm Beach, pp. 46–59.

Heidenreich B (2005) Addressing aggressive behaviour in birds. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 127–138.

Heidenreich B (2005) Solving companion parrot behaviour problems. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 219–228.

Heidenreich B (2006) How to train medical behaviours. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 135–148.

Luescher AU (2004) Rearing environment and behavioural development of psittacine birds. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 297–298.

Luescher AU (2006) *Manual of Parrot Behaviour*. Blackwell Publishing, Oxford.

Speer BL (2002) Practical clinical applications for the exam room. In: *Proceedings of the Association of Avian Veterinarians Australian Committee*, pp. 155–164.

Wilson L, Greene Linden P, Lightfoot TL (2006) Concepts in behaviour. Section II: Early psittacine behaviour and development. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing, Palm Beach, pp. 60–72.

Wilson L, Lightfoot TL (2006) Concepts in behaviour. Section III: Pubescent and adult psittacine behaviour. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing, Palm Beach, pp. 73–84.

Downloaded From Vet-eBooks.com

CHAPTER 26

INCUBATION OF EGGS

WHY INCUBATE EGGS?

Incubation allows increased production, particularly in rare or valuable species. Removing an egg shortly after it is laid often induces the hen to lay a replacement. Poor parenting by some birds means that eggs and/or chicks can be lost in the nest. Removing and artificially incubating the egg can also reduce this risk.

It was thought that hand rearing birds made them bond closer to people; therefore, artificial incubation was (and still is) widely practised to provided 'bonded' chicks for the pet market (see Chapter 25, Behavioural Problems p. 344).

In some cases, artificial incubation is used as a means of disease control. Pathogens that are not vertically transmitted through the egg, but can be horizontally transmitted, can be reduced or eliminated by artificial incubation.

WHEN TO COLLECT EGGS

Artificial incubation is rarely as efficient and effective as natural incubation. The eggs of small species (e.g. conures; cockatiels; lovebirds; budgerigars) do not do well when incubated from day zero; it is thought that vibrations from the incubator may cause decreased hatchability. In these birds, incubation should start after two weeks of natural incubation. Large birds also benefit from natural incubation for the first 2–2.5 weeks.

However, it may, in some circumstances, be necessary to collect eggs immediately (or shortly after) being laid (e.g. when the parents are destructive to the eggs or when it is desired to increase egg production – if the hen broods for 2–3 weeks, she may not lay another clutch in that breeding season).

STORING EGGS PRIOR TO INCUBATION

Some aviculturists prefer to store eggs prior to beginning incubation in order to synchronise hatching and hand rearing. However, it is usually preferable not to store eggs, as hatchability reduces on a daily basis when stored. This loss of hatching viability is species dependent. If there are no other options (and the aviculturist accepts the risk of decreased hatchability), eggs can be stored as follows:

- Temperature in the storage area should be kept between 12.8°C and 18.3°C to prevent embryonic development.
- The relative humidity should be maintained between 80% and 90%.
- The eggs should be turned 90° at least twice daily.
- Eggs should not be stored for more than seven days.

SANITATION OF THE EGGS

Eggs should not be cleaned if at all possible. Routine washing is not recommended, as this can reduce hatchability. Dry cleaning with a soft brush is the preferred means of cleaning eggs. If it is necessary to wash the eggs, a 10% quaternary ammonia or 4.2% chlorine solution can be used. The water used should be warmer than the egg, so as to prevent the egg drawing in the washing solution through its pores. Detergents must not be used on very porous eggs.

Fumigation using formalin and potassium permanganate is sometimes recommended (20–30 minutes contact with egg), this but may be harmful/carcinogenic to humans. Formaldehyde gas is also teratogenic in chickens.

Downloaded From Vet-eBooks.com

EQUIPMENT

Incubators can be either forced air (fan-driven) or convection heated (cool air is drawn in at the bottom of the incubator, warmed, and finally exhausted past the eggs through the top of the incubator). Forced air incubators give more uniform temperature control and air flow than convection heated incubators, and are preferred by most aviculturists (**Fig. 26.1**). Incubators can only add heat to the eggs' environment; they cannot cool air.

Accurate humidity control is essential. It is important to recognise that incubators can usually only add moisture to the egg environment; they rarely are able to de-humidify it. Water reservoirs in incubators can act as a growth medium for potentially pathogenic bacteria such as *Pseudomonas*, *Aeromonas* and *Klebsiella* species, and should be cleaned regularly.

Automatic turning controls are recommended, but must be checked for serviceability and vibration. Jarring and jolting of the eggs when the automatic turner is operating can reduce hatchability and increase the incidence of malpositioning.

As very few incubators can either cool air drawn in or dehumidify it, it is recommended that the incubator be kept in a clean, biosecure, air-conditioned and dehumidified room whenever possible.

INCUBATION PARAMETERS

Temperature

Temperature determines the rate of embryonic growth. The correct temperature should see the egg hatch at the same time a naturally incubated egg does. Larger eggs need lower temperatures; smaller eggs need higher temperatures. Typically, psittacine eggs develop normally if incubated between 36.4 and 37°C. As mentioned earlier, the room temperature in the incubator room must be cooler than the incubator, allowing the incubator to warm the egg rather than trying to cool it.

Humidity

Eggs lose weight during incubation due to water loss through the shell (transpiration). The rate of water loss is dependent on shell thickness, shell porosity and the water vapour concentration of the atmosphere.

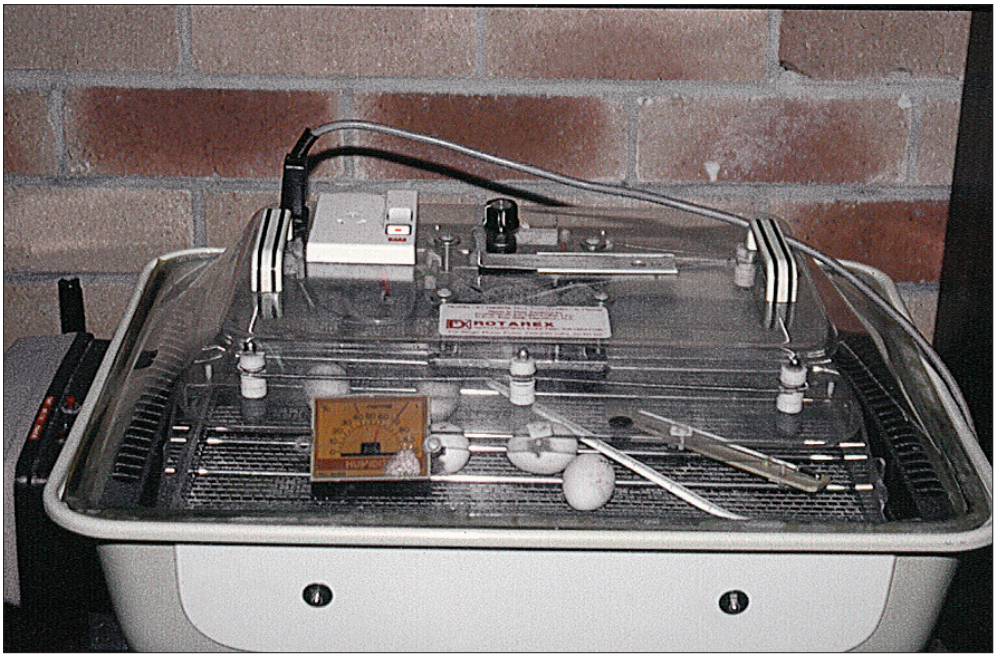


Figure 26.1 **Commercially available forced air incubator. Note that these are more effective and efficient than convection heating incubators.**

Downloaded From Vet-eBooks.com

The expected weight loss is 16–22% over the incubation period. To achieve this, it is usually necessary that the relative humidity in the incubator should be between 38 and 46%. The actual humidity in each incubator is determined by the weight loss of the eggs in the incubator (see below).

As incubators cannot dehumidify air, the relative humidity in the incubator room should be lower than in the incubator, allowing the incubator to add water to increase the relative humidity around the eggs.

Ventilation

Oxygen and carbon dioxide are exchanged across the shell (respiration). If the carbon dioxide builds up in the incubator, the chicks may suffocate. Therefore, there must be a continual infusion of fresh air into the incubator (forced air incubators are more effective than convection incubators).

Turning and positioning

Turning of the egg allows proper development of the embryo and the blood vessels in the egg, and allows better distribution of nutrients in the albumen. The old theory of the embryo 'sticking' to the shell is no longer considered valid. The eggs should be turned a quarter turn along the long axis every 4–5 hours. This is usually done mechanically, although older incubators rely on manual turning. The turning should be smooth, with no vibration or jolting of the eggs.

Poultry and ratite eggs are incubated vertically, with the air cell up; psittacine eggs are incubated horizontally with the air cell end slightly elevated. Eggs that are not positioned correctly within the incubator are more likely to develop malpositioned embryos or increased embryonic death.

MONITORING

Embryonic development can be monitored by candling (examining the egg in a darkened room with an intense focal light to transilluminate it) (**Fig. 26.2**). Candling at 7–10 days will indicate if the egg is fertile and developing properly. Clear yolks showing no signs of blood vessels or development by day seven are infertile or died early, and should be removed.



Figure 26.2 **Candling an egg. In this case the egg was infertile.**

Occasionally a 'blood ring' (extravasated blood in a ring surrounding the remnants of the embryo and embryonic circle of blood vessels) is seen. This indicates early embryonic death.

Weight monitoring should also be carried out, ideally by daily weighing, although weighing every 2–3 days can be sufficient in well-developing eggs. Eggs should lose 13–22% of their weight between setting and removal to the hatcher through evaporative loss through the shell. They may lose another 3% during hatch, although excessive weight loss at this time may dehydrate the chick, making hatch difficult. Eggs with a thin or very porous shell, or those placed in a low humidity incubator, can lose an excessive amount of weight through evaporative loss. These eggs should be placed into a higher humidity. Conversely, those with thick shells or low porosity, or those placed in a high humidity incubator, can retain moisture and lose insufficient weight. These eggs should be placed into a low humidity environment to encourage more evaporative weight loss.

HYGIENE

As incubators are warm and humid and contain a high biological content they can easily become a bacterial and fungal incubator if not maintained correctly and rigid hygiene procedures aren't followed. Water reservoirs should be cleaned daily, broken or dead eggs should be removed promptly and dust filters should be cleaned every few days or more. The incubator should be emptied, aired, and disinfected as frequently as possible, using a broad spectrum disinfectant such as F10® or Virkon®.

Downloaded From Vet-eBooks.com

RECORD KEEPING

It can be very difficult to persuade aviculturists to maintain detailed records, making it almost impossible at times to investigate incubation problems. Records that should be maintained include:

- Daily temperature and humidity readings in the incubation.
- Notes on any problems such as power failure, equipment malfunction, etc.
- For each egg, the following details should be recorded:
  - Laying date.
  - Setting date.
  - Setting weight.
  - Candling and weighing data.
  - Weight when moved to the brooder.
  - Outcome of incubation and hatching (e.g. clear egg; fail to hatch; hatched without difficulty; hatched with difficulty).
  - Viability of each chick.
- Overall the number of eggs laid, fertility and hatchability for the season and for each breeding pair should be recorded.

When these records are available, a thorough investigation into a hatchability problem is feasible. The use of egg necropsies can provide more information.

EMBRYONIC DEVELOPMENT

Embryonic growth is divided into three approximately equal phases: early, mid and late terms.

Early term

In poultry and cockatiels the following events occur:

- Day 1: blastoderm develops, becomes doughnut-shaped as cellular division occurs.
- Day 2: brain, eyes and spinal cord develop.
- Days 2–3: heart and blood vessels grow, and the extra-embryonic membranes form:
  - Amnion (from the body wall, cushions embryo in amniotic fluid).
  - Yolk sac membrane (from gut, envelops the yolk).
  - Allantois (from hind gut, acts initially as bladder).
  - Chorion (an extension of the amnion). It fuses with the allantois to form the chorioallantoic membrane (CAM), the primary respiratory organ of the embryonic chick.
- Days 3–4: body wall and viscera grow.
- Days 4–5: limbs develop.

Mid term

There is growth with little further differentiation. Feathers begin to form, the bones begin to calcify and the beak forms.

Late term

The yolk sac is drawn up into the embryo, the umbilicus is sealed, and the albumen is actively drunk by the chick. Finally, the chick positions itself and hatches.

HATCHING

Hatching is stimulated by the chick converting from chorioallantoic membrane (CAM) respiration to pulmonic respiration. Increasing carbon dioxide concentration causes the neck muscles to twitch and the chick becomes more active. The air cell expands and extends down one side of the egg 24–48 hours before internal pipping. This process is known as 'drawing down'.

The chick then penetrates the air cell (internal pipping) by rubbing its beak against the inner shell membranes until they tear. The chick is now able to breathe the air in the air cell. As the chick is jerking inside the egg, it rotates 360°, cracking the shell circumferentially (**Fig. 26.3**). This is known as the 'external pip'.

The hatch time, the interval from internal pip to hatch, is usually 36–48 hours in most species. Hatching times less than 24 hours, or more than 80 hours can indicate a problem.

Requirements for a hatcher in which an egg can be placed to hatch are:

- A lower temperature than the incubator, usually 36.4–36.9°C.
- A higher humidity, up to 70%.
- No turning.
- Adequate ventilation.

Downloaded From Vet-eBooks.com

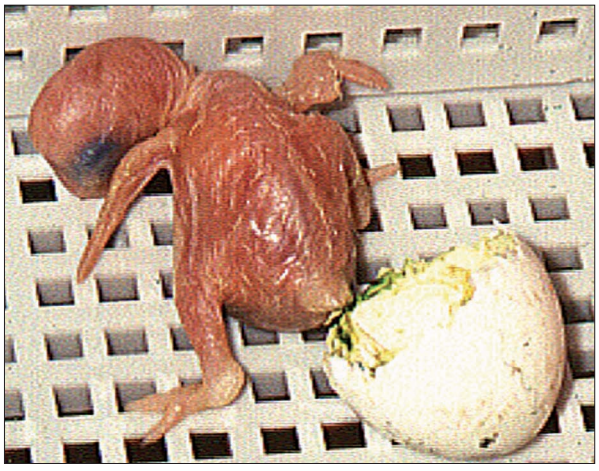


Figure 26.3 **Indian ring-neck chick in an incubator shortly after hatching. Note the circumferentially cracked egg known as the 'external pip'.**

PROBLEMS WITH INCUBATION

Embryonic mortality should not exceed 10% of fertile eggs set for incubation. This normal 10% mortality is made up of:

- Early-term deaths, 3–4%.
- Mid-term deaths, 1–2%.
- Late-term deaths, up to 4%.

Losses above these figures require investigation. The two most common causes of mortality/morbidity in the egg are malposition, the most frequent cause (see below) and inadequate moisture loss (See *Table 23.1*, p. 329).

EGG NECROPSY

All eggs that fail to hatch should be necropsied. Not only does an egg necropsy allow a determination of the stage and possible cause of embryonic death, but it also provides the clinician (and aviculturist) with a true indication of fertility.

The technique is relatively simple. The egg is firstly weighed and, if possible, the weight loss during incubation is calculated. The shell quality is examined (surface texture, porosity, and cleanliness). The egg is then candled. Candling allows identification of the air cell (mark it with a pencil), an indication of its size and mobility, and an idea as to whether an embryo or infection is present. Hairline cracks in the shell can also be identified. This information is recorded before moving to the next step.

The egg is placed vertically onto a support (an egg cup or a ring of PVC pipe is sufficient) with the air cell uppermost. The shell over the air cell is broken with a sharp pair of pointed scissors, revealing the air cell. The shell is then trimmed away to the level of the inner shell membrane forming the base of the air cell. This membrane is then peeled away with fine forceps, revealing the egg contents.

At this stage a swab for culture of bacteria and fungi can be taken from the albumen or inside the shell. If an embryo is not obviously present, examine the blastodisc on the yolk. The yolk around this disc is less dense than the rest of the yolk, so it will float uppermost. In an infertile egg the disc will appear as a small white point. A very early embryo (blastoderm) will appear as a small white 'doughnut' with a patch of yolk in the middle. Any blood vessel development at all is confirmation of fertility.

If an embryo is present, two determinations must be made: the positioning of the embryo (if it is late-term) and the stage of development.

Embryonic position is observed before removing the embryo from the egg. The normal late-term embryonic position is: head next to the air cell, right side uppermost and turned to the right, with the beak adjacent to the right wing tip and the egg tooth pointed towards the air cell; legs flexed on either side of the body and ventral to the shoulders; and the spine following the long axis of the egg. Any other position is classified as a malposition, and may account for embryonic death (See *Table 23.2*, p. 330).

The embryo is then removed from the egg and examined more closely. An assessment of the embryo's growth and development can give an indication as to whether this is an early, mid-term or late-term embryo (see above). Samples can be collected from the chorioallantois, yolk and proventriculus for culture, and tissues from multiple organs, including the membranes, can then be placed in formalin for histopathology.

Downloaded From Vet-eBooks.com

By determining the stage and cause of embryonic death, the clinician can then start to narrow down the search for the underlying cause of embryonic mortality.

FURTHER READING

Jordan R (1989) *Parrot Incubation Procedures*. Silvio Mattacchione and Co., Pickering.

Jordan R (2001) Incubation of psittacine eggs. *Seminars in Avian and Exotic Pet Medicine* **10**(**3**):112–116.

Joyner KL (1994) Theriogenology. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 748–804.

Olsen GH, Clubb SL (1997) Embryology, incubation and hatching. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philapdelphia, pp. 54–72.

Schubot RM, Clubb KJ, Clubb SL (1992) *Psittacine Aviculture: Perspectives, Techniques and Research*. Avicultural Breeding and Research Center, Loxahatchee, Fl.

Downloaded From Vet-eBooks.com

PAEDIATRICS

INTRODUCTION

Most companion birds (e.g. parrots and passerine birds) are altricial (i.e. when hatched they are blind, deaf and not feathered, and therefore totally dependent on their parents or rearer) (**Fig. 27.1**). As juveniles, their health status is determined not only by their current environment, but also by the input from their parents and how the eggs were incubated.

Pre-laying factors:

- The health of the parent birds can affect the chick via pathogens transmitted through the egg to the chick, and by the degree of transference on maternal immunity via the albumen and yolk.
- The diet fed to the parents, particularly the levels of calcium and vitamins, will have a direct effect on the vitality, development and health of chicks – even before hatch.



Figure 27.1 **Parrot chicks, such as these lorikeets, are altricial and totally dependent on their parents or rearers until weaned.**

- The maturity of the parents will affect the size of the eggs laid, the immunity passed through the egg, and the number of eggs laid.
- Parental genetics become a major issue in the breeding of mutations, where closely related birds are mated to develop certain characteristics such as colour. Unfortunately, along with desirable characteristics such as new colour, undesirable physical characteristics, such as decreased body size or physical deformities, can also occur.

Incubation factors:

- Artificial vs. natural incubation: as a general rule parent-incubated eggs tend to hatch out stronger chicks. Advances in incubation techniques (see previous chapter) are, by and large, resulting in better chicks. However, skill and experience are required to match the results seen in natural incubation.
- Temperature, humidity and hygiene in the nest box or incubator will have direct effects on the development of the chick and its hatchability and strength after hatch. (See Chapter 26, Incubation of Eggs, for further details.)
- Care in handling of the eggs by either the parents or the incubator operator, as well as the frequency and degree of rotation during artificial incubation, can affect the degree of difficulty in hatching, as well as an effect on the incidence of malpositions.

Post-hatch factors:

- If the chick has been hand reared, the following parameters in the rearing environment play important roles in the health of the chick:
  - Temperature extremes can force the chick to divert resources from its growth and health towards maintaining a constant body temperature

Downloaded From Vet-eBooks.com

- Humidity extremes can predispose a chick to dehydration, respiratory infections and skin problems (e.g. toe constriction).
- Poor hygiene will substantially increase the concentration of pathogens (or potential pathogens) in the chick's environment. With the chick's immune system still developing, this creates a high probability of infectious disease.
- Although the era of homemade hand-rearing diets is passing, nutrition of the chick is still a major factor in the health of the chick. The practice of adding ingredients to a well-balanced formula (often based on anecdotal information from other aviculturists) can have major detrimental effects on the chick.
- Management of the nursery, in particular biosecurity and the quarantine of new chicks from different sources, will determine the likelihood of introduction of infectious diseases such as Avian Polyomavirus.

Detailed knowledge of hand-rearing practices, including weaning ages, can be obtained from reputable aviculture literature.

EXAMINATION OF THE CHICK

History

As with any other medical case, a good history of the chick is an essential key in the diagnostic workup. Factors to consider stem from the earlier discussion on determinants of a chick's health. They include:

- The parents: their genetics, diet, maturity and health status.
- Incubation: was it artificial or natural? The hatchability of fertile eggs is a key indicator of incubation performance, and hopefully it can be found in the aviculturist's records.
- Hatching: if the eggs were artificially incubated, were there any problems with hatch?
- How is the nursery managed regarding hygiene, biosecurity, and the source of eggs and/or chicks?
- What type of food is been fed, how is it prepared, what volume is fed and how frequently?
- If there are any siblings or other chicks, have there been any problems or deaths within the group?
- What records does the aviculturist keep? Details on hatch dates, hatch weights, growth rates, mortalities, medications, and previous medical problems are valuable sources of information, but are sadly lacking in many cases.

Physical examination

Weight

The chick should be weighed and its weight compared with the expected weight for that age, found in growth charts (if available). All chicks will gain weight each day; it is the rate of weight gain that is more important than the actual weight. This can be monitored by expressing the chick's weight as a percentage of the expected weight for that age. If a problem exists, improvement can be seen as a gain in percentage of expected weight, rather than actual weight gain.

Posture

It is important to be aware that chicks sleep and rest in what seems to be 'awkward' positions. (For example, conure and macaw chicks often sleep on their backs.) These positions change as the chick moves; one should look for postures that do not change with movement.

Conformation

The positioning and conformation of the limbs and the spine should be checked. Common conformational abnormalities include a kinked (or 'wry') neck, scoliosis, kyphosis, tibiotarsal or femoral rotation, and anteroflexion of the toes.

Body condition

The toes and elbows in a well-nourished, healthy chick should be 'plump'. Thin toes and elbows are a good indicator in neonatal chicks of dehydration, malnourishment or disease. Palpation of the pectoral muscles is helpful; the soft keel bone at this age should be well fleshed with soft (but poorly developed) pectoral muscles (**Fig. 27.2**).

Behaviour

Pre-weaning chicks should be either resting, sleeping or calling for food. Restlessness could

Downloaded From Vet-eBooks.com



Figure 27.2 **A healthy sulphur-crested cockatoo chick.**

indicate incorrect environmental temperature or stress (e.g. excessive lighting). As they get older, the chicks still spend a lot of time sleeping, but are more interested in their environment and in socialising with nursery mates.

A feeding response (vigorous extension and bobbing of the head and neck) should be easily elicited by pressing gently at the commissures of the beak. Failure to elicit a response can be an indication of disease, hypothermia or weakness.

Skin

Prior to full feathering the normal chick's skin should be pink or pink–yellow in colour, and soft and warm to the touch. Pallor of the skin can indicate hypothermia, anaemia or illness. Erythematous skin can indicate hyperthermia or illness. Heavily wrinkled skin indicates dehydration (**Fig. 27.3**).

Crop

The normal crop should have some food in it at most times (**Fig. 27.2**). It should not be over-distended, nor should it have significant amounts of air or gas in it. Rhythmic contractions of the crop should be visible in neonatal chicks, and the crop should nearly empty in 4–6 hours in all chicks.



Figure 27.3 **Green Cheek conure chick with wrinkled skin, and poor filling of the basilic vein which are good indicators of dehydration.**

Head

The size of the head should not be excessively large in relation to body size. The beak should have a normal conformation (see Chapter 11, Disorders of the Beak and Cere). There should be no sinus swellings. The nares should be open and symmetrical.

The eyes should be symmetrical and healthy in appearance. They begin to open at 10–28 days and take several days to open completely. Most Australian and African parrots hatch with their ears open. The ears of eclectus and South American species should be open within 2–3 weeks after hatching.

Oral cavity

The oral cavity should be examined for diphtheritic plaques or other abnormalities. Its colour should be noted; it is normally pale pink.

Abdomen

In neonatal chicks the abdomen should be large and convex relative to the rest of the body (**Fig. 27.2**). The liver may be visible through the skin in very young chicks, and rhythmic contractions of the ventriculus should be visible. The duodenal loop may be visible. There should not be bruising or haemorrhage visible. The abdomen can be trans-illuminated with an intense focal light for closer inspection.

Downloaded From Vet-eBooks.com

As the chick grows, the abdomen reduces in size relative to the rest of the body. It should be concave when palpated; a convex abdomen could indicate a degree of abdominal distension.

Feather growth

Depending on the species, some chicks are hatched naked or with wispy down feathers. Others are covered in fluffy down feathers (**Fig. 27.4**). A second wave of down feather growth begins at 1–3 weeks of age and, sometimes, even later in some species.

Pin feathers begin to emerge at 2–3 weeks of age. The body contour feathers emerge over the shoulders first; the pattern of emergence after that varies between species, although usually the body contour feathers emerge at the same time or shortly before the secondary flight feathers on the wings. Primaries may begin to develop before secondary feathers, but usually mature after them. Final feather maturity is usually not complete before the bird has weaned.

Abnormalities include:

- Feathers erupting in an unusual pattern (e.g. in a circular pattern on the crown of the head, rather than running parallel along the line of the body).
- Stress bars in the opened vane.
- Abnormal colouring.
- Haemorrhage in the calamus.
- Dystrophic development.



Figure 27.4 **Yellow-tail black cockatoo chick, showing fluffy down feathers.**

Droppings

The faecal portion should be relatively well formed, light brown in colour, and not malodorous. A degree of polyuria is normal (especially in hand-reared chicks), but this should lessen as the chick ages. Excessive or persistent polyuria warrants further investigation.

Diagnostic testing

Microbiology is an important tool in assessing gastrointestinal flora. Gram stains and cultures are frequently used to assess crop or other gastrointestinal problems. Normal bacterial flora includes *Lactobacillus*, *Streptococcus*, *Staphylococcus* and *Bacillus* spp. Low numbers of *E. coli* are often normally cultured as well. Other gram-negative bacilli and *Candida* are rarely cultured from healthy chicks.

Clinical pathology can be used readily on chicks. It is important to note that compared to adults of the same species, chicks normally have:

- Lower PCV and higher white cell count.
- Lower total protein and uric acid.
- Higher CK.

Radiography is an essential tool for assessing the status of the skeletal system, but the low density of the bones and the cartilaginous growth plates in very young chicks can make this difficult.

COMMON PROBLEMS

Stunting

Aetiology

Stunting is seen in the first 30 days of life. It is usually associated with improper feeding techniques (poorly balanced or incorrectly mixed diets, inadequate amounts fed, etc.), poor environmental conditions (temperature and humidity extremes) or disease (e.g. renal disease).

Clinical presentation

Signs include subnormal weight gain, reduced muscle mass (toes, wings, back should be checked), abnormal feathering (e.g. head feathers develop in a circular pattern on the crown) and oversized head

Downloaded From Vet-eBooks.com



Figure 27.5 **A stunted black-headed caique chick suffering with a crop burn. Note the oversized head.**

relative to the size of the body (**Fig. 27.5**). Eyelids fail to open normally or when expected, and there is delayed ear opening or narrowing of the ear canal. The affected bird may suffer with chronic, recurrent infections, and be constantly calling and begging for food. As the chick gets older it often develops a globose head with an elongated slender beak. The eyes may appear exophthalmic because of the misshapen skull.

Management

The predisposing cause should be identified and treated. Nutritional inadequacies should be corrected. The prognosis is good if the problem is diagnosed early and treated successfully.

Crop stasis ('sour crop')

This is a commonly seen problem in paediatric medicine, with most sick chicks having varying degrees of crop stasis. All too often hand rearers attempt to diagnose and treat this problem without seeking to understand the underlying pathology.

Aetiology

Causes include:

- Generalised ileus (e.g. systemic illness, foreign bodies, chilling, heavy metal toxicosis, dehydration).
- Crop disorders (e.g. foreign bodies, overstretched/atonic crop, infectious ingluvitis, fibrous food impaction, or crop burns).
- Dietary problems (e.g. cold food, excessively watery food, food that settles out in the crop, overfeeding, overly dry food).

Clinical presentation

Signs include the crop failing to empty in more than six hours, regurgitation and loss of feeding response. Most chicks will be dehydrated on presentation (erythematous wrinkled skin, tenting of the skin and sunken eyes).

Aspiration of the crop contents usually reveals the sour-smelling fermenting ingesta ('sour crop') or thickened ingesta (hand-rearing formula with the fluid drawn out of it).

Faecal output may be reduced, and the stool may be unformed or pasty.

Diagnosis

This is based on crop and faecal cytology (Gram stain) and culture, haematology and biochemistry, and radiography.

Management

The cause should be identified using the means outlined above, and corrected where possible.

The crop should be emptied with a feeding tube and repeatedly lavaged with warm saline until a clear wash has been obtained. (In some extreme cases, e.g. foreign bodies, it may be necessary to perform an ingluviotomy.) It should always be assumed that these chicks are dehydrated, and they should be treated with parental fluids until crop motility has been restored.

Appropriate antimicrobials should be given as indicated by crop and faecal cytology/culture. It is overly simplistic to assume that these cases are always yeast infections; in the author's experience bacterial overgrowth is usually more common.

A crop 'bra' can be used if needed. This is a non-adhesive bandage placed under the crop and around the wings to 'lift and support' the atonic crop in order to allow gravity to assist with crop emptying (**Fig. 27.6**).

Once the crop has been emptied, in many cases it may be advisable to leave it empty for a few

Downloaded From Vet-eBooks.com



Figure 27.6 **Juvenile black cockatoo in a crop 'bra' to assist with crop emptying.**



Figure 27.7 **Juvenile black cockatoo with a crop burn. Note the initial blanching of the skin prior to necrosis.**

hours while dehydration is corrected. Initial feeds should be of small volumes of isotonic saline. If this moves through, solids can be added. Small, watery meals should be fed often. Pre-digesting the hand-rearing formula with a small amount of pancreatic enzymes can liquefy the diet without diluting it.

Motility modifiers (e.g. metoclopramide or cisapride) may assist in restoring motility, although their efficacy is poor if used without other supportive measures. Metoclopramide, in particular, is usually ineffective unless given as a constant rate infusion. In uncomplicated cases (where the chick is otherwise bright), a strong solution of fennel tea given by crop drench may assist in restoring motility.

The prognosis is good, provided prompt and appropriate therapy is provided.

Thermal injuries to the crop (Fig. 27.7)

See Chapter 16, Disorders of the Gastrointestinal Tract, p. 232.

Crop perforations

See Chapter 16, Disorders of the Gastrointestinal Tract, p. 232.

Orthopaedic problems

See Chapter 14, Disorders of the Legs, Feet and Toes, p. 201.

Beak malformations

See Chapter 11, Disorders of the Beak and Cere, p. 187.

Omphalitis

Omphalitis (infected yolk sac) is not uncommon in incubated chicks. There are two syndromes seen: a failure to draw the yolk sac into the body prior to hatch (**Fig. 27.8**); or an infection tracking up through an umbilicus that has failed to close properly (**Fig. 27.9**).

The first condition is usually seen when the incubator temperature is too high, or the humidity too low. The result is that the chick hatches before the yolk sac has been 'drawn up'. These can often



Figure 27.8 **Severe macaw chick with an externalised yolk sac.**

Downloaded From Vet-eBooks.com



Figure 27.9 **Necropsy of a macaw chick reveals omphalitis.**





Figure 27.10 **Six-week-old African grey parrot with a wry neck (a) and following the fitting of a foam neck brace to correct it (b).**

be treated by ligating the yolk sac and removing it, followed by closing the umbilicus. Unfortunately, a significant number of these chicks fail to thrive and often die within days or weeks.

The second condition occurs when the umbilicus fails to close fully after hatch and the hygiene of the brooder is poor. The resulting infection usually kills the chick in a few days; the diagnosis is usually made by necropsy. If this becomes a problem in a particular nursery, apply an iodine-based ointment to the navel at hatch can help to prevent an ascending infection.

'Wry Neck'

This term describes a condition where there is either vertebral malformation or tendon contracture in the neck of a newly hatched chick. The result is the neck is twisted to varying degrees (**Fig. 27.10a**). The neck can usually be gently turned to a normal position but reverts to its twisted position when released. Treatment is to apply a foam neck brace to hold the chick's head and neck in a more normal position (**Fig. 27.10b**).

Foreign Bodies

Chicks are by nature inquisitive animals, and it is not uncommon for them to ingest non-food items while exploring their environment. They are also vigorous feeders, and it is not uncommon for them to swallow a feeding tube that has been dislodged from a syringe. It can be surprising how quickly these foreign bodies can move from the crop into the proventriculus and ventriculus (**Fig. 27.11**).

If the foreign body is still in the crop it can often be removed through the mouth under sedation or a short anaesthetic. If this is not possible an ingluviotomy can be used to retrieve the object. Material that has moved into the proventriculus (**Fig. 27.12**) will require an ingluviotomy and removal by endoscopy or gentle flushing.

Infectious disease

Definition/overview

Infectious diseases are quite common in young chicks; their low level of immunocompetence combined with often substandard rearing practices leaves

Downloaded From Vet-eBooks.com



Figure 27.11 Radiograph of an African Grey chick who has swallowed a feeding tube (outlined by arrows).

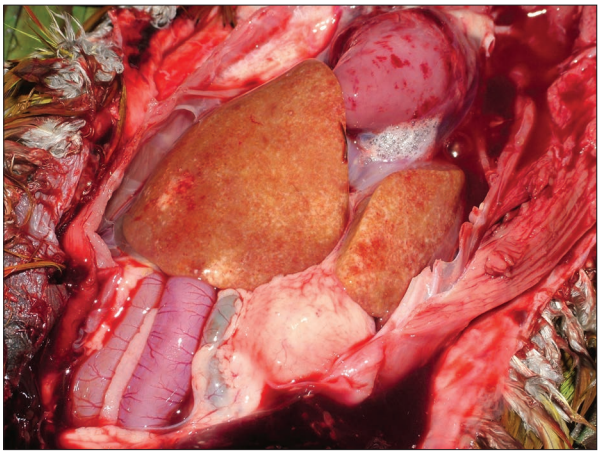


Figure 27.12 Necropsy of an Amazon parrot chick reveals adenoviral hepatitis.

them highly predisposed to infection. This same lack of immunocompetence means that the progression of an infectious disease in young birds is often rapid. Prompt and aggressive therapy is needed to save the patient.

