

Ultimate Veterinary Notes Bundle

Vet Helpful Notes
Canine Feline

Thick Forceps

Arrhythmia

Intervention/Key Points

First degree AV Block

Second degree AV Block

Third degree AV Block

Sinus Arrhythmia

Tachycardia

Supraventricular Tachycardia

ALL ABOUT

K+ AND PHOS SUPPLEMENTATION

Oral dosing

Canine Cushing

Excess Cortisol

Clinical Signs

Injury!

Arachidonic Acid

TXA2

Kidney Blood Flow & Platelet Function

Never administer K+ and Steroids together!

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Canine Feline

VET ACRONYMS

A	Definition
AAHA	American Animal Hospital Association
AAFCO	American Association of Feed Control Officials
Ab	Antibody
ABVP	American Board of Veterinary Practitioners
ACE	Angiotensin-Converting Enzyme
ACTH	Adrenocorticotrophic Hormone
AD	Right Ear
ADH	Antidiuretic Hormone
ADR	Aint Doin Right
AGID	Agar Gel Immunodiffusion
AIDS	Acquired Immunodeficiency Syndrome
ALP	Alkaline Phosphatase
ALT	Alanine Aminotransferase
ANA	Antinuclear Antibody
ARDS	Acute Respiratory Distress Syndrome
AS	Left Ear
AST	Aspartate Aminotransferase
AU	Both Ears
AUC	Area Under the Curve
AUS	Abdominal Ultrasound Exam
AVMA	American Veterinary Medical Association

B	Definition
BAR	Bright, Alert, Responsive
BCS	Body Condition Score
BDLD	Big Dog Little Dog
BLS	Basic Life Support
BID	Two Times a Day
BPM	Beats per Minute
Bp	Blood Pressure
BUN	Blood Urea Nitrogen
BW	Body Weight
Bx	Biopsy

C	Definition
C-1, C-2, C-7	Cervical Vertebrae
CBC	Complete Blood Count
CBF	Cerebral Blood Flow
CFU	Colony Forming Unit
CHF	Congestive Heart Failure
CN 1-12	Cranial Nerves I-XII
CNS	Central Nervous System
CP	Conscious Proprioception
CPR	Cardiopulmonary Resuscitation
CRTZ	Chemoreceptor Trigger Zone
CSF	Cerebral Spinal Fluid
CVP	Central Venous Pressure

Canine Feline

VET ACRONYMS

D	Definition
D+	Diarrhea
DES	Diethylstilbestrol
DHPPL	Distemper Hepatitis Parvo Parainfl Lepto
DI	Diabetes Insipidus
DIC	Disseminated Intravascular Hemolysis
DJD	Degenerative Joint Disease
DLE	Discoid Lupus Erythematosus
DM	Diabetes Mellitus
DNR	Do Not Resuscitate
DOA	Dead on Arrival
DSH	Domestic Short Hair
Ddx	Differential Diagnosis

F	Definition
FAD	Flea allergy dermatitis
FelV	Feline Leukemia Virus
FHV	Feline Herpes Virus
FIP	Feline Infectious Peritonitis
FIV	Feline Immunodeficiency Virus
FLUTD	Feline Lower Urinary Tract Disease
FNA	fine Needle Aspirate
FORL	Feline Odontoclastic Resorptive Lesion
FUO	Fever of Unknown Origin
FUS	Feline Urologic Syndrome
Fx	Fracture
FVRCP	Feline Viral Rhinotracheitis Calici Panleukopenia Virus

E	Definition
ECF	Extracellular Fluid
Ecoli	Escherichia Coli
EEG	Electroencephalogram
EKG	Electrocardiogram
ELISA	Enzyme-Linked Immunosorbent assay
EPI	Exocrine Pancreatic Insufficiency
EPO	Erythropoietin
ETT	Endotracheal Tube

G	Definition
GABA	Gamma-Aminobutyric acid
GDV	Gastric Dilatation Volvulus
GFR	Glomerular Filtration Rate
GI	Gastrointestinal
GME	Granulomatous Meningoencephalitis

H	Definition
Hb Hgb	Hemoglobin
HBC	Hit by Car
Hct	Hematocrit
HGE	Hemorrhagic Gastroenteritis
HOD	Hypertrophic Osteodystrophy
HPA	Hypothalamic Pituitary Axis
HPF	High power field
Hx	History

VET ACRONYMS



I	Definition
IBD	Inflammatory bowel disease
ICF	Intracranial fluid
ID	Intradermal
IDST	Intradermal skin testing
IgG	Immunoglobulin G
IGR	Insect growth regulator
IM	Intramuscular
IP	Intraperitoneal
IV	Intravenous
IVDD	Intervertebral disc disease

L	Definition
LD	Lethal dose
LH	Leuteinizing Hormone
LMN	Lower Motor Neuron
LPF	Low Power Field
LRS	Lactated Ringer's Solution
LVT	Licensed Veterinary Technician

J	Definition
JAAHA	Journal American Animal Hospital Association
JAVMA	Journal American Veterinary Medical Association

M	Definition
MAC	Minimum alveolar concentration
MCH	Mean Corpuscular Hemoglobin
MCHC	Mean Corpuscular Hemoglobin Concentration
MCT	Mast Cell Tumor
MCV	Mean Cell Volume
ME	Metabolizable Energy
MHz	Megahertz
MIC	Minimum Inhibitory Concentration
MLV	Modified Live Virus
MRI	Magnetic Resonance Imaging
mRNA	Messenger RNA

K	Definition
KCS	Keratoconjunctivitis Sicca
kVp	Kilovoltage Peak

VET ACRONYMS



N	Definition
NAVLE	North American Vet Licensing Exam
NPO	None Per Os
nRBC	Nucleated Red Blood Cell
NSAID	Non Steroidal Anti-inflammatory

P	Definition
PCV	Packed Cell Volume
PDA	Patent Ductus Arteriosus
PE	Physical Exam
PIE	Pulmonary Infiltrates with Eosinophils
Plt	Platelets
PMN	Polymorphonuclear Cells
PNS	Peripheral Nervous System
PO	Per OS (by mouth)
PRA	Progressive Retinal Atrophy
PRN	As Necessary
PSGAG	Polysulfated glycosaminoglycans
PT	Prothrombin Time
PTT	Partial thromboplastin Time
PtH	Parathyroid Hormone
PTHrp	Parathyroid Hormone Related Protein
PTS	Put to Sleep
PUPD	Polyuria, Polydipsia

O	Definition
O	Owner
OA	Osteoarthritis
O.D.	Right eye
OFA	Orthopedic Foundation for Animals
OR	Operating Room
O.S.	Left eye
O.U.	Both Eyes

VET ACRONYMS



Q	Definition
QAR	Quiet, Alert , Responsive
Q.I.D	Four Times a Day (NOT Q.D.!)
QOL	Quality of Life

T	Definition
T.I.D.	Three Times a Day
TMJ	Temporomandibular Joint
TPR	Temp, Pulse, Respiration
TNTC	Too Numerous to Count
TPN	Total Parenteral Nutrition
TSH	Thyroid Stimulating Hormone
TTw	Transtracheal Wash
T4	Thyroid hormone
Tx	Treatment

U	Definition
UA	Urinalysis
UPC	Urine Protein: Creatinine Ratio
UMN	Upper Motor Neuron
URI	Upper Respiratory Infection
USG	Urine Specific Gravity
UTI	Urinary Tract Infection

V	Definition
V+	Vomiting
VD	Ventrodorsal
VMD	Veterinary Medical Doctor
VWF	Von Willebrands Factor
VX	Vaccine

VET ACRONYMS



W	Definition	X	Definition
WBC	White Blood Cells	XRAY	Radiograph
WNL	Within Normal Limits		

VET DIRECTIONAL TERMS

Term	Definition
Mesial	In or near the middle of the dental arch
Axial	Pertaining to the axial skeleton (head, spine, ribcage)
Saggital	A plane parallel to the median plane of the body, separates body into right and left sides
Oblique	Slanted or inclined plane
Transverse	Separates the body into front and back
Lateral	Pertaining to a side
Ventral	Pertaining to the belly or underside. Opposite: dorsal
Dorsal	Pertaining to the back or top of body. Opposite: ventral
Cranial	Pertaining to the head
Caudal	Pertaining to the hind end or tail
Frontal	Plane that separates the body into top and bottom
Rostral	Pertaining to the nose
Palmar	Pertaining to the pads/bottoms of front feet
Plantar	Pertaining to the pads/bottoms of back feet

VET TERMINOLOGY



Body Part/Organ	Medical Terminology	Example
Air	Pneumo	pneumothorax
Anus	Anal, ano-	perianal gland
Arm	brachial, brachio	brachial plexus
Blood	Hem- hemo-hemat-	hematemesis
Chest	Thoracic, thorax, thoraco	thoracolumbar
Ear	Auricle, oto-	otoscopic
Eye	Ocular, oculo, ophthalmo-	ophthalmology
Foot	Pedal, ped- pod	pinnal-pedal
Gallbladder	Chole, chol-	cholestatic
Head	Cephalic, cephalo	oculocephalic
Heart	Carium, cardiac, cardio	pericardium
Kidney	Renal, nephric, nephro	nephrology
Lip	Cheil- labi-	cheilitis
Liver	Hepatic, hepato	hepatomegaly
Lungs	Pulmonary, pulmonic	pulmonic vein
Mouth	Oral, os, stoma, stomat-	stomatitis
Muscle	Myo-	myocardium
Nail Bed	Ungual	subungual
Neck	Cervix, cervical, cervico-	cervicothoracic
Penis	Penile	penile prolapse
Peritoneum	Periton-	Retroperitoneal
Rectum	Rectal	Rectal prolapse
Skin	Derma, integumentum	dermatitis
Spleen	Splen-	Splenomegaly
Stomach	Gastic, gastro-	gastric dilation
Testicle	Orchio, orchido	orchectomy
Bladder	Cysti-, cysto-	cystocentesis
Uterus	Hystero-, metra	hydometra, hysterectomy
Vulva	Vulvo-	vulvoplasty

VET PREFIXES/SUFFIXES



Suffixes	Meaning	Example
-algia	pain	arthralgia- painful joints
-dynia	pain	allodynia- neuropathic pain to normal stimuli
-emia	blood condition	anemia- low circulating red cell count
-ia	condition, disease	arthralgia painful condition of the joints
-itis	inflammation	arthritis- joint inflammation
-lepsy	seizure or attack	epilepsy- idiopathic seizure condition
-malacia	softening	osteomalacia- bone softening
-megaly	enlargement	hepatomegaly- liver enlargement
-oma	tumor, mass	lipoma- fatty mass
-osis	abnormal condition	osteoporosis- bone loss condition
-pathy	disease	neuropathy- nervous system disease
-penia	deficiency	osteopenia- losing bone density
-plegia	paralysis	paraplegia- paralysis of half the body
-pnea	breathing	apnea- not breathing
-sclerosis	hardening	arteriosclerosis- hardening of blood vessels
-rrhage	bursting forth	hemorrhage- bleeding out
-rrhea	flow, discharge	diarrhea- watery stool
-rrhexis	breaking open	aminorrhesis- rupture of amniotic sac
-centesis	puncture, aspiration	cystocentesis- needle aspiration of urine
-ectomy	surgical removal	pericardectomy - removal of pericardial sac
-genic	producing, forming	hallucinogenic- producing hallucinations
-graphy	process of recording	radiography- taking xrays
-lysis	separation	hemolysis- red blood cells breaking down
-opsy	view of, vision	colonoscopy- view of colon
-plasty	surgical repair	episiotomy- repairing the skin around vulva
-scopy	visual examination	cystoscopy- visual examination of bladder
-stomy	opening	esophagostomy tube- tube in esophagus
-tomy	incision, cutting	gastrotomy- incision into stomach
-uria	urine, urination	stranguria- straining to urinate

VET PREFIXES/SUFFIXES



Prefixes	Meaning	Example
A-	an, no, not, without	atony- without tone, flaccid
Ab-	away from	abduct- move away from midline
Ad-	toward, near	adduct- move towards midline
Ana-	up, apart	anabolic- building up your metabolism
Ante-	pro, pros, before, forward	antemortem- before death
Anti-	against	anti-diarrhea- prevent diarrhea
Auto-	self	autologous serum- serum from the pet
Bi-	two	bisect- divide in two
Brady	deficient	bradycardia- slow heartrate
Chromo	color	chromogenesis- the production of color
Co- Com- Con-	with, together	comorbidities- ancillary diseases
Cyano	blue	cyanosis- turning blue from lack of O2
De-	down, lack of	dehydrate- lacking hydration, dry
Deci-	one tenth	deciliter- 1/10th of a liter
Dia	through, between	Diastasis- separation of the abdomen
Dis-	apart, away from	Disability- having limitations
Dys-	bad, painful	Dysphagia- trouble eating
Ecto-	outside	ectocornea- outerlayer of the cornea
Endo-	within, in, inner	Endodontics- working within the tooth
Epi	above, upon	epidermis- outer layer of skin
Ex-	out	exophytic- extruded out
Extra-	outside of	extrathoracic- outside the chest
Erythro-	red	erythroid cell line- red cell line
Hemi-	half	hemithorax- half of the chest
Hexa-	six	hexagonal crystal- 6 sided crystal
Hyper	excessive, above	hypercoagulable- coagulates easily
Hypo-	below, deficient	hypothyroid- low thyroid function
Infra	below, beneath	infraorbital fat pad- fat below the orbit
Intra	within	Intraabdominal- within the abdomen

VET PREFIXES/SUFFIXES



Prefixes	Meaning	Example
Leuko-	White	Leukotrichia- white hair
Macro-	large	Macroglobulinemia- excess IgM production
Mal	bad	Malocclusion- irregular bite
Melano-	black	Melanocyte- pigmented cell
Meta-	change	Metacarpal bones
Micro	small	microscope- to examine small things
Mono	one	monocyte- cell with a single nucleus
Multi	many	Multiparous- producing multiple offspring
Neo	New	neonate- newborn
Pan	all	pancytopenia- all cell lines are low
Para	near, alongside	parathyroid - glands near the thyroid
Peri	surrounding	pericardial sac- sac around the heart
Post	after, behind	postmortem- after death
Pre- Pro- Pros-	before, forward	prosencephalon- part of brain in front
Poly	many, much	polydactyl- many digits
Pseudo	false	pseudopregnancy- false pregnancy
Re	back, again	rejuvenation- make younger
Retro-	behind, backward	retroperitoneal- behind the peritoneum
Sub-	less than, beneath	subcutaneous- beneath the skin
Supra-	above, upper	supraventricular- above the ventricles
Sym- Syn-	with, together	symbiotic- to exist together
Tachy-	fast	tachycardia- fast heartrate
Trans-	across, through	transplant- to move some where else
Tri-	three	Tricuspid valve- valve with 3 leaflets
Ultra-	beyond	ultrapure- very purified
Xanth-	yellow	xanthoderma- yellow skin

SPECIAL LAB TESTS IN THE VET HOSPITAL

ETSY.COM/SHOP/VETHHELPFULNOTES



LIVER



BILE ACIDS

Test of liver function. Often used to investigate a possible liver shunt

PROTEIN C

Testing for levels of a protein synthesized by the liver. Low levels suggest a portosystemic shunt

PANCREAS



PLI

Pancreatic lipase immunoreactivity: elevated in pancreatitis

TLI

Trypsin like immunoreactivity: a low value indicates EPI (exocrine pancreatic insufficiency) a disorder of the pancreas: low digestive pancreatic enzymes.

INSULIN: GLUCOSE

This test is submitted when an insulin secreting tumor is suspected. High insulin in the face of a low blood sugar suggests such a tumor is present (insulinoma of the pancreas)

THYROID



FREE T4

Normal T4 is susceptible to suppression by illness, free T4 is not

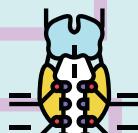
TSH

Thyroid stimulating hormone. Is elevated with hypothyroidism, it tries to make the body produce thyroid hormone.

T4 ABB

Autoimmune thyroiditis is diagnosed by high thyroid autoantibodies.

PARATHYROID



PTH

Parathyroid hormone Elevated or high normal with hyperparathyroidism. A high calcium and normal PTH means hyperparathyroidism.

PTHRP

Parathyroid related protein. This protein is expressed in some cancers that produce elevated calcium.

KIDNEY



UPC

Urine protein: creatinine ratio. Measures protein leakage across the nephron into the urine. A persistently elevated UPC indicates renal damage

FIRST A.M. USG

Urine specific gravity: test of urine concentration. Most concentrated sample of the day is typically first thing in the morning. Helps assess kidney function: normal concentration= normal kidney function

BLADDER



BRAF OR BLADDER

TUMOR ANTIGEN

A test of the urine that looks for markers of a bladder tumor

BLOOD



BUFFY COAT

A white thin layer that sits above the red cells and below the plasma in a spun blood tube. This is all white blood cells. A buffy coat exam is used to look for circulating mast cells in the blood.

COOMBS

A positive COOMBS indicates antibodies to red blood cells. Is used to help diagnose IMHA (immune mediated hemolytic anemia)

SLIDE AGGULTINATION

A drop of blood mixed with a drop of saline. If the cells clump under the microscope, that suggests they are coated with antibodies and IMHA is present

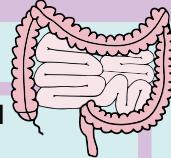
BMBT

A buccal mucosal bleeding time: measuring the time to stop a small cut in the gums. This is a test of platelet function. A delayed bleeding time is abnormal.

FLOW CYTOMETRY

A special test used to "sort" cells to identify different cell populations. Often used to investigate elevated lymphocyte counts to see if there is leukemia

GI TRACT



FECAL ALPHA 1 PROTEINASE INHIBITOR

Presence of this protein in the stool indicates likely GI disease (leaky gut) like IBD (protein losing enteropathy)

COBALAMIN AND FOLATE

Cobalamin (B12) is vital for intestinal health. Excess folate is produced by overgrowth of gut bacteria. High folate and low cobalamin can be indicators of GI disease.



ADRENALS

LDDS

Low dose dexamethasone suppression test: Used to determine if there is overactivity of the adrenal gland (cushings disease)

ACTH STIM

A test of adrenal gland function by using a hormone to stimulate the adrenal glands. Used to detect low adrenal function (addison's) or over activity of the adrenal glands (cushings)



MUSCLES/NERVES

2M ANTIBODY

Used to test for an immune mediated disease of the muscles of mastication (chewing): Masticatory Muscle Myositis

ACEYTLCHOLINE RECEPTOR ANTIBODY

Used to diagnose an immune mediated disease of nervous input to muscles: Myasthenia Gravis. Antibodies form against the acetylcholine receptor on muscle



CARDIAC

PROBNP

A test of a cardiac enzyme that increases with heart enlargement

COMMON BLOOD TESTS IN THE VET HOSPITAL



BLOOD TEST

MEASURES

WHAT FOR



CBC

RED AND WHITE CELLS,
PLATELETS

CHECK FOR ANEMIA,
HEMOCONCENTRATION,
HIGH OR LOW
PLATELETS



CHEM

KIDNEY AND LIVER ENZYMES
BLOOD PROTEINS, ELECTROLYTES
CALCIUM, PHOSPHOROUS,
CHOLESTEROL

CHECK ORGAN
FUNCTION, MEASURE
ELECTROLYTES, PROTEIN
LEVELS



I STAT

ELECTROLYTES,
IONIZED CALCIUM, LACTATE

CHECK FOR
ELECTROLYTE
DISTURBANCES, MEASURE
ACTIVE CALCIUM
FRACTION, CIRCULATION



PT/PTT

CLOTTING TIMES

MEASURE FOR
DISTURBANCES IN
BLOOD
CLOTTING



BILE ACIDS

PRE AND POST PRANDIAL (EATING)
BILE ACID LEVELS

MEASURES LIVER
FUNCTION
(HOW LIVER HANDLES
BILE ACIDS)



LDDS

SUPPRESSION OR LACK OF
SUPPRESSION OF ADRENAL
CORTISOL PRODUCTION

USED TO DETERMINE IF
A PATIENT HAS
CUSHINGS, AND
DISCERN PITUITARY OR
ADRENAL DEPENDANT
DISEASE



ACTH STIM

CORTISOL MEASUREMENT
IN RESPONSE TO STIMULATING
HORMONE

DETERMINE IF A PATIENT
HAS ADDISON'S (LOW
ADRENAL FUNCTION)



BLOOD GAS

BLOOD PH, OXYGEN, CARBON
DIOXIDE, ACID/BASE

BLOOD CIRCULATION,
VENTILATION/PERFUSION

Medical Math Conversions and Formulas

Conversions Units

Conversion Units: Weight

1 Kg = 1L = 1000 gm = 1000 ml

1 gm = 1000 mg

1 mg = 1000 mcg (micrograms)

1 pound = 16 oz

1 Kg = 2.2 lbs

1 oz = 0.0625 lbs

1 oz = 0.028 kg

1 grain = 64.8 mg

Conversion Units: Volume

1 L = 1000 ml

1 cup = 8 oz = 240 ml

1 ml = 1000 ul (microliters)

1 cc = 1ml

1 Tbs = 15 ml

1 tsp = 5 ml

1 ml = 20 drops

Conversion Units: Misc

celscius= (degree F -32) x 5/9

Farenheight = (degree C x 9/5) + 32

1% = 10 mg/ml

10% = 100 mg/ml

Common Conversions

Pounds to Kilograms: DIVIDE by 2.2

Kilograms to Pounds: MULTIPLY by 2.2

Kilograms to Grams: MULTIPLY by 1000

Ounces to Pounds: DIVIDE by 16

OR

MULTIPLY by 0.0625

Ounces to Kilograms: Convert to lbs,
then DIVIDE by 2.2

% solution to mg/ml: PUT a ZERO by the %

$mg \div mg/ml = ml(s)$

$mg \div mg/cap = cap(s)$

$mg \div mg/tab = tab(s)$

$kg \times mg/kg = mg$

Conversions Examples

POUNDS to KILOGRAMS: DIVIDE by 2.2

How many Kilograms does a 25 lb dog weigh?

$25 \text{ lb} \div 2.2 = 11.36 \text{ Kg}$

KILOGRAMS to POUNDS: MULTIPLY by 2.2

How many pounds does a 0.378 kg Kitten weigh?

$0.378 \text{ kg} \times 2.2 \text{ lbs/kg} = 0.8316 \text{ lbs}$

OUNCES to POUNDS : DIVIDE by 16 oz/lb

How many pounds does a 4.4 oz Kitten weigh?

$4.4 \text{ oz} \div 16 \text{ oz/lb} = 0.275 \text{ lbs}$

OUNCES to KILOGRAMS:

1) convert to lbs

2) $0.275 \text{ lbs} \div 2.2 \text{ lbs/kg} = 0.125 \text{ Kg}$

Formulas To Know

Injections and Oral dosing

Amount = $\frac{\text{Dose}}{\text{on Hand}}$

Flow Rate Infusion Pump

Flow Rate = $\frac{\text{Volume}}{\text{Time}}$

Drug Rate

$kg \times mg/kg/hr \div mg/ml = ml/hr$

Spike a bag with a solution

$\frac{\text{Concentration you want}}{\text{Concentration you have}} \times \text{volume you want}$

= Volume to add

$C1V1=C2V2$

Oral dosing

Order : A 20 kg dog needs 10 mg/kg of oral doxycycline twice a day for 30 days.

Doxycycline is available in 100 mg tablets. How many tablets do you dispense?

Calculate the twice daily dose. $20 \text{ kg} \times 10 \text{ mg/kg} = 200 \text{ mg per dose}$

$$\begin{array}{rcl} \text{Amount} = & 200 \text{mg} & \\ & \hline & 100 \text{mg /tab} \\ & = & 2 \text{ tablets per dose} \end{array}$$

3. Two tablets , twice a day for 30 days

= 4 tablets per day x 30 days

= 120 tablets

ORAL DOSING WITHIN TABLET SIZES:

Order: Rimadyl dosed at 2.2 mg/kg twice a day for a week for a 50.6 lb dog. The Clinic has 100 mg and 25 mg tablets on hand.

1. Convert the dog's weight to kg: $50.6 \div 2.2 = 23 \text{ kg}$

2. Solve for mg amount of medication given twice a day: $23 \text{ kg} \times 2.2 \text{ mg/kg} = 50.6 \text{ mg}$

3. Figure out how much of each tablet size you can use: **50.6 mg rounded down is 50mg**

So, you can use **1/2 of a 100 mg tablet per dose**



In other words 1/2 tablet twice daily of the 100 mg tabs = 1 tab per day x 7 days=

Seven 100 mg tabs



OR

2 of the 25 mg tablets per dose



25 mg Tablet

2 tablets per dose of the 25mg tabs = 4 tabs per day x 7 days

= Twenty Eight 25 mg tabs



You choose the 100 mg tabs because you can dispense less tablets and that is more cost-effective

If you're making the UNITS LARGER
move the decimal to the LEFT 3 X

10.0 mg → .010 gm

SMALLER unit to LARGER UNIT, makes
the number SMALLER .

If you break the pie into BIGGER pieces,
you need less pieces to make up the pie

ug -----> mg

Divide by 1000

mg -----> gm

gm -----> kg

If you're making the UNITS SMALLER
move the decimal to the RIGHT 3 X

10.0 Kg → 10,000 gm

LARGER unit to SMALLER UNIT, makes
the number BIGGER .

If you break the pie into SMALLER pieces,
you need more pieces to make up the pie

kg -----> gm

Multiply by 1000

gm -----> mg

ug -----> mg

Remember to move the DECIMAL to the LEFT 3 PLACES
when converting a SMALLER unit to LARGER unit

Move the DECIMAL to the RIGHT 3 PLACES
when converting a LARGER unit to a SMALLER unit

mcg to mg, mg to grams, grams to Kg

Kg to grams, grams to mg, mg to mcg

Injectable dosing

INJECTION ORDER: Give a patient 150 mcg of dexmedetomidine IM

How many ml do you need to draw up of a 0.5mg/ml solution of dexmedetomidine?

1. Convert Dexmedetomidine solution to mcg/ml so everything is in the same units

0.5mg/ml dexmedetomidine x 1000 mcg/mg = 500 mcg/ml solution of dexmedetomidine

MOVE THE DECIMAL 3 PLACES TO THE RIGHT

2. Calculate how many mls you need for 150 mcg

AMOUNT = $\frac{\text{Dose}}{\text{on Hand}}$

D = Desired Dose

H = Medication on Hand

A = Amount of medication you need (SOLVE for this)

AMOUNT = 150 mcg

$\frac{150 \text{ mcg}}{500 \text{ mcg/ml}}$

= **0.3 ml dexmedetomidine**

INJECTION ORDER: A 6 kg cat needs 30 mcg/kg of transmucosal buprenorphine.

Buprenorphine comes as a 0.3 mg/ml solution. What volume of buprenorphine needs to be administered to this cat?

1. First convert to the same units. $0.3 \text{ mg/ml} \times 1000 \text{ mcg/mg} = 300 \text{ mcg/ml}$ solution
2. Calculate the dose needed for the cat: $6 \text{ kg} \times 30 \text{ mcg/kg} = 180 \text{ mcg}$

3. Amount = 180 mcg (DOSE) = **0.6 ml is needed**
3. $\frac{180 \text{ mcg}}{300 \text{ mcg/ml}}$ (HAVE)

Oral Liquid dosing

Liquid Dose Order: A 3.2 kg dog needs a 2mg/kg dose of oral furosemide. Furosemide comes as a 5% solution. How much volume is the dose?

1. Convert the furosemide solution you have into mg/ml: 5% = **50 mg/ml** of furosemide
2. Calculate the dose needed in mg: $3.2\text{kg} \times 2\text{mg/kg} = 6.4\text{ mg}$ is needed **per dose**
3. Amount = 6.4mg = **0.128 ml is needed (draw up 0.13ml)**

50mg/ml



Spiking a fluid Bag

Order: Dextrose supplementation

You need to spike a bag to 5% dextrose. The fluid bag contains 450 ml of fluid. You have a 50% dextrose solution. How many ml of dextrose do you add to the bag?

Volume to add = 5% (concentration you want)

X 450 ml (volume you want)

50% (concentration on hand)

0.1×450

= **45 ml of 50% dextrose to add to 450 ml of fluid**

Thought of another way, if you had 100% dextrose, you'd need 5% of 450ml ($0.05 \times 450 = 22.5\text{ ml}$). But, you have a half-strength solution (50% dextrose) so you need double the volume: 45 ml.

Order: Insulin Infusion

You need to spike a 250 ml bag of 0.9% NaCl with 1.1 units/Kg Regular Insulin. You are treating a 10 kg dog. Humulin R is 100 units/ml. How many ml of insulin do you add to the bag?

1. How many units do you need to add to the bag: $1.1\text{ units/Kg} \times 10\text{ kg} = 11\text{ units}$

2. Calculate the dose needed in ml:

Amount = 11 units = **0.11 ml is added to the bag**

100 units/ml

CRI out of a fluid bag

Order: 2 mg/kg/day Metoclopramide CRI

A 5 kg dog needs 2 mg/kg/day added to a liter of fluids to be delivered at 22ml/hr.

How many ml of a 5 mg/ml metoclopramide do you add to a Liter of fluids?

1. Figure out how many mg given per hour:

5 kg x 2 mg/kg/day = 10 mg/day of metoclopramide or 10 mg every 24 hours

10 mg ÷ 24 hours = **0.42 mg metoclopramide per hour**

2. Figure out how many hours 1 bag lasts run at 22 ml/hr

1000 ml/bg ÷ 22 ml/hr = The bag lasts 45.45 hours

3. Mg added to the bag: 45.45 hours x 0.42mg per hour = 19.09 mg (round down to 19 mg) added to the bag

4. Convert the mg into ml: 19mg ÷ 5mg/ml metoclopramide = 3.8ml of metoclopramide to 1 liter

OR

0.42 mg metoclopramide/hr. Convert to ml/hr = 0.42 mg/hr x 1ml/5mg = 0.084 ml/hr

22 ml/hr rate = 0.084 ml metoclopramide per 22ml = 0.084/22 = 0.0038 ml metoclopramide per ml

0.0038 ml x 1000ml = 3.8 ml of metoclopramide in 1 liter

BONUS

If you have a partially used bag of fluids, you can figure out the volume to add to 1 Liter, then divide by 1000ml to know the volume added per ml. Then multiply that by the ml in the partially used bag.

Example: 1 liter bag has only **650ml** left in it. You need **3.8 ml per Liter**. OR $3.8 \text{ ml} \div 1000 = 0.0038 \text{ ml metoclopramide per ml}$ of fluid in the bag. $650\text{ml fluid} \times 0.0038 = 2.47\text{ml}$

metoclopramide added to the 650ml remaining in the bag.

Syringe Pump Infusion

Order: A dog in status epilepticus needs a Diazepam CRI run at 0.5mg/kg/hr. You're treating a 17 kg dog. Diazepam is available as a 5mg/ml solution. You want enough to last 4 hours.

17kg dog x 0.5mg/kg/hr = 8.5mg diazepam per hour

$8.5 \text{ mg/hr} \div 5 \text{ mg/ml} = 1.7 \text{ ml/hr of diazepam delivered via syringe pump}$ OR

$8.5 \text{ mg/hr} \times 1\text{ml}/5\text{mg} = 1.7 \text{ ml/hr}$

If you're planning to deliver the CRI for 4 hours, you need $1.7 \text{ ml/hr} \times 4 \text{ hr} = \textbf{6.8 ml diazepam}$

Order: Fentanyl 4mcg/kg/hr to a 48 kg dog Post-op. Fentanyl comes as a 0.05mg/ml solution

1) Convert to the same units. $0.05\text{mg/ml} = 50\text{mcg/ml}$ solution

2) $4 \text{ mcg/kg/hr} \times 48\text{kg} = 192 \text{ mcg/hr}$

3) $192\text{mcg/hr} \div 50 \text{ mcg/ml} = 3.84 \text{ ml/hr of fentanyl}$

4) If you're planning to have enough for 6 hours of fentanyl: $3.84 \text{ ml/hr} \times 6 \text{ hours} = \textbf{23.04 ml}$ of fentanyl is needed

CRI Examples

Order: Nitroprusside 2 mcg/kg/minute run at 12 ml/hr

Nitroprusside is a 25mg/ml solution

A 3.8 kg dog needs 2mcg/kg/minute of Nitroprusside added to 500 ml bag of fluids to be delivered at 12 ml/hr.

How many ml of a 25mg/ml nitroprusside do you add to a 500ml bag of fluids?

1. Figure out how many mcg given per minute:

$$3.8 \text{ kg} \times 2 \text{ mcg/kg/minute} = 7.6 \text{ mcg/minute}$$

2. Figure out how many mcg per hour:

$$7.6 \text{ mcg/minute} \times 60 \text{ minutes/hour} = 456 \text{ mcg/hour}$$

3. Figure out how many mcg per ml:

We know that 12 ml of fluid are delivered every hour. So, that means there are 456 mcg per 12 ml.

$$\text{This can be reduced to : } 456 \text{ mcg/12 ml} = 38 \text{ mcg/ml}$$

4. Figure out mg nitroprusside per fluid bag:

We have a 500ml bag. So $38 \text{ mcg/ml} \times 500 \text{ ml/bag} = 19000 \text{ mcg/500ml bag}$ or 19 mg/500 ml bag

5. Figure out ml of nitroprusside per bag:

$19 \text{ mg of nitroprusside} \times 1 \text{ ml}/25 \text{ mg nitroprusside} = 19/25 = 0.76 \text{ ml Nitroprusside added to 500ml.}$ Remember to take out 0.76ml from your bag first. So, you add 0.76ml of Nitroprusside to 499.24 ml of fluid in your bag.

Another way :

$3.8 \text{ kg} \times 2 \text{ mcg/kg/min} \times 60 \text{ min/1 hour} \times 1 \text{ mg}/1000 \text{ mcg} \times 1 \text{ ml}/25 \text{ mg} \times 1 \text{ hr}/12 \text{ ml} \times 500 \text{ ml/bag} = 0.76 \text{ ml/bag.}$ All the units get crossed out to end up with ml/bag at the end.

Now Titrate...

You're now asked to **INCREASE** the rate from 2 mcg/kg/minute to 4 mcg/kg/minute

What is the new fluid rate in ml/hour?

The fluid rate of 2 mcg/kg/minute is 12 ml/hour. If we double it to 4 mcg/kg/minute, we double the rate per hour to 24 ml/hour

Another way:

Our fluid bag has 0.76ml nitroprusside/500ml
 $0.76 \text{ ml} \times 25 \text{ mg/ml} = 19 \text{ mg nitroprusside}/500 \text{ ml} = 0.038 \text{ mg/ml} = 38 \text{ mcg/ml nitroprusside}$

We want 4 mcg/kg/minute. $4 \text{ mcg/kg/minute} \times 3.8 \text{ kg} = 15.2 \text{ mcg/minute} \times 60 \text{ min/hour} = \text{need } 912 \text{ mcg/hr.}$ $912 \text{ mcg/hr} \times 1 \text{ ml}/38 \text{ mcg} = 912/38 = 24 \text{ ml/hr}$

Make an Easy To Titrate CRI

Order:

Start Norepi at 0.1mcg/kg/minute- (norepinephrine =1mg/ml)

Weight of patient is 6.24Kg

0.1mcg/kg/minute x 624 kg = 0.624 mcg/min x 60 min/hr = 37.4 mcg/hour or .0374 mg/hr

Because NorEpi is 1mg/ml : divide 37.4 mcg by 1000 to turn the number into mg

0.0374 mg/hour which is the same as 0.0374 ml/hour of nor Epi

(0.0374 mg/hr x 1ml/mg = 0.0374 ml/hr)

If you want to draw up 10 hours of medication at that rate, multiply the number by 10

0.0374 ml/hr of Nor Epi x 10 hours = **0.374 ml** of 1mg/ml NorEpi **to last 10 hours** if run at 0.1mcg/kg/minute **

If you want your CRI to run at 1ml/hr to equal 0.1mcg/kg/minute, you want your solution to have 0.0374 mcg of Nor epi in every 1 ml delivered to the patient***

Dilute it into 10ml by adding 0.374ml (0.374mg) of Norepi to 9.63ml of d5W.
You will have 10 hours of Norepi if run at **1ml/hr = 0.0374 mg/hr**

NOW if your solution is run at 1ml/hr it equals 0.1mcg/kg/minute (0.0374 mg/hr)

If you run it at 2ml/hr you deliver 0.2mcg/kg/minute, AND
3ml/hr you deliver 0.3 mcg/kg/minute
4ml/hr you deliver 0.4mcg/kg/minute
5ml/hr you deliver 0.5mcg/kg/minute **

<https://www.etsy.com/shop/VetHelpfulNotes>

Your Own Notes

Decoding Labwork

Kidney



BUN: Blood Urea Nitrogen

Increases with kidney disease, dehydration, heart disease, poor perfusion, urinary obstruction, high protein diet; Decreases with over-hydration, liver dz

Creat: Creatinine

Increases with kidney disease and similar conditions that increase BUN. However, it is not affected by high protein diet; Normally increased in Greyhounds
Decreases with over-hydration, muscle loss

Phos: Phosphorous

Increases seen with kidney disease, increased intake from GI tract, increased vitamin D, increased in growing puppies and kittens; Decreases with increased urinary excretion, decreased GI absorption, insulin therapy, low vitamin D

Ca: Calcium

Increases with decreased kidney function, certain cancers, some toxicities, parathyroid disease;
Decreases with parathyroid surgery, low albumin, urinary obstruction

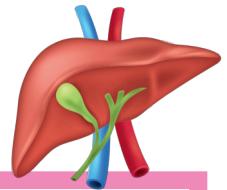
UPC: Urine Protein: Creatinine Ratio

Increased: Pre-renal: hypertension, shock, Renal: glomerular damage in kidney disease, Post-renal: UTI, active urine sediment. Screening tool in lyme positive dogs

USG: Urine Specific Gravity

Determines the kidney's ability to concentrate urine. Isosthenuria (1.008-1.012) occurs with 67% or more kidney nephron dysfunction, hyposthenuria (<1.008) can happen with Diabetes Insipidus or over-consumption of water. Hypersthenuria (>1.030 dog, >1.035 cat) indicates dehydration

Liver



ALT: Alanine Aminotransferase

Increases indicate liver cell damage. Mild increases in dogs is 2-5 x the high end of normal. ALT increases in cats indicates active cell damage because the half life is short (hours)

ALKP: Alkaline Phosphatase

Increases may indicate liver abnormality (cholestasis), Cushing's disease, rises with steroid use in dogs only, active bone remodeling after boney injury, drug induced. Elevations are normal in growing puppies and kittens

GGT: Gamma Glutamyl Transferase

Increases may represent a type of liver disease that causes cholestasis. Often increased with ALKP

AST: Aspartate Aminotransferase

Increases associated with liver diseases that produce direct cell damage. Often increases with ALT. Can also indicate muscle injury

TBili: Total Bilirubin

Increases seen with liver disease (cholestasis and liver disease) or pancreatitis; can also increase with red blood cell destruction

Bile Acids

Increases with liver dysfunction, abnormalities in blood flow to the liver or bile duct obstruction
Is a liver function test used to detect liver shunts

Glu: Glucose

Increases due to diabetes mellitus, stress response in cats. Decreases: liver disease, sepsis, poor perfusion, insulinoma

Decoding Labwork

Pancreas



Amyl: Amylase

Increases can be with pancreatitis (but not specific/sensitive marker), kidney disease, GI disease, drugs

Lipa: Lipase

Increases with pancreatitis (but not sensitive/specific), kidney disease, GI disease, drugs

Spec cPL: Canine Pancreatic Specific Lipase

A normal snap test is best to rule out pancreatitis; positive snap should have quantitative test (spec cPL) submitted to measure the value. Above 400 is consistent with pancreatitis

Electrolytes



Na: Sodium

Increases with dehydration, over supplementation (activated charcoal); Decreases with GI losses (v/d), Addison's, Kidney disease, Drugs (lasix)

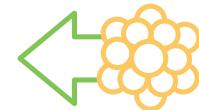
K: Potassium

Increases with kidney disease, Addison's, dehydration, kidney obstruction. Decreases: GI losses (v/d), aldosterone-secreting tumor

Cl: Chloride

Increases with dehydration, Decreases with GI losses (v/d), loop and thiazide diuretics

Lipids



Proteins



TP: Total Protein

TP is albumin + globulin. Increases can come from dehydration or inflammation. Decreases can occur from poor liver function, blood loss, GI or kidney losses

Alb: Albumin

Albumin is a measured protein and is the a major determinant of maintenance of fluid within the vascular space (oncotic pressure). Increases with dehydration; Decreases with blood loss, decreased liver production, GI and kidney disease.

Glob: Globulin

Calculated by TP-Albumin. Made by the liver and immune system. Increased with inflammation and infection; Decreases with blood loss, GI loss and immune deficiencies

Trig: Triglycerides

Commonly elevated in miniature schnauzers. Increases can be due to a recent meal, pancreatitis, Cushing's or hypothyroidism

Chol: Cholesterol

Increases due to diabetes mellitus, hypothyroidism, Cushing's, pancreatitis, nephrotic syndrome; Decreases can be due to Addison's, PLE (protein losing enteropathy), liver failure

Muscle



AST: Aspartate Aminotransferase

Can be elevated due to muscle injury as well as liver disease

CK: Creatine Kinase

Increases due to muscle damage

Helpful conversions/calculations

Head trauma fluid boluses

Hypertonic saline fluid boluses

3-7.5% HTS solution 4-6ml/kg per dog and 2-3 ml/kg per cat over 15-20 minutes. No more than twice

Mannitol is 20% solution or 200 mg/ml solution

Mannitol dose: 0.5-1gm/kg IV (2.5ml /kg to 5ml/kg) over 30 to 60 minutes

Weight conversions

1kg= 2.2 lbs Kg --> lb /2.2

1 lb= 0.45 kg Lb --> Kg x 2.2

1kg=1000gm

1gm=1000mg

1mg=1000ug

ug-->mg-->g-->Kg divide by 1000
Kg-->g-->mg-->ug multiply by 1000

calculating fluids

Maintenance fluid rate for a cat = 50ml/kg/day

Maintenance fluid rate for a dog= 60ml/kg/day

5% dehydration: $0.05 \times \text{Kg of pet} \times 1000 =$
ml fluid deficit to correct. To correct over 24 hours divide by 24 and add to fluid rate

Dog blood volume estimate 90ml/kg; shock dose is typically 25% of this over 20-30 minutes

Cat blood volume estimate 50ml/kg; shock dose is typically 25% of this over 20-30 minutes

what exponents mean

$10^3 = 1000 = \text{thousand}$

$10^6 = 1,000,000 = \text{million}$

$10^9 = 1,000,000,000 = \text{billion}$

K+ to supplement 1 L

Patient's K+ (mEq/L)	Amount K+ to add per L of 2mEq/ml
3.5-4	20 mEq (10 ml)
3-3.5	30 mEq (15 ml)
2.5-3	40 mEq (20 ml)
2-2.5	50 mEq (25 ml)
<2	60 mEq (30 ml)

% Solutions in mg/ml

100mg/ml=10% solution

10mg/ml=1% solution

1mg/ml=0.1% solution

volume conversions

1L = 33.8 oz

1tbs= 3 tsp=15ml

1 cup=240ml=8oz=16 tbs

1000 ml= 1 Kg

Basic formula tabs

Dose x Quantity x frequency of admin

Have

Order: cephalexin 200mg twice a day for 10 days

Pharmacy has 400mg tabs

How many tabs for 10 day course?

200mg x 10 days x twice daily

400mg

1/2 tab twice a day x 10 days
(1/2 tab x 2) = 1 tab per day x 10 days
= 10 total tablets dispensed

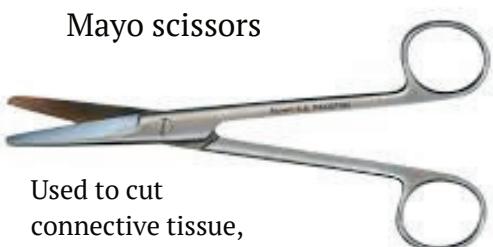
Scissors

Blunt sharp scissors



Used to cut suture. They are less expensive than Mayo or Metzenbaum scissors which should be reserved to cut tissue only.

Mayo scissors



Used to cut connective tissue, muscle, fascia

Tenotomy scissors



Used for delicate surgery

Wire Cutter scissors



Used to cut stainless steel wire

Littauer Suture scissors



Used to hook suture loops to remove stitches

Metzenbaum scissors



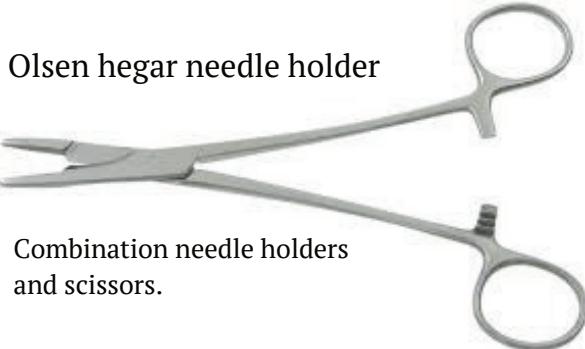
Used for fine dissection and cutting; long handle to blade ratio

Lister Bandage scissors



Used for cutting into bandage layers without harming tissues underneath

Olsen hegar needle holder



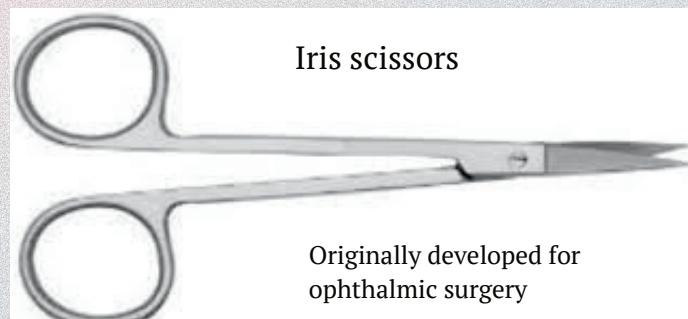
Combination needle holders and scissors.

Mayo hegar needle holder



Mayo Hagar needle holders have no scissors

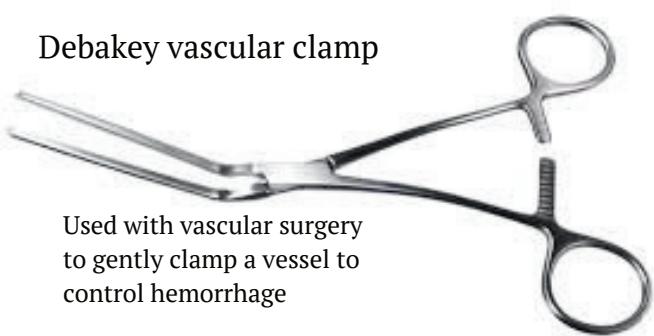
Iris scissors



Originally developed for ophthalmic surgery

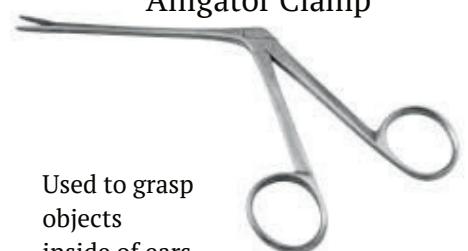
Clamps and Forceps

Debakey vascular clamp



Used with vascular surgery to gently clamp a vessel to control hemorrhage

Alligator Clamp



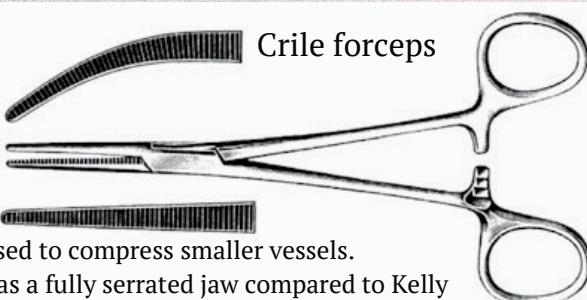
Used to grasp objects inside of ears

halsted mosquito forceps



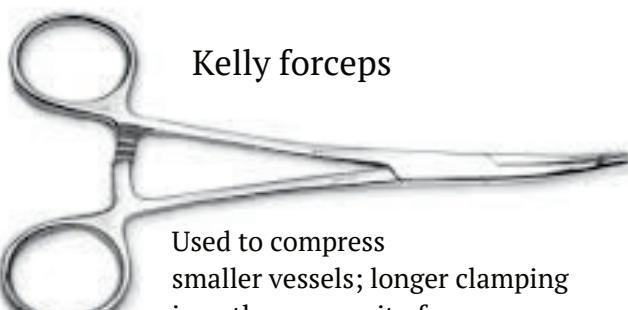
Used to compress smaller vessels

Crile forceps



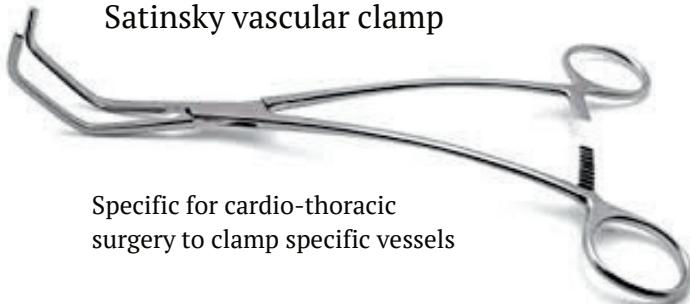
Used to compress smaller vessels.
Has a fully serrated jaw compared to Kelly

Kelly forceps



Used to compress smaller vessels; longer clamping jaws than mosquito forceps

Satinsky vascular clamp



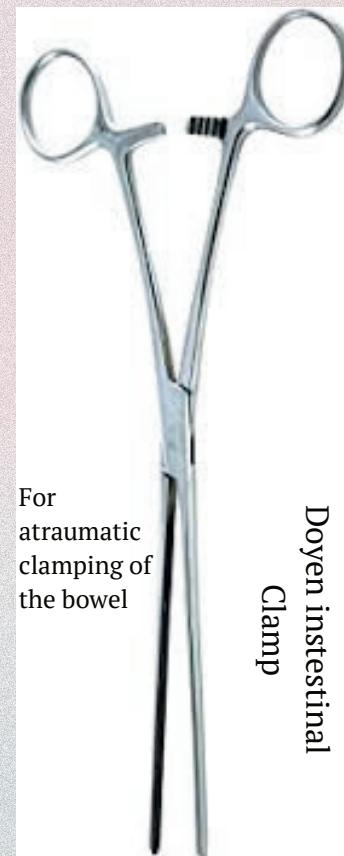
Specific for cardio-thoracic surgery to clamp specific vessels

Lovelace Lung Grasping Forceps



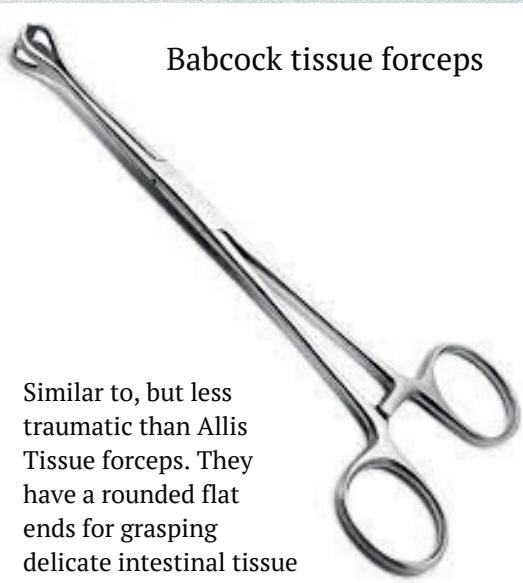
Triangular grasping area distributes force over the area for delicate tissues like the lung.

Doyen intestinal Clamp



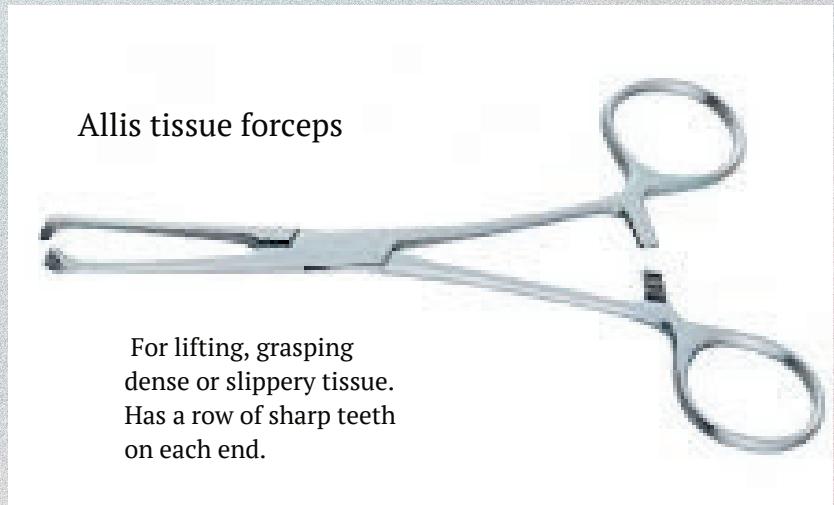
For atraumatic clamping of the bowel

Clamps and Forceps



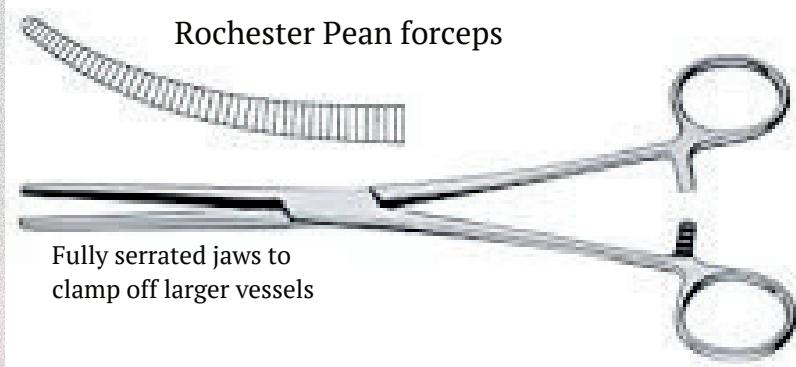
Babcock tissue forceps

Similar to, but less traumatic than Allis Tissue forceps. They have a rounded flat ends for grasping delicate intestinal tissue



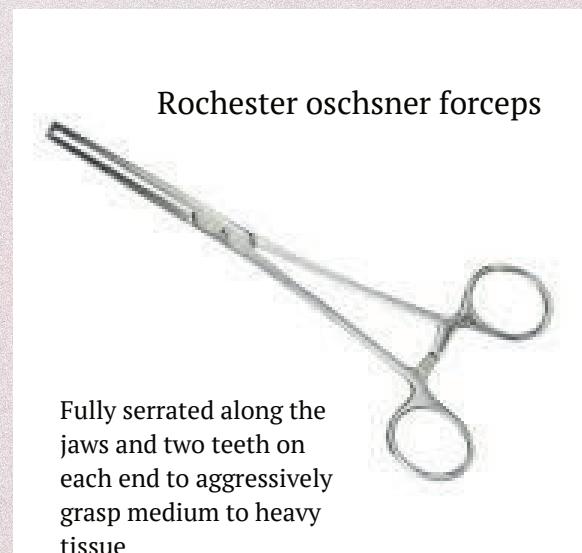
Allis tissue forceps

For lifting, grasping dense or slippery tissue.
Has a row of sharp teeth on each end.



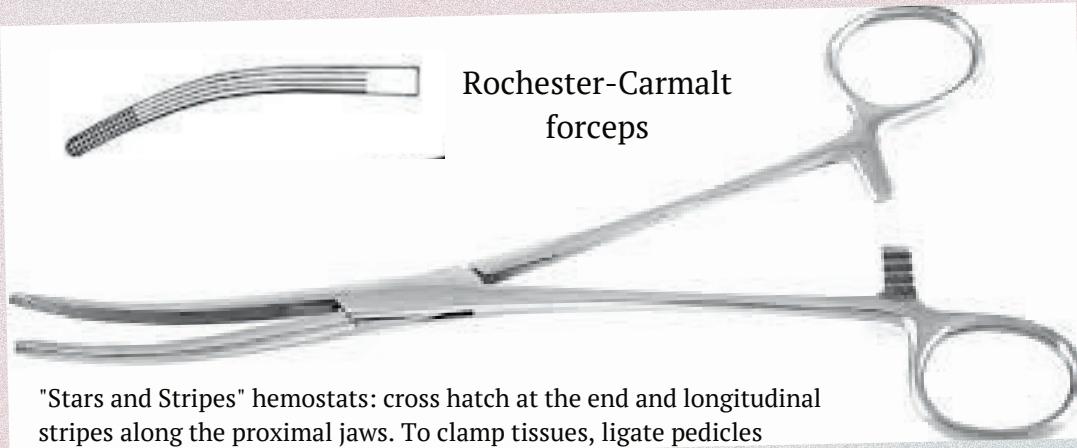
Rochester Pean forceps

Fully serrated jaws to clamp off larger vessels



Rochester oschsner forceps

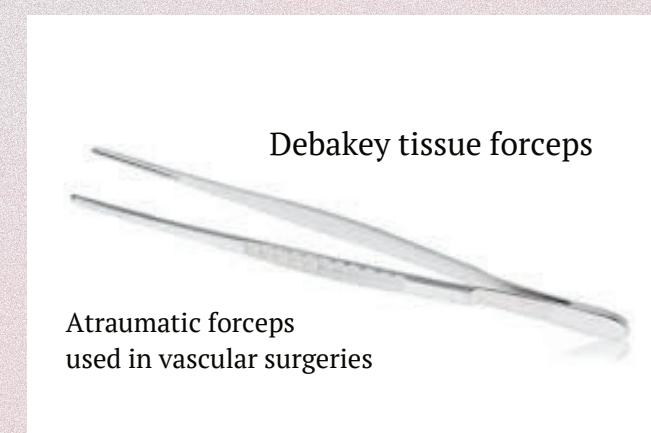
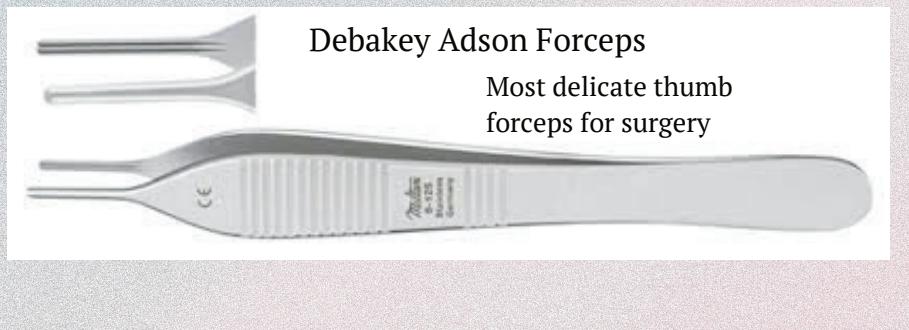
Fully serrated along the jaws and two teeth on each end to aggressively grasp medium to heavy tissue



Rochester-Carmalt forceps

"Stars and Stripes" hemostats: cross hatch at the end and longitudinal stripes along the proximal jaws. To clamp tissues, ligate pedicles

Thumb Forceps



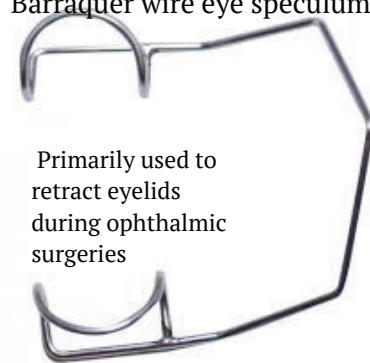
Retracting Tools

Snook Spay Hook



Primarily used to locate the ovary during a spay surgery

Barraquer wire eye speculum



Primarily used to retract eyelids during ophthalmic surgeries

A double ended retractor used to retract tissues during surgery. Has a curved fork like end.

Army Navy Retractor



Used to retract shallow incisions: skin, fat, muscle

Senn Miller Retractor

Malleable ribbon retractors



Commonly used during orbital procedure to retract orbital fat. Can be bent to adjust as needed.

Gelpi Retractors



self-retaining retractors to hold back tissues for the surgeon

Beckman weitlaner retractors



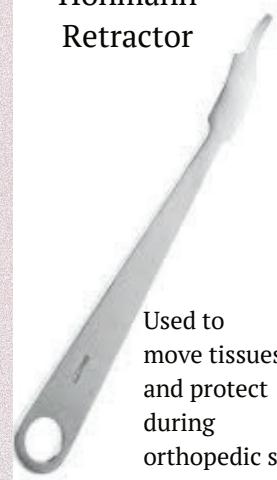
self-retaining retractors to hold open shallow incisions such as in spinal surgeries

Small Animal Vaginal Speculum



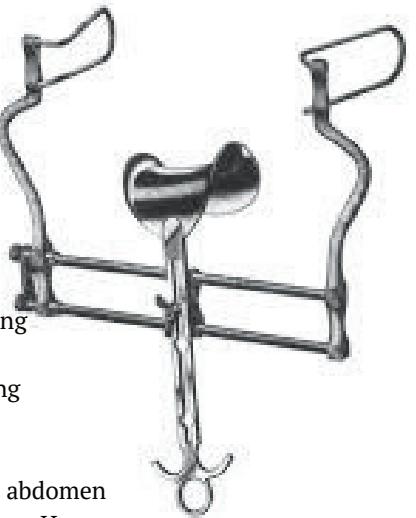
used to retract tissues to allow better visualization during female urethral catheterization.

Hohmann Retractor



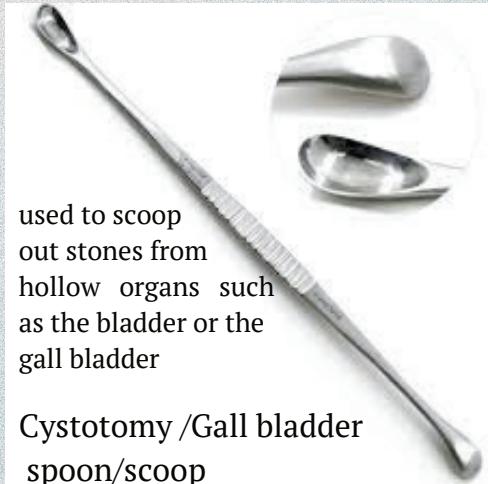
Used to move tissues and protect during orthopedic sx

Balfour Abdominal Retractors

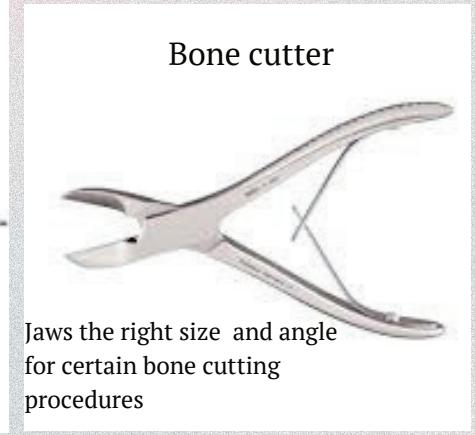
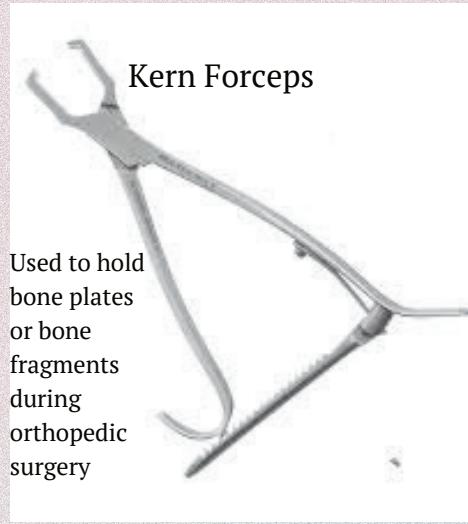
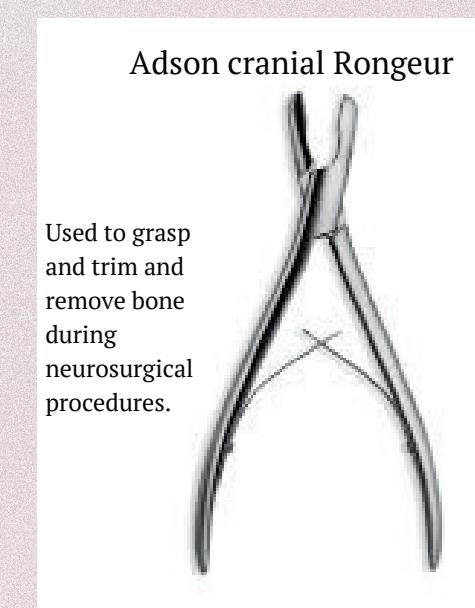
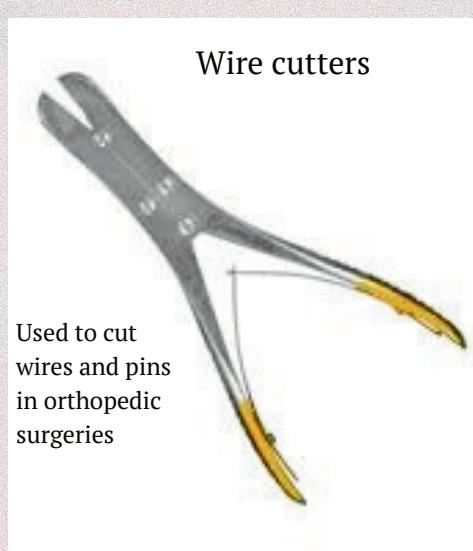
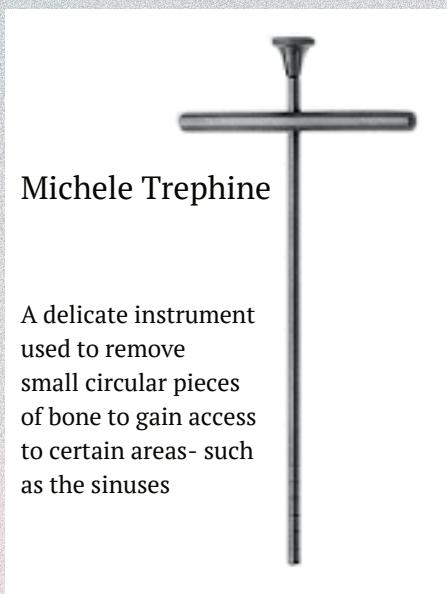


Self-retaining retractor useful during abdominal procedures to open the abdomen for inspection. Has a 7 to 10 inch spread

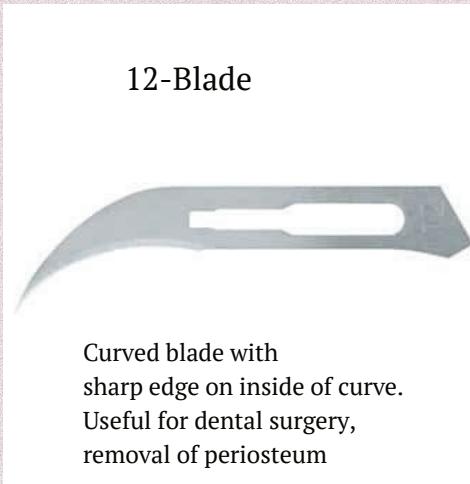
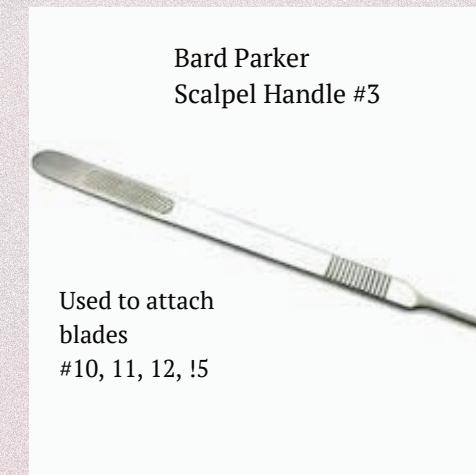
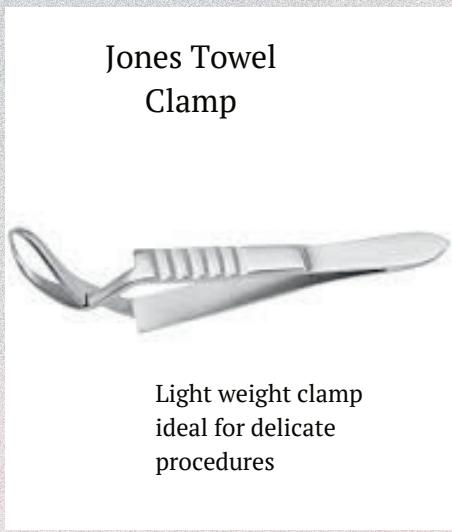
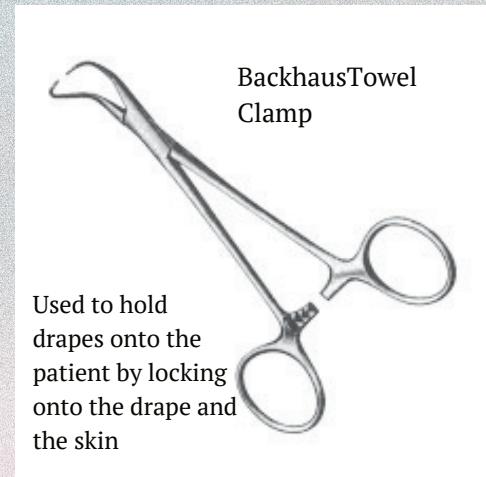
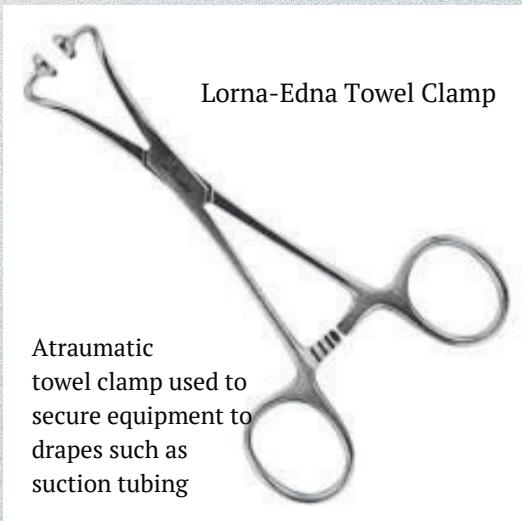
Curettes, Bone and Stone Tools plus suction tool



Volkmann Bone Curette



Towel Clamps, Blades



Suture Types

Non-Absorbable Suture



Silk

SILK: Multifilament

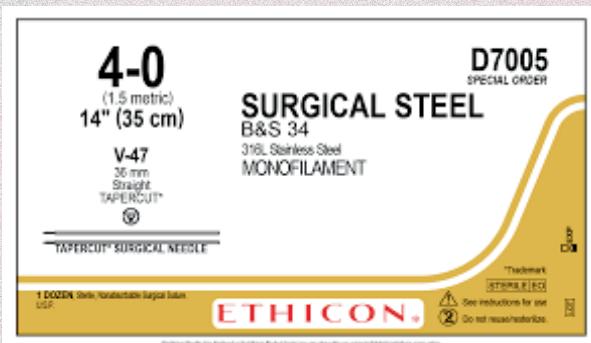
Although silk is considered a non-absorbable suture, it degrades slowly (>720 days) by phagocytosis. Silk is a good choice for vessel ligation because it does not slip off vessels. Soft; easy to work with; good knot security; high tissue reactivity. Generally limited to mouth, lips, eyelids. Where patient comfort is significantly better. Should not be used for skin closure

- |- - - - -
- | **Suture size**
- | Weakest to strongest
- | (thinnest to thickest)
- | 12-0, 11-0, 10-0, 9-0, 8-0,
- | 7-0, 6-0 5-0, 4-0, 3-0, 2-
- | 0, 1-0, 2-0, 1-0,
- | 0 1 2 3 4 5 6 7
- |
- | 2-0 -3-0 : abdominal wall
- | in small dogs and cats
- | 1-0, 0 large dogs
- |
- | Often times use 3-0-4-0
- | in SC tissues and skin
- |
- |
- |- - - - -



Nylon

Nylon: Multifilament and Monofilament inexpensive and causes minimal tissue reaction. Nylon has poor handling properties and knot security. This type of suture is a good choice for skin closure. High tensile strength.



Steel: Monofilament

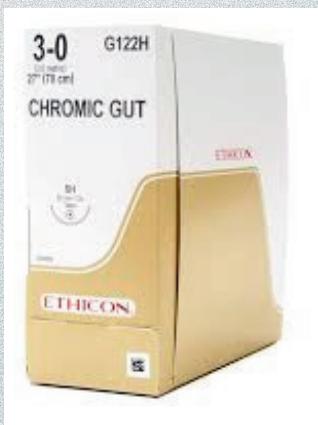
The suture is available as a monofilament and multifilament suture. Surgical stainless steel suture is indicated for use in abdominal wound closure, hernia repair, sternal closure and orthopedic procedures including cerclage and tendon repair. Biologically inert with high tensile strength.



Prolene: Monofilament, synthetic, Polypropylene: Indefinitely sticks around like steel, Poorest knot security. Can be used when there is prolonged healing and infection present. Does not adhere to tissue.

Suture Types

Absorbable Suture



Chromic Gut: Beef serosa, sheep submucosa, natural, **absorbable, monofilament.**, 90 days to absorption. Surgical gut is not recommended for closure of the GI tract or urinary bladder because of unpredictable absorption rates. It's best feature is it's rapid absorption rate. Best for tissues that heal quickly like the oral cavity.



Monocryl: **poliglecaprone 25, synthetic, monofilament**
90-120 days to absorption. Rapidly healing tissues (eg, subcutaneous and urinary bladder tissue). Good knot security and easy handling.

Most needles used in surgery are curved to allow better handling in tight spaces. The needles attached to suture are either cutting or non cutting needles. Cutting needles are used to penetrate tissue like fascia and skin.

Tapered needles are used to create the smallest hole and are good for tissue where little effort is needed to penetrate the tissue.

Suture is either multifilament (braided) or monofilament.

Multifilament suture is more pliable and easy to handle than monofilament suture. However, the braided suture is more at risk for harboring bacteria and is not good for use in infected tissues.



PDS II : **polydioxanone, synthetic monofilament**
180-210 days to absorption
Polydioxanone and polyglyconate are good suture choices for tissues such as fascia. Low tissue drag, strong knot security. Can be used in infected tissues.



Vicryl: **Polyglactin 910 , synthetic, multifilament**
56-70 days to absorption
These sutures are appropriate for vessel ligation and SC tissue closure. Soft, easy to handle. Good knot security. Preferred suture for dentistry: does not adhere plaque



Erythrocytes and Platelets

RBC: Red blood cell Red blood cell. The most numerous blood cell. Critical for oxygen delivery. Red cells also participate in acid/base balance.

HCT: Hematocrit % of blood volume composed of red blood cells. Increases: dehydration, splenic contraction, breed related, bone marrow disorders. Decreases: increased loss (hemorrhage, parasitism), increased destruction (immune mediated) or decreased production (chronic disease, nutritional)

Hg: Hemoglobin Iron containing protein inside of red blood cells that carries oxygen. Hg x 3 is a rough estimate of PCV. Increases: same as for HCT. Artifact from lipemia, use of hemoglobin derivatives (oxyhemoglobin), intravascular hemolysis. Decreases: same as for HCT

MCV: Mean Cell Volume Represents the average size of red blood cells. Increases: regenerative anemia, bone marrow disorders such as FeLV, artifact. Decreases: iron deficiency, liver failure or portosystemic shunt, hereditary microcytosis of Asian dogs, abnormal red cell shape (fragments, spherocytes).

MCH: Mean Corpuscular Hemoglobin Calculated hemoglobin per cell, irrespective of red cell size. Increases: Artifact from hemolysis, use of hemoglobin derivatives (oxyhemoglobin). Decreases: regenerative anemia, reticulocytosis without anemia (splenic contraction, compensated anemia), decreased hemoglobin production (chronic blood loss, copper or B6 deficiency)

MCHC: Mean Corpuscular Hemoglobin Concentration: Calculated hemoglobin in a specific red cell volume. Increases and decreases the same as for MCH. MCHC is considered more specific for evaluating hypochromasia (RBC's that are more pale due to decrease hemoglobin)

Retic: Reticulocyte Immature red blood cells without a nucleus that are released into the blood to correct anemia. Used to distinguish regenerative from non regenerative anemias. Increases: represent a response to anemia and the total number can reflect mild, moderate or marked responses.

Leukogram

WBC: White blood cell

Composed of the sum of the immune cells: neutrophils, lymphocytes, monocytes, eosinophils, and basophils. Increases: inflammation, infection, neoplasia, corticosteroids. Decreases: decreased production, increased tissue demand, immune mediated loss

Neutrophil: The most abundant WBC. They respond to inflammation and infection. Capable of phagocytosis.

Increases: Inflammation, infection, corticosteroids, epinephrine induced (excitement), neoplasia. Decreases: same as WBC

Lymphocytes: White blood cells derived from lymphoid tissues. Involved with cell mediated and humoral immune responses. Increases: stress induced, Addison's, lymphoid neoplasia, antigenic stimulation, more common in young animals. Decreases: corticosteroids (drug, Cushing's), infection, loss of lymphocyte rich fluid (chyle), hereditary

Monocytes: White blood cells that phagocytose, present antigen and signal cytokine production Increases: inflammation, infection, corticosteroids, neoplasia

Eosinophils: white blood cells that are involved with allergic disease and that combat parasitism and other infectious disease. Increases: allergy, infection (parasitic, fungal, FeLV), endocrine: Addison's, hyperthyroidism, neoplasia, bronchopneumopathy

Basophils: granulated white blood cells increased with allergies, parasitism, and neoplasia. The least abundant WBC.

Platelets: Also called a thrombocyte. Integral to clot formation and primary hemostasis.

Increases: increased production, corticosteroids (drug, Cushing's), neoplasia, inflammation, rebound from previous thrombocytopenia or blood losses. Decreases: decreased production, increased consumption, increased destruction

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Decoding the CBC



Erythrocytes and Platelets



Interpreting Anemia:

Anemia should signal the bone marrow to pushout immature red blood cells (reticulocytes) into the peripheral blood stream to replace losses.

However, it takes about 3 days for an adequate response.

A non regenerative anemia is anemia that is present without elevations in the reticulocyte count.

A non regenerative anemia can be due to early blood losses (< 3 days), or a malfunctioning bone marrow. However, chronic illness classically produces a non regenerative anemia and is the most common form of anemia encountered in our patients.

Interpreting Reticulocytosis:

reticulocyte percentage is not as useful to interpret degree of response- because it does not take into account the severity of the anemia.

Mild regeneration: 150-200K Dog, 80-100K Cat

Mod regeneration: 200-300K Dog, 100-200 Cat

Marked regeneration: >300K Dog, >200K Cat

Portosystemic shunts and iron deficiency anemias classically produce a "**microcytic, hypochromic anemia**" (small , pale RBC's)

FeLV in cats classically produces a non-regenerative anemia characterized by **macrocytosis** because the virus causes bone marrow problems producing red blood cells "dyserythropoiesis". Their MCV's are typically in the 60's, compared to 37-49 fL

Greyhounds: known to have **higher HCTs** and hemoglobin concentration. They are often used as blood donors. (concurrently they often have **lower platelet** counts and **elevated creatinine** due to their high muscle mass.

Asian breeds often have a **macrocytosis of their red cells** as well as a tendency to have an elevated potassium.

Platelets

Interpreting thrombocytopenia:

PLT count	Risk for hemorrhage	Relative deficiency
<10K	highest risk	marked decrease
<30K	high risk	marked decrease
30-50K	moderate risk	moderate decrease
50-100K	minimal risk	moderate decrease
>100K	marginal risk	mild decrease

Cavalier King Charles Spaniel: asymptomatic macrothrombocytopenia. Their platelet counts are low and can be a low as 30-60K. The platelets are also very large.

Leukogram

Interpreting a leukocytosis:

Stress leukogram: secondary to increased endogenous (Cushings) or exogenous steroids. Pattern: Neutrophilia, lymphopenia, eosinopenia and monocytosis. Other changes are often elevated glucose and ALP,

The opposite is seen in patients with LOW endogenous steroids (ie **Addisonian** patients): they will often have a **lymphocytosis** (in the face of illness) and a **eosinophilia** in addition to other classic electrolyte changes of high potassium and low sodium.

Physiologic leukocytosis: flight or fight response due to epinephrine release causes a mature neutrophilia and lymphocytosis. Most commonly seen in cats and young animals. No left shifting.

Left shift: presence of immature neutrophils (bands) in the blood. Cytokine signals the bone marrow of an increased demand for neutrophils so the bone marrow releases from it's storage pools immature forms (bands). Moderate to severe toxic change is often seen in the neutrophils.

Degenerative left shift: When demand for recruitment of white cells is so overwhelming (more severe infection or inflammation), then stores of mature neutrophils in the blood may be depleted and bands (immature neutrophils) from the marrow may exceed neutrophil counts. Normal or low mature neutrophils, higher numbers of bands than mature neutrophils present.

Regenerative left shift: Neutrophilia with higher numbers of mature cells than immature cells. The bone marrow has had enough time to respond to the peripheral demand for neutrophils.

Neutropenia: lower than normal numbers of neutrophils. A general rule of thumb is if mature neutrophil counts drop to 1000 or less, then that patient is at risk for sepsis. The most common causes of neutropenia in our patients is infection and secondary to chemotherapeutic drugs.

Pelger Huet anomaly: mostly the Australian shepherd, have peanut shaped (rather than trilobed) nuclei.

Canine Vaccines Schedule

Canine Core Vaccines

Canine Distemper Virus

- MLV OR Recombinant
- 3 Forms:
DAP: Distemper + Adenovirus-2 + Parvo virus
DAPP: DAP+ Parainfluenza
DAPP-L: DAPP + Lepto

** note DHPP is the same as DAPP (H is for hepatitis, A in DAPP is Adenovirus the hepatitis virus)

Canine Rabies Virus

1 and 3 year vaccines are available.
First rabies vaccine is always 1 year, regardless of using the vaccine labeled for 3 years

Administration

Beg at 6-8 weeks, sequential dosing every 3-4 weeks (q 2 weeks in shelters) until at least 16 weeks of age.

Ex: 8, 12, 16 weeks. Final dose at or after 16 weeks of age to ensure immunity

If risk of exposure is high, then an additional final dose at 18 to 20 weeks of age

Booster

Administer combination product no later than 1 year from last dose in series

Administer subsequent boosters every 3 years



Second dose scheduled no later than 1 year from first administration.
Then, every 3 years
State/local law applies

Canine Non-Core Vaccines

Bordetella Bronchiseptica

3 forms:

- Killed bacterin : SQ
- Avirulent live: Oral
- Avirulent live combined with parainfluenza: IN

Leptospirosis

Killed 4 serovar. Don't recc regular use of 2 serovar vaccine serovar: strain of leptospirosis

Administration

Intranasal (IN) vaccine may be given as early as 3-4 weeks of age. Intranasal or Oral typically given at 12 or 16 weeks once.

Subcutaneous dose: 2 doses 2-4 weeks apart.

ALERT!!! IN or Oral vx given subcutaneously can cause a fatal liver reaction!!

Initial vaccination requires 2 doses 2-4 weeks apart, starting at 8 weeks of age or older

Booster

Administer 1 year later if there is sustained exposure risk (indoors with other dogs such as kennels, daycare, grooming)
Annually thereafter

Administer 1 year later then annually thereafter

Borrelia burgdorferi

Recombinant, chimeric/recombinant, or Killed whole cell

Canine influenza (H3N2/H3N8)

Killed viral vaccine

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Initial vaccination requires doses 2-4 weeks apart
Small breed dogs <20 lbs consider delaying initial doses until CORE vaccines completed

Administer 1 year later then annually thereafter

Initial vaccination requires doses 2-4 weeks apart

Administer 1 year later then annually thereafter



Core Canine Diseases



Canine Distemper Virus

WHAT: a paramyxovirus that is shed in all secretions, but most commonly respiratory secretions. Young dogs are most at risk.

SYMPTOMS: Decreased appetite, fever, lethargy and signs of **respiratory illness** (nasal discharge, pneumonia). Can cause neurologic symptoms such as seizures ("chewing gum" seizures characterized by repetitive mouth movements) and ataxia. In later stages of the disease it can cause hyperkeratosis of the nose and foot pads **"hard pad disease"**.

DIAGNOSIS: Most commonly by **RT PCR** from conjunctival swabs, urine samples. In the early stages of the disease occasionally can find large aggregates of virus as an inclusion body in erythrocytes and leukocytes.

TREATMENT: **No specific treatment.** Supportive care.

VACCINATION: The canine distemper vaccine is highly effective and preventing infection. The vaccine is often combined with canine adenovirus (canine hepatitis), parainfluenza and parvovirus (DA2PP or DHPP).

Canine Adeno Virus Type 2

WHAT: Canine **adenovirus type 2** is one of the causes of **canine infectious tracheobronchitis**.

Canine **adenovirus (CAV) type 1** is the causes of **canine hepatitis**.

VACCINATION: The modified live vaccine only includes canine adenovirus type 2 but induces protection against CAV-1 AND CAV-2. This core vaccine is often combined with canine distemper and parvovirus and canine parainfluenza to create DA2PP or DHPP (A2 and H are interchangeable).

SYMPTOMS: Adenovirus type 2 causes respiratory symptoms. Adenovirus type 1 causes hepatitis. Symptoms include fever, vomiting, diarrhea, abdominal pain, and clotting disorders. In late stages of the disease ocular signs such as uveitis and corneal edema may occur.

DIAGNOSIS: ELISA, serology, PCR . Often diagnosed by history and symptoms.

TREATMENT: Supportive care.

Canine Parvo Virus

WHAT: Canine parvovirus is a **highly contagious disease** that is shed in the feces and is **spread via fecal-oral contact**. The virus is resistant to may disinfectants and is able to **survive in the environment for weeks to months**. Solutions of bleach 1:30 can kill the virus.

SYMPTOMS: Diarrhea, vomiting, dehydration and fever. Young dogs are most at risk. There is an increased incidence of the disease in Dobermans and Rottweilers.

DIAGNOSIS: Parvo Snap Test

TREATMENT: Supportive care to correct dehydration and control vomiting and diarrhea. The **virus** attacks rapidly dividing cells and this affects the lining of the **intestinal tract** as well as the **bone marrow**.

VACCINATION: provides very good protection. Some vaccine failure from stopping last vaccine too early (ie 14 weeks vs beyond 16 weeks when maternal interference may reduce antibody response).

Canine Rabies Virus

WHAT: Rabies is caused by rhabdovirus which is a **zoonotic virus** that affects the **nervous system**. It is most commonly transmitted through bite wounds. Rabies is endemic in **skunks, bats, foxes raccoons and coyotes**.

SYMPTOMS: Fever, difficulty swallowing, aggression, seizures, drooling, even paralysis

DIAGNOSIS: Diagnosis can only be made by submission of the brain for testing.

TREATMENT: There is no available treatment for rabies.

VACCINATION: The vaccine is available as an adjuvanted killed vaccines and non-adjuvanted recombinant vaccines. Almost 100% survival rate in vaccinated animals with a rabies antibody level greater than 0.5 IU/L. **Rabies vaccination is required by law** but the State and local laws dictate the frequency of administration (annually or every 3 years).

Important Non-core Canine Diseases

Bordetella Bronchiseptica

WHAT: Bordetella Bronchiseptica respiratory bacteria: One of the primary causes of canine infectious tracheobronchitis (kennel cough). Infection is most easily spread in crowded indoor environments like shelters, doggie daycare centers, pet shops, groomers etc.

SYMPTOMS: Dry, honking cough and sometimes nasal discharge. Bacteria are spread through respiratory secretions . This is transmitted via airborne droplets or via dog to dog contact: licking, touching noses, etc.

DIAGNOSIS: Most often diagnosed simply by history and exam findings. Can be formally diagnosed via culture of an airway wash.

TREATMENT: Supportive care treatment of cough. Rarely treated as a pneumonia.

VACCINATION: Two main forms of the vaccine are available: a modified live avirulent intranasal vaccine and an injectable inactivated antigen extract vaccine.

Canine Parainfluenza Virus

WHAT: Parainfluenza virus is a paramyxovirus that is one of the many causes of canine infectious tracheobronchitis (kennel cough).

SYMPTOMS: The main symptom is a **self limiting, non productive cough**.

DIAGNOSIS: Typically based on physical exam and symptoms. PCR from oral/ocular swabs can formally diagnose.

TREATMENT: Supportive care for cough and ancillary symptoms

VACCINATION: Available as an injectable product in combination with distemper, parvovirus and adenovirus. The injectable form only protects against clinical symptoms, not viral shedding. The **intranasal product** is available in combination with bordetella and canine adenovirus type 2 vaccines. This vaccine is considered **superior** because it protects against clinical signs and also prevents viral shedding.

Leptospirosis

WHAT: Leptospirosis is a **spirochete bacteria** called leptospira and is transmitted through contact with **infected urine shed from wildlife**: raccoons, skunks, squirrel, deer, opossum, rats, moles etc. The bacteria can survive for weeks in warm moist environments and can be spread via exposure of the animal to contaminated water, food, soil, bedding or direct contact with urine. The bacteria can cross mucous membranes via small scratches or contact with the eyes. The disease can cause kidney and liver failure and is also zoonotic.

SYMPTOMS: Fever, lethargy, inappetance, depression, PU/PD. Renal and hepatic disease.

DIAGNOSIS: PCR of blood/urine or acute and convalescent antibody titers.

TREATMENT: Antibiotics, aggressive fluid therapy and in hospital supportive care

VACCINATION: The vaccine effectively prevents significant disease and viral shedding against serovars included in the vaccine. The 4-way vaccine protects against 4 common strains and can be combined with DA2PP vaccine as DA2PP-L.

Borrelia burgdorferi

WHAT: Borrelia is the causative agent for **Lyme disease** and is a bacterium spread by **Ixodes ticks**.

SYMPTOMS: Most dogs (95%) **don't show any symptoms** of illness. Dogs that do show illness symptoms typically show signs of fever and joint swelling and joint pain. A very small subset of dogs develop a form of kidney disease that affects the glomerulus and causes protein loss into the urine. This is a swiftly moving acute form of kidney disease and can be fatal.

DIAGNOSIS: c6 Antibody test (in house snap, out-house c6 quantification)

TREATMENT: Symptoms tend to resolve within 48 hours of starting a month long treatment course of doxycycline.

VACCINATION: There are a few vaccines available for protection against Borrelia with variable efficacy. They can be recommended for those dogs with high risk of exposure to Ixodes ticks.

Tick control plays an essential role in prevention of the disease



Canine influenza (H3N2/H3N8)

WHAT: Influenza virus causes **upper respiratory disease** in dogs and possible pneumonia.

SYMPTOMS: cough, fever and possible nasal discharge.

DIAGNOSIS: PCR nasal and pharyngeal swabs

TREATMENT: Supportive care as needed; more severe cases may require hospitalization

VACCINATION: The vaccine is available as a **bivalent killed viral vaccine** that is recommended for dogs at risk of contracting the disease. Specifically dogs that are traveling or living in endemic areas or where it may be required by boarding facilities.



Feline Vaccines Schedule



Feline Core Vaccines

Vet Helpful Notes
Canine  Feline

FVRCP virus

Feline rhinotracheitis, calici, panleukopenia virus.

Contains:

- MLV Panleukopenia virus
- MLV Herpesvirus (rhinotracheitis)
- MLV Calici virus

Feline Rabies Virus

Recombinant Rabies

1 and 3 year vx (purevax)

Killed Rabies (adjuvanted)

1 and 3 year vaccines are available, but NOT recommended

Administration

3 doses are recommended between 8 to 16 weeks of age. Give every 3-4 weeks (q 2 weeks in shelters) until at least 16 weeks of age.

Ex: 8, 12, 16 weeks. If risk of exposure is high, then an additional final dose at 20 weeks of age

Single dose is usually administered at 12 or 16 weeks of age. State/Local law applies for this

Booster

Administer combination product no later than 1 year from last dose in series

Administer subsequent boosters every 3 years

Second dose scheduled no later than 1 year from first administration.

Then, every 1-3 years
State/local law applies

Feline Non-Core Vaccines

Recombinant Leukemia vx

Recombinant leukemia virus rFelv (adjuvant free)

Administration

AAFP recommends for all kittens:
Administer 1 dose as early as 8 weeks followed by a booster 3-4 weeks later

Booster

Administer 1 year later if there is sustained exposure risk (outdoors or potentially exposed to a foster/new cat)

Annually thereafter or off label use q 2-3 years if low risk

Chlamydia felis

Recc adjuvant free vaccine

Initial doses 2-4 weeks apart if indicated. However, indications for use of this vaccine are limited

Administer 1 year later then annually thereafter if sustained exposure risk

Feline Bordetella

Avirulent live intranasal vaccine

Single IN dose administered as early as 4 weeks of age if indicated. Used mostly in shelters. Indications for routine use of this vaccine are limited

Administer 1 year later then annually thereafter if exposure risk is present

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Feline Non-Core Diseases



Feline Leukemia

WHAT: Feline leukemia virus is a retrovirus that affects cats. Spread via saliva or nasal secretions usually via close casual contact: grooming, sharing food and water bowls etc. The infection can be passed onto kittens in utero. The virus enters the blood stream and lymphoid system and can either be overcome by the cat's immune system (abortive infection) or progress further by invading the rest of the body and entering the bone marrow where the cat will be persistently positive. Kittens are at higher risk for contracting the virus than adult cats with a mature immune system.

SYMPTOMS: Asymptomatic carriers can be healthy for years. Once symptomatic cats can become lethargic, anorexic and have a variety of disease symptoms including development of lymphoma.

DIAGNOSIS: Felv ELISA and IFA

TREATMENT: There is no treatment other than supportive care. Immune- modulating drugs, interferon omega can be tried.

VACCINATION: Recommended by the AAFP for all kittens as they have increased susceptibility. No vaccine is 100% protective.

Chlamydia felis

WHAT: Gram negative bacteria of cats that produces typically unilateral severe chemosis and conjunctivitis and ocular discharge which then progresses to bilateral signs. Most often affects cats under 9 months of age. Is spread by close contact with cats via ocular discharge.

SYMPTOMS: unilateral severe chemosis and conjunctivitis and ocular discharge which then progresses to bilateral signs

DIAGNOSIS: PCR on conjunctival and oropharyngeal swabs

TREATMENT: Doxycycline orally for 4 weeks. May use Clavamox as an alternative antibiotic.

VACCINATION: Recommended mostly in crowded shelter environments.

Feline Bordetella

WHAT: Aerobic gram negative bacteria of the respiratory tract. Transmission is cat to cat contact with infected nasal secretions.

SYMPTOMS: Causes upper respiratory tract signs and rarely pneumonia.

DIAGNOSIS: Isolation from oropharyngeal swabs or airway washes

TREATMENT: Doxycycline is the antibiotic of choice

VACCINATION: Since the infection generally causes only mild disease, routine vaccination is not recommended. Vaccines formulated for dogs should never be used in cats. Available as an intranasal vaccine, avirulent live culture.

Feline Core Diseases



Feline Rhinotrachetis

WHAT: Feline rhinotracheitis virus (FVR) is an infectious disease caused by feline herpesvirus type 1. The virus is shed in saliva and nasal and ocular secretions. It is considered a ubiquitous virus among cats and not all cats infected with the virus will get sick. Once infected, symptoms typically last about 10-20 days. All cats that are infected with the virus will become life-long carriers. The virus can be re-activated during periods of stress.

SYMPTOMS: Typical symptoms are: sneezing, nasal congestion and conjunctivitis. Some cats will develop eye ulcers from the virus attacking the cornea.

DIAGNOSIS: Typically based on history and physical exam findings. Can perform a PCR from conjunctival/oropharyngeal swabs

TREATMENT: Supportive care based on symptoms. Systemic or ocular antibiotics if indicated, topical or oral anti-viral therapy in some cases.

VACCINATION: Treatment is based on the severity of symptoms and can include antibiotics to treat secondary bacterial infections and anti-viral therapy. The vaccine cannot prevent infection with FHV-1 but can reduce the severity of the disease and reduce viral shedding.

Feline Calici Virus

WHAT: Feline Calici virus is the second most important respiratory virus of cats (feline herpes virus being the most important). The virus is highly contagious and infected cats shed the virus in saliva and nasal and ocular secretions.

SYMPTOMS: Symptoms include sneezing, nasal congestion, conjunctivitis and sometimes ulcers on the tongue, palate, lips or nose. Some strains of the virus can also cause a painful joints and limping will be observed. Symptoms are typically most severe in young kittens. Disease symptoms typically last for 2-3 weeks.

DIAGNOSIS: PCR from oropharyngeal or ocular swabs. May also be inferred based on clinical findings and history.

TREATMENT: Treatment is symptomatic support such as fluids, antibiotics if secondary bacterial infections are suspected and pain relief if needed.

VACCINATION: The vaccine cannot prevent infection but does reduce severity of the illness and shortens the length of illness.

Feline Panleukopenia virus

WHAT: Feline Panleukopenia virus is also known as Feline Parvovirus (FPV) or Feline Distemper. This virus is highly contagious and infected cats shed large amounts of virus in feces, vomit, urine, saliva and mucus. The virus can be transmitted on infected objects (fomites) such as bedding, food dishes etc. It can survive in the environment for up to 1 year! The virus can be killed with a dilute bleach solution used on surfaces.

Some kittens have been infected with the virus if they were in an area where a cat had once shed panleukopenia virus.

SYMPTOMS: Although many cats can be subclinical for this infection, cat under one year of age are at high risk and there is a high mortality rate in affected kittens. The virus attacks rapidly dividing cells in bone marrow, lymphoid tissue and the intestinal tract. Symptoms often include: anorexia, lethargy, vomiting, profuse watery to bloody diarrhea, bone marrow suppression (low white blood and platelet counts), and dehydration.

DIAGNOSIS: The virus can be detected using the canine snap Parvo test using feline feces.

TREATMENT: Treatment is aggressive supportive care with iv fluids, antibiotics etc. Survival is about 50% in cats receiving supportive care.

VACCINATION: Highly effective at preventing disease

Feline Rabies



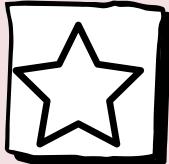
WHAT: Rabies is caused by rhabdovirus which is a zoonotic virus that affects the nervous system. It is most commonly transmitted through bite wounds. Rabies is endemic in skunks, bats, foxes, raccoons and coyotes.

SYMPTOMS: Excitation, agitation, drooling, aggression

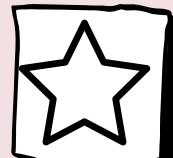
DIAGNOSIS: Diagnosis can only be made by submission of the brain for testing.

TREATMENT: There is no available treatment for rabies.

VACCINATION: The vaccine is available as an adjuvanted killed vaccines (no longer recommended based on vaccine site sarcomas) and non-adjuvanted recombinant vaccines. Rabies vaccination is required by law but the State and local laws dictate the frequency of administration (annually or every 3 years). Cats are the most commonly reported rabid domestic animal in the U.S.! The vaccine is highly effective at preventing illness.



Immunotherapy Basics



Types of vaccines

Vet Helpful Notes
Canine Feline

Killed viral vaccines

Killed vaccines may contain the entire organism or just a portion of the organism to evoke an immune response.

All canine rabies vaccines are killed/inactivated. The other killed viral vaccine is canine influenza vaccine. **All the injectable bacterial vaccines are killed** : injectable bordetella, lyme and leptospirosis.

Modified live

Modified live vaccines (also called attenuated vaccines) are vaccines that **contain live organisms** that have been altered so they cannot or are much less likely to cause disease. These live vaccines can reproduce in the animal and this causes a strong immune response. **Examples: modified live distemper, parvo vaccine, intranasal and oral bordetella vaccines.**

Recombinant

Recombinant vaccines are also called gene-deleted vaccines. They were developed to further safeguard against attenuated vaccines reverting to a form that can cause disease. The genes for a protective protein are removed from the disease causing organism and inserted into a vectored organism that won't cause disease. The vector replicates in the host animal and produces a high level of protective immunity to the disease causing organism. Examples: Recombinant feline rabies vaccine, recombinant lyme vaccine, recombinant canine distemper vaccines

Basis of Protective Immunity and vx schedules

Types of Immunity

Vaccines create **acquired immunity** through **humoral** and **cell mediated** responses. Most vaccines provide both types of responses. Some vaccines provide "**sterilizing immunity**" meaning there is virtually no chance of disease with vaccination: for example, rabies vaccines and feline panleukopenia vaccines. Not all vaccines can provide this type of robust protection. Vaccines may serve to provide a humoral and CMI local response to lessen the duration and severity of the disease. (for example feline herpes and calici viruses and bordetella vaccinations)

Humoral response

When an animal is vaccinated it creates antibodies directed at the pathogen. Antibodies adhere to the virus or bacteria for clearance by the immune system. This is how antibodies can neutralize viruses and bacteria that infect an animal.

Humoral immunity, however, takes about 1 month to develop appropriate antibody levels from vaccination.

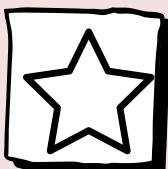
Cell-mediated response

Cell mediated immunity is the activation of phagocytes and cytotoxic t cells that act to destroy pathogens. It does not use antibodies. Cell mediated immunity is a more immediately developed after a mucosal vaccine like the intranasal bordetella vaccine. The intranasal bordetella vaccine might start working as quickly as 48-72 hours after a vaccine by activating the local immune response in the nasal cavity.

Vaccine schedules

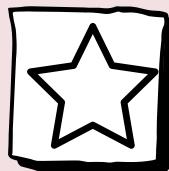
Whether an animal is vaccinated with an inactivated (killed) vaccine or a modified live/recombinant vaccine makes a difference in how long vaccines are protective. **Live and recombinant** vaccines are much more likely to create strong and **durable response**. **Killed vaccines** like leptospirosis and bordetella require **annual vaccination**. Modified live vaccines like canine distemper can last for many years. Of course, there still are some killed vaccines (like canine rabies) that do provide a strong immunity for several years.

Vaccines are given every **3-4 weeks** until **16- 20 weeks** to stimulate the immune system and stimulate immunity beyond when maternal antibodies wane. Vaccines are separated a minimum of 2 weeks apart (ideally 3-4) to give time for the immune system to respond and to avoid vaccine interference by providing a new vaccine too soon.



Over-due for vaccines

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During initial vaccine series puppy or kitten

Initial core vaccines at intervals of 3 to 4 weeks and not to exceed a 6 week interval inbetween any of these initial doses. Need at least 2 additional doses 3-4 weeks apart. Final puppy/kitten vx at or after 16 weeks of age!

During initial series of non-core vaccine

Same is due for initial 2-dose series for non-core vaccines: Need at least 2 additional doses 3-4 weeks apart.

CORE vaccine booster for dogs or Modified live-virus vaccine in cats

Administer a single dose of a combination core vaccine regardless of the number of years elapsed

Rabies vaccine booster

Varies by State, but most administer a single dose, after which the dog is considered immediately protected

Feline leukemia

Significant difference of opinion exists, but reasonable to recc re-start initial 2-dose series if more than 3 years over due

Canine Leptospirosis, Lyme or SQ Bordetella

Dogs that are within 2 years of a previous dose can receive a single dose (note some practitioners prefer 18 month interval for Lepto). Dogs exceeding this interval should re-start the 2-dose series

Canine intranasal or intraoral bordetella

Administer a single dose regardless of the number of years elapsed

Canine Influenza Virus Booster

Dogs within 2 years of a previous dose may receive a single dose. Exceeding a 3-year interval should -restart the 2-dose series

Vaccine Tips

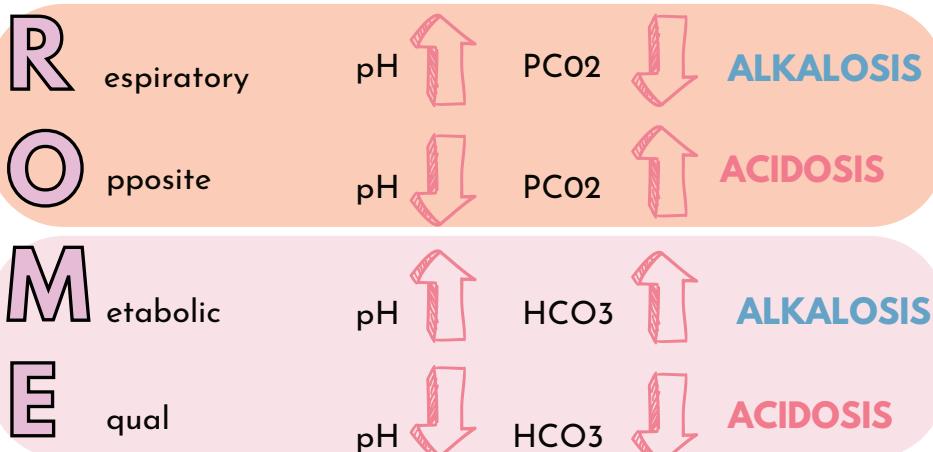
- There is **increasing risk of vaccine reactions** in small breed dogs (**under 20 lbs**) as you increase the number of vaccines. **Recommend to limit to 2-3 vaccines when possible**
- You only need 1/16th the dose of intra-nasal bordetella to induce immunity. It's okay if it always feels like most of the liquid is spraying OUT and not IN the nose ;)
- A vaccine dose is an **immunologic dose** required to stimulate the immune system. That is why the **same volume** is given to **small** dog and a **big** dog. Reducing the doses is not recommended.
- Dogs who have had a mild vaccine reaction (**Type 1 local anaphylaxis: facial angioedema or hives**) following a vaccine are generally protected by receiving a dose of **benadryl prior to vaccination** (2-4mg/kg orally or 1mg/kg parenterally).
- Dogs with **Type 1 systemic anaphylaxis** (shock) following vaccination should **not be re-vaccinated** ideally. Use of **titters** for canine distemper/parvo and feline panleukopenia is recommended instead.
- Patients with a history of **IMHA or ITP** regardless of cause should have **titters** instead of vaccination instead. Consider re-vaccinating young animals with insufficient titers. separate rabies from other vaccines.
- Most **common cause for failure to immunize** are patients who have vaccine interference from persistence of maternal derived antibodies longer than 12 weeks. This is why the **final pediatric vaccine should be at or after 16 weeks** of age. Improper handling of vaccines can lead to failure to immunize. Store vaccines at the proper temperature and give within 1 hour of reconstitution.

Arterial blood gases

normal pH
acidosis < 7.4 > alkalosis



pH	7.41	7.39	
PaCO ₂	37	31	Respiratory
HC03	22	18	Metabolic



Uncompensated
Partially Compensated
Compensated

CO₂ or HCO₃ are NORMAL
Nothing is NORMAL
pH is NORMAL

Metabolic Acidosis
Gain of acid: DKA, Uremia, EG tox, lactic acidosis
Bicarb loss: GI loss (d) renal loss

Metabolic Alkalosis:
Acid loss or Bicarb gain:
Almost always low Cl because Cl lost with H+: vomiting, pyloric obstruct, gastric suction, renal acid excretion (furosemide), iatrogenic

Resp Acidosis: Increased inspired CO₂ or decreased ventilation: breathing circuit dysfunction (exhausted soda lime), hypoventilation

Resp Alkalosis: increased ventilation: increased RR or tidal volume: hypotension, SIRS, PTE, pulm disease, excitement, exercise, pain



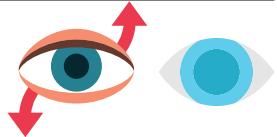
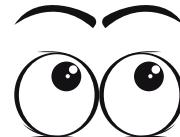
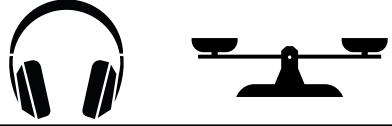
Carbonic Acid Equilibrium System

Hack :Lingual vein blood collection under anesthesia can approximate an arterial blood sample in dogs!

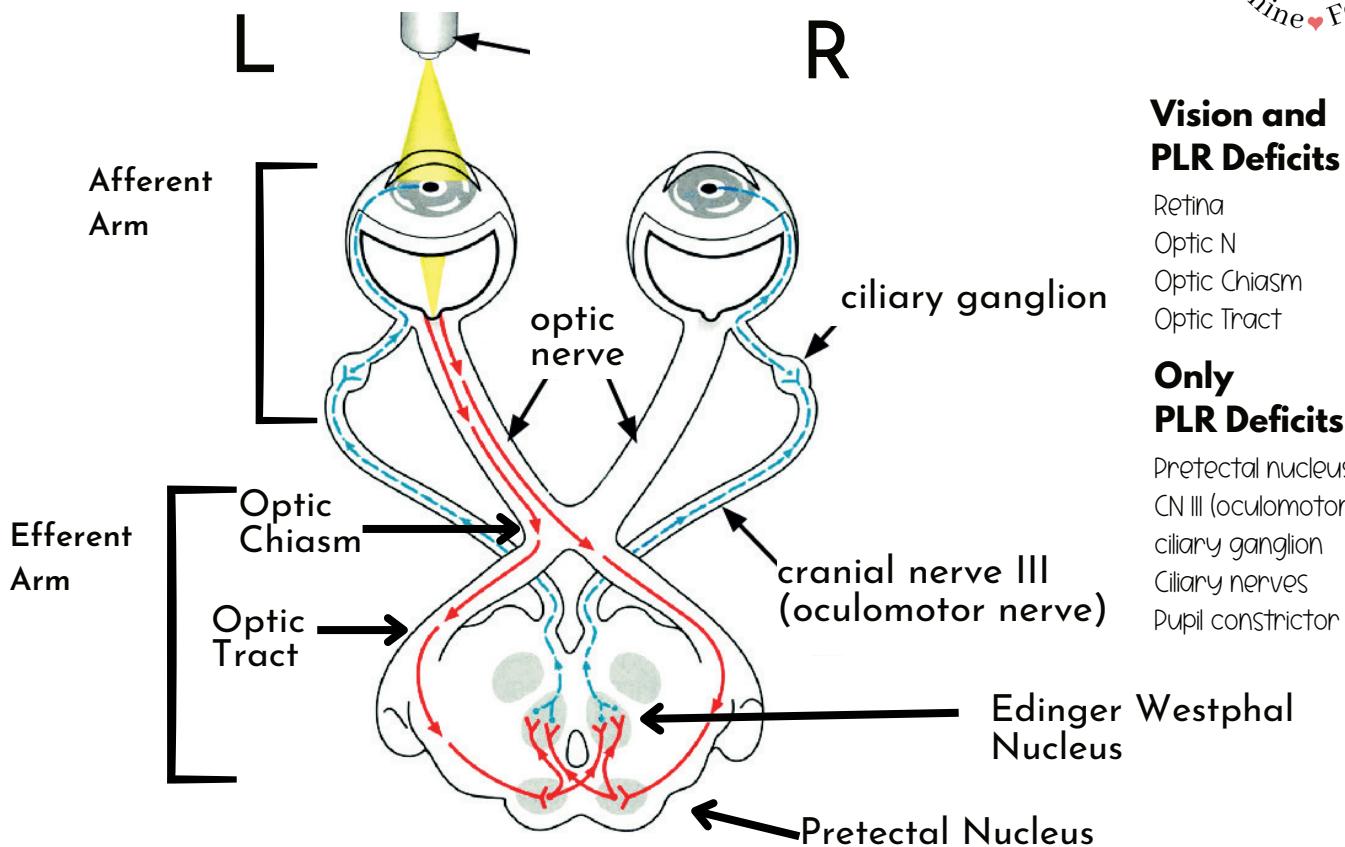
S = sensory
M = Motor
B = Both

CRANIAL NERVES

etsy.com/shop/vethelpfulnotes

SOME	I	Olfactory	smell		ON
SAY	II	Optic	acuity		OLD
MARRY	III	Oculomotor	eye movement pupil dilation		OLYMPUS
MONEY	IV	Trochlear	vertical eye movement		TOWERING
BUT	V	Trigeminal	facial expression facial sensation		TOP
MY	VI	Abducens	lateral eye movement		A
BROTHER	VII	Facial	facial movement taste		FIN
SAYS	VIII	Auditory vestibulocochlear	hearing balance		AND
BIG	IX	Glossopharyngeal	swallow		GERMAN
BRAINS	X	Vagus	gag reflex throat sensation		VIEWED
MATTER	XI	Spinal accessory	head/shoulder movement		SOME
MORE	XII	Hypoglossal	tongue movement		HOPS

PLR Interpretation



Pathway:

Light shines in the LEFT eye above. Impulse travels down the optic nerve to the chiasm. At the chiasm the impulses travel down BOTH optic tracts, synapse on the Pretectal and then Edinger-Wesphal nuclei in the mid-brain and back up both Oculomotor nerves to the ciliary ganglia. Short ciliary nerves synapse on the ciliary ganglion and innervate the pupil.

The Pupillary Light Reflex is a Sub Cortical Reflex, so you can be blind but have an intact PLR reflex! Vision requires the visual cortex (back of the brain). The PLR pathway only goes to the midbrain.

The PLR pathway has an Afferent and Efferent Arm.

Afferent arm= Retina, optic nerve

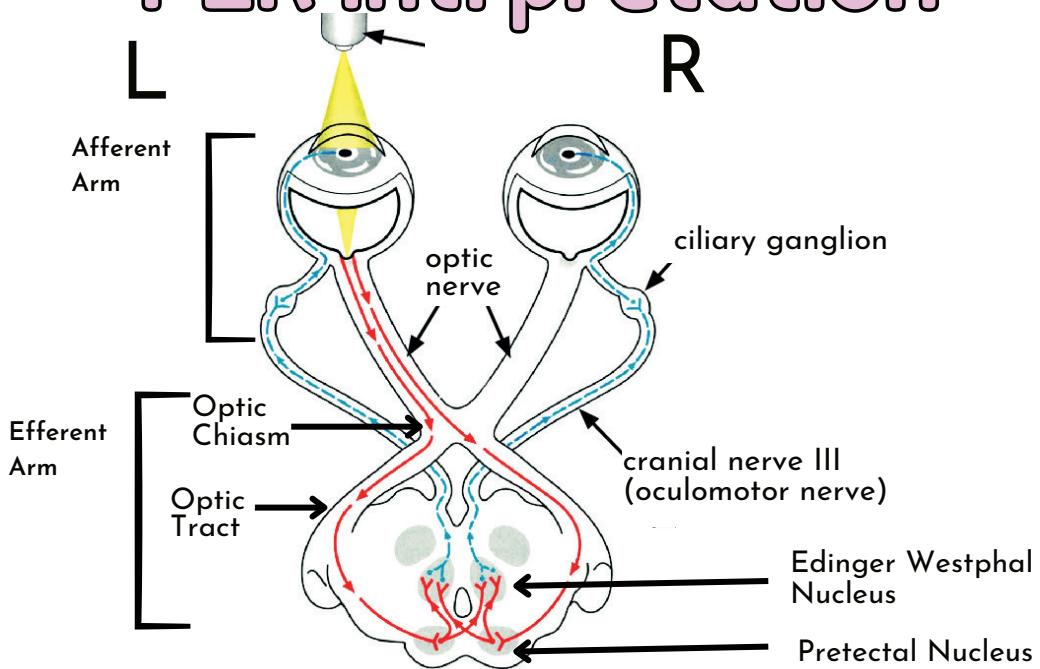
Efferent arm= Optic chiasm, optic tract, midbrain, CN III

Marcus Gunn:

A positive Marcus Gunn sign happens with a unilateral pre-chiasmal lesion (lesion in the optic nerve or retina). A light shone into a normal eye should produce constriction.

Test: Cover, uncover. Cover the affected eye in dimly lit room, then move to cover the other eye. When you move your hand, a normal eye should constrict in response to the light. If the eye DILATES, that indicates a pre-chiasmal lesion.

PLR Interpretation



EYE SIGNS WITH VARIOUS LESIONS IN THE PLR PATHWAY:

Left Optic Nerve Lesion: PUPILS SYMMETRIC at REST, BLIND in the Left eye

Light in the Left eye: No direct or consensual PLR present

1. PLR absent in the Left eye (Left eye does not constrict)
2. PLR absent in the Right eye (No consensual response)

Light in the Right eye: Direct and consensual PLRs are present

1. PLR present in the Right eye (non affected eye constricts)
2. PLR present in the Left eye (consensual response is present, affected eye can constrict)

Left Optic Tract Lesion: PUPILS SYMMETRIC at REST, BLIND in the Left eye

Light in the Left eye: Direct PLR is absent, but consensual PLR is present

1. PLR absent in the Left eye (Left eye does not constrict)
2. PLR present in the Right eye (consensual response is present)

Light in the Right eye: Direct PLR is present, consensual response in the affected eye is absent

1. PLR present in the Right eye (right pupil constricts)
2. PLR is absent in the affected Left eye (the consensual response is absent)

Left Oculomotor Nerve Lesion: PUPILS UNEVEN (Left eye dilated) at REST, VISUAL

Light in the Left eye: Direct PLR is absent, but consensual PLR is present

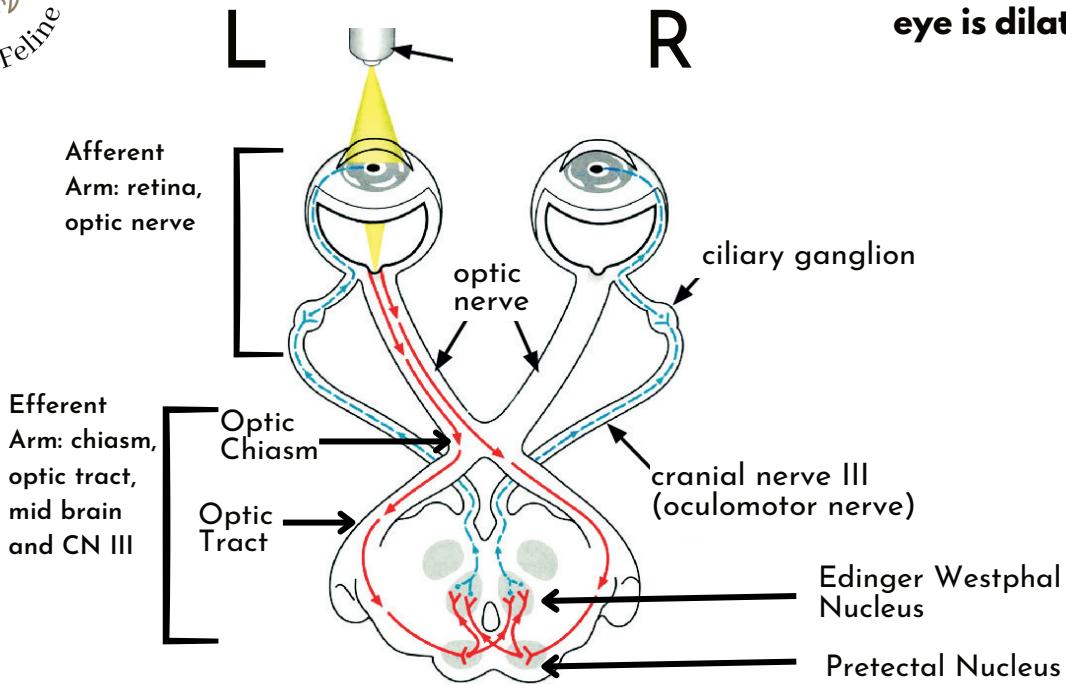
1. PLR absent in the Left eye (Left eye stays dilated)
2. PLR present in the Right eye (consensual response is present, pupil constricts)

Light in the Right eye: Direct PLR is present, consensual response in the affected eye is absent

1. PLR present in the Right eye (right pupil constricts)
2. PLR is absent in the affected Left eye (the consensual response is absent)

Anisocoria

Due to Parasympathetic Dysfunction (Abnormal eye is dilated)



Anisocoria is uneven pupil sizes. The problem can be a pupil that is abnormally small or a pupil that is abnormally dilated.

Parasympathetic Dysfunction: The parasympathetic nervous system is Rest and Digest. Pupils are normal in size. Anisocoria caused parasympathetic dysfunction will have an abnormally dilated pupil.

A light shone into the normal pupil will cause it to constrict. The larger, affected pupil will not constrict with light.

Damage to the oculomotor nerve (CN III) and/or its nucleus causes an abnormally dilated pupil. Because CN III also supplies innervation to the dorsal, medial and ventral oblique and levator muscles of the eye, the eye may exhibit ventrolateral strabismus and ptosis of the upper eyelid. Vision is present.

This defect is called ophthalmoplegia. Internal ophthalmoplegia can be due to a neoplasm of the cavernous sinus and has a poor prognosis. Often times other cranial nerves are affected (such as IV, V and VI). Other causes include: proptosis, retrobulbar dz, orbital or optic nerve neoplasm, or midbrain dz.

However, some patients have idiopathic oculomotor neuropathy which carries a good prognosis. These patients' symptoms do not deteriorate and can spontaneously improve.

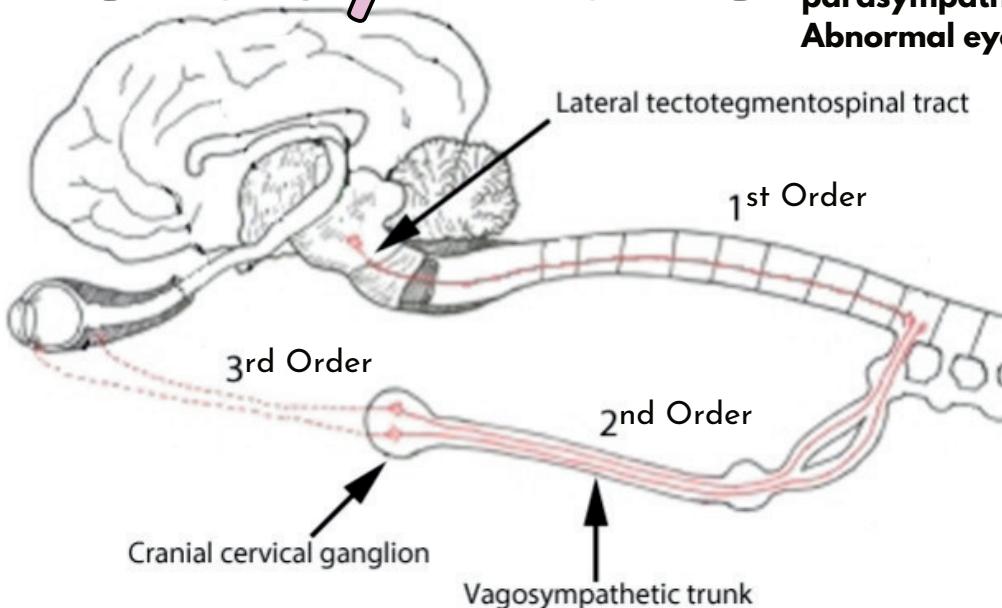
TESTING: 0.1% pilocarpine (diluted in artificial tears) applied to the affected eye will cause rapid constriction of the pupil. Pilocarpine is a parasympathetic alkaloid. In a normal eye there will be a slow, delayed constriction.

Other causes of a mydriatic eye can be posterior synechia (the iris is stuck to the cornea) or iris atrophy. Pilocarpine should not cause constriction or not cause even constriction in these cases.

Horner's Syndrome

Anisocoria due to parasympathetic dysfunction.
Abnormal eye is miotic.

Vet Helpful Notes
Canine Feline



Anisocoria is uneven pupil sizes. The problem can be a pupil that is abnormally small one one side or a pupil that is abnormally dilated on one side.

Sympathetic Dysfunction: Sympathetic innervation to the eye causes pupil dilation (flight/fight response). Horner's causes Anisocoria characterized by an abnormally constricted (miotic) pupil that won't dilate.

A light shone into the larger (normal) pupil will induce constriction. The miotic (abnormal pupil) will not be able to dilate in darkness.

The sympathetic innervation to the eye is composed of a 3 neuron pathway:

1. It starts in the hypothalamus of the brain and the first order neuron courses down the cervical spine and exits the spinal cord at spinal segments T1-T3.
2. The 2nd order neuron courses cranially through the chest cavity and out the thoracic inlet (along the jugular groove) to the cranial cervical ganglion.
3. The 3rd order neuron runs through the middle ear and ends at the periorbital muscles, third eyelid and iris muscle.

Dysfunction anywhere along this pathway leads to Horner's syndrome. A diagnosis of Horner's requires that 3 of 4 features are noted: ptosis (droopy eye from decreased tone to the eyelid muscles), miosis (constricted pupil), enophthalmos (sunken eye from weak periorbital muscles) and third eyelid elevation.

Pharmacologic Testing with 10% Phenylephrine applied to the eye can help determine which part of the pathway is affected:

3rd Order Post ganglionic lesion: signs resolve in <20 minutes (causes: otitis media, otic polyps, deep ear flushes, post-op from bulla osteotomy surgery, orbital diseases)

2nd Order Pre ganglionic lesion: signs resolve in 20-45 minutes (causes: idiopathic, brachial plexus avulsion, nerve sheath tumor, lymphoma, thoracic neoplasia(such as a mediastinal mass), traumatic jugular venipuncture, forceful choke chain use)

1st Order Upper Motor Neuron lesion: signs resolve in >45minutes (causes: neoplasia, encephalitis, myelitis, IVDD, FCE, Trauma)

NOTABLE: approximately 50% of dogs and cat with Horner's are diagnosed with idiopathic disease (no known cause). In dogs, Golden Retrievers are slightly predisposed to this disease. No treatment is needed for these patients and many spontaneously recover within 4-8 weeks. In cats, recovery can take 16 weeks or more.

1-4% of cases are due to otitis media. Miosis often persists even after otitis management.

CBC, biochemical profile (increased incidence in patients with diabetes and other metabolic diseases), otic examination, chest and neck imaging (MRI, x-rays) may all be helpful in diagnosis.

Common Cranial Nerve Disorders

II

Cranial Nerve II: Optic nerve disease

Pupils symmetric at rest but PLR (direct and consensual) and absent in affected eye.

III

Cranial Nerve III : Oculomotor nerve disease

Ophthalmoplegia. Mydriatic pupil, ventrolateral strabismus and ptosis are all commonly present. With just CN III (idiopathic neuritis in dogs) mydriasis is the only sign

V

Cranial Nerve V: Trigeminal Neuropathy "Dropped Jaw". A dog presenting with drooling and inability to close the mouth has presumptive bilateral trigeminal neuropathy that is affecting all the motor branches of the trigeminal nerve that supply the jaw. This condition is most commonly idiopathic and these dogs often spontaneously recover within a few weeks. Cancers such as lymphoma or multiple myeloma can cause this condition. Infectious causes can include polyradiculoneuritis, and encephalomyelitis and uncommonly rabies. Head trauma is also a possible cause.

Cranial Nerve V: Bilaterally weak blink reflex (palpebral reflex), one that easily fatigues, is a common symptom in Myasthenia Gravis.

Cranial Nerve V: Unilateral Trigeminal nerve disease. Unilateral atrophy of the muscles of mastication: masseter, temporal, pterygoid muscles. Typically will see a unilaterally decreased palpebral reflex, decreased nasal sensation, lack of menace, and enophthalmus. A common cause is a trigeminal nerve sheath tumor.

VII

Cranial Nerve VII : Facial nerve paralysis. Causes an absent menace response, lip droop, ear droop and hypersalivation on the affected side. Decreased blink reflex may cause KCS and corneal ulceration.

The most common cause in dogs is idiopathic facial nerve paralysis, and cocker spaniels are overrepresented. In cats, the most common cause is ear/throat polyps or deep ear infection.

The second most common cause in dogs is otitis media/interna. Hypothyroidism can also cause facial nerve paralysis.

Cranial Nerve VII: Vestibulocochlear nerve dysfunction. The vestibulocochlear nerve and its receptors are located in the inner ear. Peripheral vestibular disease affects the vestibular receptors, ganglion or nerve. The top two causes are ear disease and idiopathic vestibular syndrome. The latter is benign and resolves spontaneously within a few weeks. The cause is unknown. Clinical signs are loss of balance, leaning/falling, ataxia, positional nystagmus/strabismus, and head tilt.

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IVERMECTIN BASED HEARTWORM PRODUCTS



HEARTGARD PLUS	Ivermectin Pyrantel	HEARTWORM	ROUNDS HOOKS
TRIHEART PLUS	Ivermectin Pyrantel	HEARTWORM	ROUNDS HOOKS
IVERHART PLUS	Ivermectin Pyrantel	HEARTWORM	ROUNDS HOOKS
IVERHART MAX	Ivermectin Pyrantel Praziquantal	HEARTWORM	ROUNDS HOOKS TAPES

Ivermectin products have no whipworm protection

Slow Microfilaricidal drug

MILBEMYCIN BASED HEARTWORM PRODUCTS



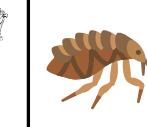
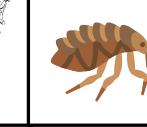
INTERCEPTOR PLUS	milbemycin praziquantal	HEARTWORM	ROUNDS HOOKS WHIPS TAPES	Prevents flea eggs from developing
SENTINEL	milbemycin lufenuron	HEARTWORM	ROUNDS HOOKS WHIPS	
SENTINEL SPECTRUM	milbemycin lufenuron praziquantal	HEARTWORM	ROUNDS HOOKS WHIPS TAPES	
TRIFEXIS	milbemycin spinosad	HEARTWORM	ROUNDS HOOKS WHIPS	ADULT FLEA



Milbemycin is most risky for causing anaphylaxis with it's FAST Microfilaricidal action

Vet Helpful Notes
Canine Feline

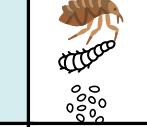
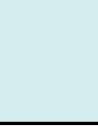
MOXIDECTIN BASED HEARTWORM PRODUCTS

SIMPARICA TRIO  	moxidectin sarolaner pyrantel	 HEARTWORM	 rounds hooks			
ADVANTAGE MULTI   	moxidectin imidacloprid	 HEARTWORM	 rounds hooks whips			 skin and ear mites
PROHEART  	moxidectin	 HEARTWORM	SLOW MICROFILARIACIDAL DRUG			

VetHelpfulNotes
Canine Feline

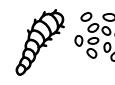
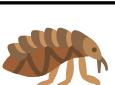
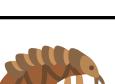
SELAMECTIN BASED HEARTWORM PRODUCTS

SLOW
MICROFILARIACIDAL DRUG

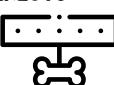
REVOLUTION   	selamectin	 HEARTWORM	 ROUNDS HOOKS			 ear and skin mites
REVOLUTION PLUS  	selamectin sarolaner	 HEARTWORM	 ROUNDS HOOKS WHIPS			 ear and skin mites

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FLEA ONLY PRODUCTS

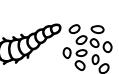
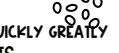
IMIDACLOPRID	neonicotinoid blocks Acetylcholine receptors in CNS	ADVANTAGE	
LUFENURON	insect growth regulator inhibits chitin synthesis	PROGRAM	
METHOPRENE	insect growth regulator juvenile hormone analog		 
DINOTEFURAN	neonicotinoid mimicks effects of acetylcholine and overstimulates cns- contact drug		
NITENPYRAM	neonicotinoid blocks acetylcholine receptors and overstimulates cns	CAPSTAR	
SPINOSAD	similar effects as neonicotinoids affects cns	COMFORTIS	 Kills fleas so fast effectively stops egg production
INDOXACARB	contact insecticide; inhibitor of acetylcholinesterase	ACTIVYL	 

COMBINATION FLEA PRODUCTS

ADVANTAGE II 	imidacloprid pryiproxyfen	  adult and larval fleas	
K9 ADVANTIX  	imidacloprid permethrin +pryiproxyfen	  adult (and larvae/eggs with Adv II)	 Repels and kills
ADVANTAGE MULTI 	imidacloprid moxidectin	  rounds hooks whips	 scabies ear mites
SERESTO 	imidacloprid flumethrin	  adult and larval fleas	 demodex
SENTINEL 	lufenuron milbemycin	  rounds hooks whips	 Repels and kills
SENTINEL SPECTRUM 	lufenuron milbemycin praziquantal	  rounds hooks whips tapes	 scabies

Vet Helpful Notes
Canine  Feline 

COMBINATION FLEA PRODUCTS CONT'D

FRONTLINE AND PARASTAR	fipronil			
FRONTLINE PLUS	fipronil methoprene	 		
FRONTLINE GOLD	fipronil methoprene pyriproxyfen	 		
VECTRA	dinotefuran pyriproxyfen	 		
VECTRA 3D	dinotefuran pyriproxyfen permethrin	 		
TRIFEXIS	spinosad milbemycin	  ROUNDS HOOKS WHIPS		
BRAVECTO- FURALANER CREDILIO- LOTILANER SIMPARICA- SAROLANER	isoxazolines	  KILLS ADULTS SO QUICKLY GREATLY REDUCES EGG COUNTS		bravecto- q 3 months top spot or chew, dogs/cats
SIMPARICA TRIO	sarolaner moxidectin pyrantel	  KILLS ADULTS SO QUICKLY GREATLY REDUCES EGG COUNTS		credilio- q monthly chew dogs/cats simparica- dog only:monthly chew simparica Trio has moxidectin and pyrantel added for HWP and deworming- only for dogs
ACTIVYL TICK PLUS	indoxacarb permethrin	 		However, Revolution plus for cats is similar- has sarolaner and selamectin
REVOLUTION	selamectin	  ROUNDS HOOKS		 EAR AND SKIN MITES
REVOLUTION PLUS	selamectin sarolaner	  ROUNDS HOOKS		 EAR AND SKIN MITES

Permethrins are toxic to cats due to insufficient glucuronide conjugation of the permethrin. causes seizures, tremors and can cause death.

Absorbed through the skin. Found in canine products such as: ADVantix, Activyl Tick plus, and Vectra 3D

HEARTWORM OR COMBINATION FLEA PRODUCT INFO

MDR-1 mutation:
found most often in collies
and Aussies

ISOXAZOLINES	Affect glutamate gaba channels to cause hyperexcitability of CNS flea/tick	bravectofuralaner credilis- lotilaner simparica-sarolaner			Causes a dysfunctional p-glycoprotein so more toxins can build up intracellularly in the brain
PYRIPROXYFEN	Inhibits molt from juvenile to adult: igr. similar to methoprene but affects ticks too	combo in advantage II and advantix II			They are more sensitive to certain drugs like ivermectin
IVERMECTIN	AFFECTS CNS TO PARALYZE AND KILLS MITES, BOTS, INTESTINAL PARASITES, MICROFILARIAE AND LARVAE OF HEARTWORM IVERMECTIN CLASS	combo product in heartgard, triheart and iverhart			however ivermectin found in HW preventatives are at low enough doses to not be problem in these sensitive breeds
MILBEMECIN	binds chloride channels to cause cns paralysis, kills microfilariae, intestinal parasites macrocyclic lactone	combo product inInterceptor, sentinel, trifexis			
SELAMECTIN	binds chloride channels to cause cns paralysis, kills microfilariae, fleas, intestinal parasites, mites: avermectin	revolution			 rounds hooks
MOXIDECTIN	binds chloride channels to cause cns paralysis, kills microfilariae.. Macrocyclic lactone	proheart or in combos to increase action Advantage Multi			 ear and skin mites
					 rounds hooks whips
					 scabies, demodex ear mites

etsy.com/shop/VetHelpfulNotes

TICK ONLY PRODUCT

AMITRAZ	Interacts with octopamine receptors in the insect nervous system to cause hyperexcitability	preventic collar	
---------	---	------------------	--

Most tick products take 24-48 hours to kill ticks.

If you are getting frequent tick exposures, then consider adding in a tick repellent collar (preventick, seresto)

or

switching to a topspot like advantix or vectra 3d with repellent effects



TICK REPELLANT PRODUCTS

AMITRAZ
FLUMETHRIN
PYRIPROXYFEN

amitraz: Preventick collar
flumethrin: Seresto collar
pyriproxyfen: Advantix, Vectra 3d

Anti-inflammatory Drugs

Vet Helpful Notes
Canine Feline

Steroids
and
Non Steroidal Anti-inflammatory Drugs

NSAIDs

- Carprofen** (Rimadyl, Vetprofen)
- Deracoxib** (Deramaxx)
- Meloxicam** (Metacam)
- Robenacoxib** (Onsior)
- Aspirin** (Bufferin, Ascripten)
- Gabaprant** (Galliprant)

Action

NSAIDs produce anti-inflammatory and analgesic effects by inhibiting the synthesis of prostaglandins via the COX enzymes.

COX-2 selective NSAIDs help keep the protective effects of prostaglandins for GI, Kidney and Platelet functions.

Injury!



Corticosteroids

Arachadonic Acid



Aspirin
Irreversibly
binds

COX-1

PG_{1,2} TXA₂



Gastric
Protection



Kidney
Blood
Flow
&
Platelet
Function



Gabaprant



Pain



PGE₂

Fever



Carprofen
Deracoxib
Meloxicam
Robenacoxib

Aspirin- mild

COX-2

Aspirin- mild

PG_{1,2} TXA₂



Gabaprant

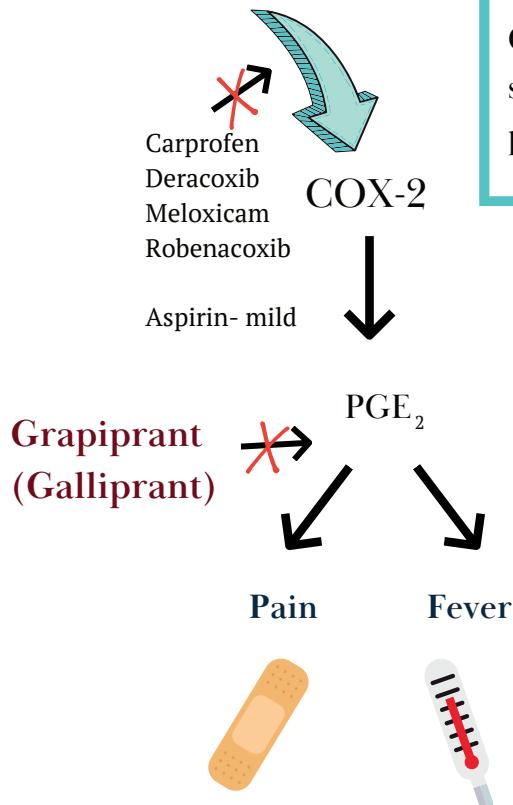


Pain



Never administer NSAIDs
and Steroids together!!!

Arachadonic Acid



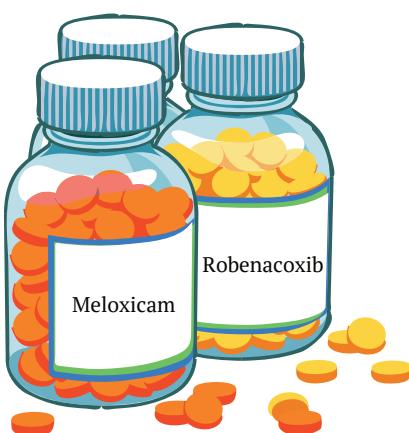
Grapiprant (Galliprant) works further down in the COX cascade so the hope is that it may be more sparing of the prostaglandins that protect the GI tract and Kidneys

Newer NSAIDS since aspirin are designed to be more COX-2 selective.

Aspirin is an "original" NSAID and is not COX-2 selective. At low doses, aspirin is more COX-1 specific. That is why low dose aspirin is used for its anti-platelet effects.

Clopidogrel, however, produces superior platelet inhibition than aspirin. It works in a different pathway

Most commonly used feline NSAIDS



Meloxicam (Metacam)
FDA approved for one-day use in cats

Robenacoxib (Onsior)
FDA approved for 3 days in cats

*Caution
CATS!!!*

- Kidney injury is observed in cats with higher doses or repeated doses of NSAIDS
- Cats have slow clearance of NSAIDS due to deficiency of the enzyme Glucuronyl Transferase

NSAIDS/Platelet Aggregate Inhibitors

etsy.com/shop/VetHelpfulNotes

Practice Pearl: When switching between different NSAIDS or between an NSAID or a steroid always wait 72 hours between medications to avoid gastrointestinal ulceration

Carprofen (Rimadyl, Vetprofen) **Dosing:** 2.2mg/kg po bid DOGS ONLY

Contraindicated in dogs with bleeding disorders (Von Willebrands), hx of serious rxns to any NSAID

Caution : geriatric patients or pre-existing chronic GI or renal diseases

GI side effects adverse effect less likely than with older NSAIDS

Rarely can cause hepatic failure

Deracoxib (Deramaxx)

Dosing: Dogs 1-2 mg/kg po SID post-op pain dose 3-4mg/kg po SID

May be useful alternative to TCC bladder carcinoma, at lower doses most Cox 2 selective;
Possible GI and renal effects

Meloxicam (Metacam):

Dosing: Dogs 0.2mg/kg po iv, sc and then 0.1mg/kg po sid
Cats 0.3mg/kg as single doses SQ no add'l doses

Cox-2 preferential; adverse effects GI and Renal effects

Robenacoxib (Onsior)

Dosing: Dogs 1-2mg/kg po sid, Cats 1 mg/kg po sid

Cox-2 specific, mostly GI side effects

Aspirin (Bufferin, Ascripten)

Dosing: Dogs 10mg/kg - 25mg/kg po bid as
anti-inflammatory; Lower doses for IMHA
Cats 1 baby aspirin q 72 hours

Contraindicated in patients w/ GI bleeds or bleeding disorders asthma, renal disease (occ used with glomerular disease); has very long half life in cats (20 hours) so dose carefully; dog sensitive to GI side effects

Grapiprant (Galliprant):

Dosing: 2 mg/kg po q 24

Prostaglandin E2 EP4 receptor antagonist. New class of NSAID for tx of OA in dogs;
Contraindicated in dogs on corticosteroids

Monitor vomiting, appetite, diarrhea

Clopidogrel (Plavix)

Dosing: Dogs: 1.1-4mg/kg po SID, Cats 1/4th tab SID

Platelet aggregate inhibitor; used to prevent thromboembolic disease in cats and hypercoagulable states in dogs. Potential to cause vomiting or bleeding. Works by inhibiting activation of platelet glycoprotein necessary for the platelet fibrinogen binding and inhibition of compounds from platelets that enhance aggregation.

Plavix and Aspirin are platelet aggregate inhibitors and are best used to treat thromboembolic disease (not as an anti-inflammatory). Aspirin works on the COX pathway but is not very selective for COX-2 like the later generations of NSAIDs. It is more likely to cause GI ulcers or renal damage than other NSAIDS in pets.



Corticosteroids



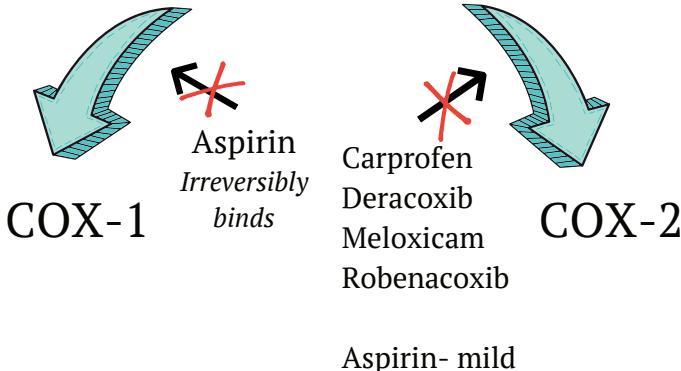
Vet Helpful Notes
Canine  Feline 

Injury!



Corticosteroids

Arachadonic Acid



Steroids (aka corticosteroids) act higher up in the COX pathway than NSAIDS do.

Prednisone is the most well known corticosteroid. Prednisone is converted to prednisolone in the liver.



PEARL: Cats do not efficiently convert prednisone to prednisolone, so they should be prescribed prednisolone.

CAUTION: active infection, Cushing's syndrome, diabetes, congestive heart failure and kidney disease.

PEARL: sustained use can lead to iatrogenic Cushing's disease

Weak potency	Intermediate potency	Strong potency
Hydrocortisone	Prednisone 4X	Betamethasone 25X
	Prednisolone 4X	Dexamethasone 30X
	Methylprednisolone 5X	
	Triamcinolone 5X	
Short duration	Intermediate duration	Long duration
Dexamethasone 8P (~ 12 hours)	Triamcinolone (10-14 days)	Depo Medrol (4-6 weeks)

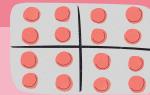
Hydrocortisone is the weakest steroid. All other forms are compared to hydrocortisone.

Dexamethasone is 30 times more potent than hydrocortisone. Prednisone and Prednisolone are 4 times as potent

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GI MEDICATIONS

VetHelpfulNotes
Canine • Feline



Antacids

H2 Blockers

Examples: Famotidine (**Pepcid**), Ranitidine (**Zantac**). All available OTC. Oral tablets and injectable.

ACTION: Block the H2 receptor on the parietal cell in the stomach to decrease acid secretion by this proton pump.

USE: Used when there is increased risk of GI ulceration such as: esophagitis, gastroesophageal reflux disease. There is controversy of opinion regarding their need in diseases such as CKD, Liver disease, pancreatitis.

RISKS: Rapid IV use of famotidine can cause bradycardia. Consider dose-reducing famotidine with renal disease. Dilute ranitidine for IV use

DOSE: 1mg/kg PO q12 hr famotidine Dogs/Cats; Ranitidine 1-2mg/kg IV or PO Dogs, 2.5mg/kg slow IV BID or 3.5mg PO BID cats

NOTABLE: Evidence suggests that Acid inhibition by H2 blockers in small animals is not as robust as Proton pump inhibitors and their use is falling out of favor.

Proton Pump Inhibitors

Examples: Omeprazole (**Prilosec**) : ORAL
Pantoprazole (**Protonix**) : INJECTABLE

ACTION: Inhibit acid secretion by the parietal cell by blocking the Proton Pump.



USE: To increase stomach pH to treat and prevent gastric ulcers

RISKS: Caution hepatic, renal disease

DOSE: Omeprazole Dogs 1mg/kg PO q 12, Cats 1-1.3 mg/kg PO q 12 hr; Protonix: 1mg/kg IV over 15 min q 12 hr

NOTABLE: Works best if given before the first meal of the day; studies have shown PPI's to be superior to reduce gastric acid than H2 blockers

Gastroprotectants

Sucralfate

Brand name: **Carafate**; comes as tablets or liquid

ACTION: A sulfated disaccharide gel that binds to ulcer craters to protect them from gastric acid. The suspension form can also be useful to treat esophagitis. Works best in an acid environment.

USE: Gastritis, Esophagitis. Comes as tablets and liquid.

RISKS: Constipation is possible

DOSE: Dogs and cats: 1/4th-1/2 of a 1 gm tablet for small dogs/cats and 1 g PO 2-4 times a day for large dogs

NOTABLE: Dissolve tablets in water before administration; can bind to other drugs, so other medications should be given 1-2 hours in advance of sucralfate. No evidence in humans to support its use is improved with an H2 blocker.

GI MEDICATIONS

Gastroprotectants



Misoprostol

Brand name: **Cytotec** ; available as a tablet

ACTION: Synthetic prostaglandin that helps prevent and heal gastric ulcers by improving mucosal blood flow and increasing gastric mucus production and bicarb production to protect the stomach lining.

USE: Major use is for helping to prevent GI ulceration from long-term NSAID use in dogs with arthritis and to heal gastritis and gastric ulceration.

RISKS: Diarrhea and possible abortion risk. Wear gloves for administration.

DOSE: Suggested dose is 3 ug/kg BID

NOTABLE: Hazardous drug, handle with gloves always

Anti-emetics



Cerenia

Brand name: **Maropitant**. Available as oral tab and injectable*

ACTION: Neurokinin receptor antagonist that blocks the action of substance P in the CNS. Metabolized by p450 enzymes. Considered a very safe drug and has improved efficacy over metoclopramide for reduction in vomiting.

USE: Anti-emetic, Anti-nausea



RISKS: Caution with hepatic disease; rare allergic reactions

DOSE: 1mg/kg SQ q day, 8mg/kg orally for motion sickness; acute vomiting. Oral dose in dogs: 2mg/kg/day and 1mg/kg/day in cats.

NOTABLE: injection can sting, so some recommend refrigerating the injection and diluting with saline. PO dose is higher because of decreased oral bioavailability

Metoclopramide

Brand name: **Reglan**. Available as oral tabs and as an injectable product.

ACTION: Has both central and peripheral nervous system effects. Centrally it acts in the brain to block the chemoreceptor trigger zone (CRTZ) by blocking dopamine action in the brain. This is mainly effective for dogs, not cats. Peripherally, it augments acetylcholine release to increase the strength of gastric contractions and the strength of the Gastroesophageal sphincter. This makes it useful for improving gastric motility and Gastric emptying.

USE: To stimulate GI motility, anti-emetic.

RISKS: Contraindicated in obstruction, seizure disorders, pheochromocytoma

DOSE: Often used in the hospital setting at a CRI 1-2mg/kg/day. Orally the dose is 0.25-0.5mg/kg po BID.

NOTABLE: Not considered very effective in cats as anti-emetic because cats don't have dopamine receptors in the brain.

GI MEDICATIONS

Anti-emetics



Ondansetron

Brand name: **Zofran**. Available as tablets and as an injectable.

ACTION: Serotonergic receptor (5-HT3) antagonist found in the CRTZ of the brain and peripherally on vagal nerve terminals.

USE: Anti-emetic

RISKS: Can mask ileus; caution with hepatic disease.

DOSE: Acute vomiting: 0.5-1mg/kg PO or IV q 8-12 Dogs, and 0.1-1mg/kg PO cats and 0.025-0.2mg/kg IV q 6-12 hours

NOTABLE: May be chosen over cerenia because of costs

Emetics

Apomorphine Ropinirole

Brand name: **Apokyn, Emedog, Clevor** Available as a tablet or injectable or eye drops

ACTION: Rapid acting, centrally mediated emetic used in dogs, but not considered effective in cats. Stimulates dopamine receptors in the CRTZ to induce vomiting. Best to use within 2 hours of ingestion of foreign material or toxin.

USE: Used to induce emesis in dogs following ingestion of foreign material or toxic matter

RISKS: Contraindicated in patients with cardiovascular shock or lack pharyngeal reflexes. Contraindicated in patients that have ingested a sharp object, strong acid or base or other caustic agent that could injure the esophagus. There may be prolonged effects in patients with hepatic disease.



DOSE: Dogs: 0.01-0.04 mg/kg IV, SQ or IM or 0.25mg/kg PO or placing a 6.25mg tablet in the conjunctival sac. Eye drops: # of drops based on weight

NOTABLE: Does not work in cats. Works well in dogs- about 90-100% of dogs will vomit.

Alpha 2 Adrenergic Agonist

Dexmedetomidine (**Dexdomitor**), Xylazine (**Rompun**)

ACTION: Used off-label in cats as an emetic.

USE: Used to promote emesis in cats; Apomorphine is ineffective in cats due to its action at dopamine receptors

RISKS: Cardiac disease, respiratory disorder, shock, liver/kidney disease. Adverse effects: bradycardia, AV block, decreased respiration, prolonged sedation.

DOSE: **Dexmed:** Cats: 6-18 ug/kg IM or 3.5 ug/kg IV; **Xylazine:** 0.44mg/kg IM

NOTABLE: Dexmedetomidine is more specific than xylazine for alpha 2 receptors than alpha 1 receptors. Alpha 1 receptors are found on vascular smooth muscle, Alpha 2 receptors are present in the brain

GI MEDICATIONS



Appetite Stimulants



Mirtazapine

Brand name: **Mirataz, Remeron**; available as oral tablet and as a transdermal gel

ACTION: 5-HT3 receptor antagonist, Tricyclic antidepressant. Antagonism of 5-HT3 receptors accounts for the anti-nausea and anti-emetic effects of the drug.