Aetiology

Infections may be bacterial (*Pseudomonas*, *E. coli*, other Gram-negative bacteria); fungal (*Candida*, *Aspergillus*); viral (polyomavirus, adenovirus, PBFD); *Chlamydia psittaci*; parasitic: protozoa (Cryptosporidia, Trichomonas, Cochlosoma, Coccidia and Atoxoplasma [in young canaries]); and nematodes (ascarids, Capillaria and Acuaria).

Clinical presentation

Signs include lethargy, loss of feeding response, pallor or erythema of the skin, dehydration, crop stasis, vomiting/regurgitation, weight loss or failure to thrive, subcutaneous haemorrhage, feathering abnormalities and sudden death.

Management

The aetiological agent should be identified and the chick treated accordingly. The patient will require supportive care (see Chapter 8, Supportive Therapy).

Avian Polyomavirus (APV)

Aetiology

Polyomavirus is a non-enveloped virus that is relatively environmentally stable. All parrots can be affected, but it is most common in macaws, conures, eclectus parrots and caiques. It is rare in African grey parrots, cockatoos and cockatiels. Passeriformes, including grass finches, canaries, goldfinches and greenfinches can also be infected. It is unknown if the virus in these birds is antigenically related to the virus in parrots, or if it can be transmitted between parrots and passeriformes.

Pathogenesis

The virus is passed horizontally in urine, faeces and respiratory and skin secretions. Vertical transmission is suspected, but not yet adequately proven. Infected non-budgerigar parrots shed the virus 2–7 days after being experimentally inoculated.

Infection does not always result in disease. Disease determinants include species of the bird, age at which it is infected and concurrent immunosuppression (e.g. PBFD). The age of peak susceptibility varies between species:

- Budgerigars: 10–25 days old.
- Conures: <6 weeks old.

Downloaded From Vet-eBooks.com

- Macaws and eclectus: <14 weeks old.
- Lovebirds: up to 1 year (concurrent PBFD may be a factor).
- Older birds usually do not show clinical signs unless secondarily immunosuppressed (e.g. PBFD).

Adult birds can be infected, but they rarely show signs of disease.

Infected birds of any age that do not develop disease will still have viral replication and shed the virus. The length of time APV is shed is determined by the age at which the bird is infected (the older the bird, the shorter the shedding period) and the species of bird:

- Budgerigars shed for up to six months, stopping at sexual maturity or the first breeding cycle.
- Conures shed for 4–8 weeks, rarely up to 16 weeks.
- Macaws shed for 6–14 weeks.
- Cockatoos shed for 8–10 weeks.

Clinical presentation

In budgerigars (the primary reservoir of APV) signs include decreased hatchability and embryonic death, abdominal distension, subcutaneous haemorrhages and feather dysplasia that may resolve after several months (primary and secondary feathers on the wings and tail are lost or fail to erupt – in the budgerigar fancy these birds are known as runners and the condition is known as French Moult) (see **Figs 10.12a** and **b**, Chapter 10, Disorders of the Skin and Feathers, p. 171). There may be neurological signs if the cerebellum is affected.

In other parrots APV causes sudden death (especially those <15 days old) or crop stasis, weakness, yellow urates, pallor, bleeding from feather follicles and subcutaneous bruising. Most parrots die, usually 10–14 days after exposure. In larger parrots, most deaths occur between 20 and 140 days old.

In estrilid finches (grass finches) the virus causes acute mortality in 2–3 day-old fledglings, young adults and mature finches. Survivors may demonstrate poor feather development and misshapen lower beaks.

Infection is rare in fringillidae finches (canaries, goldfinches, greenfinches), where three types of lesions have been described: pulmonary (firm lungs with pale areas amongst congestion); hepatic/splenic (hepatosplenomegaly, focal haemorrhages in the liver); and cutaneous (warts have been described on the beaks of canaries). Other concurrent infections are common (e.g. *Atoxoplasma*, poxvirus).

Diagnosis

Antemortem diagnosis can be achieved using PCR probes. After initial infection there is a viraemia that can be detected by blood PCR. After a short period, the virus can be detected in the cloaca by PCR. When the bird is about to stop shedding, the blood PCR becomes negative. After 6–8 weeks the cloacal shedding stops. Therefore:

- Blood positive, cloaca negative: early stages of infection, or intermittent shedding.
- Blood and cloaca positive: bird actively infected and shedding.
- Blood negative, cloaca positive: bird is about to stop shedding.
- Blood and cloaca negative: bird has cleared infection, no longer shedding.

Blood should be tested first. If it is negative, the bird should be quarantined for eight weeks and can then be considered clear. If it is positive, the bird should be retested in 2–3 months.

Gross pathology findings include hydropericardium, cardiomegaly, hepatomegaly, splenomegaly, generalised pallor (muscles may appear an orange colour) and petechial haemorrhages.

Histopathologic findings include:

- In larger parrots, clear to basophilic intranuclear inclusions can be found in the spleen, in mesangial cells in the kidneys and in Kupffer cells.
- In budgerigars inclusions can be found in most organs including the cerebellum, renal tubules, splenocytes and hepatocytes. The most prominent are often found in the feather follicles and growing feathers.
- Massive hepatocellular necrosis.
- Immune-mediated glomerulonephropathy.

Downloaded From Vet-eBooks.com

Management

Non-budgerigar aviaries: By the time the first case is recognised, most other birds will already be infected. Therefore vaccination or testing may not be of any benefit. Hand rearing should stop and chicks allowed to be parent reared, as there is less incidence of disease in parent-reared birds (except in budgerigars). All birds to be sold should be tested by blood PCR and held for two weeks after a negative result before selling.

Budgerigar aviaries: Nestlings and young birds are the reservoir of infection, therefore breeding should be stopped for four months (until all birds are at least six months old). All young birds should be removed and the adults moved to a clean environment. Disinfection is carried out with phenolics, sodium hypochlorite or stabilised chlorine dioxide.

Prevention

Non-budgerigar aviaries: It is best not to keep budgerigars, cockatiels or lovebirds without testing them. Other people's birds should not be brought into the nursery. A closed aviary should be maintained (i.e. no movement in without testing first; see Chapter 2, Husbandry, Grooming and Nutrition, p. 52). PBFD should be tested for at the same time. If birds do have to be moved out, and then back in, they must be at least 18 weeks old and, if vaccinations are being used, they should be given four and two weeks prior to movement. Traffic control is used to prevent movement from adults to chicks.

Budgerigar aviaries: A random number of birds should be tested to determine if disease is present. Only PCR-negative birds should be brought in, including own birds returning from shows.

Adenovirus

Aetiology

Avian adenoviruses are non-enveloped DNA virus that are stable outside the host and resistant to many disinfectants. Although usually considered to be opportunistic pathogens, some highly virulent strains may be primary pathogens. Adenoviruses are associated with many diseases in both companion and domestic birds, including marble spleen disease (pheasants), egg drop syndrome (chickens), quail bronchitis, and pigeon adenovirus.

In parrots it is believed the route of infection is horizontal (ingestion or inhalation), with young (immune-incompetent) birds most at risk. Although vertical transmission has not been confirmed in parrots, it is suspected to occur in poultry. As an opportunistic pathogen, adenovirus has also been associated with immunosuppressive diseases such as PBFD. It is frequently isolated from persistently infected, but asymptomatic birds.

Clinical presentation

In budgerigars and lovebirds (*Agapornis* spp.) it has been associated with nephritis and pancreatitis. Occasionally neurological signs (ataxia, torticollis, and weakness) are seen as well.

In nursery-reared parrots of all species it is occasionally seen as sporadic cases of acutely ill chicks which succumb and die within hours of presentation. Necropsy reveals a markedly enlarged, pale, mottled liver and splenomegaly (**Fig. 27.12**).

Diagnosis

Histopathology often reveals hepatic, splenic and intestinal necrosis associated with basophilic intranuclear inclusion bodies. PCR can be used to confirm the presence of adenovirus and to detect carriers.

Management

There is no vaccine available for adenovirus. Affected birds may receive supportive care but usually die despite this care.

Adenovirus are inactivated by exposure for one hour to formalin, aldehydes and iodophors.

Psittacine Beak and Feather Disease

PBFD is occasionally seen in young parrots (especially African grey parrots) as acute onset of anaemia and leucopaenia, followed by death. Necropsy reveals hepatomegaly and splenomegaly, associated with basophilic intracytoplasmic inclusion bodies. See Chapter 10, Disorders of the Skin and Feathers for more detail.

Downloaded From Vet-eBooks.com

FURTHER READING

Clubb SL (1997) Psittacine pediatric husbandry and medicine. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 73–95.

Flammer K, Clubb SL (1994) Neonatology. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 805–841.

LaBonde J (2006) Avian reproductive and pediatric disorders. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee*, pp. 229–238.

Schubot RM, Clubb KJ, Clubb SL (1992) *Psittacine Aviculture: Perspectives, Techniques and Research*. Avicultural Breeding and Research Center, Loxahatchee, Fl.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

ANALGESIA AND ANAESTHESIA

ANALGESIA

Signs and effects of pain

It is generally accepted that birds perceive pain along neurological pathways similar to those in mammals. However, as discussed earlier, birds may indicate pain in less obvious ways than domestic mammals. They appear to respond to painful stimuli in one of two ways:

- 'Fight-or-flight' responses:
  - Excessive vocalisation.
  - Wing flapping.
  - Decreased head movement.
- Conservation–withdrawal responses:
  - Immobility.
  - Closure of eyes.
  - Inappetence.
  - Fluffing of feathers.

It is thought that the 'fight-or-flight' response is more common when the bird is suffering from sudden or unexpected pain from which it is attempting to escape. In contrast, with chronic or overwhelming pain (which is perhaps more commonly seen in clinical practice) birds appear to be more likely to adopt the 'conservation–withdrawal' responses, perhaps in an attempt to minimise the further pain that struggling would induce and to avoid attracting the attention of potential predators. Care must therefore be taken not to misinterpret lack of movement or vocalisation as an indication that the bird is not in pain (**Fig. 28.1**).

In addition to these clinical signs of generalised pain, localised pain may be reflected in feather picking or other self-mutilatory behaviours over, or in the vicinity of, the painful lesion.



Figure 28.1 **Hawk with injuries and obvious bruising. Note that birds are not adept at displaying obvious signs of pain so the clinician should assume the patient will benefit from analgesia.**

While we know that pain is a response to a noxious stimulus that serves the purpose of alerting an animal to the presence of that stimulus and thereby provoking a response that removes the animal from the stimulus and therefore further injury, we also now know that it is has wider, more far-reaching consequences. Through stimulation of the adrenal gland and subsequent release of corticosterone, pain has an adverse effect on wound healing and the immune system. Survival and recovery rates of birds in pain are much lower than those receiving effective analgesia. As clinicians we therefore have not only a moral obligation to provide effective analgesia, but also sound medical reasons.

Principles of analgesia

There are two underlying principles to consider when developing an analgesic plan for a patient.

Downloaded From Vet-eBooks.com

Pre-emptive analgesia

Learning to anticipate when a patient will be painful is helpful, as pain is easier to control if the patient is treated before it becomes painful and upset. A painful event stimulates peripheral nociceptors, which transmit the 'pain' signal to the brain via central spinal nociception pathways. Continued stimulation facilitates these pathways and evokes a painful response, a process known as 'wind up'. This facilitation may outlast the initial stimulus by a prolonged period. The underlying theory of pre-emptive analgesia is to use drugs and techniques before, during and after a painful event in order to block this facilitation, preferably before it occurs. When dealing with patients who are already experiencing pain, it is not possible to apply a pre-emptive approach, which may explain why it is more difficult to get the pain under control initially, and then it requires more therapy to maintain analgesia if therapy is continued. The use of pre-emptive analgesia before performing any procedure likely to cause pain is therefore a key element in managing pain.

Multimodal therapy

The perception of pain is both a peripheral and a central event, using several types of receptors and pathways, so there is no single drug, class of drug or action that can be used as a sole analgesic agent. The concept of multimodal therapy recognises this and encourages a multi-level approach to analgesia.

1. Remove the source of pain.
   This may involve splinting a fractured limb, removing a foreign body or performing surgery with minimal tissue trauma and handling.
2. Reduce fear and stress.
   Fear and stress may enhance the nociception pathways. Providing privacy in a warm, quiet environment in order to reduce apprehension and the judicious use of anxiolytics (e.g. midazolam) may serve to relieve fear and stress and therefore enhance analgesic therapy.
3. Use several classes of drugs to treat pain at different nociceptors.
   As described above, peripheral nociceptors detect a painful stimulus and transmit, via spinal nociceptive pathways, a message to the brain where central nociceptors perceive it as pain. Inflammation in peripheral tissues may sensitise the peripheral nociceptors, invoking a more intense pain response at lower levels of pain. Using drugs that can reduce inflammation (and therefore decrease peripheral nociceptor sensitivity) while other drugs are used to block spinal pathways and/or central nociceptors can provide more comprehensive analgesia than one class of drug alone.

Analgesics commonly used in avian practice

The most commonly used analgesics in avian practice are opioids and NSAIDs. Local anaesthesia may also be used in certain circumstances.

Opioids

Some bird species appear to have more kappa opioid receptors in the forebrain than mu opioid receptors, explaining why some birds do not respond to mu agonists such as morphine, buprenorphine and fentanyl in the same manner as mammals. Therefore, kappa opioids such as butorphanol may be the more efficacious analgesics in birds.

Butorphanol (1–4 mg/kg IM or orally q6h) is currently recommended for opioid analgesia in birds. Higher doses may be hyperalgesic. In the author's experience, morphine at a high dose (1 mg/kg) provides effective analgesia in most birds with minimal side-effects. Side-effects to opioids are uncommon, but may include respiratory depression, nausea and vomiting, bradycardia and constipation. Treatment with naloxone reverses many of these unwanted effects, but also removes the analgesic effect of the drug.

Tramadol, a synthetic opioid, is becoming increasingly popular as an analgesic drug in birds. Used at doses of 10–30 mg/kg IM or PO q6–12h, it appears to provide effective analgesia. Side-effects include lethargy and mild ataxia at high dose rates.

Non-steroidal anti-inflammatory drugs (NSAIDs)

NSAIDs act on peripheral tissues and therefore are indicated when tissue damage and inflammation are the source of the pain. They may be most effective when acting synergistically with opioids.

Downloaded From Vet-eBooks.com

NSAIDs are COX inhibitors, inhibiting the production and actions of prostaglandins, prostacyclin and thromboxane (which cause hyperalgesia and sensitise nociceptors). They are arbitrarily divided into COX-1 inhibitors (inhibiting the production of prostaglandins that are important in physical function, e.g. renal perfusion, gut barrier maintenance) and COX-2 inhibitors, which inhibit the production of inflammatory prostaglandins. In theory, COX-2 inhibition will have an analgesic effect, while COX-1 inhibitors are more likely to have adverse side-effects such as renal damage and gastrointestinal damage. However, this division is not clear cut, and all NSAIDs provide analgesia and all are potentially nephrotoxic.

Commonly used NSAIDs include:

- Meloxicam 1.0–1.5 mg/kg IM or orally q12h.
- Carprofen 2–4 mg/kg orally q12h.
- Ketoprofen 2 mg/kg IM q8–24h.

Local anaesthesia

Local anaesthesia is seldom used in birds. However, local anaesthetics interrupt the transmission of pain impulses and, when used preoperatively, they can be used to block the site of tissue manipulation, thus helping to reduce central sensitization.

Cardiovascular side-effects appear to be rare at doses below 1–4 mg/kg. It may be necessary to dilute commercially available preparations in order to prevent overdosing the patient.

Although local anaesthesia reduces pain perception, it does nothing to protect the patient from stress. With many avian patients it may be better to induce general anaesthesia rather than stress the bird with restraint and local anaesthesia. However, the combination of general and local anaesthesia allows a lighter level of anaesthesia to be maintained.

ANAESTHESIA

What makes avian anaesthesia different?

There are four fundamental differences between avian and mammalian anaesthesia: the anatomy of the bird; its metabolism; its tendency to lose heat quickly; and finally, that most birds requiring anaesthesia are chronically ill.

Anatomy

Trachea

Birds have complete tracheal cartilage rings and the trachea is therefore not expandable. For this reason, only non-cuffed endotracheal tubes should be used. (If cuffed tubes are used, they must never be inflated.) Additionally, some species (notably waterfowl) have either very long, coiled tracheas or diverticula (bullae). These are anatomical adaptations allowing such birds to hold their breath under water. It also may make it difficult to mask induce these species, making intravenous or intramuscular induction advisable.

Voice production arises from the syrinx, the bifurcation of the trachea. This is located within the body and is a common site for obstructions (*Aspergillus* granulomas or inhaled bird seed).

Air sacs, non-expandable lungs and lack of a diaphragm

Respiration is achieved by movement of the sternum (up and down) and the ribs (in and out). If these movements are restricted, or if enlarged organs, fat or fluids within the body compress the air sacs, respiratory compromise can quickly develop. Therefore:

- Wherever possible place the bird in ventral or lateral recumbency. Birds in dorsal recumbency may require IPPV to properly oxygenate them.
- If any anaesthesia is likely to be prolonged more than 5–10 minutes, the patient should be intubated and ventilated, regardless of its positioning.
- Birds with ascites should be handled very carefully, and may need diuretic therapy or coeliocentesis before anaesthesia.

If the coelom is opened during surgery, the bird may be able to breathe *through* the coelom, via the air sacs. This may require increasing the oxygen flow rate and deepening the anaesthesia to overcome this. Catheterization of the caudal thoracic air sac (Chapter 4) can allow anaesthesia to be maintained

Downloaded From Vet-eBooks.com

without tracheal intubation, leaving the head free to work on.

Metabolism

The avian metabolic rate is much faster than that of mammals. For this reason, lengthy fasting is potentially dangerous, as hypoglycaemia may develop. Conversely, regurgitation and aspiration of crop and proventricular contents are very real risks. It is generally recommended to fast avian patients for 3–4 hours before anaesthesia. If necessary, crop contents can be removed by lavage prior to anaesthesia. Keeping the patient's head elevated above the level of the crop may help to prevent passive regurgitation and aspiration during anaesthesia. Blood glucose can be determined by the use of a glucometer and, if low, the administration of 5% glucose during anaesthesia can assist maintaining the patient's metabolic rate.

Heat loss

Birds begin to cool down within 20 minutes of induction. Profound hypothermia, resulting in the patient's death, can easily occur and may go unnoticed till a crisis develops. Various techniques have been developed to maintain body temperature including warmed intravenous fluids, heating pads, warmed anaesthetic gases and radiant heat. Undoubtedly, the best prevention is a well thought-out surgical plan and rapid surgical techniques.

Chronically ill patients

Most avian patients requiring anaesthesia will be chronically ill, although the masking phenomenon may mean this is not readily detected. However, the stress of physically restraining a bird to examine it may be far greater than the stress of an anaesthetic. The decision as to whether to anaesthetise a patient is ultimately the clinician's, and will be determined by the patient's physical condition and the experience of the clinician.

Procedures should be planned, endeavouring to do as much as possible in as short a period as possible. For example, with planning the patient can be anaesthetised, physically examined, radiographed, blood and culture swabs taken, fluids given and initial treatment started – all in less than ten minutes. This can be done with minimal stress to the patient and maximum return to the clinician.

The ideal requirements for avian anaesthetic agents and techniques are:

- Rapid induction and recovery in order to minimise stress and heat loss.
- Rapid metabolism and excretion, avoiding 'hangover' effects that may delay the bird's return to eating and its normal metabolic rate.
- Minimal cardiac and respiratory depressant effects, combined with good ventilation.
- Operator health and safety must remain an important consideration.

Inhalation anaesthetic agents

Halothane

Halothane manufacture is being scaled down and it is now unavailable in many countries. It is soluble in blood, fat and other soft tissues and therefore takes longer to reach effective concentration in the brain. This results in longer induction and recovery times than for isoflurane. It is excreted through the respiratory tract. Marked cardiac arrhythmias (ventricular tachycardia and ventricular fibrillation) frequently occur.

Halothane has a low anaesthetic index, meaning that higher concentrations are required to induce apnoea. Although at first this suggests greater safety, the effect is that the patient continues to breathe and therefore takes in more halothane. When combined with its solubility in the body and its cardiac effects, the result is that apnoea and cardiac arrest during deep halothane anaesthesia are often simultaneous.

Isoflurane

This is poorly soluble in blood, fat and other soft tissues, therefore induction and recovery times are rapid and controlling the depth of anaesthesia is easier to achieve. It is excreted through the respiratory tract. It does cause some cardiac arrhythmias, but not as severe or as life-threatening as those seen with halothane.

Isoflurane has a higher anaesthetic index than halothane, meaning that apnoea occurs much earlier in deep anaesthesia, preventing the spontaneous

Downloaded From Vet-eBooks.com

inhalation of more isoflurane. This lessens the probability of fatal cardiac arrhythmias developing, making it a safer anaesthetic agent.

Sevoflurane

This produces a more rapid induction and recovery than isoflurane, but not so much as to be clinically significant. It must run at a higher concentration than isoflurane to maintain surgical anaesthesia. Cardiac arrhythmias are not noted. The anaesthetic index is higher than for isoflurane, but again this is not of clinical significance. The higher concentrations required mean that sevoflurane is significantly more expensive than isoflurane, but there are only small clinical advantages.

Intravenous or intramuscular agents

Intravenous or intramuscular agents can be used, but it must be recognised that induction and recovery are variable. Recovery is often prolonged, and there can be a significant 'hang-over' effect, with the bird not regaining its normal metabolic rate and appetite for several hours or longer. It is therefore recommended that these agents only be used as an induction agent where mask induction is not feasible (e.g. waterfowl, large ratites) or in the field where gas anaesthesia may be unavailable. Clinicians must be aware of the hazards of using intravenous or intramuscular anaesthesia, and be prepared to deal with adverse effects and prolonged recovery times.

Ketamine

Ketamine alone is not recommended for anaesthesia. Although it does not appear to cause pulmonary depression or cardiovascular depression, ketamine (at anaesthetic doses) gives inadequate analgesia and muscle relaxation and is associated with spontaneous movements, muscular rigidity and violent recoveries. It is excreted through the kidneys and the recovery time is prolonged and variable. The dose is 20–50 mg/kg IM or IV.

Xylazine

When used alone, xylazine does not usually produce adequate surgical immobilization. It may be associated with bradycardia, hypotension, arrhythmias and respiratory depression. It frequently causes salivation, excitement, muscle tremors and convulsions, sometimes aggravated by noise. Recovery is prolonged, but xylazine can be reversed by yohimbine hydrochloride (0.11–0.27 mg/kg). The dose of xylazine is 1 mg/kg IM.

Xylazine–ketamine

This combination is commonly used for restraint and anaesthesia, but waterfowl (especially ducks and Canada geese), owls and accipiters do not respond well. When used together the combination is synergistic, giving a smooth induction and recovery with improved muscle relaxation and possibly enhanced analgesia. The combination can be associated with cardiac arrhythmias and hypotension. The dose is: ketamine 5–20 mg/kg, xylazine 0.5–2.0 mg/kg, IM or IV.

Diazepam

By itself, diazepam is used for sedation or seizure control. The dose is 0.05–0.15 mg/kg slowly IV, or 0.2–0.5 mg/kg IM.

Diazepam–ketamine

This combination can be used as an intravenous induction agent in large ratites, raptors and waterfowl, but it may not be a reliable maintenance agent. It can be used intramuscularly in parrots and pigeons (with a slow induction and recovery time). The dose is: diazepam 0.2–2 mg/kg, ketamine 10–40 mg/kg.

Midazolam

This can be used as a sedative and anxiolytic, or as a premedication for anaesthesia. At sedative doses it has minimal effects on blood pressure, heart rate and body temperature. The dose is 0.8–3 mg/kg IM or IV. It can be given intra-nasally for sedation at dose rates of 1–5 mg/kg.

Midazolam–ketamine

The dose for this combination is: midazolam 0.2–4 mg/kg, ketamine 10–40 mg/kg, IM.

Medetomidine–ketamine

This is a useful combination for intravenous induction of waterfowl, giving rapid induction and recovery. It is reversible with atipamezole.

Downloaded From Vet-eBooks.com

The dose is: medetomidine 60–85 µg/kg; ketamine 1.5–2.0 mg/kg IV.

Zolazepam–tiletamine

This is not recommended in birds due to violent recoveries.

Propofol

This can be used as an intravenous induction agent following sedation with medetomidine–ketamine. Apnoea is common following intravenous induction. The dose is 1–5 mg/kg IV. Intubation and IPPV are strongly recommended.

Anaesthetic technique

The anaesthetic technique should be tailored to each patient and situation. However, the technique can be broadly divided into:

- Patient preparation.
- Induction.
- Maintenance.
- Recovery.

Patient preparation

It is important than an accurate weight of the patient is obtained so that accurate calculation of drug doses can be made. Parrots should be fasted for no more than 3–4 hours. Larger birds, especially raptors, may require periods of fasting up to 12 hours. The patient's respiration is assessed by distant examination, looking for mouth breathing, tail bobbing, bilateral wing drooping, and exaggerated sternal lift as evidence of dyspnoea. If dyspnoea is present, this must be factored into the anaesthetic plan, or the anaesthesia postponed.

The patient's crop and coelom should be palpated. If the crop is full, it should be emptied by crop gavage. If there is coelomic distension it must be determined if there is ascites present (e.g. by ultrasound). If so, pre-anaesthetic coeliocentesis may be advantageous.

PCV, total plasma protein and blood glucose should be measured as minimal pre-anaesthetic testing.

If painful procedures are anticipated, pre-emptive analgesia (see above) should be given 20–60 minutes before the procedure commences.

Induction

Parrots and other small birds: Where appropriate or necessary, the patient should be wrapped loosely in a towel to prevent wing flapping and excessive struggling. Pre-oxygenation via a face mask for 1–2 minutes before induction can increase safety if there is a period of apnoea during induction. Mask induction is usually carried out with 5% isoflurane; induction usually takes approximately 20–30 seconds, after which time the anaesthetic setting can be reduced to 2–3% (**Fig. 28.2a**).

Waterfowl and other large birds: An intravenous catheter is placed in an accessible vein (e.g. the medial tarsal vein in waterfowl, the basilic vein in ratites). Induction is with intravenous drug(s) as described above.

Maintenance

For short procedures (e.g. radiography) maintenance of anaesthesia via a face mask may be all that is required. The mouth and nares should be encased in the mask, which should be sealed around the head to prevent the escape of anaesthetic gas and to assist in ventilation if required. For small patients this can be achieved by stretching a rubber glove over the mask and making a small hole for the patient's head. It is important to keep the patient's head elevated above the level of the crop throughout the procedure to prevent passive regurgitation and aspiration of crop or proventricular contents.

For procedures longer than five minutes, for compromised patients or for rare or valuable birds, intubation is advisable (**Fig. 28.2b**). It should be remembered that the avian trachea is composed of complete cartilage rings and the lumen diameter decreases caudal to the glottis (**Fig. 28.3**). Therefore, endotracheal tubes should be:

- Non-cuffed (or, if cuffed, never inflated).
- Relatively shorter than those used in mammals.
- Smaller diameter than the width of the glottis would suggest.

In some birds dripping lignocaine onto the glottis to prevent laryngospasm can make the process much easier; care needs to be taken to avoid overdosing the bird with local anaesthesia. Once intubated,

Downloaded From Vet-eBooks.com

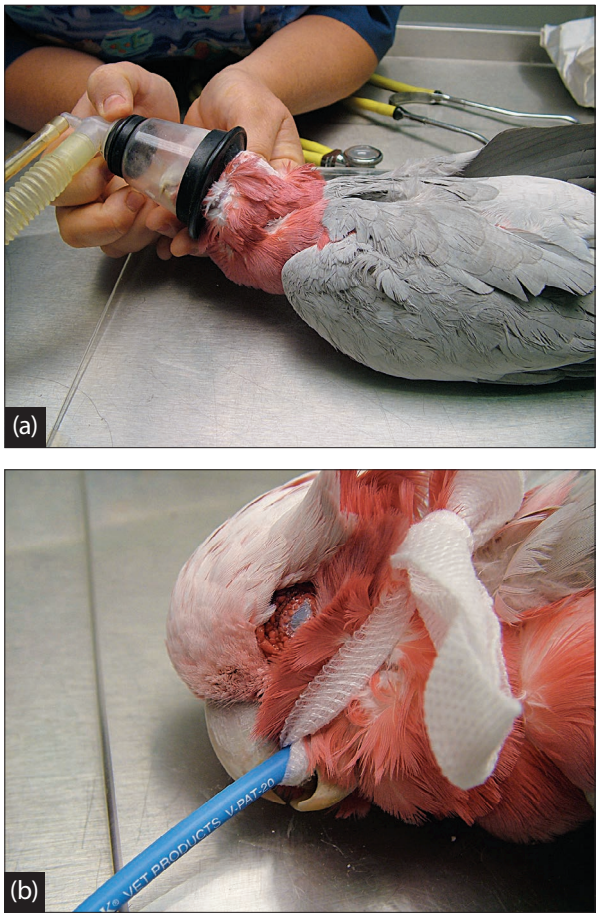


Figure 28.2 **Galah undergoing anaesthesia using mask induction (a). Note this procedure is suitable for most companion birds. Once anaesthetised, the galah was intubated (b). Note that for anything other than very short procedures this is recommended practice.**

ventroflexion of the neck must be avoided. This can bring the tip of the endotracheal tube into contact with the tracheal mucosa, causing pressure necrosis.

If the patient's condition precludes intubation (e.g. tracheal obstructions), an air sac catheter should be placed as described in Chapter 4, Clinical Techniques and illustrated in **Fig. 4.12** p. 91.

Once a stable plane of anaesthesia has been reached, the isoflurane concentration can be reduced, usually to 1.5–3%. Oxygen flow rates are typically high, usually 1–3 litres/minute even for small patients.

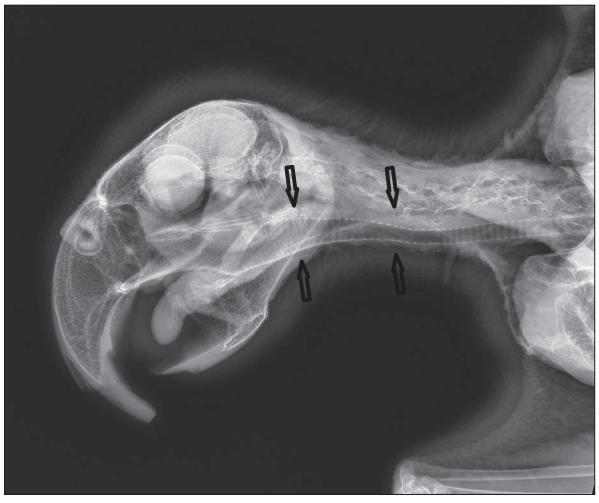


Figure 28.3 **Radiograph (lateral view) of a macaw shows how the trachea (highlighted) narrows as it progresses caudally. (Photo courtesy L Nemetz)**

The patient should, wherever possible, be maintained in lateral or ventral recumbency. When placed in dorsal recumbency the weight of the viscera can compromise the ability of the air sacs to function correctly. Sternal lift, necessary to expand the air sacs, also becomes more difficult for the patient in dorsal recumbency. Again, the patient's head should be elevated to prevent reflux of crop and proventricular contents and potential aspiration. This is easily achieved by placing a pad under the head to lift it above the level of the crop.

It should be noted that if a surgical procedure is being performed that opens an air sac to room air, spontaneous ventilation through the open air sac will result in a lightening of the anaesthetic depth. This may require an increase in both the oxygen flow rate and the level of anaesthesia to maintain a surgical plane of anaesthesia.

Support

Body temperature: Birds have a high surface to body volume ratio and will lose body heat quickly when not active or when their metabolic rate slows. The rate and amount of heat loss is directly proportional to the size of the patient (the smaller the bird, the faster and greater the heat loss). Numerous techniques have been devised to prevent hypothermia in anaesthetised patients and are listed below. It must be noted, though,

Downloaded From Vet-eBooks.com

that regardless of the thermal support provided, the patient's body temperature will start to decline after approximately 20 minutes of anaesthesia (although aggressive patient warming as described below will result in a slower rate of heat loss). One of the most important means of minimizing this heat loss is to have a plan and preparations in place before inducing anaesthesia in order to minimise the anaesthetic time. Other techniques that have been employed include:

- Warmed intravenous fluids throughout the procedure.
- Warmed and humidified anaesthetic gases.
- Radiant heat from overhead heat lamps.
- Heated air units (e.g. the Bair Hugger®).

Heat pads do not appear to be overly effective in providing heat support to avian patients and may lead to thermal burn injuries.

Circulatory support: Anaesthesia, especially when associated with surgical blood loss and/or pre-existing dehydration, can lead to hypotension and decreased vascular perfusion. Warmed fluids given subcutaneously, intravenously or intra-osseously will provide both circulatory and thermal support. Balanced electrolyte solutions such as lactated Ringer's solution, given at a rate of 10 ml/kg/hr and even up to 30–60 ml/kg/hr, appear to be safe and effective in most birds (**Fig. 28.4**).



Figure 28.4 **Cockatoo undergoing surgery receives intravenous fluids via the medial metatarsal vein.**

Birds with pre-existing anaemia or coagulopathies, or those likely to experience excessive blood loss during surgery, will benefit from a fresh whole blood transfusion (see Chapter 8, Supportive Therapy).

Respiratory support: With prolonged anaesthesia (especially in patients that develop hypothermia, hypoglycaemia or hypovolaemia), the patient's respiratory rate may slow and even stop. Cardiac arrest usually follows soon after. Adequate respiratory support, in the form of supplemental ventilation, is therefore essential for most avian anaesthetics. IPPV, performed either manually or by a ventilator, should be instituted early in the anaesthetic process rather than waiting for problems to develop (**Fig. 28.5**). Birds that are breathing by themselves while under anaesthesia will benefit from IPPV at a rate of 4–6 respirations per minute, with sufficient pressure to create normal inspiratory depth. (Excessive pressure should be avoided.) If the bird is apnoeic, IPPV should be given at a rate of 10–12 respirations per minute.

Monitoring

Monitoring of anaesthesia is vital in avian surgery (**Fig. 28.6**). For this reason all anaesthetic procedures should be monitored by a staff member assigned to this task only. Safe and effective



Figure 28.5 **Example of a commercially available respiratory ventilator. Note that these are suitable for small patients and can ease the anaesthetist's work load by ensuring that the patient is well ventilated without resorting to manual IPPV.**

Downloaded From Vet-eBooks.com



Figure 28.6 **Cockatiel about to undergo surgery is intubated, receiving intra-osseous fluids, and is been monitored with a Doppler, capnograph and pulse oximetry.**



Figure 28.7 **An anaesthetised African Grey undergoing surgery should be monitored via auscultation throughout. Note that this is an important monitoring tool.**

anaesthetic monitoring is a multimodal procedure, relying on visual assessment as well as monitoring equipment.

Manual assessment of the depth of anaesthesia requires monitoring of:

- Respiratory rate and character are the most reliable indicators of anaesthesia depth in birds. The anaesthetised bird should breathe at least once every 2–7 seconds. At a light plane of anaesthesia the respiratory rate and character can be rapid and deep. As the anaesthetic plane deepens, the respiration slows and becomes shallower. As oxygen exchange only occurs when there is movement of air through the respiratory tract, periods of apnoea can result in cardiac arrest much sooner than that seen in dogs and cats.
- Heart rate and rhythm. Decreases or changes in heart rate and rhythm are often mirrored and preceded by changes in respiration. Auscultation should be performed frequently, if not continuously (**Fig. 28.7**). The use of a Doppler probe placed over the superficial ulnar artery (on the medial surface of the antebrachium or elbow) allows constant monitoring of the heart rate and rhythm. A pulse is usually easily detected over this artery with fingertip palpation.
- The palpebral reflex (eyelid closure induced by stimulation of the ocular medial canthus or cere) disappears quickly as the anaesthetic plane deepens.
- The pedal (toe withdrawal) reflex and the corneal reflex (third eyelid movement in response to corneal manipulation) slow, but can remain present even at surgical planes of anaesthesia.

Capnography is becoming recognised as an essential means of monitoring avian anaesthesia. By measuring the concentration of expired CO_2 in each expiration, the capnometer produces a graph (capnogram) (**Fig. 28.8**) that characterises the elimination of CO_2 by the lungs. The capnogram shows the rate and depth of respiration which often may change before changes in the heart rate and rhythm. Capnometers can be either mainstream (attached to the end of the endotracheal tube) or sidestream (drawing expired air from the endotracheal tube or adaptor). Mainstream capnometers may add to the dead space in the breathing circuit, potentially a major issue in small birds. However sidestream capnometers that draw off a large amount of expired gas may exceed the respiratory volume of small birds. Microstream (50 mL/min) sidestream capnometers may be the most practical units to use in avian medicine. End tidal CO_2 ($ETCO_2$) in anaesthetised birds should lie between 30 and 45 mmHg

Downloaded From Vet-eBooks.com

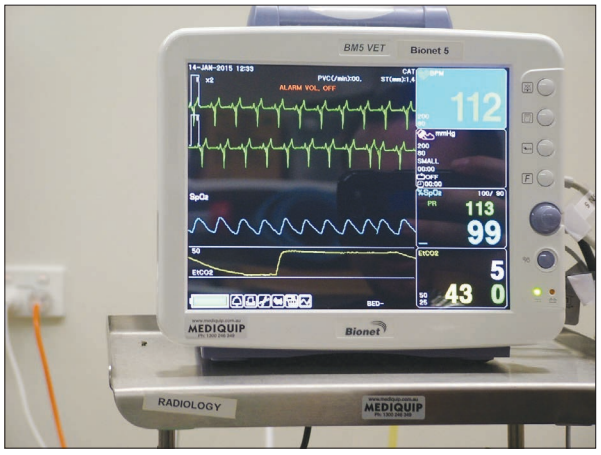


Figure 28.8 **A patient's rate and depth of respiration are monitored continuously during surgery along with heart rate and rhythm. (The ECG trace is in green, at the top. Pulse oximetry is the blue trace in the middle, while the capnograph is in yellow at the bottom.) Note that capnography is now a commonly used monitoring tool in avian surgery and electrocardiography is occasionally employed, especially for larger birds.**



Figure 28.9 **King parrot being prepared for surgery has their indirect blood pressure monitored using Doppler ultrasound. Note that the pressure cuff has been wrapped around the humerus and the crystal over the ventral aspect of the radius and ulna.**

which correlates to a $PaCO_2$ of 25–40 mmHg. Rising levels of $ETCO_2$ reflect hypoventilation and decreasing blood pH, resulting in respiratory acidosis and adverse cardiac effects.

Pulse oximetry is often considered to be unsuitable in birds because of the lack of calibration for avian blood. It may, however, provide some information on trends in blood oxygenation in avian patients and should not be discounted completely. By combining pulse oximetry (measuring oxygenation) and capnography (assessing ventilation), trends in the respiratory function of an anaesthetised patient can be closely monitored (**Fig. 28.8**).

Blood pressure monitoring is becoming increasingly recognised as a valuable tool in anaesthetic monitoring. Although direct arterial pressure measurement is ideal, it is usually not practical in private practice. Indirect blood pressure measurement techniques, based on the detection of blood flow beneath an inflated cuff, are therefore recommended (**Fig. 28.9**). Systolic blood pressure determination via ultrasonic Doppler flow detection has been found to correlate well with direct blood pressure. The blood pressure of various avian species under gaseous anaesthesia lies between 90 and 140 mmHg systolic (See Chapter 4, Clinical Techniques pp. 90–91).

Electrocardiography (**Fig. 28.8**) is occasionally employed (especially in larger birds) and is a useful tool in monitoring the heart rate and rhythm, especially when auscultation or Doppler probe placement are difficult to perform without interfering with the surgery. Electrodes should be placed quickly after induction of anaesthesia and the ECG trace monitored (see Chapter 20, Disorders of the Cardiovascular System for details on recording and interpreting ECGs).

Recovery

Depending on the type and duration of the anaesthetic, recovery to standing and perching usually takes 5–10 minutes. Full recovery usually takes 30–60 minutes.

Following anaesthesia, birds should be:

- Lightly wrapped in a towel to prevent injury from wing flapping or excessive movement during recovery.
- Placed in a darkened, quiet, heated cage and monitored closely until able to perch.

Downloaded From Vet-eBooks.com

- Extubated only when voluntary head movements are noted.
- Offered food and water as soon as the patient is fully recovered.
- Monitored for bleeding, regurgitation, dyspnoea.

Anaesthetic emergencies

Careful monitoring of respiration, heart rate and blood pressure will usually alert the avian anaesthetist to impending critical emergencies. Cardiac arrest, in many cases, is irreversible and fatal in the anaesthetised bird. However, prompt recognition and treatment of abnormalities can prevent this cardiac arrest in many cases.

Early signs of impending disaster include:

- Changes in respiratory rate and character.
- Changes in cardiac rate and rhythm.
- Hypotension.

Changes in respiratory rate and rhythm

The unique anatomy of the avian respiratory system contributes to their anaesthetic risk. Birds do not have substantial functional residual capacity (the quantity of air in the lung that is available for gas exchange at the end of expiration); 90% of the avian respiratory tract is composed of dead space (trachea, bronchi, and air sacs), and only 10% of the total intrapulmonary gas volume (located in the parabronchi and air capillaries) participates in gas exchange at any point in time. (In contrast, in many mammals only 4% of the total lung volume is dead space, and 96% of the intrapulmonary gas volume, located in alveoli, is available for gas exchange.) Birds compensate for the lack of functional residual capacity by moving air uni-directionally through their parabronchi during inspiration and expiration. Without proper airflow through the parabronchi (e.g. during apnoea or hypoventilation), efficient gas exchange cannot take place, and the physiologic effects of hypoventilation (hypoxaemia, hypercapnia) develop rapidly.

Tachypnoea (increased respiratory rate) can be due to:

- Lightening of the anaesthetic depth.
- Pain perception in the lightly anaesthetised patient.
- Pulmonary haemorrhage.
- Hyperthermia.

Dyspnoea (difficult or laboured breathing) can be due to:

- Airway obstruction (e.g. mucus plugs in the trachea or endotracheal tube).
- Poor circulatory perfusion due to heart disease, anaemia or haemorrhage.
- Restriction of body movement by internal organ enlargement, improper restraint or positioning during the anaesthetic.
- Pulmonary haemorrhage or oedema.
- Pre-existing respiratory disease.

Apnoea (complete cessation of breathing), usually preceded by a slowing of the respiratory rate, can be due to:

- Prolonged or very deep anaesthesia.
- Hypothermia.
- Inadequate ventilation.
- Hypovolaemia.

Management

Upon identifying an unexpected change in the patient's respiratory rate and rhythm, the anaesthetist should firstly check that the anaesthetic machine is functioning correctly and at what concentration the anaesthetic is being delivered. Connections to the patient should be checked to confirm that they are, in fact, connected and patent. The anaesthetist must alert the surgeon to the situation and ask if air sacs have been opened or if there is significant haemorrhage visible.

The patient should be assessed via auscultation (of the heart, lungs and trachea) and monitoring devices such as capnography, Doppler, blood pressure and ECG.

If tachypnoea is due to a lightening of the anaesthetic plane or perception of pain, the anaesthetic depth should be increased. If air sacs have been opened, the surgeon may have to temporarily 'close' the surgical site until a deeper plane of anaesthesia has been reached.

Downloaded From Vet-eBooks.com

Tachypnoea or dyspnoea due to pulmonary haemorrhage or oedema does not respond to changes in anaesthetic depth or other treatments. Consideration should be given to terminating the procedure and recovering the patient. If dyspnoea is believed to be due to a mucus plug in the endotracheal tube, the tube should be removed and replaced. If the plug is in the trachea, placing an air sac catheter may be necessary.

Apnoea or a slowing of the respiratory rate should be treated by turning the anaesthetic down (or off), leaving the oxygen on and commencing IPPV at 10–12 breaths per minute. If the patient is not intubated, this should be performed where possible, and as quickly as possible. Gentle movement of the sternum ventrally and dorsally may assist ventilation if intubation is not possible, or until it can be performed.

If hypovolaemia is suspected, fluid rates should be increased.

Changes in cardiac rate and rhythm

Changes in cardiac rate and rhythm are usually preceded by changes in rate and depth of respiration. Tachycardia usually occurs as a result of decreased anaesthetic depth.

Bradycardia should be treated similarly to apnoea (i.e. the anaesthetic is turned down or off, IPPV is begun or increased, and fluid rates are increased). Sternal compression may assist circulation and ventilation. The sternum makes direct cardiac massage difficult, if not impossible. Atropine or glycopyrrolate can be given intravenously, as can epinephrine (1:1,000) at a rate of 0.5–1.0 ml/kg IM, IV, intra-osseously or intra-tracheally.

Once cardiac arrest has occurred, resuscitation is unlikely to be successful.

Hypotension

Hypotension occurs after prolonged or deep anaesthesia or severe blood loss. A slow bolus of IV fluids followed by increasing the fluid rate, lightening the anaesthetic depth, and commencing IPPV should be implemented once a significant drop in blood pressure is noted.

FURTHER READING

Acierno MJ, Smith J, Tully TN, Migallon-Guzman D, Mitchell MA (2007) Evaluation of indirect blood pressure measurement techniques (Doppler) and the comparing point of care blood gas analyzer (I-STAT) values. In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp. 15–16.

Acierno MJ, da Cunha A, Smith J, Tully TN, Migallon-Guzman D, Serra V, Mitchell MA (2008) Agreement between direct and indirect blood pressure measurements obtained from anesthetized Hispaniolan Amazon parrots. *Journal of the American Veterinary Medical Association* **233**(**10**):1587–1990.

Boedeker NC, Carpenter JW, Mason DE (2005) Comparison of body temperatures of pigeons (*Columba livia*) anesthetized by three different anesthetic delivery systems. *Journal of Avian Medicine and Surgery* **19(1)**:1–6.

De Matos REC, Morrisey JK, Steffey M (2006) Postintubation tracheal stenosis in a Blue and Gold macaw (*Ara ararauna*) resolved with tracheal resection and anastomosis. *Journal of Avian Medicine and Surgery* **20**(**3**):167–174.

Edling, T. M, Degernes, L. A, Flammer, K, Horne, W. A. (2001) Capnographic monitoring of anesthetized African grey parrots receiving intermittent positive pressure ventilation. *Journal of the American Veterinary Medical Association* **219**(**12**):1714–1718.

Edling TM (2006) Updates in anesthesia and monitoring. In: *Clinical Avian Medicine*, *Vol 2*. GJ Harrison, TL Lightfoot (eds). Spix Publishing, Palm Beach, pp. 747–760.

Heard DJ (1997) Anesthesia and analgesia. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 807–828.

Klaphake E, Schumacher J, Greenacre C, Jones MP, Zagaya N (2006) Comparative anesthetic and cardiopulmonary effects of pre- versus postoperative butorphanol administration in Hispaniolan Amazon parrots (*Amazona ventralis*) anesthetised with sevoflurane. *Journal of Avian Medicine and Surgery* **20**(**1**):2–7.

Lichtenberger M (2005) Determination of indirect blood pressure in the companion bird. *Seminars in Avian Exotic Pet Medicine* **14**(**2**):149–152.

Lumeij JT, Deenik JW (2003) Medetomidine-ketamine and diazepam-ketamine anesthesia in racing pigeons (*Columba livia domestica*): a comparative study. *Journal of Avian Medicine and Surgery* **17**(**4**): 191–196.

Downloaded From Vet-eBooks.com

Machin KL (2005) Avian pain: physiology and evaluation. *Compendium on Continuing Education for the Practicing Veterinarian* **27**(**2**):98–109.

Paul-Murphy J (2006) Pain management. In: *Clinical Avian Medicine*, *Vol 1*. GJ Harrison, TL Lightfoot (eds). Spix Publishing, Palm Beach, pp. 233–240.

Paul-Murphy J, Hess JC, Fialkowski JP (2004) Pharmacokinetic properties of a single intramuscular dose of buprenorphine in African Grey parrots (*Psittacus erithacus erithacus*). *Journal of Avian Medicine and Surgery* **18**(**4**):224–228.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 29

SURGERY

INTRODUCTION

An understanding of surgical principles is necessary before operating on a bird. Even though there are many anatomical and physiological differences between birds and mammals, surgical principles and techniques remain similar. As discussed in Chapter 28, Analgesia and Anaesthesia, birds are prone to anaesthetic complications associated with pain perception, hypothermia, hypovolaemia and hypoxia. These anaesthetic complications are closely linked with surgical principles. In order to maximise surgical success, the following principles must be understood and applied:

- Minimise haemorrhage.
- Minimise tissue trauma.
- Minimise anaesthetic time.
- Minimise anaesthetic and metabolic complications.
- Provide postsurgical support and analgesia.

To comply with these principles, the surgeon should:

- Ensure that the patient is in the best possible condition prior to surgery.
- Develop an analgesia and anaesthetic plan that maximises patient safety and comfort (see Chapter 28, Analgesia and Anaesthesia).
- Plan the procedure so as to minimise anaesthetic and surgical time.
- Ensure that surgical preparation and patient support procedures are in place to maximise patient safety.
- Use instruments, techniques and suture materials that minimise tissue damage, blood loss and inflammatory responses.

PRE-SURGICAL ASSESSMENT AND CONDITIONING

This should take the form of a thorough physical examination and diagnostic testing.

Physical examination

This should include a comprehensive physical examination, weighing and respiratory recovery time (a healthy patient should return to normal respirations within 3–5 minutes following capture).

Diagnostics

- Haematology and biochemistries: ideally pre-operative PCV, total plasma protein, and blood glucose should be measured for most routine surgical procedures. If patients are obviously unwell, a more complete haematological and biochemical profile is indicated.
- Clotting time (crude estimate): a pin prick of the basilic vein should clot after one minute of direct pressure.
- Imaging such as radiology and ultrasound can be utilised to detect problems such as ascites and organomegaly.

Medical conditions that may necessitate postponement of surgery include respiratory distress, obesity and concurrent disease problems unrelated to the surgical condition (e.g. cardiovascular disease, infectious conditions). Clinical pathology parameters that may necessitate postponing surgery include:

- PCV <20% or >60%.
- Total plasma protein <20 g/l.
- Glucose <11 mmol/l.

Downloaded From Vet-eBooks.com

- Uric acid >700 µmol/l.
- AST >650 IU/l (in the presence of a normal CK).
- Cholesterol >18 mmol/l.

Conditioning the patient

If any of the abnormalities listed above are detected, appropriate medical therapy should be instituted (e.g. fluid therapy, blood transfusion, tube feeding, diet conversion, or weight reduction). Vitamin supplementation may be of benefit in mildly malnourished birds, but should be given several days in advance of the surgery to be of benefit. If a coagulopathy is suspected, vitamin K1 may be of benefit, but it must be administered at least 24 hours prior to the surgery.

If the patient's medical condition is critical and surgery cannot be postponed, the owner must be informed of the increased risks prior to commencing surgery.

Minimising anaesthetic time

A surgical plan should be developed. Although exploratory surgery is performed in birds, surgical time can be reduced if a plan has been developed by the surgeon. This should include:

- Revision of the appropriate anatomy if necessary.
- Determination of the extent of the lesion or disease process prior to the procedure. This can be achieved through careful physical examination, radiography and endoscopy.
- Discussion with theatre and nursing staff about the procedure, so that all personnel involved know what is happening and what their role is to be.

Prior to anaesthetising the patient the surgeon should check that all equipment and materials likely to be used in the procedure are accessible and fully functional. The author finds that a verbal rehearsal with all involved personnel at this stage detects and corrects preparation errors that could have resulted in a delay during surgery.

SURGICAL PREPARATION AND PATIENT SUPPORT PROCEDURES

Preparation of the surgical site

Feathers should be plucked; they should not be cut if at all possible. This encourages new feather growth sooner than waiting for cut feathers to moult. Soaking the feathers to be removed with the skin preparation solution prior to plucking will minimise the amount of loose feathers that can potentially contaminate the surgical site. Feather removal can be minimised by using either water-soluble lubricating gel or adhesive tape to hold back surrounding feathers.

Chlorhexidine or povidone; iodine can be used to prepare the surgical site. Alcohol-based disinfectants should not be used, as evaporative heat loss can be significant.

Draping the surgical site

Transparent drapes allow visualization of the patient and have heat retaining characteristics (**Fig. 29.1**). If adhesive drapes are not used, towel clamps may be placed around large feathers instead of through the skin.

Patient support

The provision of supportive measures, as discussed in Chapter 28, Analgesia and Anaesthesia, should be factored into the surgical plan. These measures include:

- Thermal support.
- Circulatory support.
- Respiratory support.

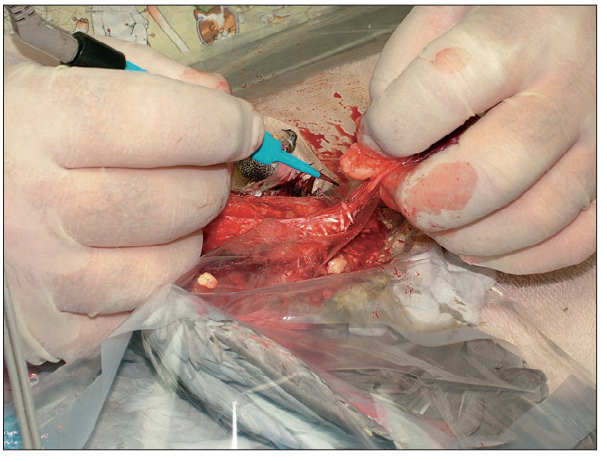


Figure 29.1 **Galah undergoing surgery. Note the use of sterile plastic drapes which limit contamination of the surgical site.**

Downloaded From Vet-eBooks.com

Minimising tissue trauma and blood loss

Instrumentation

Microsurgical and ophthalmic instruments allow for delicate and atraumatic handling of tissue (**Fig. 29.2**). Magnification, using either binocular loupes or an operating microscope, allows for much finer work than can be achieved with the naked eye. Most binocular loupes and operating microscopes come with in-built illumination that complements and enhances the magnification achieved.

Haemostasis can be achieved through the use of laser or radiosurgery (see below) (**Fig. 29.3**) and vascular clips (e.g. Hemoclip®, Weck). Sterilised cotton buds or swabs are useful for swabbing blood and other fluids, as well as gently manipulating tissues. They can also be used for gentle blunt dissection (**Figs 29.4a** and **b**).

Radiosurgery

High-frequency radiowaves are modified by a waveform adaptor and then focused from the active electrode tip to the indifferent plate (antenna). These radiowaves produce alternating electromagnetic fields that generate heat due to resistance in the tissue. This localised heat volatilises intracellular fluids, causing cell disruption along the path of the electrode (**Fig. 29.2**). A frequency of 3.8–4.0 MHz



Figure 29.2 Appropriate surgical kit for avian surgery. Note that the kit includes ophthalmic instruments and wooden cotton applicators.

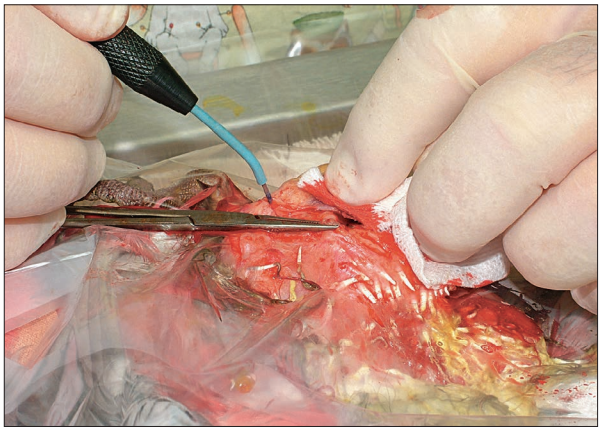


Figure 29.3 Use of radiosurgery (as shown) during a lipoma removal minimises blood loss.





Figure 29.4 Sterilised wooden cotton applicators can be used to apply pressure for haemostasis and to swab a surgical site (a) and also for blunt dissection (b).

Downloaded From Vet-eBooks.com

has been found to be optimal for tissue incision, as it focuses the energy into a minimal area.

The waveform can be:

- Fully rectified, fully filtered: pure cutting, suitable for biopsies.
- Partially rectified: coagulation, no cutting.
- Fully rectified, unfiltered: 50% cutting, 50% coagulation.
- Fulguration: destruction of tissue.

Tissue damage (lateral heat) is determined by:

- Electrode size: the greater the size, the more lateral heat and therefore the more tissue damage.
- The time the tissue is exposed to the electrode: the longer the time, the more lateral heat is created.

Radiosurgery therefore offers both haemostasis and minimal tissue damage when used correctly.

Carbon dioxide and diode laser surgery

This is slowly replacing radiosurgery. The light emitted from a laser has a wavelength of light that is absorbed by fluid in the cells, thus creating a thermal effect. The degree of penetration can be controlled, allowing for fine, controlled dissection of tissue. Advantages include:

- Haemostasis. The laser cuts and coagulates blood vessels up to about 0.5 mm in diameter. Larger vessels may have to be cauterised or ligated.
- Analgesia. As the laser cuts, it seals nerve endings and axons, reducing the sensation and transmission of pain.
- Decreased post-operative swelling. The laser seals lymphatic vessels, decreasing the extravasation of lymphatic fluid into the surgical site post-operatively.

Endosurgery

- Endoscopy has traditionally been used as a diagnostic tool in avian medicine to examine and biopsy internal organs. Recent developments have seen it used as a surgical tool, including:
  - Triangulation of various instruments using multiple ports or entry points.
  - The use of radiosurgery and/or laser surgery to ablate lesions and provide haemostasis.
  - The combination of these techniques to provide haemostasis during surgical procedures.

Advantages include:

- The combination of both diagnosis and treatment at the same time.
- Reduced surgical stress, pain and time.
- Decreased risk of infection.
- Increased anaesthetic safety.
- Faster post-operative recovery.

Suture choice

Desirable characteristics of suture material include: high tensile strength while the tissues heal; good knot security; resistance to infection; and minimal inflammatory, immunogenic, or carcinogenic reactions. A wide selection of suture materials is available to the surgeon, classified on their absorption characteristics and whether they are monofilament or braided. It is incumbent on the surgeon to understand how long different tissues take to heal, how long different sutures maintain their tensile strength, and how long before the suture is absorbed. This then affects the selection of suture material for various procedures.

Absorbable sutures of biological origin are broken down by phagocytosis, provoking a marked inflammatory response. Chromic catgut is the classic example of a biologically derived suture material. It rapidly loses its tensile strength and is quickly absorbed.

Synthetic polymers are absorbed by hydrolysis. Hydrolysis involves breaking down polymers into monomers by direct water cleavage; once broken down the monomers are then absorbed and metabolised by the body. The speed at which this occurs, and the time to complete resorption, is determined by the composition of the monomers and polymers in the material.

The characteristics of various suture materials are summarised in *Table 29.1*. When these characteristics

Downloaded From Vet-eBooks.com

Table 29.1 **Suture characteristics**

SUTURE	TYPE	>50% TENSILE STRENGTH RETAINED	TIME FOR COMPLETE RESORPTION	COMMENT
Chromic catgut	Biological monofilament	<7 days	60–90 days	• Poor knot security • Intense inflammatory reaction • Rapid loss of strength
Polyglactin 910 (Vicryl)	Synthetic braided	14–21 days	90–110 days	• increased tissue drag • Moderate inflammatory reaction • Can act as a wick for ascending infections
Polyglycolic acid (Dexon)		7–14 days	60 days	
Polydioxanone (PDS)	Synthetic monofilament	21–28 days	180–210 days	• Stiff • tendency to kink
Polyglyconate (Maxon)	Synthetic monofilament	21–28 days	180 days	• High degree of tensile memory (coil) when removed from package
Glycomer 631 (Biosyn)	Synthetic monofilament	14–21 days	90–110 days	• High degree of tensile memory (coil) when removed from package
Poliglecaprone 25 (Monocryl)		7–14 days		

are taken into account, PDS emerges as the suture material of choice for many procedures, especially for the closure of muscle and other fascia. For gastrointestinal surgery (where tissue healing times are faster) Vicryl®, Biosyn® and Monocryl® are also suitable choices.

COMMON SOFT TISSUE SURGICAL PROCEDURES

Ingluviotomy

Indication

Ingluviotomy is indicated for retrieval of foreign objects, endoscopic access to the proventriculus, placement of feeding tubes and biopsy.

Technique

The bird is positioned in dorsal/lateral recumbency, with the head elevated. The bird should be intubated. The skin is incised over the left lateral wall of the crop, close to the thoracic inlet. The skin edges are gently and bluntly dissected free of the underlying crop. A relatively avascular area is selected for an incision into the crop. The crop mucosa will often evert when the crop is opened. Care must be taken to prevent contamination of the subcutaneous tissues with any crop contents. Stay sutures may be required in some cases.

Closure

The crop is closed with two layers of 4–0 or 6–0 PDS in an inverting pattern. If unsure if a good seal has been obtained, pass a feeding tube through the mouth into the crop and gently fill the crop with saline. If there is no leakage, the saline and the tube can be withdrawn prior to recovery. Flush the subcutaneous tissues thoroughly with sterile saline to remove any crop contents inadvertently spilled during the surgery, and close the skin separately in either a continuous or interrupted pattern.

Post-operative care

Oral intake should be restricted for 6–12 hours, and then reintroduced in a controlled manner to prevent over stretching of the crop and placing tension on the suture line. After three days, if there have been no complications, normal feeding and drinking can be resumed.

Downloaded From Vet-eBooks.com

Crop fistula repair

Indications

Crop fistulae occur after crop burn due to microwaved or excessively heated and/or inadequately mixed hand-rearing food, or ingestion of caustic substances (**Fig. 29.5a**). (See also **Fig. 16.3**, Chapter 16, Disorders of the Gastrointestinal Tract, p. 232.)

Clinical presentation

Signs include slow crop emptying and erythema or blanching over the crop. In most cases it will be 3–5 days before the delineation between healthy and devitalised tissues becomes apparent, and it may take as long as 7–14 days (**Fig. 29.5b**). At this stage the fistula forms and food leaks out.

Technique

Analgesia, nutritional support and antibiotics/antifungal medications should be provided as appropriate until the demarcation between live and dead tissue is apparent.

The patient is prepared and positioned as described above for an ingluviotomy. Once the fistula has formed, the crop mucosa will have adhered to skin, forming a raised rim of granulation and fibrous tissue around the fistula. The skin is incised around the edges of this rim and the incision is continued for a short distance both cranial and caudal to the fistula. The skin edges are bluntly dissected off the crop. The fistula is excised completely, leaving fresh crop edges (**Figs 29.5c** and **d**). The crop and skin are then closed as for an ingluviotomy (**Fig. 29.5e**).

Post-operative care

Post-operative care is as described for an ingluviotomy.

Left lateral coeliotomy

Indications

This procedure provides access to the gonads, left kidney, oviduct, proventriculus and ventriculus.

Technique

The bird is placed in right lateral recumbency, with the cranial end of the body elevated 30–40° to prevent fluid entering the lungs. The wings are extended dorsally and secured into place. The left leg is abducted and drawn slightly forwards. The inguinal skin web is incised between the coelomic wall and the left leg and the leg is abducted further. This incision is continued from the sixth rib to the level of the left pubic bone (**Fig. 29.6**).

The superficial medial femoral artery and vein are cauterised where they transverse (in a dorsoventral direction) the lateral coelomic wall medial to the coxofemoral joint. The muscles (external, internal coelomic oblique and transverse coelomic muscles) are tented up and a stab incision is made with pointed scissors while protecting the viscera. This incision is extended from the pubic bone to the eighth rib. This will require transecting the last two ribs. This is done by passing a bipolar forceps around the ribs, cauterizing the intercostal blood vessels and then cutting the ribs with scissors, in turn.

A small retractor (e.g. a Lonestar) is placed to allow visualisation of the internal organs (**Fig. 29.7**).

Closure

If air sacs have been transected, they are left to heal by second intention. The muscle and skin are closed in separate layers with absorbable sutures in a continuous or interrupted pattern. No attempt is made to rejoin the transected ribs.

Post-operative care

No special post-operative care is required other than analgesia and, if appropriate, antibiotics. An adhesive dressing should be placed over the incision for 3–5 days to minimise wound infection.

Ventral midline coeliotomy

Indications

This procedure is used as an approach to the cloaca for cloacopexy, an approach to the oviduct or testes, or for biopsy of the liver and pancreas.

Technique

The bird is placed in dorsal recumbency and the legs are abducted caudally. The skin is tented and incised

Downloaded From Vet-eBooks.com

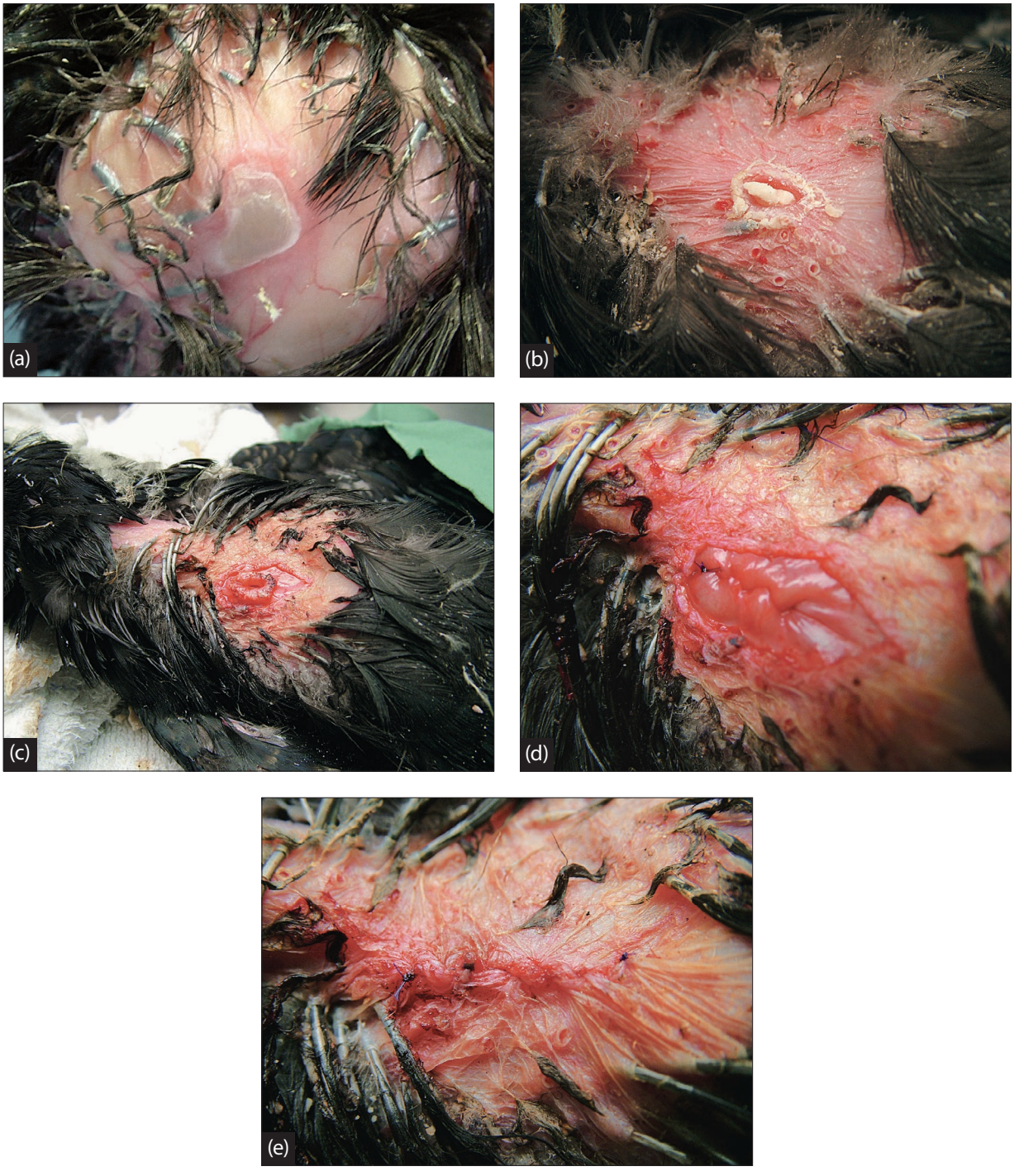


Figure 29.5 Sequence of treatment and procedures to address a crop burn in a juvenile black cockatoo. When presented (a) note the blanching of the now avascular skin and the surrounding erythema. After a few days (b) note the limit of the burn. Crop necrosis has now become evident. Surgical repair first involves dissecting the skin away from the crop and the fistula is excised (c). After excising the necrotic and granulating edges, the crop is repaired with a double-layer, continuous inverting suture pattern using a monofilament absorbable suture material (polydioxanone) (d). Finally the skin is closed in a single layer with the same suture material (e).