USE: Effective as an appetite stimulant and anti-emetic in cats.

RISKS: Contraindicated in pets on MAOI in the past 14 days (eg Selegiline). May cause sedation. Altered liver enzymes. Give q 48 hours in most cats is appropriate/effective. Some advocate daily use in cats with normal renal function.

DOSE: 1.5 inch ribbon of transdermal to inner ear q 24 hours or 1/4th of a 7.5mg tablet PO q 48 hours

NOTABLE: Can be used in conjunction with other anti-emetics; extend dosing interval in cats with liver dz. There is little data on pharmacokinetics or efficacy in dogs

Capromorelin

Brand name: **Entyce and Elura for cats**; comes as an oral solution for dogs and cats

ACTION: Ghrelin-receptor agonist that stimulates growth hormone release and causes feelings of hunger.

USE: Mainly an appetite stimulant for dogs. Can be used for cats as well.

RISKS: Acromegaly in cats, hepatic dysfunction, renal disease

DOSE: 3mg/kg PO once daily in dogs; 2 mg/kg PO daily for cats
Adverse effects: vomiting, diarrhea, inappetence, lethargy

NOTEABLE: Comes only as a liquid, no tablet form.

GI Antibiotics



Metronidazole

Brand name: **Flagyl**; comes as tablets and injectable

ACTION: Concentration dependent bactericidal agent for anaerobes;

USE: Anti-diarrheal antibiotic

RISKS: Overdoses can be neurotoxic and cause vestibular signs (Typically over 40mg/kg doses)

DOSE: 10-20 mg/kg PO BID or IV. Very low concentration in IV form: 5mg/ml

NOTEABLE: Has anti-inflammatory effects at the colon

Tylosin

Brand name: **Tylan**; comes as scoopable powder; Macrolide antibiotic

ACTION: Anti-diarrheal macrolide antibiotic with probiotic, anti-inflammatory and immunomodulatory effects

USE: used primarily for chronic diarrhea in dogs/ cats

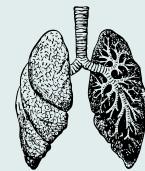
RISKS: Few listed adverse effects in dogs/cats

DOSE: Dogs and cats 20-25mg/kg PO q day

NOTEABLE: Can taste bitter as powder; can be compounded into capsules

Respiratory Medicine

Pharmacology



BRONCHODILATORS

*** Drugs ending in -terol and -ine***



Terbutaline (Brethine)

Indications: Dilate lower airways in respiratory conditions like asthma

Mechanism of action: Beta agonist that is inhaled. Stimulates Beta 2 receptors to relax bronchiolar smooth muscle and open the airways.

To Note: Excessive Beta-adrenergic stimulation may cause tachycardia by stimulating the beta receptors on the heart

CORTICOSTEROIDS

*** Drugs ending in -one***

Fluticasone (Flovent)

Indications: To reduce excessive coughing by asthma

Mechanism of action: Inhaled corticosteroid with a potency 18 times that of hydrocortisone. Has potent anti-inflammatory effects on bronchial mucosa.

To note: Fewer systemic side effects of corticosteroids such as increased thirst, urination, appetite etc. Most of the use has been established for cats with asthma, but can also be used in dogs.

Prednisolone/Prednisone (various)

Indications: Anti-inflammatory agent to reduce airway inflammation.

Mechanism of action: Glucocorticoid (oral) that is 4 times more potent than hydrocortisone. Has a variety of effects but main benefit for respiratory disease is to reduce airway inflammatory mediators.

To note: Cats must use prednisolone as their system does not efficiently convert prednisone to prednisolone. Oral steroids or injectable steroids are often used first to control ore severe asthma. Inhalers can be started at the same time as they take longer to reach effective lung concentrations.

RECOMMENDED FIRST-LINE ANTIBIOTIC CHOICES FOR BACTERIAL RESPIRATORY INFECTIONS



Acute bacterial URI in cats: Doxycycline or amoxicillin

Most common bugs: Staph, Strep, Pasteurella, E. Coli and anaerobes

Treatment length: 7 -10 days

Chronic (> 10 days duration) bacterial URI in cats:

Doxycycline or amoxicillin

If signs have persisted despite therapy with an appropriate acute URI antibiotic then further diagnostics are recommended (advanced imaging, rhinoscopy and tissue biopsy with culture, mycoplasma PCR/culture, fungal culture etc.) If antibiotic are to be continued, it is recommended to change antibiotic class

Canine infectious respiratory disease complex:

Doxycycline or amoxicillin-clavulanate

Most common bugs: B. bronchiseptica, S. equi subzoo, Mycoplasma

Treatment length: 7-10 days

Bacterial bronchitis dogs/cats: Doxycycline

Most common bugs: B. bronchiseptica dogs and cats, Mycoplasma dogs/cats

Treatment length: 7-10 days

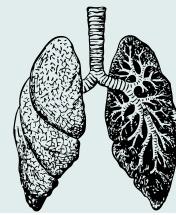
Pneumonia in animals with extensive contact with other animals: Doxycycline

Pneumonia otherwise: Fluoroquinolone and a penicillin or clindamycin pending C&S results from airway wash

Most common bugs: E coli, Pasteurella, Strep, B. bronchiseptica, Enterococcus, Mycoplasma, S. pseudintermedius, Staph, Pseudomonas

Treatment length: most textbooks recommend treat for 4-6 weeks. Shorter courses however, might be effective. Re-evaluate in 10-14 days.

Respiratory Medicine Pharmacology



VetHelpfulNotes
Canine Feline

MUCOLYTIC



Acetylcysteine 20% (Mucomyst and Acetadote)

Indications: Pneumonia, lower airway congestion

Mechanism of action: Decreases the viscosity of mucoid secretions in the bronchial tree

To Note: As a respiratory agent it is given via nebulization. Its use in nebulization therapy is controversial as efficacy studies are lacking and there is a small chance of bronchial constriction as a negative side effect.

This drug is far more often given orally as an antidote to toxicosis such as acetaminophen ingestion in cats. It acts as an anti-oxidant and can conjugate toxic metabolites.

ANTITUSSIVE



Hydrocodone-homatropine (Hycodan)

Indications: To reduce excessive coughing- most often in collapsing trachea

Mechanism of action: Opioid agonist for mu and kappa receptors. Homatropine is added to decrease substance abuse in people. In dogs hydrocodone is metabolized to hydromorphone and this has antitussive, analgesic and sedative properties.

To Note: can cause constipation, sedation, bradycardia. No established dose for cats.

BRONCHODILATORS



*** Drugs ending in -terol and -ine***

Terbutaline (Brethine)

Indications: To dilate lower airways in respiratory conditions like asthma that cause bronchoconstriction

Mechanism of action: Stimulates Beta 2 receptors which relax bronchiolar smooth muscle. May also inhibit the release of inflammatory mediators.

To Note: Excessive Beta- adrenergic stimulation may cause tachycardia by stimulating the beta receptors on the heart.

BRONCHODILATORS



Albuterol (Proventil, Ventolin)

Indications: Asthma

Mechanism of action: Beta agonist that is inhaled. Stimulates Beta 2 receptors to relax bronchiolar smooth muscle and open the airways.

To Note: As a respiratory agent it is given via an inhaler and a chamber system such as the Aerocat or Aerodawg system. This allows the medicine to be aerosolized into the chamber and slowly inhaled via an attached mask for 7 to 10 breaths. Onset of action in 15-30 minutes and duration can be as long as 8 hours.

*** **practice tip** *** the puff of the inhaler is frightening to some animals. You can puff the medication into the chamber away from the pet and *then* place the mask over their nose and mouth. *** **Alert**- some dogs present to the ER for biting an albuterol canister. This can cause acute hypokalemia because albuterol stimulates the Na^+/K^+ ATPase and this drives potassium intracellularly, dropping serum K^+ . Albuterol toxicity can also cause tachycardia and elevated blood pressure.



Aminophylline/Theophylline (generic brands)

Indications: Bronchodilator to open up lower airways and reduce inflammation/cough from tracheitis/bronchitis.

Mechanism of action: Methylxanthine bronchodilator. Non-selective PDE inhibitor which has anti-inflammatory effects and bronchodilatory effects.

To Note: Oral bronchodilator. Fluoroquinolones increase theophylline levels substantially. Do not use in dogs with seizures.

Antiarrhythmics

Some Block Potassium Channels

Class I: Sodium (SOME)

Class II: Beta Blocker (BLOCK)

Class III: Potassium Channel Blocker (POTASSIUM)

Class IV: Calcium Channel Blocker (CHANNELS)

(Class V: Work by unknown mechanism)

Class I: Lidocaine, Mexilitine

Class II: Atenolol, Propranolol

Class III: Sotolol, Amiodorone

Class IV: Diltiazem

Class V: Digoxin

Lidocaine/Mexilitine

Ventricular Arrhythmia

Atenolol/Propranolol

V tach, Feline HCM

Sotolol, amiodorone

A fib, Vtach

Diltiazem

SVT

Digoxin

A fib



Medical Uses

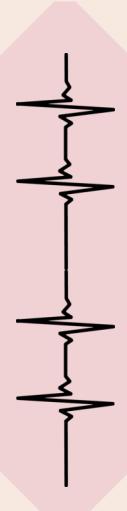
Lidocaine: blocks Na channel to prolong the action potential and slow the HR.

Atenolol: Beta 1 blocker that causes the heart to beat slower and less forcefully

Sotolol, amiodorone: Prolongs refractory period of action potential.

Diltiazem: Prevents calcium influx into cardiac cells to inhibit cardiac contractions.

Digoxin: slows AV node.



Lidocaine

Class I antiarrhythmic

Na channel blocker

Xylocaine

- Used most often in cardiac disease for animals in V Tach. Given IV
- Can also be used as a local, regional and topical anesthetic.



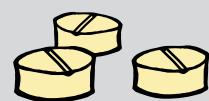
Mexitil

Class I antiarrhythmic
Na⁺ channel blocker

- Oral antiarrhythmic with similar effects as lidocaine
- Used to treat V tach and PVC's
- Often used with sotalol in dogs
- Needs to be given 3 times a day

Considerations

- Extreme caution 2nd or 3rd degree AV block
- Caution dogs with MDR1 mutation
- GI distress, (give with food), dizziness



Digoxin

Cardiac glycoside
Class V

Lanoxin

- Used primarily for heart rate control in A fib
- Found naturally in Foxglove
- Often combined with diltiazem
- Inhibits Na/K pump on cardiac muscle causing refractory AV node



Considerations

- Extreme caution with kidney disease and heart failure
- Can easily be over-dosed cause toxicity, must monitor blood levels
- Contraindicated in cats with HCM

Atenolol

Class II antiarrhythmic
Beta blocker Beta 1 selective

Tenormin

- Primarily used for ventricular hypertrophy and tachyarrhythmia in small animals
- Fairly Beta 1 selective so safe for animals with asthma

Considerations

- Contraindicated with bradyarrhythmia
- Caution with CHF, CKD
- Can cause lethargy, hypotension, diarrhea



VetHelpfulNotes
Canine Feline

Diltiazem

Class IV antiarrhythmic

Ca⁺ channel blocker

Cardizem, Dilacor

- Used most often to treat SVT and A fib
- To control A fib is used alone or with digoxin
- Inhibits cardiac muscle contractions by preventing calcium entry into cardiac myocytes

Considerations

- Contraindications: hypotension, AV block.
- Can cause GI side effects: vomiting, diarrhea, anorexia



Sotalol

Class III antiarrhythmic

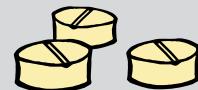
Non selective Beta Blocker and K⁺ channel blocker

Betapace

- Used to treat V tach
- Most often used in boxers with arrhythmogenic cardiomyopathy to suppress ventricular ectopy

Considerations

- May cause fatigue/dizziness, nausea and vomiting
- Can be tried in patients refractory to lidocaine and procainamide
- Beta blocking activity is 30% that of propranolol



Propranolol

Class II antiarrhythmic

Beta blocker

Inderal

- Used for acute treatment of APC's, VPC's, SVT
- Can be used to treat thyroid storm in cats



Considerations

- Contraindicated with CHF unless secondary to tachyarrhythmia
- Adverse effects bradycardia, lethargy, hypotension, bronchoconstriction
- Best if used IV; has poor oral bioavailability-so other oral beta blockers are preferred

Amiodorone

Class III antiarrhythmic

K⁺ channel blocker mostly some Na⁺ channel blocker and beta blocking properties

Pacerone

- Used for refractory V tach and prevent recurrence of A fib
- Because of the high possibility of side effects, typically only used for refractory V tach

Considerations

- Adverse effects: hepatopathy, immune dyscrasias, skin reactions
- Possible to have substantial side effects

Atropine

Parasympatholytic

Anticholinergic, antidote, Tx bradycardia

atropine sulfate

- Often used as part of pre-anesthetic protocol to reduce respiratory secretions
- Used during CPR
- Used for bradycardia, SA arrest
- Used to treat organophosphate and carbamate toxicity, muscarinic mushroom toxicity by reducing SLUD signs

Considerations

- ileus, obstructive GI disease: can reduce GI motility
- Contraindicated with tachycardia, narrow angle glaucoma, HCM

Blood Pressure Reduction

Vasodilators

Nitroprusside
Sildenafil
Pimobendan
Hydralazine

Alpha 1 Receptor Blockers

Prazosin
Phenoxybenzamine

Calcium Channel Blocker

Amlodipine

ACE Inhibitors and ARBs

Enalapril
Benazepril
Telmisartan

ACE Inhibitors

End in -pril

- Reduce formation of angiotensin II
- Inhibit aldosterone action
- Reduce blood pressure
- Reduce protein loss thru kidneys

Enalapril
Enacard

Benazepril
Lotensin

angiotensin I
ACE

angiotensin II

aldosterone

Na⁺ retention,
increased
blood volume



VetHelpfulNotes
Canine ♥ Feline

Benazepril

ACE inhibitor: ends in -pril

Use with caution in CKD

Lotensin

- Used to treat heart failure, hypertension and protein losing nephropathy
- Inhibits conversion of angiotensin I to angiotensin II. Angiotensin II is a potent vasoconstrictor and stimulates aldosterone.

Considerations

- Dual elimination in kidneys and liver unlike enalapril
- Adverse effects are uncommon but GI effects, hypotension and hyperkalemia are possible
- Avoid use in pregnancy

Enalapril

ACE inhibitor: ends in -pril

Often used when treating CHF

Enacard

- Used to treat heart failure, hypertension and protein losing nephropathy
- Inhibits conversion of angiotensin I to angiotensin II. Angiotensin II is a potent vasoconstrictor and stimulates aldosterone.

Considerations

- Mild effects to reduce hypertension. Can be combined with amlodipine to improve treatment of hypertension
- Recommended for later stages of heart failure to reduce fluid retention
- Can be used to reduce protein loss in protein losing nephropathy

Pimobendan

Ionodilator

Indicated for both preclinical heart disease and CHF

Vetmedin

- It's uses improves survival from CHF and chronic valvular disease when used with furosemide vs furosemide and enalapril
- Increases time to first onset of CHF when incorporated into therapy
- Extends survival when given to pre-clinical DCM Dobermanns

Considerations

- Inhibits PDE-III to increase calcium sensitivity and improve cardiac contractions Also has vasodilatory effects.



Sildenafil

Vasodilator and Phosphodiesterase inhibitor

Treats pulmonary hypertension

Viagra

- Used primarily to treat pulmonary hypertension which can lead to syncope and right heart failure in dogs
- Marketed as a generic, Reviato, for pulmonary hypertension and as a brand name, Viagra, for erectile dysfunction in people

Considerations

- Don't give to patients on organic nitrates like nitroglycerin
- Caution hypotension, fluid depletion, severe left ventricular outflow obstruction



Nitroprusside

Vasodilator

Can only be given IV

Nitropress

- Potent systemic vasodilator
- Used primarily to treat acute left sided heart failure
- Do not confuse with nitroglycerin (sound alike issue)



Considerations

- Blood pressure must be continuously monitored
- Can cause severe hypotension and cyanide toxicosis

Hydralazine

Vasodilator

Interferes with calcium movement

May decrease cardiac output and cause reflex tachycardia

Apresoline

- Treats systemic hypertension
- Reduces high blood pressure by vasodilation.
- Used primarily for CHF treatment for valvular heart disease



Considerations

- Not used as commonly as other vasodilators like Ace inhibitors.
- Caution: severe renal disease, intracerebral bleeding, auto-immune disease
- Often used with beta-blockers to combat reflex tachycardia

Amlodipine

Calcium Channel Blocker

May decrease cardiac output

Norvasc

- Treats systemic hypertension
- Decreases calcium influx in cardiac and vascular smooth muscle: Vasodilation

Considerations

- Drug of choice for hypertension in cats
- Adverse effects: hypotension, bradycardia and in dogs can cause gingival hyperplasia which can resolve after discontinuing the medication.

Alpha Blockers

End in: -osin, -ine

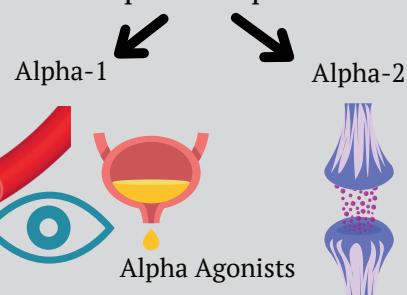
Non-selective alpha blockers
Phenoxybenzamine

Selective alpha-1 blockers
Prazosin

Selective alpha-2 blockers
Mirtazapine
Yohimbine

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Alpha Receptors



Alpha Agonists cause

- Blood vessel constriction
- Pupil dilation
- Urethral sphincter tone
- Analgesia
- Anxiolysis
- Sedative

Phenoxybenzamine

Alpha adrenergic antagonist:
ends in -osin, ine

Non selective alpha blocker

Dibenzyline

- Used mostly in small animal medicine to relax the urethra when there is urethral obstruction
- Also used for pheochromocytoma for vasodilation

Considerations

- Adverse effects: hypotension, rebound hypertension, increased IOP, tachycardia
- Slow onset of action: can take a few days

Prazosin

Alpha 1 receptor blocker

Hypotension, dizziness, lethargy and GI effects

Minipress

- Used commonly to reduce feline urethral sphincter tone in urethral obstruction. However, this drug is falling out of favor in UO treatment
- Alpha 1 blockade relaxes smooth muscle

Considerations

- Most commonly used in cats to treat functional urethral obstruction
- Can be used in dogs for reflex dyssnergia
- May also be used to treat CHF, systemic hypertension or pulmonary hypertension in dogs

ARB

angiotensin II receptor blocker

End in -sartan

Displace angiotensin II from the receptor to cause:

- vasodilation
- increase Na⁺, fluid loss, decrease K⁺ loss
- Very effective at reducing protein loss thru kidneys

Telmisartan
Micardis

angiotensin I



ACE

angiotensin II



ARB



ARBs cause low Na⁺ and elev K⁺, vasodilation in kidney

Telmisartan

ARB: ends in -sartan

FDA approved for control of hypertension in cats

Micardis

- Used to treat proteinuria, hypertension secondary to renal disease or CHF
- Blocks the action of angiotensin II. Angiotensin II is potent vasoconstrictor and stimulates aldosterone.

Considerations

- Do not use in hypotensive patients
- Do not use in patients with blood volume or electrolyte depletions
- Side effects are most commonly GI related; less common increases in liver values, azotemia, hypotension

Blood Pressure Support

Norepinephrine
Dobutamine



Dobutamine

Sympathomimetic

Synthetic catecholamine that activates B1 receptors on the heart

Dobutrex

- Causes dose dependent increases in cardiac contractility and ejection fraction to increase blood pressure
- Has a very short half life so must be given as a CRI

Considerations

- Can cause arrhythmias at high doses
- May be ineffective in patients who recently received a beta blocker



Norepinephrine

Sympathomimetic and alpha adrenergic agonist
Used to manage hypotension

Levonor

- Acts on alpha 1 and alpha 2 receptors to cause vasoconstriction
- Naturally occurring catecholamine that stimulates the sympathetic nervous system



Considerations

- Can cause peripheral and visceral vasoconstriction which may reduce blood flow to major organs
- Used to treat hypotension unresponsive to fluid resuscitation

Diuretics

Furosemide
Spironolactone
Hydrochlorothiazide



Furosemide

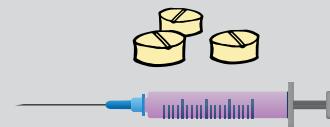
Loop diuretic

Most commonly used diuretic for CHF



Lasix

- Blocks the $\text{Na}^+/\text{K}^+/\text{2Cl}^-$ transporter on the loop of henle to inhibit 25% of NaCl reabsorption and promote diuresis.
- May cause pre-renal dehydration but this can often be corrected.
- Can cause electrolyte imbalances



Considerations

- Do not use with cardiac tamponade as it can worsen cardiac output
- Do not use with an aminoglycoside as dehydration can promote kidney injury

Spironolactone

Aldosterone antagonist

Potassium sparing diuretic

Aldactone

- Used as potassium sparing diuretic and adjunctive treatment for heart failure or ascites
- Should not be substituted for furosemide in CHF
- Competitively inhibits aldosterone at the distal tubule



Considerations

- Contraindications: hyperkalemia, addison's, acute or significant kidney disease
- Use precautions when handling: hazardous drug- use gloves

Hydrochlorothiazide

Thiazide diuretic

Used for nephrogenic DI, Ca Ox urolith prevention, and diuretic for CHF

Microzide

- Enhances excretion of Na^+ , Cl^- and water at DCT of kidney to enhance diuresis
- Decreased calcium excretion so this helps with reduction of Ca Ox stone
- Has anti-hypertensive effects



Considerations

- Caution severe kidney disease, electrolyte imbalances
- Caution Diabetes mellitus, SLE
- Do not use with hepatic impairment or hypercalcemia

Examples
Common doses
poor penetration
How it works

Aminoglycoside	Amikacin	15-20mg/kg IM, SQ, IV q24	eyes, prostate csf, pus	inhibit protein synthesis	bacteremia, abdom infections, topical otics	gram neg no anaerobic coverage	cidal conc dep
Cephalosporin	cefazolin (cefazolin (convenia) cefpodoxime (simplicef) cephalaxin (keflex))	22 mg/kg IV q12 iv 8mg/kg SQ 5-10mg/kg PO SID 22mg/kg PO BID	eyes brain testes prostate	inhibit cell wall synthesis	skin infections UTI, sx prophylaxis cefovacin: long acting parenteral 14 days	variable gram neg gram pos	cidal time dep
Tetracycline	Doxycycline Tetracycline	5-10 mg/kg po bid 22 mg/kg po tid (d) 15-22mg/kg po tid (c)	CSF	inhibit protein synthesis (30s)	rickettsial dz inflamm skin dz	gram -/+ mycoplasma, rickettsial spirochetes	static time dep
Pennicillin	Amoxicillin (amoxi tabs) Ampicillin Amoxi-Clav (Clavamox)	11-15 mg/kg q 8-12 20-40mg/kg iv q 6-8hr 14-25mg/kg po bid	eyes ,brain testes prostate	inhibit cell wall synthesis	UTI, resp infections, soft tissue infxn; ampi can tx lepto if rxn to doxy	gram neg gram pos	cidal time dep
Nitroimidazole	Metronidazole (flagyl)	10-15 mg/kg po bid	very few places	DNA damage	giardia, colitis, devitalized soft tx infection	anaerobes	cidal conc dep
Potentiated Sulfonamide	Sulfa-trimethoprim (TMS)	15-30mg/kg po q 12	pus	inhibit folate synthesis	skin infections, UTI Resp infections parasitic disease	gram neg gram pos	w/trimeth cidal time dep
Fluoroquinolone	Marboflox (zeniquin) Enrofloxacin (baytril) Pradofloxacin (veraflox)	2.75-5.5mg/kg SID 5mg/kg C 10mg/kg D 7.5mg/kg sid cat	saliva, bone csf	inhibit DNA replication	UTI, skin, soft tissue infections	gram neg gram pos	cidal conc dep
Macrolide	Azithromycin (zithromax) Erythromycin	5-10mg/kg po q 24 x 3-7 days typically dog/cat 10-20mg/kg po q 8-12 dog/cat; 0.5-1mg/kg iv TID	bone	inhibit protein synthesis (50s)	chlamydia or mycoplasma URI parasitic /rickettsial dz Eryth. prokinetic	gram pos	cidal time dep
Carbapenem	Metopenem Imipenem	8-12mg/kg SQ or IV q8 5-10 mg/kg IV q6-8	eyes brain testes prostate	inhibit cell wall synthesis	MDR UTI or soft tissue infections in hospital use only	gram neg gram pos toxoplasmosis	cidal time dep
Lincosamide	Clindamycin (antirobe)	6-11mg/kg po q 12	very few places	inhibit protein synthesis (50s)	skin infection, dental infection osteomyelitis	gram pos anaerobes Protozoal infec	static time dep
Amphenicols	Chloramphenicol	24-50 mg/kg PO q 6-8 Dog 12.5-20 mg/kg PO q 12 cat	very few places	inhibits protein synthesis (50s)	MDR infections good tissue penetration for hard to reach places	gram pos gram neg	mostly static time dep

Examples
poor coverage
action adverse rxn

Aminoglycoside					bacteremia, abdom infections, topical otics	gram neg no anaerobic coverage	cidal conc dep
Cephalosporin					UTI, sx prophylaxis cefovacin: long acting parenteral 14 days	rarely GI complications Cefovecin- long acting- so adverse rxn more dangerous	Nephrotoxic, caution dehydrated pet WHO priority drug
Tetracycline					esoph stricture, interacts antacids/dairy, caution teeth,renal/hepatic, bone development/pregnancy	poss GI side effects ampi: poor oral absorp, best iv	esoph stricture, interacts antacids/dairy, caution teeth,renal/hepatic, bone development/pregnancy



Site of Infection	Bugs at Site	Best Empiric Antibiotic
Abscess	Staph pseudintermedius, Ecoli obligate anaerobes, Pseudomonas aeruginosa, pasteurella multocida	Amoxicillin- Clavulanate: 12.5-25mg/kg q 12 Cefpodoxime 10mg/kg q 24
Anal gland	E coli, proteus mirabilis, staph pseudintermed, pseudomonas aeruginosa	Cefpodoxime 10mg/kg q 24 Amoxicillin- Clavulanate: 12.5-25mg/kg q 12 Enrofloxacin 10mg/kg q 24
Bone	Staph pseudintermedius, pseudomonas aeroginaosa, enterococcus, E. coli	Clindamycin 6-11 mg/kg q 12 Amoxicillin- Clavulanate: 12.5-25mg/kg q 12 Cephalexin 22mg/kg PO q 12
Kidney	E. coli, Staph pseudintermedius,, Streptococcus, Enterococcus	Enrofloxacin 10mg/kg q 24 Amoxicillin- Clavulanate: 12.5-25mg/kg q 12
CNS	E. coli, Staph pseudintermedius, Streptococcus, pasteurella	Amoxicillin- Clavulanate: 12.5-25mg/kg q 12 Cefpodoxime 10mg/kg q 24 Enrofloxacin 10mg/kg q 24
Prostate	E coli, B-hemolytic strep, Enterococcus spp, Staph pseudintermedius	TMS 15-30mg/kg po q 12 Enrofloxacin 10mg/kg q 24 Clindamycin 6-11 mg/kg q 12
Hepatobiliary tract	E coli, , Enterococcus spp, obligate anaerobes, staph pseudintermedius	Amoxicillin-clavulanate 12.5-25mg/kg PO q 12 Cefpodoxime 10mg/kg q 24 Enrofloxacin 10mg/kg q 24
Joint fluid	B hemolytic strep, staph pseudintermedius, pseudomonas aeruginosa, enterococcus spp	Amoxicillin-clavulanate 12.5-25mg/kg q 12 Clindamycin 5.5 mg/kg q 12 Cephalexin 22mg/kg q 12
Middle/inner ear	Pseudomonas aeruginosa, staph pseudintermedius, b- hemolytic strep, E. coli	Enrofloxacin 10mg/kg q 24 Marbofloxacin 5.5mg/kg q 24 Amoxicillin-clavulanate 14mg/kg q 12
Oral/gingival	Obligate anaerobes, pasteurella multocida, E coli	Amoxicillin-clavulanate 14mg/kg q 12 Clindamycin 5.5 mg/kg q 12 Marbofloxacin 2.75mg/kg q 24
Pyoderma	Staph pseudintermedius, pseudomonas aeruginosa, E. Coli, Staph schlefferi	Amoxicillin-clavulanate 12.5-25mg/kg PO q 12 Cephalexin 22-30mg/kg q 12 Clindamycin 11 mg/kg q 12-24
Lower Resp Tract	E coli, pseudomonas aeruginosa, Bordetella bronchiseptica, Enterococcus, Pasteurella multocida, staphpseudintermed	Amoxicillin-clavulanate 12.5-25mg/kg PO q 12 Enrofloxacin 10mg/kg q 24 PO Doxycyline 5mg/kg po q 12 or 10mg/kg po q 24
Upper Resp Tract	Staph pseudintermedius, pasteurella multocida, pseudomonas aeruginosa, E. coli	Enrofloxacin 10mg/kg PO q 24 Amoxicillin-clavulanate 12.5-25mg/kg PO q 12 Doxycyline 5mg/kg po q 12 or 10mg/kg po q 24
Lower Urinary Tract	E coli, proteus mirabilis, Staph pseudintermedius, Klebsiella pneumoniae	Amoxicillin 11-15mg/kg po q 12 Cephalexin 22mg/kg po q 12 TMS 15-30mg/kg po q12
Wounds	Obligate anaerobes, Staph pseudintermedius, E. coli, pseudmonas aeruginosa, Enterococcus spp	Amoxicillin-clavulanate 12.5-25mg/kg PO q 12 Cefpodoxime 10mg/kg 24 Clindamycin 6-11 mg/kg q 12-24hr

Opioid Selection, Dosing, Analgesia, Adverse Effects

Opioid	Common Dose	Analgesia	Agonist	Onset	Duration	Comments and Adverse effects
Decreasing Potency 	Dog and cat 1-5 ug/kg IV Most commonly used as a CRI Loading dose: Cat 5 ug/kg IV; Dog 2-10ug/kg IV CRI: Cat 5 ug/kg/hr Dog 2-10 ug/kg/hr	Excellent	full mu agonist	<1-2min	20-30 minutes	Less likely to cause adverse effects compared to other opioids.
Fentanyl 50 ug/ml	Dog and cat 0.01- 0.03 mg/kg IM, or IV. Commonly used in cat OTM at home: 0.03- 0.05mg/kg BID-TID. Dog and Cat 0.02mg/kg IV, IM	Moderate	partial mu agonist kappa antag	20-45 min	4-8 hours Dep. on pain, conc. and dose	Long duration of action but slow onset of action and minimal to no sedation; Same set of adverse effects as other opioids but much more mild . Don't use within 8 hours of surgery if planning to use full mu opioid. Partial reversal with naloxone
Buprenorphine 0.3mg/ml	Cat: 0.24mg/kg SQ, decrease if using multimodal analgesia; do not send home this high dose product Cat: two sizes: 0.4ml (1.2-3kg), 1ml (3.1-7.5kg); apply to dorsal cervical skin	III		1 hour	24 hours	Comes in a top spot applicator tube. Must wear gloves. Provides 4 days of analgesic effects.
Buprenorphine 1.8 mg/ml simbadol: feline labeled product	Dog 0.1- 0.2 mg/kg IM or IV Cat 0.1mg/kg IM or IV	III			1-2 hours	
Buprenorphine Transdermal 20mg/ml (Zorbium)	Dog and cat: 0.2-0.4 mg/kg IM or IV Dog and cat 0.4mg/kg typically for Sx and 0.2mg/kg for sedation	II		Excellent	1-5 min IV or 10-20 min IM	Similar effects as morphine, but no histamine release. At doses >0.1mg/kg in cats can cause hyperthemia.
Hydromorphone 2 mg/ml	Dog and cat: 0.2-0.4 mg/kg IM or IV Dog and cat 0.4mg/kg typically for Sx and 0.2mg/kg for sedation	IV		Mild	1 hour	Little analgesic effects. Good sedation in dogs and cats particularly if combined with a tranquilizer; Don't use within 8 hours of surgery if planning to use full mu opioid.
Butorphanol 10mg/ml	Dog and cat: 0.2-0.4 mg/kg IM or IV				20-60 min (dog) 90 min (cat)	Good alternative to morphine. NMDA antagonist. Causes panting, vocalization, vomiting, defecation. Greater cardiodepressant than morphine
Methadone 10mg/ml	Dog and cat: 0.2-0.4 mg/kg IM or IV	III		Excellent	1-5 min IV or 10-20 min IM	
Morphine 10 mg/ml 	Dog 0.25-1.0mg/kg IM or slow IV 0.5 mg/kg most common; 0.25 mg/kg old/compromised Cat 0.1- 0.3 IM or slow IV	II			1-5 min IV or 10-20 min IM	May cause vomiting after IM injection, histamine release with fast iv injection, bronchoconstriction and resp depression.
Increasing Potency					2-4 hours	etsy.com/shop/VetHelpfulNotes

Anesthetic and Sedation Drugs

Drug

Dose

Comments

Common adverse effects

Alpha-2 Adrenergic Agonists

Dexmedetomidine

Xylazine

5-10 ug/kg

0.2-1 mg/kg

Do not use with atropine as it can increase work on the heart and increased risk for arrhythmias; reversible with atipamizole. Higher doses required in cats than dogs. Do not use with epinephrine

Both can be used for emesis in cats, X is reversed with yohimbine

Bradycardia
Cardiac output reduction
Hypertension/hypotension
Vasoconstriction
AV Block

VetHelpfulNotes
Canine Feline

Benzodiazepines

Diazepam

Midazolam

0.1-0.5mg/kg

0.25mg/kg

Avoid oral use in cats: hepatic failure
May cause excitement in dogs; appetite stimulant in cats
Reversible flumazenil
Can be given IM for seizures unlike diazepam; Both D and M are anxiolytics reversible with flumazenil

Minimal cardiorespiratory effects; avoid PO use in cats: rare hepatic tox
Paradoxical excitation in some patients (mostly diazepam)
Caution midaz/etomidate in canine glaucoma

Inhalant Anesthetic Induction Agents

Isoflurane

Sevoflurane

Dog 1.5-1.8 %
Cat 1.2 to 2.2 %

dog 2.1-2.4%
Cat 2.6-3.4 %

Contraindications: Predilection for malignant hyperthermia in some dogs
Caution with increased intracranial pressure; dose dep resp. depression
More rapid induction and recovery
More easily dosed to effect for geriatrics; can mask induce; requires higher vaporizer settings

Decreased cardiac output
Decreased myocardial contractility; dose dependant respiratory depression, hypotension

Hypothermia/hyperthermia
May result in vasodilation, dose dep. resp depression

Sedatives

Acepromazine

0.02-0.05mg/kg

Doses are typically much lower than on approved label. Can potentiate effects of opiates. Tranquilizer not a anxiolytic

Peripheral vasodilation causing heat loss and hypotension

Intravenous Induction Agents

Ketamine

2-10 mg/kg

Often used in combination with diazepam or dexmedetomidine for injectable sedation
Inhibits NMDA, which can control pain as well; Also used in CRI's with dexmedetomidine. Eyes remain open and central, use eye lubricants

Caution increased ICP or intra-ocular pressure, Occasional muscle tremors/seizures

Propofol

2-6 mg/kg

Continuous monitoring required, caution for apnea. Short acting hypnotic. Can be used as an anticonvulsant; Myoclonus can occur during induction. Best if combined with a benzodiazepine.

Respiratory depression, myoclonus, repeated doses in cats can cause heinz body anemia; prolonged recovery in sighthounds

Etomidate

0.5-1mg/kg

Good choice for pre-existing cardiac conditions, head trauma or critically ill patients. Not good choice for Addisonian patients: it suppresses cortisol

muscle rigidity and myoclonus. Can be painful IV if not diluted. Causes miosis; caution with glaucoma

Alfaxalone

1-3mg/kg dog
3-5mg/kg cat

Neuroactive steroid affecting Gaba receptors
Does not provide analgesia; extralabel IM use with short and rapid duration

Resp depression/apnea, SQ/IM dosing can cause hyper-reactivity; SQ/IM dosing harder in larger pets, due to volume required

Opioids

Buprenorphine

0.01-0.03 mg/kg

Partial mu agonist, K antag often part of short term procedures or OTM; long onset of action; don't use within 8 hours of sx if likely planning to use full mu opioid

Minimal sedation, good analgesia; rare resp depression

Butorphanol

0.2-0.4 mg/kg

kappa agonist, mu antag Minimal analgesia; good sedation; anti-tussive

Sedation, ataxia

Fentanyl

05-10 ug/kg

Full mu agonist; Most potent opioid, commonly used as a bolus, or CRI; fast onset within 1-2 min, duration 20 min

Dose dependant resp, CNS and cardio depression

Hydromorphone

0.1-0.2 mg/kg

Full mu agonist
Minmal cardio/resp depression

hyperthermia in cats, vomiting, decreased GI motility, bradycardia

Morphine

0.2-0.5 mg/kg
Dog
0.2mg/kg Cat

Full mu agonist
Minimal cardio/resp depression

Histamine release (caution mast cell dz), vomiting, bradycardia, resp depression

Canine Sedation Choices

PICK ONE

YOUNG, HEALTHY

Light sedation: (use with a local block if possible)

Acepromazine 0.01-0.05 mg/kg IM or IV

Greyhounds, Boxers, Dobermanns used lower dose: 0.01-0.02 mg/kg

Maximum is 3 mg IM or 1.5 mg IV for any dog

OR

Profound sedation (orthopedic radiographs):

Dexmedetomidine 10ug/kg IM or 5 ug/kg IV

Fractious: can increase to 15-20 ug/kg IM or 7-10 ug/kg IV

And /OR add **Ketamine** 5 mg/kg IM or 2 mg/kg IV



OLDER, HEALTHY

Quiet, friendly

Midazolam 0.2 mg/kg IV or IM

Excited, painful

Add **Ketamine** 5-10 mg/kg IM or 3-5 mg/kg IV

Light sedation for nervous/fractious

Acepromazine 0.05mg/kg IM or 0.02 mg/kg IV

Fractious, healthy

Dexmedetomidine 5-10 ug/kg IM or 2-5 ug/kg IV



ANY DEBILITATED PATIENT

Midazolam 0.2mg/kg IV or IM

- Avoid **Acepromazine**
- Avoid **Dexmedetomidine**

ANY DOG WITH A HEART MURMUR

- No **Dexmedetomidine**
- Low dose **Acepromazine** if required



Reversal of Dexmedetomidine with Antisedan

Within 30 minutes, reverse dexmedetomidine with an equal volume of antisedan
Consider reversing with half volume after 30 minutes or in fractious dogs

Reversal of Hydromorphone or Morphine with Naloxone

Complete reversal - 0.01 mg/kg IV

Naloxone: Partial reversal- titrate to effect- 40 ug (0.1ml) IV per large dog , dilute to

40 ug/ml per dog and titrate for small dogs; OR consider 0.2 mg/kg **Butorphanol** instead of **Naloxone**

Reversal of Midazolam with Flumazenil

0.01-0.2 mg/kg IV - start at lower dose and repeat as needed to reverse sedation

Rough Recovery:

1/2 to 1 unit (use u-100 syringe)
per 10 lbs dexmedetomidine

Feline Sedation Choices

PICK ONE

YOUNG, HEALTHY

Light sedation:

Acepromazine (use local block if possible) or 0.02mg/kg to 0.05mg/kg IM/IV **Midazolam** 0.2mg/kg IV/IM

OR

Profound sedation (orthopedic radiographs):

Dexmedetomidine 10ug/kg IM or 5 ug/kg IV

Fractious: add **Ketamine** 5 mg/kg IM or 2mg/kg IV

OR use **Alfaxalone** 1-2mg/kg(~0.5-1ml) and combine

Alfaxalone, Ketamine and opioid in one syringe for SC or IM

OLDER, HEALTHY

Quiet, friendly

Midazolam 0.2mg/kg IV or IM

Light sedation for nervous/fractious

Acepromazine 0.05mg/kg IM or 0.025 mg/kg IV

Excited, painful

Add **Ketamine** 3-5 mg/kg IM or 2 mg/kg IV

Fractious, healthy

Alfaxalone 1-2 mg/kg(~0.5-1ml) and combine **Alfaxalone**, Ketamine and opioid in one syringe for SC or IM

ANY DEBILITATED PATIENT

- Avoid **Acepromazine**
- Avoid **Dexmedetomidine**

ANY CAT WITH A HEART MURMUR

- No **Ketamine**
- avoid **Acepromazine**
- avoid **Dexmedetomidine** - use low dose 3-5 ug/kg only if required

Can use **Alfaxalone** 1-2mg/kg, **Midazolam** 0.2 mg/kg; Do not mix **Alfaxalone** and **Midazolam** in the same syringe. Give **Alfax** SQ and **Midazolam + opioid** IM



Reversal of Dexmedetomidine with Antisedan

Use half volume of antisedan IM ONLY

Rough Recovery:

1/2 to 1 unit (use u-100 syringe)
per 10 lbs dexmedetomidine

Reversal of hydromorphone or Morphine with Naloxone

Complete reversal - 0.01 mg/kg IV

Naloxone: Partial reversal- titrate to effect- 0.0001 mg/kg- or titrate to effect, dilute to 40 ug/ml and titrate OR consider 0.2mg/kg **Butorphanol** instead of **Naloxone**

Reversal of Midazolam with Flumazenil

0.01-0.2mg/kg IV - start at lower dose and repeat as needed to reverse sedation

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Monitoring Under Anesthesia/Sedation

Circulation

- Palpation of pulses
- Auscultation of heart beat with stethoscope, doppler or esophageal stethoscope
- Assess CRT
- measure BP
- Measure blood gases

Tip: Lingual vein samples can closely approximate arterial blood sample for blood gas interpretation

- Pay attention to pulse quality
- Heart rate
- Whether there are any dropped beats when palpating pulses while ausculting
- Prolonged CRT >2-3 seconds suggest poor perfusion or dehydration
- BP systolic should be 90-140
- Blood lactate should be <2; lactate increases with poor perfusion
- PaCO₂ should be 35-45; Low PaCO₂ happens with acidosis and poor perfusion

Ventilation

- Observe chest wall movements
- Observe excursion of re-breathing reservoir bag
- Auscult lung sounds
- Observe for fogging of endotracheal tube or face mask
- PaCO₂, Bicarbonate
- End- tidal CO₂

- Monitor for presence or absence of respiration
- Monitor regularity and frequency of respiration
- Monitor pattern and depth of respiration
- PaCO₂ and Bicarb decrease with respiratory alkalosis (hyperventilation) and both increase with respiratory acidosis (hypoventilation)
- End -tidal CO₂ should be 35-45; it rises with hypoventilation, increased body temperature Falls with hyperventilation or decr body temp

Oxygenation

- Assess mucous membrane color and
- tongue color
- Monitor SpO₂

- Pink (adequate oxygenation)
- Pale or cyanotic (inadequate oxygenation)
- Low SpO₂ <95%

Anesthetic depth

Assessment of:

- palpebral, corneal and swallowing reflexes
- eyeball position
- jaw tone
- muscle and anal tone
- Response to painful stimulation
- Purposeful movements

Light anesthesia:

- strong palpebral and corneal reflexes
- Central eye position
- Swallowing, muscle twitching, purposeful movements
- Increase in heart and RR with or without vocalization



Moderate anesthesia:

- Ventral rotation of eyeball
- Loss of palpebral reflex with sluggish corneal reflex
- Moderate jaw tone



Deep Anesthesia:

- Muscle relaxation and loss of jaw tone
- Central eye position
- No corneal reflex
- No response to surgical stimulation
- Slow HR and RR, weak pulses



ASA Classes

ASA I Normal, healthy

ASA II Mild to moderate systemic disease

ASA III Severe systemic disease, but still active

ASA IV Severe systemic disease and incapacitated

ASA V Moribund, terminally ill

ASA-E Emergency



ANESTHESIA GUIDE

DRUGS AND FLUIDS PRE-OP PREP



Veterinary

Pharmacy Fast Facts

PAIN/SEDATION

Torb	10 mg/ml	0.2 - 0.4 mg/kg IV/IM/SQ
Midaz	5 mg/ml	0.2 mg/kg IV/IM
Ace	10 mg/ml	0.01-0.02 mg/kg IV/IM/SQ
Fentanyl	50 mcg/ml	3-5 mcg/kg IV/IM
Hydro	2 mg/ml	0.1 mg/kg IV/IM/SQ
Morphine	10 mg/ml	0.2-0.4mg/kg IV/IM/SQ
Ketamine	100 mg/ml	3-5 mg/kg IV/IM
Buprenor	0.3 mg/ml	0.01 -0.04 mg/kg IV/IM/SQ
Methadone	10 mg/ml	0.3 -1 mg/kg IV/IM/SQ
Bupivacaine	2.5 - 5 mg/ml	1-2 mg/kg 15 min prior
Lidocaine	20mg/ml	2-4 mg/kg 5 min prior

NAUSEA

Cerenia	10 mg/ml	1 mg/kg IV/SQ OR 0.1 ml/kg
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INDUCTION

Alfaxalone	10 mg/ml	2-6 mg/kg IV/IM
Dexdom	0.5 mg/ml	5-10 mcg/kg IV
Propofol	10 mg/ml	2-6 mg/ml IV

REVERSALS

Atipamezole	5 mg/ml	= volume as dex
Naloxone	0.4 mg/ml	0.01 -0.04 mg/kg
Butorphanol	10 mg/ml	0.05 -1 mg/kg

NSAIDS

Carprofen	50 mg/ml	2.2 mg/kg q 12
Meloxicam	5 mg/ml	0.2 mg/ml SQ load (dog)
Onsior	20 mg/ml	2 mg/kg SQ OR 0.1ml/kg

EMERGENCY DOSES

Dextrose Boluses

Symptomatic hypoglycemia with BG <60
Give 1ml/kg 50% dextrose, diluted 1:4 over 10 min

Emesis Induction

Apomorphine 0.03 mg/kg IV, can be repeated

Anticonvulsants

Midazolam (5 mg/ml): 0.4 mg/kg IV/IN

Diazepam (5 mg/ml) : 0.5 mg/kg IV/Rectal

Phenobarb (65 mg/ml or 120 mg/ml):

3-5 mg/kg IV for 3 doses ; total of 9-15 mg/kg

Levetiracetam (100 mg/ml): 30 mg/kg IV

Antiarrhythmics

Lidocaine: 2mg/kg IV SLOWLY

Allergic Rxn

Diphenhydramine 2 mg/kg IM

+/- Dex-SP 0.1 mg/kg IV

Fluid Therapy

Maintenance Fluid Therapy

2 ml/kg/hr OR 40-60 ml/kg/day

Shock Bolus

90 ml/kg in dogs, divided into 30 ml/kg bolus IV
60 ml/kg in cat, divided into 20 ml/kg bolus IV

SQ Fluid Administration

20-30 ml/kg SQ +/- accounting for dehydration

Surgical Fluid rate

3-5 ml/kg/hr

CPCR

Epinephrine (1 mg/ml)

0.1 ml per 10 Kg

Atropine (0.54 mg/ml)

1 ml per 10 kg



EQUIPMENT PRE-OP PREP

Endotracheal Tubes

WEIGHT (KG)	ET TUBE SIZE (MM)
1	3
2	3.5
3.5	4
4+	4.5

WEIGHT (KG)	ET TUBE SIZE (MM)
2	5
3.5	5.5
4.5	6
6	6.5
8	7
10	7.5
12	8
14	8.5
16	9
18	9.5
20	10
25	11
30	12
40+	14-16

Anesthetic Machine

- Primary Oxygen Source Checked
- Back-up Oxygen Available
- Oxygen Alarm Working
- Flowmeters Working
- Vaporiser Attached and Full
- Anesthesia Machine Leak Tested
- Scavenging Checked
- Monitoring Equipment Functioning
- Emergency Equipment and Drugs Checked
- Pop off valve open unless using ventilator

Reservoir Bag

WEIGHT (KG)	BAG SIZE
0-4.5 Kg	1/2 L Bag
4.6-9 Kg	1 L Bag
9.1-27.2 Kg	2 L Bag
27.3- 54.4 Kg	3 L Bag

Drugs & Equipment Needed

- HEAT SUPPORT
- ENDOTRACHEAL TUBES (CUFFS CHECKED)
- LARYNGOSCOPE / LIDOCAINE / STYLET / SUCTION
- EMERGENCY EPINEPHRINE
- ATROPINE
- REVERSALS (ATIPAMEZOLE / NALOXONE / BUTORPHANOL)
- IV CATHETERS
- ISOTONIC CRYSTALLOIDS
- FLUID ADMINISTRATION SET
- CPCR ALGORITHM AVAILABLE



ANESTHESIA GUIDE

PERI-OP PREP



Intubation & Ventilation

- ✓ Et Tube length: tip of the nose to the thoracic inlet
- ✓ Apply sterile lube to the ET cuff
- ✓ Visualize vocal folds during placement
- ✓ Proof of proper placement:
 - ECO2 registering
 - condensation in ET tube during exhalation
 - movement of the reservoir bag
- ✓ Cuff inflation: Listen for air leakage while assistant gives a manual breath
- ✓ If assisting with manual breaths, close pop off and squeeze the reservoir bag to 15-20 cm H2O
- ✓ If manually ventilating (during apneic periods or to reduce elevated ETCO2), give 1 breath every 5-7 seconds

WEIGHT	BREATHING CIRCUIT
	Non-Rebreathing System
< 3 KG	<ul style="list-style-type: none"> Ayre's T Piece Mod Jackson-Rees Bain
3-10 KG	<ul style="list-style-type: none"> Ayre's T Piece Mod Jackson-Rees Bain Pediatric circle (not pediatric universal F)
	Rebreathing System
10-20 KG	<ul style="list-style-type: none"> Adult Circle Adult Universal F (not if ETT is > 9.5mm) Adult Circle
>20 KG	<ul style="list-style-type: none"> Adult Circle



02 FLOW RATE

Non-Rebreathing System

O2 FLOW RATE	200 ml/kg/min never less than 500 ml/min
O2 FLOW RATE	30 ml/kg/minute Generally run at 1 L/min for most patients Never less than 500 ml/min



Iso

Sevo



1.2-1.3%

2.3%



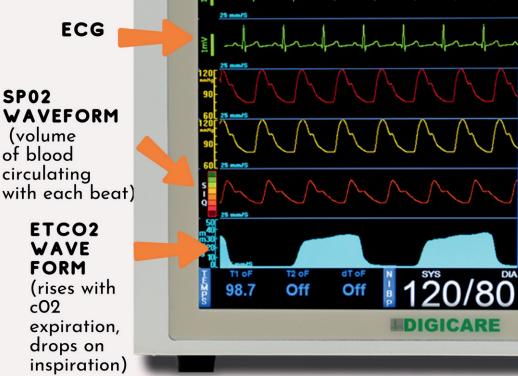
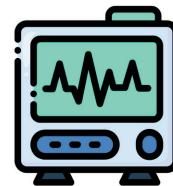
1.3-1.6%

2.6-3.4%



NORMAL VITAL SIGNS UNDER ANESTHESIA

Large dog HR	60-160
Small dog HR	70-180
Cat HR	90-200
Resp Rate:	8-12
SPO2:	>95%
MM color	pink
CRT	1-2 sec
ETCO2	35-45
BP	>80 mmHg Sys >60 MAP
Temp	>98 deg F



INTRA-OP MONITORING

LIGHT ANESTHESIA:



- Strong palpebral and corneal reflexes
- Central eye position
- Swallowing, muscle twitching, purposeful movements
- Increase in heart and RR with or without vocalization

MODERATE ANESTHESIA:



- Ventral rotation of eyeball
- Moderate jaw tone
- Loss of palpebral reflex with sluggish corneal reflex

DEEP ANESTHESIA:



- Muscle relaxation and loss of jaw tone
- Central eye position
- No corneal reflex
- No response to surgical stimulation
- Slow HR and RR, weak pulses

Bradycardia

Intervention:

- Administer Atropine 0.02-0.04 mg/kg
- Determine possible cause
- Most common are drug induced (anesthetic gas, narcotics).
- Reduce anesthetic gas, may need to reverse medications: Naloxone, Flumazenil

Tachycardia

Intervention:

- Might be insufficient anesthetic depth: increase gas
- Patient might be painful: add opioid?
- Check for hypotension
- Check PCV for hemorrhage

VETHHELPFULNOTES

Hypertension

Intervention:

- Evaluate anesthetic depth
- Administer supplemental oxygen
- Consider increasing gas inhalant

Excessive Depth

Intervention:

- Decrease Anesthetic gas flow rate
- Increase IV fluid rate/provide boluses as needed to raise BP
- Provide additional heat support
- Consider reversal medications

Hypotension

Intervention:

- First decrease gas if possible
- If bradycardia, 1/2 dose glyco/atropine
- Address pain
- Kiss of ketamine or dexdom to reduce anesthetic gas needs
- 10 ml/kg crystalloid bolus
- Consider using pressors: dopamine: 2-10 mcg/kg/min
dobutamine: 1-10 mcg/kg/min
Ephedrine: 0.05-0.1 mg/kg IV



ECG Interpretation



Vet Helpful Notes
Canine Feline

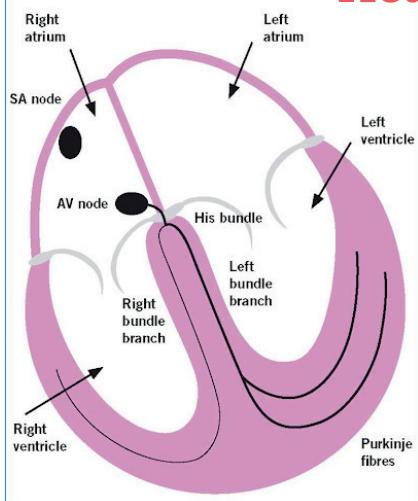
Normal heart rate ranges

Adult dog	70-160 beats/minute
Giant breeds	60-140 beats/minute
Toy breeds	70-180 beats/minute
Puppies	70-220 beats/minute
Adult cats	120-240 beats/minute

Six basic questions to help with electrocardiogram interpretation

- What is the heart rate (slow, normal, fast)?
- What is the rhythm (regular, regularly irregular, irregular)?
- Is there a QRS complex for every P wave?
- Is there a P wave for every QRS complex?
- Are they consistently and reasonably related?
- What is the morphology of the QRS complex (narrow and upright = supraventricular or wide and bizarre= ventricular)?

Heart Conduction



Conduction starts at the SA node in the right atrium and propagates to the AV node, down through the bundle of His and finally to the Purkinje fibers of the Ventricles.

Partial or complete blockage of electrical activity at the AV node is a form of AV Block (Types 1-3).

Junctional escape beats are started at the AV node.

Premature ventricular complexes occur at various locations in the ventricles.

Heart Arrhythmia Classification

Grade	Ventricular Arrhythmia
0	No ventricular arrhythmias
I	Unifocal, infrequent VPCs (<30/hour)
II	Unifocal, infrequent VPCs (>30/hour)
III	Multifocal VPCs
IVa	Ventricular couplets
IVb	Ventricular triplets or nonsustained ventricular tachycardia
V	R-on-T

Arrhythmias due to faulty conduction in the AV node

Partial or complete blockage of electrical activity at the AV node is AV Block (Types 1-3).

Junctional escape beats are started at the AV node.

Supraventricular arrhythmias

SVT (Supraventricular tachycardia)

Atrial Fibrillation

Bradycardia

Heart block

Ventricular Arrhythmias

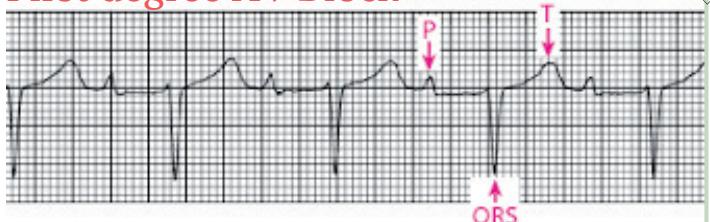
Junctional escape beats arise from a high ventricular location

V Tach

Left and Right BBB

VPC's (same as PVC's): monomorphic, (all look the same, so they come from the same hyper-exciteable focus in the ventricle) polymorphic (VPC's are differently shaped, meaning they come from different locations in the ventricle= more sinister arrhythmia) fusion, bigeminy, trigeminy

First degree AV Block



1st degree: In 1st degree AV block, conduction across the AV node or the bundle of His is merely slowed--the P-R interval is thus prolonged. The normal P-R interval is less than 0.14 seconds in a dog and 0.08 seconds in a cat.

First degree block is usually benign and caused by increased vagal tone or drugs like beta blockers. It is usually reversible and does not typically require treatment.

2nd degree AV Block



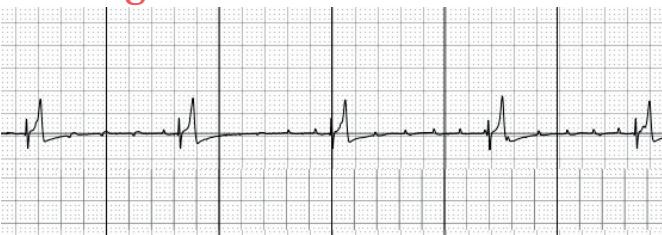
In second degree AV block some impulses are conducted normally, while others are “blocked” at the AV node. The AV node is located at the junction between the atria and ventricles.

Some P waves have no associated QRS.

In 2nd degree Mobitz type I AV block, the P-R interval is variable. It indicates high vagal tone and is less concerning.

In 2nd degree Mobitz type II block, the P-R interval is elongated with NO variation. This indicates a more serious conduction disorder. It may progress to 3rd degree block and require a pacemaker.

Third degree AV Block

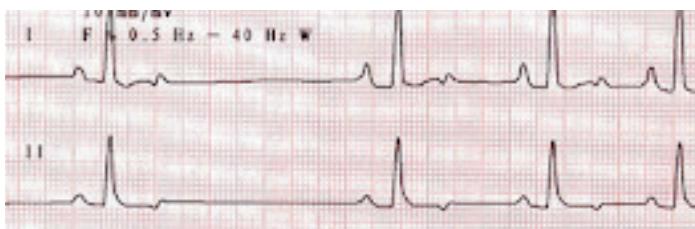


In third degree AV block all impulses are blocked at AV node. This results in a heart rate that is too slow (bradycardia) because it is generated entirely by the ventricles (ventricular escape rhythm).

The escape rhythm is much slower than the rate of firing coming from the SA node or AV nodes. Cats with 3rd degree AV block have HR's of 80-130bpm. Dogs have a HR of 20-60bpm.

Intervention: Pacemaker.

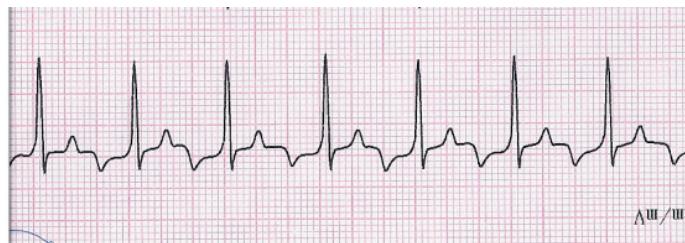
Sinus Bradycardia



Sinus arrhythmia is caused by high vagal tone. It is abolished with atropine.

The atropine response test is performed by administering 0.04 mg/kg of atropine IV and repeating an ECG 15 minutes later to determine if the sinus bradycardia was abolished. Typically this rhythm does not require treatment.

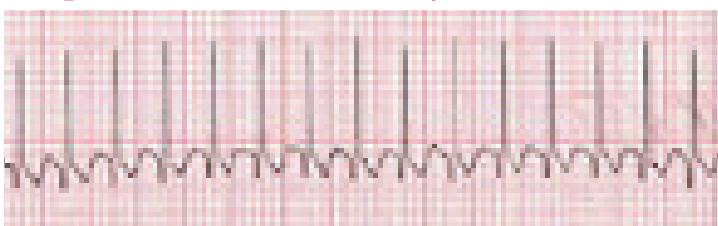
Sinus Tachycardia



Sinus Tachycardia is a very common rhythm disturbance in small animals due to stress and excitement. Unrelenting sinus tachycardia may be due to CHF and high sympathetic drive. Animals in pain or with fevers may have persistent sinus tachycardia. You must treat the underlying illness.

Heart rates with sinus tachycardia are slower than in SVT. Also, P and T waves are easy to distinguish in sinus tachycardia.

Supraventricular Tachycardia



Sustained supraventricular tachycardia (SVT) can be a life-threatening rhythm disturbance in the dog.

This rhythm disturbance occurs most often in animals with primary noncardiac diseases. SVT is distinguished from Sinus Tachycardia by its higher heart rate and because the P and T waves cannot be distinguished. Treatment: IV diltiazem.



Arrhythmia



Intervention/Key Points

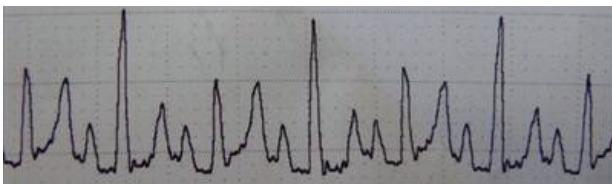


Ventricular Tachycardia



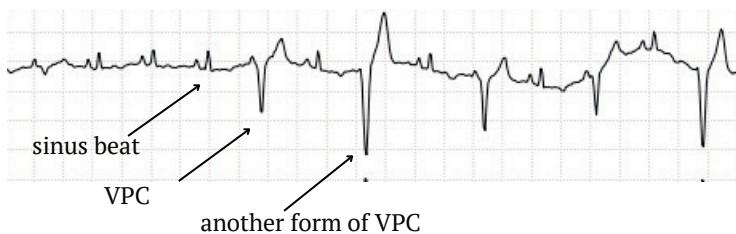
Ventricular tachycardia significantly decreases cardiac output. It can worsen congestive heart failure (CHF) and contribute to weakness or collapse. Sustained v-tach lasting >30 seconds is an indicator of medium to high risk for sudden death (due to development of ventricular fibrillation). Treat with lidocaine.

Electrical Alternans



Electrical alternans is characterized by an alternating size of the QRS complex and/or occasionally the T wave, which can change from beat to beat. This ECG sign generally suggests a large volume of pericardial effusion is causing the heart to move back and forth inside the pericardium. Sinus tachycardia is common with cardiac tamponade (pressure of the fluid around the heart). Treatment: pericardiocentesis.

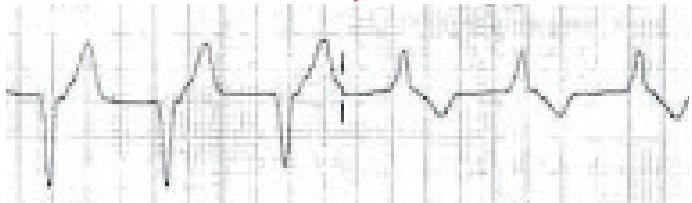
Polymorphic VPC's



Differently shaped VPC's (polymorphic), indicating they are complexes arising from more than one excitable foci on the ventricle. Polymorphic VPC raise the concern for progression to a malignant arrhythmia. Treatment is recommended if you see any of the following:

- Hemodynamic alterations are present: syncope, weakness, collapse, hypotension, exercise intolerance, respiratory effort, restlessness, worsening of heart failure.
- Pairs or triplets of VPC's and/or polymorphic VPC's which indicate an increased risk of progression to ventricular fibrillation.
- Sustained/fast ventricular tachycardia (>180-200 bpm)
- R-on-T phenomenon (R wave falling very close to the T wave of the preceding QRS complex)

Idioventricular Rhythm

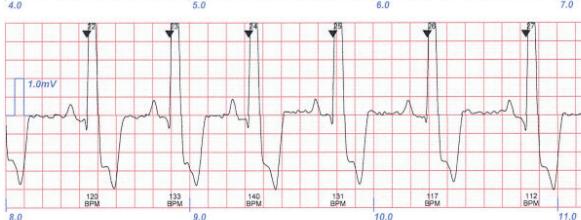


This is a ventricular arrhythmia that occurs in systemically ill animals (e.g., vehicular trauma, splenic disease, GDV) at a rate less than 160 beats per minute.

No direct anti-arrhythmic therapy is indicated if all of the following criteria apply: HR and blood pressure are within normal ranges and clinical signs of hypotension are absent (weakness, syncope) and no other premature ventricular ectopic beats are occurring.

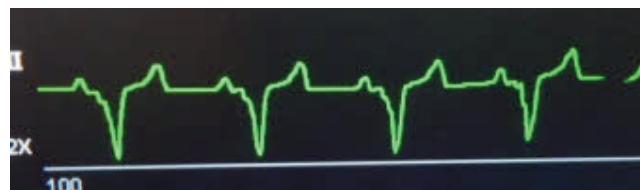
These ventricular beats are not premature. They are depolarizing similar to the normal sinus rate.

Left Bundle Branch Block



Left bundle branch block, in contrast to a right bundle branch block, is a more serious wave form and indicates more serious heart disease is present. LBB does not always require treatment but further investigation is recommended. Like a RBB, the LBB is delayed electrical conduction through the purkinje/HIS fiber system on the left side of the heart. It has the opposite deflection as the RBB and also has a p wave to distinguish it from Ventricular tachycardia.

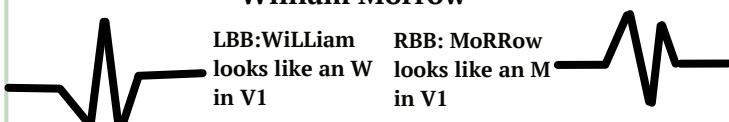
Right Bundle Branch Block



"William Morrow"

LBB: WillAmm
looks like an W
in V1

RBB: MoRRow
looks like an M
in V1



Right bundle branch block looks like a scary VPC rhythm, but is actually a harmless rhythm caused by delayed electrical impulses through the right ventricle. The delayed impulses through the right ventricle causes the wave form to look wide and bizarre. What distinguishes this wave form from a VPC is the preceding p wave.



Arrhythmia



Intervention/Key Points



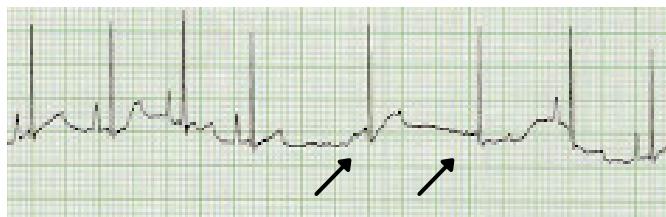
Atrial Fibrillation



Hyperkalemia

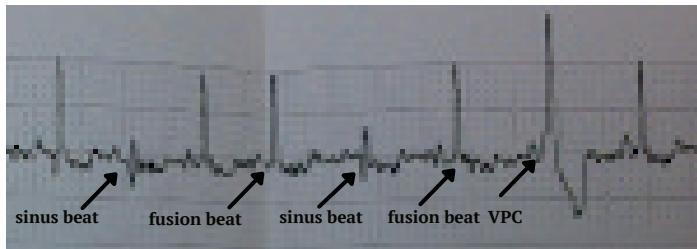


Junctional Escape Beat



Junctional beats. Look like more normal complexes without the preceding p wave

Fusion Beats



Isoarrhythmic Dissociation



VPC's firing at same rate as sinus beats

Red arrows show sinus beats. Can see p waves of sinus beats landing very close to the VPC's and firing at the same relative rate as the VPC's. Both VPC's and sinus beats are firing independently.

Electrocardiography (ECG) is required to definitively diagnose AF. This is determined by a lack of identifiable P waves (an irregular bumpy baseline called an F wave) and an irregular ventricular rate characterized by a variable R-to-R interval. Usually, the QRS complexes are narrow and predominately upright in lead II and generally the HR is elevated. Treatment: slow the HR with betablockers, diltiazem or digoxin.

ECG changes in mild hyperkalemia include increased amplitude, narrowing of the T wave, and shortening of the QT interval. Moderate hyperkalemia causes prolongation of the PR interval and widening of the QRS.

As hyperkalemia progresses, P waves decrease in amplitude, become wide, and eventually disappear. Bradycardia may be observed, but is less pronounced in cats. The QRS may merge with the T wave creating a sine wave appearance.

Treatment: iv calcium gluconate, iv dextrose +/- insulin

Junctional beats arise from the junction between the atria and the ventricles at the Bundle of His. They have a more supraventricular look, but unlike a supraventricular beat, they don't have a p wave (because they arise from below the atria). Junctional escape beats typically are benign and don't require treatment.

Fusion beats are a combination of a normal sinus beat merged with a VPC. On first glance it may appear that there is only one VPC present.

The fusion beats are the intermediate sized waveform. They indicate more VPC's are happening in this patient than immediately meets the eye.

Treatment recommendations vary depending on the patient's clinical status.

Atria and ventricles are beating at the same rate by their independent pacemakers but are not associated with each other. Happens a lot in cats, and is typically a benign rhythm. Can be abolished by accelerating the rhythm with atropine. This rhythm rarely causes hemodynamic compromise and generally does not require treatment.

It is not 3rd degree AV block because both the atrial pacemaker and the ventricular or junctional pacemaker are firing at a relatively normal and similar rate. It is not really understood why isoarrhythmic dissociation occurs. It can happen in healthy cats.

ANESTHETIC Interventions

NORMAL VITAL SIGNS UNDER ANESTHESIA

Temp	97-102 F	
Large dog	HR 60-160	MM color: pink
Small dog	HR 70-180	CRT: 2-3 sec
Cat	HR 90-200	ETO2: 35-45mmHg
Resp Rate:	8-20 BPM	BP >80 mmHg Syst
SPO2:	>95%	>60 MAP

Excessive Anesthetic Depth

SIGNS:

- Minimal Jaw Tone
- Central Eye Position
- Lack of Corneal Reflex
- No response to surgical stimulus
- Bradycardia
- Slow Respiratory Rate
- Hypotension



INTERVENTION:

- Decrease Anesthetic gas flow rate
- Increase IV fluid rate/provide boluses as needed to raise BP
- Provide additional heat support
- Consider medications as indicated
(see below for more details)

Too light

INTERVENTION:

- Increase gas flow rate
- Manually ventilate
- Administer propofol 0.5-1mg/kg IV over 30 sec

Pale mucous Membranes

IF YOU NOTICE GUM PALLOR, CHECK:

- anesthetic depth
- Signs of circulatory problems:
HR (too high or too low) or BP (low)
- If these signs are normal, collect a PCV/TS

Bradycardia:

LG BREED DOG < 60

SM BREED DOG <70

CAT <90

Dexmedetomidine

causes low HR

Dogs: 30-60, cats 80-100

INTERVENTIONS

- Administer Atropine 0.02-0.04 mg/kg
- Determine possible cause
most common are drug induced (anesthetic gas, narcotics).
- Reduce anesthetic gas, may need to reverse medications: Naloxone, Flumazenil

Tachycardia:

LG BREED DOG >160

CAT >200

SM BREED DOG >180

INTERVENTIONS

- Might be insufficient anesthetic depth:
increase gas
- Patient might be painful
- Check for hypotension causing reflex tachycardia

Hypotension

HYPOTENSION:

BP < 80MMHG SYSTOLIC
MAP < 60MMHG

SIGNS:

Increased CRT >2 second

Poor pulse quality

Tachycardia or Bradycardia

Cool extremities and Body temperature

ROUGH ESTIMATES OF BLOOD PRESSURE:

Absence of a peripheral pulse indicates systolic pressure < 60mmHg

INTERVENTION:

- First decrease the amount of gas if possible
- Try a 10-20 ml/kg crystalloid bolus
- Increase the fluid rate
- A colloid bolus can be given next if there is inadequate response
- Consider using PRESSORS:

DOPAMINE: 2-10 MCG/KG/MIN

DOBUTAMINE: 1-10 MCG/KG/MIN

EPHEDRINE: 0.05-0.1 MG/KG IV

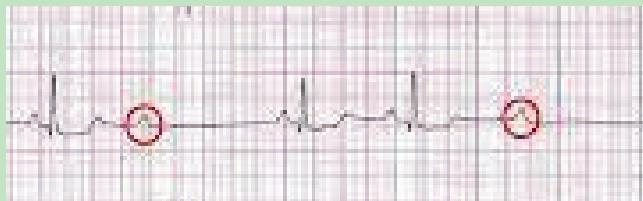
- If nothing is working to correct the hypotension, the procedure may need to be aborted

Hypertension

- Evaluate anesthetic depth
- Administer supplemental analgesia
- Consider increasing gas inhalant



COMMON ANESTHETIC ARRHYTHMIAS



2ND DEGREE AV BLOCK

Drugs that can lead to AV block
slow the HR: Dexmedetomidine, methadone,
hydromorphone, fentanyl

2nd degree AV block is when there are p waves and dropped QRST complexes. This is a common anesthetic arrhythmia and generally does not require intervention. However, high grade 2nd degree block, (when there are many p waves with dropped QRST complexes) is more dangerous. This block is more likely to lead to 3rd degree block (complete dissociation between p waves and QRST complexes). A low HR or hypotension warrants treatment. Administer atropine.

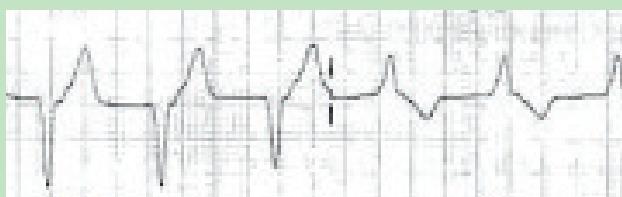


SINUS TACHYCARDIA

Heart rates with sinus tachycardia are typically slower than in SVT. Also, P and T waves are easier to distinguish.

Sinus Tachycardia is a very common rhythm disturbance in small animals due to stress and excitement. Unrelenting sinus tachycardia may be due to CHF and high sympathetic drive. Animals in pain or with fevers may have persistent sinus tachycardia. Treat underlying illness.

Check for pain, hypoxia, etc.



IDIOVENTRICULAR RHYTHM

Ventricular beats are not premature. They are depolarizing at the sinus rate.

An idioventricular (IV) rhythm is called an accelerated IV rhythm if the HR is fast. Generally this rhythm indicates systemic illness is present such as: GDV, splenic tumor or pancreatitis. Often don't have to treat this rhythm. However, you should monitor the BP, HR and ECG until the rhythm converts back. Also keep an eye on oxygenation, lyses, acid/base, hydration and lactate levels.



MULTIFOCAL VPC'S

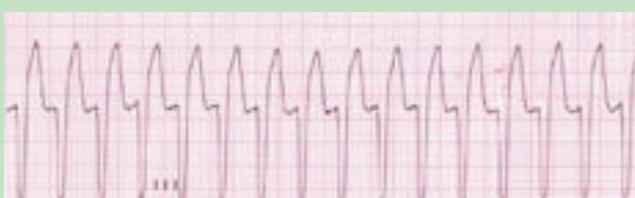
Irregular rhythm with premature ventricular beats. Pairs or triplets of VPC's increase concern level.

Polymorphic VPC's raise the concern for progression to a malignant arrhythmia. Treatment is recommended if you see any of the following:

-Hemodynamic alterations are present:

Hypotension, Tachycardia, Respiratory effort.

-Pairs or triplets of VPC's and/or polymorphic VPC's indicate an increased risk of progression to ventricular fibrillation.



V TACH

This is an urgent concern! Treat immediately.

Ventricular tachycardia significantly decreases cardiac output and contributes to weakness or collapse. Sustained v-tach lasting >30 seconds is an indicator of medium to high risk for sudden death (due to development of ventricular fibrillation). Treat with lidocaine if HR >150-180. Start with a 2mg/kg iv bolus. Hypotensive V tach indicates either cardiac illness or systemic illness. If no response within a minute or two, give a second dose. After that, if there is no conversion, switch to another anti-arrhythmic drug like : procainamide, diltiazem or propranolol.