Downloaded From Vet-eBooks.com

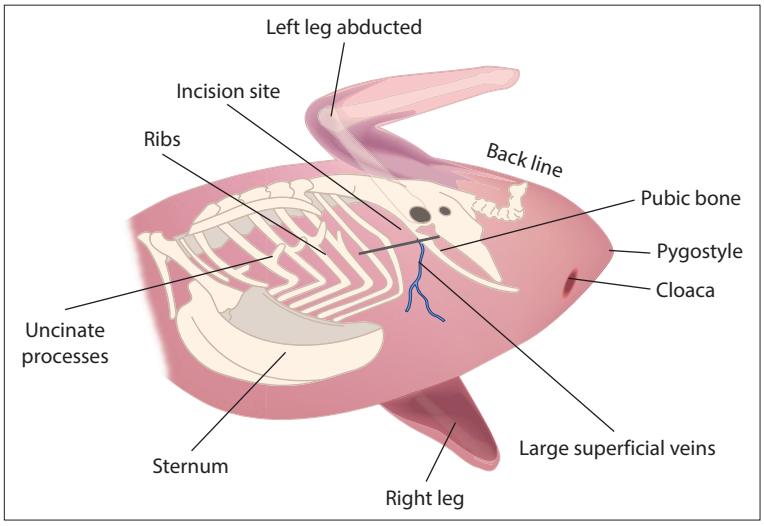


Figure 29.6 Schematic diagram highlighting the anatomical landmarks for a left flank coeliotomy.

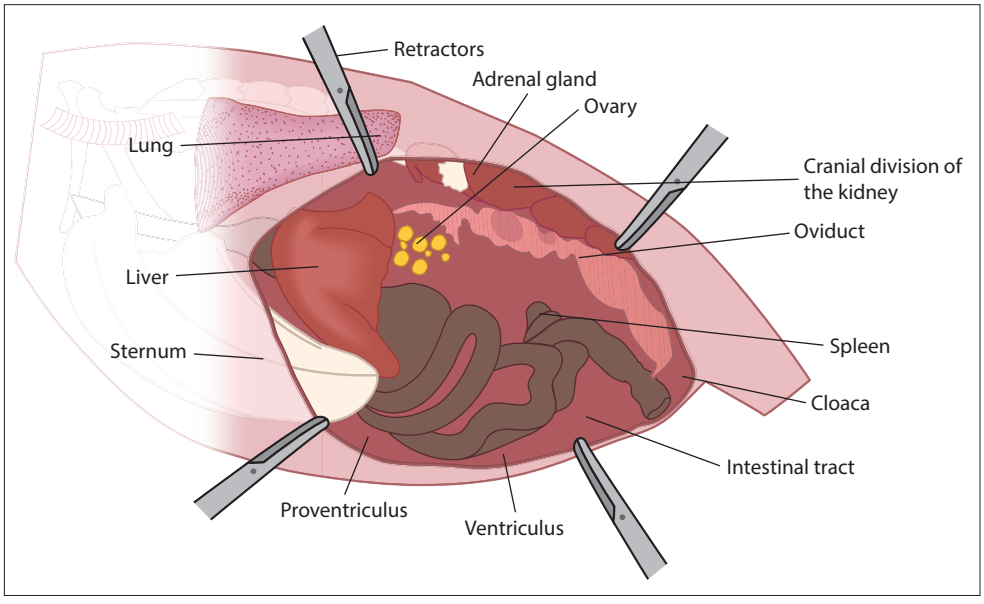


Figure 29.7 Illustration of the typical view inside the coelom during a left flank coeliotomy.

in the ventral midline from the sternum to a point level with the pubic bone. (**Fig. 29.8**).

The linear alba is tented and incised in a craniodorsal direction, avoiding iatrogenic damage to the underlying viscera (**Fig. 29.9**). To allow greater exposure, the incision can be extended laterally at the proximal and distal ends of the incision to create flaps. This will form a 'C' shaped incision with a unilateral flap or an 'I' shaped incision with bilateral flaps. (Note that while lateral flaps increase exposure they also allow increased anaesthetic gas and heat loss through the open coelomic cavity.) Again, a retractor can be used to increase exposure.

Closure

The muscle and skin are closed in separate layers with absorbable sutures in a continuous or interrupted pattern. No attempt is made to appose the peritoneum.

Downloaded From Vet-eBooks.com

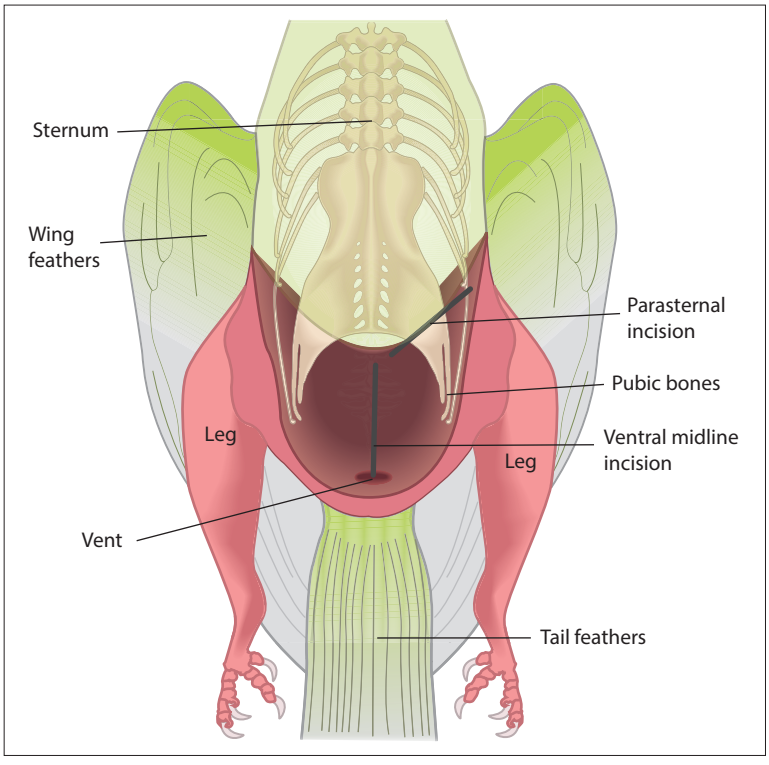


Figure 29.8 Schematic diagram indicating appropriate incision sites for a ventral midline coeliotomy.

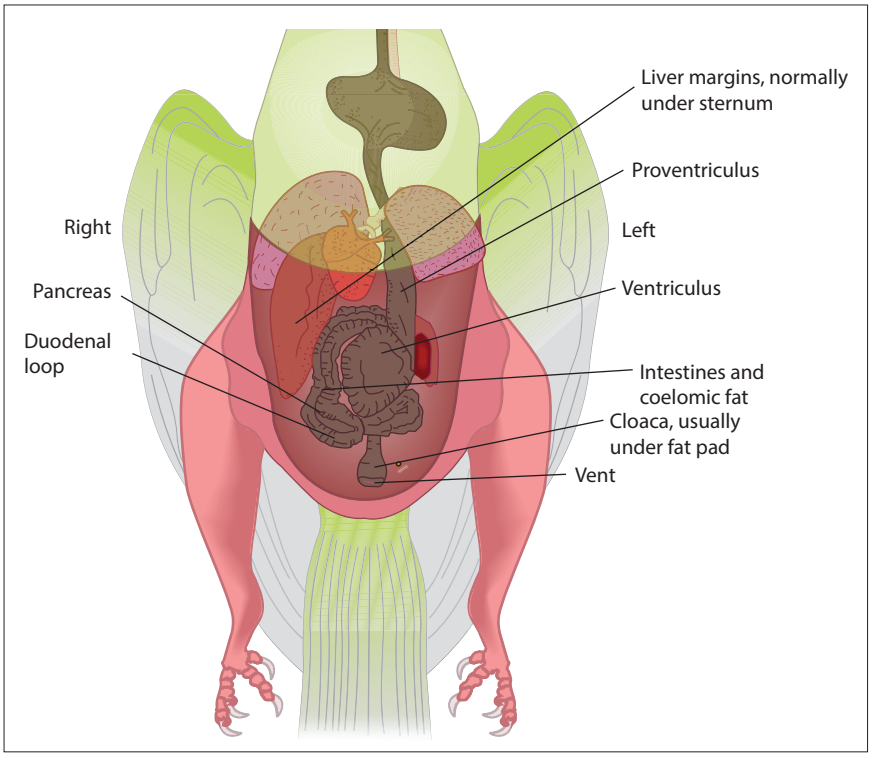


Figure 29.9 Illustration of the typical view inside the abdomen following a ventral midline incision.

Downloaded From Vet-eBooks.com

Post-operative care
Post-operative care is as for a left flank coeliotomy (see above).

Pancreatic biopsy
Indications
This is used for diagnosis of pancreatic disease.

Technique
With the bird in dorsal recumbency, a midline coeliotomy is performed as described above. The duodenal loop, found on the right side of the coelomic cavity, is gently exteriorised. Care must be taken not to pull too hard on the duodenal loop as this may damage the blood supply to both the duodenum and the splenic lobe of the pancreas.

The pancreas is examined for gross focal lesions. If seen, they are carefully biopsied. If no focal lesions are visible, the distal edge of the ventral lobe of the pancreas, at the apex of the duodenal loop, is carefully reflected to reveal underlying vasculature. Once these blood vessels are located and avoided, the end of the ventral lobe is removed with iris scissors and fixed in formalin. Minimal bleeding usually results.

The duodenum is then replaced into the coelom, and the skin and muscle are closed as described above.

Post-operative care
Post-operative care is as described above. Iatrogenic pancreatitis does not appear to be a feature of avian pancreatic biopsies as they are with mammals.

Proventriculotomy
Indications
This is used for retrieval of foreign bodies from the proventriculus or ventriculus.

Approach
A left flank coeliotomy is performed as described above. The proventricular suspensory ligaments are broken down. Two stay sutures are placed in the tendinous part of the ventriculus and it is brought up into the surgical field and attached to the skin. If possible, the rest of the coelom is packed off with saline-soaked gauze. The triangular lobe of liver overlying the proventricular isthmus is identified and gently reflected with a sterile cotton swab.

A stab incision is made into the isthmus and extended with iris scissors. Suction is used to empty fluid from the proventriculus and ventriculus. If necessary, an endoscope can be placed in the incision to ensure all foreign objects have been removed.

Closure
The proventricular incision is closed with 6–0 or 8–0 synthetic monofilament absorbable suture in two layers: the first should be appositional, the second inverting. The liver is tacked down over the incision. The coelom is closed routinely.

Post-operative care
Post-operative care is as described above for a left flank approach. Pre-, peri- and post-operative antibiotic coverage is advisable.

Ventriculotomy
Indications
This technique is indicated for the removal of foreign bodies (e.g. metal or cotton fibres) in the ventriculus.

Approach
A ventral midline approach as described above is utilised. The ventriculus is brought up to the incision with careful traction, and stay sutures are placed in the muscle of the ventriculus. In birds with a well-muscled ventriculus a stab incision is made through the muscle aponeurosis, seen as a white fibrous band. In birds with a poorly muscled ventriculus (e.g. lorikeets) a stab incision is made on the caudal wall of the ventriculus. Either incision is extended in a dorsoventral direction to gain access to the ventricular lumen.

Closure
The ventricular wall is closed with closely-spaced single interrupted PDS sutures. The surgical area is carefully flushed or cleaned with moistened Q-tips and the coelom closed as described above.

Post-operative care
Post-operative care is as described for a proventriculotomy.

Downloaded From Vet-eBooks.com

Coelomic hernia repair

Aetiology

Coelomic herniation is caused by a combination of increased intra-coelomic pressure (fat, ascites and organomegaly) and weakened muscles due to hormonal influences, obesity, lack of exercise and chronic malnutrition. It is commonly associated with females during the breeding season, as enlargement of the ovary and oviduct increases intra-coelomic pressure. Although most hernias occur on the ventral midline, they can also be seen on the lateral body wall and dorsal to the vent.

Precautions

If possible, the bird should be converted to a formulated diet before surgery in order to achieve significant weight loss. Reproductive activity can be reduced by hormonal and behavioural manipulation before surgery. These modifications and treatments may be sufficient to reduce the size of the hernia and avoid surgery.

The veterinarian should be aware that viscera may underlie and be attached to the skin. Replacing viscera back into the coelom may result in 'abdominal compartment syndrome' leading to decreased venous return and hypotension, and may also compress the air sacs leading to dyspnoea.

Technique

The bird is positioned according to the location of the hernia. The skin is incised over the hernia, taking care not to cause iatrogenic trauma to underlying viscera (which may have adhered to the skin). The skin over the hernia is gently and bluntly dissected until the borders of the body wall around the hernia are identified. Any adhesions to these borders are dissected away to free them from the hernia contents.

If possible, a salpingohysterectomy is performed and as much intra-coelomic fat as possible is removed before closing. This lessens the risk of abdominal compartment syndrome and air sac compression.

Closure

The hernia deficit is closed with a monofilament absorbable suture in a simple interrupted pattern. The anaesthetist should watch closely at this stage for any changes in the bird's respiratory pattern (indicating excessive pressure on the air sacs) or slowing of the heart rate (indicating hypotension).

Very large hernias, or those that cannot be closed without cardio-respiratory compromise, may require the use of non-absorbable mesh inside the body wall, attached to the muscles, last ribs, sternum and pubis (**Fig. 29.10**).

The skin is closed routinely.

Post-operative care

Post-operative care is as for other coelomic surgeries. If mesh has been used, antibiotic coverage for 10–14 days should be considered. The owner should be advised that re-herniation, either at the surgery site or elsewhere, is possible if the bird's weight and reproductive activity is not addressed and controlled.

Cloacopexy

Indication

This is indicated for chronic cloacal prolapse, associated with hypersexuality in male cockatoos, or excessive straining due to:

- Intestinal parasites.
- Retained egg.
- Adenomatous polyp.
- Bacterial enteritis.
- Neoplasia.
- Coelomic mass.
- Cloacal hyperplasia.



Figure 29.10 **Black cockatoo chick undergoing a mesh repair of a coelomic hernia.**

Downloaded From Vet-eBooks.com

Technique

A ventral midline incision is made as described above, although positioned more caudally. The prolapsed cloaca is replaced and an assistant places a gloved finger (large birds) or Q-tip (small birds) into the cloaca to define its extent and to lift it to the coelomic wall. Intra-coelomic fat ventral to the cloaca is removed.

Using monofilament non-absorbable material, sutures are placed through the full thickness of the cloacal wall and then around the last rib (at the junction of the sternal and vertebral portions) on each side, or through the cartilaginous border of the sternum. All sutures are pre-placed and tied once all are in position, anchoring the cloaca in a reduced position with the cloacal wall apposed to ribs, albeit more cranial than normal.

Following the placement of these sutures, the ventral cloacal serosa is incised to the level of the submucosa and tacking sutures are placed through the coelomic wall to suture the submucosa to the coelomic muscle. Some of these sutures are incorporated into the coelomic wall closure.

Post-operative care

Post-operative care is as for other coelomic surgeries.

Prognosis

This technique attaches the ventral aspect of the cloaca to the ribs and body wall. However, the dorsal cloaca is not dealt with. Therefore, if the bird continues to strain, this tissue can still prolapse. As time goes on, with continued straining, the sutures may break or cut through the attached tissues and prolapse of all the cloacal tissue can occur.

Cloacoplasty/ventoplasty

Indications

This procedure provides temporary or permanent narrowing of the vent opening as treatment for cloacal prolapse (**Fig. 29.11**) or atony. A purse-string suture is contraindicated in birds as the vent is a lateral slit, not a circle.

Technique

If a permanent narrowing is required, the mucocutaneous border on the lateral third of the dorsal and ventral vent lips is trimmed on both sides, leaving the middle third untouched If a temporary closure is required, this step is not indicated. The lateral third of the vent is closed on both sides with one to two vertical mattress sutures (see **Fig. 16.13**, Chapter 16, Disorders of the Gastrointestinal Tract, p. 247), leaving the middle third open for faeces and urine to be expelled.



Figure 29.11 **Cockatoo with a cloacal prolapse.**

Post-operative care

The bird should be carefully observed to ensure it is able to defecate normally. If a temporary procedure has been performed the sutures can be removed after 2–4 weeks. Care must be taken with egg-laying hens that either its reproductive drive is switched off or that the bird is monitored carefully for dystocia.

Cloacotomy

Indications

Cloacotomy is used for debriding cloacal papillomas or removing cloacoliths.

Technique

The bird is placed in dorsal recumbency. A moistened cotton bud is inserted into the cloaca to outline the structure. An incision is made through the skin, cloacal sphincter and cloacal mucosa from the vent to the cranial end of the cotton bud. This opens the ventral wall of the cloaca, exposing the coprodeum, urodeum and proctodeum.

Downloaded From Vet-eBooks.com

Closure
The mucosa is closed with a continuous suture pattern, the sphincter is closed with a single mattress suture and the skin is closed routinely.

Post-operative care
A topical antibiotic ointment such as silver sulphadiazine can be introduced into the cloaca twice daily to sooth the cloacal wall and provide local antibiotic coverage.

Salpingohysterectomy
Indications
This is indicated for chronic egg laying and any oviductal disease that cannot be managed medically (e.g. pyometra, yolk peritonitis, or coelomic herniation).

Pre-surgical evaluation and conditioning
Yolk peritonitis and underlying liver, lung and kidney disease all carry a higher risk of surgical complications. An enlarged oviduct (due to hormonal influences) fills the left coelom and makes surgery difficult. Therefore, if time permits, the patient's nutritional status should be improved and the reproductive cycle 'turned off' through behavioural, social and environmental modification and hormonal therapy (see Chapter 23, Disorders of the Reproductive Tract).

Technique
The left lateral approach gives best exposure, but the ventral midline approach can also be used depending on the surgeon's preferences. The ovary is left alone; the blood supply is tightly adherent to major blood vessels (the cranial renal artery and the caudal vena cava), making removal without an operating microscope extremely dangerous.

The infundibulum is identified and gently retracted out through the incision. A large blood vessel running between the infundibulum and ovary must be identified and ligated, and the relatively avascular ventral suspensory ligament is broken down. The more vascular dorsal suspensory ligament is broken down by sharp dissection and the blood vessels (the cranial, middle and caudal oviductal arteries) are cauterised or ligated as needed.

The oviduct is retracted through the incision and the junction with the cloaca is identified (if necessary, a cotton bud or gloved finger is inserted into the cloaca to delineate the structure). Two vascular clips or sutures are placed across the oviduct near this junction, and the oviduct is removed. Closure is as described previously.

Post-operative care
Post-operative haemorrhage from ovarian or oviductal vessels is a major and common complication with salpingohysterectomy. Often these vessels are not identified at the time of surgery but bleed once the patient starts to move around. Weakness or obvious blood loss should be investigated with this in mind. Re-entering the coelom, identifying and ligating the bleeding vessels, is the most effective way of remedying this problem. The surgeon should be prepared to give the patient a blood transfusion.

If there has been ascites or coelomic bleeding, it is not uncommon for the patient to be mildly dyspnoeic and/or coughing up blood/fluid for a few days post-operatively. (This is due to fluids entering the air sacs and then the lungs.) This needs to be monitored carefully and, if necessary, supportive measures provided (such as an oxygen cage). In severe cases it may be necessary to re-explore the coelom surgically.

If there are no complications, post-operative care is similar to that used for a coeliotomy (see above).

Follow-up
Continued ovulation and yolk release with subsequent yolk-related peritonitis has been reported in several birds following salpingohysterectomy. This may be due to a persistent right oviduct or failure to remove larger follicles during surgery. Follow-up surgery may be required, although in many cases hormonal manipulation and anti-inflammatory medications may control this issue.

Orchidectomy
Indications
This procedure may be carried out to treat orchitis or testicular neoplasia, or for behavioural modification.

Downloaded From Vet-eBooks.com

Technique

The approach is via either a left lateral or bilateral coeliotomy or a ventral midline coeliotomy with cranial flaps. The testis is gently retracted ventrally and the mesorchium over the testicle carefully incised to allow it to retract and expose the blood supply to the testicle. Vascular clips are placed across the dorsal blood supply and the testicle is excised, leaving the vascular clip in place. Radiosurgery unit is useful for this part of the procedure, cauterising small blood vessels that may have been missed in the ligation. The coelomic incision is closed as previously described.

Post-operative care

Post-operative care is as for other coelomic surgeries.

Follow-up

If all testicular tissue is not removed, regrowth is common. Some male characteristics are retained, indicating that testosterone may be produced in other tissues.

Enucleation

Indications

Enucleation is indicated for severe, irreversible panophthalmitis, perforating corneal ulceration, or neoplasia.

Precautions

This procedure is more difficult in birds than in mammals because of the relatively larger globe compared with the size of the orbit. The optic nerve is short; excessive traction can result in contralateral blindness. The area is very vascular and the surgeon should anticipate haemorrhage.

Technique

The lids are sutured together in a simple continuous pattern. A circumferential incision is made through the skin (not the conjunctiva) 1–2 mm from the lid margins. Note that the ligamentous attachments at the medial canthus are firm. Haemorrhage can be expected in this area and at the lateral canthus.

Dissection is carried out between the palpebral conjunctiva and the bony orbit, as it is not feasible to identify and transect each individual muscle. The sutured eyelids are manipulated to provide traction on the globe.

In some birds with a large globe, it may be necessary to collapse the globe prior to enucleation. In some species with thin orbital bone, it is feasible to extend the skin incision from the lateral canthus to the auditory meatus and transect the orbital rim at the lateral canthus. This opens the orbit and allows easier retraction of the globe.

Where feasible, a vascular clip is applied blindly to the optic stalk (incorporating the nerve and blood vessels). Care must be taken to apply minimal traction to the globe at this point to avoid damage to the contralateral optic nerve. After the stalk is clipped, it is transected and the eye removed. If it is not feasible to place a vascular clip, the globe is retracted with the use of sharp dissection. Haemorrhage can be expected at this stage, and can usually be controlled by placing a vascular clip directly on to the now visible optic stalk. If this is not feasible, packing the orbit with absorbable gelatine sponges (e.g. Gelfoam®) is usually effective.

Closure

The eyelids are sutured in a simple interrupted pattern.

Post-operative care

Post-operative bleeding, epistaxis and sneezing is common and usually self-limiting. Excessive bleeding (indicated by marked distension of the skin over the orbit) may indicate the need for surgical exploration to locate the source of the bleeding.

It is not unusual for glandular material to be inadvertently left in the orbit, and for a mucoid discharge to persist after surgery. In these cases, surgical exploration of the orbit may be required to locate and remove this tissue.

Repairing skin lacerations

Skin lacerations are not uncommon in birds, with injuries inflicted by predator attacks, sharp objects, or even other birds. Some of these injuries are simple lacerations; others are more complex injuries associated with extensive soft tissue and even bone damage.

Downloaded From Vet-eBooks.com

Avian skin is closely attached to underlying muscle and bone. Loose skin in most birds can be found on the neck and the inguinal area, leaving most of the skin on the torso, head and limbs relatively immobile. This makes undermining and mobilising skin to close wounds more difficult than similar surgery in many mammalian species.

The skin is thin and often has a fatty subcutaneous layer. Subcutaneous suturing does not hold well and is therefore of limited value. Post-operative swelling is not as severe in birds as mammals, and sutures can therefore be tied tighter and closer together.

Avian heterophils lack lysozymes, meaning that avian pus is usually caseated and therefore does not drain from a contaminated wound readily. The use of drains in these wounds may not be of significant benefit.

Skin laceration repairs in birds therefore require that the surgeon:

- Thoroughly debrides and flushes the wound. In most cases, the use of sterile saline is preferable as a flushing solution – chlorhexidine and povidone iodine, even diluted, can be toxic to granulation tissue and should be avoided if possible.
- Mobilises available skin where possible to achieve primary closure without tension on the wound edges.
- Avoids the use of drains unless combined with a flushing or irrigation system. If necessary, dead space should be obliterated surgically.

Where primary closure cannot be achieved, either skin grafts and flaps, or healing by second intention is required. Using the same surgical techniques employed in mammals such as dogs and horses, skin grafting and flaps are feasible alternatives in birds. If skin grafting is not employed, good healing can often be achieved by secondary intention. In this situation, avoid the underlying subcutis and muscle drying out by the use of wet or hydrocolloid dressings (e.g. Duoderm®).

Aggressive post-operative antibiosis and analgesia will significantly improve morbidity and mortality rates in these patients.

ORTHOPAEDICS

General considerations

Bones

Avian bones are lightweight, but possess great aerodynamic strength. They have thin brittle cortices, which will not provide sufficient holding power for bone screws. Some, but not all, are pneumatic. Fractures are frequently open and comminuted due to minimal soft tissue coverage.

The blood supply to bones arises from periosteal, medullary, metaphyseal and epiphyseal blood vessels. The periosteal blood supply is very important in callus formation, especially in pneumatic bones such as the humerus and femur, and its importance may exceed that of the medullary blood supply in those bones.

Joints

If joints are immobilised for long periods of time, contracture of ligaments and tendons can result in a permanently reduced range of movement. Fracture callus may impinge on joint range of motion, as may adhesions of ligaments and tendons.

Muscles

Powerful flight muscles can cause rotational deformity of long bones during the early healing phase.

Bone healing

Healing of bones is a combination of:

- Primary healing. Bone to bone healing through the Haversian system, with minimal callus formation. This is only achieved with rigid fixation with perfect bone apposition.
- Endosteal callus formation. This occurs rapidly where bones are well aligned. It is the most important part of bone healing.
- Periosteal callus formation. This occurs when fractures are not aligned and there is movement at the fracture site.

Obviously primary healing is the desired goal of fracture repair, but it is only occasionally achieved. Movement of the limb, combined with the strong pull of muscles (especially in the wing) and the thin

Downloaded From Vet-eBooks.com

cortices of avian bone make rigid fixation difficult to achieve. However, even if primary healing is not attained, endosteal healing makes for a solid fracture repair that can be very functional.

The rate of healing is dependent on:

- Displacement of bone fragments. Segmental fractures will heal well so long as periosteal blood supply is intact. If devitalised, the fragment can be incorporated into the fracture site as a cortical bone graft. Healing is slower, as cancellous bone first bridges the gap, then the segment is demineralised and becomes cancellous itself. Healing may take 9–18 weeks.
- The blood supply to the bone. As mentioned above, the blood supply to bones arises from the periosteal, medullary, metaphyseal and epiphyseal vessels. The periosteal blood supply is the predominant source of blood to the pneumatic bones (e.g. humerus and femur) while non-pneumatic bones (e.g. the radius, ulna and tibiotarsus) may be more reliant on medullary or metaphyseal and epiphyseal blood vessels. The disruption of a bone's blood supply, either by trauma or surgical repair of a fracture, may result in delayed healing or even complete failure to heal (a non-union). The presence of medullary bone (osteopetrosis or hyperostotic polyostosis) in non-pneumatic bone means that their blood supply will be compromised even more by trauma, and, in the author's experience, fractures of these bones in hyperoestrogenic hens carries a guarded prognosis.
- Presence of infection. Osteomyelitis may result in a sequestrum of dead bone. Sequestra can add to the stability of a fracture and should not be removed until a bony callus has formed, unless they are acting as a nidus of infection.
- Movement at the fracture site creates a large haematoma and a large cartilaginous bridge. This can delay or even prevent healing.

Stable, well aligned fractures heal faster in birds than in mammals. Clinical stability of a fracture (often achievable through a firm fibrous callus at 2–3 weeks) may precede radiographic evidence that bone is healed (4–8 weeks). Healing times are approximately as follows:

- External coaptation:
  - 1 week: palpable callus, movement still palpable.
  - 2 weeks: movement considerably reduced.
  - 3 weeks: no movement, endosteal callus present.
  - 5–8 weeks: healed, remodelling beginning.
- Internal fixation:
  - 2 weeks: union present.
  - 3 weeks: remodelling beginning.

Principles of orthopaedic surgery

Keeping in mind the issues discussed previously, orthopaedic surgery should aim for:

- Minimal soft tissue damage so as to preserve the periosteal blood supply.
- Accurate alignment of the bone so as to maintain the length of the bone, and rotational and angular orientation.
- Rigid stabilisation of the fracture by either splinting the limb or surgery.
- Encouragement of early return to normal function to prevent 'fracture disease' (permanent contracture of muscles, tendons, ligaments and joints).

Types of fracture repair

Fractures can be repaired by either external coaptation, surgery, or a combination of both. The selection of the technique is a function of the extent and location of the fracture, the requirement for full return of function, the skill and experience of the clinician and the costs for both veterinarian and client.

External coaptation

External coaptation (bandaging and splinting) can be used for many limb fractures, either as a temporary splint before or after surgery, or as the sole treatment for a fracture. Figure-of-eight bandages are frequently used for antebrachial and metacarpal fractures; humeral fractures are immobilised by strapping the wing to the body; tibiotarsal and tarsometatarsal fractures can be treated with Robert Jones bandages, Altman splints, or stirrup splints. These techniques are described in Chapter 4, Clinical Techniques. Clinicians need to be aware of the benefits and disadvantages of these techniques:

Downloaded From Vet-eBooks.com

Benefits of external coaptation (see Figs 4.14–4.19, Chapter 4, Clinical Techniques, pp. 92–93) include:

- Decreased chance of infection.
- Less damage to regional vascularity.
- Minimal surgical and anaesthetic risk in high-risk (e.g. trauma) patients.
- Can be used in patients too small to feasibly operate on.
- It is inexpensive and rapid.

Disadvantages of external coaptation include:

- 'Fracture disease' is common (restricted joint motion and soft tissue contraction resulting in joint ankylosis).
- Healed bone may be misaligned (shortened or rotated).
- The lack of rigid immobilisation may result in slower healing, usually by periosteal callus, and delayed or non-union is a major complication.
- Pressure necrosis can develop if the bandages are applied too tightly.
- Slippage of the bandage can result in the fracture becoming a fulcrum point, resulting in movement at the fracture site and delayed or non-union.

The selection of the means of repair is dependent on these advantages and disadvantages, as well as the location of the fracture. For example, distortion produced by the powerful flight and leg muscles makes external coaptation a poor choice for humeral and femoral fractures.

Surgical repair

Intramedullary pins: Stainless steel pins or polymer rods can be used. (Polymer rods are introduced in a retrograde fashion and held in position with bone cement). Intramedullary pins maintain alignment and length of the bone, but may lack rotational stability (**Fig. 29.12**). This can be overcome by:

- Combining the pinning with semi- and full-cerclage wire, but great care must be taken not to disrupt the periosteal blood supply.
- Stack pinning with several small pins (most applicable to fractures of the humerus and femur).

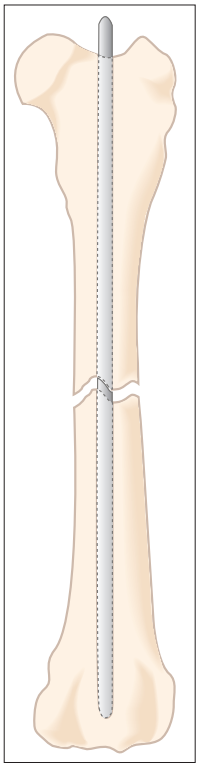


Figure 29.12 **Diagram showing the correct placement of an intramedullary pin in the femur.**

- Combining with an external skeletal fixator to create a 'tie-in' fixation (see below).

Pin diameter should equal half to two-thirds of the medullary canal. Excessively large pins can interfere with endosteal blood supply, which may cause avascular necrosis or iatrogenic fractures.

Intramedullary pins can be placed either:

- Retrograde: the pin is inserted through the fracture site and advanced out either the proximal or distal end of the bone (avoiding the joint), and then the direction is reversed and the pin guided into the other fragment. This technique is usually used for femoral, humeral and ulnar fractures.
- Normograde: beginning at a natural end of the bone, the pin is advanced through the fracture site and into the other fragment. This is usually used for tibiotarsal, proximal humeral or distal ulnar fractures.

Downloaded From Vet-eBooks.com

The pins should be removed when the bone has healed.

Plates: Lightweight plates such as 'string of pearl' plates, cuttable plates, and finger plates may lend themselves to avian fracture repairs. Advantages of plates include:

- Early return to function, resulting in minimal fracture disease.
- Rigid immobilisation leads to primary bone healing.
- They provide rotational stability as well as maintaining the length and alignment of the bone.

However, there are disadvantages to the use of bone plates, including:

- The cortices of most (but not all) avian bones are too thin to engage the thread of the most bone screws. In some cases this can be overcome by filling the medullary canal with bone cement before placing the screws, but the exothermic reaction as the cement cures can be detrimental to the bone. Titanium bone screws (which form a bond between the bone and the screw) can be used, but present a major financial hurdle.
- Most plates are often too large and too heavy for avian bones.
- There are technical difficulties involved in the placement and removal of plates and screws, involving a longer surgical time, and therefore increased risks of tissue compromise and prolonged anaesthesia.
- It may be difficult to obtain soft tissue coverage of the plate and screws.
- Bone plates and screws are expensive.

External skeletal fixation: External skeletal fixation (ESF) (**Fig. 29.13a**) is lightweight, strong, inexpensive and adaptable to many fractures. In closed fractures, it can reduce the risk of osteomyelitis. It provides both rotational and lengthening stability.

The equipment needed includes:

- Transfixation pins: positive-profile threaded pins inserted through the predrilled holes have been found to maintain solid bone-to-pin interfaces for prolonged periods (up to three months) in some birds.
- A connecting bar.
- Means of connecting pins to bar: clamps are available, but dental acrylic and similar hardware materials have been used successfully.

Types commonly used in birds are the Type I device, with half pins connected on one side of the limb only (**Fig. 29.13b**), and the Type II device, with full pins running through the bone and connected on both sides of the limb.

The most distal and proximal pins are inserted first in a medial to lateral direction. Ideally, 2–3 pins are placed through each side of the fracture at an angle of 30–90° to the bone, and the connecting bar is then applied.

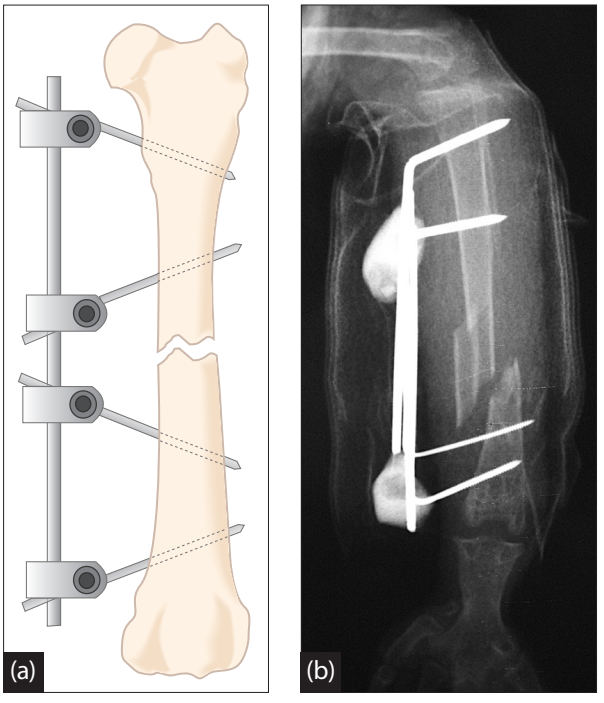


Figure 29.13 **Diagram showing how a Type I external skeletal fixation should be applied to address a fracture (a). Radiograph of a cockatoo with a Type I external skeletal fixation in place to repair a tibiotarsal fracture (b).**

Downloaded From Vet-eBooks.com

Tie-in fixator: A tie-in fixator (TIF) is a combination of an intramedullary pin linked to external fixator pins. It can be used to repair diaphyseal and periarticular fractures of all avian long bones except the tarsometatarsus (**Fig. 29.14**). The intramedullary pin selected should fill 50–65% of the medullary cavity. It can be inserted in either a normograde or retrograde technique past the reduced fracture (**Fig. 29.15a–c**), and emerges from the proximal end of the fracture. Positive-profile ESF pins are inserted perpendicular to the bone at locations where they will not be interfered with by the intramedullary pin (**Fig. 29.15d**). The protruding end of the intramedullary pin, where it emerges from the skin, is bent at 90° and rotated so that it is in the same plane as the ESF pins (**Fig. 29.15e**).

All pins are joined by a connecting bar (**Fig. 29.15f**). This connecting bar can be either a separate piece, or it can be formed from the intramedullary pin (if long enough) bent again at 90° to run down the length of the limb (**Fig. 29.15g**).

The degree of fixation achieved with this technique is such that primary bone healing often results (i.e. no periosteal callus) so long as the fracture is well aligned.

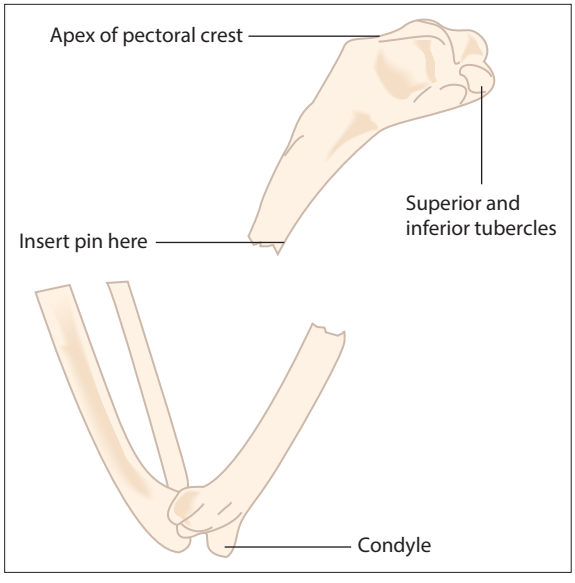


Figure 29.14 Illustration of a typical diaphyseal fracture.

Selection of means of fixation

See *Table 29.2* at the end of this section for a summary of options.

Wing

An inability to adequately immobilise the shoulder joint, as well as distortion produced by the powerful flight muscles, makes external coaptation a poor choice for humeral fractures if a return to flight is desired. If this is not a prerequisite, external coaptation may well be a suitable technique so long as the owner has realistic expectations of the bird's ability to fly afterwards.

Coracoid, scapula, clavicle: There are two schools of thought for the repair of fractures involving the pectoral girdle. Conservative treatment (strapping the wing to the body) may give good results if the coracoid is not severely displaced. However, surgical reduction and fixation of the coracoid with an IM pin may be indicated if it is displaced enough to press on to the trachea or the heart.

Leg

Proximal humerus: ESF is difficult due to the lack of room in the proximal fragment. A tension band method using wire (in a figure-of-eight configuration) and an IM pin has worked, as has a TIF.

Midshaft humerus: IM pinning or a TIF using a single ESF pin placed in the distal humerus are suitable repair methods for midshaft fractures of the humerus. Bone plating may be suitable for large birds (>3 kgs).

Distal humerus: Cross-pinning from the distal humerus, perhaps combined with a Type I ESF, can be effective in repairing these fractures.

Antebrachium: External coaptation may be used if either the ulna or radius is still intact, however fractures involving both bones will do better if surgically repaired. External coaptation often results in a synostosis joining the two bones, making pronation of the antebrachium difficult and thereby restricting flight.

Downloaded From Vet-eBooks.com

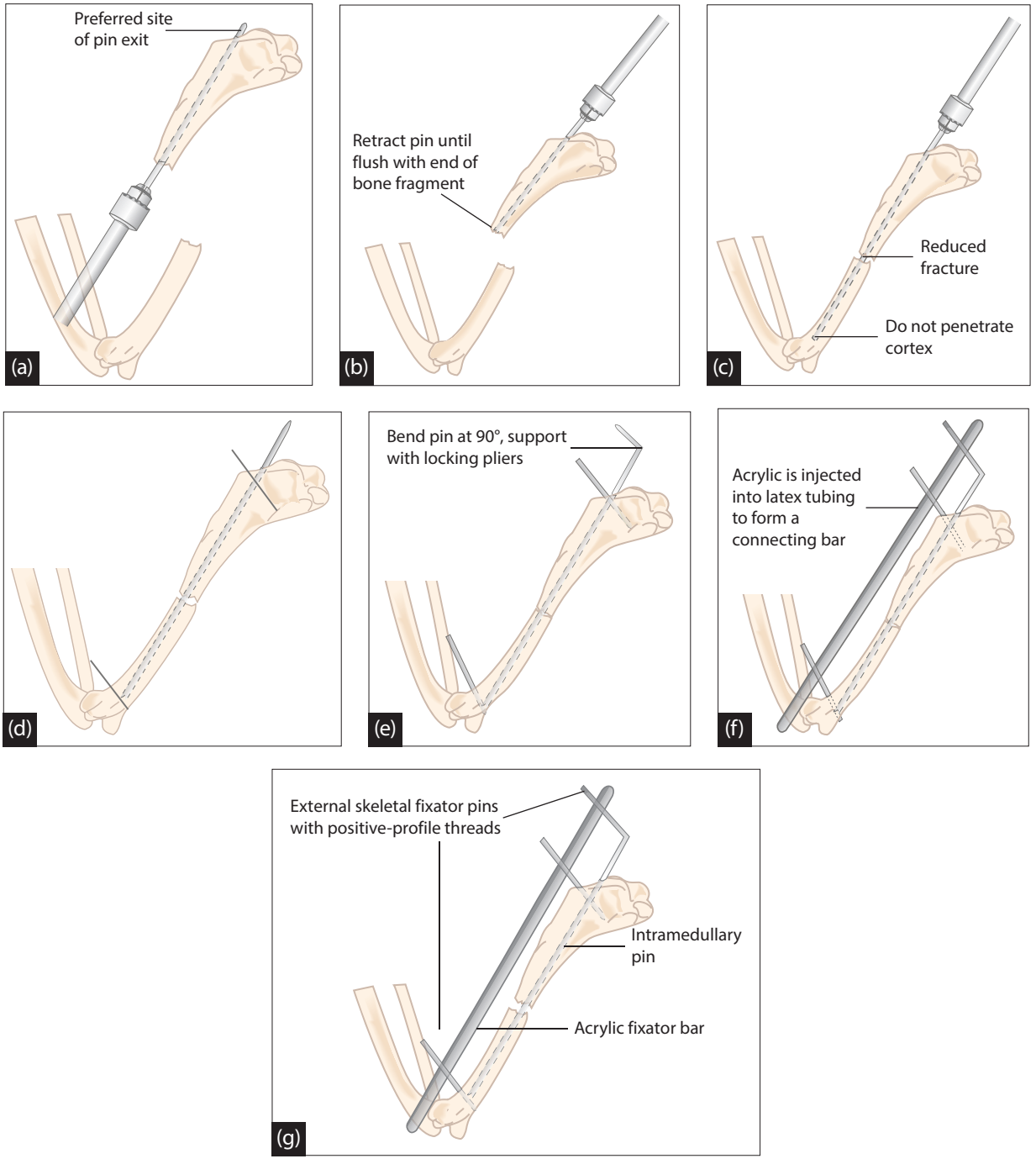


Figure 29.15 Schematic sequence of necessary steps required to address a diaphyseal fracture Step one (a). Retrograde placement of an intramedullary pin into the proximal fragment. Step two (b). The pin is withdrawn proximally until the pin is flush with the fracture line. Step three (c). The fracture is reduced and the pin advanced into the distal fragment. Step four (d). Positive profile threaded ESF pins or K-wires are placed at both ends of the bone, perpendicular to the bone. Step five (e). The proximal end of the pin, protruding from the bone, is bent at 90° in the same plane as the ESF pins. Step six (f). A connecting bar (pin or latex tube filled acrylic) is used to join the ESF pins and the end of an intramedullary pin that has been bent over. The completed tie-in fixator (g).

Downloaded From Vet-eBooks.com

Table 29.2 **Selection of means of fracture repair for different limb injuries**

		MEANS OF REPAIR						
LIMB	**LOCATION**	**EXTERNAL COAPTATION**	**IM PIN**	**TYPE I ESF**	**TYPE II ESF**	**TIF**	**PLATING**	**OTHER**
Wing	Coracoid	**X**	x					
	Proximal humerus		x			**X**		x[1]
	Mid-shaft humerus		x	x		**X**	x	
	Distal humerus		x	x		**X**		x[2]
	Antebrachium	x	**X**	x		x		
	Metacarpus	**X**	x	x				
Leg	Proximal femur		x			x		**X**[1]
	Mid-shaft femur		**X**			x	x	
	Distal femur		x			x		**X**[2]
	Tibiotarsus	x	x	x	x	**X**	x	
	Tarsometatarsus	**X**		x	x			

[1] Tension band wiring
[2] Cross pinning

Intramedullary pins, ESF or TIF are suitable means of repair.

Metacarpus: External coaptation, Type I ESF or TIF are indicated, depending on the size of the bird.

Leg fractures

An inability to immobilise the coxofemoral joints, as well as distortion produced by the powerful leg muscles, makes external coaptation unsuitable for femoral fractures.

Proximal femur: A TIF is usually successful, but tension band wiring using an IM pin and cerclage wire can also be used.

Midshaft femur: IM pinning, or a TIF with a single ESF pin in the distal femur, is indicated. Plating can be used in large birds (>3 kgs). The bulk of the leg muscles makes ESF pinning unsuitable for fractures in this position.

Distal femur: Cross-pinning is used in combination with an IM pin.

Tibiotarsus: Fixation may be achieved with an IM pin, Type or I or II ESF, a TIF or external coaptation.

Tarsometatarsus: The lack of a medulla in the tibiotarsus in most birds makes surgical repair difficult in most cases. External coaptation (**Fig. 29.16**) is suitable in small birds, or a Type II ESF can be used in larger birds.



Figure 29.16 Lorikeet with an external coaptation in place to repair a tarsometatarsal fracture.

Downloaded From Vet-eBooks.com

Post-operative management and complications

Where possible, external coaptation should be used to prevent iatrogenic damage to a fracture site. Excessive wing flapping or premature weightbearing on a repaired limb can, and frequently does, undo the best efforts of any surgeon. This external coaptation should be placed immediately post-operatively, before the patient is recovered from anaesthesia.

Analgesia should be provided (see Chapter 28, Analgesia and Anaesthesia) for 1–2 weeks post-operatively.

Antibiotic coverage is provided using:

- Cephalosporins or enrofloxacin for five days post-operatively for uncomplicated fracture repairs.
- Clindamycin or amoxicillin-clavulanic acid can be used for open, comminuted and infected fractures.
- Antibiotic-impregnated methylmethacrylate beads can be used in some fractures if infection is likely.

Joint contracture and ankylosis is a common complication of orthopaedic repair. Physical therapy (passive range of movement exercises) can help to prevent or overcome this, and may have to be done under anaesthesia, starting at two days (for humeral fractures) or ten days (for other fractures). Five-minute sessions should be given twice weekly for two weeks.

Patagial (wing web) contraction is common and can restrict the ability of the bird to extend its wing. This can be overcome by use of rigid fixation (allowing early return to function), passive physical therapy, ultrasound massage and physical massage.

The fracture should be radiographed at seven days and again at three weeks. If re-alignment is necessary, it must be done before ten days. Dynamic destabilisation (partial dismantling of fixation), where appropriate, can be started at 21 days if healing is well underway. Sequestra can be seen radiographically at 21 days and can be removed surgically when their extent is clear.

Healing should be complete by six weeks and all fixation removed.

Approaches to the bones of the wing

Coracoid and clavicle

A ventral approach is used. A skin incision is made along the caudal edge of the furcula starting laterally and continuing medially along the lateral edge of the keel for the first one-fifth or one-sixth of the length of the keel. An incision is made through the superficial pectoral muscle along the caudal edge of the furcula. Cranial and caudal pectoral vessels and nerves are seen between the superficial and deep pectoral muscles. The deep pectoral muscle is incised and reflected along the clavicle and keel. Haemorrhage from the clavicular artery, which supplies part of the pectoral muscles (encountered at caudal midpoint of furcula), must be controlled.

Muscles of the coracoid are now visible: the supracorocoideus and corobrachialis caudalis. These can be separated to get better exposure of the coracoid. Multiple small intramedullary pins are introduced at the fracture site and exteriorised through the point of the shoulder, then the pins are normograded back through the distal fragment taking care not to perforate the pericardium and heart.

The supracorocoideus and corobrachialis caudalis are sutured if separated. The pectorals are reattached to the periosteum in separate layers. The skin is closed.

Proximal humerus

A dorsal approach is used in fractures of the proximal third. The feathers are plucked over the proximal, medial and ventral humerus. In raptors the scapular coverts insert into a fascia, which continues from the cutaneous costohumeralis muscle. This should be maintained if possible, as they may be involved in critical aspects of flight. The skin is incised along the shaft of the humerus.

Caution should be exercised at this stage, as:

- The axillary nerve is deep to the propatagialis complex in the proximal third of the humerus.
- A branch of subscapular artery is medial and cranial to nerve.
- The radial nerve crosses the humerus two-thirds of the way down the shaft, near the site of insertion of the deltoideus major muscle.

Downloaded From Vet-eBooks.com

The propatagialis muscle can be transected in the distal third or one can bluntly dissect through it, avoiding the muscle branch of the axillary nerve. The deltoideus may be retracted proximally, but it needs to be reattached to bone when the procedure is completed.

There are two techniques for repair: stack pinning in birds <300 g or tension band in birds >300 g. In the latter technique, two K-wires are inserted retrograde dorsally and ventrally to the pectoral crest, then the fracture is reduced and the pins are advanced into the distal fragment. Two holes are drilled, one in the humerus 1 cm distal to the fracture, the other through the pectoral crest proximal to the K-wires. Cerclage wire is passed through the holes, made into a figure-of-eight and tightened.

If the deltoideus was elevated, a hole is drilled through the bone to allow a suture to reattach the muscle to the bone. The propatagialis muscle is sutured if it was transected.

Distal humerus

There are two approaches, both of which can be used to reduce and fix internally fractures of the humerus in the midshaft to distal third. The dorsal approach is more commonly used.

Dorsal approach: The bird is placed in sternal recumbency with the affected wing extended. The feathers are plucked from the pectoral crest to the proximal radius/ulna.

The radial nerve should be palpated before making the skin incision. The skin is incised from the proximal third of the humerus to the olecrannon fossa and ventral epicondyle, or even more caudally (**Fig. 29.17a–d**). The deltoideus and triceps insertions on the caudal aspect of the humerus are identified, with the radial nerve emerging between them. The biceps brachii and the tendon of the tensor propatagialis pars brevis are identified on the cranial side of humerus. Distally, three tendons are identified originating from the antebrachium: cranially, the extensor metacarpi radialis; caudally, the extensor metacarpi ulnaris; and between them, the supinator and common digital extensor.

In distal fractures, two K-wires can be used to cross-pin through the dorsal and ventral epicondyles. These can then be tied into an ESF.

Ventral approach: This is used for distracted fractures of the distal two-thirds of the humerus (**Fig. 29.18a–c**).

The bird is placed in dorsal recumbency with the affected wing extended. The feathers are plucked from the ventral humerus and dorsally and cranially over the pectoral crest.

The biceps brachii muscle is palpated cranial to shaft of humerus. The ulnar and radial vessels and medianoulnar nerve run deep to or on the caudal edge of this muscle, as does the superficial basilic vein. To avoid these structures, the skin incision is made either over the belly of the biceps muscle or over the humeral shaft caudal to the vessels and nerves. The excision is continued distally to the elbow. The biceps is elevated and retracted with the vessels and nerve. The triceps can be elevated caudally for better exposure.

Closure is by simply suturing skin and superficial fascia as a single layer.

Proximal radius and ulna

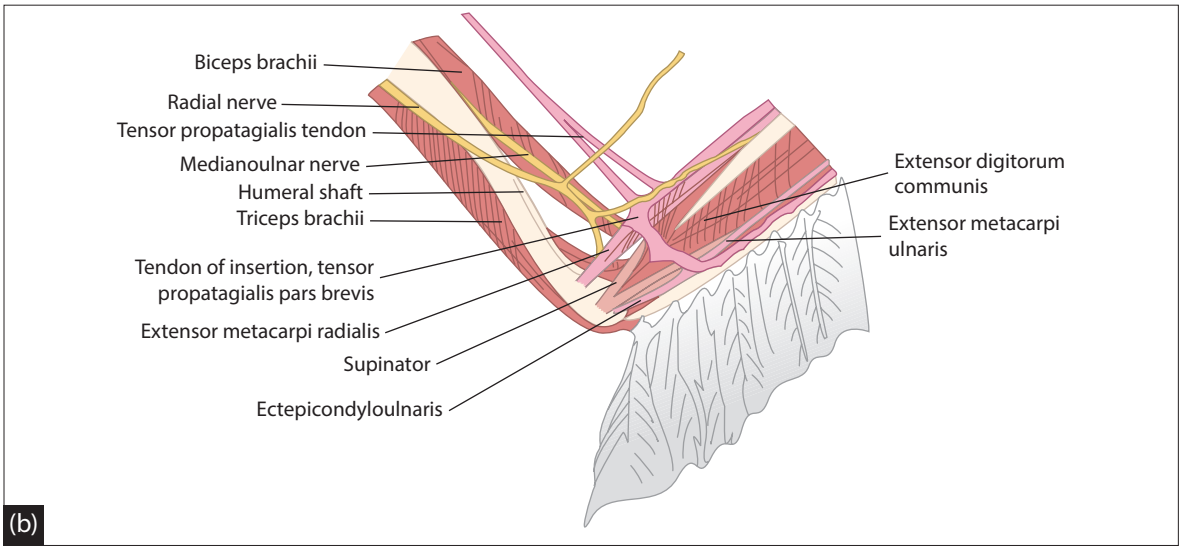
A dorsal approach is used for proximal radius and ulna fractures and elbow dislocation (**Fig. 29.19a–d**).

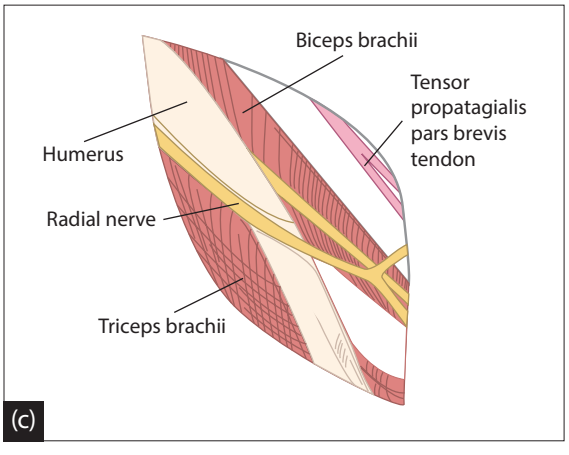
The bird is placed in sternal recumbency and the affected wing is extended. The feathers are plucked from mid humerus to the distal antebrachium.

A curvilinear skin incision is made from the distal humerus, between the radius and ulna, and extending as far as needed for exposure. Care should be taken to avoid branches of the radial nerve and the insertion of the tensor propatagialis pars brevis tendon. The supinator muscle is retracted cranially and the extensor digitorum muscle is retracted caudally. For better exposure of the radial head, the tendon of insertion of the tensor propatagialis pars brevis must be transected. If there is still not enough exposure, the supinator muscle is transected at its distal third, avoiding the deep radial nerve. The ulna can be exposed by incising between the retinaculum of the extensor metacarpi ulnaris and the muscle itself. The branch of the deep radial nerve and the interosseous dorsalis

Downloaded From Vet-eBooks.com







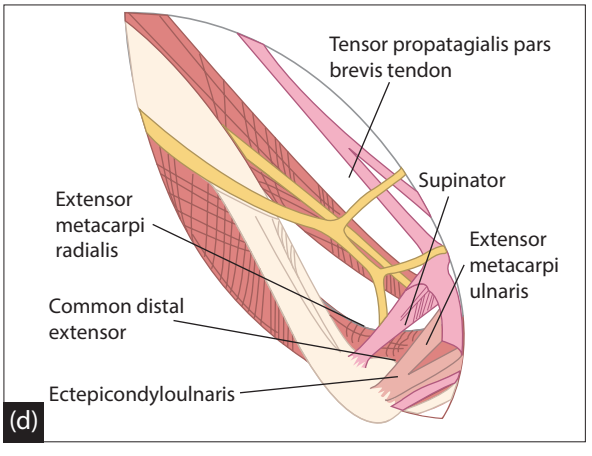


Figure 29.17 **Schematic sequence to illustrate a *dorsal* approach to the *distal humerus* (a). Avian anatomy of the distal humerus (dorsal view) (b). The initial incision, note the superficial structures (c) which is then extended distally to reveal structures overlying the dorsal elbow (d).**

Downloaded From Vet-eBooks.com

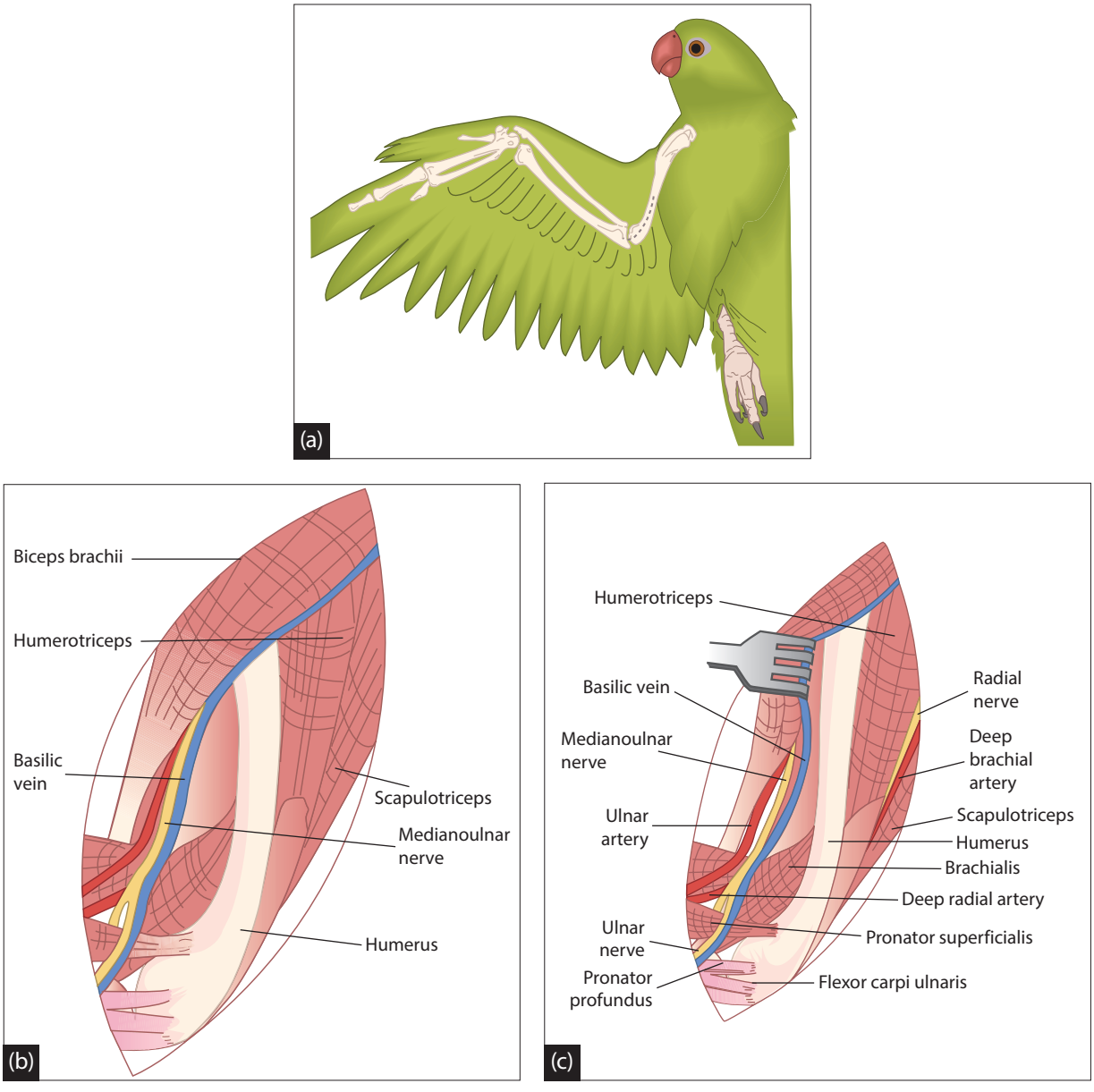


Figure 29.18 **Schematic sequence to illustrate a *ventral* approach to the *distal humerus* (a). Initial incision, note the superficial structures (b). Reflection of the biceps brachii gains better exposure of the humeral shaft (c). If necessary, the humerotriceps can be dissected off the caudal humerus for more exposure.**

artery and vein that emerge here and run along caudal aspect of ulna should be avoided.

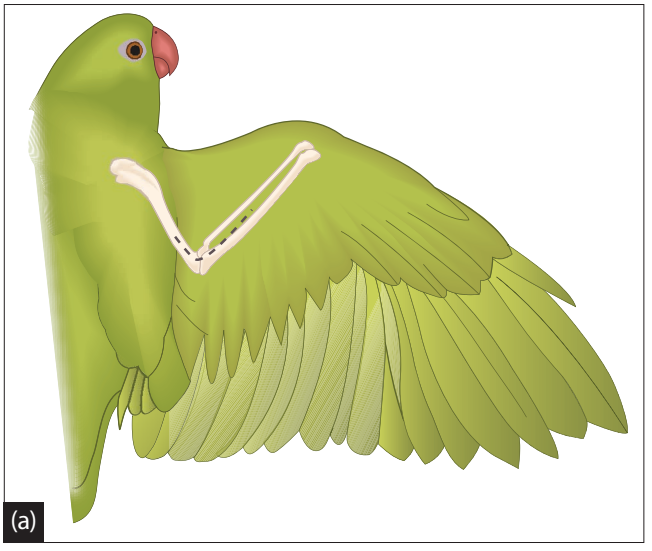
Distal radius and ulna

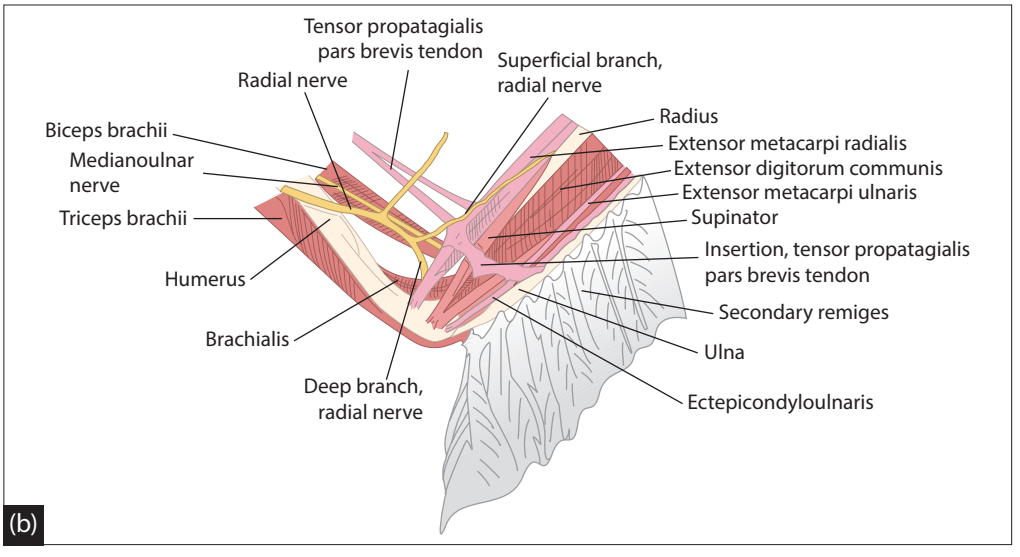
A dorsal approach is used for open reduction of fractures of the radius and ulna and luxations of these bones (**Fig. 29.20a–c**).

The bird is placed in sternal recumbency and the affected wing is extended. The feathers are plucked from the bone, but the secondary feathers are left intact.

The skin is incised between the radius and the ulna. If necessary, the extensor metacarpi radialis, on the cranial surface of the radius, and the extensor

Downloaded From Vet-eBooks.com





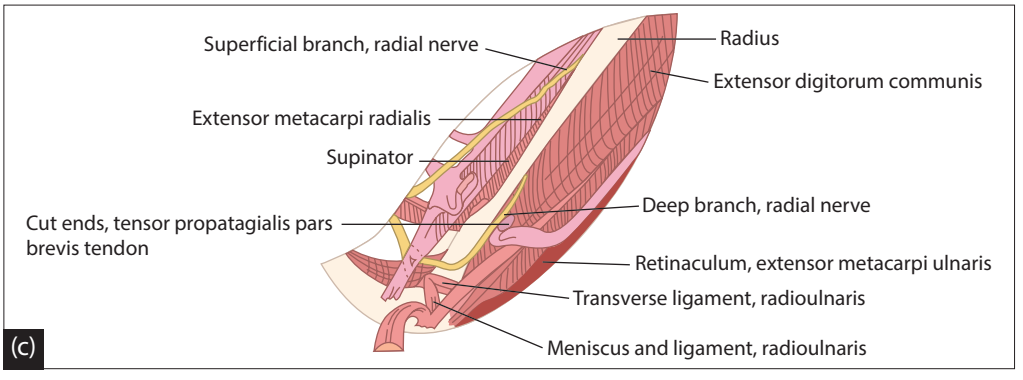


Figure 29.19 **Schematic sequence to illustrate a *dorsal* approach to the *proximal radius and ulna* (a). Anatomy of the elbow and proximal radius and ulna (dorsal view) (b). Dorsal approach to the proximal radius (c).** ***(Continued)***

Downloaded From Vet-eBooks.com

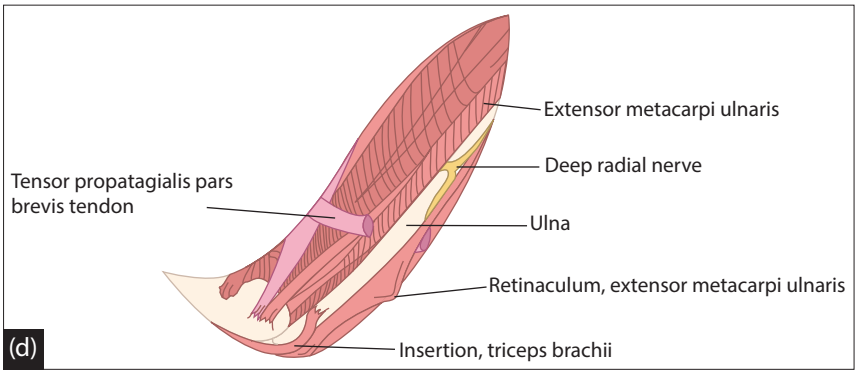
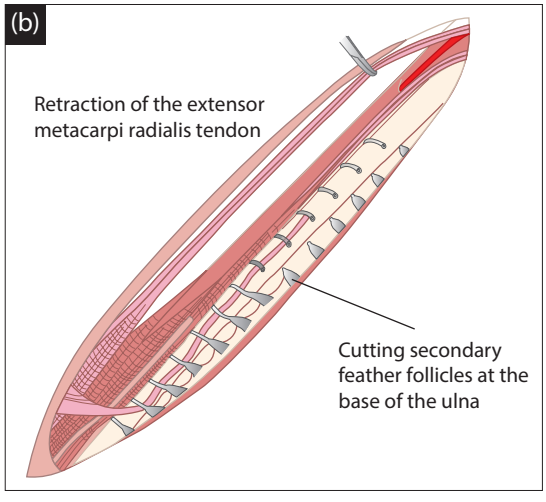


Figure 29.19 (Continued) **Dorsal approach to the proximal ulna (d).**





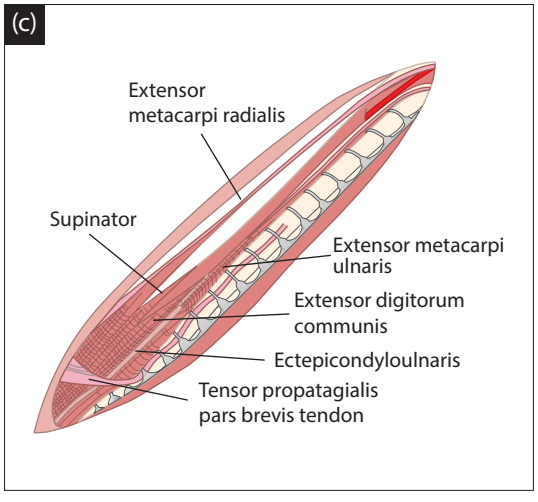


Figure 29.20 **Schematic sequence to illustrate a *dorsal* approach to the *distal radius and ulna* (a). Dorsal approach to the distal radius (b). Dorsal approach to the distal ulna (c).**

Downloaded From Vet-eBooks.com

metacarpi ulnaris, on the dorsal surface of the ulna, can be retracted. If it is necessary to repair the ulna, the calamus of the secondary feathers between bone and skin can be cut, avoiding the follicle.