Important drug/fluid doses

Atropine

Dogs and Cats 0.02 to 0.04 mg/kg IV, IM, SQ
Faster onset, shorter duration of action than Glyco

Glycopyrrolate

Dogs and Cats 0.005 to 0.01 mg/kg IV, IM, SQ
Slower onset, lasts longer than atropine

Flumazenil

Dogs and Cats 0.01 mg/kg IV; Repeat hourly as needed

Naloxone

Dogs and Cats 0.01-0.04 mg/kg IM or IV; Give 1/4 of the calculated dose every 3-4 minutes until desired effect achieved
Re-dose every 1-3 hours if needed

Dobutamine CRI

Dog 2-10 mcg/kg/min
Cat use low end of the range

Sample recipe for 2 mcg/kg/minute dose
1. Add 60 mg of Dobutamine (4.8ml of a 12.5 mg/ml solution) to a 250 ml bag of 0.9% Saline
This provides a 0.24 mg/ml solution

2. If you give 0.5 ml/kg/hour it will provide 2 mcg/kg/minute

Dopamine CRI

Dog 1-10 mcg/kg/min
Cat use low end of the range

Sample recipe for 0.002 mg/kg/minute dose
1. Add 60 mg of Dopamine (1.5ml of a 40 mg/ml solution) to a 250 ml bag of 0.9% Saline
2. If you give 0.5 ml/kg/hour it will provide 0.002 mg/kg/minute

Surgical Crystalloid Rate: 5 ml/kg/hour dogs, 3ml/kg/hr cats

Crystalloid Bolus for hypotension: 5-10ml/kg

<https://www.etsy.com/shop/VetHelpfulNotes>

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Blood Film/Hematology Cheat Sheet

Blood Film Basics

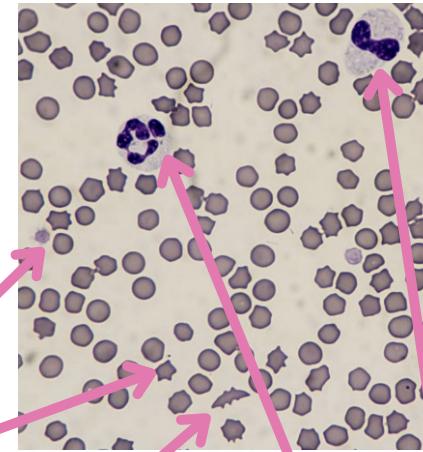
- First scan the slide using a 10x or 20X objective
- Screen for rouleaux or agglutination
Confirm that the mature neutrophil is the predominant nucleated cell type
- Record if there are any nucleated RBC or immature neutrophils and if there are any large or atypical leukocytes
- Corrected WBC (thou/ μ L) = obtained nucleated cell count \times (100 \div [nRBC + 100])**
- Identify any platelet clumps- pay particular attention on feline blood smears!
- Platelet estimate**
dogs: # platelets in a 100X field \times 15,000
cats # platelets in a 100x field \times 20,000
Count several fields and use the average.
Normal for dogs: 8-15 plt/HPF
Normal for cats: 10-12 plt/HPF
- Crude WBC estimate**
Healthy pet: 18-50 WBC per LPF (10X) in the monolayer. Approx 330 cells/ μ l in this estimated area
- Leukocyte differential.** Find good spot in monolayer and count 100 leukocytes, separating into numbers of neutrophils, lymphocytes, monocytes, eosinophils and basophils. Multiply the % of each leukocyte type by the total WBC from the machine count (after correcting for nRBCs) will give you the absolute count
- Erythrocyte evaluation:** Search for agglutination or rouleaux. Count NRBC's to correct WBC.
- Check morphology on 100x oil immersion, slightly behind feathered edge, evaluating for poikilocytes
- TIP: spherocytosis cannot be identified in cats!

etsy.com/shop/VetHelpfulNotes

Platelet

Echinocyte

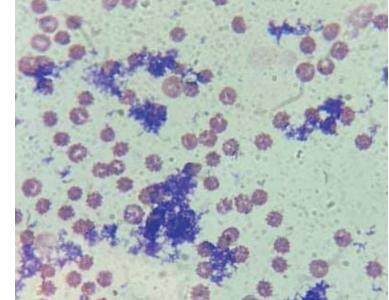
Schistocyte



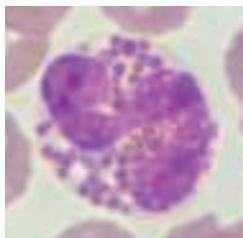
Band

Stain Precipitant

Purple stippling. Don't confuse with bacteria! Sepsis is bacteria INSIDE white blood cells or macrophages.

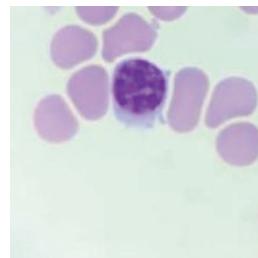


Distinguishing Features in Canine/Feline Hematology

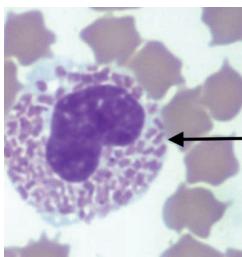


Canine eosinophil
Has round, pink granules

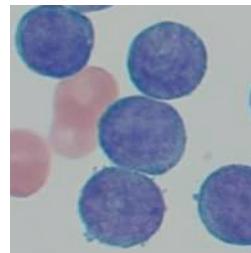
VetHelpfulNotes
Canine Feline



Lymphocyte:
slightly bigger than an RBC
and about the size of a
neutrophil



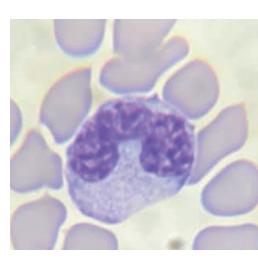
Feline eosinophil
Feline granules are
more bar like than
the canine
granules. Has a
segmented nucleus



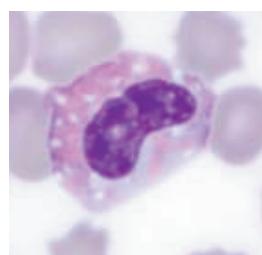
Lymphoblasts
Much larger
than an RBC. These are
almost twice the size of
an RBC



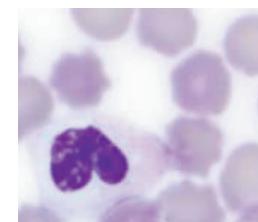
Grey eosinophil
Color variation seen
most often in Greyhounds
and sometimes Labs



Monocytes:
Distinguished by size: much
larger than an RBC and also
cytoplasm has bluish
cytoplasm. Imagine fitting
4-5 RBC into one monocyte



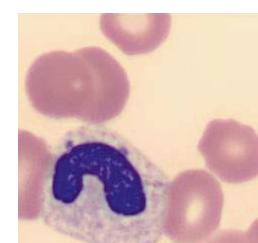
**Hyposegmented eosinophil:
Pelger Huet**
Distinguish from a monocyte by the
more distinct granules inside; should
see all neutrophils and eos as
hyposegmented. Common in certain
canine breeds



metamyelocyte
Immature neutrophil
Looks like a monocyte but is
smaller and does not have
bluish cytoplasm; not a band
which is more horseshoe
shaped. This is bean shaped



Normal Neutrophil
Has segmented nucleus
These are called polymorpho
nuclear cells (PMNs)



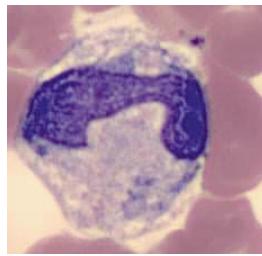
Band Neutrophil:
Nucleus is not segmented,
but is horseshoe shaped
Nucleus is indented 50%
all the way around



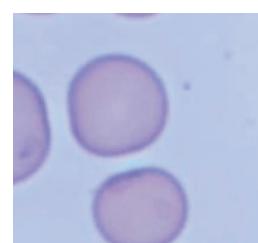
**Hypersegmented
Neutrophil:**
Not a signs of toxic
change, just a neutrophil
in circulation awhile



Normal canine RBC
Has central pallor. Feline
RBC often do not



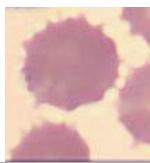
**Toxic Band
Neutrophil:**
Lacey, blue cytoplasm,
Dohle bodies



Normal feline RBC
Has no central pallor.

Poikilocyte cheat sheet

Echinocytes



More regularly spiculated
Secondary to artifact/aged blood snake bite,
bee stings, some drugs

Acanthocytes



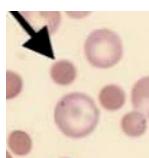
Irregularly spiculated, blunt tips
Alteration in lipid membrane or fragmentation
injury cancer (HSA) vasculitis, DIC, GI, liver,
various other causes.

Eccentrocyte



Eccentric central pallor represents
oxidation injury: acetaminophin, propofol
(cats), onions, zinc, mothballs

Spherocyte



Smaller, round RBCs with no central pallor.
Due to removal of membrane parts by
macrophages, most commonly IMHA

Keratocyte



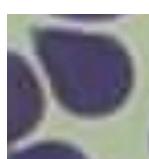
Bite or blister cells. Results from oxidative
injury to cell membrane

Schistocyte



RBC fragment from altered blood flow due to: Liver
shunt, changes to vessels, RBC fragility

Dacryocyte



RBC that is tear drop shaped. Thought to be due
to myelofibrosis in dogs

Target cell



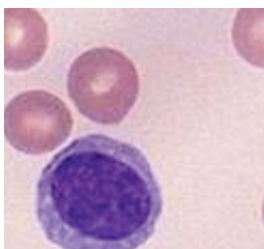
Only really observed in dogs. A clump of
hemoglobinized cytoplasm in the center of the cell.
Can be seen with anemias, liver disease and
hypothyroidism

Stomatocyte



A mouth like central area of pallor that can be
artifact or due to anemia, liver disease, lead
poisoning. Can be hereditary in Alaskan
Malamutes, Standard and Miniature Schnauzers

Nucleated Cells Canine/Feline



Lymphocyte 9-12 um

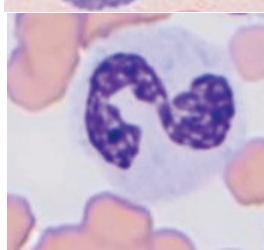
Light blue cytoplasm, eccentric, round nucleus.

Increasing in size

Slightly smaller than neutrophils. Larger than RBC's.

Cells of the immune system: B and T cells.

B cells produce antibodies, T cells release signals (cytokines) to attack invaders.



Neutrophil 12-15 um

Lobulated nucleus with pale pink or light blue cytoplasm.

Primary defense against microorganisms. Accumulate at the site of inflammation or infection. The most numerous of all the WBC's.

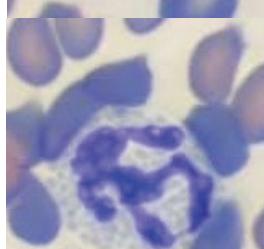


Eosinophil 12-20 um

Lobulated nucleus with orange-red granules in the cytoplasm.

The same size or slightly larger than a neutrophil.

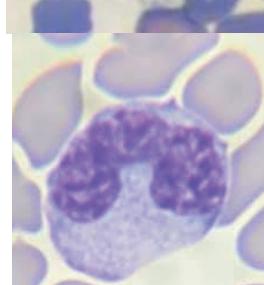
Canine granules are round, Feline granules look more bar-shaped. Function in hypersensitivity reactions- they are attracted to histamine release. They also play a large role in fighting parasitic infections (flukes and nematodes)



Basophil 12-20 um

Ribbon like folded nucleus with light purple/grey cytoplasm and discrete dark granules. About the same size as eosinophils, but eos have pinkish granules and less ribbon like lobulated nucleus.

They release histamine in hypersensitivity reactions. Rarest of all WBC. Hard to find!



Monocyte 15-20 um

Irregular lobulated nucleus with grey to grey-blue cytoplasm. The largest of the circulating WBC. Imagine fitting 4-5 RBC's inside a monocyte. They're big!

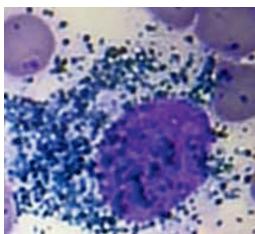
Circulate in the blood until they are needed in inflamed or infected tissues. Here they mature into macrophages which are capable of engulfing microorganisms or other cells.

Basophil. Purple granules, ribbon like nucleus

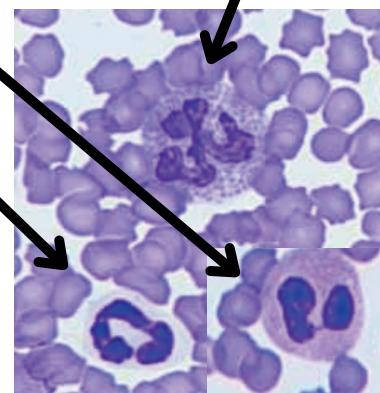
Eosinophil. Pink granules, lobulated nucleus

Neutrophil. No granules, lobulated nucleus

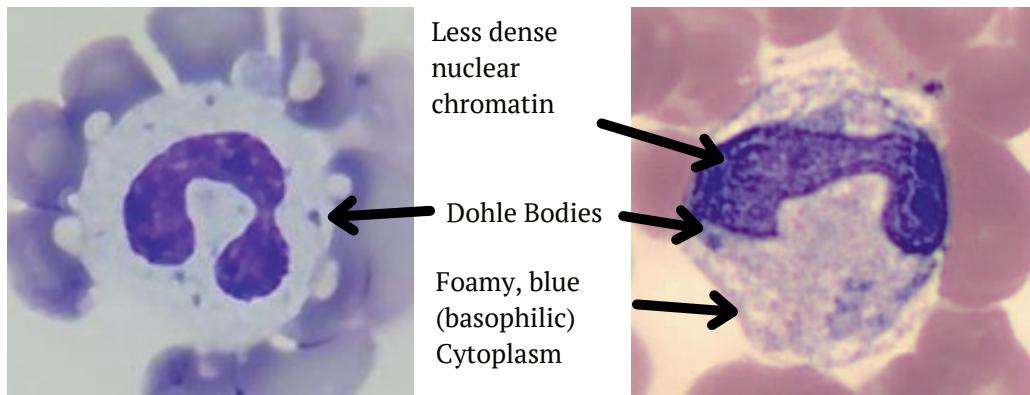
Mast cell ~10 um



Round nucleus, dark purple granules. Mast cells can occasionally be seen in a blood film of a normal dog. However, any circulating mast cells (typically noted on the feathered edge) in a cat is abnormal! Most often due to splenic or GI splenic tumors!



Toxic Changes to Neutrophils



Toxic Changes:

1. Streaky blue cytoplasm (cytoplasmic basophilia)
2. Dohle bodies
3. Foamy cytoplasm (cytoplasmic vacuolization)
4. Less dense nuclear chromatin

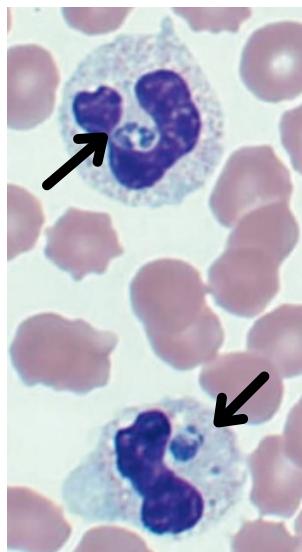
Toxic Neutrophil examples. The left WBC is not a band because the nucleus does not have parallel sides and the nucleus indented at least a third of the way on one side.

Toxic changes seen: foamy cytoplasm and lots of little dark punctate regions called Dohle bodies are present. Dohle bodies are pieces of ribosome and endoplasmic reticulum.

Toxic changes are graded 1+ to 3 or 4+ and is based on the presence and number of changes within the neutrophils that indicated more rapid maturation (signaled by cytokines) to meet increased demand (inflammation, infection). Unlike the name implies, toxicity is not damage to the WBC from bacteria.

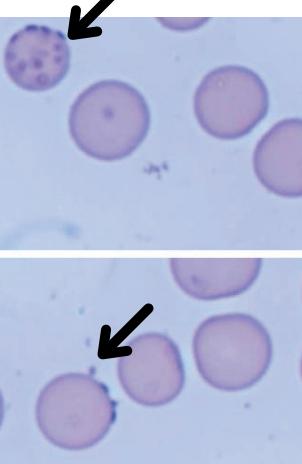
Toxicity is changes within the cell that occur during demand for more rapid maturation.

Hematologic Parasites



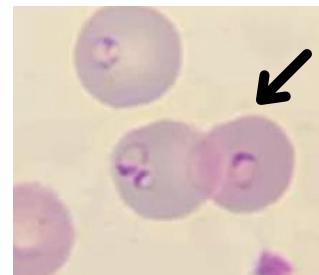
Anaplasma phagocytophilum or **Erhlichia ewingii** morulae within neutrophils.

Look the same cytologically. Must use PCR or SNAP test to distinguish. These are bacterial infections spread via ticks. Can see morulae before the snap is positive



Mycoplasma haemophilus:

Epicellular bacterial parasite of cat RBC's. Causes hemolytic anemia and is spread by fleas

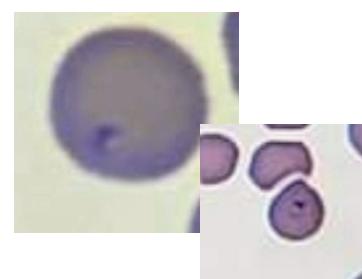


Babeisa piroplasm within RBC's.

Tick transmitted protozoal parasite. Causes hemolytic anemia in dogs



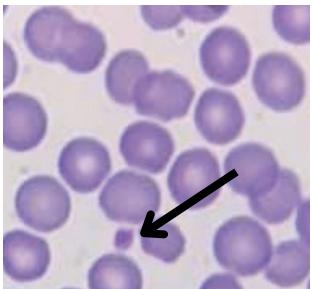
Distemper virus inclusion. Very very rare to find. Large aggregates of viral particles can be found in RBC's.



Cyttauxzoon felis:

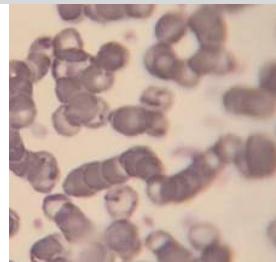
Often fatal protozoal parasite of cat RBC's. Transmitted by ticks. Looks like Babesia gibsoni, but Babesia gibsoni does not affect cats.

Stuff about RBC's -n-Platelets



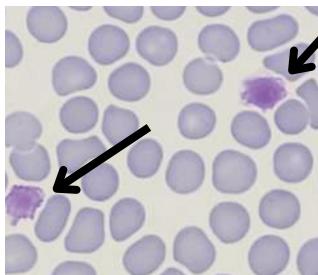
Normal platelets

Considerably smaller than RBC's



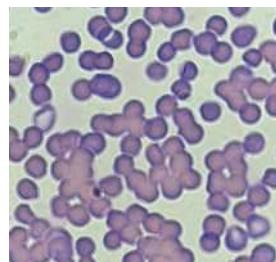
Rouleaux

Normal stacking. RBC's line up like coins. This is not agglutination



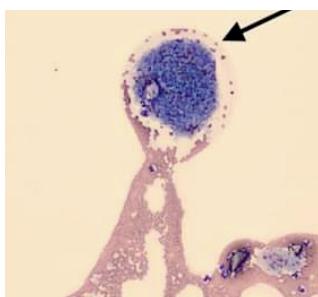
Macroplatelets

Approaching the size of RBCs. Keep in mind Cavalier King Charles Spaniels normally have macroplatelets and decreased platelet numbers (often 60-100K)



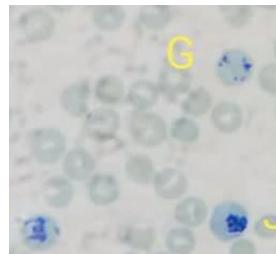
RBC agglutination

Hallmark sign of IMHA on a saline agglutination test. Cells coated in antibodies clump together and are removed from circulation.



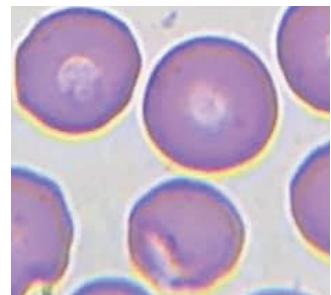
Platelet clump

Common in cats at feathered edge, low resolution example. Analyzers frequently count low numbers of platelets in cats. Look for clumps.



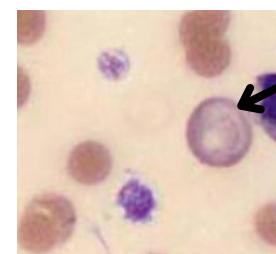
Aggregate and punctate reticulocytes:

Stained with new methylene blue. They are larger than the surrounding RBC's RNA precipitates as chunks or aggregates. They indicate a regenerative response. Correspond to polychromatophil on a routine blood film.



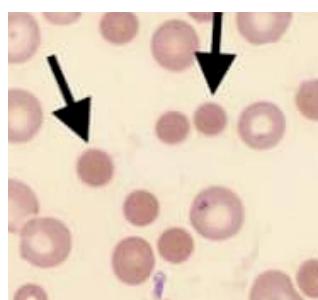
Normal canine RBC

Has central pallor. Feline RBC often do not. This is why you cannot identify spherocytosis in cats.



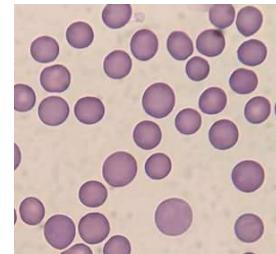
Polychromatophils

Are reticulocytes not stained with new methylene blue. They are larger and darker than normal RBC's



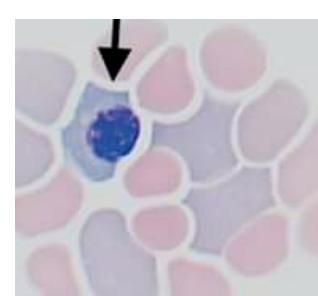
Spherocytes

Very small and round, no central pallor. Hallmark of IMHA. Spherocytes indicate piece of cell membrane were removed from damage. Can't see spherocytes in cats due to their lack of normal central pallor



Anisocytosis in a blood film:

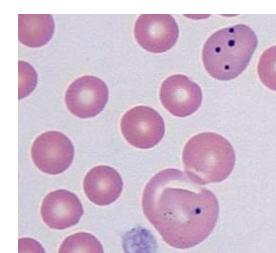
Variation in cell size seen as a regenerative response to anemia. Immature RBCs are larger than mature RBCs.



Nucleated RBC

Low numbers can be seen in dogs, or in strongly regen anemia. Analyzer often calls them lymphocytes because of the retained nucleus. Must correct for NRBC on your manual diff.

TIP: NRBC's with apoptotic eosinophils/ leukocytes= heatstroke



Howel Jolly Body

Nuclear remnants. More common in splenectomized animals. Can be normal in healthy cats.

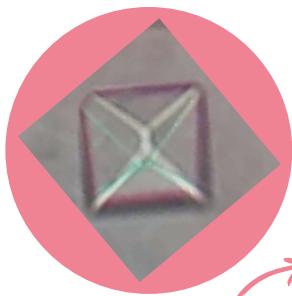


Heinz bodies

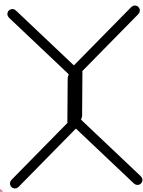
Represent oxidative injury to hemoglobin in RBC's. Visualized best with new methylene blue stain. Hemoglobin of cats is more susceptible to oxidant injury than other species. Well known with onion toxicity and repeated propofol use in cats.

Canine and Feline Urinalysis

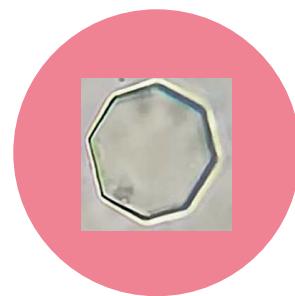
Struvite: Magnesium Ammonium Phosphate



Struvite
Looks like an envelope , not
like a cross (how to
distinguish from Calcium
Ox Dihydrate)



Struvite
Classic shape is
coffin shaped



Struvite
can sometimes have many
sides. Distinguish from
cystine which always has 6
sides

Struvite crystals are also called **magnesium ammonium phosphate** or **triple phosphate crystals**. They are the most common crystal seen in canine and feline urine and can be seen in normal urine

Struvite stones in dogs are formed by bacteria that split urea into ammonium and phosphate and increase the urine pH. PEARL: Dogs almost always produce struvite stones from a urease-positive bacterial infection. Management is via elimination of the infection and a dissolution diet that acidifies the urine. Once the stone is dissolved and the UTI cleared, dogs do not need to remain on the urinary diet. Cats, however, form sterile struvite stones and benefit from lifetime urinary diet management to maintain optimum pH and reduce ash (mineral matter) in the diet.

Calcium Oxalate: Dihydrate and Monohydrate

Calcium Oxalate Dihydrate



**Calcium oxalate
dihydrate**
Squares with
intersecting lines. Most
common form

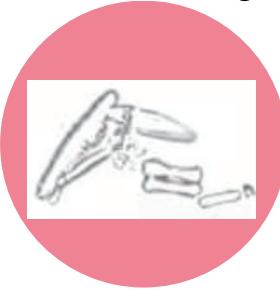
Calcium Oxalate Monohydrate



**Calcium Oxalate
monohydrate**
Dumb-bell shape



**Calcium Oxalate
monohydrate
hemp seed variety**
not very common



**Calcium Oxalate
monohydrate
Picket fence form** most
commonly seen with
Ethylene glycol toxicity

Super saturation of the urine with calcium can cause calcium oxalate crystals or stones.

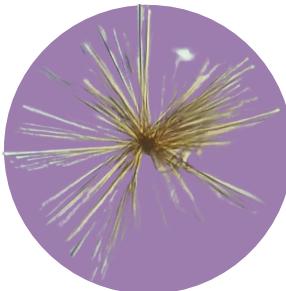
However, these crystals can form in normal urine and in a range of urine pH's. Calcium oxalate crystals can also form over time in stored urine. Some animals with hypercalcemia (hyperparathyroidism, lymphoma) or animals with increased calcium excretion from drugs (steroids, furosemide) may be predisposed to developing calcium oxalate crystals and stones. Miniature Schnauzers are pre-disposed to calcium oxalate stone formation. Picket fence shaped Calcium oxalate monohydrate crystals with acute kidney failure is suspicious for ethylene glycol toxicity.

Canine and Feline Urinalysis

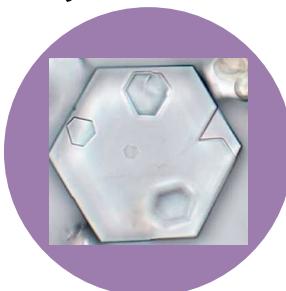
Ammonium Biurate



Bilirubin



Cystine



Calcium Phosphate



Ammonium Biurate or **Ammonium Urate** appear as yellowy-brown irregularly shaped crystals that have little spiky projections. They are sometimes referred to as thorny apples. Rarely, if ever, seen in normal dogs or cats of non-predisposed breeds (Dalmations, English Bulldogs and Black Russian Terriers that have a genetic tendency to produce excess uric acid. Animals with liver disease also can produce increased renal excretion of ammonium urate. **PEARL:** If you see these crystals in non predisposed breeds, check their liver enzymes. Urate stones can be dissolved on a purine (amino acid) restricted, alkalinizing diet like U/D. **PEARL:** 60% of all urate stones are in Dalmations.

Bilirubin crystals are most often seen in canine urine. It's not uncommon for there to be some bilirubin in normal canine urine (most often very concentrated urine). In dogs, they are most often of no clinical concern. **PEARL:** *Bilirubin crystals in feline urine is abnormal and liver disease should be suspected and investigated.*

Cystine crystals form in animals with a genetic dysfunction in renal tubular reabsorption of cystine and other amino acids. This genetic disease does not affect kidney function, but predisposes to cystine stone formation. Cystine crystals are flat and hexagonal with 6 sides. Do not confuse with the struvite crystals that can also form flat crystals- these crystals have more than 6 sides.

Calcium phosphate crystals form in neutral or alkaline urine. Uncommon to see. Can happen in animals with excessive dietary calcium and phosphorous, animals with metabolic disorders that promote high calcium such as hyperparathyroidism.

Common Crystals

- Struvite
- Bilirubin
- Calcium Carbonate
- Calcium Oxalate dihydrate
- Calcium Oxalate monohydrate (dumbbell and hemp seed forms)

Uncommon Crystals

- Calcium Oxalate monohydrate (picket fence forms)
- Ammonium biurate
- Cystine

Canine and Feline Urinalysis



Urine DipStick

Urinalysis starts with evaluating a urine dipstick. **PEARL:** the test pads for urine specific gravity, urobilinogen, nitrate and leukocytes are not useful in veterinary patients!

pH: normal urine pH is between 6-7.5.

Alkaline urine (over 7.5) may result from urinary infections with urease producing bacteria. It's a good idea to investigate alkaline urine samples for evidence of infection (white blood cells, bacteria).

Protein: There should be little to no protein present in normal urine. The filter unit of the kidney, the glomerulus, should not filter large plasma proteins such as albumin and globulin. A negative test is reliable, but there can be false positive reactions on the test pad.

Persistent positive protein in urine can be assessed by measuring the urine protein concentration with a urine protein: creatinine ratio. Healthy dogs should have a UPC <0.5 and healthy cats < 0.4.

Blood: This test pad detects heme containing substances such as lysed red blood cells, or myoglobin from muscle damage. Corroborate with a urine sediment exam.

Bilirubin: Can be normal to see in canine urine. Dogs have a low renal threshold so bilirubin can be spilled into the urine from hemoglobin breakdown. *Bilirubin is always abnormal in cats!*

Glucose: Not normally present in healthy urine. Can increase with diabetes mellitus and stress hyperglycemia

Ketones: Normally not detected in healthy urine. Test pad most sensitive to only a few ketone bodies. Indicates excess fat metabolism and is most often found in unhealthy diabetic patients.

Urine Sediment

A urine sediment exam is best performed as both a dry mount and a wet mount preparation.

Wet mount: 5-10 ml spun at 1500 rpm for 5 minutes. Remove most of the supernatant, leaving 2-3 drops of supernatant to mix with the sediment plug. Gently tap the tube with your finger to re-suspend the contents. Transfer one drop with a disposable pipette onto a slide and place a coverslip. A drop of stain may be used to improve nuclear detail and improve identification, but stains may also add contaminants and dilute the sample. *PEARL: wet mounts are best to identify casts and crystals.*

Dry mount: samples can be prepared the same way, but allowing the sample to dry completely (air dry or using a cool hair dryer). Some people like to take a cotton tipped applicator to swab the bottom of the tube to roll onto the slide. Another popular method is to push the tiny drop of sediment halfway across the slide with another slide to create a line (don't aim to smear like a blood film-- once dry, the line has a good number of cells to examine).

PEARL : dry mounts are best for identifying bacteria and cells. Having trouble with sediment washing off your slide? Try using serum to coat your slide and let it dry. Once dry, apply the suspended sediment. This encourages material to stick to the slide better. Any serum can be used. Slides can be coated and kept in the freezer for future use.

Canine and Feline Urinalysis

Urine Sediment

Red Blood Cells

0-5/HPF

Healthy urine can have up to 5 RBC's per HPF. RBC's look like donuts on wet mount. They are almost half the size of WBC's.

Smaller cells are RBC's
They are 1.5- 2 smaller than WBC's



Larger cell is a neutrophil. They look more rounded on a wet mount

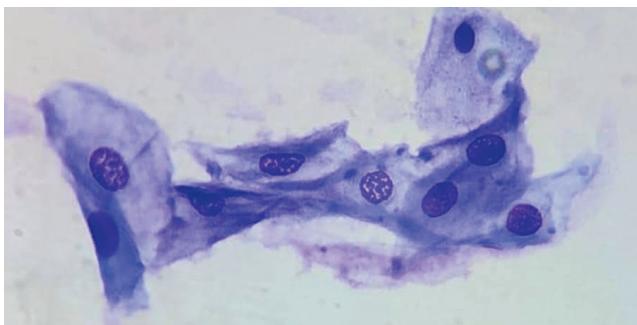
White Blood Cells

0-5/HPF

Healthy urine can have up to 5 WBC's per HPF. WBC's are much bigger than RBC's (1.5-2x the size of RBC's). Pyuria= 6-10 neutrophils per HPF.

Squamous Cells

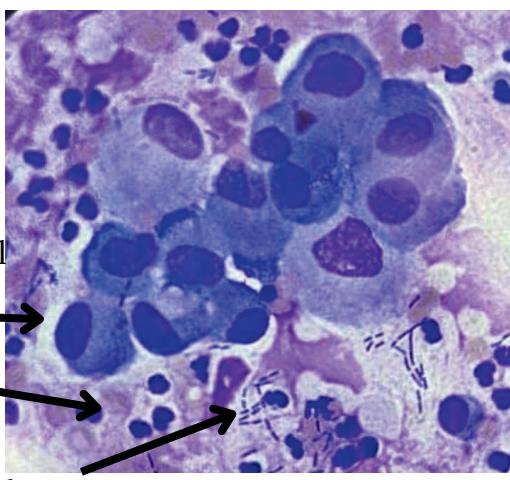
Low numbers of epithelial cells can be seen in healthy urine. More can be seen with urine obtained via catheterization as cells can slough. Increased numbers due to inflammation from infection as well. They line the distal urethra and genital tract of females.



Flat, with irregular borders and a smaller central nucleus

Transitional Cells

Transitional cells line the bladder, renal pelvis, ureters and urethra. Increased numbers can be seen with bladder inflammation



Transitional cells

Neutrophil

Rod bacteria

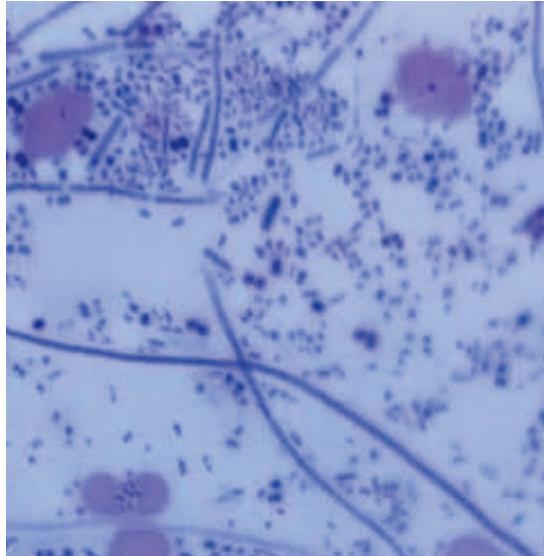
Transitional cell clusters look more rounded with larger nuclei. Here you can compare their size to the surrounding neutrophils. They are substantially larger than a neutrophil. Rod bacteria are also present in this sample.

PEARL: if you are unsure of the significance of clusters of transitional cells you can submit a BRAF test on the urine to detect a molecular marker of transitional cell carcinoma. Imaging of the bladder with ultrasound is also recommended.

Canine and Feline Urinalysis

Urine Sediment

Bacteria



Both rod bacteria (present in long chains that look like ropes) and cocci in clusters

The puffy purple blobs are degenerate neutrophils

PEARL: brownian motion causes particles suspended in liquid to appear to move as the particles undergo motion as they spread out. Movement does not mean bacteria!! Cocci don't move because they don't have flagella. The movement is physics.

Casts



Casts are tubular structures composed of mucoproteins secreted by cells lining the renal tubules and collecting ducts.

Low numbers (0-1/LPF) can be seen in normal urine.

Casts can be described as cellular, granular and waxy. It is presumed that these represent different stages of time the cast was present in the tubule before it was shed in the urine.

Casts in normal animals are generally few to none and typically are hyaline or granular.

Cellular casts indicate acute tubular injury. They are composed of many cellular pieces within the tubular structure.

EVALUATION FOR BACTERIAL OR YEAST OTITIS:

1. Insert cotton-tipped applicator into otic canal and gently rotate remove debris. Samples should be collected from each ear. Roll sample onto a glass slide.

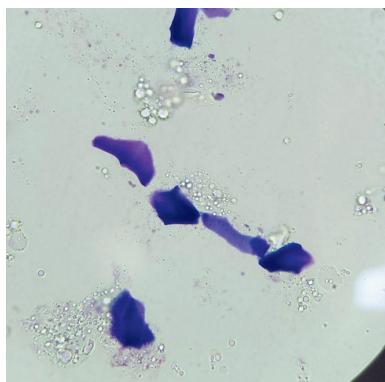
A time saving tip:

Roll samples from each ear onto the same slide, so you only have to dip ONE slide. By convention it's easiest to put the left sample on the left side and the right sample on the right side. 2. Samples do NOT need to be heat fixed

2. Stain slide with Diff-Quik and let air dry

3. Scan slide on 10X objective lens to find representative areas. Then move up to the 100X objective lens. Yeast and bacteria are often found near or on top of clumps of keratinocytes.

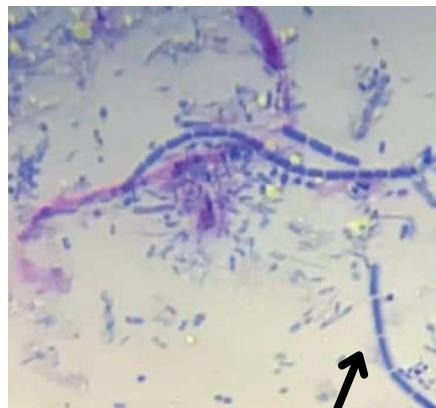
4. Examine 5-10 areas to estimate the numbers of infectious organisms
0-2 yeast/40X field and 0-4 or 5 cocci/40X can be normal



Normal ear swab. Normal keratinocytes, beaded oily/waxy debris. No infectious organisms present here.

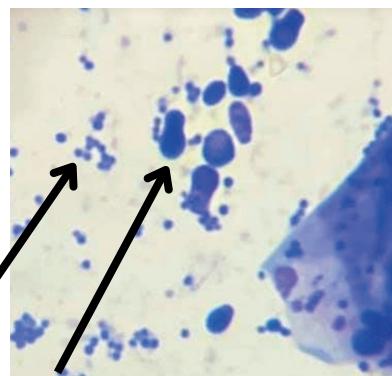


Typical yeast infection. There may be a few neutrophils present (or none). On this slide there is one keratinocyte in the middle of the field.



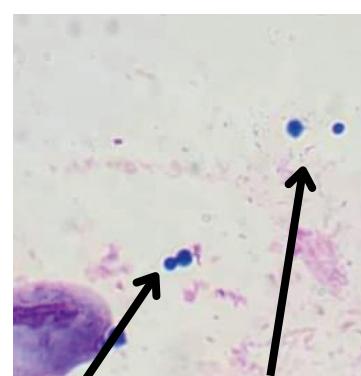
Cocci and rods present.
Rods can form long chains

Vet Helpful Notes
Canine Feline

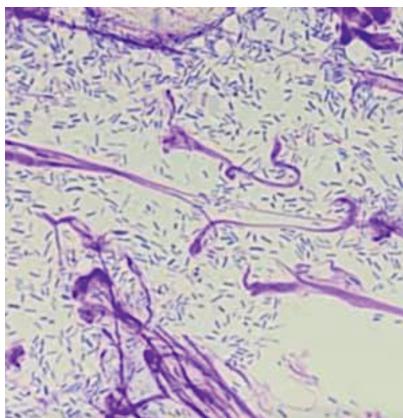


Diplococci

Yeast look like foot prints
Cocci look like purple dots
Certain strains of bacterial cocci join together in two's and are called diplococci



Budded yeast and non-budded yeast (budding is the process of yeast growth)



Numerous rods
(TNTC = too numerous to count)
Streaming neutrophils- ribbons of nuclear material from broken neutrophils

Rod bacterial infections are most commonly from pseudomonas infections.
They often look like yellow pus in the ears.

Typically there is a heavy amount of white blood cells (neutrophils) present which can either look like intact cells or just strands of nuclear material from broken cells.



EVALUATION FOR OTODECTES (EAR MITES):

1. Use cotton-tipped applicator to gently remove large amount of debris from otic canal. Roll onto glass slide. Add a few drops of mineral oil and then place coverslip.
2. Scan entire slide with 4X or 10X lens for mites or eggs.



On lower power you can easily identify ear mites (*Otodectes*). They look like they have suction cups on their legs.

These mites feed on the skin and debris lining the ear canal and are highly transmissible by close casual contact between pets.

Treatment Cats: Milbemite, Advantage Multi, Revolution, Feline frontline, Feline Bravecto

Treatment Dogs: Revolution, Simparica, Bravecto, Nexgard

Vet Helpful Notes
Canine Feline

Otics

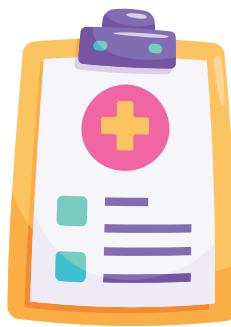
Dexamethasone
Betamethasone
Mometasone } similar potency

Triamcinolone
Prednisolone
Hydrocortisone

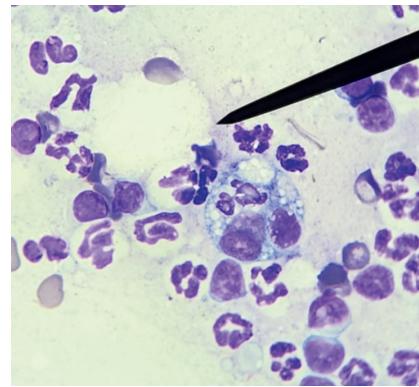
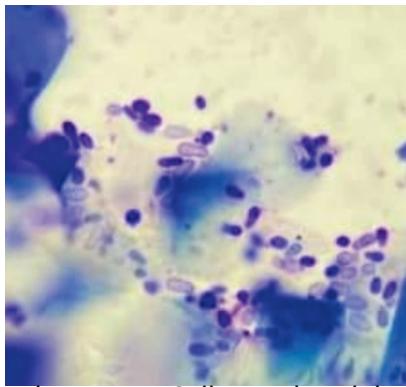
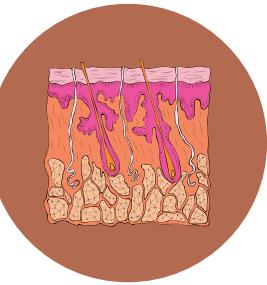


Brand Name	Manufacturer	Steroid	Antibiotic	Antifungal
Mometamax	Merck	Mometasone 1mg/gm	Gentamicin	Clotrimazole
Otamax	Merck	Betamethasone valerate	Gentamicin	Clotrimazole
Mozotic	Patterson	Mometasone 1mg/gm	Gentamicin	Clotrimazole
Easotic	Virbac	Hydrocortisone	Gentamicin	Miconazole
GentaOtic	Butler Schein	Betamethasone	Gentamicin	None
Animax	Dechra	Triamcinolone	Neomycin	Nystatin
Tresaderm	Merial	Dexamethasone 1mg/ml	Neomycin	Thiabendazole
Baytril Otic	Bayer	NONE	enrofloxacin	Silver sulfadiazine
Posatex	Merck	Mometasone	orbifloxacin	Posaconazole
Surolan	Vetoquinol	Prednisolone	PolymyxinB	Miconazole
Zymox HC	Pet King	Hydrocortisone 1%	Enzymes; Lysozyme, lactoferrin, lactoperoxidase	NONE
Claro	Bayer	Mometasone	Florfenicol	Terbinafine
Osnuria	Elanco	Betamethasone	Florfenicol	Terbinafine

Ear Cleaners

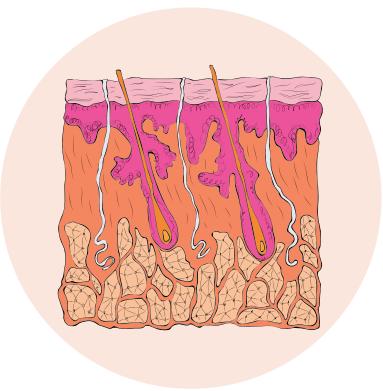


Type of Ear cleaner	Indications	Concerns	Products
Drying agents	Cleansing, drying. For prophylaxis and to deodorize	May sting due to alcohol content Avoid in ulcerate, inflamed ears (such as ears with rod bacterial infections)	CleaRxTreatment Dryer • MalAcetic Otic • Otic Clear • Otic-Clens • OtiRinse Ear Solution • Oti-Soothe Ear Cleansing Solution • UltraOtic Rinse
Antiseptics	Antimicrobial for Yeast and bacteria. Triz EDTA is useful for pseudomonas to de-stabilize the cell wall which allows medication to penetrate	High concentrations of chlorhexidine is contraindicated with ruptured ear drums (ototoxicity) Triz EDTA is a more basic solution which can predispose to yeast overgrowth. Later formulations added Keto for this reason.	TrizEDTA Aqueous and Crystals Flush • TrizEDTA+Keto • MalAcetic Otic • Otocetin Solution • Otic Clear
Cerumenolytics	Ceruminolytic (helps dissolve ear wax)	May predispose to yeast overgrowth	Cerumene • Epiklean Ear Cleanser • KlearOtic • Douxo Micellar
Combination products	Drying and disinfection. Effective for mildly waxy/inflamed ears. Can be used as a preventative for recurrent ear infections	Alcohol in duoxo micellar may be stinging to irritated ears	Epi-Otic Advanced • Douxo Micellar



Yeast dermatitis. Collect with a slide impression or using clear acetate tape if skin is dry. Typically you only find a few yeast on a skin slide. Finding even one or two yeast on a skin slide diagnoses yeast dermatitis. However just a few yeast on an ear cytology can be normal.

Inflamed skin will have lots of neutrophils. The longer inflammation is present, macrophages will be recruited. This foamy macrophage is eating a neutrophil.

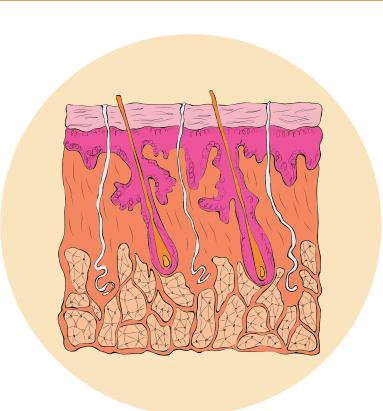


Scabies:
This is a very difficult mite to find on skin scrape. It can take multiple attempts

Scabies Mites: these mites are zoonotic which means that they can infect humans too! If you suspect scabies, always ask your client if they are experiencing any itching too. This mite infestation is very very itchy. It often causes redness and crusting lesions on the skin. Common locations are the ear margins, lateral elbows and hocks.

The pinnal-pedal reflex was coined for this infection. When you rub the ear margin, infected dogs instinctually often start scratching with their hind limbs.

Scabies has a host preference for dogs, foxes, coyotes and wolves. It rarely causes infection in cats. Fox and Coyote populations may serve as reservoirs for this parasite. Treatment: revolution, advantage multi and off-label use of the isoxazoline class of drugs.



Demodex:
Cigar shaped mite

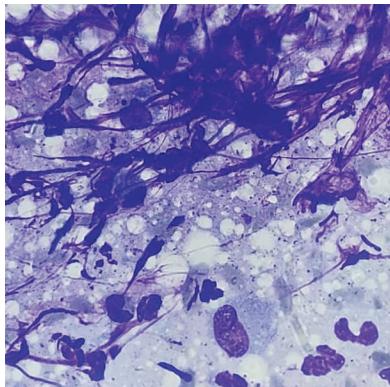
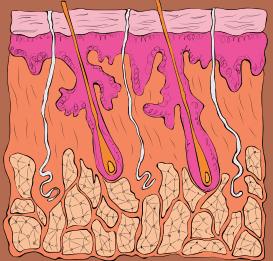
Demodex mite. This can be found by skin scrape. It can be helpful to squeeze the skin as they burrow around hair follicles. Hair plucking can also be used to find these organisms.

Canine demodex is considered to be caused by a genetic or immune disorder that allows mites present normally on the skin in very low number to proliferate on the skin.

Puppies can develop demodex due to their immature immune system. Adults that are immunocompromised may also develop demodicosis.

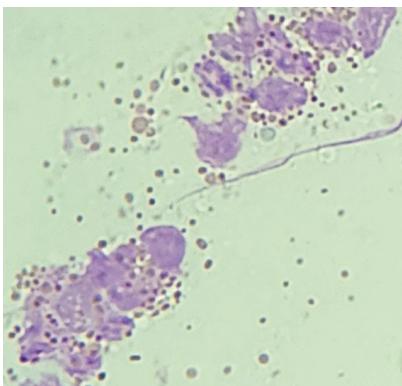
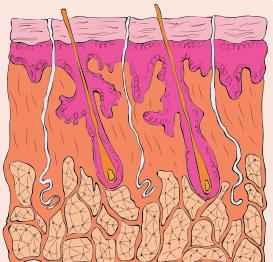
Localized lesions can spontaneously regress within 6-8 weeks. Smaller lesions can be treated with benzoyl peroxide shampoo or gel.

Mitacidal drugs like the isoxazoline classes of drugs (simparica, nexgard and bravecto) and advantage multi can be used off label to treat demodex.

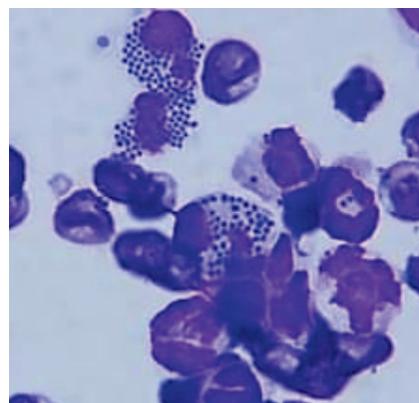
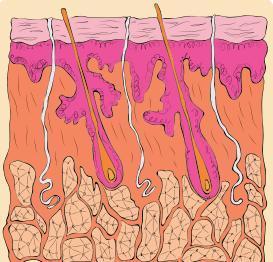


Neutrophils are the most common white blood cell seen on cytology. It's common to see them broken on the slide. The long purple tendrils are the broken nuclei. These are called streaming neutrophils.

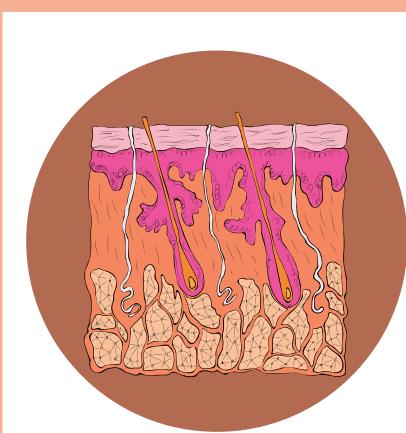
Vet Helpful Notes
Canine ♥ Feline



Eosinophilic dermatitis.
Eosinophils look like neutrophils with pink granules. Eosinophils are an allergy cell and they can sometimes also be seen on cytology of skin lesions/rashes



When you see neutrophils eating up cocci, that indicates that there is a bacterial skin infection present



Vet Helpful Notes
Canine & Feline notes

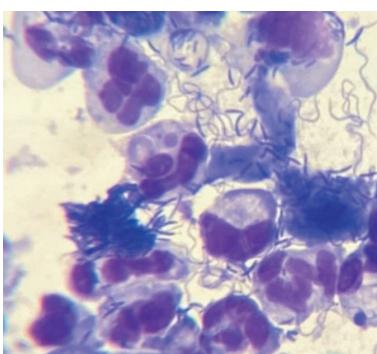
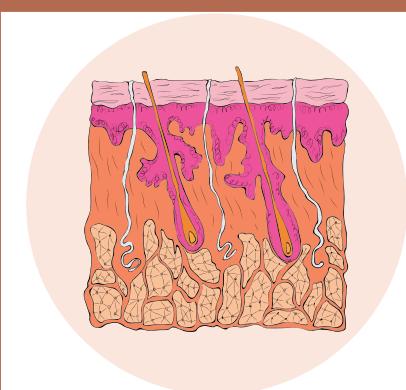
Incidental findings



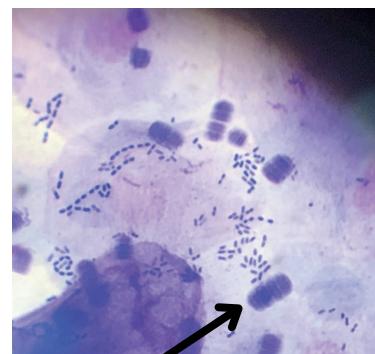
Curvularia soil fungus.
They look like croissants!



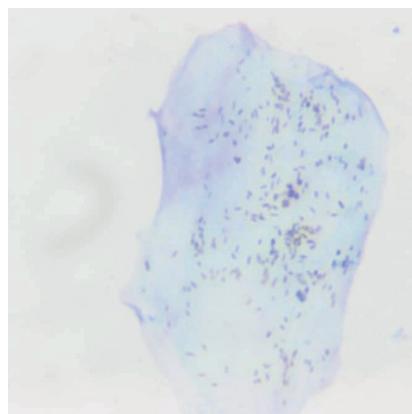
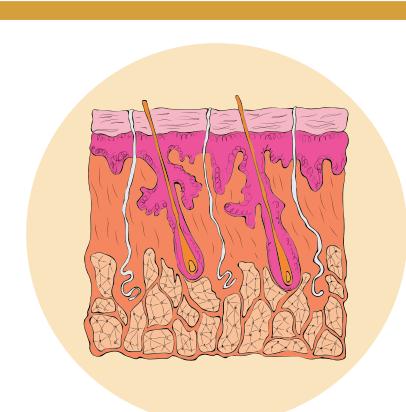
Alternaria a common
soil and plant fungus.
They have alternating
septae and a club!



Oral spirochetes commonly
found around the mouth in
cats. (part of the oral
bacterial flora)

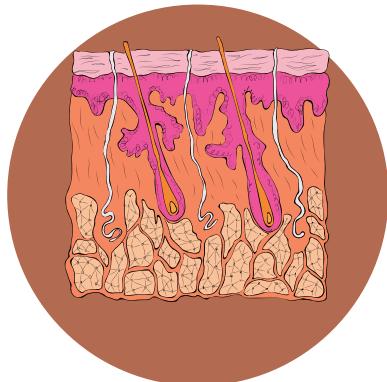


Conchiformibius (formerly
simonsiella) are large stacks of rods
that look like fat cigars and are
normally part of the oral flora. They
indicate licking of the skin.



Melanin granules
on epithelial cell.
These are brown
pigmented spots, not
stained blue cocci

Dermatophytosis



M.canis

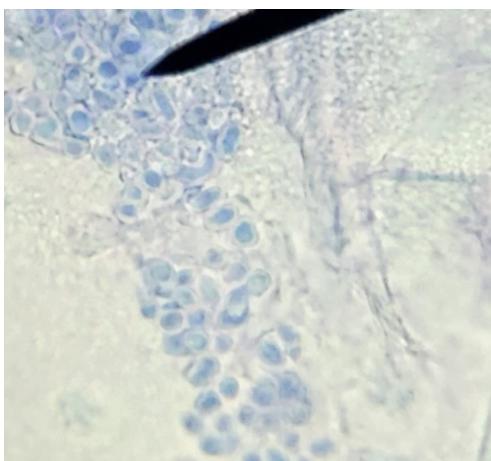
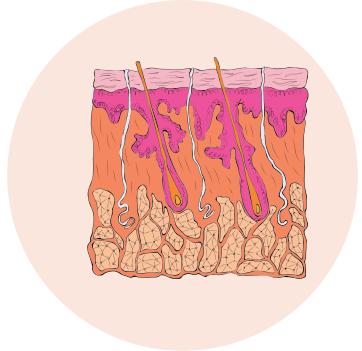
The top three dermatophytes seen in dogs and cats are:

1. Microsporum canis- by far the most common: can *fluoresce*
2. Microsporum gypseum : does not fluoresce
3. Trichophyton mentagrophytes: generally will not fluoresce

Infection via contact with infected hair/scale, infected animals, soil or fomites

Microsporum canis is the most common cause of ringworm in dogs and cats. Infected hairs can fluoresce with a woods lamp. Pluck hair from around a lesion and place on a glass slide. Add a drop of mineral oil and place a cover slip. View on 10X objective. Infected hairs are wider than normal hairs due to spores coating the shafts. M canis produces "cuffs" of spores on the outside of infected hairs indicating ectothrix invasion.

Dermatophytosis

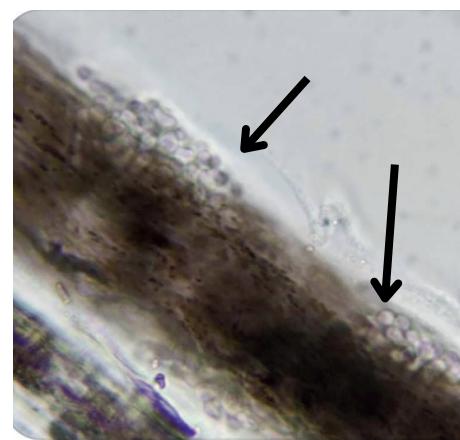
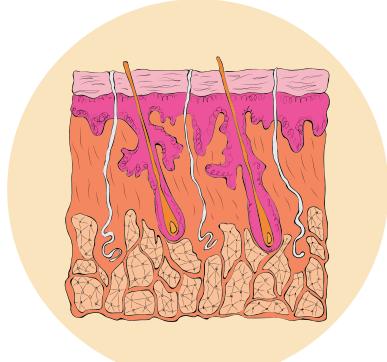


Tape prep cytology from the ear of a cat with ringworm. These are ringworm arthroconidia. (Purple spores with a clear zone around it).

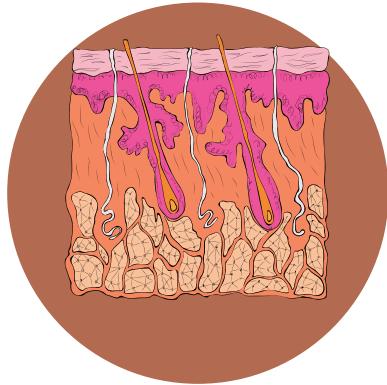
VetHelpfulNotes
Canine Feline

Dermatophytosis

Hold the woods lamp within 2-4 cm from the skin and move slowly to examine the hair shafts. Only hairs, not crusts, should glow apple green.



Trichogram of microsporum canis shows: cuffs of arthrospores seen on hair cuticle (ectothrix invasion)

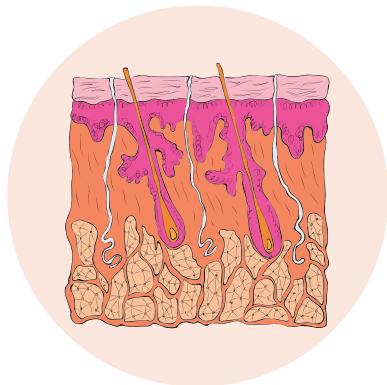


Fungal Kerion

Dermatophytosis



Fungal kerions are raised, fleshy round lesions. They are typically from *T. Mentagrophytes* or *M. gypseum*. Dogs often get fungal kerions on their muzzle or feet from digging in contaminated soil. If there is a small break in the skin, there is an increased risk for infection. Because these dermatophytes infections are deep under the dermis, routine testing of hairshafts, woods lamp review and fungal culture may be negative. These are often diagnosed via biopsy.

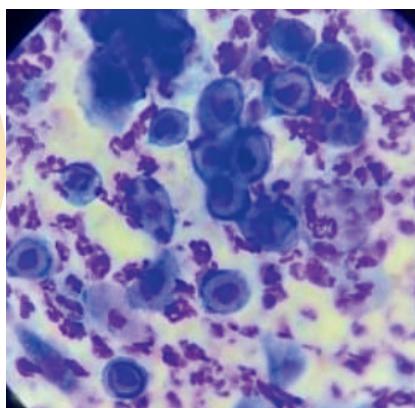
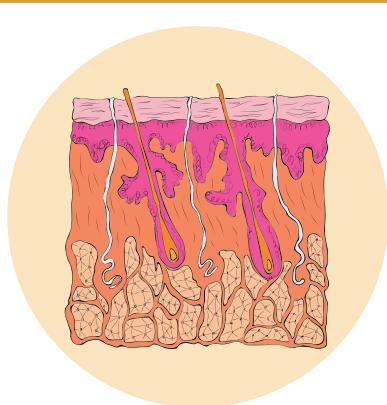


Auto-immune skin disease

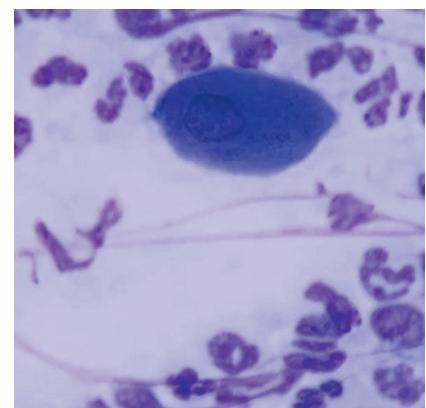
Pemphigus



Pemphigus is an immune mediated skin disorder of the attachments between keratinocytes on the surface of the skin. This leads to pustules and crusts that lift up on the skin. Cytology can help diagnose the disease by looking for acantholytic keratinocytes. These are "floating" keratinocytes that are rounded up because they have lost their attachments to other skin cells. They often appear quite large in comparison to a background of neutrophils. Common location for skin changes are the nasal planum, dorsal muzzle, pinnae, periocular skin and paw pads.

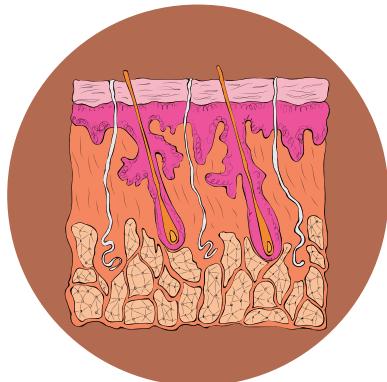


Acantholytic keratinocytes are rounded, nucleated, skin cells that have lost their attachments to each other. This is due to immune mediated attack on these attachment proteins.



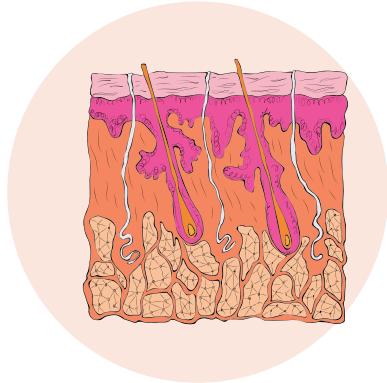
Cytology is taken from a pustule or under a crust.

Pemphigus



Pemphigus in cats : crusting and scaling is often located around the claws, nasal planum and the pinnae.

Mycobacterium: rare bacterial skin infection

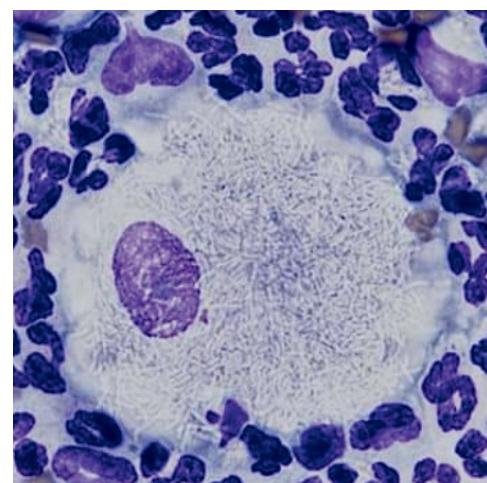
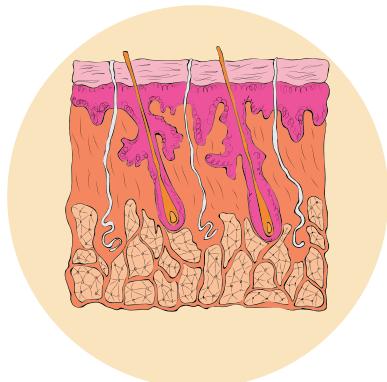


Mycobacterium

Mycobacterial infections are very rare infections caused by gram positive bacilli and can cause nodular and draining cutaneous lesions.



Mycobacterium: rare bacterial skin infection



They have a characteristic "clear" rods under the microscope.
Here you see numerous clear rods inside a macrophage.
Certain strains of mycobacteria are causes of leprosy and tuberculosis.
Some strains take a very long time to culture.

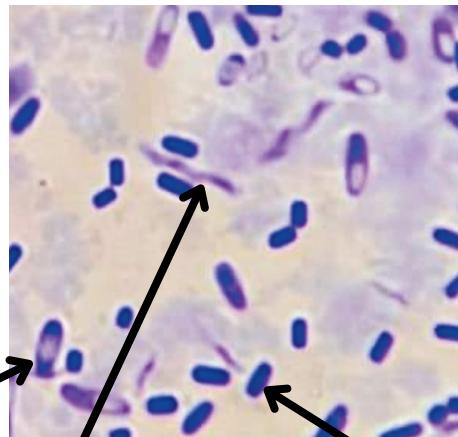


Fecal cytology is easily performed in the clinic and can compliment fecal flotation.

A dry prep is made by rolling some fecal material and fecal mucus on a slide. Fecal cytology can't identify fecal pathogen overgrowth, because relative numbers of organisms might be reduced making one population look more abundant. Also bacterial numbers might look more or less prevalent depending on thin or thick preparations. However fecal cytology can be used to look for spirochete bacteria that may indicate campylobacter infection and to screen for giardia or



Clostridium look like paperclips

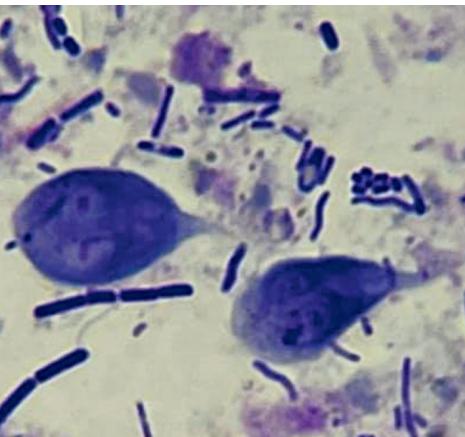


Campylobacter look like seagulls or squiggles

Fecal cytology can identify two fecal bacterial pathogens by shape: clostridium and campylobacter. Clostridium can be found in patients with and without diarrhea.

However, the presence of Campylobacter organisms is a significant finding, especially if the patient has diarrhea.

Typcial rod bacteria



Giardia is a single celled protozoal organism. It has a characteristic tear drop shape with a "monkey face". Giardia move with a jerky pattern under the microscope. It is most likely to cause diarrhea in young animals with an immature immune system. Giardia is a zoonotic disease, but canine/feline strains rarely cause human disease.

A similar looking organism is more rare and is called Tritrichomonas. It has a "falling leaf" style of movement. It is found most often in cats.



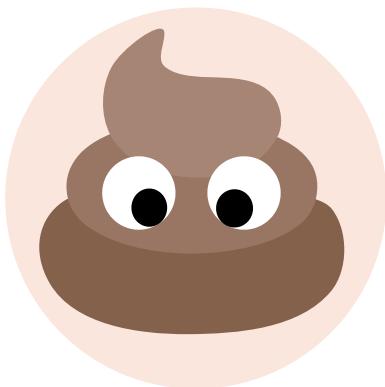
Incidental findings



Vet Helpful Notes
Canine & Feline

Grain mite. Grain mites can be found in canine feces when dogs are fed food that may have not been stored properly to allow grain mites to develop in the food. Grain mites are not a pathogen of dogs, but a "pass through" organism.

Eimeria look like isospora, but have a micropyle cap (plug at the top). They are rabbit coccidia. Eimeria are also "pass through" organisms. They often signify that dogs are consuming rabbit feces.



Incidental findings

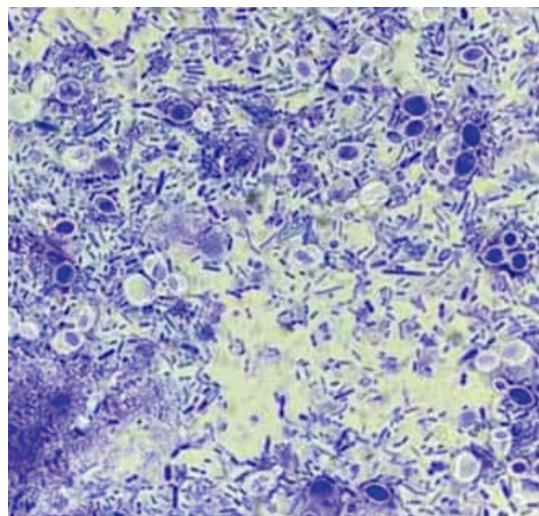


Cynicloomyces gluttanatus

Presence of these yeast organisms are common findings and likely are not pathogens in dogs. They are also a "pass through" organism of dogs and are the result of rabbit feces ingestion.



Incidental findings



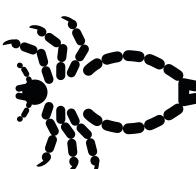
Incidental yeast

Yeast: basophilic staining round organisms with a clear capsule. Mostly considered benign and an "incidental finding" in fecal cytology.

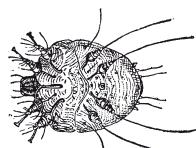
ECTOPARASITES : CANINE/FELINE

LESION DISTRIBUTION	CLINICAL SIGNS	DX	TREATMENT OPTIONS	GOOD TO KNOW
FLEAS: <i>CTENOCEPHALEDES FELIS, CTENOCEPHALEDES CANIS</i>				
Caudal dorsum caudomedial thighs ventral abdomen 	Pruritus, variable depending on how allergic the pet is to flea saliva. can cause: miliary dermatitis (multifocal crusted papules), erythema and local hairloss	Flea comb, wet paper test characteristic caudo-dorsum distribution of erythema and papular crusting and pruritus	Treat the environment. treat the pet with an adulticide and an IGR: example: selamectin, spinosad q 2 weeks alternating or fipronil and lufenuron alternating. or, use one of the newer isoxazoline classes of drugs: furalaner	Ingestion of fleas can lead to tapeworm (diplydium caninum); seeing tape proglottids- recommend empiric flea treatment as well as tx for tapes

PEDICULOSIS BITING: *TRICHODECTES CANIS FELICOLA SUBROSTRATUS* SUCKING: *LINOGANTHUS SETOSUS*

Biting lice: affects dorsum sucking lice: tips of ears, anterior back	Variable pruritus hair loss, papules anemia can occur with sucking lice in heavy infestations in small animals	Flea comb, magnifying glass 	Fipronil spray, vectra 3D in dogs off label use of isoxazolines, selamectin 	Fipronil spray can be used off label in pets under 8 weeks of age but is labeled for use in pets 2 days old or older in europe
--	---	--	--	--

SARCOPTIC MANGE: *NOTOEDRES CATI, SARCOPTES SCABEI*

Elbows, hocks, ears 	Marked pruritus, papules, crusting, excoriations, hair loss hx of itchy people in the home as well	Skin scrape multiple; sarcoptes antibody is available but can be positive in dogs with a house dust mite sensitivity	Selamectin combined with imidacloprid/ moxidectin q 2 wks for 2-3 treatments or off label use of furalaner day 0 and day 28	Scoropotes can be hard to find on skin scrapes so empiric therapy is often warranted.
---	---	--	---	---

ECTOPARASITES : CANINE/FELINE

LESION DISTRIBUTION	CLINICAL SIGNS	DX	TREATMENT OPTIONS	GOOD TO KNOW
---------------------	----------------	----	-------------------	--------------

DEMODECOSIS: D. CANIS, D. INJAI, D. CATI (FOLLICULAR) D. GATOI (SURFACE)

Haired skin generalized or localized



Alopecia, comedones follicular casts, secondary bacterial pyoderma
D. gatoi in cats is highly pruritic and cats barber

Skin scrape, hair pluck

Weekly lime sulfur dips 6-8 weeks or imidacloprid/moxidectin q 2 weeks for 3 months
off label: topical bravecto

Localized demodex can spontaneously resolve; generalized demodex (> 5 sites) may indicate an underlying disease/condition affecting immune function

CHYLETIELLOSIS : C. PARSITIVORAX, C. BLAKEI C. YASAGURI

Found most often on dorsum

Pruritus, scale +/- walking dandruff

Examine scales



Fipronil spray
selamectin
lime sulfur dips
off label: furalaner

Affects ears and occasionally other sites



Pruritus, otitis with dark brown coffee ground discharge

Aural cytology of exudate

Revolution
imidacloprid/moxidectin

Concurrent otic therapy may speed resolution

OTODECTIC MANGE: O. CYANOTIS

Anywhere on skin

Often asymptomatic, occasionally raised inflammation locally and rarely joint pain, lethargy, malaise

Visual exam



Fipronil spray
isoxazolines
selamectin/sarolaner
pyrithethrins
pyriproxyfen

Faster tick kill provides better benefit. However most tick products kill within 24-48 hours; lyme disease takes 36-48 hours of attachment to transmit disease

TICKS

IXODES, DERMACENTOR, RHIPICEPHALIS

Anywhere on skin

Often asymptomatic, occasionally raised inflammation locally and rarely joint pain, lethargy, malaise

Visual exam



Fipronil spray
isoxazolines
selamectin/sarolaner
pyrithethrins
pyriproxyfen



Parasites found in Stool

Parasites of the Gastrointestinal Tract

Nematodes

Hookworm

Ancylostoma caninum



Uncinaria looks the same but is 25% bigger

Stomach worm

Physaloptera

40-35um



Roundworm

80-85um

75-55um

70-80um

65-75um

Toxocara Canis

Baylisascaris Procyonis

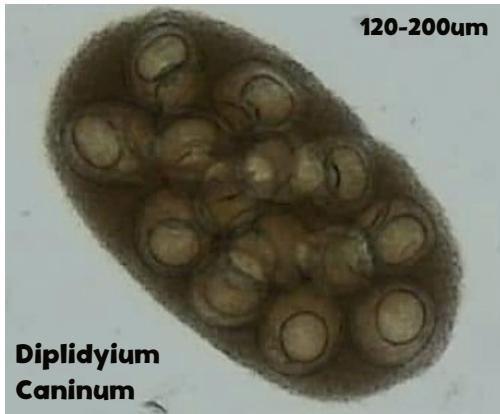
Toxocara Leonina

Toxocara Cati

Whipworm

Trichuris vulpis (whipworm):
Bipolar plugs, more symmetrical than *Eucoeus aerophilus* (cappillaria);
Trichuris is bigger as well.

90-44um



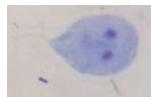
Diplidium Caninum

Protozoa

Giardia

8-14um

8-12um



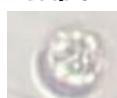
8-14um

30-37um



Different stages of sporogony

10um



4-6um



Toxoplasma gondii

Cryptosporidium

www.etsy.com/shop/VetHelpfulNotes

Cestodes



Spirometra

30-35um



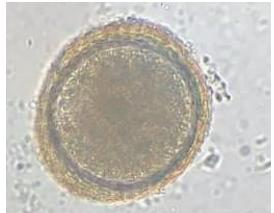
Taenia & Echinococcus



Comparison of Size and Characteristics of Ova Found in Stool

Vet Helpful Notes
Canine Feline

Roundworm Nematodes



Toxocara canis



Baylisascaris procyonis



Toxocara Leonina



Toxocara cati

Protozoa

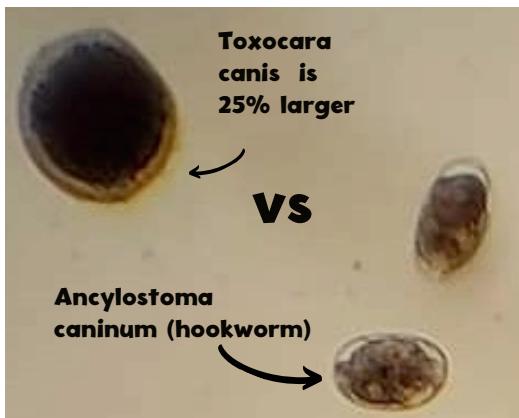
Cestode

Protozoa



Toxo

Crypto



Grain mite egg. Bigger than Hookworm ova and hard to see "egg packets"

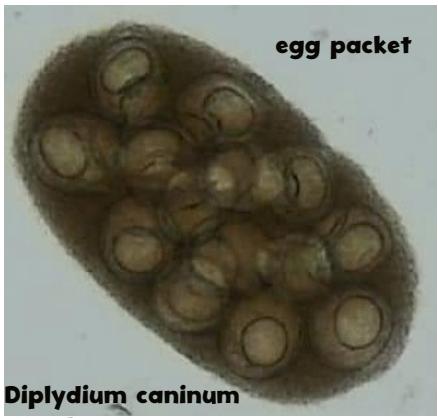


Hookworm and Grain Mite eggs look alike and are sometimes confused

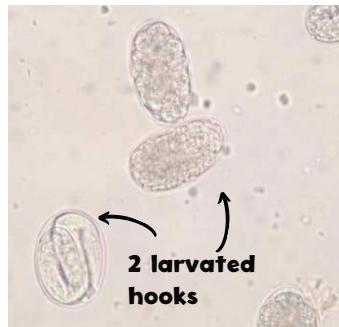


Ancylostoma caninum (hookworm)
Compared to coccidia (Isospora)

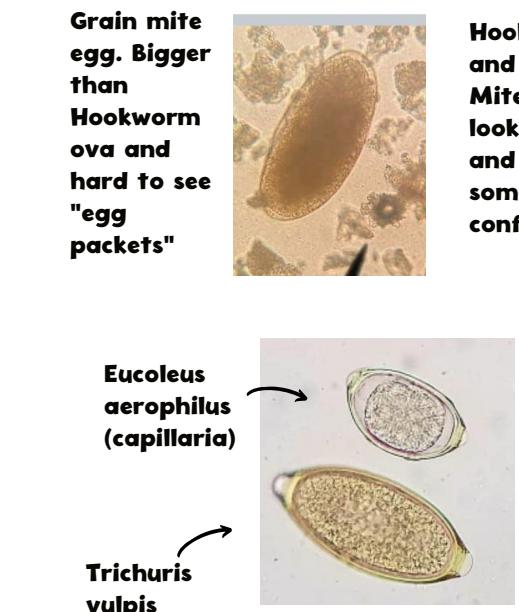
Size matters!
They look alike, but



Diplydium caninum
40-45um per egg,
1-63 eggs/packet



Hookworm ova and larvated hooks



Trichuris vulpis



Trichuris vulpis (whipworm)
Bipolar plugs, symmetrical
Bigger than Eucoelius aerophilus (capillaria);



Eucoelius aerophilus (capillaria)
Bent/curved, asymmetrical bipolar plugs



Size Matters!



Biggest to smallest



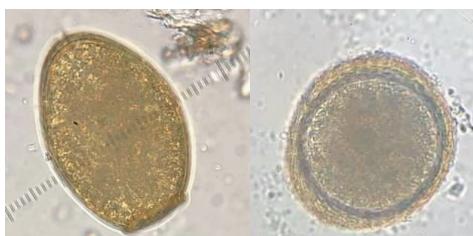
Aleurostrongylus Abstrusus 400um
&
Filaroides 240-290um
Lungworm/Strongyle



Diplydium caninum
120-200um
40-45um per egg &
1-63 eggs/packet
Tapeworm



Trichuris vulpis
90x44 um
Whipworm



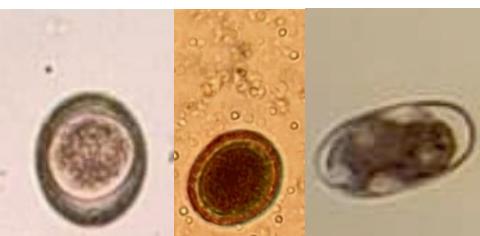
Paragonimus Kellikotti
85x55umum
Trematode (Fluke)



Toxocara canis
80x85 um
Canine Round



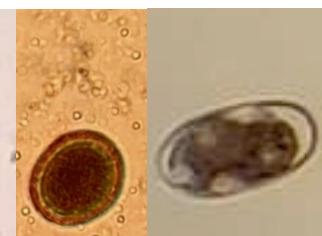
Baylisascaris procyonis
80x85um
Racoon Round



Eucoleus aerophilus (cappillaria)
70 x 35um
Lungworm



Toxocara Leonina
70x 80 um
Canine & Feline Round



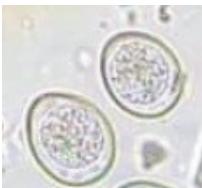
Toxocara cati
65x75um
Feline Round



Ancylostoma caninum
60 x 40m
Hookworm



Physaloptera
45x35 um
Stomach worm



Isospora
30-37um
Coccidia



Taenia
30x35um
Tape



Tritrichomonas
8x 14
trophozoites
Protozoa



Giardia
8x 14
trophozoites
Protozoa



Giardia
cysts
8-12um
cysts
Protozoa



Toxoplasma
10um
Protozoa



cryptosporidium
4-6um
Protozoa

Respiratory and Urinary Tract Parasites

Nematodes

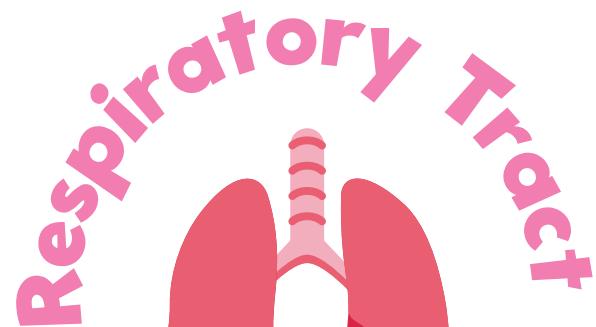
**Aelurostrongylus
Abstrusus
&
Filaroides**



Strongyle
has kinked
tail

Eucoleus aerophilus
(formerly *capillaria*)
is bent/curved compared
to *Trichuris Vulpis*
(whipworm).

You can see bipolar plugs
are slightly angled
because
the curve of the top side
versus the bottom are
different.



Trematodes

**Paragonimus
kellicotti**



Trematodes
have a cap
(operculum)

Nematodes of the Urinary Tract

Found most often on a urine
sediment exam. Can be found
in stool contaminated with urine

**Pearsonema
feliscati**
Found in felines
only

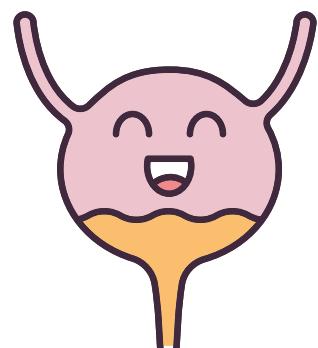


**P.
plica**
65-
25um

**Pearsonema
plica**
found in
canines and
felines



**P.
feliscati**
55-30 um



Common Nematodes



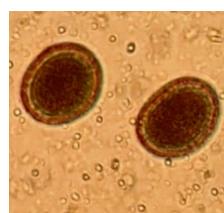
Toxocara Canis



Toxocara Leonina



Baylisascaris Procyonis



Toxocara Cati



Ancylostoma Caninum



Uncinaria
about 1.5 x
the size of
Ancylostoma



Physaloptera

Roundworm (Ascarids)

Toxocara canis and **Leonina** are canine roundworms (ascarids), **Toxocara cati** and **T. Leonina** are feline roundworms (ascarids). Dogs are infected via ingestion of larvated eggs in a contaminated environment. They can also be infected by ingestion of other animals that are infected (eg: birds, rodents) or transplacental infection to puppies and kittens (not **T. Leonina**). **T. Leonina** causes few symptoms. The larvae migrate through the liver and lungs and are coughed and swallowed to land in the intestine.

All puppies and kittens should be dewormed regularly starting at 2 weeks of age. Deworm every 2 weeks until broad spectrum parasite control begins. This is a zoonotic disease that can cause visceral and ocular larval migrans in people.

Adult worms look like spaghetti in stool or vomitus.

Baylisacaris: ascarid of raccoons that can infect dogs.

Zoonotic: Can cause neurologic disease in humans.

Treatment: fenbendazole, milbemycin oxime, pyrantel pamoate.

Hookworm

Ancylostoma caninum, **A. Tubaiforme** (cats only), **A. braziliense**, **Uncinaria stenocephala**.

Infection via ingestion of third stage larvae in contaminated environment, ingestion of rodents, cockroaches, nursing an infected mom, skin penetration. During development, some of the infective larvae remain dormant in the host tissues and may be re-activated later. (A common feature of racing Greyhounds).

Most mature in the small intestine. Immature and adult worms can attach to the small intestine. Adult worms are small, 1-2 cm long and 1/2 cm in diameter. Infected puppies and kittens may develop anemia and ill thrift. Untreated puppies and kittens may die of the infection. Pneumonia may develop in young puppies as the larvae migrate through the lungs. **A. caninum** causes more serious disease than **A. braziliense** in dogs.

Zoonotic: Hookworm is zoonotic and can cause visceral larva migrans. Humans can be infected by direct skin penetration of the hookworms shed by dogs and cats.

Control is best achieved by prompt removal of feces from the environment, covering sandboxes so cats can't defecate there and treating dogs and cats.

Treatment: fenbendazole, milbemycin oxime, moxidectin, pyrantel pamoate.

Stomach Worm

Physaloptera

Coyotes, foxes normal definitive hosts. Can infect dogs and cats via ingestion of intermediate hosts: crickets, cockroaches, beetles or paratenic hosts (amphibians and reptiles, other mammals).

Adult nematodes develop in the stomach. Larvated eggs passed in stool and develop into third stage infective larvae once inside insect intermediate hosts.

May cause chronic vomiting in dogs/cats. Adult worms are 3-6 cm in length.

Eggs don't float well even with centrifugation so can underestimate the prevalence of this infection.

Treatment: removal of worms via endoscopy and treatment with pyrantel q 2 weeks for 3 treatments.

NOT zoonotic.

Common Nematodes



Trichuris Vulpis

Whipworm

Whipworms: *Trichuris vulpis* occurs in the dog, fox and coyote.

Whipworms are rare in cats in North America. Hosts are infected by ingesting embryonated eggs in the soil. Larvae hatch from eggs in the small intestine and penetrate the mucosa of the intestine and then move to the cecum.

Adult worms are 4.5-7.5cm long with most of their body composed of a whip like thin lash at the end of a shorter body.

Eggs are football shaped with prominent bipolar plugs and a smooth shell.

They must be differentiated from other cappilarids (*Eucuolus* : a lungworm and bladder worm species) by their size and shape. *Trichuris* is a little bigger and has a symmetric shape.

Most infections are asymptomatic but it can cause diarrhea with blood, dehydration and anemia.

There is NO transplacental or transmammary transmission.

Infected animals may have symptoms long before they shed detectable eggs into their feces.

Treatment: drontal plus, fenbendazole.

NOT zoonotic.

Lung worms

***Eucoleus*, formerly called *Cappillaria aerophila*. Affects dogs and cats.**

Transmission is fecal -oral. No known

intermediate hosts. Earthworms might be a paratenic host.

Larvae from ingested eggs hatch in the small intestine and migrate to the lungs. Adult worms are 1.5-4 cm long. Eggs are 60umx 30um (smaller than *Trichuris Vulpis*) and have assymmetrical bipolar plugs.

Young animals are most susceptible when infected. Can cause cough, nasal discharge, dyspnea and sometimes pneumonia.

Treatment: ivermectin or imidacloprid/moxidectin.

Zoonosis: Rare reports of human transmission.

***Aleurostrongylus Abstrusus*.** Common in cats. Cats are the definitive host but mice, rodents frogs, toad, snakes lizard and birds can be transport hosts. Cats eat transport hosts and ingested larvae end up in the intestine and migrate to the lungs. Worms are 1/2 to 1 cm in length. First stage larvae are passed in the stool and have a characteristic kinked tail. Clinical signs often absent. But can cause asthma, cough and dyspnea. Can be diagnosed by fecal flotation. A baermann on fresh fecal samples or on sputum.

***Filaroides* spp** are canine lungworm. There is no intermediate host. Infective via feces or sputum. Causes wart like nodules on the trachea and bronchii. Treated with fenbendazole.

Treatment: fenbendazole, imidacloprid/moxidectin.

NOT zoonotic.

Bladder worm

***Pearsonema plica*: dogs and cats, *Pearsonema feliscati*: just cats.** Eggs are passed in the urine and first stage larvae must be ingested by an earthworm. Dogs or cats ingest the earthworm and the adults develop in the urinary bladder.

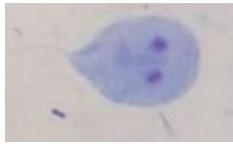
Kidney worm

***Dioctophyme renale*: kidney worm** have a very pitted surface and adults live in the kidneys. Dogs and cats are infected by eating fish or frogs.

Treatment: fenbendazole and ivermectin.

NOT zoonotic.

Protozoa



Giardia

Giardia is acquired by ingestion of cysts in fecal contaminated water, food or fomites.

Dog strains do not infect cats and cat strains do not infect dogs.

Types of giardia or strains are called assemblages. The motile form is a trophozoite and it attaches to the lining of the intestine. The organism is intermittently shed. The trophozoites are very small at

~15x 11um in size. Cysts are ellipsoidal and non motile and are about 12um x 8um in size with a thick refractile wall. Dogs can be asymptomatic with infection. Diagnosis is made by direct fecal smear, fecal flotation, or fecal ELISA.

Treatment: fenbendazole, metronidazole, drontal plus. ELISA test can remain positive after treatment for variable periods of time, best to look for cysts/trophozoites to determine treatment failure. Reinfection is common in areas with high environmental contamination.

Zoonotic: Rare for human transmission from dogs or cats.

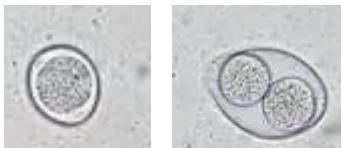


Tritrichomonas

Parasite lives in colon and cecum. There is no cyst stage that lives outside the host for extended periods of time like giardia. Many cats are asymptomatic carriers. Infection and disease is most common in catteries. Most common symptom is diarrhea which can last weeks, months, years. Age of onset is 0-24 months. Most common in long haired cat breeds and cats under 1 year of age. Transmission is fecal-oral. Diagnose with direct fecal smear, fecal culture or fecal PCR. Can be mis-identified as giardia, but the T. foetus movement is jerky and erratic vs the falling leaf movement of giardia.

Treatment is Ronidazole.

NOT zoonotic.



Isospora (Coccidia)

Coccidial infections are common in dogs and cats. Young animals are far more likely to become infected with coccidia than older animals. Transmission by ingestion of sporulated oocysts from contaminated environments or by predation. Can cause diarrhea. Dogs may be asymptomatic too. Sporulated oocysts are infective. The organisms live in the cells lining the intestine.

Treatment : sulfadimethoxine, ponazuril

NOT zoonotic.



Toxoplasmosis

Toxoplasmosis is a well known and important zoonotic parasite. The protozoal parasite can infect dogs, but rarely so. It is most likely to produce disease in immunocompromised cats. It causes multisystemic infection with granulomatous inflammation, fever, weight loss and lethargy. Cats with clinical toxoplasmosis are unlikely to be shedding oocysts in their stool.

Cats and dogs become infected by ingesting infected mammals or birds with bradyzoites encysted in their tissues or ingestion of sporulated oocysts contaminated by feline feces (soil, water, vegetation). Oocysts shed by cats become infective 1-5 days later. cats only actively shed cysts for 1-3 weeks. Most cats that are shedding oocysts are seronegative at the time. Antibody testing might help determine timing of infection (recent or in the past). Most seropositive cats (IgM or IgG) have completed their oocyst shedding and unlikely to repeat shedding or represent a source of human infection.

Treatment: clindamycin 10-12mg/kg bid x 2-3 weeks.

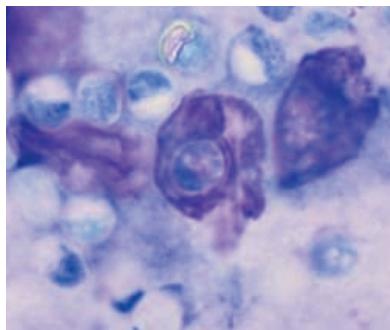
Prevention: prevent hunting in cats, don't feed raw food, remove fecal matter daily from litter boxes.

Zoonosis: Direct contact with cats is not considered a risk factor, particularly if they are kept indoors and fed a commercial non raw diet. Cats that test positive to Toxo are less likely to shed oocysts than a cat that is seronegative.

Do not have immunocompromised or pregnant women scoop the litterbox. FACT most human infections are due to eating under-cooked meat or shellfish or contact with infected soil from gardening.

Protozoa

Cryptosporidium



Crypto is a protozoan parasite that infects a wide number of vertebrate species. Humans are less

likely to be infected by Crypto from dogs and cats than other species. The organisms are tiny at 4-6 um in diameter and can be difficult to identify under the microscope without a trained eye. Diagnosis is via finding oocysts on a fecal float, fecal smear, or antibody or antigen tests.

The organism causes a self-limiting diarrhea that is worse in immunocompromised animals. Transmission of the infection is via ingestion of sporulated oocysts from fecal-contaminated environments. Most cases of cryptosporidium in dogs and cats are not clinical (do not cause diarrhea).

Treatment: Tylosin, Azithromycin

Zoonotic: disease spread most often by strains that do not commonly infect dogs and cats but more commonly cattle or pigs.

Cestodes

Spirometra



Indirect life cycle that requires two intermediate hosts before becoming infectious to a dog or cat. When an infected dog

or cat defecates near water, the egg contacts water and the embryo hatches and is ingested by a copepod (a type of zooplankton) which is the first intermediate host. This is then consumed by a non fish vertebrate. The spirometra further develops and embeds in the tissue of the non fish vertebrate (amphibians, reptiles, birds). A cat or a dog has to ingest these infected animals to become infected.

Eggs are operculate and measure ~65-45um.

Symptoms in dogs and cats are diarrhea, weight loss and vomiting and typically resolve with anthelmintic therapy.

Treatment: praziquantal.

Zoonosis is considered rare.

Taenia



Requires intermediate hosts. Dogs and cats infected shed proglottids laden with eggs in their stool. Dogs and cats are infected when they ingest infected intermediate hosts. Different strains of Taenia require different intermediate hosts: rabbits, rodents, sheep most often.

Adult taenia are in the small intestine. Disease in dogs and cats with Taenia is rare and most dogs and cats don't have significant disease complications from infection.

Taenia eggs are 24-40um in diameter with a thick striated egg shell. Cannot physically distinguish Taenia eggs from Echinococcus eggs with microscopic exam!

Treatment is praziquantel. Prevention of predation/scavenging activity.

Zoonosis: Isolated reports of human infection with Taenia but risk is very low in humans.

Cestodes

Echinococcus



E. granulosus : dogs and cats

E multilocularis: cats

Cestode that requires specific intermediate hosts. Definitive host shed proglottids in feces. When eggs are consumed by the intermediate hosts, then large hydatid cysts develop. Dogs and cats are usually infected when they ingest these cysts during predation/scavenging rodents.

Adults of echinococcus are very small (2-7 mm) and reside in the small intestine. Eggs passed in stool are infective to the intermediate host. Larvae penetrate the intestinal wall and migrate to liver, lungs and other organs.

E. multilocularis produces multilocular hydatid cysts that aggregate and proliferate in the tissue. Highly invasive and can mimic malignant cancer in the infected intermediate host. The definitive host is infected when they consume the infected intermediate host.

Echinococcus occasionally causes multilocular cysts in dogs- but reports are rare.

E granulosus is only known to infect dogs and wild canids following ingestion of cysts. Intermediate hosts: goats, sheep, cattle, pigs, horses

Dogs that do develop multilocular hydatid cysts have a poor prognosis. **Eggs cannot be distinguished from Taenia spp.** Proglottids are unlikely to be observed grossly. PCR identification can help diagnose.

Treatment: praziquantel for elimination of intestinal stages. **Prevention:** limit opportunities for ingestion of prey or scavenging.

Zoonotic: Dogs and cats infected with these tapeworms do present a zoonotic risk, although the risk is low. Humans can be an intermediate host to E. Granulosus and E. multilocularis. Recc monthly treatment of dogs and cats with praziquantel.



Dipylidium Caninum

Affects cats and dogs who ingest fleas during grooming. The tapeworm segments (proglottids) are shed in the stool of infected dogs and cats about 2-3 weeks after infection. The dipylidium egg packets are found within the proglottids and contain clusters of 25-30 eggs.

Proglottids are visible to the naked eye and look a little like cucumber seeds. The entire tapeworm is about 15-70cm in length and reside in the small intestine.

Fecal flotation tests are often negative in infected animals because the eggs and proglottids heavy and don't float well. Diagnosis is often by identify proglottids , not in a fecal flotation.

Most proglottids are seen in the perianal area, bedding or stool.

Treatment: praziquantel

Control and prevention: control fleas and lice.

Zoonotic: There are rare reports of infection in children after flea ingestion. The disease is typically mild and readily treated.

Paragonimus kellicotti

Platynosomum fastosum

Nanophyetus salmincola

Heterobilharzia americana



Flattened end is the operculum

Trematode eggs have an operculum or a cap. Trematodes of dogs and cats have an indirect life cycle requiring one or two intermediate hosts to reach an infective stage for dogs and cats.

Dogs infected with *P. kellicotti* shed eggs in their stool. The eggs hatch in water and penetrate a snail which is then ingested by a crayfish (paragonimus) salmon fish (nanophyetus salmincola), lizards/reptiles (platynosomum fastosum). When a dog eats an intermediate host, they become infected.

Paragonimus adults develop in cyst in the lungs of dogs and cats. Dogs and cats can be asymptomatic or have symptoms of coughing, dyspnea, pneumothorax etc.

Nanophyetus adults are found in the small intestine of dogs and cats. *N salmincola* is not associated with symptomatic disease. But this fluke may carry *Neorickettsia helminthoeca* which is the agent of Salmon poisoning, a virulent disease of the Pacific Northwest.

Platynosomum fastosum: liver fluke of cats in Florida and southeastern US and HI. Chronic infection leads to enlarged bile ducts and gall bladder and biliary hyperplasia and eventual liver failure.

Heterobilharzia americana live in the mesenteric and hepatic veins. This is acquired because these penetrate the skin of dogs that wade in water. They induce granulomatous inflammation and cause diarrhea, vomiting weight loss.

Trematode infection in dogs and cats is rare compared to nematodes and cestodes. However, because they can cause severe disease control is essential.

Trematode eggs are less buoyant and require concentrating ova with sedimentation not flotation.

The different trematode species are identified as trematode by the characteristic opercula but differentiated by size and other morphologic characteristics.

Treatment: praziquantal, fenbendazole, Tetracyclines.

Prevention: prevent pedation/scavenging.

NOT zoonotic.

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Antibiotics for Corneal Ulcers in Dogs and Cats

www.etsy.com/shop/VetHelpfulNotes

Causes of Corneal Ulcers:

- Trauma
- Trichiasis
- Distichiasis
- Ectopic cilia
- Foreign bodies
- Cranial nerve dysfunction
- Ectropion
- KCS
- Eyelid tumors
- Blepharitis
- Herpes virus
- Lagophthalmus

Superficial uncomplicated Corneal Ulcer

Should heal within 7 days, usually 3-5 days; recc treat q 6 hours

PEARL: The most critical treatment for a corneal ulcer is removal of the primary cause. Therefore, if an ulcer has not healed in 7 days, change your diagnosis, not your antibiotic

Melting Ulcer

Widen antibiotic spectrum

Example: on BNP add Moxiflox or Oflox

Example: on On Erythromycin add Oflox or Tobra

Add Serum or Vetrix EyeQ amniotic

Increase frequency to q 2 hours administration

Cats

If an ulcer has not healed in 7 days, categorize it as:



About the same

Deep/green/melting

loose edges

Look harder for cause (ex FHV-1)

C&S
increase antibiotic therapy

Debride and add antiviral

Dogs

If an ulcer has not healed in 7 days, categorize it as:



About the same

Deep/green/melting

Loose edges

Look harder for a cause

C&S
increase antibiotic therapy

Debride and/or grid Keratotomy

Ophthalmic Antibiotics

Bacitracin- peptide antibiotic: gram positive bacteria (might cause feline anaphylaxis)

Chloramphenicol- (no particular class): Broad spectrum, Not good for pseudomonas (resistant)

Erythromycin- Macrolide: Gm pos Staph and Strep, mycoplasma, chlamydophila. Not good for pseudomonas

Gramicidin- Peptide antibiotic: Gram pos (not grm pos bacilli like corynebacterium) and some gram neg organisms

Neomycin- aminoglycoside: Broad spectrum, Not good for: pseudomonas and mycoplasma (resistant) may cause feline anaphylaxis

Moxiflox/Ofloxacin- fluroquinolone: Broad spectrum: Staph, Strep, Pseudomonas, Chlamydophila, Haemophilus, variable efficacy against mycobacteria and anaerobes, Not good for some strains of staph and strep (resistant). Better against Gm neg than Gram Pos

Polymyxin B- polypeptide antibiotic: Gram neg bacteria, mostly used in combination with other abx (Caution feline anaphylaxis)

Terramycin- tetracycline: Mycoplasma, chlamydia, some gram negatives (proteus, pseudomonas)

Tobramycin-aminoglycoside: Gram neg bacteria (pseudomonas) and Gram pos staphylococci (limited); not good for Strep

The most common bacterial isolates from cats with bacterial keratitis:



~ 66% Gram positive: Mostly staphylococcus (cocci)

~33% Gram-negative: Mostly pseudomonas (rods)

Good first line ophthalmic antibiotic choice:

Cats: **Erythromycin** or ofloxacin (caution with Terramycin due to added Polymyxin b)

The most common bacterial isolates from dogs with bacterial keratitis:



~75% Gram positive Staphylococcus> Streptococcus> Corynebacterium (rod)

~25% Gram negative: Mostly Pseudomonas, E. coli (rods)

Good first line ophthalmic antibiotic choice:

Dogs: **Triple Antibiotic** like: Bacitracin, Neomycin, polymyxin (BNP), ophthalmic or Neomycin, polymyxin, gramicidin

Glaucoma



Pathophys

Glaucoma is elevation in intraocular pressure (IOP) that is incompatible with normal ocular function. IOP is the result of a balance between production and drainage of aqueous humor.

- **Primary Open Angle Glaucoma:** Gradual/progressive increase in IOP over 2-4 years. Early/moderate stages have IOPs between 25-40mmHg and variable clinical signs. Advanced stages IOP's 50-60's (buphthalmia, lens luxation/sublux, optic disc degeneration, vision loss)
- **Primary Closed Angle Glaucoma (Most common primary cause in dogs):** abrupt increase in IOP. IOP's increase to 30mmHg or higher. Vision often impaired or lost by the time they present to the vet. The fellow eye typically follows about 8 months later.
- **Secondary Glaucoma:** Altered aqueous humor dynamics due to other causes such as: uveitis, neoplasia, trauma, lens luxation etc. Most common cause of glaucoma in cats!

Clinical signs

Episcleral injection, painful eye, corneal edema
Dilated pupil, may be sluggish to respond to light
Lens luxation may be present
Buphthalmos from long standing glaucoma
Cats: Rarely show ocular redness!! Look for epiphora, varying blepharospasm, mydriasis, reduced PLR, corneal edema

Treatment

Dogs: Goal of emergency therapy is to **reduce IOP to <20mmHg within a few hours**

Acute Glaucoma treatment (no anterior lens luxation present)

- 1-2 drops **prostaglandin analogue** (ex Xalatan) followed by **Dorzolamide** 5 minutes later.
- Repeat Xalatan in 30 minutes if IOP >30mmHg
- **If IOP still elevated >30mmHg in an hour** (90 minutes after starting) **add in mannitol**

Mannitol: Draw blood sample prior to mannitol to check lytes. Prepared IV mannitol: 2gm/kg or 1 gm/kg with heart disease. Give slowly IV over 20 minutes through a filter set. Should decrease IOP within an hour. Withhold food/water for 4 hours.

Continue Dorzolamide BID to TID and start Pred Acetate BID to counteract inevitable uveal inflammation

- Dorzolamide (Trusopt): carbonic anhydrase inhibitor
- Dorzolamide:Timolol (Cosopt): in combo with timolol (beta blocker)
- Timolol: caution with heart block, bradycardia, asthma, chronic bronchitis

Some Ophthalmologists prefer the use of an emergency Acqueocentesis: drawing off about 0.1ml of aqueous humor by inserting a narrow gauge needle in a shallow angle into the anterior chamber at the limbal margin

Cats: Typically not acute, due most often to secondary cause.

Cats don't respond to prostaglandin analogues! Use topical timolol, dorzolamide ; treat underlying cause

Etiology

- **Primary Glaucoma:** Inherited abnormal metabolism of the outflow system, or physical pupillary blockage and changes to the drainage areas
- **Secondary Glaucoma:** Increased IOP associated with previous or current eye disease that obstructs outflow pathways. Typically unilateral and not inherited. Examples: uveitis, lens luxation, intraocular neoplasia
- **Congenital Glaucoma:** increased IOP associated with inherited anomalies. Develops soon after birth. Very rare.

Diagnostics

- **Tonometry:** IOP is **>30mmHg (dogs) >25mmHg (cats)** or a greater than **10mmHg pressure difference between the two eyes.**
- Gonioscopy to evaluate the iridocorneal angle to determine if the angle is open or narrowed.
- Ophthalmoscopy to evaluate the retina
- PLR /menace to assess for vision loss
- Lens evaluation to screen for lens luxation
- **Cats:** screen for secondary causes: hypertension, infectious dz, neoplasia, pre-existing uveitis, trauma

Summary/key points

Most canine glaucomas are closed angle (not slowly progressive glaucoma as seen in people). Therefore annual tonometry is generally not useful.

- Once glaucoma has developed in one eye it is useful to prophylactically treat and monitor the fellow eye.
- **Prophylactic treatment** of the unaffected eye can **delay glaucoma** from a median of 8 months to a median of **31 months** with treatment.
- In cats most glaucoma is due to uveitis or neoplasia, but open angle glaucoma occurs in the Siamese breed.
- Not all cats with glaucoma present with a red eye or any signs of ocular discomfort!!
- Prostaglandin analogues contraindicated with anterior lens luxation because they can worsen drainage
- **Medical therapy typically ineffective within a year.**
- Surgical intervention can prolong vision but has a poor long term success rate.

Cats: 95-95% are secondary to uveitis, neoplasia, sometimes trauma, intraocular hemorrhage, etc.

Anterior Uveitis

Pathophys

Uveitis is any condition that causes uveal tract inflammation. This leads to breakdown of the blood/aqueous barrier/blood-retinal barrier and allows protein and cellular accumulation

Clinical signs

Episcleral erythema, +/- photophobia, +/- aqueous flare, miosis or anisocoria, rubeosis iridis, iris bombe
IOP <10 mmHg OR a difference of 10mmHg or more between the 2 eyes is supportive
+/- corneal edema. Remember, Hyphema or hypopyon always = uveitis!

Treatment

If the patient has normal pressures (8-18mmHg) in the face of obvious anterior uveitis, start a topical steroid (1% pred acetate or 0.1% dexamethasone) and dorzolamide BID as a preventative for possible pressure spikes.

Always check a fluorescein stain to ensure no corneal ulceration is present before starting topical steroids.

If there is a corneal ulcer, topical NSAIDS can be used and an oral steroid until the ulcer is healed.

Pressure <8mmHg: start pred acetate 3-4x/day

Pressure 8-18mmHg : pred acetate and add in dorzolamide BID

Pressure >18mmHg pred acetate and add in dorzolamide TID

Basic plan:

1. 1% pred acetate q 4-6 hrs +/- dorzolamide
2. Tropicamide bid to TID or atropine as needed to keep pupil dilated
3. Flurbiprofen or diclofenac BID
4. +/- oral NSAID if safe (or oral prednisone if confident won't cause worsening of occult underlying disease)
5. Doxycycline 5mg/kg bid x 14 days dogs OR Azithromycin or Clindamycin in cats

Once clinical signs improve (normal IOP, no redness, injection, no more cellularity) you can start by tapering the oral NSAID or steroid. Then taper the topical steroid. Tapering off all meds usually takes weeks to months.

Etiology

Infectious: bacterial, viral, fungal, parasitic

Idiopathic/immune-mediated: up to 60%-70% of dogs and cats

Other: Coagulopathies, Diabetes mellitus, Hyperlipidemia, Hypertension, Lens-induced uveitis (eg, cataracts), Primary neoplastic disease (eg, ocular melanoma), Secondary neoplastic disease, Trauma, Ulcerative keratitis

Diagnostics

Tonometry: **IOP under 10**, unless there is concurrent glaucoma which can cause a normal IOP even with obvious signs of uveitis (flare, hyphema, etc.)

Other eye tests should include fluorescein staining to look for corneal ulceration.

Minimum database: CBC, Chemistry, Urinalysis, Bp

Infectious disease testing cats: Felv/FIV, Cryptococcus antigen, Bartonella Ab by IFA, FCoV antibody, Toxoplasma IgG and IgM

Infectious disease testing dogs: tick titers, fungal, protozoal, bacterial and viral diseases as indicated by area, travel history etc.

Consider imaging as well: chest xrays, AUS

Summary/key points

Feline uveitis is the most common cause of glaucoma in cats

Most uveitis cases in dogs and cats are idiopathic (no underlying cause determined)

Of the cats with a diagnosed cause, infectious disease is most common; dogs rarely have infectious uveitis

Most causes of uveitis in dogs is idiopathic (up to 60%); other non-infectious causes are: lens induced, neoplastic, traumatic, corneal ulcers, pigmentary uveitis of golden retrievers, uveodermatologic syndrome.

Up to 70% of cats with uveitis have an underlying systemic disease or associated systemic disease: Felv, FIV, FIP, Bartonella, Toxo, Fungal disease

If there is Hyphema or Hypopyon = uveitis



TOP TOXINS



TOXIN	OUTCOME IF UNTREATED	TOXIC DOSE	DECONTAMINATE?	TREATMENT	MONITORING	OF NOTE
GRAPES AND RAISINS	Renal failure can occur within 24-48 hours if a toxic amount is ingested	Lowest documented toxic grape or raisin dose is: 0.32 to 0.65 oz/kg Avg grape: 7 grams Raisin small 0.5 gm med 1 gm large 1.5 gm	Yes, emesis followed by activated charcoal.	Following decontamination, aggressive IVF for a minimum of 48 hours. For oliguric dogs, administer dopamine or furosemide.	Monitor renal values and urine production.	Not all dogs will develop renal failure if exposed to grape/raisins. It is considered possibly an idiosyncratic reaction.
CHOCOLATE	Mild signs: vomiting, diarrhea, polydipsia with lower ingestions Severe signs with higher ingestions: cardiac seizures, arrhythmias, seizures, hyperthermia	Mild signs with 20 mg/kg of theobromine and caffeine (both are methylxanthines) Cardiotoxicity at 40-50mg/kg Seizures > or = to 60mg/kg. 1 oz of milk chocolate per pound may be lethal!	Yes, emesis followed by activated charcoal. Because of enterohepatic recirculation of methylxanthines, repeat charcoal doses every 12 hours if symptomatic.	Following decontamination, correct dehydration control vomiting with anti-emetics, control body temperature and treat any seizures, arrhythmias	Monitor acid/base and electrolyte derangements, monitor heart with ECG, manage body temperature	Placement of a urinary catheter or frequent walks to encourage urination because methylxanthines can be re-absorbed from the bladder.
XYLITOL	Hypoglycemic seizures, acute liver failure, death	>75-100 mg/kg ingested: pet should be hospitalized and baseline BG measured. >500mg/kg should have baseline liver values	Induction of emesis if asymptomatic and under vet supervision. Charcoal is not effective as it does not bind well to the xylitol sugar	Induction of emesis if asymptomatic Dextrose supplementation as needed until BG's normal for 12-24 hours. K and phos q 4-6 hours	Monitor BG's q 1-2 hours for 12 hours. Evaluate liver values q 24 hours. Tx until pet can maintain normal BG w/o dextrose. Provide dextrose for >500 mg/kg ingestions. Monitor for coagulopathy, evidence of liver insufficiency	Prognosis for uncomplicated hypoglycemia is good. Prognosis for severe hepatotoxicosis is guarded to poor.



TOP TOXINS



TOXIN	OUTCOME IF UNTREATED	TOXIC DOSE	DECONTAMINATE?	TREATMENT	MONITORING	OF NOTE
LILY	Acute renal failure in cats within 12-36 hours of exposure. Death within 3-5 days.	No toxic dose known, but less than 1 leaf or small amounts of pollen can cause clinical effects. Ingestion of 2-3 leaves have been fatal.	Yes, emesis followed by activated charcoal with sorbitol	Fluid diuresis at twice maintenance rates for at least 48 hours to maintain urine flow and prevent obstruction of renal tubules with sloughed epithelial cells.	Baseline CBC, chemistry and urinalysis. Urine epithelial casts within 12-18 hours of exposure and isosthenuria. Proteinuria and glucosuria can occur. Monitor elevation in creatinine, compared to BUN may be seen.	Once anuria develops hemodialysis or peritoneal dialysis are the only potential treatment options. Disproportionate elevation in creatinine compared to BUN may be seen.
ANTI COAGULANT RAT BAIT	Clinical signs of bleeding will occur within 3-5 days of ingestion. Symptoms can include: lethargy, weakness, anemia, dyspnea, cough, hemoptysis, abdominal distension, seizures, and paralysis.	Second generation products: brodifacoum, bromadiolone, difenacoum and difenacoum require only 1 feeding. First generation products like warfarin typically require several feedings.	Yes, emesis ideally within 4 hours. Emesis unlikely to be effective if >8 hours since ingestion. Follow with activated charcoal. Don't decontaminate if pet is symptomatic for 14-28 days. warfarin 14 days bromadiolone 21 days Brodifacoum- 28 days Plasma transfusion if coagulopathic. Whole blood transfusion if PCV < 25	Mainstay of treatment is Vitamin K1 antidiote. Dose: 3.5 mg/kg by mouth q 12 hours with a fatty meal to enhance absorption for 14-28 days. warfarin 14 days bromadiolone 21 days Brodifacoum- 28 days If coagulopathic, monitor clotting times and PCV and q 1-4 hours following plasma transfusion. Baseline PT for any asymptomatic pet, then check PT 48-72 hours after last dose of vitamin K. If PT is increased then extend vitamin K course and recheck q 48-72 hrs	If coagulopathic, monitor clotting times and PCV and q 1-4 hours following plasma transfusion. Baseline PT for any asymptomatic pet, then check PT 48-72 hours after last dose of vitamin K. If PT is increased then extend vitamin K course and recheck q 48-72 hrs	Never give vitamin K1 IV: as it can cause anaphylaxis. Vit K is safest and best absorbed orally.
BROMETHALIN RAT BAIT	Neurotoxin which affects Na/K pumps and leads to cerebral edema. Causes vomiting, tremors, ataxia, paresis/paresis 1-7 days after ingestions of sublethal dose. Larger ingestions cause seizures, hyperthermia, depression and death within 4-8 hours.	Sublethal dose: 1.67-2.5 mg/kg Larger doses: 3.65-6.25 mg/kg <ul style="list-style-type: none"> • A bromethalin concentration of 0.01% equals 0.1 mg of bromethalin in each gram of bait. 	Early decontamination with emesis and multiple doses of activated charcoal (q 8 hours for 24-48 hrs) is recommended. Not recommended to treat if patient is obtunded or seizing as they cannot protect their airway.	Anti emetics, gastro-protectants, IV fluids, methocarbamol, anticonvulsants, IV lipid emulsion (within 4 hours of ingestions), mannitol have all been used in these patients.	Monitor for clinical signs improving. Treat as needed for any GI symptoms and neurologic symptoms that develop.	In the U.S., the Environmental Protection Agency has limited the sale of 2nd generation rodenticides. Therefore there may be increasing toxicities with neurotoxin or vitamin D rat baits.



TOP TOXINS



TOXIN	OUTCOME IF UNTREATED	TOXIC DOSE	DECONTAMINATE?	TREATMENT	MONITORING	OF NOTE
CHOLECALCIFEROL (VITAMIN D3)	Found in certain rat baits. Organ dysfunction secondary to tissue mineralization. Weakness, ataxia, anorexia, vomiting, PU/PD.	0.1 mg/kg. Doses greater than 0.5 mg/kg causes soft tissue mineralization secondary to elevated calcium and phosphorous	Clinical signs with doses as low as 0.1 mg/kg. Doses greater than 0.5 mg/kg causes soft tissue mineralization secondary to elevated calcium and phosphorous	IVF at twice maintenance rates for 24 hours to safely administer multiple doses of activated charcoal. Dose 0.1-0.5 mg/kg: cholecalciferol 0.3-1 g/kg PO q 8 hrs for >0.5 mg/kg dose; charcoal with sorbitol q 8 hours for up to 3 treatments	Dose 0.1-0.5 mg/kg: SC fluid therapy and outpatient lab monitoring: baseline calcium (ionized ideal), phosphorus, pcv/tp, bun and creat, lytes, urinalysis 3-4 days. clinically affected pets, GI support as needed, if elev phos: Aluminum hydroxide 30-90mg/kg po with food divided/day Hypercalcemia: bisphosphonates like pamidronate (single IV dose) prednisolone 1mg/kg po q 12 hr, furosemide 2mg/kg q 8-12 hrs)	After ingestion, it's converted to active vitamin D3 which increases calcium and phosphorous gut absorption and absorption from renal tubules and mobilization from bone
ETHYLENE GLYCOL	Acute renal failure within 18-36 hours 3 clinical signs stages: Neurologic symptoms from alcohol inebriation within 30 minutes for 12 hours. Vomiting from EG irritation of GI lining within 6-12 hours Isthmenuria and PU/PD within 3 hours	Toxic dose not established lethal dose of undiluted antifreeze is 4.4-6.6 ml/kg in dogs and 1.4 ml/kg in cats	Yes, induce emesis if ingestion within 1 hours. Charcoal within 1-3 hours of ingestion	Formipazole (4MP) in dogs or ethanol in cats. 4MP (Formipazole) inhibits alcohol dehydrogenase to interfere with toxin metabolism but may be ineffective in cats. 4MP: q 12 hrs x 36 hours 20mg/kg IV slow 15-30 min, then 15 mg/kg IV at 12 and 24 hrs, then 5 mg/kg at 36 hrs. Ethanol (ie vodka): 8.6ml/kg 7% ethanol bolus, 100 mg/kg/hr upto 200 mg/kg/hr CRI Fluid tx support, furosemide for oliguria	Can test for EG at 0.5-12 hours post ingestion, negative results do not mean much, just pos Monitor for severe acidosis, electrolyte derangements, tachypnea, tachycardia, CNS depression, seizures, pulmonary edema AG >25 mEq/L, crystalluria: ca oxalate crystals (monohydrate) hypocalcemia, low serum pH	Mild reversible clinical signs have been found following ingestion of prey/carriorn that consumed the toxin mortality rates for cats as high as 90%, dogs 60-70% Can shine UV or cobalt light on fur around mouth, urine or vomitus to see if it glows.



TOP TOXINS



TOXIN	OUTCOME IF UNTREATED	TOXIC DOSE	DECONTAMINATE?	TREATMENT	MONITORING	OF NOTE
CARPROFEN	<p>Dose dependant effects. Lower doses cause GI side effects (vomiting, diarrhea, GI ulceration).</p> <p>Higher doses can cause acute kidney injury.</p> <p>Really high doses can cause neurologic effects:</p> <p>seizures, stupor.</p>	<p>Cats: 4-8 mg/kg more mild GI signs 8mg/kg or above can cause acute renal failure</p> <p>Dogs: 20 mg/kg: significant GI signs</p> <p>40 mg/kg: Renal effects possible; mid transient ALT and ALP elevations</p> <p>100mg/kg more severe elevations in ALP/ALT and clinical illness</p> <p>Kidney damage possible as low as 48 mg/kg</p> <p>Neurologic signs (seizure, stupor) at high ingestions (280 mg/kg and above studied)</p>	<p>Yes, emesis ideally within 2 hours of ingestion.</p> <p>Then activated charcoal. Multiple doses are beneficial due to enterohepatic recirculation. First dose with sorbitol, then up to 2 more doses without a cathartic q 8 hours.</p>	<p>Aggressive administration of GI protectants for 7 to 10 days. Dogs: use misoprostol for 3-5 days. Don't use in pregnant dogs and humans must wear gloves. Sucralfate 30 min before antacid therapy.</p> <p>Crystalloid fluids at 2x maintenance rate for 48 hours to promote diuresis. If bun/creat are normal at 48 hours can taper fluids over 24 hours and recheck renal values at 72 hours.</p> <p>Antiemetics, SAM-e, +/- antibiotics</p>	<p>baseline CBC, chemistry and UA.</p> <p>For hospitalized pets, daily assessment of BUN, creat, PCV, liver enzymes, urine output and sediment, hydration status.</p> <p>Normal values related to hepatic and renal function measured 72 hours post exposure are not expected to elevate further.</p> <p>Monitor for signs of ulceration (melena), abdominal pain, anorexia, anemia</p>	<p>Nearly 90% of the drug is bioavailable after ingestion. Peak plasma concentration within 1-3 hours. Carprofen is highly protein bound, so it can increase other protein bound drugs given! Elimination half life in dogs is 8 to 18 hours. Cats have a much higher half life due to limited metabolism via glucurionidation.</p>
FELINE PERMETHRINS	<p>Ataxia, muscle tremors, hypersalivation, seizures and hyperthermia.</p> <p>Permethrins are spot ons for dogs. Any amount can cause toxicity in cats, even close contact between a treated dog and a cat</p> <p>seizures, start to wash off the product with liquid dish soap like Dawn</p>	<p>No. Topical side effects don't require emesis or activated charcoal.</p> <p>However, after immediately treating any severe trembling or seizures, start to wash off the product with liquid dish soap like Dawn</p>	<p>Stabilize tremors with IV methocarbamol or diazepam if seizing, then immediately bathe with dish soap like Dawn</p> <p>If methocarbamol /diazepam, cooling for hyperthermia does not resolve symptoms then there are anecdotal reports of response to intralipid IV emulsion.</p>	<p>Monitor for improvement in tremoring, development of seizures. Monitor for hyperthermia. IV fluid support to help reduce hyperthermia and protect kidneys from rhabdomyolysis</p>	<p>With prompt treatment clinical signs can resolve within 24-72 hours.</p> <p>Don't forget to try intralipid if symptoms cannot be controlled with more traditional therapies.</p>	

Vet Helpful Notes
Catline Refine

USE OF CHARCOAL

A cathartic is often added to activated charcoal to speed transit through the GI tract. The longer charcoal sits in the GI tract, the longer it has a chance to pull fluid into the GI tract and increase risk for hyponatremia. If charcoal sits in the GI tract too long, it may possibly release the toxin and render the charcoal ineffective. Sorbitol can increase cramping and diarrhea and should not be used if a pet is dehydrated or having diarrhea. Generally, it is recommended to give charcoal with sorbitol as the first dose and follow up doses to be regular charcoal.



TOP TOXINS



TOXIN	OUTCOME IF UNTREATED	TOXIC DOSE	DECONTAMINATE?	TREATMENT	MONITORING	OF NOTE
MARIJUANA	Dogs who ingest or inhale THC have clinical signs of stupor, ataxia, possible seizures, urine dribbling and GI symptoms. Fatalities are rare.	A true LD 50 (toxic dose) has not been established, but dogs have ingested 3000 to 9000 mg/kg without fatality.	Yes emesis ideally within 2 hours of ingestion. Then activated charcoal if patient is alert.	Treatment is mostly supportive care for clinical signs such as IVF therapy and thermoregulation. Diazepam as needed to control agitation, tremors or seizures. THC is highly lipid soluble, so there is anecdotal evidence that intralipid can be beneficial: initial bolus of 20% lipid emulsion (15 mL/kg), followed by a constant-rate infusion (0.05-0.25 mL/kg/min; not to exceed 10 mL/kg) over 30 minutes.	Clinical signs develop within 1-3 hours and can last 36-48 hours. Monitor Resp rate, cardiac function and body temperature. Look for improvement in neurologic symptoms in most dogs within 24 hours, but up to 72 hours.	The accuracy of urine drug screens have not been validated for detecting THC in dogs. THC is mostly eliminated through the feces, only 20% is eliminated through urine.
SSRI ANTIDEPRESSANTS	Increased serotonin to the nervous system causes: lethargy or agitation, vomiting, ataxia, tremors, seizures, hypertension and tachycardia. Death is rare however.	The minimum lethal oral dose of sertraline in dogs is reported as 80 mg/kg. Paroxetine doses up to 3 mg/kg have no effect on heart rate: mild ECG changes at 10 mg/kg intravenously. The lethal dose for paroxetine is unknown. The minimum lethal dose of fluoxetine in dogs is greater than 100 mg/kg	Yes, emesis ideally within 2 hours of ingestion. Then activated charcoal if patient is alert.	Cyproheptadine is a serotonin antagonist: give orally/rectally 1.1 mg/kg in dogs. If cyproheptadine is not effective, can use diazepam or phenobarbital. IVF to support renal function and BP. Tachycardia (heart rate over 180) can be controlled with propranolol at a dose of 0.04 to 0.06 mg/kg given slowly IV.	Monitor for CNS stimulation: agitation, depression, ataxia, tremoring, seizures, hyperthermia, diarrhea.	These drugs are highly protein bound so IVF therapy does not do much to enhance excretion. When dogs ingest more than one kind of antidepressant, the risk of serotonin syndrome goes up. Signs generally resolve in 12 to 24 hours and prognosis is generally good with supportive care.

EMETIC TREATMENT

At home can use: 3% hydrogen peroxide. Use an unopened bottle (or it may not be effective). Dose: 2 mL/kg, no more than 45 mL.

Apomorphine 0.04 mg/kg IV, 0.08 mg/kg SQ or 0.25 mg/kg in conjunctival sac. Clevor eye drops (ropinoreole) is an FDA approved eye drop for inducing emesis in dogs over 1.8 kg and older than 4.5 months of age. Following emesis, it's a good idea to administer maropitant as you typically will want to give the patient activated charcoal PO.

VetHelpfulNotes
CatLine
DogsLine
Reline

All About Seizures

Seizure Basic DDX

First seizure < 1 year : PSS, Hydrocephalus, Toxin

First seizure 1-5 years Epilepsy, Toxin, late blooming PSS

First seizure > 5 years of age: intracranial cause: neoplasia, infarct, toxin

EPILEPSY IS LIKELY IF:

- Seizures developed between the age of 6 months and 6 years
- Normal between seizures
- No metabolic causes found (hypoglycemia, hypocalcemia, liver disease (shunt, liver failure) electrolyte disturbances, uremic effects on the brain from renal failure, hypoxia, hypothyroidism)
- Ideally meet these rules AND normal MRI imaging and CSF

When to start Anti-epileptic Drugs (AED)?

1. Structural brain lesion present or history of brain disease or injury
2. Cluster seizures
3. A seizure longer than 5 minutes
4. Three or more seizures in 24 hours,
5. Two or more seizures in 6 months
6. Prolonged, severe seizures or unusual post-seizure (post-ictal) periods

Which drug to choose?

No evidence that any single anti-epileptic drug (AED) works better in people.

Drug selection based on tolerability, cost, ease of treatment etc.

Time to reach Steady State Drug Levels

When to check efficacy and drug concentrations?

Drug concentrations should be checked after steady-state concentrations are achieved

This happens after 5 drug half-lives (varies from drug to drug).

DRUG	TIME TO REACH STEADY STATE
Bromide	4 months
Phenobarbital	2 weeks
Zonisamide	2 weeks
Levetiracetam	Rapid onset, but half life too short to reach steady state

Because the half life of bromide is so long, a level at 1 month is recommended (when the level should be 50% of the steady state level)- this will allow time to adjust the dose if the target level is not likely to be reached.

Because Levetiracetam has a half life too short for steady state concentrations, it's recommended to monitor after 1 week to see how pet is responding

PHENOBARBITAL

Rapidly absorbed within 2 hours and Maximal plasma concentration in blood within 4-8 hours of oral dose
Metabolized by the liver and 6 weeks after starting, levels may fall due to increasing activity of p450 liver enzymes. Relatively inexpensive, well-tolerated

DOSE:

DOGS : 2.5mg/kg PO BID

CATS: 1-3 mg/kg PO BID or 1/2 to 1 tablet of 15mg/tab PO q 12hr



MONITORING:

Level at 2 weeks (steady state) and at 6 weeks (new steady state) and then q 6 months thereafter
Once drug reaches steady state levels don't fluctuate much throughout the day, but if you want trough levels, check in the a.m. before dosing on a fasted dog. For Peak levels, check the level 4-5 hours after dosing.

If >2 seizures in this period recheck level and then 2 weeks after each dose change

TIPS

** DO not use a serum separator tube (this will falsely LOWER pb levels measured). Use a plain red top tube ***

*** should not use in pets with liver dysfunction*** Instead choose Bromide and/or Levetiracetam

*** increases the clearance of other AED's like Levetiracetam and zonisamide***

*** Hepatotoxicity is not reported in cats on Pb so if you see elevated liver values, investigate ASAP ***

Eight studies combined showed:

82% of dogs achieved >50% seizure reduction rate

31% cumulative seizure- free

15% failure to respond

Adverse effects: hyper-excitability, restlessness, sedation: typically resolve within 1-2 weeks

Polydipsia, polyphagia, increase in ALP as soon as 2 weeks after initiating ; labwork recc q 6 months to monitor for hepatotoxicity.

Rare, idiosyncratic reaction: IMHA, neutrophenia, thrombocytopenia- within 1st 6 months of tx

POTASSIUM BROMIDE (KBR)

Slowly metabolized in the dog with median elimination half life of 15 day

DOSE: Starting dose of 40 mg/kg/day recommended

MONITORING:

at steady state between 6-12 weeks, then annually or if >3 seizures before next scheduled evaluation

Can collect a sample at any time point< 2 hours after dosing

*** for more rapid drug levels, you can LOAD dogs with 600 mg/kg of drug over 24-48 hours while also giving a maintenance dose of 30 mg/kg/day. 24 hours after finishing the 24-48 load, most dogs will be at therapeutic drug levels*** Caution here is severe sedation and ataxia that takes time to improve

TIPS:

DIET affects drug concentrations!!! So keep the diet the same (high chloride diets result in excess renal clearance and lowered drug concentrations!!)

Serum Chloride levels are often falsely elevated as some machine's cannot discern chloride and bromide ions

DON'T use KBR in cats: can cause SEVERE ASTHMATIC REACTION

DON'T use in dogs prone to PANCREATITIS

[ETSY.COM/SHOP/VETHHELPFULNOTES](https://etsy.com/shop/vethelpfulnotes)

74% of dogs >50% seizure reduction

52% of dogs were seizure-free in 6 month treatment period

Adverse effects: polydipsia, polyphagia, lethargy, ataxia with increasing doses, gastric irritation

caution with renal disease as there may be decreased clearance

LEVETIRACETAM (KEPPRA)

No known drug interactions for Levetiracetam

Serum concentrations of this drug are not routinely measured based on the wide therapeutic index and short half life. The drug is well tolerated with infrequent adverse effects.

DOSING:

Cats: 20mg/kg po q 8 hours; or one 500mg extended release tablet daily
Dogs: 20mg/kg po q 8 hours, up to 30mg/kg po q 8 hours

TIPS:

Can be used in cats as a second agent when phenobarbital is ineffective OR as the sole therapy if phenobarbital is not well tolerated. Also useful to send home for intermittent use if needed following break through seizures.

Typical tablets are immediate release and must be dosed three times a day.

May utilize extended release tablets to ease dosing to BID. When using extended release tablets, do not split or crush.

The smallest tablet is 500mg which makes using ER in small dogs difficult.

Also comes as a liquid 100 mg/ml solution

Adverse effects: generally well tolerated with infrequent adverse effects: sedation, hyperactivity, ataxia are possible

ZONISAMIDE

Typically just used for dogs; limited use in cats with anecdotal success. Metabolized mostly by hepatic enzymes.

Zonisamide clearance is increased by almost 50% when used with phenobarbital.

Serum zonisamide level should be monitored 1-2 weeks after treatment initiation or dosage adjustment (peak is 3-4 hours after zonisamide administration).

DOSING:

Dogs 5-15 mg/kg PO q 12 hours; average dose is 10 mg/kg PO BID

Cats refractory to phenobarbital: 6-17 mg/kg PO ONCE daily

Available forms: 25 mg, 50 mg and 100 mg capsules

Adverse effects: sedation, ataxia, vomiting, increased ALT and decreased T4 possible; it is a sulfonamide drug but is unlikely to cause KCS

Breakthrough Seizure Treatment at Home

May add in Keppra 20 mg/kg every 8 hours or 30mg/kg ER q 12 hours (dogs) for 24 hours past the last seizure.

CLUSTER BUSTERS !!!!

To stop cluster seizures at home, may give Midazolam in pre-loaded plastic syringes topped with a nasal atomizer (available on amazon).

Dose: 0.5mg/kg intranasally up to 3 times in a 24 hour period or hourly for 3 hours

OR rectal Valium dosed at 1 mg/kg once an hour for 3 hours after a seizure

OR

clorazepate 1 mg/kg po TID x 2 days following a seizures



EMERGENCY SEIZURE MANAGEMENT

When a seizing pet arrives, check a spot BG immediately and provide diazepam or midazolam 0.25 mg/kg IV

BG <40 Give 0.5-1ml of 50% dextrose. Dilute it first 1:3 with saline.

Can repeat the benzodiazepine up to 3 X in the first 10-30 minutes

If continued seizures, start a phenobarbital load (if the patient is not already on PB)
Give 20 mg/kg IV divided into 4-5 mg/kg IV doses every 30 minutes to 1 hour for 4 doses

If seizures continue to be a concern, you may need to start a CRI:

Diazepam CRI: 0.5 mg/kg/hr for 6-10 hours

Then reduce to 0.25 mg/kg/hr for 6-10 hours

Or

Propofol CRI: 6-12 mg/kg/hr for 6-10 hours

Then wean by 50% for 6 hours, then STOP

Continue maintenance Phenobarbital at 2 mg/kg IV BID



Random Notes

Breeds known to have a higher incidence of refractory epilepsy:

German Shepard

Australian Shepard

Bull Terrier

Border Collies have a 24% incidence of a mutation in the MDR1/ABCB1 gene which causes resistance to PB

Other Collie breeds may have this as well

In breeds known for refractory epilepsy (this means adequate seizure control has not been achieved with at least 2 anticonvulsant drugs administered simultaneously at maximum/safe drug levels), you might initiate treatment with TWO drugs.

What is considered "good control" of seizures? At least 50% longer duration between seizures

When to consider potentially weaning off an anticonvulsant entirely?

If a pet is seizure-free for at least 1 year after starting AED. However, it can be risky to wean off an AED in a patient with a known structural brain disease or a pet whose seizures were hard to control initially.

HEART FAILURE

MYXOMATOUS MITRAL VALVE DISEASE CLASSIFICATION

STAGE A

Dogs with a higher risk of heart failure but no structural abnormalities and no heart murmur present. No drug or diet recommended.

STAGE B1 & B2

B1:

Asymptomatic. Structural heart disease is present but no radiographic or echocardiographic evidence of cardiac remodeling. (ie heart murmur only)

Treatment: No drug or diet recommendations yet.

Recommend re-evaluation by chest x-rays or echo in 12 months.

B2: 30% of dogs in B2 will progress to CHF

Asymptomatic. Structural heart disease is present and LA and LV enlargement sufficient to recommend treatment before onset of clinical symptoms.

- heart murmur > or= 3/6
- La:Ao > 1.6
- VHS >10.5

Ideally, all these criteria would be met before initiating life long treatment. Or evidence of interval change in cardiac size on radiography

Adding Pimobendan at Stage B2 delays progression to CHF by about 15 months and increases overall survival times!!

Treatment of Stage B2

Pimobendan 0.2-0.3mg/kg po q 12 hr

ACEI therapy 0.5mg/kg PO q 12 hr

Dietary therapy

STAGE C

Chronic, home based therapy:

Furosemide to effect: often 2mg/kg q 12 hr

Some clinicians substitute torsemide at 5 to 10% of furosemide dose (0.1-0.3mg/kg q 24hr) for dogs whose response to furosemide in hospital was not great.

Measurement of creatinine, BUN, lytes 3-14 days after initiating furosemide at home in CHF.

ACEI : continue or start 0.5mg/kg po bid

Pimobendan 0.25-0.3mg/kg po bid

ADD:

Spironolactone 2mg/kg PO q12-24 (better aldosterone antagonists)

If **Afib** is present, **diltiazem +/- digoxin** titrated to achieve heart rate control (< 125bpm)

Cough suppressants and **bronchodilators** as needed.

Diet to maintain weight, restrict sodium intake, supplement if needed potassium (more common w/ torsemide); monitor serum magnesium if needed.

STAGE C CONT'D

C:

Dogs with past or current heart failure associated with structural heart disease.

Diagnosis: CXR and preferably echo.

Acute hospital based therapy:

Furosemide 2mg/kg IV or IM followed by 2mg/kg iv or im until RR and effort substantially improved or until total dosage is 8mg/kg within 4 hours.

Life threatening pulmonary edema (froth, severe dyspnea): Furosemide CRI : 0.66-1mg/kg/hr after initial 2mg/kg bolus.

Pimobendan 0.25-0.3mg/kg po bid

Oxygen supplementation

Centesis of effusions (thoracic, abdominal if present and impeding respirations)

Butorphanol for anxiety 0.2-0.25mg/kg IM or IV or **buprenorphine 0.01mg/kg and ace 0.01mg/kg**, or morphine, hydrocodone

If patients fail to improve with these measures:

Dobutamine 2.5-10 ug/kg /min CRI (starting low and increasing incrementally) to improve ventricular function. Monitor for tachycardia or ectopy with continuous ECG. **CRI of Nitroprusside 1-15 ug/kg /min for up to 48 hours** for life-threatening or poorly responsive pulmonary edema

- No beta blockers in the face of active CHF

- No Nitroglycerin with active CHF

STAGE D

if >8mg/kg of furosemide needed
then move to Stage D .

Acute hospital based therapy:

If creatinine < 3mg/dl can use Furosemide initial bolus 2mg/kg/IV followed by additional boluses or a CRI 0.66-1mg/kg/h until RR and effort decreases or for maximum 4 hours.

OR use Torsemide at 5-10% the furosemide doses (0.1-0.2mg/kg q 12 to 24 hr).

Cavitory centesis as needed to relieve respiratory distress/discomfort.

Oxygen therapy

May need vigorous afterload reduction in patients that can tolerate arterial vasodilation:

Nitroprusside 1 ug/kg/min titrated q 15-30 min to max of 10-15 ug/kg/min for 12-48 hours. Continuous ECG and BP measurement.

PO drugs: **hydralazine** 0.5-2mg/kg po with nitroprusside, and titrating to effect.

Re-evaluate creatinine 24-72 hours later.

Sildenafil if pulmonary hypertension is present: 1-2mg/kg po q 8

May add in a third dose of **pimobendan 0.3mg/kg PO q 8 hour**.

Some also add in a bronchodilator to treat pulmonary edema in hospitalized patients

HEART FAILURE

(CHRONIC VALVULAR HEART DISEASE)

STAGE D

Chronic, home based therapy:

Furosemide to effect: Some will increase to TID dosing

Or switch to torsemide if no longer adequately responsive to furosemide. Dose at 5 to 10% of furosemide dose (0.1-0.3mg/kg q 24hr) for dogs whose response to furosemide in hospital was not great.

Measurement of creatinine, BUN, lytes 12-48 hours after increased furosemide or torsemide dose

ACEI : continue or start 0.5mg/kg po bid



Pimobendan increased by some to a **third daily dose at 0.3mg/kg**

Spironolactone 2mg/kg po q12-24 (better aldosterone antagonism)

Some may use additional afterload reducing agents with **hydralazine** or **amlodipine**.

If **Afib** is present, **diltiazem +/- digoxin** titrated to achieve heart rate control (< 125bpm)

Sildenafil 1-2mg/kg po q8 hours to address **pulmonary hypertension** and clinical signs secondary to exertion and ascites management

Cough suppressants and bronchodilators as needed for intractable cough

Diet to maintain weight, restrict sodium intake, supplement if needed potassium (more common w/ torsemide); monitor serum magnesium if needed.

Heart failure: failure of the heart to pump and distribute blood appropriately leading to tissue hypoxia.

Congestive Heart Failure: cardiac dysfunction resulting in increased venous /capillary pressures which then cause edema or effusions.

80% of dogs that develop CHF will only survive 1.5-2 years from diagnosis.

80% of CHF in dogs is due to Mitral Valve disease.

Left heart failure: life threatening condition caused by venous congestion in the lungs caused by increased left atrial pressure. This pressure occurs when the left ventricle can't adequately fill or eject blood.

Common conditions in dogs and cats leading to left sided CHF:

1. **Myxomatous mitral valve disease:** causes increased LA pressure . Most common in Cavaliers, Chihuahuas, Min Pin, Yorkies and other small breed dogs

2. **DCM (dilated cardiomyopathy):** causes increased LA pressure. Most common in : Dobies, Boxers, Danes, G sheps, Labs and other large and giant breed dogs

3. **HCM: (hypertrophic cardiomyopathy)**

Thickening of the left ventricle. Most commonly affects cats: Maine coon, Persian and American domestic shorthair

Right heart failure: increased pressure in the vena cava and right atrium and pulmonary vessels.

This can be due to:

1. myocardial failure: DCM, pulmonic insufficiency (blood leaks back into the RA from the pulmonary arteries)

2. Volume overload into RA from valvular regurgitation or obstruction: Tricuspid insufficiency (blood leaks back into RA as it's supposed to move into the RV) or obstruction (Pulmonic stenosis or heart worms blocking pulmonary arteries)

3. Increased myocardial stiffness: pulmonary hypertension, constrictive pericarditis or pericardial effusion

Classically right sided heart failure leads to ascites. On physical exam, you may notice bounding jugular veins and a distended abdomen.

Pulmonary hypertension can cause poor cardiac output and syncope. Dogs with chronic pulmonary disease (cough) can develop pulmonary hypertension from the secondary fibrosis and airway disease.

HEART FAILURE

DIAGNOSIS

Diagnosis of heart failure is best identified by chest x-rays.

Dogs: Classic findings of CHF is interstitial-alveolar infiltrates in the perihilar and/or caudo-dorsal lung fields in dogs.

Cats: Ventral or diffuse interstitial to alveolar pattern. Can see pleural effusion via fissure lines, retraction of lung lobes and effacement of cardiac silhouette. (The heart is best evaluated on a VD view if pleural effusion is present because the lungs fall away from the heart). * In cats, heart failure can have a variety of appearances *

Both: Left atrial enlargement and generalized cardiomegaly. These findings are more difficult to see in cats but a valentine shaped heart on the VD/DV view and a VHS >8 indicates cardiac enlargement. *Lack of cardiac enlargement on chest x-rays does not rule out CHF in cats.*

NT-Pro BNP testing



A ProBNP snap test can be a useful bedside test to help discern respiratory disease from cardiac disease in a dyspneic cat. If the snap test spot is the same color as the control spot the ProBNP is estimated to be 150-200pmol/L If it is darker, the estimate is the ProBNP is >200 pmol/L

Good to know:

The quantitative snap gives the most helpful information- but is a send- out test.

MOST CATS with:

SEVERE HCM have a ProBNP >900

MODERATE HCM have a ProBNP 270-900 pmol/L

A value above 270 pmol/L is considered the cut-off for CHF in cats. In one study, 90% of cats in HF had a proBNP >300.

A value above 100 pmol/L is abnormal and an echo is recommended to determine if there is subclinical disease.

Heart murmurs and cats:

- Approximately 15% of cats (1 in 6) have heart disease
- Only about 30% of cats with heart disease will have a heart murmur!
- 1 out of 4 apparently healthy cats may have a murmur, but only 25% of these cats will have evidence of cardiomyopathy by echocardiogram

Utility of proBNP to detect subclinical heart disease in cats:

proBNP measurement thus far has not been proven to be reliable to detect HCM in clinically normal Main Coon Cats!

However, as a general rule, a proBNP in an apparently healthy cat that is under 100 means that clinically significant heart disease is UNLIKELY

An even better cut off is to be under 44pmol/L

The GOLD STANDARD to identify cardiac disease remains an echocardiogram study.

NT proBNP as a Canine Screening Tool for heart disease in apparently healthy dogs:

No clear benefit to identify dogs with mitral valve disease. A heart murmur in a small dog does a better job identifying likely MMVD.

Screening tool for DCM? Not accurate enough to allow breeders to use this to screen for DCM. Still need to rely on Echo and Holter monitoring. An NT ProBNP >900 improved accuracy in detecting subclinical DCM but was not sensitive enough to detect all the subclinical DCM dogs.

ProBNP: Not useful to detect subclinical ARVC

Boxer Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC): an inherited (genetic) ADULT onset heart muscle disease that leads to arrhythmias and can cause CHF and heart failure. Genetic testing can determine if a Boxer has inherited one copy (heterozygous) or two copies (homozygous) of the mutant gene.

Heterozygous/Homozygous: at 3 years of age an annual Holter monitor screening is recommended. Homozygous: do not breed.

Doberman Pinscher: One of the most common breeds to develop Dilated Cardiomyopathy (DCM).

There are two identified mutations that can lead to this disease- although a Doberman without the mutation can still develop the disease. Recommendations are to monitor all Dobermanns starting at 3 years of age with yearly Holter monitoring and echocardiograms.

Two subsequent Holters with 50-300 VPC's is indicative of DCM.

If costs are prohibitive, then yearly proBNP testing and 5 minute ECG recordings are recommended.

Canine and Feline Diabetes Mellitus

Insulin	Trade Name	Syringe
Insulin Zinc (Lente)	Vetsulin	U-40
	Humulin N Novalin	U-100
PZI	ProZinc	U-40
	Lantus	U-100

How to Suspend

Roll
PZI
NPH



Shake
Insulin Zinc



No mixing
Glargine



Can use insulin up to 3-6 months if kept in fridge and no discoloration or flocculent changes (regardless of expiration)

Canine Treatment

Porcine Zinc (Vetsulin)

0.25 u/kg q 12 hour to start, round to the nearest whole U

- most dogs well controlled on 0.5 U/kg

other option

NPH (Humulin N, Novalin N)

0.25 u/kg- 0.5 u/kg q 12 hr

Larger dogs: best at lower end to start

Smaller dogs: best at higher end to start

Canine/Feline Diabetes

Diabetes Mellitus: Protracted Hyperglycemia due to loss or dysfunction of pancreatic Beta cells OR decreased sensitivity of tissues to insulin OR both.

Canine diabetes:

Most similar to Type 1 diabetes. It is most often secondary to immune mediated destruction of the pancreas or vacuolar degeneration within the pancreas or pancreatitis.

Remission is not anticipated

Intact female dogs are an exception as spaying may reverse insulin-resistance of diestrus.

Risk factors dogs:

Obesity, Cushing's, hypothyroidism

Breed risk: Australian Terriers, beagles, Samoyed, Keeshonden

*** PEARL *** always counsel owners that diabetic dogs typically go blind from cataracts within the first year. Cats do not.

Feline diabetes:

Most similar to Type II diabetes and remission is possible with insulin and diet (mostly within the first 6 months).

Cats usually suffer from insulin resistance, islet amyloidosis or lymphocytic/plasmacytic pancreatitis.

Risk factors in cats:

Acromegaly, CKD, obesity

Breed risk: Burmese

Risk factors for dogs and cats:

dental disease, systemic infection, pregnancy/diestrus, medications: steroids, progestins, cyclosporine)

Feline Treatment

Lantus (Glargine)

0.5 U/kg q 12 hr : BG >360 at diagnosis

0.25 U/kg : BG <360

Most cats started on 1-2 U BID

Some clinicians start all cats out on 1 U BID regardless

other option

PZI (ProZinc)

0.5 U/Kg q 12 hr

typical starting dose is 1-2 U/cat

acceptable window: dose q 12 hr +/- 2 hours

Canine and Feline Diabetes Mellitus



Diagnosis of DM:

Clinical signs: PU/PD/PP, weight loss; sometimes lethargy, weakness

Cats often develop impaired ability to jump and/or dropped hocks due to peripheral neuropathy from glucose toxicity to the neurons

Labwork: hyperglycemia (>250), glucosuria, stress leukogram (neutrophilia, lymphopenia, eosinopenia), elevated cholesterol and triglycerides; in dogs : elevated ALP and ALT are common.

Goal of Treatment: Controlling BG below the renal threshold for as much of the day as possible in order to improve clinical signs of DM and avoid clinically significant hypoglycemia!!!

Renal Threshold: <200 mg/dl in dogs; <250-300 mg/dl in cats!

Example Diet Choice in Dogs: Hills W/D (high fiber diet), RC Diabetic, Purina DM

Example Diet Choice in Cats: Purina DM or Hills MD (high protein, low carb), RC Diabetic: wet food preferred

Monitoring

Newly Diagnosed:

BGC= Blood Glucose Curve



BGC 7-14 days after treatment started; q 2 hours for 12 hours

if a cat is on lantus can check bg's q 3-4 hours for 12 hour interval

** measure BG hourly if BG drops below 150 mg/dl during the curve **

BGC If pet is exhibiting clinical signs (pu/pd, weight loss), increase insulin by 10% or 1/2 U whichever is greater) and recheck in 1 month

If the pet is exhibiting clinical signs and the dose is > 0.5 U/kg BID in dogs or 2 U/cat BID then run a BGC

Curve Results: Nadir above 150

Nadir >150mg/dl with no clinical signs: if maintaining or gaining weight: no change to dose; consider stress hyperglycemia, consider repeat BGC in a month. If able, check BGC at home.

Nadir >150mg/dl with no clinical signs: but pet has lost weight, consider increasing dose by 10% or 1/2 U/dose and BGC in 7 to 14 days.

Nadir > 150 mg/dl with clinical signs :

Dogs: increase dose 10-25% and BGC in 7-14 days

Cats: increase dose 0.5-1 U/dose BGC in 7 to 14 days



Nadir optimum 80-150mg/dl with no clinical signs: maintain dose

Nadir optimum 80-150mg/dl with clinical signs: if performed in hospital, consider BGC at home or placing a continuous glucose monitor

Canine and Feline Diabetes Mellitus



Curve Results: Nadir below 80 mg/dl

If BG is every <80mg/dl the insulin dose should be reduced regardless of symptoms!!

Nadir <80 mg/dl

-With clinical signs of hypoglycemia:



CAT: BGC is never >250mg/dl in a cat: consider remission. Discontinue insulin and monitor for clinical signs of DM, recheck urine glucose and or BGC in 3-7 days

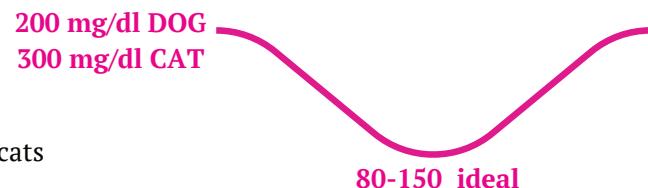


In 3-5 days if BG remains <250mg/dl and/or urine glucose negative, continue to monitor for recurrence of symptoms and remain off insulin

If clinical signs of DM recur and urine glucose positive, start cat at 1 U/cat q 24 hours and BGC in 7 to 14 days.

DOG: Discontinue insulin for 12 hours. Don't re-start until BG>250mg/dl, and decrease insulin by 10-50%. BGC in 24 hours. If hypoglycemia persists, repeat as above.

If no longer hypoglycemic, maintain the new lower dose. BGC in 7 to 14 days.



NOTES

First aim for regulation is an acceptable Nadir!