For ulnar fractures, intramedullary pins are introduced through the fracture site, retrograded out of the olecranon (avoiding the elbow) and normograded into the distal fragment. They can then be tied into a TIF.

Radius

A ventral approach is used for distal radial fractures only (**Fig. 29.21a–d**). The dorsal approach is preferred for proximal fractures or ulnar fractures.

The bird is placed in dorsal recumbency and the affected wing is extended. The feathers are plucked from the ventral antebrachium.

The superficial ulnar artery is palpated distal to the elbow joint. Starting distally to this artery, an incision is made over the caudal aspect of the radius between the extensor metacarpi radialis muscle anteriorly and the extensor digitorum communis over the intra-osseous space. Numerous major arteries, veins and nerves, as well as muscle bellies and tendons, are in this area. Care must be taken not to damage them. In order to expose the radius, the belly of the pronator superficialis must be reflected cranially away from the pronator profundus, taking care to avoid the arteries, veins and nerves in this area.

If the fracture is displaced, intramedullary pins are inserted through the fracture site out towards the carpus (avoiding the joint) and then retrograded back through the proximal fragment.

Ulna

A dorsal approach can be used for simple ulna fractures.

The bird is placed in sternal recumbency. Covert and down feathers are plucked from the caudal and dorsal aspect of antebrachium, leaving the secondary feathers in place.

An intramedullary pin is inserted at right angles to the skin on the caudal aspect of the ulna between the second and third last secondary feathers. As the trochar of the pin cuts into the cortex, the angle of the pin is gradually changed so that it becomes aligned with the ulna as it penetrates into the medullary cavity. The pin can then be guided, by either a closed or open approach, into the distal fragment.

A slight variation of this technique is to use a larger pin to gain entrance to medullary cavity, and then introduce a blunt-tipped smaller pin. This prevents accidental penetration of the carpal joint.

Metacarpus

A ventral approach is preferred due to the insertion of primary feathers on the dorsal surface (**Fig. 29.22a–d**).

The skin is incised between two metacarpals. The abductor digiti majoris muscle lies between two major tendons (deep and superficial digital flexors). These two flexor tendons are retracted cranially and the muscle transected and reflected. Soft tissues, tendons and blood vessels are separated in order to approach the main, or primary, metacarpal bone.

Approaches to the bones of the leg

Femur

Two approaches are described. Vultures and gallinaceous birds have a well-developed iliotibialis lateralis muscle that covers the underlying structures; this muscle is not as well developed in parrots, raptors and owls.

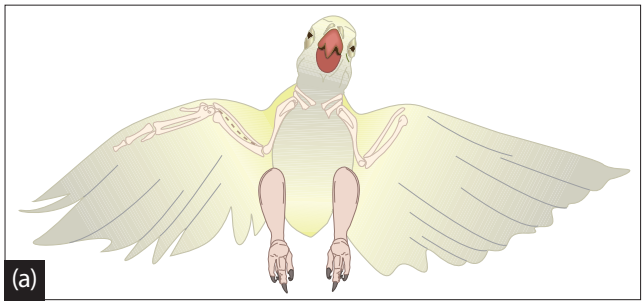
The bird is placed in partial sternal or lateral recumbency. The femoral shaft is palpated and an incision made over it (**Fig. 29.23a–c**) from the femoral trochanter to the lateral condyle. In vultures and chickens the iliotibialis lateralis is bluntly separated, being careful not to go too far caudally and damage the sciatic nerve. In parrots, the iliotibialis lateralis (cranially, overlying the femorotibialis externus) and the iliofibularis (caudally, overlying the sciatic nerve) are separated. Retraction of the iliotibialis lateralis reveals the femorotibialis externus (cranial) overlying the femoral shaft, and the iliofibularis lying caudal to it. The femorotibialis externus can be retracted cranially to expose the femur. The ischiatic vein lies caudal to the femur in the middle third.

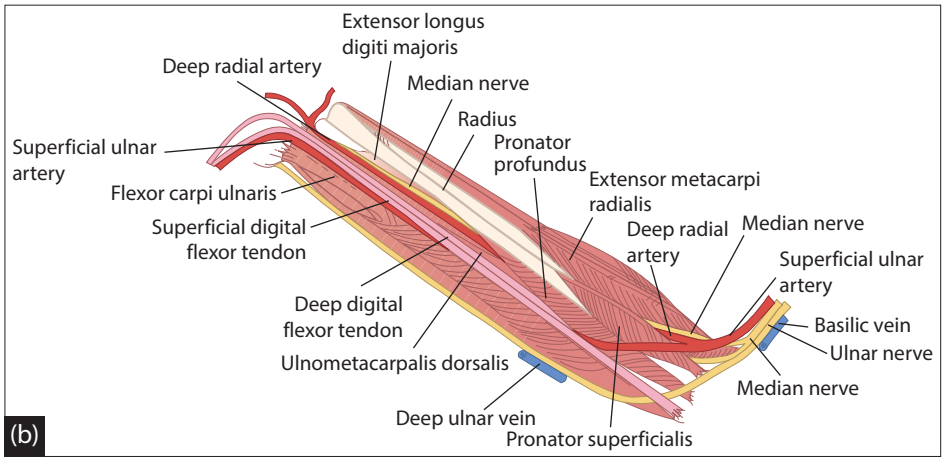
Tibiotarsus

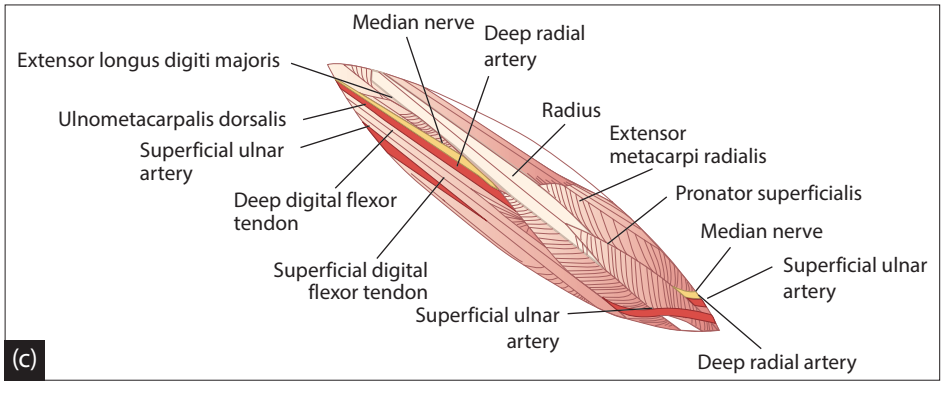
There are four repair methods:

- Intramedullary pins retrograded out of the hock, then back up towards the stifle.

Downloaded From Vet-eBooks.com







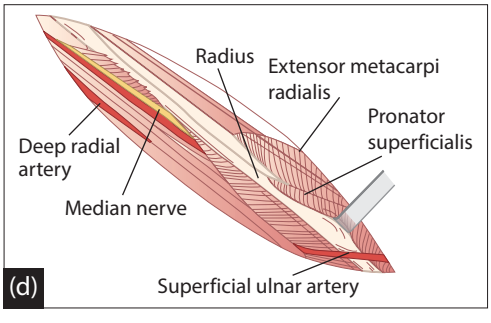


Figure 29.21 **Schematic sequence to illustrate a *ventral* approach to the *distal radius and ulna* (a). Anatomy of the distal radius and ulna (ventral view) (b). Initial incision, ventral approach to the distal radius and ulna (c). A deeper dissection exposes the distal radius (d).**

Downloaded From Vet-eBooks.com

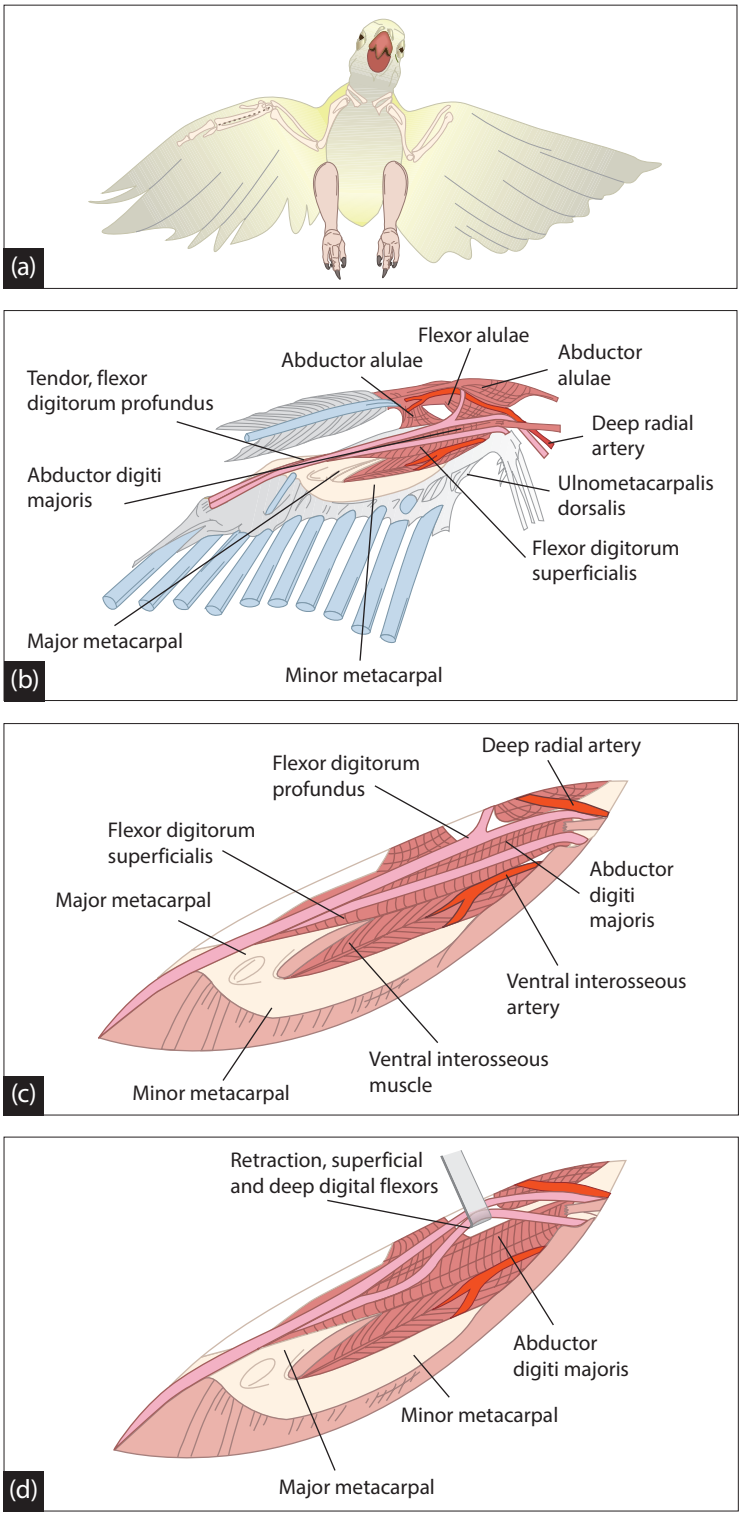


Figure 29.22 Schematic sequence to illustrate a *ventral* approach to the *metacarpus* (a). Anatomy of the metacarpal region, ventral view (b). Note the superficial structures of the ventral metacarpal region (c). Retraction of the deep and superficial digital flexors exposes the major metacarpal bone (d).

Downloaded From Vet-eBooks.com

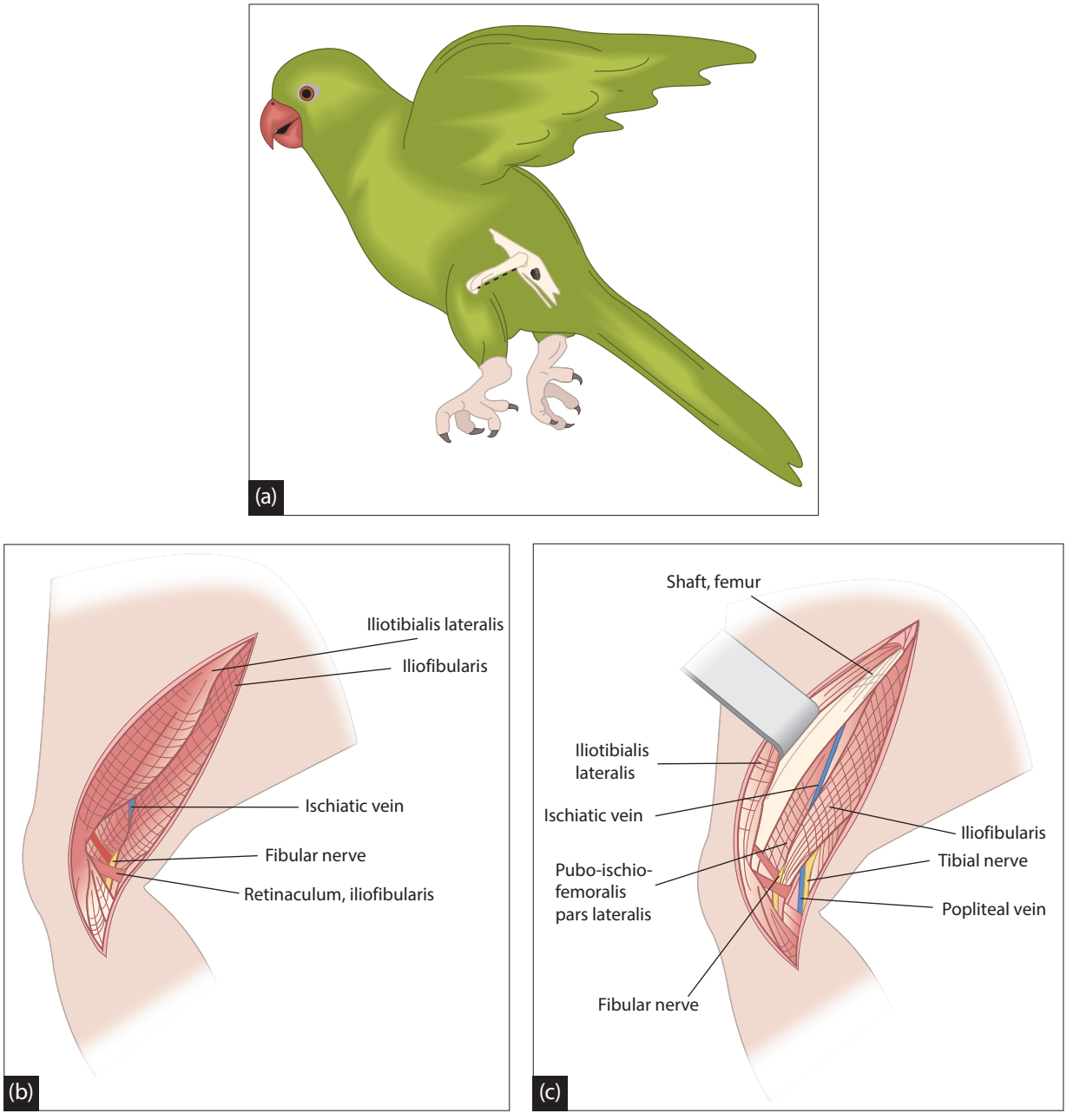


Figure 29.23 **Schematic sequence to illustrate a *lateral* approach to the *femur* (a). After the initial incision, note the superficial structures (b). Retraction exposes the shaft of the femur (c).**

- Intramedullary pins are introduced from the tibial crest and normograded down through the proximal fragments and then into the distal fragments.
- ESF.
- TIF incorporating the latter two techniques.

A medial approach is used because of the muscle bulk on the lateral side (**Fig. 29.24a–c**). Care must be taken, as the medial metatarsal vein crosses the hock and then runs caudally behind the tibiotarsal. Incise over the shaft of the tibiotarsus from just cranial to the hock to the medial femoral condyle.

Downloaded From Vet-eBooks.com

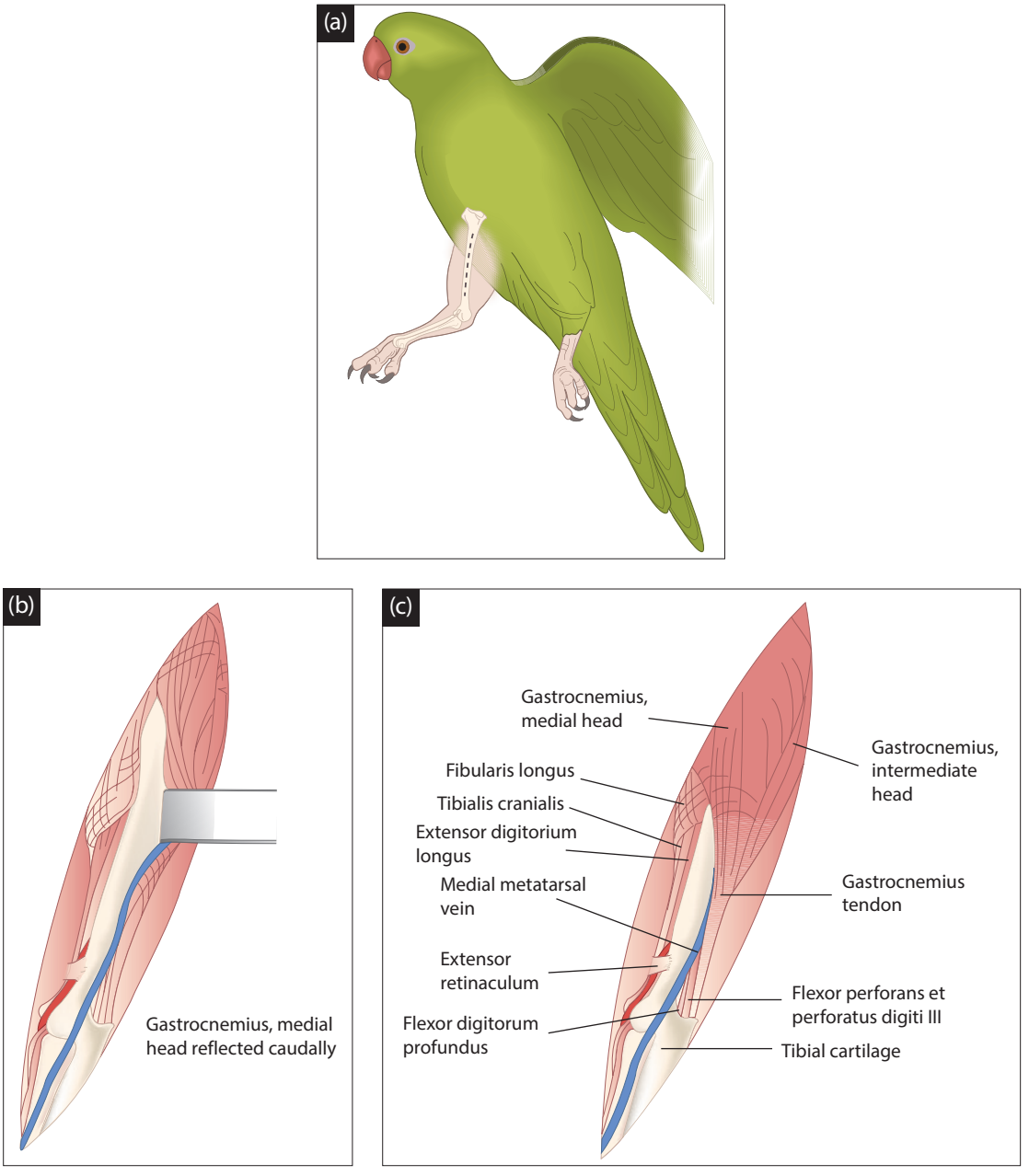


Figure 29.24 **Schematic sequence to illustrate a *medial* approach to the *tibiotarsus* (a). After the initial skin incision note the superficial structures (b). Retraction of the medial head of the gastrocnemius exposes the proximal shaft (c).**

The cranial complex of muscles (fibularis longus and tibialis cranialis) are separated from the medial head of the gastrocnemius. This division is easier to see in raptors than in parrots. To close the surgery site, this attachment is sutured before closing the skin.

Tarsometatarsus

Fractures of the tarsometatarsus tend to be open fractures and are best repaired with ESF. Closed fractures, especially in parrots, lend themselves to external coaptation.

Downloaded From Vet-eBooks.com

If intramedullary pins are used, they should be introduced through the fracture and exteriorised retrograde laterally or medially to the joint, then passed normograde back into the distal fragment. The bone is U-shaped in cross-section, with a groove up the back. Flexor tendons run in this groove; extensor tendons, arteries and nerve supply run on the cranial aspect of the shaft; veins are on the medial and lateral sides. A lateral approach is used, with an incision made along the shaft.

Joint surgery

Fractures and luxations of joints have a guarded prognosis for a full return to normal function.

Shoulder joint

The shoulder joint is not a very stable joint and luxation is not uncommon. It is often accompanied by an avulsion fracture of the ventral tubercle of the proximal humerus, and sometimes coracoid fractures; careful radiographic assessment is warranted. If the ventral tubercle has been avulsed, surgical reattachment of the tubercle with a K-wire often results in a stable joint. If the tubercle is not avulsed, closed reduction is often successful. Regardless of the technique used, the wing should be strapped to the body for 10–14 days.

Elbow joint

Luxation of the elbow may be dorsal, caudal or caudo-dorsal (ventral luxations are usually associated with fracture of the radius). The presence of open wounds and fractures has been associated with a poor prognosis for return to normal function.

Reduction is accomplished by flexing the elbow while internally rotating the radius and ulna and applying pressure to the dorsal (lateral) aspect of the radial head to force it into apposition with the dorsal (lateral) humeral condyle. The elbow is then gently extended; if the ligaments are not severely damaged the joint should click into place. If the joint is stable following reduction, it may be supported with a figure-of-eight bandage for 7–12 days.

If closed reduction is not possible or the luxation readily reccurs, open surgical reduction is recommended. Through a medial approach to the elbow joint, the common digital extensor is sutured to the scapulotriceps tendon distal to the elbow. A figure-of-eight bandage is then used to immobilise the joint for two weeks while it heals.

If further stabilisation is required, a transarticular external skeletal fixation device may be applied to maintain reduction. At least two pins are placed in the humerus and two in the ulna. The elbow is flexed into a normal folded position and the pins connected (proximal humeral pin to the distal ulnar pin and the distal humerus pin to the proximal ulnar pin).

Coxofemoral joint

Coxofemoral luxation is relatively uncommon, but can be repaired surgically. The bird is placed in lateral recumbency and feathers are plucked over the dorsolateral femur and pelvis.

A skin incision is made over the dorsolateral crest of the ilium, extending over the femoral trochanter. The iliotibialis lateralis muscle (cranially) and iliofibularis muscle (caudally) are separated from distal to proximally to the iliac crest, cutting the common tendinous insertion of these muscles on the ilium. The musculotendinous insertion of the iliofemoralis externus and the iliotrochantericus caudalis is transected, leaving enough tissue for reattachment. These two muscles lie dorsal to the acetabulum.

The bird is turned 180° to view the transparent membrane covering the joint, being aware of the branch of the femoral artery crossing this membrane and the femoral vein and nerve deep to it. The luxations can now be reduced and the joint capsule sutured in 2–3 locations. Transected muscles and tendons are reattached and stabilisation sutures are placed from the trochanter to the dorsolateral iliac crest (caudal to the central axis of the femur) and from the trochanter to the cranial rim of the acetabulum. The sutures are placed through the bone and, with the stifle maintained in a normal standing position, the sutures are tightened. These sutures prevent external rotation of the femur. The skin is closed routinely. Post-operatively the limb should be supported in a spica splint or a sling for 7–10 days. It is best to maintain the bird in a cage with smooth walls and a perch near the floor to discourage attempts to climb.

Downloaded From Vet-eBooks.com

An alternative to open reduction is to perform a femoral head and neck ostectomy (FHO). The approach to the hip described above is used and the femoral head and neck are removed with an appropriate sized osteotome, scissors or oscillating saw, and the bone smoothed with a rongeur or file. Stabilising sutures (as described above) are used to prevent external rotation of the femur.

Following FHO early use of the leg is encouraged to promote the formation of a fibrous pseudoarthrosis. Passive range of motion exercises can be started the day after surgery.

Femoral-tibiotarsal joint

Luxations of this joint are not uncommon and can be congenital or acquired. Three techniques have been described:

1. In young birds with a developmental luxation, IM pins are introduced, via a closed cranial approach to the joint, into the femur and the tibiotarsus such that the ends protrude through the cranial aspect of the joint. The joint luxation is reduced, the joint flexed into a normal perching/standing position, and the pins are locked together with acrylic (the excess lengths of pin are removed). This technique does damage to articular cartilage and infection may track along the pins, resulting in infection.
2. A type I or II ESF is constructed across the joint, with two pins in the femur and two in the tibiotarsus. Again, the joint is flexed into a normal perching/standing position. The pin in the proximal femur is connected to the pin in the distal tibiotarsus and the distal femoral pin is connected to the proximal pin in the tibiotarsus.
3. A lateral approach to the joint is used to expose the distal femur and the proximal tibiotarsus and reduce the luxation. A transverse hole is then drilled through the distal femur and the proximal tibiotarsus. A strong suture is placed through these holes (from lateral to medial in the distal femur and medial to lateral in the proximal tibiotarsus), creating a mattress suture that will provide collateral support (but not cranio-caudal stability) to the joint. If needed, a Type I ESF as described above can be placed to provide additional support.

After 2–4 weeks the pins can be removed; the joint has usually ankylosed and stabilised.

Tibiotarsal-tarsometatarsal joint

This is often a developmental luxation seen in young birds. The tarsometatarsus may be rotated through 90° such that the cranial aspect of the bone is now facing laterally or medially, with the toes reflecting this misalignment.

A lateral approach is made over the joint and the joint is opened with a stab incision using a scalpel. The articular surface of the joint can be debrided with a bone curette, and the tarsometatarsus gently but firmly rotated back into a normal position. The joint is flexed into a normal perching position and one or two K-wires are then introduced through the distal tibiotarsus into the tarsometatarsus to stabilise it. The joint and the skin are then closed and the foot bandaged. After three weeks the joint should have arthrodesed and the pins can be removed.

FURTHER READING

Altman RB (1997) General surgical considerations. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 691–703.

Altman RB (1997) Soft tissue surgical procedures. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 704–732.

Altman RB (1997) Radiosurgery (Electrosurgery). In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 767–772.

Bennett RA, Kuzma AB (1992) Fracture management in birds. *Journal of Zoo and Wildlife Medicine* **23**(**1**):5–38.

Bennett RA (1997) Orthopedic surgery. In: *Avian Medicine and Surgery*. RB Altman, SL Clubb, GM Dorrestein, K Quesenberry (eds). WB Saunders, Philadelphia, pp. 733–766.

Bennett RA, Harrison GJ (1994) Soft tissue surgery. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 1096–1136.

Bowles HL, Odberg E, Harrison GJ, Kottwitz JJ (2006) Surgical resolution of soft tissue disorders. In: *Clinical Avian Medicine*, *Vol 2*. GJ Harrison,

Downloaded From Vet-eBooks.com

TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 775–830.

Helmer P, Redig PT (2006) Surgical resolution of orthopedic disorders. In: *Clinical Avian Medicine*, *Vol 2*. GJ Harrison, TL Lightfoot (eds). Spix Publishing Inc, Palm Beach, pp. 761–774.

Hernandez-Divers SJ (2005) Minimally invasive endoscopic surgery of birds. *Journal of Avian Medicine and Surgery* **19**(**2**):107–120.

Martin HD, Ritchie BW (1994) Orthopedic surgical techniques. In: *Avian Medicine: Principles and Application*. BW Ritchie, GJ Harrison, LR Harrison (eds). Wingers Publishing, Lake Worth, pp. 1137–1170.

Orosz SE, Ensley PK, Haynes CJ (1992) *Avian Surgical Anatomy: Thoracic and Pelvic Limbs*. WB Saunders, Philadelphia.

Downloaded From Vet-eBooks.com

This page intentionally left blank

Downloaded From Vet-eBooks.com

CHAPTER 30

ONCOLOGY

INTRODUCTION

Advances in the prevention, diagnosis and treatment of infectious diseases, malnutrition, and other non-infectious diseases of birds have meant longer and healthier lives for companion birds. With this rise in longevity comes a rise in degenerative diseases including neoplasia. The incidence of neoplasia in bird is unclear; while uncommon it is considered that neoplastic diseases account for between 2–6% of cases presented to diagnostic laboratories.

It is now accepted that certain altered genes (oncogenes), whether inherited or caused by DNA damage, are a primary cause of neoplastic transformation in normal cells. In tumour cells, they are often found at high levels. While most normal cells undergo a programmed form of rapid cell death (apoptosis) when critical functions are altered, activated oncogenes can cause those cells to survive and proliferate instead. Most oncogenes require an additional step, such as mutations in another gene or environmental factors such as carcinogens (chemicals, irradiation), viral infections or dietary influences, to cause cancer.

There also appears to be a hereditary predisposition to some cancers, with some avian species are proportionally over-represented with certain neoplastic diseases, summarised in *Table 30.1* below.

TUMOUR CLASSIFICATION

Tumours can be classified by the cell types within the tumour:

- Carcinomas: malignancies of the epithelial cells.
- Sarcomas: malignancies of mesenchymal cells.
- Discrete or round cell neoplasms: the only common neoplasm of this type is lymphoid neoplasia.

(Note that poorly differentiated neoplasms produce cells having features of malignancy, but the cells are difficult to classify as carcinomas or sarcomas.)

They can also be classified by their tissue of origin:

- Ectodermal – skin, skin glands, mucous membranes.
- Endodermal – epithelium of pharynx, digestive tract, respiratory tract, urinary tract.
- Mesodermal – connective tissue, bone, cartilage, muscle, blood, blood vessels, lymphoid organs, notochord, pleura, pericardium, kidneys, gonads.
- Teratomas (neoplasm of mixed tissue types, none of which originate from the site where the tumour is found) and dermoid cysts (fibrous wall lined with stratified epithelium, containing feather follicles, glands, and nerve elements, usually found in the skin, ovary and eye) are generally considered rare.

Diagnosis

Physical examination

Many neoplasms (e.g. lipomas, fibromas) are discrete masses easily detected on physical examination. In other cases, where the neoplasia is more diffuse (e.g. multicentric lymphosarcoma), physical examination may reveal nothing more obvious than weight loss.

Clinical pathology

Anaemia (regenerative and non-regenerative) is often present with chronic neoplasia. The presence of neoplastic cells in a blood smear, as is seen with leukaemia, along with a leucocytosis can be diagnostic. In other cases of neoplastic disease there may be no inflammatory response.

Biochemistry changes may reflect organ development (e.g. in hepatic or renal neoplasia) but is not pathognomonic for neoplasia.

Downloaded From Vet-eBooks.com

Table 30.1 **Commonly seen tumour types by bird species**

BIRD SPECIES	TUMOUR TYPES
African grey parrot (*Psittacus erithacus*)	• Cutaneous papilloma • Squamous cell carcinoma
Amazon parrot (*Amazona* spp.)	• GI papillomas, adenocarcinoma • Bile duct carcinoma • Lipoma
Budgerigar (*Melopsittacus undulatus*)	• Renal adenocarcinoma • Thyroid neoplasia • Ovarian/oviductal adenocarcinoma • Sertoli cell tumour • Lipomas • Xanthomas • Fibroma/fibrosarcoma • Uropygial adenoma/adenocarcinoma
Cockatiel (*Nymphicus hollandicus*)	• Ovarian/oviductal adenocarcinoma • Squamous cell carcinoma • Fibroma/fibrosarcoma
Cockatoo (*Cacatuidae* spp.)	• Soft tissue sarcoma • Cloacal polyps • Lipoma
Lovebird (*Agapornis* spp.)	• Soft tissue sarcoma • Fibrosarcoma • Lymphosarcoma
Macaw (*Ara, Anodorhynchus, Cyanopsitta, Primolius, Orthopsittaca*, and *Diopsittaca* spp.)	• GI papillomas • Adenocarcinoma
Galah or rose-breasted cockatoo (*Eolophus roseicapilla*)	• Lipomas

Diagnostic imaging

Radiology, ultrasound, CT and MRI are all useful adjuncts in the diagnosis of neoplastic disease.

Molecular diagnostics

Given the known involvement of certain viruses (e.g. herpesvirus) in the development of some neoplastic diseases, the use of molecular diagnostic testing such as PCR can assist in the determining the presence or aetiology of some neoplastic diseases.

Biopsy and cytology

Once the presence of a neoplastic mass has been confirmed or suspected, the use of cytology, incisional biopsy, or excisional biopsy can assist in further defining the disease process. As well as routine staining techniques, the use of immunohistochemistry and other techniques can be used to classify the disease more accurately, often guiding the selection of treatment.

Treatment options

Generally it can be said that treatment options for avian neoplasia currently include:

- Surgical excision, including amputation (**Fig. 30.1**).
- Surgical excision with adjuvant chemotherapy.
- Chemotherapy.
- Radiation.
- Photodynamic therapy.