Ideal peak is no higher than 200 mg/dl in dogs and 300 mg/dl in cats

Somogyi (over-swing) phenomenon results from BG dropping under 60 mg/dl or drops too quickly. This causes counter-regulatory hormones to act to increase BG (cortisol, epinephrine, glucagon). If Somogyi is observed, insulin dosage should be decreased. Once nadir > 80 mg/dl then Somogyi won't be triggered.

Alpha Track II is the portable BG monitor of choice for the most accurate results in dogs and cats

Continuous Glucose Monitors (CGM) like the FreeStyle Libre are being used more and more. They collect interstitial glucose measurements with acceptable correlation to blood glucose levels. Most CGM last 5-10 days after placement in veterinary patients.

Urine Glucose Home Monitoring for Cats

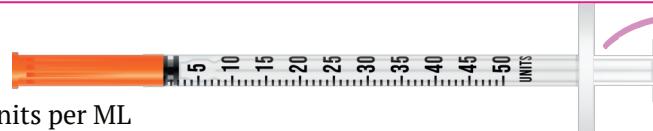
(UG dipstick measurements in dogs have low accuracy)



Negative for glucose for 2 days in a row: concern that insulin is too high. Reduce insulin by 0.5-1 unit and recheck in 2-3 days

1000-2000+ mg/dl color change : cat should have clinical signs. Increase insulin by 1 unit BID and recheck in 5-7 days.

Insulin syringes:



U-100 syringes hold 100 units per mL

U-40 syringes hold 40 units per mL

1 unit from a U-40 syringe is the same as 2.5 U-100 units (e.g. 2 U-40 units is 5 units from a u-100 syringe)



0.5 cc
U-100 syringe
Holds 50 units

When ordering U-100 syringes, it's typically best to order a 0.3 cc syringe. The syringe above is a 0.5 cc u-100 syringe. This means it fits 50 units and each unit dash is closer together. A 0.3cc u-100 syringe is easier to see the separation of units and makes drawing up 1/2 unit possible to eye-ball. Most humans need a syringe that holds more units, but cats don't!

ALL ABOUT DKA

DIAGNOSIS

Hyperglycemia > 200 mg/dl, **metabolic acidosis** on a blood gas, evidence of **glucosuria** with **ketones** in the **urine** OR blood **serum**.

Hyperglycemic Hyperosmolar Syndrome Diagnosis: Severe Hyperglycemia >600mg/dl or 33.3 mmol/L, hyperosmolality > 350 mosm/kg, severe dehydration, lack of ketones, mild acidosis, CNS depression. Long term survival with HHS is very low, around 12%. Most common in cats, not dogs. Focus is on rehydration first, then gradual use of insulin. Avoid dropping BG too quickly or cerebral edema can occur.

DKA frequently occurs in newly diagnosed diabetics. Prolonged hyperglycemia and insulin deficiency causes the body to breakdown fat for energy. This is because insulin is not available to drive sugar into the cells. Free fatty acids are converted into ketones. When fat is mobilized, there is an excess of free fatty acids and fat is then stored in the liver. Excess ketones build up. Ketones are strong acids and lead to metabolic acidosis. Ketones and glucose in the urine lead to the loss of sodium, potassium and water into the urine. This leads to dehydration and electrolyte abnormalities.

LABWORK ABNORMALITIES

CBC: there may be a non regenerative anemia, leukocytosis, left shift

Biochemical panel: Hyperglycemia, ALP and ALT, AST May be elevated (from storage of excess fat in the liver or even concurrent pancreatitis), cholesterol may be elevated. Azotemia may also be present because of dehydration.

Low Na, Low Cl can occur secondary to elevated glucose causing water retention and subsequent dilution of Na/Cl.

Metabolic acidosis shifts phosphorous extracellularly. However once insulin is started, this drives phosphorous back into the cells and can quickly drop the phos levels! Very low phosphorous (< 0.5mmol/L) can cause hemolytic anemia, seizures and poor cardiac function.

UA: glucosuria, proteinuria, ketonuria. UTI's are common. Ketones may not be detected, as the most common ketone: Beta hydroxybutyrate is not detected on the dipstick

Blood gas: metabolic acidosis (decr bicarb and pH)

Serum Osmolality: can be elevated.

Calculated with this equation:
Serum OSM: $2(\text{Na} + \text{K}) + (\text{Glucose} / 18) + (\text{Bun} / 3)$
normal is typically 280-310

Corrected serum Na: Na falls temporarily in response to hyperglycemia. To correct for the sodium use the equation:

Measured Na meQ/L + 0.016 x serum glucose in mg/dl - 100

UNDERLYING DISEASES

If your patient is NOT a newly diagnosed diabetic, chances are an underlying disease pre-disposed them to develop dysregulation of their diabetes and subsequent DKA. Here are some common causes to screen:

DOG 71%

CAT 93%

Pancreatitis	UTI	Hepatic lipidosis	Neoplasia
Cushings	Hypothyroidism	cholangiohep	pancreatitis
Neoplasia	pneumonia	UTI	CKD

Ideal diagnostics: CBC, chem, UA + culture, coags, VBG, doppler BP, chest/abdomen imaging

TREATMENT

1. Correct Dehydration: Isotonic Fluid Therapy (LRS or 0.9% NaCl or Norm R) Good Rule of Thumb: pick a fluid with a Na close to the serum level (after correction).

If $\text{Na} < 125-120 \text{ mmol/L}$ or $> 170 \text{ mmol/L}$ only adjust Na by 0.5 mmol/hr or $10-12 \text{ mmol/day}$

1. Shock fluid therapy may be warranted if the patient has cardiovascular shock (hypotension, brady/tachycardia).

Full shock dose is 90 ml/kg: start with 1/4th to 1/3rd value and reassess parameters.

Correct dehydration, provide maintenance needs and replace ongoing losses over 6-24 hours.

a) % Dehydration $\times \text{Bw in Kg} \times 1000 = \text{ML}$

add that to

b) 20ml/kg/day insensible losses

add that to

c) 20 to 40 ml/kg/day maintenance sensible losses

add that to

d) any estimated fluid losses from vomiting, diarrhea, polyuria

2. Electrolyte supplementation

Monitor serum K and Phos q 4-6 hours until K is in normal range and Phos is > 1.5 , then q 6-24 hrs. See charts for more.

3. Regular insulin therapy. See chart for more.

First, you hydrate a patient for several hours and this will help to initially drop the BG and correct dehydration. There is faster resolution of DKA with starting insulin within 6 hours but no change in time to discharge.

CLINICAL SIGNS

- Pu/PD
- Inappetance
- Nausea
- vomiting
- Muscle wasting
- Weight loss
- Lethargy
- Fruity odor of ketones
- Possible abdominal pain

ALL ABOUT DKA

MONITORING

Monitor:

Physical exam: resp rate/effort, HR, pulse quality

Hydration status, appetite

Electrolytes: K, phos, +/- Mg, blood gas and acid/base status

Ketones: serum, urine: ketones take longer to clear from the urine, so monitoring serum ketones can be more sensitive for resolution

Urine dipstick does not measure the most common ketone: betaHB so under-estimates ketosis; you can add serum to your urine dipstick. If the pad does not change color, some advocate to add a drop of hydrogen peroxide to the test pad after to see if the pad changes color. H2O2 converts BetaHb to acetoacetate which is detected on the dipstick.

When to switch to long acting insulin?

- If the patient is well-hydrated
- Ketones are gone or only trace
- Patient is eating consistently
-

Start long acting insulin, but keep the pet on 1-2 more hours of regular insulin infusion before stopping

K+ AND PHOS SUPPLEMENTATION

Serum K concentration (mEq/L)	KCL dose meQ/L	rate of K+ supplementation
> 3.5 (maintenance)	20	0.05-1 mEq/kg/hr
3-3.5	30	0.2 mEq/kg/hr
2.5-3	40	0.3 mEq/kg/hr
2-2.5	60	0.4 mEq/kg/hr
<2	80	0.5 mEq/kg/hr

Serum Phos concentration (mg/dl)	K phos dose
2-2.5	0.03 mmol/kg/hr
1.5-2	0.06 mmol/kg/hr
1-1.5	0.09 mmol/kg/hr
<1	0.12 mmol/kg/hr

CHEAT WAY to supplement KCL and KPhos:

Determine the mEq amount of K needed.

(If potassium and phos are relatively normal or low normal prior to adding insulin, then use the higher end of K supplementation.)

By mEq add 1/2 Kcl and 1/2 KPhos

Cheat way example: If K is 3.2, need to add 30mEq per L

Give 1/2 mEq from KCl and 1/2 mEq from KPhos

15 mEq K x ml/2mEq KCl = **7.5ml KCl**

15 mEq K x ml/4.4 mEq KPhos = **3.4ml KPhos**

INSULIN CRI

Blood glucose mg/dl mmol/L	Insulin: 0.9% NaCl Mixture rate in ml	other fluid
>250 mg/dl >14 mmol/L	10	0.9% NaCl
200-250 mg/dl 11.1-13.9 mmol/L	7	0.45% NaCl + 2.5% dextrose
150-200 mg/dl 8.3-11.1 mmol/L	5	0.45% NaCl + 2.5% dextrose
100-150 mg/dl 5.5-8.3 mmol/L	5	0.45% NaCl + 2.5% dextrose
<100 mg/dl < 5.5 mmol/L	0	0.45% NaCl + 5% dextrose

2.5% solution of dextrose: add 50ml of 50% dextrose to a 1L bag

5% solution of dextrose: add 100 ml of 50% dextrose to a 1L bag

Add 2.2 U/kg of humulin R to a 250 ml bag for dog or cat (some prefer to add 1.1 U/kg for cats)

Run 50ml of fluid through the line as insulin binds to plastic. Use a new line every 24 hours

NEW study for use of glargin IM and SQ glargin to manage DKA:

2 u gargin/cat SQ on initiation of fluid and electrolyte replacement

1 u/cat IM 1-2 hr later, repeated q 4 hours if BG if > 250mg/dl (14mmol/L). Provide IV dextrose if BG <250mg/dl (14 mmol/L)

SAMPLE CALCULATION

How much Kphos and KCl (in ml) to add to a 100 ml burette?
BW= 10 kg Phos = 1.6 mg/dl K = 3.4 mg/dl Fluid rate: 60ml/hr

Phos supplementation need: 0.06mmol/kg/hr
K supplementation need: 0.2mEq/kg/hr

Phosphorous:

0.06 mmol/kg/hr x 10kg = 0.6 mmol/hr

0.6mmol/hr x hr/60ml x 100ml/burette = 1 mmol/100ml burette

1mmol/burette x ml/3mmol= **0.3ml/burette from KPhos**

0.3ml kphos/100ml burette x 4.4 mEq/K/ml = 1.3 mEq K/burette

Potassium:

0.2mEq/kg/hr x 10kg = 2mEq/hr

2mEq/hr x hr/60ml x 100ml/burette = 3.3 mEq/100ml burette

3.3mEq/burette- 1.3 mEq K from Kphos= 2 mEq K from KCl

2 mEq K in burette x ml/2 mEq/L K = **1 ml from KCl**



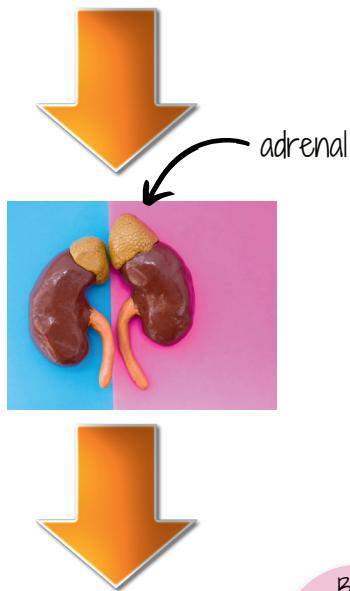
Canine Addison's

Etiology

Addison's (Canine hypoadrenocorticism) is typically the result of immune-destruction of the adrenal gland layers that produce mineralocorticoids (aldosterone) and glucocorticoids (cortisol).

Stim any dog with a baseline cortisol < 2ug/dl (55 mmol/L)

ACTH STIM



Cortisol
< 2 ug/dl or 55 mmol/L
is consistent
with Addison's

- These two layers are in the cortex (outer layers) and are called:
 - zona fasciculata: cortisol
 - Zona glomerulosa: aldosterone
- The syndrome occurs once 80 to 90% of the adrenal cortex tissue is destroyed
- The adrenals are two tiny organs that sit right above the kidneys.

Baseline cortisol screening :
Cortisol < or = to 1 ug/dl (28 nmol/L)
is highly sensitive and specific
for Addison's disease

Clinical Signs

Addison's disease has been coined as the "great pretender" because it can mimick a variety of common diseases in the dog and can be challenging to diagnose.

Clinical signs are often waxing/waning and most dogs have chronic vague symptoms and it's not until there is an acute illness develops that it is diagnosed.

Most common reported **symptoms** are:

- Poor appetite (88-95%)
- Lethargy/depression (85-95%)
- vomiting/regurgitation (68-75%)
- Weakness (51-75%)
- Weightloss (40-50%)
- Diarrhea (35%)
- PU/PD (17-25%)
- Collapse (10%)

Quick Round-up

Canine Hypoadrenocorticism

- Aldosterone:** mineralocorticoid which is responsible for excreting K (potassium) from the kidneys and reabsorbing Na (sodium) and Cl (chloride). 90% of Addison's patients have abnormal electrolytes at initial diagnosis (high K, low Na)
- Cortisol:** is a glucocorticoid which affects almost every tissue in the body. It is vital to maintain most biological functions.
- 10% of Addisonian patients (atypical addisonian) only have a glucocorticoid deficiency, and will have normal electrolytes at diagnosis.
- The **classic electrolyte changes** seen in Typical Addisons' disease are:
 - elevated **K** (up to 95%)
 - Low **Na** (up to 86%)
 - Low **Cl** (about 40%)
- Na:K ratio** normally between 27:1 and 40:1
95% of addison's patients have ratios <27:1, sometimes <20:1 and ratios <15:1 are strongly supportive

Common Breeds:

great danes, all poodles, westies, Portuguese water dogs, bearded collies, Nova Scotia duck tolling retrievers, soft-coated wheaten terriers

Canine Addison's

Addisonian Crisis

- Addisonian crisis is due to severe hypovolemia, dehydration, hypotension, and electrolyte derangements.
- The goal is to correct the hypovolemia, hypotension and correct elevated potassium and low blood sugar, and provide corticosteroid supplementation.

Diagnosis

- Gold standard diagnosis is with an ACTH stim test. This test uses adrenocorticotropic hormone (ACTH) to assess the ability of the adrenal glands to produce the hormone cortisol.
- The classic ACTH stim protocol uses synthetic ACTH : Cortrosyn (cosyntropin): Take a blood sample, administer 250ug (1 vial) IV and then collect a 1 hour post sample
- A lower dosage can be used: 5 ug/kg IV to conserve Cortrosyn.
- A reconstituted vial can last up to 21 days in the fridge. Cosyntropin can also be stored in aliquots and frozen (-20 celcius) in plastic syringes for up to 6 months.

Summary/Key Pts

Addison's disease can present with a variety of vague clinical symptoms.

Most Addison's patients have classic electrolyte derangements of high serum potassium and low sodium/chloride.

Atypical Addison's disease has normal electrolytes and is therefore more difficult to diagnose.

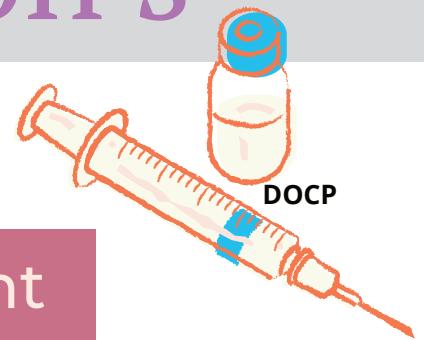
A good screening tool for any dog with vague symptoms is to take a baseline cortisol. Because cortisol is a stress hormone, it is expected to rise in pets who are not well. However, in an Addisonian patient, they have too little cortisol. **A baseline cortisol over 2 ug/dl rules on Addisons disease. A baseline cortisol of under 1ug/dl is highly sensitive and specific for Addison's disease.** However, this should always be confirmed with a full ACTH stim test.

You can go ahead and treat a suspected Addisonian patient with 0.2 mg/kg Dexamethasone IV. This does not affect the ACTH stim results!

In the hospital dexamethasone is often started at 0.2mg/kg bid for the first day, then 0.2mg/kg sid the next few days until the pet is eating.

Prednisolone and Hydrocortisone WILL alter the ACTH stim results.

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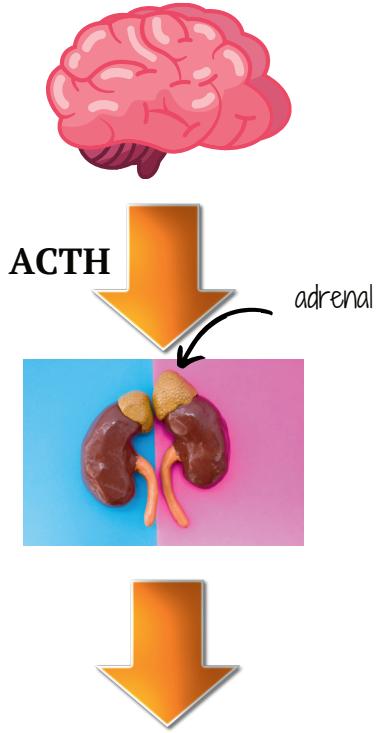
Treatment

- Most addisonian patients will require mineralocorticoid replacement. (atypical addisonian's will not need DOCP)
- **DOCP** (dosocycorticosterone pivalate) , also called Percorten-V is given IM or SC.
- There is debate about whether to start DOCP during the addisonian crisis. Some rely just on intensive fluid therapy and glucocorticoid therapy and then start DOCP once the patient is stabilized.
- **TO START**
Initial dosage of DOCP is 2.2 mg/kg every 25 days.
- Because DOCP is a pure mineraolocorticoid, glucocorticoid supplementation is also necessary.
- **Prednisone is often given at physiologic doses: 0.22mg/kg BID.** This can be tapered later to reduce PU/PD. The pred dose should be increased 2-10 times during times of stress (ie boarding, vet visits, illness etc).
- **ADJUSTMENTS**
 - Following treatment for an addisonian crisis, dogs receiving DOCP should have a recheck history, exam, body weight and chem panel **12 days and 25 days** after initial injection.
 - Minimum database should include: **Na, K, BUN/Creat ,CBC or at least PCV/TS.**
 - **12 day** recheck is to assess the **Peak action of DOCP**. If **hypokalemia** or **Hypernatremia** occur, the next **DOCP** dose should be **decreased by 5 to 10%**.
 - The **25 day check** evaluates **DOCP frequency**. If **hyperkalemia** and /or **hyponatremia** exist, the dosing frequency should be **decreased by 1 day**. Repeat these checks as needed until a good dose/frequency is established.
 - **Abnormal electrolytes = adjust DOCP**
 - **Vomiting, diarrhea, inappetance/lethargy means pred dose should be adjusted higher.**

Canine Cushing's

Etiology

Cushing's is the most common endocrine disease of dogs. Its medical term is: Hyperadrenocorticism



- Cushing's is caused by over production of the steroid hormone, cortisol, from the adrenal glands.
- This is most often triggered by a tumor of the pituitary gland in the brain which over-secretes the hormone ACTH.
- ACTH: Adrenocorticotrophic stimulating Hormone

Pathophys

Pituitary Dependant Hyperadrenocorticism (PDH):

- 80-85% are due to a benign pituitary adenoma:
- Over-secretion of ACTH (adrenocorticotrophic hormone) by a benign, small tumor of the Pituitary gland in the brain. This is a microadenoma.
- PDH results in bilateral adrenal enlargement which can be measured on an abdominal ultrasound exam.
- 20% of PDH results in a progressively enlarging (macroadenoma) of the pituitary which can lead to neurologic decline (head pressing, circling, seizures etc.)

Adrenal Dependant Hyperadrenocorticism (ADH)

- The other 15-20% of cases are caused by an adrenal gland tumor (ADH).

Clinical Signs

Common symptoms: PU/PD/PP/Panting, abdomen distension, alopecia, hepatomegaly, muscle weakness, systemic hypertension

- 30% of dogs show only one symptom: PU/PD, 10% show only hair coat abnormalities
- Less common symptoms: lethargy, hyperpigmentation and comedones, thin skin, calcinosis cutis, poor hair regrowth, insulin resistant diabetes
- Rare symptoms: thromboembolism, facial nerve palsy, ligament rupture

Common breeds:

miniature poodle, dachshund, terrier breeds, boxer, beagle.

Canine Cushing's

<https://www.etsy.com/shop/VetHelpfulNotes>

Diagnostics

Gold standard test to diagnose Cushing's is the **Low Dose Dexamethasone**

Suppression Test (LDDS). It has a much higher sensitivity for detecting canine Cushing's than an ACTH stim test.

LDDS is the initial screening test of choice to determine if a dog has Cushing's.

The **ACTH stim test** is the test of choice to diagnose iatrogenic Cushing's from steroid use.

LDDS: Dexamethasone: 0.015 mg/kg given IV and tests taken at time zero, 4 and 8 hours.

Suppression to less than 50% of baseline confirms PDH

25% of dogs with PDH won't suppress at 4 or 8 hours, so an ACTH concentration is recommended.

Normal to elevated cACTH is consistent with PDH. A low cACTH is consistent with an adrenal tumor.

Summary/Key Pts

The most common symptom of Cushing's is PU/PD. The characteristic labwork changes are a **stress leukogram** (neutrophilia, decrease eosinophils) and mild elevations in **platelet** numbers and **HCT**. A biochemical profile typically shows an elevated **ALP**, elevated **cholesterol** and **triglycerides**.

A diagnosis of Cushing's often takes multiple tests and these typically include baseline biochemistry profile, CBC and a low dose dexamethasone suppression test. **Abdominal imaging** with an ultrasound can be useful to screen for enlargement of one or both adrenal glands.

Most Cushing's disease is caused by a **microadenoma** of the pituitary gland. It is more rare to develop a **macroadenoma** (an enlarging mass in the brain). These patients are best treated with **radiation**. Adrenal gland tumors are more rare but are best treated with surgery.

Treatment of Cushing's is recommended for those patients with **intolerable clinical signs**.

Treatment



Adrenal-directed therapies: mitotane (lysodren) and trilostane (Vetoryl) are the most commonly used agents for Canine Cushing's treatment.

Mitotane: causes necrosis of the layer of the adrenal gland that produces cortisol.

Initial therapy: 50mg/kg divided q 12 hr for 5-10 days until water consumption decreases to less than 100ml/kg/day. Then the dog should be re-evaluated and an ACTH stim performed.

Dogs without PD or dogs whose water cannot be monitored should be administered mitotane for a maximum of 5-7 days prior to the ACTH stim.

In dogs with significant non-adrenal illness, the dose may be reduced by 50% and adjusted based on clinical and hormonal response.

Maintenance 50mg/kg q 7 to 10 days or 25mg/kg q 3 days. Monitor: ACTH stim 1 month after initiation of maintenance therapy and then q 3-4 months thereafter. Goal: pre and post cortisol 2-6 mcg/dl.

Trilostane: blocks adrenal steroid synthesis. Well tolerated and adverse effects mild/self limiting (diarrhea, vom, lethargy).

Initial therapy: 1-2mg/kg q 12 hr. Administer with food. Best to use brand name Vetoryl.

ACTH stim on days 10, 30 and 90 after initiation of treatment then 30 days after each dose adjustment. Samples for testing should be between 2-5 hours post trilostane dosing at usual hour and be maintained for each subsequent test.

Goal: post cortisol less than 6-9 mcg/dl and remission of clinical signs. New Monitoring: pre-pill cortisol just prior to when the morning pill is given.

New Monitoring Protocol:

Well controlled clinically, pre-pill cortisol > 1.4-2 ug/dl can continue dose.

If cortisol is < 1.4-2 ug/dl, or pet is ill: ACTH needed

If pet is clinical: pu/pd/pp and pre-pill cortisol is >3.0 ug/dl, can increase dose by 10-20% and recheck in 2-4 weeks.

If clinical but pre-pill cortisol is <3.0 ug/dl: ACTH stim

Canine Hypothyroidism

PATHOPHYS

Decreased thyroidal production of thyroid hormones T4 and T3. T4= protein bound Thyroid Hormone - not biologically active ft4= free T4 (unbound Thyroid Hormone)- able to penetrate cells and bind receptors- the active hormone!

T3= secretory protein bound product of thyroid gland but not useful to evaluate thyroid function!

Over 90% of hypothyroidism is due to primary immune destruction of the thyroid gland.

When T4 decreases from thyroid disease, then Thyroid Stimulating hormone increases (cTSH) to try to activate the thyroid gland to produce more thyroid hormone.

CLINICAL SIGNS

Lethargy, mental dullness, weight gain, exercise intolerance, alopecia, obesity.

TREATMENT

Initial dose: 0.02mg/kg bid

Maximum of 0.8 mg q 12 hours

Measure peak post pill T4 (4-8hrs) post-pill measured 2-4 weeks after starting therapy.

Once BID dosing has corrected clinical signs and post- pill T4 in acceptable range, might be able to change to once daily dosing. If clinical signs return, then BID dosing must be started again.

OF NOTE

Sight hounds (Greyhounds, Whippets, Salukis etc) and Basenjis have T4 and ft4 ed below normal reference ranges. High TSH is a better indicator of hypothyroidism in these breeds.

T4 alone has no place as a screening tool for hypothyroidism.

Only valuable to run a T4 if there is clinical and biochemical evidence of hypothyroidism!

Incidentally found low T4 without clinical and biochemical signs of hypothyroidism is not treated. Instead: 1) Counsel owners about clinical symptoms to watch for 2) Consider screening for non-thyroidal illness.

Don't test for hypothyroidism if a dog is on these medications: Test only after they have been off for 6-8 weeks.

Most common drugs that lower T4:

Phenobarbital	Zonisamide
TMS	Clomipramine
Aspirin	Glucocorticoids

RISK FACTORS

Young to middle- aged dogs , average age of 7 years. Hypothyroidism is uncommon to develop in senior dogs.

Senior dogs with decreased T4 most likely have euthyroid sick syndrome. (Falsely low T4 due to non-thyroidal illness)

Breed predispositions:

golden retrievers, Doberman pinschers. Heritable thyroiditis: beagle, Borzoi, golden retriever, Great Dane, Irish setter, Doberman pinscher, old English sheepdogs.

DIAGNOSTICS

Low serum T4 is sensitive (picks up 90-95% of cases), but it's not specific (only 70-75% are truly hypothyroid).

Rule out hypothyroidism if T4 is in the upper 50% of the reference range. Typically, if T4 is normal, the dog is not hypothyroid. The exception is that 15% of dogs develop auto-antibodies to the thyroid gland and this can falsely increase serum TT4.

Free T4 (ft4) is unbound T4. measurement is more accurate than total T4 (TT4) measurement because ft4 is not affected by non-thyroidal illness. Free T4 is best measured by Equilibrium Dialysis (ED) because it is not altered by auto-antibodies.

Thyroid Stimulating Hormone (cTSH) is only elevated in 65-75% of cases- so it's not sensitive as a screening test. But, an elevated TSH with a low T4 or ft4 = diagnosis.

TGAA: Thyroglobulin Auto Antibody. 10 to 15% of hypothyroid dogs have elevated TGAA due to autoimmune destruction of the thyroid gland. It's presence does not mean that the pet is hypothyroid, just that autoimmune disease is present.

TGAA can cause false increases in T4 and T3. In this case, rely on ft4 (ED) for determination of hypothyroidism. Most dogs with increased TGAA will eventually develop hypothyroidism.

Ideal diagnostic panel includes: T4, ft4(ED) and TSH. If results are inconclusive (ie TSH is normal, but T4, ft4 are low), then, if pet appears hypothyroid, consider 2-3 month therapeutic trial to see if there is a marked clinical response. The jury is still out whether or not you need to taper off thyroid meds after a trial is deemed ineffective. Likely it is of no benefit.

Also common in hypothyroid dogs:
Low-ish HCT and high-ish cholesterol

Feline Hyperthyroidism

Pathophys

The thyroid gland located on the ventral neck, produces thyroxine (T4) as its main secretory product. Only a fraction of thyroid hormone is not protein bound: free T4 and this is metabolically active. Thyroid hormone helps with heat production, metabolism of food and increasing sympathetic nervous system tone. Unlike dogs, 96-98% of feline cases are due to benign thyroid adenomas. Thyroid carcinoma accounts for only 1-2% of all cases of feline hyperthyroidism.

Clinical signs

Clinical history: weight loss despite increased hunger (polyphagia). In addition, cats may have a history of diarrhea, increased vomiting, PU/PD and hyperactivity. Affected cats often have decreased BCS and decreased muscle condition and a poor haircoat. 80% of cats have a palpable thyroid mass (goiter). Other PE findings may include tachycardia, heart murmur, gallop or other arrhythmia, and dehydration.

Diagnosis

Ideal database: Total T4 (TT4), CBC, Biochemical profile, urinalysis with culture, chest x-rays and blood pressure. Findings: diagnosis is confirmed by measurement of a single increased TT4. Early hyperthyroidism or concurrent non-thyroidal illness may have a high normal or borderline elevated T4 (in the upper half of the reference range). Repeat measurement in 4-8 weeks or after concurrent illness has been treated.

Can submit free T4 (fT4) to investigate borderline elevated T4. Free T4 is less affected by non-thyroidal illness, but it is important to always evaluate with a TT4 because there is an increased risk of a falsely elevated fT4.

Nuclear scintigraphy can also be used to confirm hyperthyroidism in cats with concurrent illness. Scintigraphy is performed by administering a subcutaneous dose of a radionuclide (Technetium) and 20 minutes later using a gamma camera to take an image. Scintigraphy will highlight the over-active thyroid tissue. It is useful to gauge how much over-active thyroid tissue there is to help quantify a dose for I-131 treatment (rather than relying on commonly used "per-patient" dosages). If there is ectopic thyroid tissue (active thyroid glandular tissue in more than one location further down the neck) then scintigraphy can identify this. (Note: surgical thyroidectomy will not be effective if there is extra-nodal thyroid tissue) that cannot be removed. Also, very enlarged thyroid masses that have broken out of the thyroid capsule and a TT4 value 5-10X the normal range is most consistent with thyroid carcinoma.

Possible lab findings: polycythemia due to increased erythropoietin (epo) production, stress leukogram (lymphocytosis, eosinopenia). Mild to moderate increases in ALT, AST and ALP. Azotemia, hyperphosphatemia and hypokalemia.

Etiology

Forty years ago feline hyperthyroidism was virtually non-existent! The marked rise in this endocrine disorder is not truly understood but exposure to environmental factors that disrupt thyroid function has been suggested. Risk for hyperthyroidism increases with age (most cats are over age 7), use of cat litter, eating a diet >50% canned, eating a fish based food are all identified risk factors. Some studies have suggested a 2-3 fold increase in hyperthyroidism in cats on mostly an all-canned food diet. Studies have documented higher serum concentrations of certain compounds (PBDE's and PFA's) in hyperthyroid cats.

Treatment

Medicine: Methimazole - most common antithyroid drug in the U.S. Inhibits synthesis of thyroid hormone. Comes as tablets, but can be compounded into a liquid or a transdermal gel to apply to the inner ear, alternating ears. Must titrate to effect with labwork monitoring 3 weeks after starting and any dose-adjustments. Typically adjust dose about 25% up or down based on results. Once T4 normalized, labwork is monitored q 3-6 months. Dose requirements tend to rise over the years. Most cats require <10mg per day. Adverse reactions occur in up to 20% of cats and tend to happen in the first 3 months. Common side effects: anorexia, vomiting, lethargy. *Transdermal gel has reduced GI side effects.* Rarely: leukopenia, eosinophilia. Rare: bone marrow suppression, icterus, facial pruritus. Typical starting dose: 1.25-2.5mg per cat PO q 12 hr.

Diet: Prescription food made with severely reduced iodine levels to <0.3ppm: Hills Y/D diet. Acceptable protein/phosphorous levels for early CKD. Can be fed to non-hyperthyroid cats as well, but they should be fed a few tablespoons of a non-iodine restricted diet. Not a good choice for outdoor cats or any cat that you can't be sure is not eating treats or other food. Will render the diet ineffective.

Surgery: ablation of the thyroid tissue. Technetium scan recommended prior to make sure no ectopic thyroid tissue. Risk removing parathyroid glands (which are tiny, only a few mm in size) and causing severe hypocalcemia.

Radioactive iodine treatment (I-131): Considered gold standard treatment. It is relatively safe and cures the condition. Radioactive isotope administered as a SQ injection. Requires in-hospital isolation for a few days. First 2 weeks at home: limit close interaction (<1 foot) to 1 hour/day and hold onto scooped waste for 2 weeks for radiation to decay.

Summary/key points

Systemic hypertension has been reported in 12-36% of hyperthyroid cats. Typically the hypertension is not severe and once the patient is treated, the hypertension may resolve.

Hyperthyroidism can mask renal disease by increasing renal blood flow and GFR. However, untreated hyperthyroidism can worsen CKD. It's recommended to normalize T4 even if it unmasks renal disease for optimum patient care. If T4 remains slightly high, there is activation of RAAS which can lead to increased cardiac output, volume overload, glomerular sclerosis and renal hypertension. However, if there is marked azotemia after instituting medical therapy, then definitive therapy with I-131 may not be recommended.

Don't forget that in cats with an elevated proBNP can have transient elevations in this cardiac enzyme due to hyperthyroidism. If you treat hyperthyroidism, their proBNP may normalize as well.

Most radioactive iodine centers use 2-4 mCi per cat. Hypothyroidism is a potential complication of I-131 therapy. Permanent hypothyroidism occurs in less than 5% of treated patients. Scintigraphy prior can help a clinician use a more targeted dose for the patient based.

FeLV in Cats

Outcomes
of FeLV in a
multi-cat
household



Transmission: vertically from infected queens to kittens and horizontally from cats who live together: sharing food bowls, grooming each other or fighting

PCR detects genome-integrated FELV provirus

Elisa snap antigen tests for free p27 antigen in the blood

IFA detects antigen in WBC and platelets

The median survival after diagnosis for FeLV positive cats was 2.4 years
50% die in 2 yrs
80% die in 3 yrs

Transmission takes weeks to months to develop viremia and then propagate to other tissues



Progressive Infection
Persistent viremia



Regressive Infection
Transient viremia; antigen negative but PCR positive (Can only amplify a minute amount of virus)



Abortive Infection
No viremia

FeLV-associated disease

Progressive Infection: Infection not contained. Virus replicates in lymph nodes and bone marrow. Eventually develop FeLV-associated disease

Signs: fever, lymphadenopathy, leukopenia, diarrhea, seizures, ocular diseases
This may take months to years

Managing Positive Healthy Cats

FeLV + cats are 62X more likely to develop lymphoma/leukemia

Examine twice yearly

Monitor for weight loss

Perform a CBC twice a year and chemistry/ua/FF once a year

Spay/neuter, vaccinate

Control endo and ecto parasites

Reactivation possible

Regressive Infection: Cat remains infected, but reverts to an aviremic state. Can take up to 4 months.

- No antigen or culturable virus in circulation
- FeLV proviral DNA may be detectable in blood via PCR (ie negative snap test, +/- positive PCR)
- unlikely to shed virus or develop FeLV-associated disease

No viremia detectable Abortive infection

A **negative test result** is much **more reliable** than a positive result due to the lower prevalence of the disease

Who is tested:
Any new kitten or cat adoption

A cat/kitten with a negative test but possible recent exposure, re-test in 30 days

Recent studies using real-time PCR have shown that 5-10% of cats **negative** on soluble antigen tests were **positive** for FeLV provirus by **PCR** (regressive infection)

Kittens most at risk for contracting FeLV
Adult cat much more resistant to infection

If **SNAP Pos but IFA or PCR neg**, recheck ELISA in 90 days

Antigen NEG
PCR NEG

Antigen POS
PCR NEG

Antigen NEG
PCR POS

Antigen POS
PCR POS



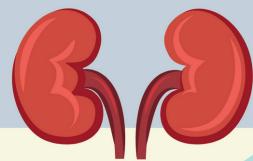
Uninfected

Regressive

Progressive

$\sim 1.0 \times 10^6$ copies/ml

$\sim 1.0 \times 10^6$ copies/ml



CKD Diagnosis and Treatment



Causes of Kidney Injury

- obstruction/stone
- heritable renal disease (polycystic kidney dz, Fanconi syndrome, renal dysplasia, etc.)
- infection (pyelo, lepto, rickettsial dz)
- inflammation/immune mediated
- ischemia
- Toxin (NSAID, lily, EG, etc.)
- vascular injury (hypertension)
- idiopathic/unknown

Distinguishing Acute vs Chronic CKD

Acute

- Clin signs more severe than expected from degree of azotemia
- Leth, vomiting
- Normal or enlarged kidneys
- Normal PCV or regen anemia
- Potassium normal to high

Chronic

- Azotemia more severe than clinical signs
- Decr appetite, PU/PD, weight loss
- small, irregular kidneys or BKLK (big/little kidney)
- NCNC Non regen anemia
- Cats: low potassium
Dogs: mild hyperkalemia

Diagnosis of Stage 1 or Early Stage 2 CKD

One or More of these diagnostic findings

- Creat increasing within reference interval over time and no pre-renal cause evident
- Persistent SDMA >14 ug/dl
- Abnormal kidney imaging
- Persistent renal proteinuria (Dogs > 0.5, Cats >0.4)

Diagnosis of Stage 2-4 CKD

Both of these diagnostic findings

- Increased Creatinine and SDMA
- PLUS
- USG < 1.030 Dogs, < 1.035 Cats

Risk Factors that worsen disease

Proteinuria, Hypertension and Hyperphosphatemia can increase progression of kidney disease if left untreated

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Stage	Dog		Cat		Survival Statistics	MST = Median Survival Time
	CREAT	SDMA	CREAT	SDMA		
1 clinical signs mild or absent	<1.4	<18	<1.6	<18	Cats: were excluded Dogs: MST over 400 days (over 1 year)	
2 clinical signs mild or absent	1.4-2	18-35	1.6-2.8	18-25	Cats: Median 3.1 yrs, up to 8.5 yrs from diagnosis Dogs: 0.5 yrs to 1.1 yrs MST	
3 several extrarenal clinical signs may be present	2.1-5	36-54	2-5	26-38	Cats: Avg 1.8 yrs, some up to 5.7 yrs Dogs: 0.3-0.5 yrs MST	
4 systemic clinical signs and increased risk of uremic crisis	>5	>54	>5	>38	Cats: 1.16 months MST Dogs: 2 weeks to almost 3 months	

Remember:

- 75% of nephrons are lost once Azotemia is detected in labwork
- 66% of nephrons are lost once isosthenuria is present from CKD
- Extra-renal factors
 - Creatinine: decreased with muscle wasting; increased in muscled breeds like Greyhounds
 - BUN: Increased with GI bleeding or high protein diet, decreased with malnutrition, severe protein restriction or liver failure

Pre-Renal Factors

decreased renal perfusion in dehydrated, hypovolemic or hypotensive patients

Post-Renal Factors

Unilateral uterine obstruction

CKD Diagnostics

Serum biochemical profile with electrolytes and acid-base measurement
 Complete blood count with reticulocyte count
 Urinalysis and UPC
 Urine culture and susceptibility
 Urinary tract imaging

Nutrition, Monitoring & Advanced Therapies**Prescription Renal Diets**

- Reduced protein, phosphorus, and sodium
- Increased B vitamins, fiber and omega-3 FA's
- Prescription renal diet reduced risk of uremic crisis by 72% in dogs compared to dogs fed a maintenance diet
- Rec to begin Rx Renal diet when creat is $>/= 2.0$

Monitoring

- Q 4 months Renal values, electrolytes, PCV
- U culture whenever CKD acutely worsens
- Blood pressure measurement q 6 months

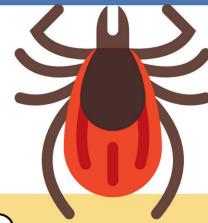
Transplantation/Dialysis

Renal transplantation so far is best developed for cats. The right candidate cat must be in free from other serious illnesses including cancer and heart disease. Fractious cats are not well suited given that medication must be administered regularly and most programs incorporate adoption of the donor cat

Dialysis removes hematogenous wastes and gives the kidneys time to recover from acute kidney injury. It can be used for chronic kidney disease, but is intensive, costly and only effective for a period of time. It requires about 2-3 treatments per week for 5-6 hours to perform on a patient with CKD.

CKD complications	Medications for CKD	Dog	Cat
GI complications (GI/oral ulceration, vomiting, decr appetite)	Omeprazole Maropitant Appetite Stimulant Ondansetron Sucralfate	1mg/kg po bid 2mg/kg po sid Entyce: 3mg/kg po q 24 0.5-1mg/kg po 1-8-12 hr 1/4-1gm po q8hr	1mg/kg po bid 1mg/kg po sid Mirtazapine: 2mg transdermal daily 0.1-1mg/kg po q 12 hr 1/4 gm po q 8hr
Hyperphosphatemia	ALOH	60-90mg/kg/day po divided mixed into food- gel powder	60-90mg/kg/day po divided mixed into food
Anemia	Darbopoeitin Iron dextran	0.45ug/kg SQ q week, till target 10-20mg/kg IM, monthly	1 ug/kg SQ weekly, until target 50mg per cat, monthly
Hypertension	benazepril amlodipine	0.25-1mg/kg po q 12 hr 0.2-0.5mg/kg po q 24 hr	0.25-0.5mg/kg po q 12-24hr 0.625mg-1.25mg per cat q 24hr
Proteinuria	benazepril telmisartan EPA +DHA	0.25-1mg/kg po q 12 hr 1mg/kg po SID 50mg/kg po divided	0.25-0.5mg/kg po q 12-24hr 1mg/kg po q 24hr 50mg/kg po divided

TICK BORNE DZ (TBD)



Predisposed Breeds

Tick borne illness occurs more often in certain breeds, possibly due to a genetic pre-disposition or lifestyle.

Examples:

German Sheperds, Sib husky:	Ehrlichiosis
Goldens and Labs:	Lyme Nephritis
Greyhounds:	Babesia canis
Pit Bulls:	Babesia gibsoni
American Fox Hound:	Babesia & Leishmeniasis

Most Common TBD

After Lyme, Anaplasma and Ehrlichiosis are the next most common infections in the U.S.

Co-infected dogs (more than 1 TBD) are more likely to be ill.

Less common TBD include:

RMSF
Bartonellosis
Babesia
Hemotropic mycoplasmosis
Cytotoxoon felis
Hepatozoonosis

Lyme fast FACTS

Lyme disease is the most common TBD in the U.S. It is a bacterial infection with a bacterium called *Borrelia* transmitted by the bite of a tick.

Although some areas of the U.S. have 70-90% seropositive dogs, overall only 5% of Lyme seropositive dogs develop clinical signs of illness!

Less than 2% of the 5% of Lyme positive dogs develop Lyme nephritis (immune complex glomerulonephritis)

Because Lyme borreliosis travels via tissue migration and generally not in the blood stream, seropositive dogs can still be blood donors.

Cats almost never get ill from Lyme disease

Lymes Low Down

Of the dogs that develop Lyme disease symptoms, the two main diseases they develop are severe joint inflammation and pain (synovitis) and more rarely, severe kidney failure from glomerulonephritis.

Most common Lyme Symptoms

Fever
Anorexia
Painful Joints
Swollen Joints
Intermittent or shifting Leg Lameness
Swollen Lymph nodes
Lethargy

With **Lyme nephritis**, rapid kidney failure occurs due to antigen/antibody complexes deposited into the kidney glomeruli which cause severe inflammation and rapid loss of kidney function. There is an increase incidence in Goldens and Labrador Retrievers. Prognosis is grave as most dogs do not survive.

Ticks that carry Lyme:

Ixodes scapularis and *pacificus* depending on region

Incidence of human TBD is highest in Spring and Summer months when ticks are most active and nymphal ticks are most present.

How long to transmit borrelia: It takes 36-48 hours of tick attachment before Lyme's can be transmitted

Frontline (Fipronil) takes 24 hours (minimum) to kill an attached tick.

Most Isoxazoline class drugs (nexgard, simparica, Bravecto) kill ticks after 4 hours of attachment

Tick products

Products that kill after attachment:

Oral: isoxazolines (Nexgard, Simparica, Bravecto) FAST kill in about 4 hours
Topical: Fipronil - SLOW kill - about 24 hours

Products that prevent tick attachment/repel:

Topical: Permethrin in Advantix, Vectra 3D
Collars: Flumethrin (Seresto collar)
Amitraz (preventic collar now off market)
Oral: none available

For heavily infested areas, it may be a good idea to double up on tick protection by combining a repellent collar and an oral preventative or topspot product.

TICK BORNE DZ (TBD)



Lymes common ?'s

Should every positive dog be treated?

The majority of the panelists of the Consensus Statement on Lyme's disease in dogs do not recommend treatment of non-clinical lyme-positive dogs unless a urine screen is positive for an elevated UPC. Reasoning: 1. potential over-use of antibiotic therapy, 2. borrelia is not reliably cleared from the tissues with treatment anyway and many dogs will be come re-infected at a later point anyway.

The panelists who do treat asymptomatic Lyme positive dogs do so based on the results of a quantitative c6. Based on an elevated c6 test, treatment may be instituted in the hopes that it may lower the risk for antigen-antibody complexes depositing into the kidneys.

How to monitor a lyme positive dog?

Although routine treatment for lyme positive dogs is not universally recommended, a yearly UPC check is recommended.

Should dogs be given the Lyme vaccine?

About half the panelists recommend the Lyme vaccine and the other half prefer focusing on prevention strategies.

Reported efficacy for various Lyme vaccine options have highly variable efficacy ranging from 50-100% infection prevention.

Panelists who recommend the vaccine cite the fact that natural infection does not provide any immunity.

Panelists who do not recommend the vaccine cite the inconsistent efficacy and duration of immunity of the current vaccines.

Finally, some also express caution about sensitizing a dog to developing vaccinal antibody-antigen complexes after lyme vaccination.

Can Lyme disease go away on its own?

It takes a certain amount of borrelia bacteria from a tick bite to enter the host to cause a true infection. Some dogs will test positive from low dose exposure to the organism and will later test negative once their antibodies are no longer present. This may be due to naturally clearing the organism or because the organism "hid" from detection by the immune system.

If the Lyme SNAP is positive, can the dog develop symptoms later? Yes. The antibodies can develop in 3-5 weeks, but symptoms take 2-5 month! So a positive Lyme dog may not develop symptoms for 1-4 more months.

How long does it take to transmit TBD?

Borellia (Lyme) 24-48 hours attachment

Anaplasma phago 6-36 hours

RMSF: 3-6 hours up to 24 hours

Ehrlichiosis: as early as 3-6 hrs up to 24 hours

*** Hepatozoonosis acquired by dogs ingesting the tick! ***

Common owner ?'s

I found a tick on my dog . Did the tick prevention not work?

Most tick preventatives don't prevent attachment, but will kill the tick within a few hours of attachment and hopefully before they can transmit infection.

Because the tick has to bite to be killed, some prefer to use a product with repellent properties (or combining products if visiting more heavily tick infested areas).

If you leave mouth parts behind when removing a tick, can it still transmit disease?

As long as you remove the main body of the tick, they can no longer transmit infection. The borrelia organism lives in the midgut of the tick and will no longer transmit infection if only the head and mouthparts are left behind. Sometimes, leaving the head/mouthparts behind will cause a secondary irritation or swelling.

Other tick infections

Ehrlichia canis and chaffeensis: infect monocytes and lymphocytes

E canis acute disease lasts 3-5 weeks. Symptoms: fever, anorexia, splenomegaly, lymphadenopathy

Most common lab abnormality with e.canis is anemia and thrombocytopenia. The second phase is the asymptomatic carrier phase. With some chronic cases, can have cyclical anemia and thrombocytopenia or pancytopenia. No chronic illness with E. chaff or ewengii.

Ehrlichia ewengii: primarily infects neutrophils but thrombocytopenia is the most consistent laboratory finding, clinical signs typically happen within 7 to 14 days of infection. Acute illness only: fever, reluctance to walk, joint effusion, lethargy, anorexia, central nervous system signs (head tilt, tremors, anisocoria) also can occur. Positive snap test may not occur until 2-3 weeks post infection. So, a negative snap does not rule out the disease when a pet is symptomatic. Re-test in 2-3 weeks.

Note: polyarthritidis from E. ewengii can't be distinguished from Anaplasma as the morulae look the same. Both take 1-2 weeks for symptoms to arise. So, can see morulae BEFORE symptoms develop!

TICK BORNE DZ (TBD)



Tick Pathophys

Rickettsia: Cause endothelial damage to organs, thrombocytopenia, hyperglobulinemia, bone marrow depletion (pancytopenia)

examples of Rickettsia:

Ehrlichia canis

Ehrlichia ewingii

Anaplasma platys

Rickettsia rickettsia (RMSF)

Babesia: cause intra and extravascular hemolysis

Mycoplasma: hemoplasmas are epierythrocytic parasites spread via arthropod vectors (often fleas, but also ticks, lice etc) **M haemofelis** and **M. haemocanis** can cause anemia. Most often causes anemia in splenectomized dogs. In cats can cause potentially fatal acute hemolytic anemia.

Borrelia (Lyme): Can cause arthropathy and in some rare cases, severe kidney damage

Response to doxycycline in acute infection with rickettsia, babesia, mycoplasma and borellia should occur within 24-48 hours!

Hepatozoon: ingested ticks. Sporozoites enter blood and lymphatic system causing organ damage via severe inflammation

Francisciella (Tularemia): infected macrophages invade organs : spleen, liver, lymph nodes and skin to cause microabscesses

Cytozoonosis: protozoal infection of cats. The organism infects macrophages and sporozoites reproduce to form large shizonts in the cat. Shizonts rupture to release piroplasms that infect red blood cells and cause fever, lethargy, leukopenia, thrombocytopenia, hyperbilirubinemia and hyperglobulinemia.

Tick Treatment Recommendations

Lyme Ehrlichia Anaplasma } Doxycycline 5-10mg/kg po bid x 28 days

Babesia Either imidocarb or atovaquone & azithromycin depending on Babesia species infecting

Hepatozoon Ponazuril then Decoquinate long term
Tularemia streptomycin and tetracycline
Cytozoonosis atovaquone and azithromycin

4DX test Results

ASYMPTOMATIC

Lyme positive: consider Quant c6; UA:UPC to screen for proteinuria; patient may develop symptoms in the next 1-4 months.

Anaplasma positive: CBC with blood film to screen for thrombocytopenia, anemia; look for morulae in neutrophils or platelets. Symptoms tend to develop before antibody positive on SNAP tests. Negative SNAP, normal CBC is likely prior exposure.

Ehrlichia positive: CBC with blood film to look for thrombocytopenia, anemia; may also screen for proteinuria, hyperglobulinemia

Types of Ticks

Brown Dog Tick (Rhipicephalus sanguineus)
E. canis, anaplasma platys

Lone Star Tick (Amblyomma americanum)
E. ewingii, Cytozoonosis

Deer Tick -Black Legged (Ixodes scapularis)
Anaplasma phagocytophaga, Borrelia burgdorferi

American Dog Tick (Dermacentor variabilis)
E. canis, RMSF, Tularemia (Francisciella)

Gulf Coast Tick (Amblyomma maculatum)
Hepatozoonosis

American Dog tick



Deer Tick



Brown Dog tick



Lone Star Tick



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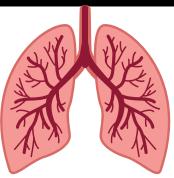


Veterinary Emergency CONDITIONS

Emergency

PULMONARY CONDITIONS

ASPIRATION PNEUMONIA



WHAT IT IS

Inadvertent inhalation of gastric acid, oropharyngeal secretions or food. Typical risk factors are regurgitation after anesthesia, frequent vomiting, esophageal disease, seizures or vomiting.

DIAGNOSIS

Chest x-rays frequently show an alveolar infiltrate in the right middle lung lobe. The right cranial lung lobe is also commonly infiltrated. This is due to how straight the bifurcations of the bronchi are and their susceptibility to passive aspiration.

OF NOTE

Aspiration pneumonia happens more commonly in dogs than in cats



SYMPTOMS

Coughing, nasal discharge, increased respiratory rate/effort. Lethargy, decreased appetite, fever.

TREATMENT

Hospitalization for iv antibiotics, supportive care as needed (oxygen, fluid therapy, nebulization)

Uncomplicated pneumonia example antibiotic option: ampicillin, amoxi/clav or cefazolin

More complicated pneumonia add in enrofloxacin

Right middle lung lobe consolidation from pneumonia



megoesophagus is a risk factor for aspiration pneumonia

PNEUMOTHORAX



WHAT IT IS

Pneumothorax is air in the pleural space which collapses the lungs. The lungs normally inflate due to negative pressure in the chest cavity. Air can collect in the chest cavity from trauma to the chest or damage to the lungs causing air to leak out and into the chest cavity.

DIAGNOSIS

Lateral chest x-ray showing the heart lifted off the chest.

This video demonstrates the loss of the "glide sign"

SYMPTOMS

Tachypnea and dyspnea

OF NOTE

Huskies are at increased risk for spontaneous pneumothorax from pulmonary bullae rupture.

The most common cause of pneumothorax is trauma from blunt force to the chest (HBC, horse kick, etc.)



Heart lifts off the chest

Round Bulla



TREATMENT



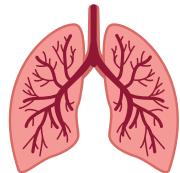
Thoracocentesis. Think of the thorax as a box. Shave a square in the middle of the box. After aseptically preparing the skin, use a 22 gauge 3/4 to 1.5 inch needle and advance with the bevel pointing ventrally. Once there is air that can be evacuated, direct the needle parallel to the chest by pushing the hub of the needle down.

A chest tube may need to be inserted if multiple thoracocentesis are required to keep air from continuing to fill the chest.

Pulmonary bullae may need surgical removal if air continues to leak. If that is not an option sometimes a blood patch pleurodesis can work to create a biologic "bandaid". This is the administration of the patient's own blood into the chest cavity (5-10ml/kg). It works 50% of the time.

Emergency

PULMONARY CONDITIONS



PULMONARY EDEMA

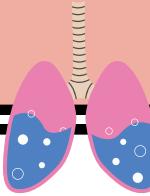
WHAT IT IS

Pulmonary edema is fluid that leaks out of pulmonary vessels into the lungs. The most common cause is due to left sided heart failure. Left heart failure happens when the left heart has reduced diastolic filling which then backs up fluid into the pulmonary vessels.

Non cardiogenic edema is caused by altered pulmonary vessel permeability. This can happen from electric cord injury, choking, seizures or acute respiratory distress syndrome.

DIAGNOSIS

Pulmonary edema is diagnosed primarily by x-rays of the lungs. Edema shows up as interstitial to alveolar infiltrates. Alveolar infiltrates distributed in the dorso-caudal lung fields without cardiomegaly is most consistent with non-cardiogenic edema. Alveolar infiltrates in the hilar area is most consistent with heart failure. Always correlate with clinical signs and history.



SYMPTOMS

Tachypnea and respiratory distress.

B-lines are also called lung rockets. They are like flashlight beams.

OF NOTE

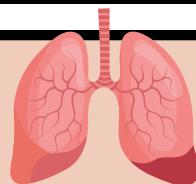
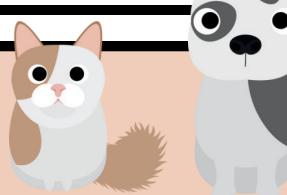
T-Fast can diagnose pulmonary edema with over 3 b-lines per hemithorax quadrant.



TREATMENT

Cardiogenic pulmonary edema is treated with oxygen, rest and diuretics. Vasodilators can be used with caution (as long as patients are not hypotensive). IV fluid therapy is not recommended.

Non cardiogenic pulmonary edema treatment is not as straightforward. Most will rapidly improve with oxygen and supportive care. However, use of diuretics like furosemide remains controversial for non-cardiogenic causes.



PLEURAL EFFUSION

WHAT IT IS

Fluid accumulation in the pleural space. This fluid can be a transudate, modified transudate, or exudate.

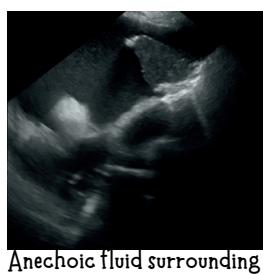
Transudates: clear, low tp $<2.5\text{mg/dl}$ and low cellularity. They are typically from hypoalbuminemia or overhydration.

Modified transudates: tp of 2.5-5 and can be due to heart failure, neoplasia, or inflammatory conditions like pancreatitis.

Exudates: tp >3 and can be from pyothorax, FIP, hemothorax and chylothorax.

DIAGNOSIS

Diagnosis is via imaging with chest x-rays or a chest ultrasound.



Anechoic fluid surrounding retracted lung



Retracted lung lobes surrounded by fluid

SYMPTOMS

Tachypnea and difficulty breathing.

TREATMENT

Pleural effusion is typically drained via thoracocentesis. The underlying cause is treated differently depending on the cause:

FIP: antiviral therapy

Pyothorax: antibiotics, chest tube management in cats

Pyothorax in dogs is a surgical condition treatment

Heart failure: oxygen, diuretics etc.

Chylothorax: thoracic duct ligation, rutin

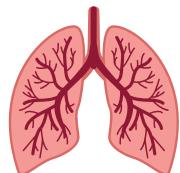
OF NOTE

Pleural effusion is a much more common diagnosis in cats than in dogs.

The top two most common causes of pleural effusion are CHF (41%) followed by neoplasia (26%). The other common causes were: pyothorax, FIP, idiopathic chylothorax, trauma, and non traumatic diaphragmatic hernia.

Emergency

PULMONARY CONDITIONS



ASTHMA



WHAT IT IS

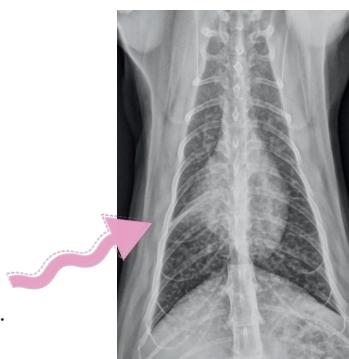
Asthma is seen most commonly in cats. It is an inflammatory condition that affects the lower airways causing constriction of the bronchioles.

DIAGNOSIS

Diagnosis is based on a history of a cough, wheezing and chest x-rays that show excess bronchiolar markings in the lower airways.

OF NOTE

Frequently there is right middle lung lobe consolidation from mucus plugging and collapse.



SYMPTOMS

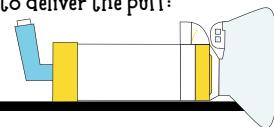
Frequent coughing that is similar to a cat having a hairball without the hairball.

There may be an increased respiratory rate and effort with severe cases.

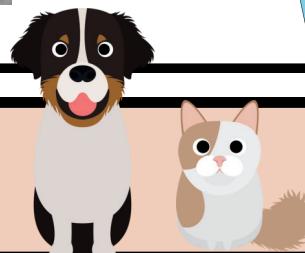
TREATMENT

Steroids are a cornerstone to treat the airway inflammation that triggers asthma. Oxygen and injectable steroids may be needed in emergency cases. In emergencies, injectable bronchodilator therapy with terbutaline 0.01mg/kg or an inhaled puff of albuterol as a second line bronchodilator choice.

Chronic at home therapy: fluticasone inhaler 110mcg/puff bid. Use a mask/chamber system to deliver the puff.



KENNEL COUGH



WHAT IT IS

Also known as infectious tracheobronchitis, is a honking cough caused by a few possible infections- the most common bacterial causes: bordetella bronchiseptica gram negative coccobacilli. Other gram negative bacteria can also cause this infection (e. coli, pseudomonas, klebsiella). Mycoplasma induced kennel cough is unclear. Viral causes include canine adenovirus, influenza, distemper and herpes virus. Together, this is also called canine infectious respiratory disease complex (CIRDC).

DIAGNOSIS

Many times the diagnosis is based on symptoms and history alone. Pneumonia must be diagnosed with imaging (x-rays). A nasopharyngeal swab can be used to isolate infectious causes via respiratory PCR panel: Canine Distemper Virus (CDV), Canine Herpes Virus (CHV), Canine Parainfluenza Virus (CPV), Canine Respiratory Coronavirus (CRoV), Canine Adenovirus-2 (CAv-2), Influenza A, Bordetella bronchiseptica, and Mycoplasma spp. However, routine PCR testing is mostly important at the population level as individual therapy most often will not change.

TREATMENT

Many times it is just supportive care (cough suppressants, quarantine at home). If there is pneumonia present, then antibiotics such as: clavamox, doxycycline or enrofloxacin are often utilized. Doxy and Clavamox are considered the first line treatment choice for CIRDC. Antitussives are not recommended if treating for pneumonia. It's important to cough to help bring up and clear the infected material in the lungs.

SYMPTOMS

Kennel cough produces a classic goose honk tracheal cough followed by a retch (often produces white foamy liquid from the trachea.)

In many cases patients still feel well. Some patients can develop a secondary bronchopneumonia with more severe signs of fever, purulent nasal discharge and lethargy/anorexia.

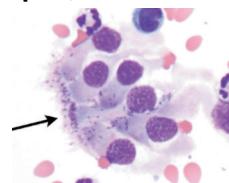
OF NOTE

The disease typically develops 5-10 days after exposure to other susceptible or affected dogs. The cough typically lasts about 5 days, but can linger for up to 3 weeks.

Not all infected dogs will cough, so kennel cough can develop from exposure to a non symptomatic dog. Many dogs can still shed infectious bacteria/viruses for up to 3-4 weeks after infection.

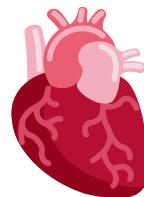
The human bordetella is whooping cough: Bordetella Pertussis

Bordetella are coccobacilli seen on tracheal wash



Emergency CARDIAC CONDITIONS

CONGESTIVE HEART FAILURE (CHF)

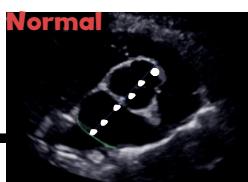


WHAT IT IS

CHF is the inability of the heart to function to maintain sufficient cardiac output to the body. With left sided heart failure there is increased pressure in the left atrium. This is most often from back-flow of blood through a leaky mitral valve. Increased pressure backs up into the pulmonary veins which leaks fluid into the lungs. With right sided heart failure there is increased pressure in the right atrium. This backs up into the vena cava running through the liver. Hydrostatic pressure causes fluid to weep into the abdomen and cause ascites.

DIAGNOSIS

Diagnosis of left heart failure is demonstration of edema in the lungs and an enlarged heart on x-ray. An echocardiogram can also be used to confirm leaky valves, chamber enlargement and poor contractility. On an ER basis, a quick ultrasound exam can check for a dilated left atrium with an La:Ao ratio. La= left atrium. Ao= Aorta. Normal La:Ao is 1.5 or less.



SYMPTOMS

Soft cough, increased respiratory rate and effort, fatigue, audible murmur.

TREATMENT

Acute treatment: furosemide, oxygen, pimobendan
2nd line drugs may include arterial vasodilators and pressors to increase blood pressure (dobutamine, dopamine)
Chronic treatment: furosemide, pimobendan, ace-inhibitor, spironolactone

OF NOTE

Monitor for azotemia and electrolyte changes (hypokalemia for example) on cardiac drugs and adjust or implement supplementation as needed.

Prognosis: Median survival after first CHF episode with DMVD is 11 months.



HYPERTROPHIC CARDIOMYOPATHY (HCM)

WHAT IT IS

Hypertrophic cardiomyopathy is the most common form of heart disease in the cat. It is concentric thickening of the heart wall muscle that leads to decreased chamber size of the left ventricle. The excess blood that cannot fit in the left ventricle backs up into the left atrium. This dilates the left atrium from excess blood received. This excess blood backs up into the pulmonary veins leading to lung congestion. Blood that pools in the enlarged atrium leads to clot formation and these can be released from the heart and block off blood supply to the legs if they get trapped in the distal aorta.

DIAGNOSIS

Pulmonary edema and/or pleural effusion with an enlarged, valentine heart on x-ray. Definitive diagnosis is via echocardiography demonstrating atrial enlargement and poor contractility. Sometimes "smoke" in the atrium from blood pooling in the enlarged chamber. A quick ER test is measuring the La:Ao ratio. (see above) A probnp snap test: Almost all cats with CHF will have a positive SNAP test. So, if it's normal, it rules out CHF

Normal right lateral VHS in a cat should be 6.9 to 8.1. In addition, a short axis wider than 4 vertebral bodies is highly supportive of atrial enlargement.

SYMPTOMS

Tachypnea, dyspnea, hypothermia, occasionally arrhythmia.

TREATMENT

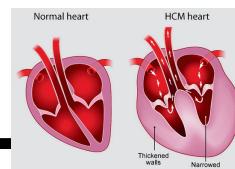
Oxygen, furosemide are the mainstays of hospital therapy. Pimobendan is increasingly being used when there is left ventricular systolic dysfunction, significant pleural effusion, concern for renal insufficiency, or severe refractory pulmonary edema.

At home therapy typically includes an ace inhibitor like benazepril once the cat is eating fine at home.

OF NOTE

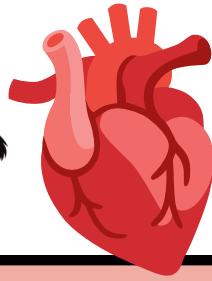
Many cats with heart disease have no audible murmur. And many cats with grade I-II murmurs do not have heart disease! (Rather they have an innocent physiologic flow murmur).

A murmur in a cat is much less predictive of heart disease than in the dog. Most cats with a HCM do not have structural heart disease. About 1/3 of cats with heart disease have no HCM.



Emergency CARDIAC CONDITIONS

PERICARDIAL EFFUSION



WHAT IT IS

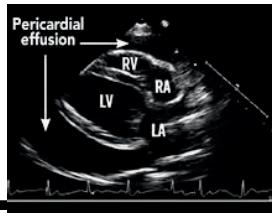
Fluid build up within the pericardial sac. The fluid is most often hemorrhagic. The two most common causes are idiopathic pericarditis or neoplasia. Congestive heart failure and hypoproteinemia can cause pericardial effusion but rarely enough to cause tamponade (compression of the heart by the fluid). Very rarely seen in cats.

DIAGNOSIS

idiopathic pericarditis: med to large breed male dogs (90% are male). goldens <3 years and St. Bernards. non clotting hemorrhagic effusion.
Neoplasia: Primarily older dogs
Rt atrial mass; hemangiosarcoma
Aortic body tumor: chemodectoma
Echocardiogram to identify masses.

OF NOTE

Fluid cytology is of little use.
But blood should be non-clotting. If the blood clots, it may be from a chamber.



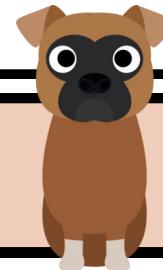
SYMPTOMS

Collapse, weakness, syncope, lethargy. Abdominal distension from ascites, muffled heart sounds. Weak femoral pulses, pale mucous membranes. Tamponade is when the fluid within the heart sac causes enough compression to reduce cardiac output.

TREATMENT

Pericardiocentesis to improve cardiac output immediately. Subtotal pericardectomy is recommended if idiopathic pericarditis is suspected.

[Here is a video on how to perform a pericardiocentesis](#)



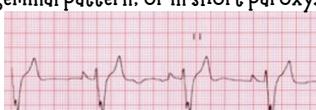
ARRHYTHMOGENIC RT VENTRICULAR CARDIOMYOPATHY

WHAT IT IS

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an inherited myocardial disease seen in dogs, primarily Boxers. The disease is characterized by fatty replacement of the myocardium which causes ventricular arrhythmias, and the potential for syncope or sudden death. In Boxers, ARVC is a familial disease inherited in an autosomal dominant pattern.

DIAGNOSIS

A workup includes a physical examination, minimum database, ECG, echocardiogram, and 24-hr Holter monitor. An abdominal ultrasound may also be recommended in older animals to rule out the abdominal disease that can lead to ventricular arrhythmias (e.g., splenic lesions or adrenal masses). Regular screening in predisposed animals is recommended. Most dogs with ARVC are asymptomatic until they develop syncope or even sudden death. ECG findings: Left bundle branch block morphology VPCs isolated, as couplets, bigeminy pattern, or in short paroxysms or sustained runs of ventricular tachycardia.



Holter monitoring is the current screening method of choice for ARVC, and the results have diagnostic, prognostic, and therapeutic importance. Echocardiography might show RV systolic dysfunction and RV chamber enlargement. Some dogs will also develop LV enlargement and wall thinning.

SYMPTOMS

PE may be normal or dogs may have an arrhythmia with pulse deficits. A left sided heart murmur or signs of CHF (dyspnea, tachypnea, jugular vein distension, crackles)

TREATMENT

There is no absolute number of VPCs that automatically warrant medical therapy. Trigger to begin antiarrhythmic drugs is often dictated by clinician preference. Therapy is generally started in dogs with clinical signs attributed to their arrhythmia, frequent ventricular ectopy on 24-h Holter monitoring, or in those that exhibit malignant arrhythmias with increased complexity (R-on-T phenomenon, couplets, triplets, frequent bigeminy, ventricular tachycardia). Early treatment is often sotalol. Pimobendan and mexiletine may also be used.

A >80% reduction in arrhythmia is needed to definitively suggest treatment efficacy.

OF NOTE

Mean age of diagnosis of five to seven years in Boxer dogs. Syncope or weakness in a middle-aged to older Boxer or English bulldog should prompt a diagnostic workup for ARVC.

Emergency VASCULAR CONDITIONS

FELINE AORTIC THROMBOEMBOLISM



WHAT IT IS

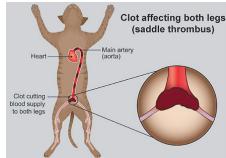
Thrombus formation secondary to blood pooling in an enlarged Left atrium that lodges in a distal blood vessel when it's sent out of the heart. This is most often secondary to hypertrophic cardiomyopathy. Other less likely causes are other heart diseases, infective endocarditis or tumor emboli. A common location for the thrombus to land is at the caudal trifurcation of the aorta (as it bifurcates to both hind limbs). This is called a saddle thrombus.

DIAGNOSIS

Hyperglycemia and azotemia are frequently encountered. Azotemia may be pre-renal from poor perfusion and shock. CK and phosphorous elevations from muscle damage. Hyperkalemia from reperfusion injury. A blood glucose difference of greater than 30 mg/dl between an unaffected limb and affected limb is 100% sensitive & 90% specific for ATE. Cats with an initial body temperature over 98.9°F have a greater than 50% chance of survival to discharge.

OF NOTE

Sadly most cats with ATE have no known prior history of heart disease. There is a 37.5% discharge rate with bilateral pelvic limb paralysis. Euthanasia rates are high. 75% of FATE cases are male cats.



SYMPTOMS

Affected cats that suffer from a saddle thrombus present with acute pain, vocalization and inability to properly move one or both hind limbs. The affected hind limbs are cool to the touch due to decreased or absent circulation. More than 3/4ths of cats present with 2 limbs affected. 20% have only 1 limb affected, and only 1-2% have 3 limbs affected.

40-70% of cats have concurrent CHF and may be dyspneic, tachypneic, have crackles and open mouth breathing. An echo can show "smoke" in the atrium (tiny suspended clots)

TREATMENT

Immediate: provide oxygen and analgesia. Assess for and treat CHF if present (chest x-rays, furosemide). Antithrombotics (low MW heparin) q 6 hr SQ. Clopidogrel: 75mg loading dose, then 1/4th tab q 24 hr. Physiotherapy when more stable. Monitor blood gases closely the first 48-72 hours for hyperkalemia and acidosis secondary to reperfusion injury.

HYPERTENSION



WHAT IT IS

High blood pressure is defined as a blood pressure over 160 mmHg in dogs and cats. Common causes are: Idiopathic, CKD, hyperthyroidism, hyperadrenocorticism, diabetes mellitus, pheochromocytoma and hyperaldosteronism. Concern for untreated hypertension is target organ damage (TOD) to the kidneys, eyes and brain.

DIAGNOSIS

Doppler BP using a cuff 30-40% the circumference of the site (commonly the antebiarm, tail or tarsus). A cuff too large will underestimate the BP and a cuff too small will overestimate BP. Take BP measurements in lateral or ventral recumbency. Use the average of 5-7 consecutive readings. Try to perform in a calm quiet environment.

OF NOTE

The goal of hypertension treatment is to avoid target or end organ damage (TOD). This means damage to the eyes, the heart, kidney and brain. Kidney: proteinuria, azotemia. Brain: seizures, stroke. Eyes: retinal detachment, hemorrhage, retinal vessel tortuosity. Heart: left ventricular hypertrophy, CHF. BP > 180 and no TOD, recheck 2x in 14 days; if still elev recc tx. BP 160-180 recheck 2x in 2 months, if still > 160 recc tx; if < 160 recheck in 3-6 mo.

SYMPTOMS

Sign of TOD: hypertensive retinopathy, hyphema, seizures, altered mentation, and focal neurological deficits, renal abnormalities (eg. proteinuria and azotemia), and cardiovascular abnormalities (eg. LVI, gallop rhythm or other arrhythmia, murmur, and epistaxis). Hypertension may initially be asymptomatic.

TREATMENT

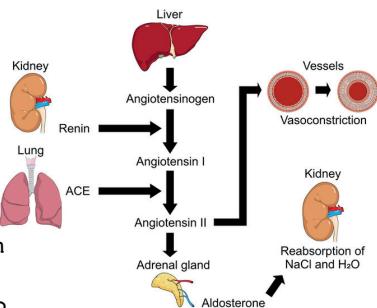
Most common to start RAAS inhibitor: ACE or ARB for dogs;

Goal is minimum of under 160; ideally 140 mmHg

If ineffective by 3-4 weeks, then add Calcium channel blocker.

In cats, it's still recommended to start with CCB.

For hypertensive crisis, can check as soon as 3-5 days and if ineffective, add second agent.



Emergency URINARY CONDITIONS



URINARY OBSTRUCTION



WHAT IT IS

A life threatening urinary blockage that has a high survival rate to discharge if treated. It happens mostly in male, indoor cats. Male cats have a narrow urethra that can get plugged/obstructed by mineral grit, mucus or spasming. Azotemia and hyperkalemia are major changes noted in labwork.

DIAGNOSIS

Palpation of a turgid, painful and enlarged urinary bladder with symptoms of straining to urinate with little to nothing produced.



OF NOTE

Temperature under 96.6 degrees F 98% predictive of a serum potassium $> 8 \text{ meq/L}$ Also HR below 120 is indicative of hyperkalemia. Post obstructive diuresis occurs within 48 hours for 88% of cats w/ UO. Can last up to 3 days after UO treatment. Relapse rate is highest in the first week after medical management. Rate is reported 15-40%.



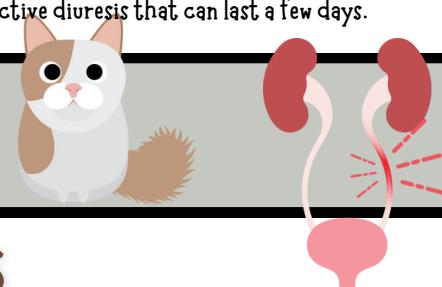
SYMPTOMS

Initial symptoms are straining to urinate with little to nothing produced. It progresses to vomiting, lethargy and abdominal discomfort. Lab work can show azotemia (elevated kidney values) and elevated potassium. If potassium elevation is severe, then it can affect the cardiac pacemaker by reducing its excitability and cause life threatening arrhythmias and bradycardia.



TREATMENT

Sedate and pass a urinary catheter to release the obstruction. Saline is used to flush the obstruction into the bladder. K⁺ values > 7.5 need treatment, values > 9 are life-threatening. Calcium gluconate: protects the heart from arrhythmias caused by elevated potassium. IV dextrose +/- insulin to drive potassium back into cells. Bladder might be drained via cystocentesis to relieve pressure. Sometimes a sacrococcygeal block can help with analgesia and unblocking. After unblocking iv fluid diuresis to address azotemia and post obstructive diuresis that can last a few days.



URETERAL OBSTRUCTION

WHAT IT IS

Ureteral obstruction is a problem encountered primarily in cats. It's a stone formed in the kidney that lodges in the ureter. The primary stone type is calcium oxalate. These stones obstruct the urine outflow from the kidney and as a result cause pressure damage to that kidney.



DIAGNOSIS

Imaging studies with abdominal x-rays can screen for mineral dense stones in the region of the ureter. Abdominal ultrasound can identify a stone in the ureter and hydronephrosis of the ipsilateral kidney (fluid back up building up in the center of the kidney).

OF NOTE

Most cats with ureteral obstruction develop azotemia because they already had mild chronic kidney disease. (It takes 75% nephron loss to cause azotemia to develop).

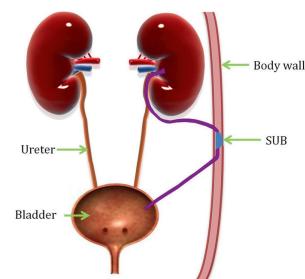
Ureteroliths don't dissolve in cats because they are composed of calcium oxalate. Further more, a stone in the ureter is not bathed in enough urine to allow for a dissolution diet to be effective.

SYMPTOMS

Ureteroliths typically are diagnosed when the cat is acutely symptomatic for kidney failure (vomiting, lethargy, decreased appetite, PU/PD, acute abdominal pain etc.) or incidentally on examination by palpating an enlarged, possibly painful kidney on one side and a small, irregular kidney on the other side.

TREATMENT

Unfortunately, medical management is only 10% effective (it relies on iv fluid diuresis and medications like mannitol to increase the blood volume and push the stone down the ureter). A ureteral bypass system called a SUB (subcutaneous ureteral bypass system) can be placed surgically to salvage the affected kidney. This creates a "fake" ureter to re-establish urine flow.



Emergency URINARY CONDITIONS

FELINE INTERSTITIAL CYSTITIS

WHAT IT IS

Feline Interstitial Cystitis (FIC) is a syndrome of sterile bladder inflammation without an identifiable cause (no infection, no stone, no anatomic defect etc). It's used interchangeably with the term idiopathic cystitis and is one of many causes of FLUTD (feline lower urinary tract disease).

DIAGNOSIS

FIC is the most frequent cause of FLUTD (feline lower urinary tract disease) and accounts for about 45-65% of cases of FLUTD. FIC can be non-obstructive or obstructive, but 80-90% are non-obstructive thankfully. It is a diagnosis of exclusion.

OF NOTE

Two to 15% are frequently recurring or persistent cases. Male cats are most at risk for developing FIC and some studies show that overweight cats and cats between 2-7 years old are most at risk. Cats with FIC are more easily alarmed/nervous and live indoors. FIC cats frequently eat dry food and don't drink as much water.



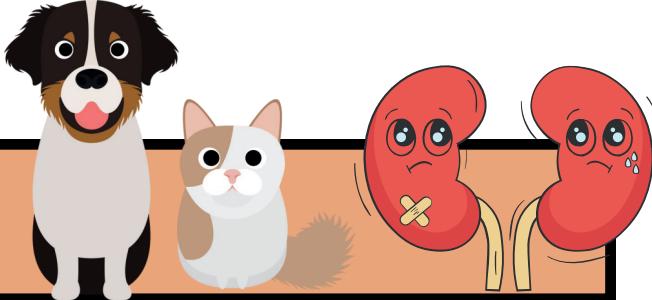
SYMPTOMS

FIC is similar to a condition seen in women. Neurogenic input to the bladder (stress, etc.) can trigger bladder wall inflammation which then leads to symptoms of straining to urinate, bloody urine, and small frequent urinations.

TREATMENT



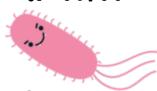
FIC is typically self-limiting and an episode can from a few days to a couple of weeks. There is no specific treatment for FIC but practitioners usually treat for pain with analgesics like gabapentin, administer SC fluids to improve hydration and switch to an all wet food diet. They recommend reducing stressors in the home by increasing the number of litter boxes, providing stress reducing supplements like zylkene and pheromones like Feliway.



PYELONEPHRITIS

WHAT IT IS

Pyelonephritis is a kidney infection that frequently causes azotemia. It is typically bilateral and due to an ascending infection from the bladder.



DIAGNOSIS

Systemic signs of lethargy, fever in addition to labwork that supports infection (leukocytosis, positive urine culture, and supportive ultrasound findings). A urine culture may be negative with lower burden infections. A culture directly from urine in the center of the kidney would be the most diagnostic, but this is an invasive procedure and is rarely performed. Instead reliance on urine culture and imaging studies of the kidneys that show renal pelvis dilation are typically utilized.

OF NOTE

Pyelonephritis is more common in cats than in dogs and e.coli is the most common pathogen found.



SYMPTOMS

Pyelonephritis can cause kidney failure and flank pain with PU/PD.

TREATMENT



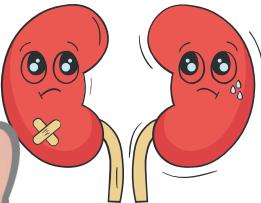
A broad spectrum antibiotic used to recommend 4-6 weeks of treatment, but newer ISCAID recommendations suggest 10-14 days may be sufficient. Preferably select an antibiotic which can properly penetrate the kidney such as a fluoroquinolone or a third generation cephalosporin antibiotic. Urine should be cultured before administration of antibiotics, 5-7 days into treatment and again 3-5 days after treatment has ended. Then monthly for 3 months is recommended.

Febrile, anorexic or azotemic animals should be hospitalized for iv fluid therapy and intravenous antibiotics. A goal is to diurese for 48 to 72 hours and reduce azotemia by 50% or more.



Emergency URINARY CONDITIONS

LEPTOSPIROSIS



WHAT IT IS

Infection with a spirochete bacteria shed in the urine of affected wildlife, most notably rodents like the common rat but also squirrels, fox, deer, opossums and raccoons. Leptospirosis causes acute liver and kidney damage. Zoonotic (can be transferred to people!)

DIAGNOSIS

CLINICAL CRITERIA. Onset of nonspecific fever, lethargy, polyuria, polydipsia, anorexia, or some combination of these signs

AND 2 OR MORE OF THE FOLLOWING:

BLOOD:  Neuts, low platelets, azotemia,  liver enzymes, PLI or CK
URINE: glucose in urine w/o elev BG, active urine sediment
CHEST X-RAYS: infiltrates
AUS: pancreatitis, perirenal fluid or hyperechoic cortices
ECG: arrhythmia

4 fold rise in titer 2 weeks after acute titer submission or positive PCR from urine and blood

*the SNAP Lepto test can be positive if a dog has received the Lepto vaccine within the past 12 weeks *



SYMPTOMS

Illness symptoms (fever, lethargy, pu/pd, inapp) +/- gi signs (pancreatitis, enteritis) pulmonary signs (pulmonary hemorrhage), ocular signs (uveitis, conjunctivitis), oliguria/anuria, icterus, hemorrhage (coagulopathy)



TREATMENT

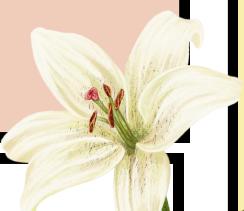
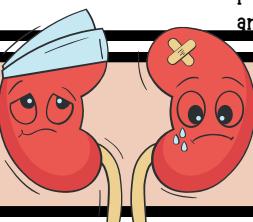
Initial treatment with IV penicillin derivative followed by oral doxycycline to rid the carrier stage of kidney infection. Considered infectious to humans and animals until at least 48 hours of antibiotic therapy. Treat AKI with appropriate supportive care. Monitor urine output, BP, pain control and for respiratory symptoms.

OF NOTE

Cats can get infected but generally don't get sick! Must wear personal protective equipment when working with a sick patient while they are still infectious (first 48 hours into antibiotic tx)



LILY TOXICITY



WHAT IT IS

Acute kidney injury occurs in cats secondary to ingestion of any part of the plant (stem, leaves, petal, pollen or water from the vase). The exact dose or toxic element is not known.

DIAGNOSIS

Even if no kidney injury is documented, if there is suspicion of having ingested any part of a lily, the cat should be treated. BUN, creat, phos, K rise 24-72 hours after ingestion. Glucose and casts can be seen in the urine within 18 hours.

OF NOTE

Easter Lily, Tiger Lily, Japanese Showy Lily, Day Lily are toxic



Cala Lily, and Peace Lilies are NOT toxic



SYMPTOMS

Salivation, vomiting, inappetance, depression leading to polyuric then anuric renal failure.

TREATMENT

Most successful outcomes start treatment within 6 hours of exposure or at least before anuric renal failure develops. Once anuric renal failure has developed, then the best outcome is achieved with dialysis.

If a cat is brought in soon after ingestion - decontamination (emesis) followed by administration of activated charcoal with sorbitol, iv fluid therapy for at least 48 hours to maintain urine flow and prevent tubular obstruction with sloughing epithelial cells. Causes renal tubular necrosis but the basement membrane of the tubule remains intact and the renal tubular epithelium can regenerate with prompt treatment. Baseline, 24, 48 and 72 hour renal values recommended.

Current fluid therapy recommendations for acute kidney injury suggest that following appropriate rehydration, that maintenance rate of fluid is sufficient. Higher rates of fluid therapy may lead to kidney edema and tubular swelling, potentially worsening an already injured kidney.

Emergency URINARY CONDITIONS

TCC

WHAT IT IS

Transitional cell carcinoma is most common bladder tumor in dogs and is very aggressive.

There is a 21 fold increased risk of TCC in Scottish Terriers and 3-5 fold increased risk in Eskimo dogs, Shetland Sheepdogs, West Highland White Terriers, Keeshonds, Samoyeds, and Beagles

DIAGNOSIS

Imaging with an ultrasound can identify a mass in the bladder. Urinalysis from traumatic catheterization to characterize the abnormal cells. Fine needle aspiration or cystocentesis is generally not recommended for fear of seeding cancer through the needle path. Cystoscopy and biopsy of the mass.

BRAF is a urine test that can identify an important gene mutation found in the cancerous cells



SYMPTOMS

Hematuria (blood in the urine), stranguria (slow painful urination), dysuria (difficult, painful urination), pollakiuria (frequent small urinations).

TREATMENT

Treatment is rarely curative, so most treatments are aimed at palliation of symptoms and improving quality of life. NSAIDs can help to alleviate symptoms and dogs on NSAIDs live longer than dogs that are not treated with NSAIDs. The addition of chemotherapy with NSAIDs can extend the disease free interval to 1 year or 6 months if there is urethral or prostate involvement.

OF NOTE

The mass, in dogs, typically develops in the trigone region (neck) of the bladder where the ureters attach. This location is often not resectable with surgery. Many dogs have concurrent urethral involvement and 20% of dogs have metastasis at the time of diagnosis.

BLADDER STONES

WHAT IT IS

Bladder stones occur in both dogs and cats. There are a variety of stone types but the two most common are struvite and calcium oxalate. Struvite stones can be dissolved with a dissolution diet, but calcium oxalate stones cannot.

DIAGNOSIS

Bladder stones are diagnosed with imaging (x-rays or ultrasound) but the stone type cannot be identified without removing the stone and submitting it for stone analysis.



OF NOTE

Female dogs are 12-50X more likely to have struvite uroliths vs male dogs who are 3 x more likely to have Calcium oxalate uroliths. Feline uroliths are approximately 50/50 of each stone type.

Most canine struvite uroliths are caused by urinary tract infections. However, struvite uroliths are sterile in approximately 95% of feline cases!



SYMPTOMS

Bladder stones may not cause any symptoms or can cause intermittent symptoms of hematuria, stranguria and pollakiuria.

TREATMENT

Struvite stones should be medically dissolved (in dogs this means a dissolution diet paired with the appropriate antibiotic for at least 1 month after dissolution of the stones). In cats, a dissolution diet alone is typically effective. As long as cats continue to create struvite crystals, a stone diet is recommended. However, cats over age 10 rarely form stones.

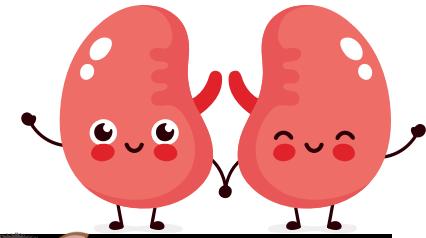
Most stones will dissolve in under 3 weeks on the appropriate dissolution diet. Calcium oxalate stones must be removed surgically as dissolution is ineffective.

Adding moisture to the diet is a cornerstone of urolith prevention. A urinary diet can help prevent sterile struvite stone formation.

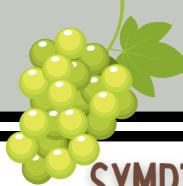
To minimize Calcium oxalate urolith recurrence: decrease urine concentration, avoid urine acidification, avoid diets w/ excess protein.



Emergency URINARY CONDITIONS



GRAPE TOXICITY



WHAT IT IS

Some dogs (but not all dogs) can develop acute renal failure after ingesting grapes or raisins. Oliguric (decreased urine production) or anuric (no urine production) renal failure can occur within 24-48 hours of ingestion.

DIAGNOSIS

History of exposure and renal failure diagnosed on labwork evaluation.

OF NOTE

Serum creatinine tends to rise early and disproportionately to the serum urea nitrogen levels! Once anuria develops, many dogs are euthanized.

The lowest reported amount to cause AKI have been 19.6 g/kg for grapes and 2.8 g/kg for raisins. Four to five grapes can induce AKI in a small dog (<8.5 kg).

Cats do not appear to develop renal failure from grape / raisin ingestion.

The average grape weighs 5 grams



SYMPTOMS

Vomiting and diarrhea can occur within 6-12 hours of ingestion. Other symptoms include anorexia, dehydration, polydipsia, abdominal pain and trembling.

TREATMENT

Aggressive and early decontamination with emesis induction followed by activated charcoal administration.

Following decontamination, IV fluid diuresis for at least 48 hours to help prevent further renal tubular damage by flushing out tubular cells that are sloughing. Monitoring blood pressure, urine production and renal and electrolyte parameters q 24 hours until 48-72 hours post ingestion.



ETHYLENE GLYCOL



WHAT IT IS

Ingestion of a product containing ethylene glycol (antifreeze) causes intractable renal failure preceded by GI and neurologic symptoms.

DIAGNOSIS

Vomiting, lethargy, ataxia with a noted possible exposure (leaking radiator or chewed container of ethylene glycol), fluorescence with a black light of residue on the face/paws, vomitus or urine.

Ethylene glycol test kits are available that can detect EG intoxication 1-12 hours after ingestion.

TREATMENT

Emesis if <1 hour (and patient is not seizing or obtunded).

Activated charcoal if under 3 hours from ingestion.

IV fomepizole (4-MP) or ethanol to compete with EG for alcohol dehydrogenase. Ethanol can be hard liquor like Vodka.

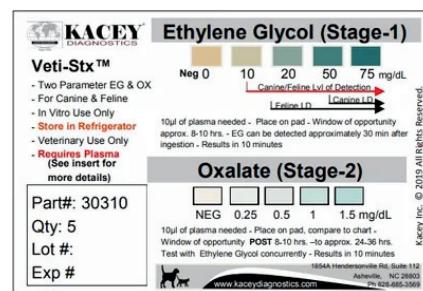
Fomepizole: Dogs: administer every 12 hours for 36 hours. Dosing is 20 mg/kg slow IV over 15 to 30 minutes, then 15 mg/kg slow IV at 12 and 24 hours, then 5 mg/kg at 36 hours.

Cats or dogs: ethanol-8.6 ml/kg 7% ethanol solution bolus, maintain at 100 mg/kg/hr up to 200 mg/kg/hr constant-rate infusion

SYMPTOMS

- 1 Stage 1: Lasts 1-12 hours. Neurologic signs- stupor, disorientation, ataxia, pu/pd. Isosthenuria can occur in this stage, metabolic acidosis developing.
- 2 Stage 2: Lasts 12-24 hours. CNS depression, seizures, pulmonary edema possible. High anion gap and severe metabolic acidosis
- 3 Stage 3: As early as 12 hours post exposure in cats but at least 24-72 hours post exposure. Oliguric/Anuric renal failure. Azotemia, depression, anorexia, vomiting.

OF NOTE

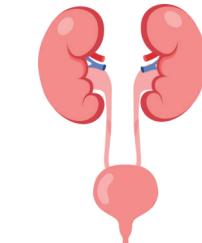


Calcium oxalate monohydrate crystal

Although there is an antidote to ethylene glycol, this toxicity is deadly without prompt diagnosis and treatment within the first ideally 6-12 hours. Monohydrate calcium oxalate urinary crystals can develop within 3 hours (cats) and 6 hours (dogs). These look like picket fence posts.

Emergency URINARY CONDITIONS

URINARY TRACT INFECTION



WHAT IT IS

Up to 27% of dogs will develop a UTI, but only 1-2% of cats!. A UTI is most commonly a bacterial infection in the bladder (rare cases can be yeast). The cause is bacteria present on the skin which ascends into the urethra and into the bladder. Females are more at risk than males because the pathway to infection is easier than the circuitous route in a male dog or cat.

DIAGNOSIS

The gold standard for diagnosing a UTI is a sterile sample collected directly from the bladder (cystocentesis) and then submitted for a culture and sensitivity.

OF NOTE

Cats under age 10 RARELY develop a UTI because cats with normal kidney function have highly concentrated urine which prohibits the growth of bacteria. Cats that develop UTI's tend to have dilute urine from conditions like kidney disease, hyperthyroidism, diabetes or a predisposing cause like perineal urethrostomy surgery or urinary catheterization.

SYMPTOMS

Dysuria : painful urination

Hematuria : blood in the urine

Stranguria: straining to urinate

Pollakiuria: frequent small urinations

TREATMENT

Sporadic cystitis (simple UTI): empiric tx : Amoxi or TMS for < 7 days (3-5 days)

Recurrent UTI: 3 or more infections in 12 months or less

Can be reinfection (new bug within 6 months of last antibiotic dose) or relapsing (same bug within 6 months of the last antibiotic dose).

Causes of recurrent UTI: functional/structural issues, internal nidus, altered immune function, ineffective antibiotic therapy

Prostatitis: empiric tx: 4-6 weeks fluoroquinolone, TMS or clinda

Pyelonephritis: 2 weeks of fluoroquinolone or 3rd gen cephalosporin

URETHRAL PROLAPSE



WHAT IT IS

Protrusion of the urethral mucosa from the tip of the penis.

DIAGNOSIS

Visualization of prolapsed tissue at the tip of the penis.



TREATMENT

Prolapsed mucosa can be reduced by passage of a urethral catheter and placement of a loose purse-string suture for up to 5 days. Applying hypertonic saline or a sugar solution may aid in reducing the prolapsed tissue. In the dog, recurrence is common. Surgical treatment with urethropexy is recommended along with castration.

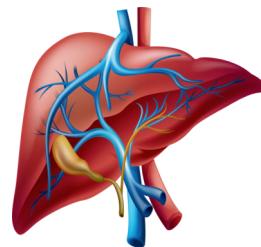
SYMPTOMS

Clinical signs and physical abnormalities include excessive licking of the prepuce/penis, bleeding from the prepuce/penis, stranguria, dysuria, and hematuria.

OF NOTE

Occurs most commonly in the English Bulldog. Rarely reported in cats. Most often in young, male, brachycephalic dogs.

Emergency HEPATIC CONDITIONS



HEPATIC LIPIDOSIS



WHAT IT IS

The most common acquired liver disease in the cat. The onset is due to a catabolic state triggered by an obese cat that stops eating for 2-7 days. This causes breakdown of fat and the excess energy is deposited and stored in the liver. Fat deposition in the liver causes severe cholestasis.

DIAGNOSIS

In a fat yellow cat that is anorexic, feed first and diagnose later!

But to diagnose:

Initial diagnosis is based on physical examination findings (jaundice, hepatomegaly) and history of anorexia

Definitive diagnosis of HL is based on the following:

1. clinicopathologic features (especially hyperbilirubinemia and increased liver enzymes)
2. ultrasonographic confirmation of hepatic parenchymal hyperechogenicity and hepatomegaly
3. cytology of a US-guided hepatic aspirate (after vitamin K1 administration)

SYMPTOMS

Weight loss, anorexia, vomiting, weakness, icterus and dehydration.

TREATMENT

Fluids with potassium supplementation based on electrolyte levels.

You may need to supplement phosphorous as well if it is below 2mg/dL.

Remember, enteral feeding may precipitate hypophosphatemia because existing phosphorous stores are depleted and the surge of insulin with refeeding triggers uptake of phosphate into cells. D/C phos supplementation when phos reaches 2 mg/dL. Monitor every 2-6 hours until stable.

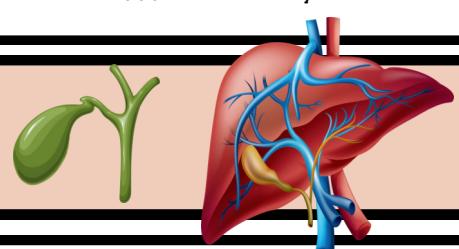
Enteral feeding (NG or esophagostomy tube if needed to go home with tube) - 25% RER up to full RER in 3-5 days.

OF NOTE

Half of cats with HL will have vitamin K deficiencies leading to prolonged PT. This is due to recycling of the vitamin by the liver via enterohepatic circulation.

Chemistry panels show marked incr in ALP. GGT normal to mildly incr; mild to marked incr in ALT/AST.

GALL BLADDER MUCOCOELE



WHAT IT IS

A gall bladder mucocoele is distension and accumulation of thick inspissated bile sludge in the gall bladder. An association has been found between dyslipidemias (hypertriglyceridemias and hypercholesterolemias) and mucocele formation.

Certain breeds that are predisposed to idiopathic hyperlipidemia like Shetland sheepdogs and miniature schnauzers are at greater risk. Endocrinopathies known to aggravate lipid metabolism such as Cushing's may be a risk factor.

SYMPTOMS

On physical examination, specific findings may include abdominal pain, icterus, fever, vomiting, anorexia, tachypnea, and tachycardia. A CBC in these dogs is usually unremarkable. Serum biochemistry panels may reveal elevated activities of ALP, ALT, aspartate aminotransferase (AST), and gamma-glutamyl transpeptidase (GGT), as well as increased concentrations of blood urea nitrogen (BUN) and total bilirubin.



TREATMENT

Surgical treatment via cholecystectomy (gall bladder removal) is recommended for dogs diagnosed with a GBM. This recommendation is because at this stage, the gallbladder is diseased and at risk for rupture. The semisolid mass of bile-laden mucus is not likely to respond to choleretic medical management.

In dogs without clinical signs, surgery can be scheduled on an elective basis.

Medical management ursodeoxycholic acid :10 to 15 mg/kg, PO, divided into 2 doses per day, and s-adenosylmethionine : 20 mg/kg, PO, on an empty stomach. Ideally these patients return once or twice a month for ultrasonographic monitoring of their GBM.



OF NOTE

Perioperative mortality rate ranges are very high for this procedure. They range from 21% to 32% because the risk for bile peritonitis and sepsis is high.

Success rates for medical management alone is low. If hypothyroidism is diagnosed, management with levothyroxine may help to improve gallbladder emptying. Low fat diets may also improve biliary flow, especially in patients with confirmed hypertriglyceridemia.

Emergency HEPATIC CONDITIONS

CHOLANGIOHEPATITIS

WHAT IT IS

Cholangiohepatitis is seen most commonly in cats versus dogs. It is the second most common cause of liver disorders in the cat. Cholangitis is inflammation of the portal region of the liver and bile duct. Cholangiohepatitis is cholangitis with extension of inflammation into the periportal areas in the liver.

Two main variants are neutrophilic and lymphocytic-plasmacytic (L/P) inflammation.

Neutrophilic cholangiohepatitis is caused by an ascending bacterial infection from the intestine into the gallbladder and liver. L/P cholangiohepatitis is caused by autoimmune disease attacking the bileducts and liver.

DIAGNOSIS

Liver biopsy and culture is recommended for diagnosis. Mild neutrophilia, mild increased bilirubin, elevated ALP and ALT

OF NOTE

Feline cholangitis/cholangiohepatitis often coexists with inflammation of the pancreas, and duodenum (triaditis).



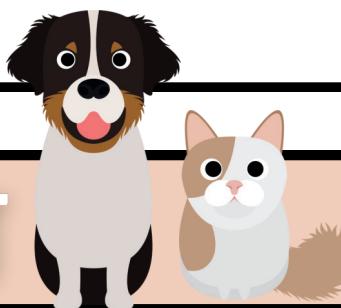
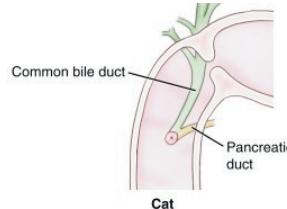
SYMPTOMS

Symptoms can vary in more chronic cases to almost no signs to more severe signs in acute cases: fever, depression, lethargy and anorexia.

TREATMENT

L/P cholangiohep is treated with steroid +/- other immune modulating drugs. Neutrophilic cholangiohepatitis is treated with antibiotics based on liver or bile culture. Liver protectants such as SAM-E and ursodiol to thin gall bladder secretions are also often used.

Common empiric antibiotics used are amoxi-clav and metronidazole.



POTOSYSTEMIC SHUNT

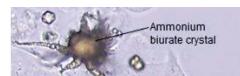
WHAT IT IS

A portosystemic shunt is an abnormal communication between the portal (liver) circulation and the systemic circulation. There are 2 categories of liver shunts: extrahepatic and intrahepatic. Most dogs are born with the shunt (congenital) but some develop (or acquire) the shunt later. Normally, blood from the intestinal tract, spleen, and pancreas is deposited into the portal vein. This blood is filtered and detoxified in the liver through the portal vein. When the liver is deprived of this blood, it gets smaller in size due to the lack of hepatotrophic factors.

Microvascular dysplasia (MVD) is a congenital vascular anomaly that is not a large shunting vessel, but a problem within the branches of the portal circulation: the branches attenuate too quickly. This leads to decreased vital blood supply to the liver.

DIAGNOSIS

Some patients are clinically normal but have elevated liver enzymes on routine blood work (ALT, ALP). Serum bile acids are often >90mmol/L. Other biochemical changes can include hypoalbuminemia, low cholesterol and decreased BUN. On x-rays the liver is often smaller than normal. A portosystemic shunt can be identified with imaging of the liver and vasculature. This is most commonly identified with an ultrasound study, or a CT scan. If you suspect MVD (no shunting vessel is obvious) the a blood test measuring protein C can be performed. Dogs with a shunting vessel have a low level of protein C. Protein C is normal with MVD. An absolute diagnosis of MVD is via liver biopsy, Venoportography, or angiography.



SYMPTOMS

Clinical signs of hepatic encephalopathy can be mild (very calm or quiet) or more obvious: post prandial head pressing, seizures, and somnolence

TREATMENT

Gradual closure of the shunting vessel with an ameroid constrictor, cellophane band or intravascular coil to treat extra and intrahepatic shunting vessels. Closure of the shunt should happen slowly to hopefully prevent life-threatening portal hypertension. MVD has no surgical option. Treatment is medical management with: a protein restricted liver diet, and lactulose to acidify the colonic lumen and trap and excrete excess ammonia as well as reduce the bugs that produce excess ammonia. Metronidazole may also help to reduce ammonia producing gut microbes: 7.5mg/kg po bid.

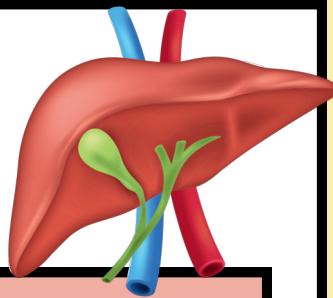
OF NOTE

Protein breakdown into nitrogen by gut microorganisms produces ammonia. Ammonia travels to the brain to cause symptoms of hepatic encephalopathy. Measuring ammonia is difficult because it must be processed immediately as it is a volatile compound. It's not frequently performed.



Emergency HEPATIC CONDITIONS

LIVER TOXICITY



WHAT IT IS

The liver is vulnerable to toxins because it is the initial organ to process blood from the intestinal tract after eating. The common liver toxins include xylitol, acetaminophen, amanita mushrooms, blue green algae and cycads.

SYMPTOMS AND DIAGNOSIS

Suspect exposure combine with appropriate symptoms. Common symptoms are vomiting, diarrhea, and lethargy. Other signs: acute liver enzyme elevations, coagulopathy, hemorrhage, hypoglycemia, depression.

TREATMENT

Emesis if recent ingestion is suspected, activated charcoal (charcoal is not much use with xylitol).

Cholestyramine for cyanobacterium to bind microcystin

Acetylcysteine for acetaminophen toxicity

Supportive care treatment as needed for blood sugar control, plasma for clotting support

OF NOTE

Xylitol (canine only, cats are not affected): 500mg/kg causes liver failure. 50mg/kg causes hypoglycemia

Acetaminophen: Cats are most susceptible to the toxic effects given their inability to glucuronidate to a non-toxic metabolite. It causes NAPQI to build up and damage red blood cells causing life-threatening methemoglobinemia.

Amanita mushrooms: Ingestion of 1 cap can be lethal to dogs/cats/humans

Cyanobacterium (blue green algae): Dogs are most commonly exposed after swimming or drinking in infected water which causes hepatic necrosis; some varieties are neurotoxic

Cycad plants have cones with seeds. 1-2 seeds can kill a medium sized dog if ingested: hepatocellular necrosis



LIVER NEOPLASIA



WHAT IT IS

Metastatic neoplasia (cancer) is more common than primary liver tumors. Metastasis tend to arise from the spleen, pancreas and GI tract. The most common primary liver tumor in dogs is hepatocellular carcinoma. Over 60% are solitary massive hepatocellular carcinomas. These are slow to metastasize and potentially can be removed (particularly if on the left side, away from the giant vessels). The nodular or diffuse hepatocellular carcinoma forms, metastasize 90% of the time. The most common primary liver tumor in cats are: biliary cystadenoma, adenoma and carcinoma.

DIAGNOSIS

Massive, solitary hepatocellular carcinoma of dogs is often found incidentally as dogs don't often have any symptoms until later in the disease. Imaging with ultrasound is the most common method to identify liver tumors.

Biopsy is required for a formal diagnosis/interpretation.

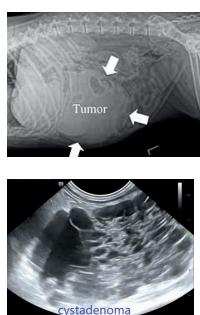
Multiple liver masses they could just be benign nodular change (hyperplastic regenerative nodules) which has a good prognosis and does not require surgical removal. Additionally, ill defined nodular areas can also be from benign vacuolar hepatopathy. The best imaging modality for the liver is CT.

OF NOTE

Cats develop benign liver tumors more often than dogs.

Half of all feline liver tumors are benign biliary cystadenomas!

The most common neoplastic tumor is a bile duct carcinoma. Biliary cystadenoma- feline ultrasound (and it's the 2nd most common liver tumor type in dogs).



SYMPTOMS

There may be no symptoms or symptoms of lethargy, weight loss, decreased appetite, vomiting, abdominal distension and jaundice.

TREATMENT

Surgical removal of canine massive hepatocellular carcinoma; even incomplete surgical margins did not affect survival times!

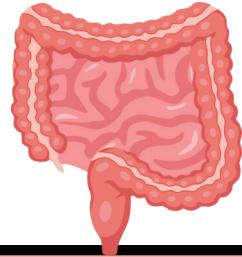
Surgical removal of biliary cyst adenomas in cats is recommended if possible.

Surgery is not recommended with metastatic liver neoplasia.

A recent study of liver "lesions" detected by ultrasound prior to splenectomy for a splenic mass in dogs showed: a higher probability of those lesions being benign than malignant.

Emergency GASTROINTESTINAL

GI LYMPHOMA



WHAT IT IS

Lymphoma (LSA) is the most common cancer in cats and the most common location is in the GI tract. Two main types are high grade and low grade lymphoma (LSA). Low grade (indolent, small cell) GI LSA can be difficult to distinguish from IBD as both have small lymphocytes invading the intestinal architecture. LGL has a slow course and cats may survive several years. High grade LSA has a more acute onset with

DIAGNOSIS

Abdominal ultrasound is often used to determine if there is classic intestinal thickening of the muscularis or if there is intestinal lymphadenopathy. PARR may be helpful to determine if there are antigen re-arrangement present on a cytology sample. A negative PARR does not rule out GI LSA. Cytology can be performed if there are prominent/enlarged GI lymph nodes. Gold standard diagnosis is based on full thickness (surgical biopsies) but endoscopic biopsy can often reveal a diagnosis.

OF NOTE

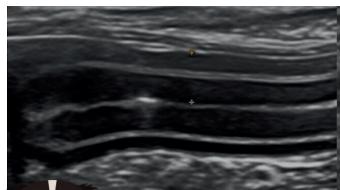
Low grade LSA has a slow course and cats may survive several years. High grade LSA (large cell) has a more acute onset and cats are often feeling poorly on presentation. Often there is an abdominal mass and sometimes peritoneal effusion present. Multi-agent therapy with CHOP or COP is recommended. (cyclophosphamide, Doxorubicin (hydroxydaunorubicin), Vincristine (Oncovin), Prednisolone).

SYMPOMTS

Clinical signs are typically vague: weight loss, inappetance, vomiting, diarrhea. Bloodwork is often unremarkable. Abdominal palpation sometimes reveals ropey or palpably thickened intestines. Occasionally there may be a mass effect.

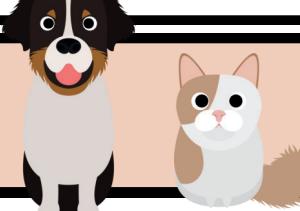
TREATMENT

With HGL Multi-agent therapy with CHOP or COP is recommended.. 50-65% will respond with a MST of 6-12 months. In LGL chlorambucil, prednisolone and cobalamin supplementation if needed. There is an 80% response rate and MST of 1-3 years.



Thick muscularis approaches the same thickness as the submucosa.

OBSTRUCTION



WHAT IT IS

Intestinal obstruction is a blockage in the bowel and is an emergency. The most common causes of obstruction that are extra-luminal are intussusceptions.

Neoplastic masses can also cause extraluminal or intraluminal obstructions.

In dogs, foreign body ingestions are frequently the cause.

Young indoor cats and young large breed dogs are more likely to present for foreign body ingestion (in cats, linear fb's are far more common than in dogs).

DIAGNOSIS

Imaging helps provide the most support for an obstruction. Plain x-rays have some value but often foreign bodies are not radio-opaque. Diagnosis based on x-rays relies on interpreting gas patterns. Abdominal ultrasound is frequently the cornerstone for emergent diagnosis as this can follow the GI tract lumen. In young cats, trying to look under the tongue is an important part of the physical exam.

SYMPTOMS

The most common symptom is vomiting. Other symptoms include diarrhea, abdominal pain and inappetance.

TREATMENT

Sometimes foreign material can move through the GI if the patient is admitted and placed on iv fluids. However, if the GI tract appears compromised and the patient has any signs of infection / peritonitis then surgery is the best choice. Sometimes linear foreign bodies can be cut from their attachment and then milked into the colon.

Here is a "red rubber" catheter trick to use on linear foreign bodies: <https://www.kickassvets.com/blogs/redRubber.php>

OF NOTE

Animals with signs of peritonitis are at higher risk of sepsis. With enterotomies and resection surgeries, the highest risk of dehiscence is 3-5 days post op.

Diagnosing septic peritonitis: intracellular bacteria noted on cytology of effusion . Also An effusion lactate concentration of 4.2 mmol/L was 72.2% sensitive and 84.2% specific for the diagnosis of septic peritonitis in one study.



Emergency GASTROINTESTINAL

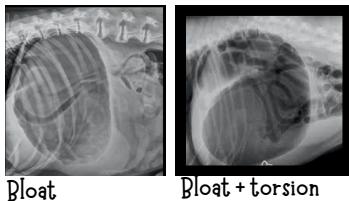
GASTRIC DILITATION VOLVULUS

WHAT IT IS

GDV is life threatening enlargement of the stomach with food and gas (bloat) followed by torsion (twisting) in a clockwise fashion around the distal esophagus. Depending on the degree of twisting, the duodenum is often trapped between the distal esophagus and the stomach. Gas is trapped in the stomach and often there is outflow obstruction and splenic entrapment/engorgement as the spleen is intimately connected to the stomach.

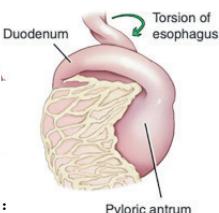
DIAGNOSIS

The classic diagnosis is via a right lateral x-ray showing a popeye arm or a "double bubble" effect. The pylorus shifts into a dorsal position on the x-ray.



OF NOTE

Poor prognostic indicators: symptoms for >6 hrs
Lactate >6 mmol/L initial. 58% survival rate
Indicates gastric necrosis more likely and longer hospitalization times.
Initial lactate <4 mmol/L: higher survival, less complications
Drop in lactate by 40% or more after fluid resuscitation: improved odds



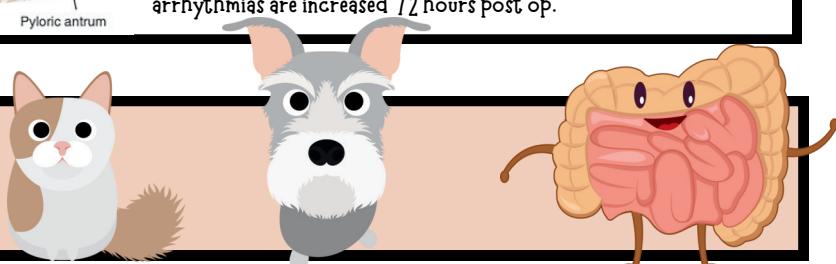
SYMPTOMS

GDV is life threatening enlargement of the stomach with food and gas (bloat) followed by torsion (twisting) in a clockwise fashion around the distal esophagus. Depending on the degree of twisting, the duodenum is often trapped between the distal esophagus and the stomach. Gas is trapped in the stomach and often there is outflow obstruction and splenic entrapment/engorgement as the spleen is intimately connected to the stomach.

TREATMENT

Prompt decompression with trocharization and surgery. Initial steps: Lactate/blood gas, CBC, biochem panel, right lateral abdomen, chest xrays in an older dog. ECG to look for arrhythmias. Fluid resuscitation, pain management, gastric lavage and surgery to derotate the stomach and pexy. Assess the splenic health and in some cases remove the spleen if there appears to be lack of effective blood supply. ECG monitoring before and after surgery as ventricular arrhythmias are increased 72 hours post op.

PLE



WHAT IT IS

Protein Losing enteropathy is a condition of excessive loss of albumin (the major serum protein), through the intestinal tract. Low albumin is the hallmark change. However, other bloodwork changes include low globulin, calcium, cholesterol, and lymphocyte counts. A diverse range of underlying causes exist for this disease. However, certain breeds are at increased risk: Yorkies, Soft Coated Wheaten Terriers, Norwegian Lundehunds, and Basenjis.

Chronic GI diseases that can cause PLE include: IBD, lymphangiectasia, GI LSA, histoplasmosis, pythiosis and GI parasites.

DIAGNOSIS

Although the levels of globulin loss vary, panhypoproteinemia helps to distinguish this disease from PLN (protein losing nephropathy). Hyperechoic mucosal stippling or striations are associated with lacteal dilation, having a sensitivity of 75% and a specificity of 96% for PLE.



SYMPTOMS

The most common symptoms are vomiting, diarrhea, inappetence and weight loss. Ascites, edema and pleural effusion can develop if albumin levels drop very low. These dogs are also at risk for clots because they may lose anti-thrombin through their GI tract.

TREATMENT

Plasma infusions for severely low proteins. B12, appetite and nausea support, ultra-low fat or hypoallergenic diet, and immunosuppressants (often a steroid with a second line agent like cyclosporine, or mycophenolate).

OF NOTE

Poor prognostic indicators: serum albumin <2, low cobalamin (B12), high or low BUN, chronic vomiting, low body weight. Survival rates are reported to be under 50%. Of course, this depends on the underlying cause and response to treatment.

Emergency GASTROINTESTINAL

INTUSSUSCEPTION

WHAT IT IS

A condition where a portion of a loop of bowel telescopes into an adjacent portion of a loop of bowel. This is seen most commonly in bowels that are inflamed. Puppies with diarrhea are susceptible. Common inciting causes include: parasitic or other GI infections, intestinal foreign bodies, masses or recent intestinal surgery. The intussuscepter swallows the intussusceptum.

DIAGNOSIS

Imaging with ultrasound can detect a "target sign" when the intussusception is visualized in cross section. On abdominal x-ray sometimes you may see a crescent shaped gas structure from a trapped pocket of gas inside an intussusception. Sometimes palpation of a sausage like loop of bowel is possible. Dogs or cats will typically have episodes of vomiting or diarrhea prior to an intussusception event. These invaginations can be temporary (and self-limiting) or chronic (requiring surgical reduction).



SYMPTOMS

Vomiting, diarrhea, abdominal pain.

TREATMENT

If the intussusception is not self-limiting, then surgical reduction is often needed. If the bowel has been strangulated, sometimes resection/anastomosis is required. Some surgeons will perform enteroplication surgery to tack the bowel loops together to help prevent further intussusception.

OF NOTE

Most common in dogs under 1 year of age. The ileocolic junction is the most common location. Most times no definitive cause is identified.

ULCER

WHAT IT IS

GI ulceration is seen most often in dogs. It is a disruption in the normal mucosal barrier and occurs most often in the stomach or duodenum. It can be caused by steroids, NSAIDS, cancers (gastrinoma, disseminated mast cell disease, gastric carcinoma), and systemic disease (addison's, liver and renal diseases).

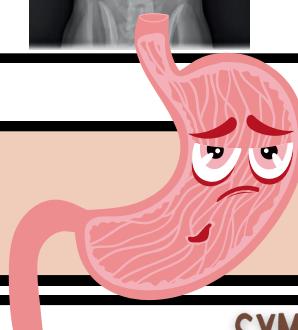
Steroids and NSAIDS interfere with COX1 production of beneficial prostaglandins that help to protect the stomach lining.

DIAGNOSIS

Abdominal ultrasound is probably the most useful way to quickly detect GI ulceration by looking for local thickening with a crater in the center.

OF NOTE

NSAID administration, neoplasia, and hepatic disease are the most common reported causes of gastroduodenal ulceration or perforation in dogs.



SYMPTOMS

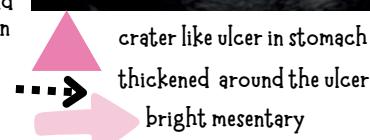
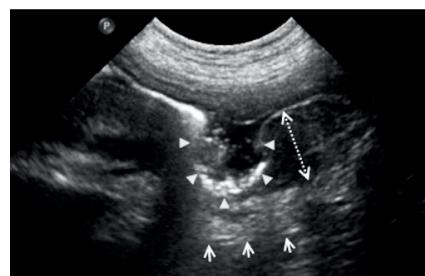
The most common symptoms are vomiting, hematemesis, melena and anemia.

TREATMENT

Treatment is aimed at removing or reducing circumstances that promote ulceration (ie identifying and treating renal or hepatic diseases, neoplasia etc.), supporting gastric mucosal perfusion and reducing gastric acidity, and protecting the ulcer.

Common drugs used are:

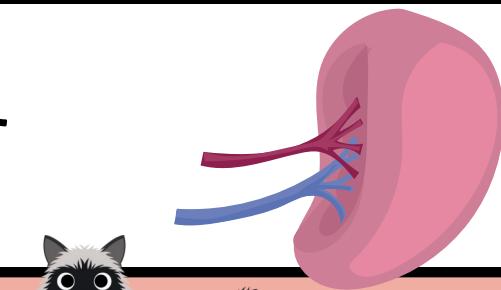
- H2 inhibition (omeprazole) or H2 antagonism (famotidine). Omeprazole is superior to famotidine at reducing acidity.
- Prostaglandin analogues like misoprostol to increase mucosal circulation and mucosal surface turnover.
- Cytoprotective drugs like sucralfate to bind and coat the ulcerated areas in the stomach/duodenum.
- Surgery is not frequently performed, but will be pursued if there is a complete perforation or severe hemorrhage.



Emergency

SPLENIC

FELINE SPLENIC MAST CELL DZ



WHAT IT IS

Mast cell tumors are the most common cause of splenic tumors in cats. The other common location is in the GI tract. Other common splenic cancers are lymphoma and hemangiosarcoma. In cats with signs of multiple myeloma, 50% have splenic or liver involvement.

DIAGNOSIS

Circulating mast cells almost always indicate serious disease in cats (mast cell tumor in the spleen or GI and sometimes other cancers (LSA or HSA). Diagnosis is via splenic needle biopsy.

TREATMENT

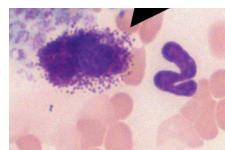
Treatment of choice is splenectomy even with cutaneous spread. 30% of cats with splenic MCT have cutaneous mast cell tumors. Median survival is up to 2 years in cats with splenectomy. Addition of chemotherapy is of unknown benefit.

SYMPTOMS

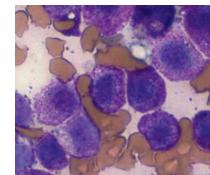
The most commonly recorded clinical signs are weight loss, anorexia, vomiting, and lethargy. One study showed splenomegaly was noted in 44% of cats upon physical examination and 30% had concurrent cutaneous MCT.

OF NOTE

Anemia and mastocytemia were noted in 47% and 35% of evaluated blood samples, respectively, and metastasis to the liver was present in 52% of biopsy or fine needle aspirate samples. Unlike other cancers, surgery can still be beneficial even with metastasis to the skin. And in some cases, splenectomy will cause regression of the cutaneous masses.

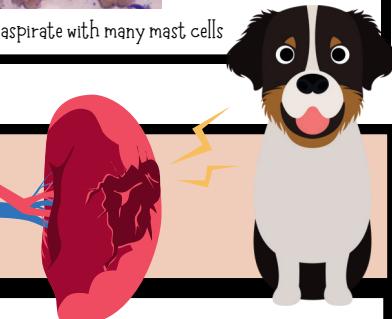


Circulating Mast cell



splenic aspirate with many mast cells

CANINE HEMANGIOSARCOMA



WHAT IT IS

Canine hemangiosarcoma (HSA) is the most common tumor of the spleen in dogs. It is a tumor of blood vessels and can lead to blood vessel fragility and bleeding. The double 2/3rds rule: 2/3rds of all splenic tumors in dogs are malignant. Of those malignant tumors, 2/3rds are HSA. Most HSA's are assumed to have spread microscopically at the time of diagnosis. The 2/3rds rule only applies to ruptured splenic tumors. For splenic masses found incidentally ("incidentalomas"), only 30% are malignant.

DIAGNOSIS

FNA is not recommended for cavitated splenic tumors as it is low yield and imparts risk for hemorrhage. Definitive diagnosis is via histopathology of the entire spleen submitted after surgical removal. Because of the high incidence of metastasis, screening chest x-rays should be taken prior to surgery. While full echo is generally not recommended as a screening tool prior to surgery, a T-FAST for pericardial effusion is reasonable screening tool. If metastasis is already visible, surgery is generally not recommended given the poor prognosis for any meaningful survival time. Other perioperative diagnostics should include clotting times, CBC and biochemical panel. Some patients will require a blood transfusion. Peri and post-operative ECG monitoring for arrhythmia is also recommended.



accelerated idioventricular rhythm is common with splenic disease

SYMPTOMS

Unfortunately, a common scenario for diagnosis is after the splenic tumor ruptures. The dog presents urgently with acute anemia, pale gums, panting, lethargy and hemorrhagic effusion in the abdomen.

TREATMENT

Splenectomy and chemotherapy in patients without gross metastasis is the treatment of choice.

OF NOTE

Prognosis: less than 10% of dogs with aggressive treatment survive a year. With surgery alone, survival is predicted to be only 1-3 months. In dogs treated with surgery and chemotherapy, expected survival increases to 6 to 9 months.

Following surgery, addition of a medicinal mushroom blend I'm-Unity or K9 Immunity plus may increase survival times. For dogs with metastatic nodules, Yunnan Baiyao (a Chinese herbal supplement) may help decrease hemorrhage.

A 2024 study showed liver nodules present in 20% of patients with splenic masses, but NO association between liver lesions found on ultrasound and metastatic disease.

Emergency

PANCREATIC

CANINE PANCREATITIS

WHAT IT IS

Pancreatitis is inflammation of the pancreas. It can be acute or chronic. Most acute cases present with symptoms: anorexia, weakness, vomiting, abdominal pain, fever are the main symptoms in dogs. In addition to breed related factors, predisposing factors include: high fat diets, hypertriglyceridemia of min schnauzers ($>860 \text{ mg/dL}$), Diabetes, hypothyroidism and cushing's disease as well as certain drugs.

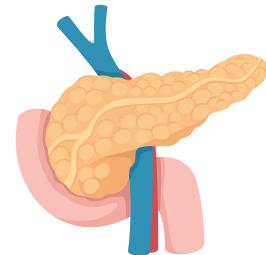
DIAGNOSIS

The best way to diagnose pancreatitis is with imaging of the pancreas to show inflammation of the organ. The most common imaging modality used is ultrasound which shows a hypoechoic (darker looking) pancreas with a bright surrounding mesentery. Serum amylase and lipase are not specific enough to be reliable. However, pancreatic lipase immunoreactivity (PLI) is specific for pancreatic inflammation. canine PLI $<200 \text{ mg/L}$ rules out pancreatitis, and $>400 \text{ mg/L}$ supports pancreatitis.

Hyperlipidemia is a risk factor in dogs but not cats.



Abdominal ultrasound shows a hypoechoic (dark) pancreas with hyperechoic (bright) surrounding mesentery



SYMPTOMS

Most acute cases present with symptoms: anorexia, weakness, vomiting, abdominal pain, fever are the main symptoms in dogs. In cats, signs are less specific as the main two symptoms are anorexia and lethargy.

TREATMENT

Supportive care treatment as needed to promote hydration, provide pain relief, enteral feeding with low fat diets (in dogs), high protein diets (cats) anti-nausea medications.

Panobell-PAL (fuzaplatid) is approved for use in acute pancreatitis in dog. It is an iv injection given once a day for 3 days to help prevent migration of neutrophils into the pancreas that occurs in acute pancreatitis. It can help these dog recover more quickly but it should be used early in the disease process to work well.

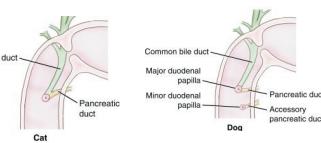
OF NOTE

Acute pancreatitis: Min schnauzer, yorkie, other terriers

Chronic pancreatitis: Cavaliers, cockers, Boxers, Collies

Feline pancreatitis: Siamese cats

Extrahepatic bile duct obstruction can occur in both dogs and cats if the pancreas swells and closes off the bile duct. This causes sharp elevations in T bili and jaundice.



FELINE PANCREATITIS



WHAT IT IS

Pancreatitis is inflammation of the pancreas. Most cases of pancreatitis are idiopathic but there are some known risk factors or triggers. Clinical pancreatitis is more common in dogs. Cats frequently are found to have pancreatitis, but a smaller subset develops clinically apparent disease.

DIAGNOSIS

Abdominal ultrasonography is the most commonly utilized modality to diagnose pancreatitis. A hypoechoic pancreas with hyperechoic surrounding mesentery is a recognized common set of features. Liver enzymes ALT, AST, and bili may be increased due to biliary tree inflammation, bile duct obstruction, or hepatic inflammation.



OF NOTE

ACVIM Consensus Statement for Feline Pancreatitis

95% of cases of pancreatitis in cats is considered idiopathic. Unlike dogs, a normal fPLI should not be used to rule out pancreatitis, however, a positive test is more specific for pancreatitis.

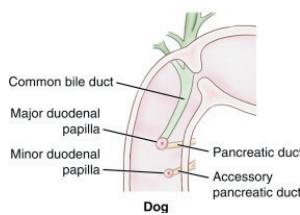
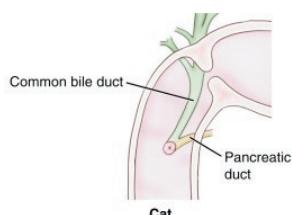
Cats have a merged common and pancreatic duct. This sometimes leads to a backwash of pancreatic fluid into the biliary tree. This causes inflammation and infection of the biliary tree and liver (cholangitis/ cholangiohepatitis).

SYMPTOMS

In cats, signs are less specific as the main two symptoms are anorexia and lethargy. In some cases, the pancreatic swelling leads to extrahepatic bile duct obstruction and causes jaundice.

TREATMENT

Treatment is largely supportive care unless an inciting cause can be directly treated. Fluid therapy to hydrate and correct electrolyte imbalances, appetite stimulants, prokinetic agents (metoclopramide, erythromycin, cisapride) if there is GI stasis, antiemetics. Antacid therapy is no longer routinely recommended unless there is suspicion for esophagitis or upper GI bleeding. Pain management (fentanyl, buprenorphine, etc) and enteral nutrition are cornerstones of treatment. Cats have a higher tolerance to dietary fat than dogs do. But caution feeding the long-term diet to cats when in hospital to reduce food aversion.



Emergency

PANCREATIC

INSULINOMA

WHAT IT IS

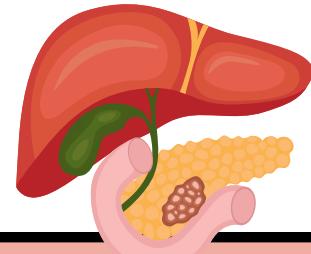
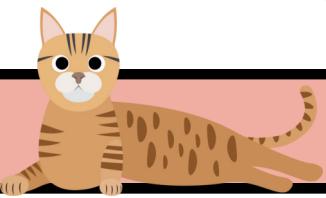
Insulinoma is a tumor of the pancreas that secretes inappropriate amounts of insulin leading to profound hypoglycemia.

DIAGNOSIS

Gold standard diagnosis is not possible without histopathology of an insulin-secreting tumor in the pancreas. But suspicion for insulinoma comes from documenting hypoglycemia (<40mg/dl) and an insulin: glucose ratio demonstrating inappropriate secretion of insulin despite low blood sugar. An ultrasound study showing a nodule in the pancreas or a CT scan can be helpful. Ultrasound studies are much less sensitive, and only detect about 50% of insulinomas. CT studies increase visibility of insulinomas to about 70%.

OF NOTE

Insulinoma is one of the rare tumors where surgical excision is still recommended even when there is metastasis present. About 50% of dogs will have metastasis at the time of diagnosis. However, removal of the tumor will substantially help clinical signs that result from hypoglycemia. Median survival times in dogs with mets is about 7 months. Without mets is about 2 years.



SYMPTOMS

Physical exam signs are not present. But symptoms can be: weakness, ataxia, extreme fatigue after exercise, and collapsing episodes. Seizures can occur later in the disease process.

TREATMENT

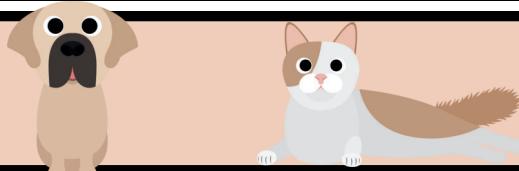
Gold standard treatment is surgical removal of the tumor. Dogs that can't have surgery can be medically managed with frequent, small meals. Treatment with drugs like prednisone, diazoxide, and octreotide can also help stabilize blood sugar levels. Streptozotocin is recommended for dogs with metastatic disease to control symptoms.

Insulinoma Survival Times Reported in the Literature: 1993 – 2013

OVERALL	MEDICAL MANAGEMENT	SURGICAL MANAGEMENT	ADJUVANT THERAPY (STREPTOZOTOCIN)
Polton et al (2007) ² MST: Stage I: 785 d Stage II: 547 d Stage III: 217 d	Polton et al (2007) ² MST: 196 d	Polton et al (2007) ² MST: 785 d	Northrup et al (2013) ¹² (all stages): MPFS: 196 d MST: 308 d
Trifonidou et al (1998) ³ MST: 258 d MDFI: 244 d	Tobin et al (1999) ⁷ MST: 74 d	Tobin et al (1999) ⁷ MST: 381 d	Moore et al (2002) ¹¹ (stage II or III) MTN: 163 d
	Dunn et al (1993) ¹⁰ MST: 14 mo	Dunn et al (1993) ¹⁰ MST: 14 mo	

MDFI = median disease free interval; MPFS = median progression free survival; MST = median survival time; MTN = median time of normoglycemia

DKA



WHAT IT IS

Diabetic Ketoacidosis occurs from prolonged hyperglycemia and insulin deficiency. This leads to breakdown of fat for energy because insulin is not driving glucose into the cells. The fatty acids are converted into ketones. These excess ketones build up and cause blood acidosis. Ketones and glucose are excreted in the urine and lead to loss of sodium, potassium and water. This causes dehydration and metabolic acidosis.

DIAGNOSIS

Ketonuria, hyperglycemia, acidosis on blood gas evaluation
Checking for ketones in the blood serum is more sensitive than in the urine.



OF NOTE

~70% of dogs with DKA are discharged from the hospital. The median duration of hospitalization was 6 days in one study. A feline study documented a mortality rate of 26% for feline DK or DKA patients. Azotemia, metabolic acidosis, and hyperosmolality were more severe in those patients than died compared to those that survived. Recurrence of DK or DKA was documented in 42% of these cats.

Concurrent illnesses frequently seen with DKA in dogs: uti, pancreatitis, Cushing's, hypothyroidism, neoplasia, pneumonia.

Cats with DKA: Hepatic lipidosis, cholangiohep, UTI, pancreatitis, CKD, neoplasia. Some cats develop diabetes after receiving steroids.

SYMPTOMS

PU/PD, inappetance, nausea, vomiting, lethargy, weight loss.

TREATMENT

1. Correct dehydration
2. Supplement potassium and phosphorous
3. Regular insulin therapy (in most cases)
4. supportive care (for nausea, appetite etc)



Sick pets with DKA are typically hospitalized. A pet that has urine ketones and hyperglycemia that is still eating can be started on long acting insulin at home.

Potassium and phosphorous will drop quickly when starting insulin because insulin drives these electrolytes intracellularly. Monitoring carefully is most important in the first 12-24 hours. Severe hypophosphatemia can cause acute anemia.

Emergency



NEURO

SEIZURES



WHAT IT IS

Uncontrolled electrical activity in the brain causing abnormal muscle movements (focal areas or full body tonic/clonic movements). Epilepsy does happen in cats as well as dogs, but with far less frequency.

DIAGNOSIS

A seizure is typically based on its classic tonic/clonic movements. But partial seizures can be more challenging to determine the cause. Lack of awareness during the movement is common.

Sometimes syncope can be mistaken for a seizure. Some distinguishing features: seizures commonly happen during sleep, not syncope. While urination can happen with both, other signs such as vomiting, drooling can happen with seizures. Behavior changes are frequently seen immediately before or after a seizure. Abnormal heart rhythm, pulse or murmur is more commonly detected if the sign was syncope.

Epilepsy is seizure activity with no abnormal brain imaging or obvious underlying disease that would cause seizures.

In dogs epilepsy typically develops between 6 months and 6 years of age.

Epileptic dogs should remain neurologically normal between seizures.

OF NOTE

Emergency seizure management: Check spot BG immediately and provide diazepam/midazolam 0.25mg/kg IV
If BG <40 give 0.5-1 ml/kg diluted 1:3 with saline. If seizures continue, start to load with phenobarbital (if pet not already on it). Give 20 mg/kg IV divided in to 4-5mg/kg IV doses q 30 min to 1 hour for 4 doses. Can use diazepam CR if needed.

SYMPTOMS

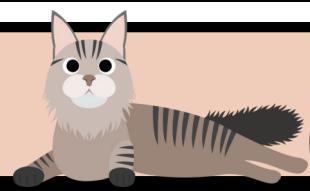
Seizures can be simple (partial uncontrolled motor movement- fly biting, abnormal vocalization, only a few muscles contracting involuntarily, etc.) or complex (generalized abnormal muscle movements with loss of awareness). Seizures most frequently happen during changes in brain activity- excitement, feeding, falling asleep or waking up.

TREATMENT

Treatment with anti-seizure medications are started if there are cluster seizures, a seizure longer than 5 minutes, 3 or more seizures in 24 hours, two or more seizures in 6 months, prolonged or unusual post-ictal events. Drug selection varies based on tolerability, cost, and ease of use.

In dogs the most common drugs used are: phenobarbital, zonisamide and levetiracetam. Cats most commonly use phenobarbital or zonisamide. They cannot use potassium bromide because they can develop severe fatal bronchitis. Levetiracetam in cats must be dosed TID. They are too small to use the ER formulations that allow BID dosing.

VESTIBULAR DZ



WHAT IT IS

The vestibular disease controls balance. The two main categories of vestibular disease are central (in the brain) or peripheral (middle/inner ear).

DIAGNOSIS

Accompanying neurologic symptoms are often helpful to localize the vestibular event as either peripheral or central.

Peripheral

Head tilt
Ataxia
Rotary, horizontal nystagmus

usually >60bps
normal mentation
Normal CP's
VII, occasionally V
Possible Horner's

Central

Head tilt
Ataxia
Rotary, horizontal, vertical nystagmus, changing direction is possible

Usually <10bps
abnormal mentation
CP deficits
Multiple
No Horner's

SYMPTOMS

Symptoms of vestibular disease are a head tilt and circling and falling to one side and nystagmus of the eyes.

TREATMENT

For peripheral disease symptomatic treatment with assistance to eat/drink/eliminate, anti-nausea medication/vertigo (cerenia, meclizine), treatment as indicated for any otitis. Treatment as indicated for any central causes.

OF NOTE

Idiopathic vestibular disease tends to occur acutely in dogs male or female ages 12-13. Signs typically last a few days for up to a few weeks. Some dogs have a residual mild head tilt.

Causes of central disease:

Metronidazole toxicity (usually >60mg/kg/day)

Cerebrovascular accident

MUE (meningoencephalitis of unknown etiology)

brain tumor

trauma

hypothyroidism can cause both central and peripheral dz

structural brain disease (chiari malformation, hydrocephalus)

The most common cause of peripheral vestibular dz is otitis media/interna, followed by idiopathic ("old dog") vestibular disease.

Baseline tests: CBC/chem/t4, BP, good otoscopic and neurologic exams

NEURO

BRAIN TUMOR

Emergency



WHAT IT IS

Brain tumors happen more commonly in dogs older than 9 and cats older than 12. Meningiomas and gliomas are the most common brain tumors in dogs. Head conformation in dogs is a risk factor for some specific tumor types. Meningioma is more common in the long-nosed breeds (e.g. golden retriever), whereas glioma is common in the short-nosed breeds (e.g. boxer, Boston terrier). Meningioma is the most common brain tumor type in cats.

DIAGNOSIS

Diagnosis is based off of imaging with MRI. Biopsy is not always due to location.

SYMPTOMS

Common symptoms: altered mentation (e.g. mental blunting, stupor, coma), behavior changes, seizures, loss of coordination and circling. Seizures are the most common clinical sign in dogs and cats usually have vague or nonspecific signs (anorexia and lethargy being most common). If a dog has its first seizure after 4 years of age, a brain tumor should be considered as a disease differential.

Meningiomas arise from the leptomeninges, they sit between the dura and the brain. They are large, extra-axial masses. Gliomas arise from the brain parenchyma. They are intra-axial.

TREATMENT

Goals for treatment of brain tumors are tumor removal or size reduction and control of secondary effects (mentation changes, seizures etc).

Surgical resection and radiation therapy are the common definitive methods used to treat canine and feline brain tumors.



OF NOTE

Head conformation in dogs is a risk factor for some brain tumor types! Meningioma is more common in the long-nosed breeds (e.g. golden retriever), whereas glioma is common in the short-nosed breeds (e.g. boxer, Boston terrier). Meningioma is the most common brain tumor type in cats.

Median survival times for dogs after radiation:

Glioma 9-14 months Meningioma 20-24 months

Cats that have sx removal of their meningiomas have a good long term prognosis

IVDD



WHAT IT IS

Intervertebral disc disease is a ruptured or bulging disk in the spine.

DIAGNOSIS

A neurologic exam and imaging with MRI or myelogram are the best ways to formally diagnose IVDD.

TREATMENT

Mild paresis and pain can be managed with strict rest and pain relievers: gabapentin, anti-inflammatories. Surgery is recommended for Grades III and IV IVDD. There is a 50-60% chance of recovery if surgery is performed within 48 hours of loss of pain recognition (grade V).

OF NOTE

Chondrodystrophic dogs have premature degeneration of their intervertebral discs.

Of the chondrodystrophic dogs that develop a herniated disc one quarter of them are in the Daschund breed.

SYMPTOMS

Symptoms can be mild back pain and stiffness, reluctance to rise or climb stairs/get up on furniture, to more severe signs of paresis or paralysis.

Grade I disc disease is characterized by spinal pain only and no neurologic impairment.

Grade II disc disease is characterized by mild to moderate weakness (paresis). The patient can still walk but has proprioceptive deficits (diminished ability to know where their feet are).

Grade III disc disease is neurologic weakness so severe the dog cannot walk. These dogs can still move their back legs however. They may also have difficulty urinating.

Grade IV disc disease is paralysis with urine retention. The dog can still feel sensation to their back legs with a pinch on their toes.

Grade V has the most grave prognosis because there is complete paralysis and lack of sensation of deep pain.

Proprioception spinal reflexes muscle tone	UMN absent/reduced present good	LMN absent /reduced absent/reduced poor, rapid atrophy
C1-C5	UMN	UMN
C6-T2	LMN	UMN
T3-L3	normal	UMN
L4-S1	normal	LMN
S1-S3	normal	some LMN lesions

Anterior Uveitis

Pathophys

Uveitis is any condition that causes uveal tract inflammation. This leads to breakdown of the blood/aqueous barrier/blood-retinal barrier and allows protein and cellular accumulation

Clinical signs

Episcleral erythema, +/- photophobia, +/- aqueous flare, miosis or anisocoria, rubeosis iridis, iris bombe

IOP <10 mmHg OR a difference of 10mmHg or more between the 2 eyes is supportive
+/- corneal edema. Remember, Hyphema or hypopyon always = uveitis!

Treatment

If the patient has normal pressures (8-18mmHg) in the face of obvious anterior uveitis, start a topical steroid (1% pred acetate or 0.1% dexamethasone) and dorzolamide BID as a preventative for possible pressure spikes.

Always check a fluorescein stain to ensure no corneal ulceration is present before starting topical steroids.

If there is a corneal ulcer, topical NSAIDS can be used and an oral steroid until the ulcer is healed.

Pressure <8mmHg: start pred acetate 3-4x /day

Pressure 8-18mmHg : pred acetate and add in dorzolamide BID

Pressure >18mmHg pred acetate and add in dorzolamide TID

Basic plan:

1. 1% pred acetate q 4-6 hrs +/- dorzolamide
2. Tropicamide bid to TID or atropine as needed to keep pupil dilated
3. Flurbiprofen or diclofenac BID
4. +/- oral NSAID if safe (or oral prednisone if confident won't cause worsening of occult underlying disease)
5. Doxycycline 5mg/kg bid x 14 days dogs OR Azithromycin or Clindamycin in cats

Once clinical signs improve (normal IOP, no redness, injection, no more cellularity) you can start by tapering the oral NSAID or steroid. Then taper the topical steroid.

Tapering off all meds usually takes weeks to months.

Etiology

Infectious: bacterial, viral, fungal, parasitic

Idiopathic/immune-mediated: up to 60%-70% of dogs and cats

Other: Coagulopathies, Diabetes mellitus, Hyperlipidemia, Hypertension, Lens-induced uveitis (eg, cataracts), Primary neoplastic disease (eg, ocular melanoma), Secondary neoplastic disease, Trauma, Ulcerative keratitis

Diagnostics

Tonometry: **IOP under 10**, unless there is concurrent glaucoma which can cause a normal IOP even with obvious signs of uveitis (flare, hyphema, etc.)

Other eye tests should include fluorescein staining to look for corneal ulceration.

Minimum database: CBC, Chemistry, Urinalysis, Bp

Infectious disease testing cats: FeLV/FIV, Cryptococcus antigen, Bartonella Ab by IFA, FCoV antibody, Toxoplasma IgG and IgM

Infectious disease testing dogs: tick titers, fungal, protozoal, bacterial and viral diseases as indicated by area, travel history etc.

Consider imaging as well: chest x-rays, AUS

Summary/key points

Feline uveitis is the most common cause of glaucoma in cats

Most uveitis cases in dogs and cats are idiopathic (no underlying cause determined)

Of the cats with a diagnosed cause, infectious disease is most common; dogs rarely have infectious uveitis

Most causes of uveitis in dogs is idiopathic (up to 60%); other non-infectious causes are: lens induced, neoplastic, traumatic, corneal ulcers, pigmentary uveitis of golden retrievers, uveodermatologic syndrome.

Up to 70% of cats with uveitis have an underlying systemic disease or associated systemic disease: FeLV, FIV, FIP, Bartonella, Toxo, Fungal disease

If there is Hyphema or Hypopyon = uveitis

Glaucoma

Pathophys

Glaucoma is elevation in intraocular pressure (IOP) that is incompatible with normal ocular function. IOP is the result of a balance between production and drainage of aqueous humor.

- **Primary Open Angle Glaucoma:** Gradual/progressive increase in IOP over 2-4 years. Early/moderate stages have IOPs between 25-40mmHg and variable clinical signs. Advanced stages IOP's 50-60's (buphthalmia, lens luxation/sublux, optic disc degeneration, vision loss)
- **Primary Closed Angle Glaucoma (Most common primary cause in dogs):** abrupt increase in IOP. IOP's increase to 30mmHg or higher. Vision often impaired or lost by the time they present to the vet. The fellow eye typically follows about 8 months later.
- **Secondary Glaucoma:** Altered aqueous humor dynamics due to other causes such as: uveitis, neoplasia, trauma, lens luxation etc. Most common cause of glaucoma in cats!

Clinical signs

Episcleral injection, painful eye, corneal edema
Dilated pupil, may be sluggish to respond to light
Lens luxation may be present
Buphthalmos from long standing glaucoma
Cats: Rarely show ocular redness!! Look for epiphora, varying blepharospasm, mydriasis, reduced PLR, corneal edema

Treatment

Dogs: Goal of emergency therapy is to **reduce IOP to <20mmHg within a few hours**

Acute Glaucoma treatment (no anterior lens luxation present)

- 1-2 drops **prostaglandin analogue** (ex Xalatan) followed by **Dorzolamide** 5 minutes later.
- Repeat Xalatan in 30 minutes if IOP >30mmHg
- **If IOP still elevated >30mmHg in an hour** (90 minutes after starting) **add in mannitol**

Mannitol: Draw blood sample prior to mannitol to check lytes. Prepared IV mannitol: 2gm/kg or 1 gm/kg with heart disease. Give slowly IV over 20 minutes through a filter set. Should decrease IOP within an hour. Withhold food/water for 4 hours.

Continue Dorzolamide BID to TID and start Pred Acetate BID to counteract inevitable uveal inflammation

- Dorzolamide (Trusopt): carbonic anhydrase inhibitor
- Dorzolamide:Timolol (Cosopt): in combo with timolol (beta blocker)
- Timolol: caution with heart block, bradycardia, asthma, chronic bronchitis

Some Ophthalmologists prefer the use of an emergency Acqueocentesis: drawing off about 0.1ml of aqueous humor by inserting a narrow gauge needle in a shallow angle into the anterior chamber at the limbal margin

Cats: Typically not acute, due most often to secondary cause.

Cats don't respond to prostaglandin analogues! Use topical timolol, dorzolamide ; treat underlying cause

Etiology

- **Primary Glaucoma:** Inherited abnormal metabolism of the outflow system, or physical pupillary blockage and changes to the drainage areas
- **Secondary Glaucoma:** Increased IOP associated with previous or current eye disease that obstructs outflow pathways. Typically unilateral and not inherited. Examples: uveitis, lens luxation, intraocular neoplasia
- **Congenital Glaucoma:** increased IOP associated with inherited anomalies. Develops soon after birth. Very rare.

Diagnostics

- Tonometry: **IOP is >30mmHg (dogs) >25mmHg (cats) or a greater than 10mmHg pressure difference between the two eyes.**
- Gonioscopy to evaluate the iridocorneal angle to determine if the angle is open or narrowed.
- Ophthalmoscopy to evaluate the retina
- PLR /menace to assess for vision loss
- Lens evaluation to screen for lens luxation
- Cats:** screen for secondary causes: hypertension, infectious dz neoplasia, pre-existing uveitis, trauma

Summary/key points

Most canine glaucomas are closed angle (not slowly progressive glaucoma as seen in people). Therefore annual tonometry is generally not useful.

- Once glaucoma has developed in one eye it is useful to prophylactically treat and monitor the fellow eye.
- **Prophylactic treatment** of the unaffected eye can **delay glaucoma** from a median of 8 months to a median of **31 months** with treatment.
- In cats most glaucoma is due to uveitis or neoplasia, but open angle glaucoma occurs in the Siamese breed.
- Not all cats with glaucoma present with a red eye or any signs of ocular discomfort!!
- Prostaglandin analogues contraindicated with anterior lens luxation because they can worsen drainage
- **Medical therapy typically ineffective within a year.**
- Surgical intervention can prolong vision but has a poor long term success rate.

Cats: 95-95% are secondary to uveitis, neoplasia, sometimes trauma, intraocular hemorrhage, etc.

Antibiotics for Corneal Ulcers in Dogs and Cats

www.etsy.com/shop/VetHelpfulNotes

Causes of Corneal Ulcers:

- Trauma
- Trichiasis
- Distichiasis
- Ectopic cilia
- Foreign bodies
- Cranial nerve dysfunction
- Ectropion
- KCS
- Eyelid tumors
- Blepharitis
- Herpes virus
- Lagophthalmus



Superficial uncomplicated Corneal Ulcer

Should heal within 7 days, usually 3-5 days; recc treat q 6 hours

PEARL: The most critical treatment for a corneal ulcer is removal of the primary cause. Therefore, if an ulcer has not healed in 7 days, change your diagnosis, not your antibiotic

Melting Ulcer

Widen antibiotic spectrum

Example: on BNP add Moxiflox or Oflox

Example: on Erythromycin add Oflox or Tobra

Add Serum or Vetrix EyeQ amniotic

Increase frequency to q 2 hours administration

Cats

If an ulcer has not healed in 7 days, categorize it as:



About the same

Deep/green/melting

loose edges

Look harder for cause (ex FHV-1)

C&S
increase antibiotic therapy

Debride and add antiviral

Dogs

If an ulcer has not healed in 7 days, categorize it as:



About the same

Deep/green/melting

Loose edges

Look harder for a cause

C&S
increase antibiotic therapy

Debride and/or grid Keratotomy

Ophthalmic Antibiotics

Bacitracin- peptide antibiotic: gram positive bacteria (might cause feline anaphylaxis)

Chloramphenicol- (no particular class): Broad spectrum, Not good for pseudomonas (resistant)

Erythromycin- Macrolide: Gm pos Staph and Strep, mycoplasma, chlamydophila. Not good for pseudomonas

Gramicidin- Peptide antibiotic: Gram pos (not grm pos bacilli like corynebacterium) and some gram neg organisms

Neomycin- aminoglycoside: Broad spectrum, Not good for: pseudomonas and mycoplasma (resistant) may cause feline anaphylaxis

Moxiflox/Ofloxacin- fluroquinolone: Broad spectrum: Staph, Strep, Pseudomonas, Chlamydophila, Haemophilus, variable efficacy against mycobacteria and anaerobes, Not good for some strains of staph and strep (resistant). Better against Gm neg than Gram Pos

Polymyxin B- polypeptide antibiotic: Gram neg bacteria, mostly used in combination with other abx (Caution feline anaphylaxis)

Terramycin- tetracycline: Mycoplasma, chlamydia, some gram negatives (proteus, pseudomonas)

Tobramycin-aminoglycoside: Gram neg bacteria (pseudomonas) and Gram pos staphylococci (limited); not good for Strep

The most common bacterial isolates from cats with bacterial keratitis:



~66% Gram positive: Mostly staphylococcus (cocci)
~33% Gram-negative: Mostly pseudomonas (rods)

Good first line ophthalmic antibiotic choice:

Cats: **Erythromycin** or **Tobramycin** (caution with Terramycin due to added Polymyxin b)

The most common bacterial isolates from dogs with bacterial keratitis:



~75% Gram positive Staphylococcus> Streptococcus> Corynebacterium (rod)

~25% Gram negative: Mostly Pseudomonas, E. coli (rods)

Good first line ophthalmic antibiotic choice:

Dogs: **Triple Antibiotic** like: Bacitracin, Neomycin, Polymyxin (BNP), ophthalmic or Neomycin, Polymyxin, Gramicidin



Medication Rules

Vet Helpful Notes
Canine Feline

Drugs You Can't Crush or Break

Extended release tabs are meant to dissolve slowly

Examples:

- Theophylline ER
- Keppra (Levetiracetam ER)
- Proin ER

Breaking enteric coated tabs: this can lead to a bitter taste or degradation in the stomach. *The coating is meant to protect the stomach lining*



Some examples of enteric coated meds:

- Budesonide
- Erythromycin
- Omeprazole capsules (granules)
- Potassium Citrate tabs

Drugs irritating to mucous membranes:

- Piroxicam
- Bisphosphonates (alendronate)

Handle with Gloves!!