Surgery, including cryosurgery, is usually reserved for localised and accessible skin or limb neoplasia. It is important to ensure that adequate margins should be taken but as this is not always

Downloaded From Vet-eBooks.com



Figure 30.1 **Radiograph of a galah reveals an osteosarcoma in the humerus. Note that such a lesion will require amputation, possibly with adjuvant chemotherapy.**

feasible without amputation, adjuvant chemotherapy or radiation is often used. Knowing the tumour type and behaviour is essential for predicting the likelihood of metastatic disease.

The basic concept of chemotherapy is that the drugs used target rapidly dividing cells, making cancer cells most susceptible to chemotherapy when actively dividing. However, not only do these drugs target the tumour cells, they may also affect the bone marrow, gastrointestinal mucosa and the feather follicle cells causing unwanted side-effects. When cancer cells are not dividing, they are partially/completely resistant to chemotherapy, making it often wise to combine drugs that have differing actions to maximise the chances of successful treatment with little/no resistance developing.

There are several classes of chemotherapeutic drugs summarised in *Table 30.2* below.

Radiation therapy uses an external beam of radiation to destroy the proliferative capacity of the tumour without irreparable damage to surrounding normal tissues. This is not always possible because of the radiation response of the surrounding normal tissue, which often dictates the maximum tolerable dose that may be administered to a patient. There are two forms of external beam radiation therapy, classified based on the energy of the photon emitted. They are

- Orthovoltage radiation, a lower energy form of radiation which is more beneficial for treating surface tumours but acute radiation side-effects can be severe and quite uncomfortable for the patient.
- Megavoltage radiation, a higher energy radiation, is obtained from linear accelerators and cobalt machines. The penetrating ability of megavoltage radiation allows radiation therapy to be applied to deeper tumours for which orthovoltage would not be an option, as well as superficial tumours. Megavoltage radiation evenly distributes the radiation dose throughout the tissues in the radiation treatment field with a predictable interaction in tissues.

Another form of external radiation is plesiotherapy, i.e. beta (electron) irradiation with a Strontium-90 applicator via direct contact. Electrons produced have minimal penetration (<1 mm) and as such can only be used for very early, small superficial lesions such as squamous cell carcinoma. Strontium therapy can be repeated since there is minimal normal tissue damage. This form of therapy has been used to successfully treat uropygial squamous cell carcinomas in budgerigars (**Fig. 30.2**).

Key elements to consider when planning external beam radiation include:

- *Total radiation dose:* This is generally based on the tumour being treated and what normal tissues may be affected. Recommendations are usually based on treatment regimens for dogs and cats.
- *Fractionation:* Dividing the total dose into increments, which reduces long-term radiation side-effects
- *Treatment time:* Treatment over longer periods of time may help spare acutely affected tissues, but may allow for tumour re-population

Downloaded From Vet-eBooks.com

Table 30.2 **Summary of available chemotherapy drugs by class**

CLASS	EXAMPLES	ACTION	USES	COMMENTS
Vinca alkaloids/ anti-mitotics	Vincristine	Arrests cell division in metaphase.	• Leukaemic neoplasia myeloma • Soft tissue sarcomas • Mast cell tumours	• If given IV – perivascular irritation can be severe. • Excreted in bile.
Anti-tumour antibiotics	Doxorubicin	Binds to DNA and inhibits synthesis of nucleic acids and cell division.	• Carcinoma • Sarcoma • Round cell tumours	• If given IV – irritant if extravasated. • Adverse effects include: • Bone marrow suppression. • GI tract effects. • Cardiotoxic effects. • Nephrotoxic effects. • Hypersensitivity reactions. • Excreted in bile.
Alkylating agents	Cyclophosphamide Chlorambucil Lomustine	Combines with DNA, damaging it.	• Lymphomas • Carcinomas • Sarcomas	• Can be given orally or IV. • Myelosuppressive. • Inactivated in liver and excreted in urine
Platinum drugs	Cisplatin Carboplatin	Inhibits DNA synthesis and may have some alkylating effects.	• Osteosarcoma • Carcinoma	• Given IV or intra-lesional. • Can be nephrotoxic, myelosuppressive, and neurotoxic. • Carboplatin is not as toxic as cisplatin. • Renal excretion.
Other	L-Asparaginase	Asparaginase catalyzes the conversion of L-asparagine to aspartic acid and ammonia, depriving the leukemic cell of circulating asparagine, which leads to cell death.	• Leukaemia and lymphosarcoma	• Can be given IM or SC.



Figure 30.2 **Budgerigar with a preen gland carcinoma which should respond well to Strontium-90 therapy.**

Photodynamic therapy utilises a plant extract or synthetic drug which, after injection, is selectively taken up by neoplastic cells. When irradiated by a laser, there is a reaction that results in cellular death. However, this technique requires a dedicated laser and direct access to the tumour, making it unsuitable for deep tumours. Patients may be photosensitive for 1–2 weeks after and need to be kept out of direct light. There are only limited reports of its use in avian medicine, but it has been used to treat a sarcoma successfully.

FURTHER READING

Reavill DR. (2004) Tumors of pet birds. *Veterinary Clinics of North America: Exotic Animal Practice* **7**:537–560.

Zehnder AM, Hawkins MG, Koski M, Kent MS. (2010) Therapeutic considerations for squamous cell carcinoma: an avian case series In: *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, p. 71.

Downloaded From Vet-eBooks.com

APPENDIX 1: FORMULARY

EMERGENCY DRUGS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Anti-muscarinic agent used to treat bradycardia.	Atropine	0.02 mg/kg IM or IV	May thicken respiratory secretions.
	Glycopyrrolate	0.01 mg/kg IV	May thicken respiratory secretions.
Alpha- and beta-adrenergic agonist agent used systemically for treating anaphylaxis and cardiac resuscitation.	Adrenaline (epinephrine)	0.01–0.1 mg/kg IV	Care with hypovolaemic patients (not a substitute for adequate volume replacement).
CNS stimulant used to stimulate respiration.	Doxapram	2 mg/kg IM, IV	Not a substitute for ventilatory support. It will not increase respiratory frequency, but will increase tidal volume and oxygen consumption leading to decreased oxygen saturation. Consequently its use is often not recommended.
Opiate antagonist.	Naloxone	0.01–0.04 mg/kg IV q2–3 minutes to effect.	At reversal dosages, naloxone can negate opioid analgesic effects. The reversal effect may last for a shorter time than opioid effect, so repeated dosing may be needed.
Benzodiazepine antagonist.	Flumazenil	0.05–0.1 mg/kg IV *or* IM	Flumazenil is used for the reversal of benzodiazepine effects after overdoses or toxic effects.
Fluids	Crystalloid fluids (0.9% saline)	10 ml/kg IV bolus	Shock therapy.
	Colloid fluids	5 ml/kg IV bolus	Shock therapy.

Downloaded From Vet-eBooks.com

ANTIBIOTICS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Penicillin • Bactericidal. • Well distributed in extracellular spaces especially in inflamed tissues. • Do not readily penetrate the eye and CNS. • Excreted by the kidneys, largely unchanged, therefore high concentrations can be found in the urine. • Allergic reactions (anaphylaxis) have been reported, possibly from the procaine component in procaine penicillin. • Potentially synergistic in combination with aminoglycosides.	Amoxicillin	100 mg/kg q8 hours 200–800 mg/l water	Effective against Gram-positive bacteria, especially *Staphylococcus* spp.; most (all) Gram-negative bacteria are resistant.
	Ampicillin	100 mg/kg IM q6 hours	
	Amoxicillin/ Clavulanic acid	125 mg/kg PO q8–12 hours	Clavulanic acid inhibits beta-lactamase (bacterial enzyme that inactivates many penicillins).
		60–120 mg/kg IM q12 hours	
	Carbenicillin	100–200 mg/kg IM q8 hours	Improved spectrum against *Pseudomonas* spp., and other Gram-negative bacteria. Oral form has poor bioavailability.
	Ticarcillin	200 mg/kg IM q8 hours	Similar to carbenicillin; much more active against *Pseudomonas* spp. Also found in combination with clavulanic acid and used at the same dose. Parenteral administration only.
	Piperacillin	100–200 mg/kg IM q8–12 hours	Good activity against most Gram-negative bacteria including *Pseudomonas* spp., *Klebsiella* and *Enterobacter.* Piperacillin with tazobactam is commonly used to overcome beta-lactamase containing bacteria. Parenteral administration only.
	Piperacillin/ tazobactam		
Cephalosporins • Three generations effective against both Gram-positive and Gram-negative bacteria. • Well distributed in extracellular spaces. • Do not readily penetrate eye and CNS, except for cefotaxime and ceftazidime. • Synergistic with aminoglycosides.	Cephalothin	100 mg/kg IM q6 hours	First generation cephalosporin. Effective against most Gram-positive cocci, many Gram-negatives and some anaerobes.
	Cephalexin	50–100 mg/kg IM *or* PO q6–12 hours	
	Cefoxitin	75–100 mg/kg IM q8–12 hours	Second generation: Increased Gram-negative activity.
	Ceftazidime	50–100 mg/kg IM q6–8 hours	Third generation. Penetrates CSF.
	Ceftiofur	10–20 mg/kg IM q12 hours	Third generation with activity against *Pasteurella, E coli, Streptococcus, Staphylococcus* and *Salmonella* spp.
	Cefotaxime	75–100 mg/kg IM q8–12 hours	Third generation. Expanded Gram-negative spectrum. Penetrates CSF.

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Chloramphenicol • Bacteriostatic. • Highly lipid soluble, therefore good tissue penetration, including CNS and eye; tissue concentrations often exceed serum levels. • Reversible dose-related anaemia, CNS depression, loss of appetite has been reported in chickens, turkeys and ducks. • Caution with people handling the drug. • Not to be used in food-producing animals.	Chloramphenicol palmitate (oral); Chloramphenicol succinate (injectable)	50–75 mg/kg IM *or* PO q6–12 hours	Broad spectrum against *Chlamydophila*, *Mycoplasma*, Gram-positive and Gram-negative bacteria, and some protozoa; many avian Gram-negative isolates are resistant. Oral form (palmitate ester) – erratic blood concentrations. Injectable forms (succinate, propylene glycol based) – more predictable serum concentrations.
Aminoglycosides • Bactericidal. • Synergistic with penicillins and cephalosporins. • Excellent spectrum against Gram-positive and Gram-negatives. • Ineffective against anaerobic bacteria and in proteinaceous environments such as abscesses and exudates. • Poor penetration into CNS and eye. • Nephrotoxic; in dehydrated birds and birds with compromised renal function the dose should be reduced or a less toxic drug selected.	Amikacin	10–20 mg/kg IM q12 hours	• Greater activity against Gram-negatives including some resistant to gentamycin and tobramycin. • Achieves higher serum concentrations than gentamycin but is less toxic so levels are better tolerated. • Less toxic side-effects and is the aminoglycoside of choice for use in birds.
	Gentamycin	5–10 mg/kg q12 hours	More toxic than amikacin. Not generally recommended due to narrow margin of safety.
	Tobramycin	5 mg/kg q12 hours	Pharmacology is similar to gentamycin but has greater activity against *Pseudomonas* spp. Neurotoxicity and nephrotoxicity may develop.
Quinolones • Bactericidal. • Most avian Gram-negative pathogens, some Gram-positive pathogens, most *Mycoplasma* and possibly *Chlamydophila* are sensitive. • Not effective against anaerobes. • Achieves high levels everywhere especially in the liver and urinary tract; tissue concentrations may exceed serum concentrations. • May cause permanent articular defects in juveniles–use with extreme caution in growing birds. • Scattered anecdotal reports of aggressive, irritable behaviour in Amazon parrots treated with quinolones. • IM injection causes pain and necrosis at the site of injections. • Toxic reactions may be species-specific.	Enrofloxacin	10–30 mg/kg IM or PO q12 hours	• Excellent activity against *Mycoplasma*, some Gram-positive and most Gram-negative bacteria. However, *Pseudomonas* spp., resistance is common. Not suitable for anaerobic infections • May have anti-chlamydial activity but while treatment eliminates clinical signs it may not clear the carrier state. • Can cause vomiting in raptors when given orally. • If given in water, enrofloxacin may not achieve therapeutic levels except for highly susceptible bacteria. • Only a single IM injection should be given, as repeated injections cause significant bruising and muscle necrosis.
	Ciprofloxacin	15–40 mg/kg PO q12 hours	Antibacterial spectrum similar to enrofloxacin.
	Marbofloxacin	2.5–15 mg/kg PO or IM q24 hours	Used in raptors, as it does not appear to cause nausea.
	Orbifloxacin	15–20 mg/kg PO q24 hours	Antibacterial spectrum similar to enrofloxacin.

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Trimethoprim-sulpha derivatives • Bacteriostatic. • Excellent Gram-positive and Gram-negative spectrum. • *Pseudomonas* spp., and some strains of *Enterobacteriaceae* are resistant. • May be effective against some *Coccidia* spp. • Has been used in combination therapy for *Sarcocytis* infection. • Wide extracellular distribution. • Some birds (especially macaws) suffer GI upset and will regurgitate 1–3 hours after an oral dose. Incidence can be reduced if the drug is added to a small amount of food. • Sulphas are excreted via the same pathway as uric acid. In dehydrated birds and those with compromised renal function sulphas may form crystals and damage renal glomeruli. Therefore if serum uric acid is elevated, select another drug.	Trimethoprim-sulphadiazine (veterinary formulations).	50–100 mg/kg (of combined product) PO q12 hours *or* 8–20 mg/kg IM q12 hours *or* 475–950 mg/l water for 5–7 days	Sterile abscesses and irritation have been reported following the use of the injectable veterinary products.
	Trimethoprim-sulphamethoxazole (medical formulation).	100–100 mg/kg (of combined product) PO q12 hours	
Tetracyclines • Bacteriostatic. • Spectrum includes many Gram-positive organisms; poor efficacy against most avian Gram-negative isolates. • Used for *Chlamydophila* and *Mycoplasma*. • Wide volume of distribution. • Side-effects: • Anorexia, vomiting, diarrhoea. • Immunosuppression. • Hepatotoxicity (rare). • Localised tissue reactions to doxycycline injection formulations. • Alteration of gut flora especially yeast overgrowths and secondary bacterial infections. • Chelates calcium in gut and bone; dietary calcium interferes with the oral uptake of tetracyclines. Use cautiously in baby birds for extended periods of time.	Oxytetracycline	10–50 mg/kg IM once every 3–5 days	• Poorly absorbed when given orally, therefore oral formulations are not recommended. • IM well absorbed and widely distributed, but it is irritating and will cause necrosis at the injection site.
	Chlortetracycline	40–50 mg/kg PO q12 hours *or* 1500 ppm in food or water	• Poor acceptance. • Immunosuppressive. • Can be used for flock treatment of chlamydiosis, but doxycycline is preferred.
	Doxycycline	25–50 mg/kg PO q24 hours *or* 100–500 mg/l water (African grey parrots may require doses up to 800 mg/l) *or* 60–100 mg/kg IM once weekly	• More lipophilic than other tetracyclines – absorbed better and faster through the GI tract and greater bioavailability. • May offer significant advantages over other tetracyclines for treating chlamydiosis.

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Macrolides and lincosamides • Bacteriostatic. • Indicated for *Pasteurella*, *Bordetella*, some Mycoplasma, *Campylobacter* spp. and *Clostridia* spp. and obligate anaerobic bacteria. • Often used for susceptible URTI and osteomyelitis. • Active against Gram-positive organisms and anaerobes, but virtually all aerobic Gram-negative bacteria are resistant.	Erythromycin	60 mg/kg PO q12 hours	Active against chlamydiosis in people but not effective at the dose levels used in birds. Injectable form may cause severe tissue irritation.
	Clindamycin	25–150 mg/kg q12 hours	Used occasionally to treat osteomyelitis caused by susceptible Gram-positive pathogens. Only oral forms are used in avian medicine because of injection site necrosis with injectable formulations.
	Lincomycin	75–100 mg/kg q12 hours *or* 500–750 mg/l water	Usually combined with spectinomycin. It has been used to treat respiratory and GI infections caused by Gram-positive bacteria and *Mycoplasma*.
	Azithromycin	50–80 mg/kg PO q24 hours	New generation macrolide; it appears to be active against intracellular infections including *Chlamydophila, Toxoplasma, Plasmodium* and *Cryptosporidium.*
	Tylosin	10–40 mg/kg IM q8–12 hours *or* 200–500 mg/l water	Used predominantly for suspected *Mycoplasma* infections and *Pasteurella.* Intramuscular injections can be very irritating.
Metronidazole • Bactericidal. • Effective against many Gram-positive and most Gram-negative obligate anaerobes. • Ineffective against aerobic bacteria. • Highly effective against many motile protozoa. • Well absorbed from GI tract. • Highly lipophilic and penetrates bones, CNS and abscesses.	Metronidazole	10–50 mg/kg PO q12–24 hours	Frequently used for anaerobic infections, (including *Clostridial* infections) and motile protozoa (*Trichomonas, Giardia* and *Cochlosoma* spp.)

Downloaded From Vet-eBooks.com

ANTIFUNGAL DRUGS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Azoles • Inhibits ergosterol synthesis, increasing cellular membrane permeability; also causes secondary metabolic effects and growth inhibition. • Fungistatic, so months of therapy are often required. • Take several days to reach steady-state concentrations.	Ketoconazole	20–30 mg/kg PO q12 hours for 10–30 days. It should be administered with food.	• For *Candida* infections. • Probably not as effective with *Aspergillus* infections as other azoles. • Widely distributed to tissues, but is highly protein-bound and does not penetrate CNS or eye fluids. • Water insoluble unless dissolved in acid first. • Toxic side-effects seen in mammals are rarely seen in birds, but may be associated with hepatotoxicity.
	Enilconazole	Nebulisation solution; enilconazole. 100 mg:9 ml DMSO:90 ml NaCl Topically: dilute 1:10 and apply q12 hours	• Administered topically or nebulised.
	Fluconazole	1.5–10 mg/kg PO q12 hours *or* 10–20 mg/kg PO q24–48 hours	• Effective against *Candida* (especially mycelial form), *Aspergillus* and *Cryptococcus* spp. • Penetrates eye, CNS and CSF • Best safety margin of the azoles.
	Itraconazole	5–10 mg/kg PO q24 hours given with food for at least one month after signs have resolved.	• Used for gastrointestinal and cutaneous candidiasis, dermatophyte infections. • Maybe more effective than other azoles and amphotericin-B in *Aspergillus* infections. • Poorly distributed to CSF, ocular fluids and plasma (tissue concentrations are higher than plasma). • Although less toxic than amphotericin-B, it appears that African grey parrots may be sensitive to this drug, causing anorexia and depression. It is not recommended to use it in this species, or to use it at 2.5–5 mg/kg PO q24 hours.
	Miconazole	5 mg/kg intra-tracheally q12 hours five days *or* Apply topical gel q12 hours	• Can be used topically, intra-tracheally, or nebulised. • Many toxic side-effects: cardiac arrhythmias; cardiac arrest if given IV too quickly.
	Clotrimazole	2 mg/kg intra-tracheally q24 hours *or* 10 mg/ml NaCl as a nasal flush q12 hours	• Poorly absorbed, therefore topical use or nebulised only. • Effective against *Aspergillus* spp. • It may cause local irritation.
	Voriconazole	12–18 mg/kg PO q12 hours. Higher doses may be needed to maintain plasma concentrations during long-term treatment	• High oral bioavailability. • Can cross into the CNS.

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Terbinafine • An allylamine; inhibits squalene epoxidase, an enzyme that is critical for fungal sterol and cell wall synthesis.	Terbinafine	10–15 mg/kg PO q12–24 hours *or* Nebulisation (1 mg/ml; 500 mg terbinafine + 1 ml acetylcysteine + 500 ml distilled water) for 30 minutes q8–12 hours.	• *Aspergillus* and dermatophytes. • Good oral absorption; distributed well to fat and skin.
Polyenes • Acts by binding to sterols (primarily ergosterol) in the cell membrane and alters the permeability of the membrane allowing the loss of intracellular potassium and other cellular constituents.	Amphotericin B	1.5 mg/kg IV q8 hours for 3–5 days *or* Intra-tracheally 1 mg/kg q12 hours *or* Nebulise at 0.3–1 mg/ml for 15 minutes q6–12 hours *or* 100 mg/kg PO q12 hours 10–30 days for the treatment of *Macrorhabdus ornithogaster.*	• Fungicidal. • Active against both yeast and hyphal fungi. • Used orally for the treatment of *Macrorhabdus ornithogaster*. • Not absorbed orally; for aspergillosis it must be administered IV, topically, or by nebulisation. • Widely distributed when given IV, but only minor systemic absorption with aerosol or topical administration. • Nephrotoxicity is common in mammals but uncommonly reported in birds. • Topical solutions must be diluted before flushing into closed spaces (e.g. sinuses) to prevent irritation. • Combine with itraconazole for best response.
	Nystatin	200,000–300,000 units/kg PO q8–12 hours	• Not systemically absorbed. • Must come into contact with yeast to be effective, therefore tube feeding may bypass oral lesions. • Do not mix with hand-rearing formula to ensure concentration and contact time is maximised.
Fluorinated pyrimidines • inhibit macromolecule synthesis	5–fluorocystine (Flucytosine)	60 mg/kg PO q12 hours for birds >500 g; 150 mg/kg PO q12 hours for birds <500 g	• Rarely used. • Can be used with amphotericin B to treat aspergillosis. • Resistance develops rapidly when used alone.

MYCOBACTERIAL TREATMENT PROTOCOLS

- Protocols involve combinations of antituberculous drugs, as resistance to a single drug can develop rapidly.
- Because these organisms can only be killed during replication, which occurs once every 16–20 hours, treatment often lasts nine months or longer. Organisms can also persist for months in caseous lesions and macrophages where replication seldom occurs.
- Therapy is aimed at achieving the highest possible blood levels of drug rather than maintaining constant blood levels. Therefore, the highest tolerable doses of drugs are given once daily.
- Treatment protocols include the following combination of drugs, most given orally once daily:
  - Amikacin 20 mg/kg q24 hours IM; Enrofloxacin 30 mg/kg.
  - Enrofloxacin 30 mg/kg; Ethambutol 30 mg/kg; Rifabutin 15 mg/kg.
  - Clarithromycin 55 mg/kg; Rifabutin 6 mg/kg; Ethambutol 30 mg/kg; Enrofloxacin 30 mg/kg.
  - Clarithromycin 55 mg/kg; Rifabutin 15 mg/kg; Ethambutol 30 mg/kg; Enrofloxacin 30 mg/kg.
  - Clarithromycin 55 mg/kg; Rifabutin 15 mg/kg; Ethambutol 30 mg/kg.

Downloaded From Vet-eBooks.com

ANTIVIRAL DRUGS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Acyclovir • Used for treating outbreaks of herpesvirus infection.	Acyclovir	80–330 mg/kg PO q8 hours for a minimum of seven days	• Works better on in-contact birds rather than affected birds. • Injectable form is strong irritant.
Interferon • Group of small protein and glycoprotein cytokines naturally produced by the immune system following natural infection or vaccination. • Interferons protect the bird by suppressing cell proliferation, inhibiting viral replication and augmenting the activity of macrophages and T lymphocytes. • They are species specific and a mammalian interferon would not be expected to have a significant action in birds. However, cross-species reactivity has been reported in birds.	Avian Interferon	1,000,000 units IM q24 hours 90 days *or* 1,000,000 units IM every 2–7 days for three treatments	• Initially the use of interferon was limited due to the difficulty in manufacturing the protein in large enough quantities but the recent development of recombinant DNA technologies has made interferon economic and easy to produce. • Not commercially available at time of writing.

ANTIPROTOZOAL DRUGS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Nitroimidazoles • Used against motile protozoa (*Trichomonas, Giardia, Cochlosoma, Spironucleus, Histomona* spp.). • *Candida* overgrowth after prolonged therapy can occur, especially in cockatiels.	Carnidazole	20–30 mg/kg PO once	• Safe with good efficacy, but must be given on an empty stomach otherwise vomiting may occur.
	Ronidazole	6–10 mg/kg PO *or* 60–600 mg/l water for seven days	• Treatment of choice for *Trichomonas*; dose can be increased to 600 mg/l water if resistance develops.
	Dimetridazole	200–400 mg/l water for five days	• Toxic if overdosed: depression, anorexia, ataxia, seizures (incorrect water preparation or excessive water intake, e.g. hot weather, feeding chicks). • Do not use in Pekin robins and finches. Use with care in lorikeets and mynahs (use lower dose).
	Metronidazole	50 mg/kg PO q12 hours–q24 hours *or* 40–80 mg/l water for three days	Frequently used for motile protozoa (*Trichomonas, Giardia* and *Cochlosoma* spp.).

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Sulphonamides • Used to treat Coccidia.	Sulphachlorpyridazine	100–400 mg/l water for 3–5 days	• Contraindicated with: • Dehydration. • Liver disease. • Renal disease. • Treatment periods greater than two weeks may require supplementation with folic acid. • All treatments should be repeated after five days to allow for the prepatent period of coccidian.
	Sulphadimethoxine	20–50 mg/kg PO q12 hours for 3–5 days *or* 250–500 mg/l water for 5–7 days	
	Sulphadimidine	50–150 mg/kg PO q12 hours *or* 3330–6660 mg/l water for five days	
	Sulphamethazine	75–185 mg/kg PO q24 hours for three days *or* 125 mg/l water for three days	
	Sulphaquinoxaline	100 mg/kg PO q24 hours for three days *or* 250–500 mg/l water for 5–7 days	
Benzeneacetonitrile derivatives • Used to treat Coccidia. • All treatments should be repeated after five days to allow for the prepatent period of coccidian.	Clazuril	2.5 mg tablet per pigeon *or* 7 mg/kg PO q24 hours for 2–3 days	Single dose may suppress oocyst excretion for two weeks.
	Diclazuril	10 mg/kg PO q24 hours on Day 0, 1, 2, 4, 6, 8 and 10 *or* 5 mg/l water	Can be used for treating *Toxoplasma* spp.
	Toltrazuril	25–75 mg/l for five days *or* 7–15 mg/kg q24 hours for three days *or* 20–35 mg/kg PO once	Has also been used to treat *Atoxoplasma* in canaries.
Amprolium and **amprolium-ethopabate** • Used to treat Coccidia • Inhibits the active transport of thiamine into the cell. Coccidia are 50 times as sensitive to this inhibition as the host.	Amprolium	15–30 mg/kg q24 hours for 1–5 days *or* 50–100 mg/l water for 5–7 days	• Resistance is common. • Treatment periods greater than two weeks may require supplementation with folic acid. • All treatments should be repeated after five days to allow for the prepatent period of coccidian.
	Amprolium-ethopabate		
Pyrimethamine • Used for: • Toxoplasma. • Atoxoplasma. • Sarcocystis. • Leucozytozoonosis.	Pyrimethamine	0.5 mg/kg PO q12 hours 30 days	

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Quinacrine HCl • Used for: • Atoxoplasma. • Plasmodium.	Quinacrine HCl	5–10 mg/kg PO q24 hours 10 days	Overdosage may cause hepatotoxicity.
Chloroquine • Used for: • Plasmodium. • Other blood parasites.	Chloroquine	10–25 mg/kg PO as a first dose, then 5–15 mg/kg at 6,18 and 24 hours	Use in conjunction with primaquine.
Primaquine • Used for: • Plasmodium, in combination with chloroquine. • Sarcocystis.	Primaquine	0.3–1 mg/kg PO q24 hours for 3–10 days	Use in conjunction with chloroquine.
Mepacrine HCl • Used for: • Plasmodium in canaries	Mepacrine HCl	0.24 mg/kg PO q12 hours	

INTERNAL PARASITICIDES

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Fumarate reductase inhibitors • Benzimidazole derivatives. • Used mainly for treating nematodes. • Interferes with energy metabolism; prevents the parasite from using sugars. • Usually requires several days of treatment, although single high doses can be effective. • Low toxicity, but can affect haematopoietic cells and intestinal epithelium. • Do not use during breeding, as embryotoxic effects have been observed. • Toxicity varies with species and drug	Albendazole	10–50 mg/kg PO once	• Can be used for treating Microsporidia, as well as some nematodes. • Can be toxic in raptors at doses over 25 mg/kg
	Thiabendazole	40–100 mg/kg q24 hours seven days *or* 100–500 mg/kg once	• Less effective than fenbendazole
	Cambendazole	60–100 mg/kg q24 hours 3–7 days	
	Fenbendazole	25–50 mg/kg once *or* 8–10 mg/kg daily for 3–4 days *or* 50 mg/kg/day ×3 days (may treat *Giardia*).	• May also be effective against some cestodes, trematodes and *Giardia*. • Can interfere with feather growth during moulting. • Can be toxic to bone marrow, causing leucopaenia.
	Mebendazole	10–25 mg/kg q12 hours for five days *or* 10–20 mg/l water for 3–5 days	• Effective against some cestodes and trematodes.
	Oxfendazole	10–40 mg/kg once	
	Febantel	30 mg/kg once	

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Imidithiazoles • Used against nematodes. • Stimulates cholinergic receptors, causing paralysis of parasite. • Regurgitation, anorexia, diarrhoea, neurological signs (ataxia, head tilt and torticollis) are occasionally seen.	Levamisole	20–40 mg/kg PO once *or* 100–200 mg/l water for three days, repeat in two weeks.	• Not recommended in finches, lories or debilitated birds. • Care should be taken during hot weather or occasions where water intake is likely to be high, as this can lead to overdose.
Gamma amino butyric acid (GABA) interfering drugs • Macrocyclic lactones. • Used against: • *Ascaridia* and other nematodes. • Blood-sucking external parasites. • *Cnemidocoptes*. • Microfilaria. • Toxicity is low, but overdosing and idiosyncratic reactions include severe depression, inactivity, excessive sleeping, and neurological signs. • Budgerigars, African finches and European finches may be more sensitive.	Ivermectin	200–400 µg/kg IM PO or topically, once	Toxicity may be higher if the drug is injected (particularly via the IM route).
	Doramectin		
	Moxidectin		
Adenosine triphosphate synthesis blockers • Block synthesis of ATP and produce paralysis by interfering with energy metabolism.	Niclosamide	50–100 mg/kg PO once, repeat in 10–14 days	• Used for treating cestodes and trematodes. • Praziquantel is more effective.
	Rafoxanide	10 mg/kg PO	• Used for treating trematodes and cestodes.
Miscellaneous	Praziquantel	10–20 mg/kg PO or IM once	• Used for treating cestodes and trematodes. • Caution with IM in finches; sudden death has been reported. • Very unpalatable.
	Piperazine	100–250 mg/kg PO once *or* 1000–2000 mg/l water for three days	• Used for *Ascaridia* only. Resistance is common.
	Pyrantel tartrate Pyrantel pamoate Pyrantel embonate	7–25 mg/kg PO once	• Used for nematodes. • Poorly absorbed from the GI tract. • Good safety margin.
Paromomycin	Paromomycin	100 mg/kg PO q12 hours 7 days	• Used for treating *Cryptosporidia*

Downloaded From Vet-eBooks.com

EXTERNAL PARASITICIDES

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Pyrethrins and synthetic pyrethroids	Pyrethrin Permethrin	Spray or wash	• Often combined with insect growth regulators.
Piperonyl butoxide	Piperonyl butoxide is usually combined with a pyrethrin or permethrin.	Spray or wash	
Organophosphates	Malathion Maldison	Spray or wash	• Toxic; use is not recommended because of hazards to birds and owners.
Fipronil	Fipronil	3 mg/kg spray or spot-on application.	• Not registered for use in birds. • Spray works better than spot-on application. • Beware of inhalation or dermal absorption of the alcohol base. • Beware of the drying effect the alcohol base may have on feathers.
Carbaryl 5%	Carbaryl	Dust lightly *or* Add 1–2 teaspoons to nesting material.	• Carbamate flea powder. • Use is illegal in many countries.
Gamma amino butyric acid (GABA) interfering drugs • Macrocyclic lactones. • Used against: • Blood-sucking external parasites. • *Cnemidocoptes*. • Toxicity is low, but overdosing and idiosyncratic reactions include severe depression, inactivity, excessive sleeping, and neurological signs. • Budgerigars, African finches and European finches may be more sensitive.	Ivermectin	200–400 µg/kg topically, IM or PO once	• Toxicity may be higher if the drug is injected (particularly via the IM route).
	Moxidectin		

Downloaded From Vet-eBooks.com

HORMONAL THERAPIES

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Reproductive hormones	Cabergoline	10–20 µg/kg PO q24 hours	• Inhibits prolactin release by direct stimulation of dopamine receptors in prolactin-releasing cells in the anterior pituitary.
	Chorionic gonadotropin (HCG)	500–1,000 units IM on Day 1, 3 and 7 and then every two weeks as required.	• LH agonist • Continued use is limited by formation of anti-HCG antibodies.
	Deslorelin	4.7 mg implant SC	• GnRH agonist, will last for 6–18 months if environmental, behavioural and dietary changes are made.
	Leuprolide acetate	100–700 µg/kg every two weeks for three treatments, then as required	• GNRH agonist • Must be combined with environmental, behavioural and dietary changes for effect.
	Medroxyprogesterone acetate	5–25 mg/kg IM every 4–6 weeks	• Historically used for feather-picking behaviour, but side-effects (PU/PD, polyphagia, diabetes mellitus, hepatopathy, weight gain, sudden death) have led to its use been discontinued. • At very low doses it may be useful in suppressing ovulation.
	Megestrol acetate	2.5 mg/kg PO q24 hours for seven days, then weekly as required	• Seldom used because of severe side-effects, similar to medroxyprogesterone.
	Oxytocin	0.5–1.0 IU/kg repeated every 30–60 minutes	• Used to stimulate oviductal contractions, but is not a naturally occurring hormone in birds and it may not be effective. • Use is contraindicated if uterovaginal sphincter is not dilated • Should be used in conjunction with calcium gluconate injections.
	Prostaglandin E2 (Dinoprost)	0.02–0.1 mg/kg applied topically into the cloaca	• Used to induce egg laying in egg-bound birds; simultaneously relaxes utero–vaginal sphincter while contracting oviduct.
Thyroxine	Levothyroxine	5–200 µg/kg PO q12 hours	• May induce moult. • Monitor for cardiotoxicity.
Insulin	Short-acting insulin NPH insulin Ultralente insulin	Dose rates vary considerably, and should be based on the observed effects. They range from 0.067–3.3 U/kg q12–24 hours	• For insulin-dependent diabetes. • The immediate use of short-acting insulin (0.1–0.2 U/kg) can be used to initially stabilise the patient, but long-term control at home usually required longer-acting insulin (NPH or ultralente).

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Sulfonylureas	Glipizide	0.5 mg/kg PO q12 hours	• Stimulates the beta cells in the pancreas to secrete additional endogenous insulin. • Extra-pancreatic effects include enhanced tissue sensitivity of circulating insulin.
Glucocorticoids • Previously recommended for the treatment of shock and as an anti-inflammatory drug • Side-effects are severe: • PU/PD. • Catabolism. • Liver and kidney disease. • Immunosuppression. • Topical application seems to be as severe as, or worse than, parenteral administration.	Dexamethasone	2–4 mg/kg s/c, IM, IV once	• With the possible exception of prednisolone sodium succinate, the use of glucocorticoids in birds is dangerous and cannot be recommended. • Only indication may be immune-mediated disorders such as IMHA.
	Hydrocortisone	10 mg/kg IM once	
	Methylprednisolone acetate	0.5–1 mg/kg IM once	
	Prednisolone	0.5–1 mg/kg IM once	
	Prednisolone sodium succinate	2–4 mg/kg IV once	
	Triamcinolone	0.1–0.5 mg/kg IM once	

DRUGS USED TO TREAT LIVER DISORDERS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Anti-inflammatory • Anti-fibrotic • anti-inflammatory	Colchicine	0.04–0.2 mg/kg PO q24 hours	• May cause nausea and vomiting in some birds.
Chelating agent • Chelates iron	Deferoxamine	100 mg/kg PO, IM or SC q24 hours	• Preferred iron chelator for iron storage disease. • Avoid in birds with renal disease.
Diuretic • Reduce ascites	Furosemide	0.15–2 mg/kg IM, SC, PO q12–24 hours	• Overdose can lead to dehydration and electrolyte abnormalities. • Lorikeets are very sensitive to this drug; use with caution.
Lactulose • Does not treat liver disease. • Reduces absorption of ammonia from intestine by altering the pH of the intestinal lumen and promoting an osmotic catharsis. This reduces the ammonia levels presented to the liver. • Best results are seen in carnivorous birds (e.g. raptors), as vegetable protein lacks many encephalopathic precursors.	Lactulose	150–650 mg/kg PO q8–12 hours	• Can cause diarrhoea.
Antioxidant • Unproven remedy that offers good promise in avian medicine. • Antioxidant. • Enhances protein synthesis and hepatocellular regeneration. • Protective effect against hepatotoxins. • Suppresses fibrogenesis. • Promotes fibrolysis.	Silibinin (silymarin; milk thistle)	50–75 mg/kg PO q12 hours	• Use a low alcohol or alcohol-free base.

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Hepatoprotective drugs	Ursodeoxycholic acid	10–15 mg/kg PO q24 hours	• Bile acid. • Cytoprotective. • Reduces involvement of hepatocytes and biliary epithelium in inflammatory process. • Changes mix of bile acids to eliminate toxic bile acids from liver.

DRUGS USED TO TREAT KIDNEY DISORDERS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Decrease production of uric acid	Allopurinol	10 to 15 mg/kg PO q12–24 hours	• May worsen renal disease. • Maintain good hydration.
Reduce inflammation associated with articular gout	Colchicine	0.04–0.2 mg/kg PO q24 hours	• May cause nausea and vomiting in some birds.
Anti-inflammatory	Aspirin	1 mg/kg PO q24 hours	• Used together for the treatment of membranous glomerulonephritis.
	Omega-3 and -6 fatty acid supplementation, mixed in a ratio of Omega 6:Omega 3 of 6:1	0.1 ml/kg PO q12 hours	

DRUGS USED TO TREAT CARDIOVASCULAR DISORDERS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Angiotensin converting enzyme (ACE) inhibitors • Block the formation of angiotensin II, thereby blocking the renin–angiotensin–aldosterone system. • They also have a diuretic effect.	Enalapril	0.5–1.25 mg/kg PO q12 hours	• Side-effects could include hypotension, reflex tachycardia, dehydration, gastrointestinal disorders, renal dysfunction and hyperkalaemia.
	Benazapril	0.5 mg/kg PO q12 hours	
Cardiac glycoside • Indicated for myocardial dysfunction, chronic mitral insufficiency, chronic volume overloads. • Contraindicated for hypertrophic cardiomyopathy, ventricular tachycardia, and sinus or atrioventricular node disease.	Digoxin	0.02–0.05 mg/kg PO q24 hours	• Adverse effects relate to myocardial toxicity, therefore patients should be monitored for clinical improvement and via ECG for prolonged PR-time. Serum concentrations should be measured after one week of therapy, or one week after the dose is changed. The dose can be increased if the serum concentration is less than 0.8 mg/l 8–10 hours after dosing.
Positive Ionotrope • Similar in effect to digoxin, but with less risk of adverse effects.	Pimobedan	0.15 mg/kg PO q12 hours	

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Beta blocker • Used to treat ventricular and supraventricular tachycardia.	Propranolol	0.2 mg/kg q24 hours	
Diuretics • Reduce ascites associated with liver disease or congestive heart failure.	Furosemide	0.15–2 mg/kg IM, SC, PO q12 hours–q24 hours	Overdose can lead to dehydration and electrolyte abnormalities. Lorikeets are very sensitive to this drug; use with caution.
	Spironolactone	1–2 mg/kg PO q12 hours	Used in patients with congestive heart failure who do not adequately respond to furosemide and ACE inhibitors, or who develop hypokalaemia on other diuretics.
VASCULAR DRUGS	Pentoxifylline	10–25 mg/kg PO q6–12 hours	Increases erythrocyte flexibility and reduces inflammation.
	Isoxsuprine	5–10 mg/kg PO q24 hours	Peripheral vasodilation.

DRUGS USED TO TREAT GASTROINTESTINAL DISORDERS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Intestinal motility modifiers • Used for gastrointestinal motility disorders including slow emptying crops, crop stasis and regurgitation or vomiting.	Metoclopramide	0.5–2 mg/kg PO or IM q6hrs–q12 hours	• Increases force and frequency of gastric contractions, relaxes pyloric sphincter, and promotes peristalsis in the duodenum and jejunum. • Usually only effective if given with IV fluids as a constant rate infusion.
	Cisapride	0.5–1.5 mg/kg PO q8–12 hours	• Stimulates gastrointestinal motility.
Intestinal protectants • Used when proventriculitis or gastric ulceration is present.	Cimetidine	5 mg/kg IM q12 hours	• Reduces gastric secretion of HCl and pepsin. • May impair the metabolism of concurrently administered drugs. • Potential side-effects: depression, diarrhoea, tachycardia, respiratory failure.
	Ranitidine	1 mg/kg PO q8–12 hours	
	Sucralfate	25 mg/kg PO q8 hours	• A complex disaccharide that reacts with stomach acid to form a complex that binds to proteins associated with an ulcer and produces a protective layer that protects the ulcerated mucosa from gastric acids and microbial pathogens.

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Laxatives and cathartics • Used to remove foreign bodies from the gastrointestinal tract.	Mineral oil (paraffin oil)	5 ml/kg PO	• Acts unchanged as an emollient laxative. Care must be taken to prevent aspiration; give by crop gavage. It can be mixed with peanut butter in a ratio of one part mineral oil to two parts peanut butter.
	Methylcellulose	5 ml/kg PO q12 hours	• Absorbs water and swells. Care must be taken not to mix too thickly or give too much, as it can absorb enough water to block the gastrointestinal tract
	Magnesium sulphate (Epsom salts)	0.25–1.0 g/kg PO q24 hours for 1–2 days	• Retain or attract water via osmotic forces. Must be used with caution
	Lactulose	150–650 mg/kg PO q8–12 hours	• Has a laxative effect through osmotic catharsis. • In birds with caecae, lactulose passes to the caecae where saccharolytic microflora ferment lactulose to produce acetic, lactic and other organic acids which lowers the pH of the caecal content.
	Vegetable oils (e.g. castor oil, raw linseed oil, olive oil)	15 ml/kg	• Act as irritant purgatives; • Forms sodium and potassium salts of released fatty acids after hydrolysis by pancreatic lipase, making them irritant soaps.

ANTICONVULSANT THERAPY

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Barbiturate	Phenobarbital	1–7 mg/kg PO q12 hours	• Mild sedative effect used for long-term seizure control. • May cause deep sedation and ataxia. • Dose is best adjusted by response to treatment.
Others	Potassium bromide	25–80 mg/kg PO q24 hours	• Used for long-term seizure control alone, or in conjunction with phenobarbital. • May take up to 90 days to establish steady state.
	Levetiracetam	50 mg/kg PO q8 hours *or* 100 mg/kg q12 hours	• Well tolerated. • Dose and frequencies may need escalation based on differences in species and individuals, and drug levels should be monitored.
	Zonisamide	10 mg/kg PO q12 hours	• Used in conjunction with other anticonvulsant drugs such as levetiracetam.

ANAESTHETICS AND SEDATIVES

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Sedatives • Used for sedation or as a pre-medication for anaesthesia. • Often used in conjunction with an opioid (e.g. butorphanol).	Diazepam	0.05–0.15 mg/kg IV *or* 0.2–0.5 mg/kg IM	• Can be used as a pre-medication prior to anaesthesia if given 10–20 minutes before induction. • Onset of action 10–20 minutes when given IM. • Onset of action when given intra-nasally is three minutes. • At sedative doses there is minimal effects on blood pressure, heart rate and temperature.
	Midazolam	0.5–1.5 mg/kg IV *or* 0.8–3 mg/kg IM *or* 2–5 mg/kg intra-nasally	

Continued

Downloaded From Vet-eBooks.com

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Local anaesthesia	Lignocaine	2–8 mg/kg SC	• The shorter acting local anaesthetics (e.g. lignocaine) have a faster onset, shorter duration of effect, lower potency and higher therapeutic index than the long-acting local anaesthetics (e.g., bupivacaine). Lignocaine causes CNS effects (i.e. seizures) before cardiac effects are seen. However, bupivacaine induces adverse cardiac effects at lower doses than its CNS effect. • Note that topical application can reach systemic concentrations in birds greater than those achieved in mammals.
	Bupivacaine	1–2 mg/kg SC	
	EMLA (eutectic mixture of local anaesthetics) 5% cream Lignocaine 2.5% Prilocaine 2.5%	2–4 mg/kg topically	
Injectable anaesthetics • See Chapter 28, Analgesia and Anaesthesia p. 375 • Injectable anaesthesia is not recommended in companion birds; regimes and doses are included here to assist veterinarians without access to inhalation anaesthesia.	Ketamine	20–50 mg/kg IM, IV	• Ketamine alone is not recommended for anaesthesia. • Inadequate analgesia and muscle relaxation. • Associated with spontaneous movements and muscular rigidity. • Not associated with pulmonary depression or cardiovascular depression. • Violent recoveries. • Lack of coordination. • Excitement. • Head shaking. • Wing flapping.
	Xylazine	1 mg/kg IM, IV	• Does not usually produce adequate surgical immobilisation. • May lower the arrhythmogenic threshold to endogenous catecholamines. • Respiratory depression. • Hypotension. • Bradycardia. • Excitement and convulsions. • Prolonged recovery. • Muscle tremors, salivation, and movement in response to noise. • Reversible with yohimbine 0.11–0.27 mg/kg IM.
	Xylazine–Ketamine	Ketamine 4.4 mg/kg + Xylazine 2.2 mg/kg) IV *or* Ketamine 20–30 mg/kg + Xylazine 2.5–4.0 mg/kg IM	• Used for restraint and anaesthesia. However, waterfowl, (especially ducks and Canada geese), owls and acciptres, do NOT respond well to this combination. • Combination is synergistic, giving a smooth induction and recovery with improved muscle relaxation and enhanced analgesia.
	Ketamine–Diazepam	Ketamine 10–40 mg/kg Diazepam 0.2–2 mg/kg IV, IM	• Can be used as an intravenous induction agent in large ratites, raptors and waterfowl, but may not be a reliable maintenance agent. • Can be used intramuscularly in parrots and pigeons, with a slow induction and recovery time.
	Ketamine–Medetomidine	Ketamine 1.5–2.0 mg/kg + Medetomidine 60–85 µg/kg IV	• Useful for inducing waterfowl. • Can be reversed with atipamezole.
	Ketamine–Midazolam	Ketamine 10–40 mg/kg Midazolam 0.2–2 mg/kg IM	
	Propofol	1–5 mg/kg IV	• Can be used as an intravenous induction agent following sedation with medetomidine–ketamine. • Apnoea is common following IV induction. Intubation and IPPV are recommended.
	Alfaxalone	5–10 mg/kg IV *or* 10–20 mg/kg IM	• Brief apnoea on induction if not given slowly. • Short duration. • Note that these doses are anecdotal; no controlled studies on the use of alfaxalone in birds have been conducted.

Downloaded From Vet-eBooks.com

ANALGESICS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Opioids Some bird species appear to have more kappa opioid receptors in the forebrain than mu opioid receptors, explaining why some birds do not respond to mu agonists, such as morphine, buprenorphine, and fentanyl, in the same manner as mammals. • Therefore kappa opioids, such as butorphanol, may be the more efficacious analgesics in birds.	Butorphanol	1–4 mg/kg IM, PO q6–8 hours	• Higher doses may be hyperalgesic. • Side-effects are uncommon, but may include respiratory depression, nausea and vomiting, bradycardia and constipation.
	Morphine	1 mg/kg IM q12 hours	
	Tramadol	10–30 mg/kg IM, PO q6–12 hours	
		2–5 mg/kg IV	
Non-steroidal anti-inflammatory drugs (NSAIDs) • NSAIDs act on peripheral tissues, and therefore are indicated when tissue damage and inflammation are the source of the pain. • They may be most effective when acting synergistically with opioids. • All have the potential to cause renal damage and gastrointestinal ulceration.	Carprofen	2–4 mg/kg PO q12 hours	
	Flunixin	2–4 mg/kg IM, PO q12 hours	Renal side-effects and gastrointestinal ulceration is common; use with caution in all species
	Ketoprofen	2 mg/kg IM q8–12 hours	Not commonly used
	Meloxicam	1.0–1.5 mg/kg IM, PO q12–24 hours	Available as a palatable oral suspension or an injectable form. • Oral suspension can be diluted (1:5) with sterile water or methylcellulose to give a stable oral solution for small birds.
Other	Gabapentin	3 to 11 mg/kg PO q12–24 hours	• Used to treat neuralgic pain.

ANTIDOTES AND OTHER DRUGS USED IN POISON CASES

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Antidotes	Atropine	0.01–0.1 mg/kg IM, repeat every 3–4 hours as required	• Organophosphate and carbamate toxicosis. • Does not cause pupillary dilation.
	Calcium EDTA (edentate calcium disodium)	10–50 mg/kg IM q12 hours 3–5 days	• Treatment of choice for lead and zinc toxicosis. • Often recommended as an oral treatment, but efficacy is uncertain. • Ongoing therapy may be required for chronic lead toxicosis (e.g. twice weekly for 6–8 weeks after initial stabilisation).
	Deferoxamine	20–100 mg/kg IM, PO q24 hours 3–5 months	• Preferred chelator for iron storage disease. • May take three months to achieve effect. • May cause reddish discoloration of urine.
	Dimercaprol (BAL)	25–35 mg/kg q12 hours PO 3–5 weeks	• Heavy metals other than lead or zinc. • Rarely used.
	Dimercaptosuccinic acid (DMSA)	25–35 mg/kg PO q12–24 hours for three weeks	• Oral chelation of lead. • Can be used with CaEDTA.
	D-Penicillamine	30–50 mg/kg PO q12 hours 1–3 weeks	• Preferred chelator for copper toxicosis, but can be used for other heavy metal toxicosis. • Can cause nausea and vomiting
	Pralidoxamine	10–100 mg/kg IM, repeat after six hours	• Organophosphate toxicosis; contraindicated for carbamate toxicosis. • Must be used with 24–36 hours of exposure.
	Vitamin K	0.2–2.2 mg/kg IM q6–8 hours till stable, then q24 hours orally for 2–4 weeks, depending on toxin	• Warfarin toxicosis.
Adsorbents	Activated charcoal	2,000–8,000 mg/kg PO	• May be mixed with hemicellulose to form a bulk laxative to remove toxins from the gastrointestinal tract.

Downloaded From Vet-eBooks.com

PSYCHOTROPIC DRUGS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Benzodiazepines • Inhibit dopamine and potentiate GABA. • Muscle relaxants. • Anxiolytic. • Can interfere with learning.	Diazepam	0.5 mg/kg PO q8–12 hours *or* 0.25–0.5 mg/kg IM, IV	
	Lorazepam	0.1 mg/kg PO q12 hours	
Butyrophenones • Inhibit dopamine. • Used for self-mutilation and feather-picking.	Haloperidol	0.1–0.4 mg/kg PO q12–24 hours *or* 1–2 mg/kg IM every 2–3 weeks	• May cause anorexia or depression.
Antihistamines • Inhibit histamine receptors, producing sedation.	Diphenhydramine	2–4 mg/kg PO q12 hours	• May work best if feather-picking is allergic in origin.
	Hydroxyzine	2 mg/kg PO q8 hours	
Progestins • Potentiate GABA. • Have a calming effect and anti-inflammatory effect. • Multiple side-effects	Medroxyprogesterone acetate	5–25 mg/kg IM every 4–6 weeks	• Often used for feather-picking behaviour. • Side-effects include PU/PD, polyphagia, diabetes mellitus, hepatopathy, weight gain, sudden death.
	Megestrol acetate	2.5mg/kg PO q24 hours seven days, then weekly as required	• Seldom used because of severe side-effects, similar to medroxyprogesterone.
Tricyclic antidepressants • Potentiate serotonin, giving a sedative and, anticholinergic activity. • Alleviate anxiety and depression. • Use with care because of side-effects including constipation and arrhythmias.	Amitriptyline	1–5 mg/kg PO q12–24 hours	• Use for at least 30 days to assess effect.
	Clomipramine	0.5–2.0 mg/kg PO q12–24 hours	• Side-effects may include: • Regurgitation. • Drowsiness. • Death (possibly with pre-existing arrhythmias). • Adjust dose after 2–3 weeks.
	Doxepin	0.5–1.0 mg/kg PO q12 hours	• Adjust dose after 14 days.
Serotonin specific re-uptake inhibitors • Have serotonergic effects. • Some effect in birds.	Fluoxetine.	0.4–3.0 mg/kg PO q12–24 hours	• May cause sedation.
Narcotic antagonists and agonist/antagonists • Act at the opiate centres in the brain, blocking endorphin response to self-injurious behaviour. • May be useful in some bird species.	Naltrexone	1.5 mg/kg PO q8–12 hours for 1–18 months	• Contraindicated if liver disease is present. • May need to increase dose.

Downloaded From Vet-eBooks.com

NUTRITIONAL SUPPORT

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Calcium • Used to treat hypocalcaemic diseases, including hypocalcaemic tetany, egg binding, metabolic/ nutritional bone disease.	Calcium borogluconate	50–100 mg/kg IM or slowly IV	
	Calcium carbonate	3–7 mg/kg feed	• Calcium requirements vary considerably between species and according to reproductive status. • For example, budgerigars have very low calcium requirements compared to chickens; egg laying hens require more calcium than inactive hens or cocks.
	Calcium glubionate	20–25 mg/kg PO q24 hours *or* 750 mg/l water	
	Calcium gluconate	5–10 mg/kg slowly IV *or* 10–100 mg/kg IM	• For hypocalcaemic tetany or egg binding. • Dilute with saline before injecting.
Iodine	Lugol's iodine	0.2 ml/l water	• Used to treat thyroid hyperplasia (goitre) in budgerigars.
Iron	Iron dextran	10 mg/kg IM repeated weekly	• Used for treating anaemia, although not used as frequently as it once was. • Use with caution in species prone to iron storage disease (e.g. mynahs, toucans, lorikeets, etc.).
Vitamins	Vitamin A	Cockatiels and budgerigars should be dosed at 2,000–5,000 IU/kg IM weekly; those less susceptible may benefit from doses of 20,000–30,000 IU/kg IM weekly.	• There is considerable variation in recommended doses for vitamin A due to species susceptible to vitamin A toxicosis • Dietary sources: • Fruit (carotenoids; provitamin A). • Spinach. • Fish. • Rodents. • Seed and insects are low in vitamin A. • Beware of over supplementing vitamin A.
	Vitamin B1 (Thiamine)	1–2 mg/kg IM, PO q24 hours	• Dietary sources: • Grains. • Leafy green plant material.
	Vitamin B7 (Biotin)	0.05 mg/kg PO q24 hours 1–2 months	
	Vitamin B12	0.25–0.5 mg/kg IM weekly	• May cause orange/pink urine and urates.
	Vitamin D3	3,000–6,000 IU/kg IM weekly	• Requires 11–45 minutes of unfiltered sunlight (or UVb at 290–320 nm) per day to activate vitamin D3 synthesis in the skin. • Beware of over supplementation.
	Vitamin E	0.06 mg/kg IM weekly	• Dietary sources include green plants. • Sunflower oil, hazelnuts, almonds have insufficient levels of vitamin E, and the effect of the vitamin E is negated by polyunsaturated fatty acids. • Low in whole prey (rodents and fish).
	Vitamin K1	0.025–2.5 mg/kg IM q12 hours *or* 0.2–2.2 mg/kg IM q6–8 hours till stable, then q24 hours orally for 2–4 weeks, depending on toxin	• Dietary sources: • Apple peel. • Spinach leaf. • Termites. • Large fig parrots (*Psittaculirostris* spp.) require vitamin K supplementation in their diet.

Downloaded From Vet-eBooks.com

CHEMOTHERAPY DRUGS

CLASS	DRUG	DOSE AND ROUTE	COMMENTS
Vinca Alkaloids/Anti–mitotics • Arrests cell division in metaphase.	Vincristine	0.75 mg/m^2 IV q7 days ×3 *or* 0.1 mg/kg IV q7–14 days *or* 0.5 mg/m^2 IV, then 0.75 mg/m^2 q7 days ×3	• Given IV – perivascular irritation can be severe. • Excreted in bile.
Anti–tumour antibiotics • Binds to DNA and inhibits synthesis of nucleic acids and cell division.	Doxorubicin	50–60 mg/m^2 IV *or* 2 mg/kg IV *or* 30 mg/m^2 IV q2 days	• Given IV – irritant if extravasated. • Adverse effects: • Bone marrow suppression. • GI tract effects. • Cardiotoxic. • Nephrotoxic. • Hypersensitivity reactions. • Excreted in bile.
Alkylating agents • Combines with DNA, damaging it.	Cyclophosphamide	200 mg/m^2 IO q7 days *or* 300 mg/m^2 PO once	• Can be given orally or IV. • Myelosuppressive. • Inactivated in liver and excreted in urine.
	Chlorambucil	1 mg/kg PO q3 days	
	Lomustine		
Platinum drugs • Inhibits DNA synthesis and may have some alkylating effects.	Cisplatin	1 mg of cisplatin/cm^3 of tissue when given intra-lesionally *or* 15–27 mg/kg IV	• Given IV or intra-lesional. • Can be nephrotoxic, myelosuppressive, and neurotoxic. • Carboplatin is not as toxic as cisplatin. • Renal excretion.
	Carboplatin	125 mg/m^2 IV slow bolus q2–3 weeks. Dilute to a comfortable volume with 5% dextrose *or* 15 mg/kg IO *or* 5 mg/kg intralesional	
Other • Leukaemic cells and some other suspected tumour cells are unable to synthesise the amino acid asparagine (normal cells are able to make their own asparagine); Asparaginase catalyzes the conversion of L-asparagine to aspartic acid and ammonia, depriving the leukemic cell of circulating asparagine, which leads to cell death.	L-Asparaginase	400 IU/kg IM q7 days *or* 1650 IU/kg SC once	• Used in the treatment of leukaemia and lymphosarcoma. • Can be given IM or SC.

Downloaded From Vet-eBooks.com

APPENDIX 2: REFERENCE INTERVALS FOR COMMONLY KEPT COMPANION BIRDS

SPECIES	AMAZON	BUDGERIGAR	CAIQUE	CANARY	COCKATIEL	COCKATOO
PCV (l/l)	0.41–0.53	0.44–0.58	0.47–0.55	0.45–0.55	0.43–0.57	0.40–0.55
WBCs (x10^9/l)	5–17	3–10	8–15	3–10	5–11	5–13
H (%)	31–71	40–68	39–72	21–60	46–72	45–72
L (%)	20–67	22–60	20–61	20–65	26–60	20–50
E (%)	0	0	0	0–1	0–2	0–2
M (%)	0–2	0–2	0–2	0–1	0–1	0–2
B (%)	0–2	0–2	0–1	0–1	0–1	0–1
AST(IU/l)	150–350	150–375	120–365	130–350	120–400	140–360
CK (IU/l)	120–425	120–370	120–390	55–350	160–420	150–420
Bile acids (µ mol/l)	30–150	30–120	10–110	20–90	40–110	30–110
Uric acid (µ mol/l)	130–260	285–765	150–630	245–750	200–650	225–655
Glucose (mmol/l)	14–21	12–25	9.4–21	8.9–20	12.6–24.4	11.4–23.2
Total protein	26–45	20–45	25–35	30–45	20–50	25–49
Calcium (mmol/l)	2–3.4	2–2.8	2.1–2.8	1.5–3.4	2–2.7	2–2.9

H = heterophils; L = lymphocytes; E = eosinophils; M = monocytes; B = basophils. (SI units)

Adapted from: Fudge A (2000) *Laboratory Medicine: Avian and Exotic Pets.* WB Saunders, Philadelphia

SPECIES	CONURE, *Aratinga* SPP.	CONURE, *Pyrrhura* SPP.	ELECTUS, RED-SIDED	GREY PARROT	LORIES	LOVEBIRDS
PCV (l/l)	0.42–0.55	0.42–0.55	0.42–0.55	0.42–0.53	0.47–0.55	0.44–0.55
WBCs (x10^9/l)	6–11	5–10	9–15	6–13	8–13	7–16
H (%)	44–72	46–71	46–70	45–73	39–60	40–56
L (%)	20–49	22–53	23–57	19–50	22–70	20–53
E (%)	0	0–1	0–1	0–1	0–1	0–2
M (%)	0–1	0–2	0–1	0–2	0–2	0–1
B (%)	0–2	0	0–1	0–1	0–1	0–1
AST (IU/l)	140–350	140–270	140–340	110–340	140–370	130–360
CK (IU/l)	150–370	140–280	130–410	140–410	180–400	160–400
Bile acids (µ mol/l)	10–90	10–90	30–110	10–100	20–100	10–90
Uric acid (µ mol/l)	130–690	190–740	120–650	120–650	120–700	195–635

Continued

Downloaded From Vet-eBooks.com

SPECIES	CONURE, *Aratinga* SPP.	CONURE, *Pyrrhura* SPP.	ELECTUS, RED-SIDED	GREY PARROT	LORIES	LOVEBIRDS
Glucose (mmol/l)	9/3–21	11/9–22	12–22	14/2–20	10.5–21.5	12.2–22
Total protein	24–45	35–50	30–45	25–45	19–40	18–35
Calcium (mmol/l)	2–2.8	2–3.1	2–3	2–3.5	2–2.9	2.1–2.9

H = heterophils; L = lymphocytes; E = eosinophils; M = monocytes; B = basophils. (SI units)

Adapted from: Fudge A (2000) *Laboratory Medicine: Avian and Exotic Pets*. WB Saunders, Philadelphia

SPECIES	MACAW	MYNAH	PIONUS	QUAKER (MONK PARAKEET)	RING-NECK PARROT (INDIAN)	SENEGAL
PCV (l/l)	0.42–0.56	0.38–0.50	0.45–0.54	0.45–0.58	0.45–0.54	0.45–0.60
WBCs (x10^9/l)	10–20	8–12	5–13	8–17	8–14	6–14
H (%)	50–75	45–64	55–74	47–70	40–70	44–73
L (%)	23–53	21–55	19–70	20–63	30–55	22–70
E (%)	0	0–4	0–1	0–1	0–1	0–2
M (%)	0–1	0–1	0–1	0–4	0–1	0–1
B (%)	0–1	0–1	0–1	0–3	0–1	0–1
AST (IU/l)	65–170	200–350	130–360	130–380	150–390	120–330
CK (IU/l)	90–360	250–420	120–410	190–400		150–400
Bile acids (µ mol/l)	7–100	30–100	15–90	20–90		20–100
Uric acid (µ mol/l)	110–700	135–710	120–685	130–710	195–700	135–710
Glucose (mmol/l)	11.7–20	12.5–20.5	12–20/5	11.5–22	12.2–20	14.2–20
Total protein	24–44	30–45	20–43	25–35		30–42
Calcium (mmol/l)	2.1–3	2–2.7	2–2.6	2.1–2.7	2.1–2.6	2.1–2.7

H = heterophils; L = lymphocytes; E = eosinophils; M = monocytes; B = basophils. (SI units)

Adapted from: Fudge A (2000) *Laboratory Medicine: Avian and Exotic Pets*. WB Saunders, Philadelphia

Downloaded From Vet-eBooks.com

APPENDIX 3: BIOLOGICAL VALUES FOR SOME COMMON COMPANION BIRD SPECIES

SPECIES	WEANING AGE (DAYS)	SEXUAL MATURITY	ADULT BODY WEIGHT (G)	LIFE EXPECTANCY (YEARS)
Alexandrine parrot	85–100	2–3 years	200–300	25–35
Amazon, blue-crowned	90–120	4–6 years	620–1000	50–70
Amazon, blue-fronted	90–120	4–6 years	360–490	50–70
Amazon, double yellow-headed	90–120	4–6 years	460–700	50–70
Amazon, yellow-naped	90–120	4–6 years	470–800	50–70
Budgerigar	30–40	6–9 months	35–45	7–12
Caique	95–105	2–3 years	120–150	30–40
Canary	21	10 months	15–25	6–12
Cockatiel	40–50	8–12 months	80–100	10–15
Cockatoo, bare-eyed (little corella)	110–120	3–4 years	450–600	30–40
Cockatoo, greater sulphur-crested	120–150	3–4 years	850–950	50–70
Cockatoo, lesser sulphur-crested	80–90	2–3 years	310–380	40–60
Cockatoo, Major Mitchell	80–90	2 years	350–450	40–60
Cockatoo, medium sulphur-crested	100–120	3 years	275–350	40–60
Cockatoo, rose-breasted (galah)	85–95	2–3 years	250–320	20–40
Cockatoo, citron-crested	85–95	2–3 years	300–400	50–60
Cockatoo, Moluccan	100–120	3–5 years	700–1000	50–60
Cockatoo, umbrella	95–105	5–6 years	550–650	50–60
Conure, *Aratinga* spp.	55–75	2–4 years	110–130	15–25
Conure, *Pyrrhura* spp.	55–75	9–18 months	55–85	15–25
Eclectus, red-sided	80–90	3 years	380–410	25–30
Grey parrot, Congo	100–120	4–6 years	390–500	50–60
Grey parrot, Timneh	95–105	3–5 years	280–360	25–35
Jardine parrot (red-fronted)	95–105	3–4 years	200–250	20–25
Lorikeet, rainbow	60–70	1–2 years	130–150	15–20
Lory, chattering	85–100	2–3 years	160–180	30–35
Lory, red	85–100	2–3 years	100–130	25–30
Macaw, blue and gold	90–110	5–7 years	1000–1200	50–80
Macaw, green-winged	110–140	3–5 years	1050–1320	60–90
Macaw, Hahn's	70–85	2–3 years	130–150	25–30

Continued

Downloaded From Vet-eBooks.com

SPECIES	WEANING AGE (DAYS)	SEXUAL MATURITY	ADULT BODY WEIGHT (G)	LIFE EXPECTANCY (YEARS)
Macaw, hyacinth	180–270	5–7 years	1200–1700	60–90
Macaw, military	110–120	2–3 years	860–1100	50–60
Macaw, scarlet	90–110	5–7 years	950–1150	50–80
Meyer's parrot	70–90	2 years	80–110	25–35
Mynah bird	60	2–3 years	180–260	12
Pionus, blue-headed	70	2–3 years	250	20–25
Pionus, white-capped	70–100	2 years	130–160	25–30
Quaker (monk parakeet)	50–60	1 year	120–140	20–30
Ring-neck parrot (Indian)	70–90	2 years	110–120	25–30
Senegal parrot	70	2–3 years	125	15–25

Downloaded From Vet-eBooks.com

SECOND EDITION

Avian Medicine and Surgery in Practice

Companion and Aviary Birds

Avian Medicine and Surgery in Practice is an invaluable quick reference resource for clinicians and a useful study guide for veterinary students.

In this practical and beautifully illustrated book, early chapters cover physical examination, advice on interpreting diagnostic tests, and avian anatomy and physiology. Disorders affecting the different body regions and systems make up the majority of the book from the external—skin, feathers, eyes, legs and feet—to the internal including the gastrointestinal tract and the cardiovascular system. Further aspects of avian medicine discussed in the book include behavioural problems, incubation of eggs, paediatrics and surgery.

Written by an expert with more than 30 years of clinical experience in avian medicine, the new edition is thoroughly revised with updated diseases, new and expanded clinical techniques, and over 100 new color illustrations. It also adds four important new chapters: *Husbandry, Grooming and Nutrition, Diagnostic Imaging, Endoscopy, and Oncology* as well as new sections on cardiovascular anatomy and neuroanatomy.

Praise for the previous edition:

"Several avian books are available for practitioners working with pet birds and most claim to be comprehensive and easy to use. Unfortunately, many don't succeed in accomplishing both aims. This book does."

–Julia Whittington, DVM, University of Illinois College of Veterinary Medicine in *Doody's Review Service*

"The author clearly draws on a wealth of experience and has written a book with much to recommend…"

–*Journal of Small Animal Practice*

"…chapters are extremely useful for any veterinarian, as they vividly describe the basics of avian medicine and how to apply them... many practical tips and scenarios... [images] are of high quality and are well labelled.."

–*Australian Veterinary Journal*

CRC Press
Taylor & Francis Group
an informa business
www.crcpress.com

6000 Broken Sound Parkway, NW
Suite 300, Boca Raton, FL 33487
711 Third Avenue
New York, NY 10017
2 Park Square, Milton Park
Abingdon, Oxon OX14 4RN, UK

K24223
ISBN: 978-1-4822-6020-5
90000
9 781482 260205

Downloaded From Vet-eBooks.com