Cyclophosphamide: carcinogen and birth defects

Mycophenylate: carcinogen and birth defects

Finasteride: Can cause birth defects

Misoprostol: can cause spontaneous abortions

Chloramphenicol: can cause aplastic anemia in some people



ER : extended release
= DR: delayed release

Ya Know....

Any drug in a blister pack, is probably moisture sensitive, and should not be mixed with water (Papich 2005), nor should lipophilic drugs, such as itraconazole or diazepam. (Check the product insert to see whether a drug is highly lipophilic)

If a drug is scored, the drug in each half should be equal amounts

Special Ways to Give Drugs

Doxycycline can cause esophageal strictures if if they are not swallowed into the stomach. Always Flush after with water



Sucralfate tablets should be mixed with water and given as a slurry.



Phosphate binders should be given with food to help bind the phosphate in the food.



Pancreatic enzymes should be mixed into food 15 minutes before serving. This helps to "pre-digest" the food.

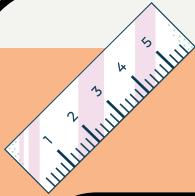


Human Drugs Harmful to Pets



Estrogen gels	: bone marrow toxicity
Voltaren cream	: renal failure cats
Rogaine	: cardiovascular tox, vomiting
Aspirin	: renal/GI side effects
Pepto bismol	: renal/GI side effects





Medication Rules

Transdermal (TD) Medications:

Transdermal drugs that are effective in humans tend to have total daily dosages *less than 50 mg* per day vs transdermals for pets which should *not be over 25 mg*.

In humans, the stratum corneum barrier limits transdermal delivery of most drugs in permeation enhancers to about 1 mg per cm² of skin.



Veterinary transdermal formulations of drugs that require higher daily dosages are less likely to be fully absorbed through the relatively small surface area of the pinna.

FACT:



Typical dosages for human transdermals are very low
Examples:

**clonidine for hypertension
(Catapres-TTS 0.1-0.3 mg per day)**

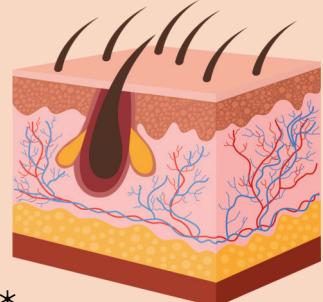
Selegiline for depression (Emsam 6-12 mg delivered over 24 hours)

**Nicotine for smoking cessation
(NicoDermCQ ≤ 21 mg per day).**



Transdermal options

- Cerenia
- Fentanyl
- Mirataz
- Methimazole
- Amlodipine
- Nitroglycerin
- Amitriptyline
- **Dexamethasone ***



*steroids applied TD to the pinna may damage the ear cartilage over time

note: the only **studied** veterinary compounded TD is methimazole.

All others should be used cautiously as variability may be marked

TD Properties should be:

- Drug should be <800 Daltons to pass thru the skin
- Drug should be lipophilic and hydrophilic
- Drug should have a wide therapeutic index
- A condition where the response is not needed immediately

Drugs that won't work

- Medications requiring dosages over 25mg
- Pro-drugs needed to be split by Gut microbes (example sulfasalazine)



Medication Rules

Vet Helpful Notes
Canine ♥ Feline

PEARLS

Drugs with transmucosal action:

Diazepam, midazolam, ketamine, buprenorphine, Dexmedetomidine



PROBIOTIC FACTS

- Never get probiotics scooped from a tub. Only individually sealed! (Blister packs or individual pouches.) Live microorganisms are sensitive to moisture.

** Caveat: Probiotics in food are heat and moisture resistant bacterial spores such as bacillus

- A human pediatric dose is 5 billion cfu's per dose. (like in Proviable)
- Visbiome has 112 billion cfu's/dose
- Fortiflora uses less bacteria but is considered an immune stimulant

Fortiflora	Calming Care	Lactoquil	Proviable DC
100 million	1 billion	1.5 billion	5 billion
1 strain	1 strain	9 strains	7 strains

Good to Know

Some Omeprazole DR capsules can be opened and split for use. They contain 1.1mg enteric coated pellets that you can count out separately and use for smaller patients.



Clavamox recommends against splitting. If you do, wrap the foil tightly around the remaining 1/2 tablet.

The amoxicillin and clavulanic acid may not be evenly dispersed in the tablet

Diets supplemented with zinc, like aluminum, can chelate fluoroquinolones.



Random Facts

Give with food

Itraconazole
NSAIDS
Steroids
Antacids
Insulin



Give on an empty stomach

Sucralfate
Cyclosporine
Levothyroxine

Don't give antacids with:

Sucralfate
Aluminum hydroxide

Don't give together

NSAIDS with steroids

Don't give fluoroquinolones with:

Sucralfate, aluminum hydroxide, zinc supplemented diets, milk products



Sucralfate and **aluminum hydroxide** contain aluminum that **forms complexes with many other drugs** in the GI tract, which markedly decreases drug absorption

Doxycycline and fluoroquinolones are poorly absorbed in the presence of calcium, as well as iron, zinc, or magnesium found in multivitamins. Do not give with milk products.

Cyclosporine needs to be microemulsion and name brand is best: Atopica or Neoral. It should be given 1 hour before or 2 hrs after a meal

The calcium in dairy products may also decrease fluoroquinolone bioavailability. This has been observed in nursing kittens





Medication Rules

Drug Beware



Toxicity

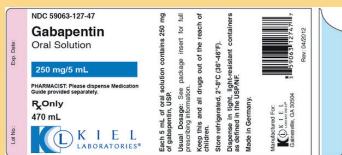
Some manufactured liquid rx's have xylitol!

Ex: Gabapentin suspension

Some supplements too:

Ex: Metamucil gummies

BIRCH SUGAR = XYLITOL



Avoid sugar-free products



Ketoconazole !

Ketoconazole and itraconazole are best absorbed at acidic pH; therefore, **do not combine** these drugs with: **antacids** Interestingly, increased gastric pH does not affect the absorption of fluconazole

Ketoconazole inhibits a cytochrome P450 enzyme (CYP3A), which helps to metabolize drugs. Ketoconazole has **high potential for drug-drug interactions**.

Ketoconazole is also an inhibitor of p-glycoprotein, an important drug efflux transporter in the gut and biliary tree. Ketoconazole can therefore **decrease the bioavailability and/or clearance of many drugs**:

Ketoconazole can allow lower doses of cyclosporine and save \$! (About 50% lower)

Recommended dosages: cyclosporine: 2.5-5 mg/kg/day; ketoconazole: 10 mg/kg/day. Monitor ALT and clinical response. Whole blood cyclosporine can be measured at steady state (by one week).

Digoxin: **ketoconazole** can lead to **digoxin toxicity**.

Amitriptyline, midazolam: ketoconazole could increase sedation.

Phenobarbital

Phenobarbital is a major P450 enzyme inducer in humans and dogs. **Phenobarbital speeds the metabolism** of many drugs in dogs, which decreases their efficacy. Examples:

Glucocorticoids, ketoconazole, clomipramine, chloramphenicol, lidocaine, etodolac, theophylline, digoxin, propranolol, and many others...



However, phenobarbital causes minimal cytochrome P450 enzyme induction in the cat, and therefore these P450-mediated drug interactions are unlikely in the cat.



Drug Interactions

Gi bleed:

Do not give an NSAID and a steroid together, wait at least 3 days before changing from one NSAID to another or changing from an NSAID to a steroid and vice versa

Serotonin Syndrome:

Can happen if you give more than one drug that increases serotonin or an over-dose:

Symptoms: Agitation, restlessness, vomiting, tremors, hypertension from too much serotonin

SSRI + MAOI most risky

SSRI

Fluoxetine
Citalopram
Paroxetine

TCA

Amitriptyline

SNRI

Venlafaxine

MAOI

Selegiline

Antidote:
cyproheptadine

OTHERS

Buspirone
fentanyl
metoclopramide
tramadol
methadone
ondansetron
trazodone

Medication Rules



Caution MDR-1

Genetic condition that causes drug and toxin build up and even cross over the blood brain barrier

Breeds most at Risk

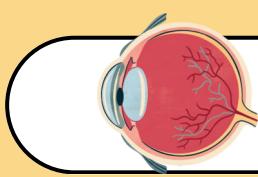
Australian Shepherd	50% risk
Collie	70% risk
Long Haired Whippet	50% risk



Caution drugs

Acepromazine
Apomorphine
Butorphanol
Cyclosporine
Imodium

Maropitant
Ondansetron
Grapiprant
Ivermectin



Eye Meds



Always separate eye drops by 5 minutes

When to choose an ointment vs drop:

Ointments provide better contact time. So, they are great for a condition like pannus.

When treating a complicated ulcer with multiple medications, drops are preferred as an ointment might interfere with penetration

Eye drops that could cause feline anaphylaxis

- Terramycin (polymyxin B)
- Neopolygram/Neopolybac (Bacitracin/Neomycin)

Steroid eye drops comparison

Dexamethasone 0.1% better for surface inflammation
Pred acetate 1 % best for uveitis



Good artificial tear/lubricating eye drops:

Optixcare + hyaluron

Prostaglandin eye drops (like Latanoprost):

Some sources suggest they don't work well in cats
However, they are often contra-indicated in feline glaucoma as uveitis is often a component of their disease
A potent miotic used in an eye with uveitis can increase pupillary block and potentiate glaucoma

Medications delivered as an eye drop:

Clevor eye drop to induce vomiting
Desmopressin to treat diabetes insipidus in dogs

Eye drops are given by mouth to treat disease:

Pilocarpine ophthalmic given orally to treat xeromycteria

Eye drops used as ear drops:

Tobramycin ophthalmic can be useful to treat rod infections in the ears where there is concern for a ruptured ear drum (TM)

Melting Ulcer

- Either serum or plasma can be used on melting corneal ulcers.
- Serum or plasma should be refrigerated and used for 3 days max OR sterile filtered through a 0.22 micron syringe filter and used for 5 days max
- If the corneal ulcer is melting then serum from a different animal or species should be used "heterologous serum". Melting usually is caused by the patient's own immune reaction so no fuel on that fire "autologous serum" Serum will not help a deep non-melting ulcer or sequestrum much
- Don't collect serum from diabetic dogs (high sugar content might "feed bacteria". Also, use serum from a dog with "healthy" eyes.

KCS Drugs

It's generally recommended to start treating KCS with Cyclosporine. You can start with optimune ointment (0.2% cyclosporine). If that is ineffective you can move up to 2% cyclosporine compounded drops.

Cyclosporine 2% or Tacrolimus 1% are often chosen for more difficult to manage cases (STT <10mm)

Cyclosporine compounded ophthalmic

The preferred oil by many ophthalmologists on VIN is MCT (medium chain triglyceride) oil. Olive and Corn oil can be irritating sometimes

Oil based drops, may leave greasy residue around pet's eyes which can bother owners.

You can also choose aqueous drops (water based) but this must be shaken well to keep it in suspension.



Medication Rules



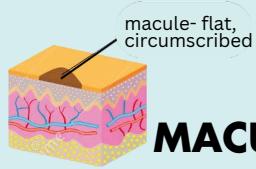
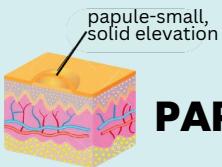
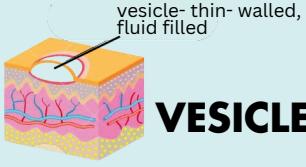
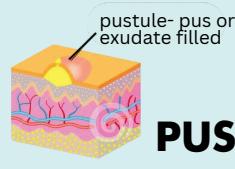
Common Drug Contraindications

Condition	Drug to Avoid	Possible Complication
Diabetes	Steroids	Reduced sensitivity to insulin
Asthma	beta blocker	Bronchospasm
Heart disease	Steroids	Congestive heart failure
Corneal Ulcer	topical steroid	Decreased corneal healing
Kidney disease	NSAID	Decreased renal blood flow
MDR-1	see chart previous page	Increased drug side effects such as sedation and seizures
Diarrhea/vomiting	NSAIDS	Increased risk for GI ulceration
Glaucoma caused by uveitis/ant lens lux	Latanoprost	Increased risk for pupillary block and worsening glaucoma, lens entrapment
Epilepsy	Penicillin, Chloramphenicol, Fluoroquinolones, some HW prev, Metoclopramide some SSRI's	Reduced seizure threshold
Mast Cell Tumor	Morphine	Histamine release

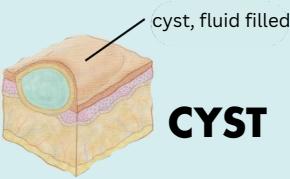
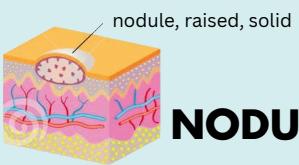


Lesions of the skin

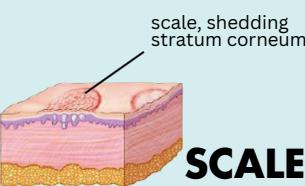
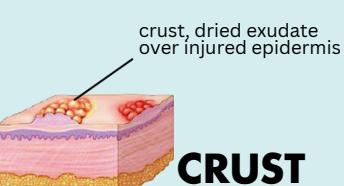
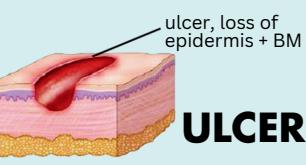
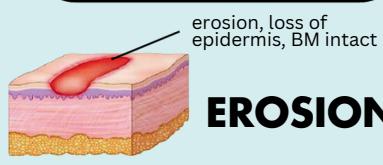
Definitions

**MACULE****PAPULE****VESICLE****PUSTULE**

A circumscribed, flat lesion with color change up to 1 cm in size that is not palpable

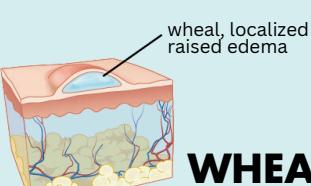
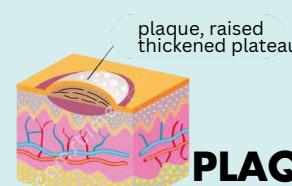
**CYST****NODULE**

An abnormal, usually noncancerous growth filled with liquid or a semisolid substance

**SCALE****CRUST****ULCER****EROSION**

Segmental or more extensive loss of the epidermis, including the basement membrane

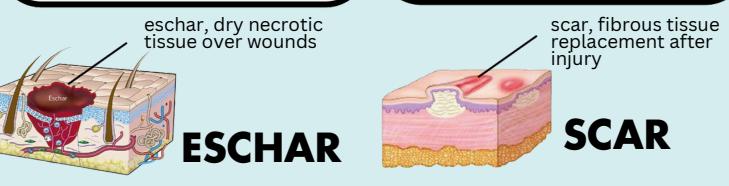
Partial loss of the epithelium, with the basement membrane left intact

**WHEAL****PLAQUE**

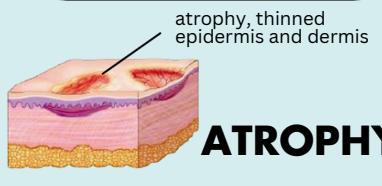
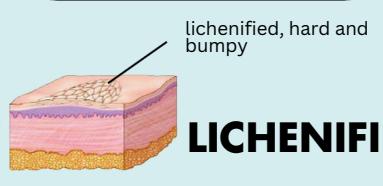
Increased shedding of stratum corneum as a result of abnormal exfoliation

**ESCHAR**

Necrotic tissue that can develop on severe wounds. It is typically dry, black, firm

**SCAR**

An area of fibrous tissue that replaces normal skin after an injury

**ATROPHY****LICHENIFIED**

Degeneration and thinning of the epidermis and dermis. Wrinkled and shiny skin.

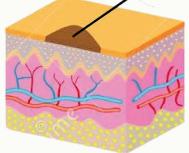
Thickened, hardened area of skin. It may have a bumpy texture and be darker

Lesions of the skin



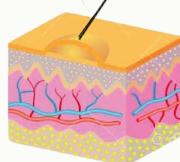
MACULE

macule- flat, circumscribed



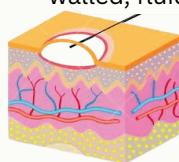
PAPULE

papule- small, solid elevation



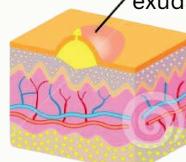
VESICLE

vesicle- thin-walled, fluid filled



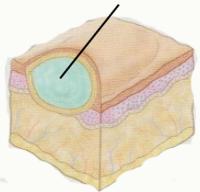
PUSTULE

pus-tule- pus or exudate filled



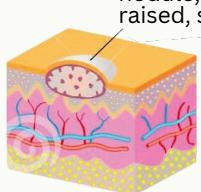
CYST

cyst, fluid filled



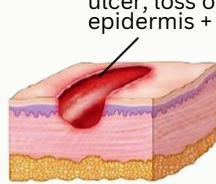
NODULE

nodule, raised, solid



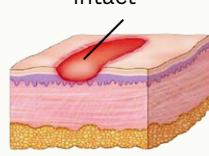
ULCER

ulcer, loss of epidermis + BM



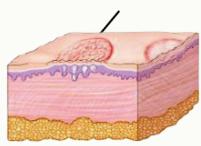
EROSION

erosion, loss of epidermis, BM intact



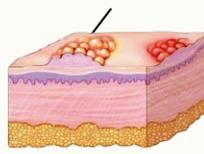
SCALE

scale, shedding stratum corneum



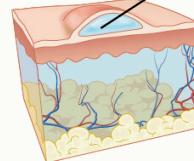
CRUST

crust, dried exudate over injured epidermis



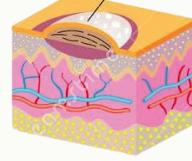
WHEAL

wheel, localized raised edema



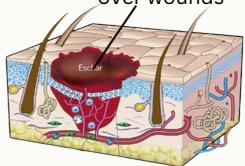
PLAQUE

plaque, raised thickened plateau



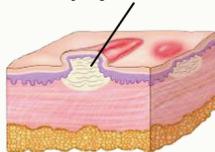
ESCHAR

eschar, dry necrotic tissue over wounds



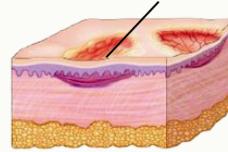
SCAR

scar, fibrous tissue replacement after injury



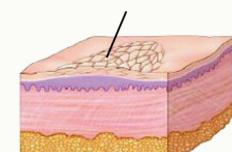
ATROPHY

atrophy, thinned epidermis and dermis



LICHENIFIED

lichenified, hardened, bumpy skin texture



Steal my strategy

Quickly and Confidently Diagnose
Lumps and Bumps Presented to you
in Veterinary Practice



NICOLE COHEN, DVM DABVP

The OLP Method

The OLP method stands for :

Origin, Location, and Pet Specifics

We're not trained clinical pathologists, yet we are tasked by our clients every day to help guide them when faced with a new lump or bump. It doesn't make sense to take every patient to surgery to remove every lump. So what do we do?

We have to hone our skills at evaluating each patient. In each exam, we should determine what the lump is most likely to be and provide a professional recommendation regarding options and next steps based on its expected behavior.

Using the **OLP** method will help enormously to whittle down the possibilities and give you confidence in your recommendation.

Let's get started!

We start with **ORIGIN**

The first task is to decide if the origin of the mass is **neoplastic** or **inflammatory**. This starts with a **fine needle aspirate** (FNA).

Inflammatory nodules exfoliate predominantly red blood cells and white blood cells like neutrophils, eosinophils, and macrophages.

If you suspect the mass is inflammatory, then it would be expected to regress. If you suspect the mass is infected (you see intracellular bacteria inside of neutrophils) then a course of antibiotics may help to treat the bump. If you see a lot of eosinophils, an antihistamine trial might help as well.

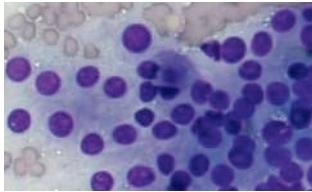
You can discuss a recheck exam in 1-3 weeks if the mass persists to re-aspirate once the inflammation has subsided or to surgically biopsy.

If you don't suspect an inflammatory nodule on your FNA, and you see a population of cells on your slide, then your task is to do your best to identify the suspected neoplasia. Don't forget, neoplasia can be benign OR malignant.

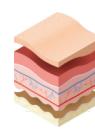
Almost all neoplastic masses can be broadly characterized into 3 groups:

EPITHELIAL, MESENCHYMAL AND ROUND CELL TUMORS.

Epithelial Tumors



Come from skin/hair/gland components



Cells are shed in clusters and look as if they share a pool of cytoplasm
Majority are benign, but the most well known aggressive epithelial tumors are:

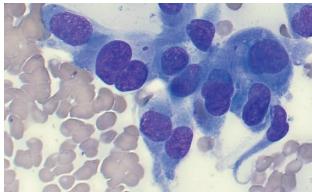
Squamous cell carcinoma (such as of the mouth in cats)

Fibrosarcoma (such as vaccine associated fibrosarcomas of cats)

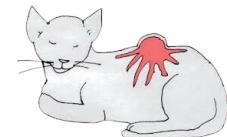
Anal Sac Adenocarcinoma of dogs

Mammary adenocarcinoma of dogs and cats

Mesenchymal Tumors



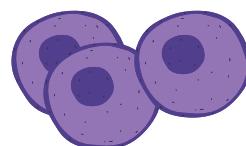
Come from connective tissue: fat, bone, muscle, cartilage, fibrous tissue



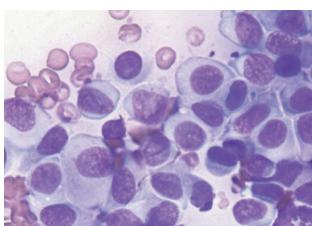
Cells are spindle shaped with wispy edges and often have ovoid nuclei

Malignant mesenchymal tumors end in "sarcoma". The majority are low to intermediate grade with a low chance of distant metastasis.

They can be difficult to completely remove, however. Many have long tumor tentacles and if not fully removed may grow back and cause local tissue destruction.



Round Cell Tumors



Named for their round cell type. They have distinct borders.

There are 6 kinds if you include melanoma:

Mast Cell: Malignant in dogs but most are cured with surgery. High grade tumors are more likely to be located in the oral/perioral, prepuce, scrotum and perineal regions. Most are benign in cat but should be removed.

Plasma Cell: Generally don't metastasize. Should be surgically removed. Rarely associated with multiple myeloma. Good to screen for elevated globulins on bloodwork if there are multiple plasma cell tumors.

Lymphoma: "lumps" are often enlarged lymph nodes. Malignant and should be treated with chemotherapy

Histiocytoma: Benign tumors which regress within 2-3 months

Transmissible Venereal Tumor: Rare to metastasize, but should be treated with chemotherapy/surgery. Can spread via direct contact.

Melanoma: Oral melanoma locally invasive and >60% are metastatic

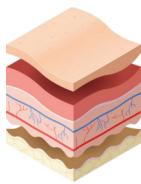
Nail bed melanoma is locally invasive, lower rate of metastasis 30-60%

Cutaneous melanomas are generally benign, but those at mucocutaneous junction can be more aggressive

1. Epithelial tumors come from:

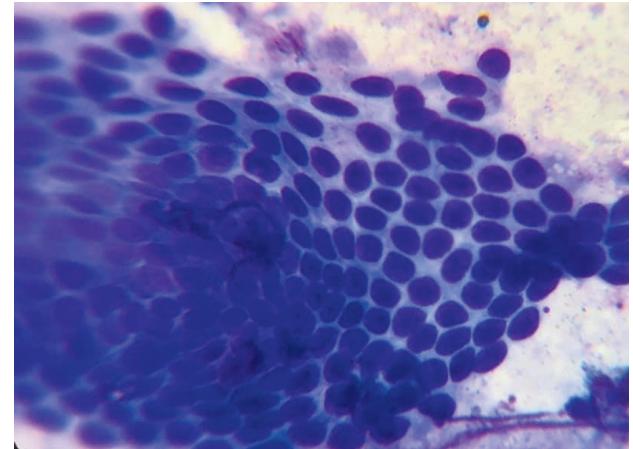
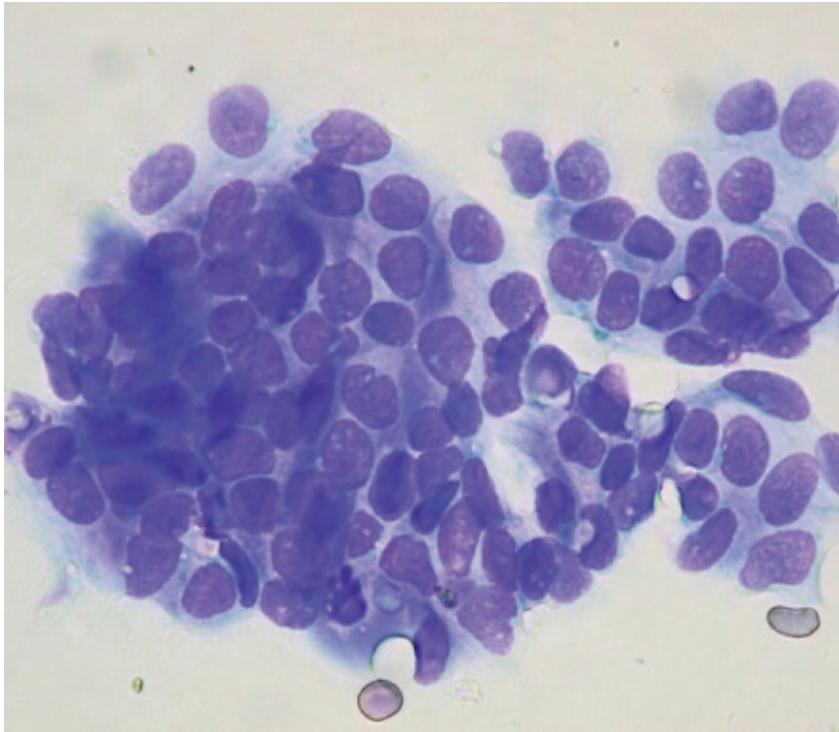
SKIN

HAIR COMPONENTS
AND GLANDS.



CYTOLOGY CHARACTERISTICS of Epithelial tumors

On cytology, epithelial tumors usually shed cells in clusters. This is because epithelial cells have tight junctions and tend to stay attached to each other.



Epithelial Tumor Cytology Characteristics

- Cells in clusters
- Distinct cell borders that are round to polygonal
- Round nuclei
- When they group, they appear as if they share a common cytoplasm pool

Pages 4 and 5 provide examples of some common epithelial tumors you will see in practice and their behavior.

Common Epithelial Tumors



Sebaceous adenoma

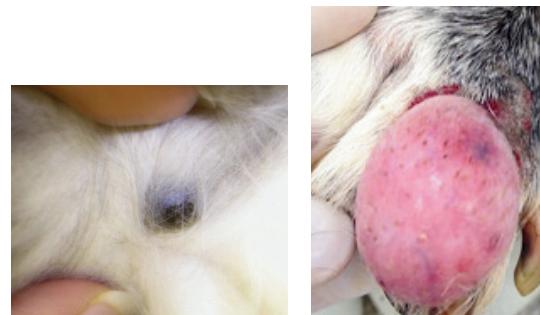
Cauliflower like lesions which are benign proliferations of sebaceous tissue. They often secrete oily waxy material.



Trichoblastoma/trichoepithelioma

Benign proliferation of hair follicle components. Although they are locally invasive, they are cured with surgical removal. These can sometimes be large! **

Trichoblastomas (formerly basal cell tumors) are the most common pigmented skin tumor of cats**



Squamous cell carcinoma- Malignant

In white cats with excess sun exposure, SCC most commonly affects the skin on the ear tips, the nose, eyelids and lips. It can look like a non healing scab and later progress to ulceration and necrosis. Surgery provides the best long term treatment.



Papilloma

A papilloma is a benign lesion induced by the canine papilloma virus. It causes a cauliflower like growth most often in the mouth. It typically affects young dogs with an immature immune system. These can regress on their own.



Meibomian gland adenoma

The vast majority of eyelid tumors in dogs are benign sebaceous tumors of the eyelid margin. Eyelid tumors are not common in cats.



Common Epithelial Tumors



Anal sac adenocarcinoma- Malignant

A mass near the anus in the 4 o'clock or 8 o'clock position. These tumors can cause hypercalcemia and are malignant.

These tumors spread first to local iliac lymph nodes and surgical removal + chemotherapy is the recommended treatment.



Mammary adenocarcinoma- Malignant

Mammary tumors in cats are almost always malignant (85%) and require extensive surgical removal (typically a full chain mastectomy) followed by chemotherapy. In contrast, only 50% of mammary tumors in dogs are malignant.

Mammary tumors cannot be diagnosed as benign or malignant on cytology. They require histopathology.



Apocrine cystadenoma (cystomatosis)

These are benign blueish tinted cysts of the ceruminous glands. They most commonly are found lining the ears. But they can develop in other areas as well.



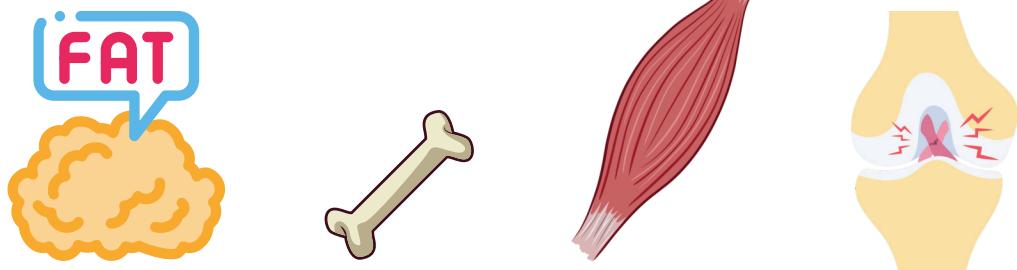
Perianal adenoma

A tumor of the perianal sebaceous glands of dogs. They occur in dogs that are not neutered because testosterone influences the growth of these masses. Treatment is local resection and neutering. Cats don't have these perianal sebaceous glands.



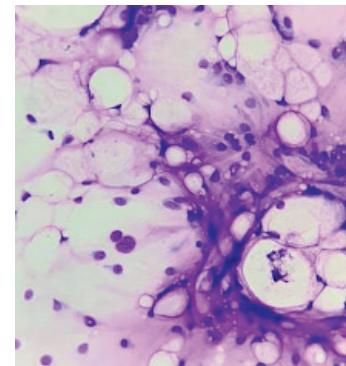
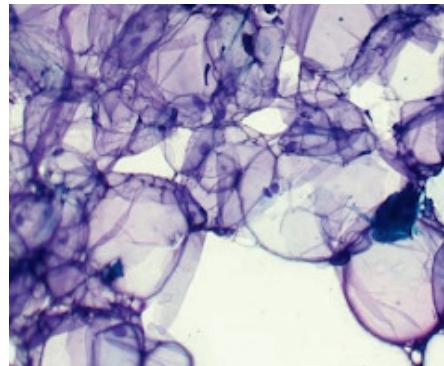
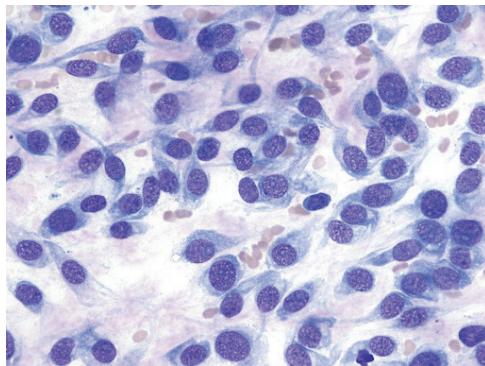
2. Mesenchymal tumors arise from connective tissue

FAT
BONE
MUSCLE
CARTILAGE
FIBROUS TISSUE



Malignant mesenchymal tumors are called sarcomas.
Benign mesenchymal tumors end in -oma.

Healthy mesenchymal tissue does not exfoliate well. However, malignant mesenchymal tissue exfoliates readily.

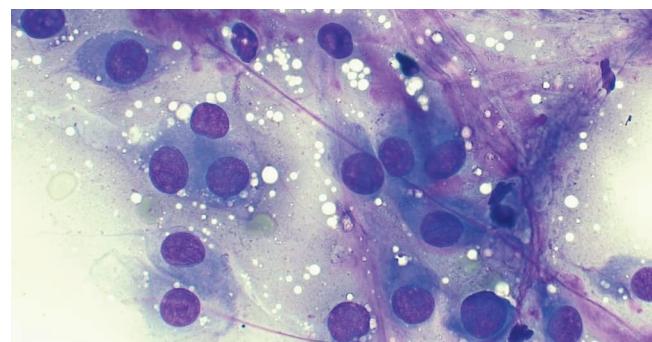
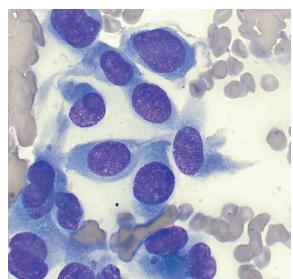


Mesenchymal cells tend to be individual, spindleoid or wispy cells with an indistinct border. They often have ovoid nuclei.

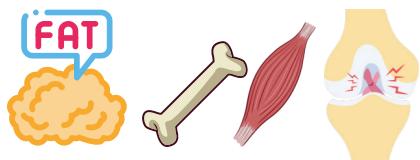
By far, the most commonly diagnosed mesenchymal tumor in practice is the lipoma. A benign lipoma is above to the left. The far right is a liposarcoma. This has many nucleated adipocytes.

Mesenchymal tumor cytology characteristics

- Spindleoid or wispy cells
- Indistinct borders
- Ovoid nuclei are common



Common Mesenchymal Tumors



Lipoma/Liposarcoma

Lipomas are common fatty tumors seen most commonly in dogs and sometimes in cats. Malignant liposarcomas are very rare but do respond well to complete excision.



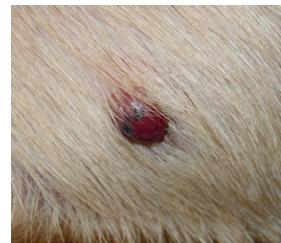
Fibrosarcoma/fibroma

Vaccine associated fibrosarcoma are aggressive mesenchymal tumors induced by vaccination with leukemia or rabies vaccines. These were thought to be due to reactivity to the adjuvant in older vaccines, but can happen even with purevx vaccines. Even with radiation following aggressive resection, 30-40% recur at the site.



Hemangiosarcoma/hemangioma

Hemangiomas are benign tumors of the blood vessels and tend to occur in light colored dogs on their ventrum from sunbathing. Hemangiosarcomas are very aggressive tumors that can affect many tissues like the spleen, liver, heart and skin.



Soft tissue sarcoma (STS)

Include fibrosarcoma, myxosarcoma, hemangiopericytoma and peripheral nerve sheath tumors. They are locally invasive but most grades (I and II) are rare to metastasize. Complete excision can be difficult. Grade III tumors are most likely to metastasize.



-Fibrosarcoma/fibroma

This is a type of STS. A tumor free surgical margin of 3mm predicts a low rate of local recurrence and also decreased likelihood of this being a grade III STS. Even with less than 3mm surgical margins, grade I and II STS have low rates of local recurrence at 7% and 35% respectively.



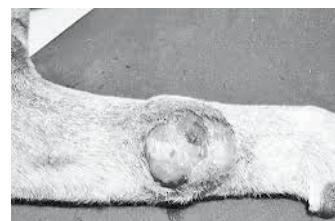
-Peripheral nerve sheath tumor (PNST)

These tumors arise from the tissues surrounding nerves. Found mostly in dogs, the most common location is the brachial plexus (80%). This leads to front limb lameness and atrophy. Another common location is the trigeminal nerve leading to marked masticatory muscle atrophy.

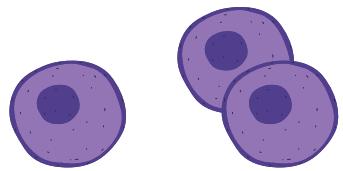


-Hemangiopericytoma

Most commonly develop on the distal limb in dogs. Distant metastasis is rare, but the mass can be difficult to fully remove depending on its size and location. Radiation in addition to surgery may be recommended.



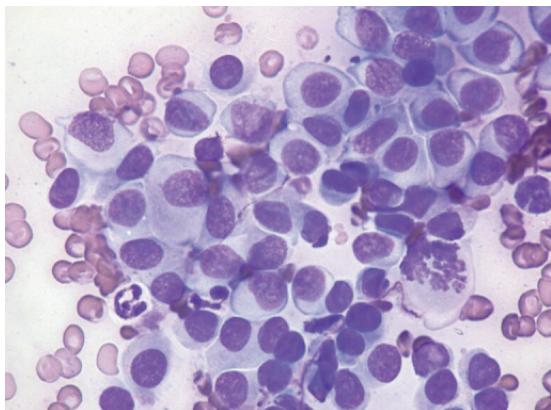
3. Round cell tumors + melanomas



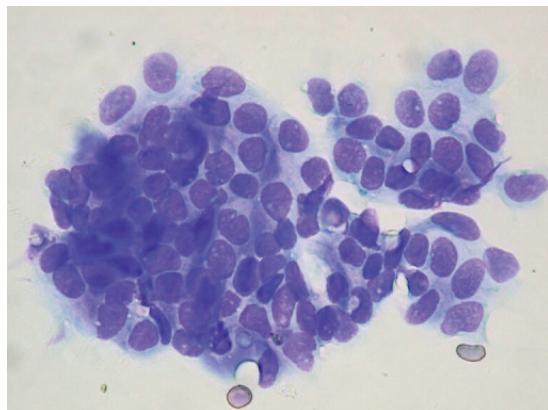
Round cell tumors have features that include:

- A round cell shape
- An arrangement of cells individually or in sheets

Melanomas are grouped into this category because they are often round cells, but sometimes can vary in appearance

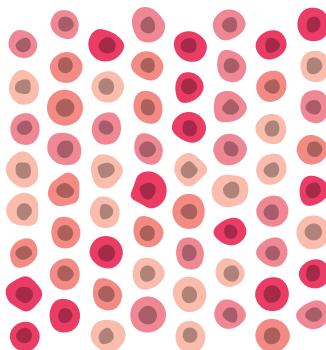


Plasmacytoma with distinct round cell edges



Epithelial tumor. Cells almost share the same pool of cytoplasm

Round cells have distinct borders separating their cytoplasm. The epithelial tumor on the right has numerous almost bare nuclei that seem to float in a shared cytoplasm.



Round Cell tumors

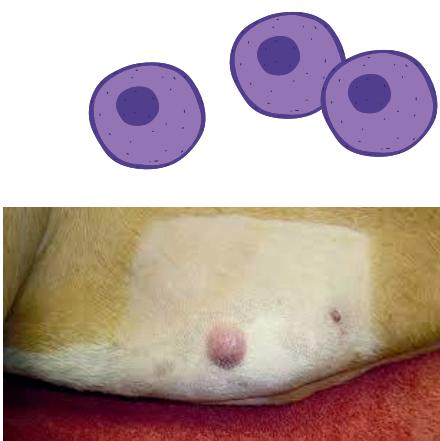
Mast cell tumor-Malignant

Cutaneous tumors are graded low and high grade which predicts their biologic behavior. Low grade tumors just require complete excision. High grade tumors are more likely to metastasize so additional chemotherapy is needed for improved outcomes. Histopath is required for grading, not cytology alone.

Cutaneous MCTs on the muzzle, oral/perioral, prepuce and scrotum are suggested to have more aggressive behavior. Subcutaneous MCTs are not graded and are less aggressive.

Plasma cell tumor (cutaneous)

Requires complete surgical removal, but generally don't metastasize. Rare in the cat. Usually on the head and distal limbs.



Lymphoma- Malignant

Many varied presentations, but enlarged lymph nodes are often present. With high grade B and T cell LSA, 80% present with generalized peripheral lymphadenopathy.

Histiocytoma

These generally occur in dogs less than 2 years of age and look like a red button with an eroded surface. They commonly occur on the front half of the body and will spontaneously regress within 3 months. Rare in cats.

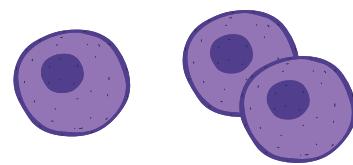
TVT- Malignant

Flower like or nodular tumor near the genitalia of dogs. Transplantation to other dogs or areas on the body by direct contact. Rare to metastasize, but should be surgically removed and followed up with chemotherapy.

Melanoma- Malignant

The most common oral tumor and the second most common digital tumor in dogs. Cutaneous melanomas are generally benign but should be removed. Oral and digital melanomas behave more aggressively. Rare in cats.

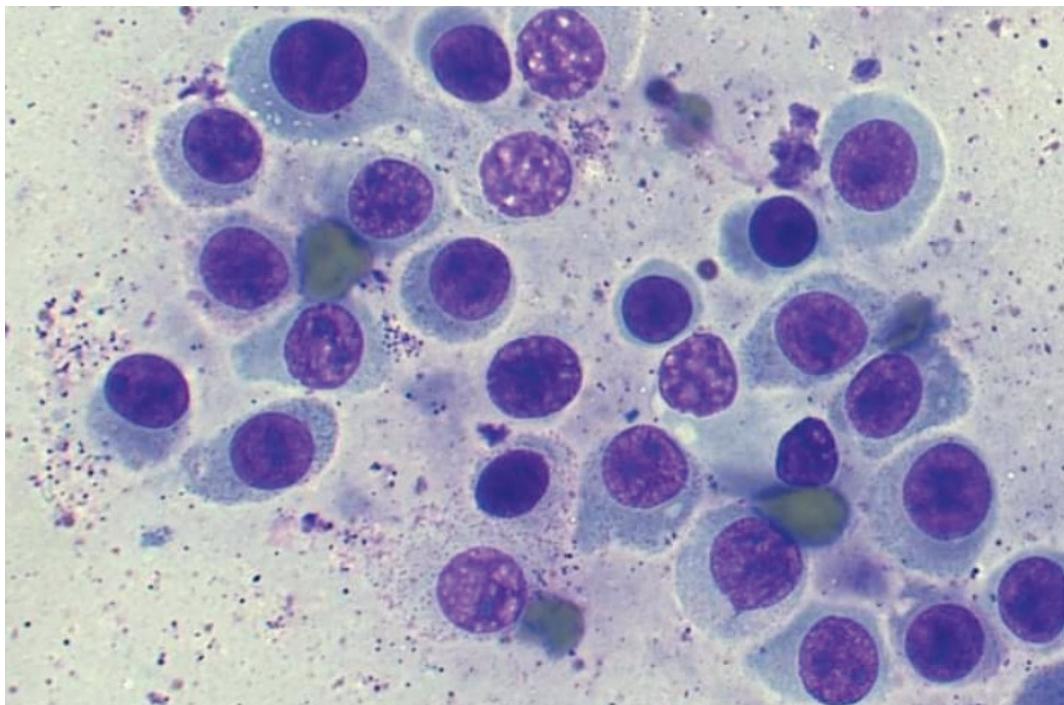
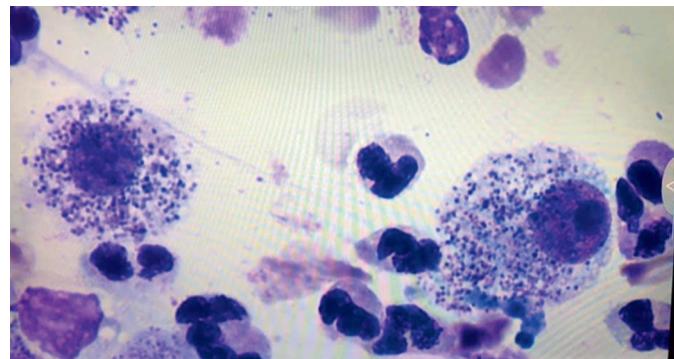
Round Cell tumors



Mast cell tumor-Malignant

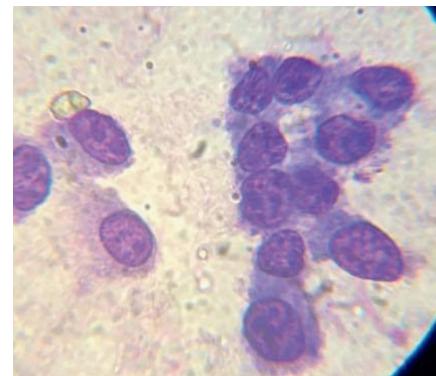
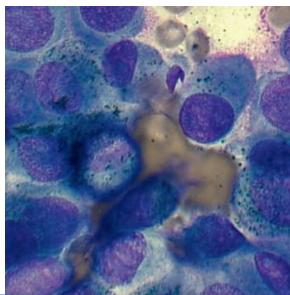
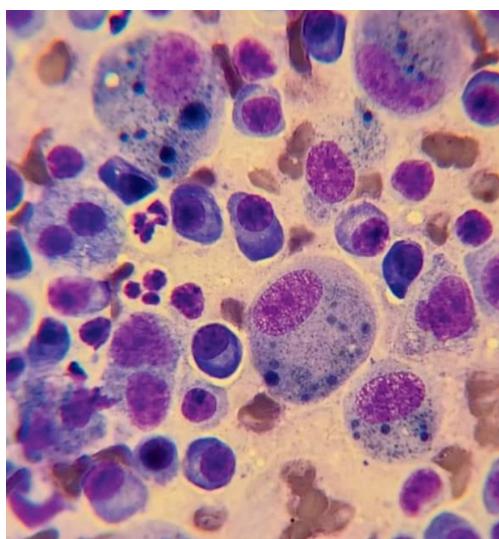
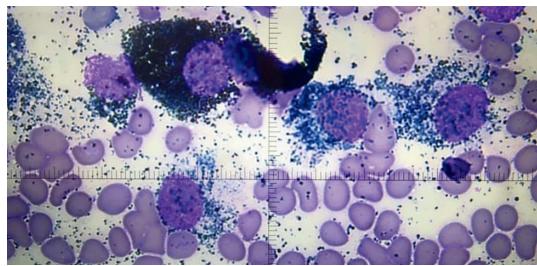
Round cells with classic even purple granules to the right.

The example below is a poorly staining mast cell tumor. You can still see many granules in the surrounding area.



Melanoma- Malignant

Melanoma has a varied appearance. Classically they appear as round cells, but they can also look epithelial or spindleoid sometimes as well. Their granules are dark blue to greenish brown.



Amelanotic Melanoma

Some malignant melanomas don't contain melanin. This is an amelanotic melanoma from an oral tumor in a dog.

Round Cell tumors

Plasma cell tumor (cutaneous)

Plasmacytomas are benign round cell tumors derived from β cells in dogs (rare to see cutaneous plasma cell tumors in cats). Found most commonly on the skin of the head or limbs. Occasionally they can be found in the mouth or rectal mucosa. Typically alopecic, pink and round and 1–2 cm. Skin plasmacytomas should be surgically removed but are rare to metastasize. Oral plasma cell tumors can behave in an aggressive fashion. Very rarely they are associated with multiple myeloma (β cell tumor in the bone marrow).

Plasma Cell cytology characteristics

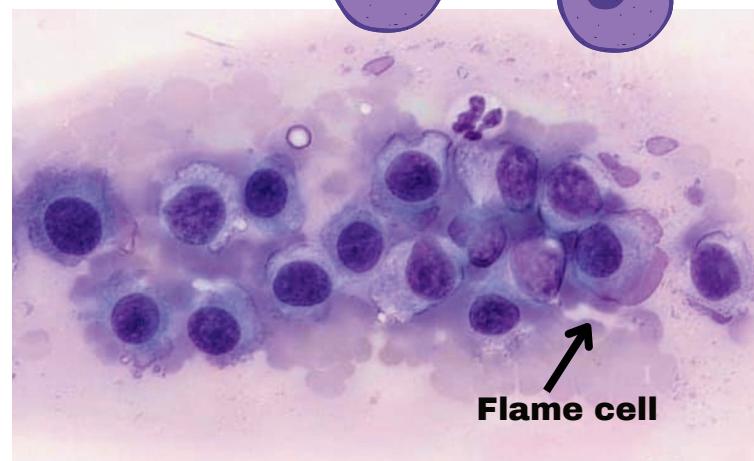
- Clumped chromatin pattern like a choco chip cookie
- Non uniform cells, with some criteria of malignancy seen (binucleation, variation in nuclear size and cell size)
- Defined cytoplasmic edges compared to a histiocytoma
- Blue cytoplasm
- Eccentric nuclei
- Perinuclear clear zone
- Found in older patients (5–11 years old)

Histiocytoma

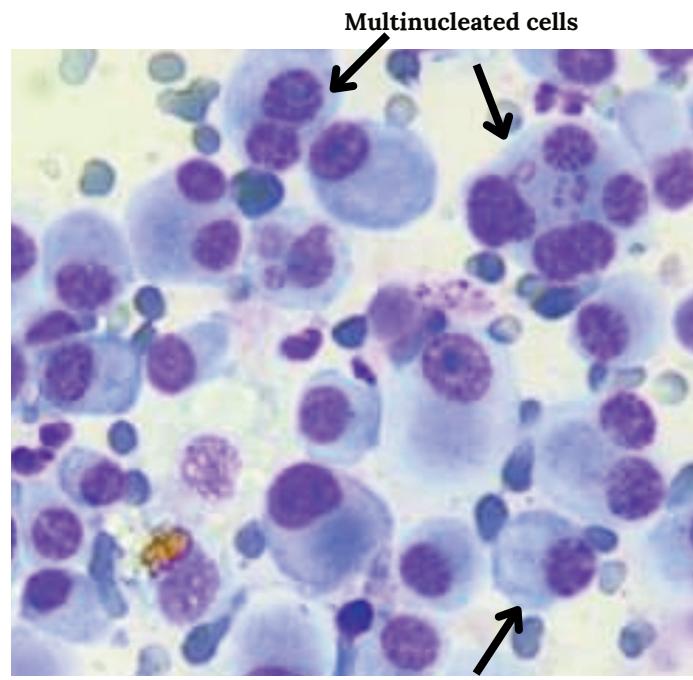
Benign skin growth in dogs that goes away on its own in 1–3 months. Occurs most commonly in dogs under 2 years of age and usually on the front half of the body. Typically partially alopecic and less than 2 cm in diameter. Rare in cats.

Histiocytoma cytology characteristics

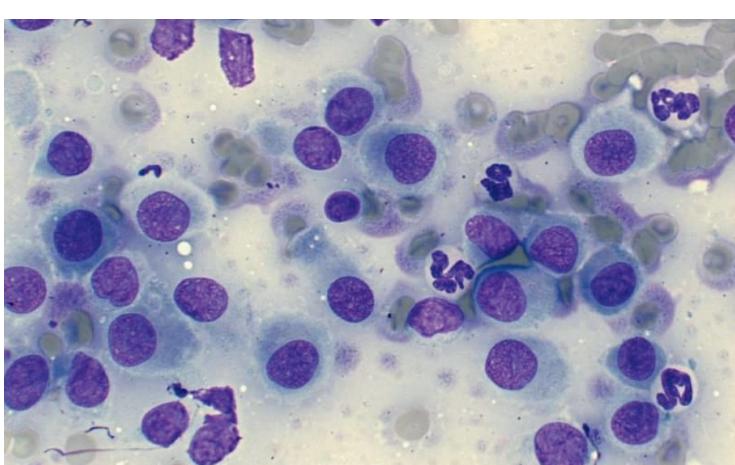
- Cell border fades away
- Uniform in appearance (compared to plasmacytomas)
- Pericytoplasmic clearing (clear zone along the edge of the cytoplasm)
- Central nuclei
- Med stain cytoplasm
- Usually younger patients (under 2 years)



Flame cells are plasma cells with an outer pink rim due to precipitated immunoglobulins from the β cells.



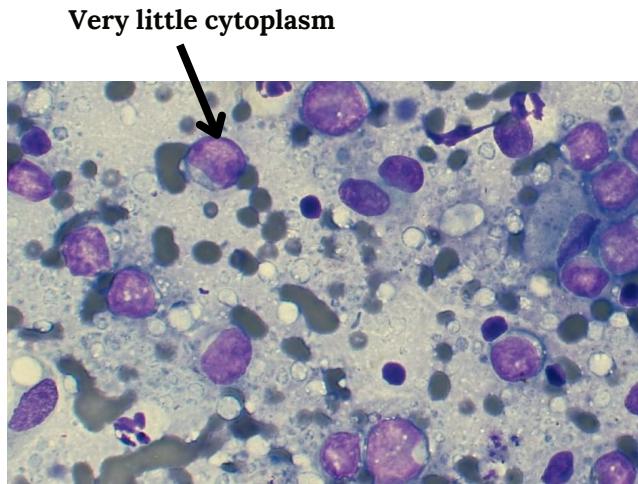
eccentric nuclei



Round Cell tumors

Lymphoma- Malignant

Many varied presentations, but enlarged lymph nodes are often present. With high grade B and T cell LSA, 80% present with generalized peripheral lymphadenopathy.

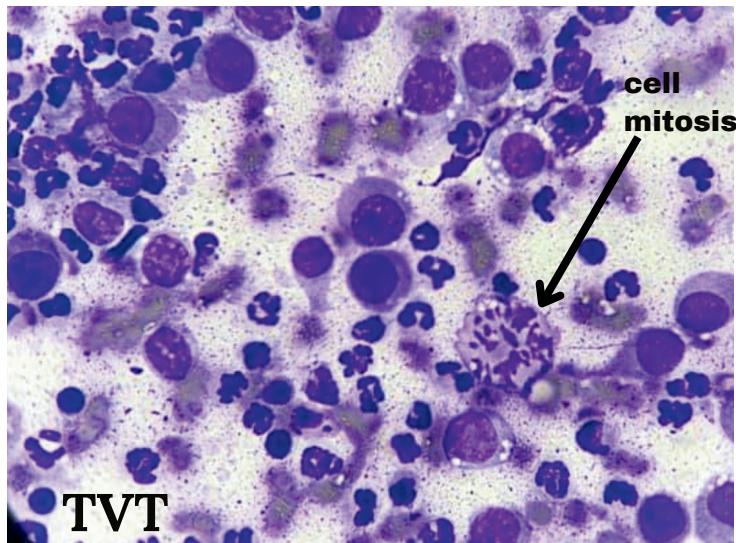


Lymphoma

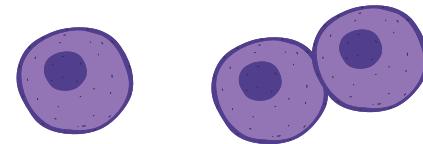
TVT- Malignant

Flower like or nodular tumor near the genitalia of dogs. Transplantation to other dogs or areas on the body by direct contact. Rare to metastasize, but should be surgically removed and followed up with chemotherapy. Sometimes chemo alone is effective.

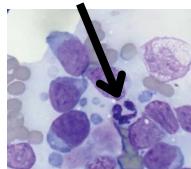
Cytoplasmic vacuoles, clumped chromatin (chocolate chip cookie) pattern, frequent mitoses are characteristic features.



TVT

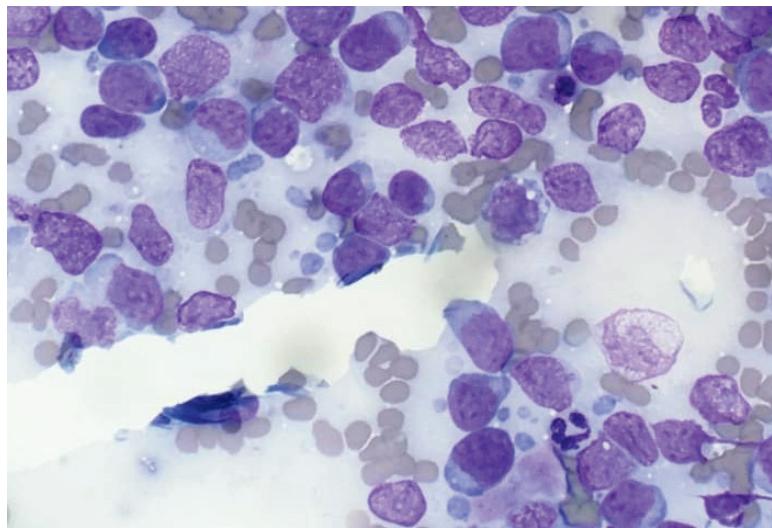


neutrophil smaller than blast nuclei



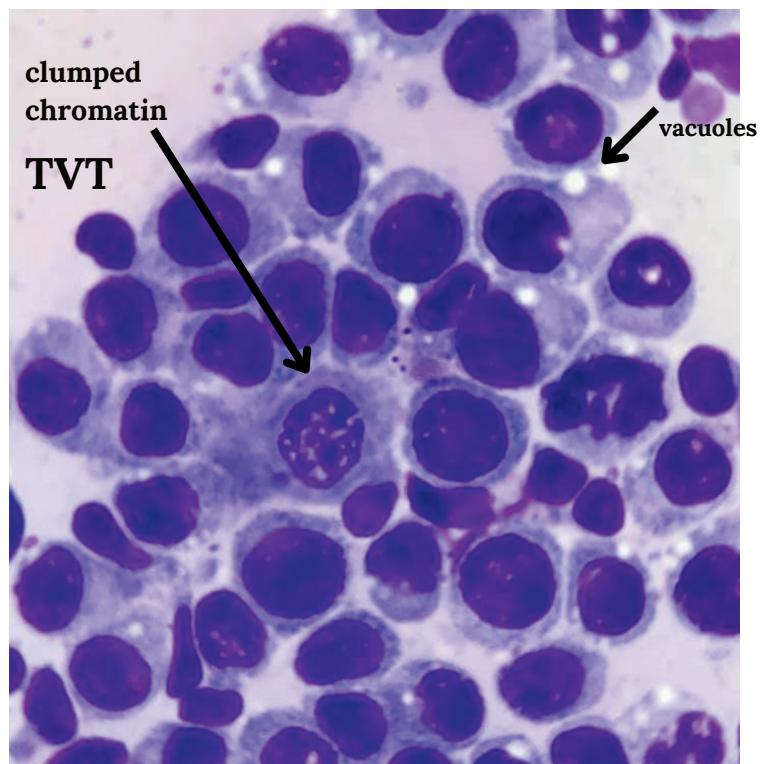
Lymphoma

Blast nuclei are larger than a neutrophil
80% or more are intermediate to large sized



Lymphoma cytology characteristics

- Blasts are round cells with very little cytoplasm.
- Nuclei are larger than a neutrophil.



TVT cytology characteristics

- Vacuolated round cells
- Clumped chromatin (chocolate chip cookie)
- Frequent mitoses

The next important consideration following tissue ORIGIN is LOCATION of the mass. The location of the mass can predict the likelihood of certain tumors. Below are several examples.

Location



Anus

Perianal adenoma. Common in uncastrated male dogs.



Anal gland

Anal Sac Adenocarcinoma of dogs



Ear

Ceruminous gland adenocarcinoma, Cystadenoma, SCC



Eyelid

Meibomian gland adenoma



Digit

Dogs: #1 SCC #2 Melanoma

Rare in cats, multiple digits: digit-lung syndrome



Mammary

Dogs: 50% adenoma 50% adenocarcinoma.

Cats: only 15% are benign, rest are malignant.

Siamese are affected at a younger age with a higher rate of lymphatic spread



Nasal planum

Cats: SCC



Nasal cavity

Cats: SCC Adeno carcinoma, lymphoma

Dogs: Adenocarcinoma, rare: sarcoma



Oral cavity

Cats: SCC is most common. >90% of all oral tumors in cats are malignant

Dogs: #1 Melanoma #2 SCC #3 FSA

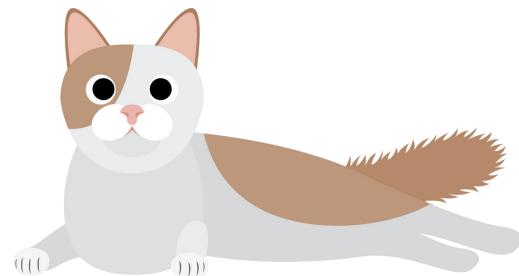
SCC= squamous cell carcinoma, LSA= lymphoma, ADC= adenocarcinoma, FSA= fibrosarcoma

Location



Pinna

Cats: SCC



Pharynx

Cats: LSA, then ADC

Dogs: numerous types, mostly malignant



Penis

Dog: SCC and TVT



Salivary Gland

Dog: ADC most common; cats- not common



Tonsil

SCC most common in cats/dogs; higher in urban areas with increased pollution



Testicle

Dogs: interstitial tumor most common, then seminoma and sertoli.

Low metastatic potential, castration typically curative



Tongue

Dogs 50% SCC but rare

Cats: SCC



Thyroid

Dogs ~75% malignant carcinoma and not functional; Cats usually benign adenoma and functional

SCC= squamous cell carcinoma, LSA= lymphoma, ADC= adenocarcinoma, FSA= fibrosarcoma, TVT= transmissible venereal tumor



Pet Specifics

The final consideration following tissue ORIGIN and LOCATION of the mass is the PET SPECIFIC information.

For example:

Is the pet a dog or a cat?

Is the mass ulcerated or not?

Is the mass associated with a loss of function?

Is the mass growing quickly?

Are there any lab abnormalities?

Is the pet a prone breed?



Some pet specific examples

- A digital tumor in a dog is more common than in a cat. The number one canine digital tumor is a SCC, followed by melanoma. Cats rarely get digital tumors, but if you see one, you should take some chest x-rays as metastasis from primary lung tumors happens to the toes.
- Mammary tumors in dogs are benign half the time, but almost always malignant in cats.
- Thyroid tumors in cats are almost always benign but not so in dogs. Dog thyroid tumors rarely express thyroid hormone. However cat tumors are typically diagnosed due to an elevated t4 measured on their bloodwork.
- Oral tumors in cats are almost always malignant and typically SCC. In dogs, melanoma is the most common oral tumor.
- Peripheral Nerve Sheath Tumors will often affect function because they affect the nervous enervation to the nearby muscles. 80% of PNST affect the brachial plexus.
- Masses that grow quickly or change shape/character (soft to firm , etc.) are more concerning and should be evaluated or rechecked.
- Certain tumors trigger high calcium . In cats this is typically due to lymphoma or SCC. In dogs, this is most often due to lymphoma and Anal Sac Adenocarcinoma. So if you have a dog with hypercalcemia, make sure to perform a rectal exam!
- Boxer dogs and French Bulldogs are prone to mast cell tumors but they are most often low grade.
- Canine mast cell tumors on the muzzle, oral/perioral area, prepuce and scrotum are associated with more aggressive behavior. Subcutaneous mast cell tumors are not graded and behave in a more benign fashion.
- Feline mast cell tumors are usually tiny dermal bumps a few mm only and most commonly arise on the head and neck. They rarely metastasize, but should be surgically removed. If you see multiple dermal mast cell tumors, then it may be worthwhile to screen for visceral MCT that has metastasized to the skin.
- Vaccine associated fibrosarcomas in cats are extremely aggressive. 60-80% recur with surgery alone and 30-40% with surgery and radiation.

FAQ's

What do I do if I start my investigation with an FNA and it's non-cellular? Just blood and no other cells present?

If it's an aspirate of a dermal bump that is reasonably small (~1cm or less) and is not growing quickly, I would first inform the owner that the bump is not expected to be a round cell tumor because they exfoliate cells easily. A skin mass that is small and non-diagnostic on FNA is most likely to be benign. So, I would offer either removal or monitoring. If the patient is healthy and you would likely anesthetize to perform another necessary procedure like a dental cleaning, then removal and submission for histopath would be quick and easy. I would, however, feel comfortable monitoring the bump for any changes in size (growth over 30%,) or character which would necessitate removal for biopsy at that time.

If your FNA is non diagnostic, but the mass is firm and large (over 2 cm and either dermal or subcutaneous), then I would recommend incisional biopsy or surgical removal if it's possible to obtain at least 2 cm margins. Since the mass did not exfoliate any cells readily, it could be an epithelial tumor or possibly a sarcoma. Most sarcomas are locally invasive but slow to metastasize. I would inform the owner that a diagnosis must be made with an incisional biopsy but that most of the tumor possibilities would be slow to grow. There should be time to make an informed decision and plan appropriately for surgery. (ie a 2 cm mass on the distal limb would be harder to remove with adequate margins and a sarcoma that requires wide margins might be best performed by a specialty surgeon).

What do I do for my patient if the FNA looks inflammatory?

If the FNA appears inflammatory and it is a very small mass (less than 1 cm) then you can advise the owner to monitor for regression. If the lump is ulcerated or appears swollen/infected you may choose to send home an antibiotic or recommend antihistamines if there are a lot of eosinophils present and recheck the mass in 1-2 weeks.

What do I do if my initial FNA is just clear fluid and the lump drains completely? Small, solitary cysts are rarely malignant. I would inform the owner that the mass is a cyst and likely benign. Cysts will not go away after drainage. They retain a secretory cyst lining and will fill back up with fluid. We do not advocate periodic drainage as this can introduce infection. They rarely cause irritation or discomfort to the pet. An option is always to surgically remove the cyst and submit it for formal histopathology.

What if I'm not really sure if it's a round cell or just macrophages vs epithelial etc. ? We are not always going to be correct in our first step determining if the mass is epithelial, mesenchymal or a round cell.

Once you've scanned your own slides to make sure that there are cells to interpret, then it is always recommended to submit these slides for formal cytology to your reference lab. Sometimes you will misinterpret a mass as round cell when it's actually epithelial or even just inflammatory. However, in most cases, you will be able to rule out a mast cell if you don't see characteristic cells. You can also provide your clinical impression based on the patient and the location of the mass. For example, cells that look like round cells from an oral tumor in a dog you should discuss melanoma as the most common oral tumor in dogs. (Even if the cells don't appear pigmented.) Conversely, a mass that arises from a 1 year old dog that is 1 cm and ulcerated on the surface and exfoliates cells that appear to be round cells is most likely a benign round cell tumor like a histiocytoma or possibly a plasmacytoma.

Do you recommend an FNA on every new lump?

In some cases you may feel confident not pursuing an FNA. Some examples are viral papillomas, sebaceous adenomas, or skin tags. Often times these smaller skin tumors don't exfoliate cells well. You can always offer the FNA but warn the client that they may not exfoliate cells well. If the aspirate appears non-diagnostic, you can use your best clinical judgement. (ie, recommend surgical biopsy vs monitoring).

What do you do if you see a nodule arise near a vaccine site in a cat?

Surgically biopsy these immediately if any of the following apply:

1. They are present for longer than 3 months
2. They are > 2cm
3. They are growing in size 4 weeks after vaccination



Moving Forward

Veterinary medicine is a practice. It takes a lot of repetition and experience to gain confidence. My hope is that this guide will help you gain more comfort approaching masses in practice.

A few tips to help you grow your skills:

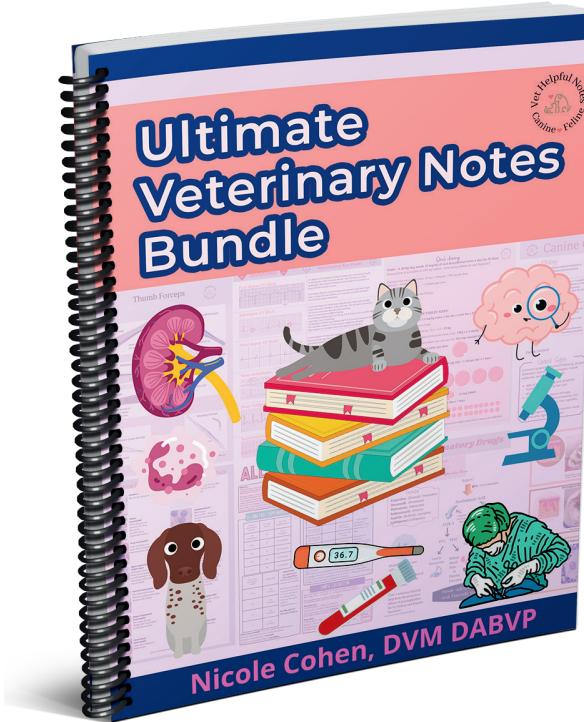
Join a veterinary cytology Facebook group. These groups post cytologies and discuss their characteristics and diagnoses. Often times these groups are moderated by Clinical Pathologists.

Read up on E Clin Path online for expert information on all things cytology.

Seek out continuing education on these topics.

In practice, keep a collection of your own cytology slides. You can keep these labeled in a case to refer back to or train others with these slides.

Always compare your tentative cytologic diagnosis with the results from the clinical pathologist report when you get your submission results.



I hope you found this guide helpful.

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Ultimate Notes Bundle

Lesions of the cornea



Improve your descriptions

LIPID DYSTROPHY



CORNEAL DYSTROPHY



NEOVASCULARIZATION



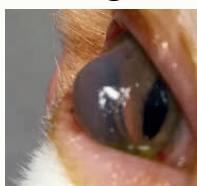
PANNUS



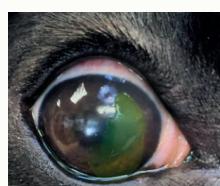
SEQUESTRUM



BULLOUS KERATOPATHY



INDOLENT ULCER



PERFORATED CORNEAL ULCER



MELTING ULCER



PIGMENTARY KERATITIS



EOSINOPHILIC KERATITIS



ENDOTHELIAL DISEASE



STROMAL ULCER



DESCETMETOCOELE



DERMOID



TROPICAL KERATOPATHY



Lesions of the cornea

Definitions



LIPID



CALCIFIC CORNEAL DYSTROPHY



NEOVASCULARIZATION



PANNUS

Usually inherited bilateral deposits centrally of lipid/cholesterol. Rarely requires treatment

Calcium/mineral deposits in the cornea. Can lead to painful sloughing and require treatment to heal the cornea.

Formation of a blood supply across the corneal surface to aid healing

Immune mediated growth of inflammatory and vascular tissue on the cornea or third eyelid. Exacerbated by UV light



SEQUESTRUM



BULLOUS KERATOPATHY



INDOLENT ULCER



PERFORATED CORNEAL ULCER

Focal region of necrosis and pigmentation

Blister like swelling on the cornea caused by edema. Most commonly seen in cats

Spontaneous chronic corneal epithelial defects (SCCED). Superficial, non-infected and chronic corneal ulcers.

Ruptured cornea that is often plugged up by the iris



MELTING ULCER



PIGMENTARY KERATITIS



EOSINOPHILIC KERATITIS



CORNEAL ENDOTHELIAL DEGENERATION

An infected corneal ulcer that causes softening of the cornea secondary to destructive enzymes.

Long-standing inflammation of the eye surface which leads to pigment deposits on the cornea

Eosinophilic infiltrate on the cornea and conjunctiva in cats. It often looks white in color.

The innermost layer of the cornea functions to pump water out of the cornea. When these pumps breakdown, the cornea becomes edematous



STROMAL ULCER



DESCET METOCOELE



DERMOID



TROPICAL KERATOPATHY

A deeper corneal ulcer involving both the superficial and stromal/mesenchymal connective tissue of the cornea

A deep corneal ulcer that is nearly full thickness, sparing the final layer of the cornea (which does not take up stain)

Skin like tissue growing on the eye

Variably sized round lesions on the cornea. These lesions develop in tropical locations and their cause is unknown. No treatment is needed.

Iridial & Conjunctival Abnormalities



Improve your detection

ANTERIOR SYNECHIA



POSTERIOR SYNECHIA



IRIS FRECKLE



IRIS NEVI



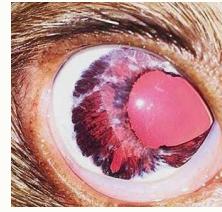
HETEROCHROMIA IRIDUM



IRIS MELANOMA



COLOBOMA



PPM



DYSCORIA



RUBEOSIS IRIDIS



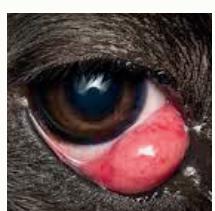
CHEMOSIS



EPISCLERAL INJECTION



CHERRY EYE



CONJUNCTIVITIS



SUBCONJUNCTIVAL HEMORRHAGE



SYNBLEPHARON





Lesions of the Iris and Conjunctiva



ANTERIOR SYNECHIA



POSTERIOR SYNECHIA



IRIS FRECKLE



IRIS NEVI

Adhesion between the iris and the cornea. Typically due to injury or infection.

Adhesion between the iris and the lens.

A feline specific condition where the iris becomes pigmented. A freckle is small focal deposit.

Iris nevi are larger than iris freckles. They are not superficial like freckles and affect deeper structures in the iris.



HETEROCHROMIA IRIDUM



IRIS MELANOMA

Two different colored eyes due to lack of pigment in one eye. The Husky is the most common breed.

Melanocytic tumor of the iris. Can be hard to distinguish from melanosis. Tips: uveitis, pupil shape changes, raised lesions are suspicious.



COLOBOMA

A congenital hole in the iris due to incomplete formation. Genetic in dogs such as the Australian Shepard.



PPM

Persistent pupillary membranes. Remnants of a fetal structure called the pupillary membrane. They generally cause no harm.



DYSCORIA



RUBEOSIS IRIDIS

A misshapen pupil. Can be due to iris atrophy or scarring secondary to previous or current inflammation in the eye

Iris neovascularization typically caused by uveitis.



CHEMOSIS

Swelling or edema of the conjunctiva



EPISCLERAL INJECTION

congestion of the deep episcleral vessels that run 90 degrees to the limbus. They represent an external sign of intraocular disease.



CHERRY EYE



CONJUNCTIVITIS



SUBCONJUNCTIVAL HEMORRHAGE



SYNBLEPHARON

Prolapse of the third eyelid gland that lives behind the nictitating membrane (3rd eyelid)

Inflammation of the conjunctiva from infection or allergies.

Ruptured blood vessels beneath the conjunctiva. Most commonly due to trauma (vehicle trauma, attack, strangulation)

Adhesion of the palpebral conjunctiva to the bulbar conjunctiva of the eye. Most common in kittens with herpes virus.



Uveal, Retinal & Lens Abnormalities



Improve your detection

UVEAL CYSTS



HYPOPYON



HYPHEMA



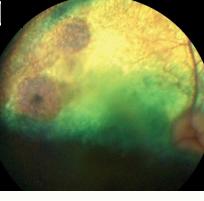
ASTEROID HYALOSIS



GRANULOMATOUS UVEITIS



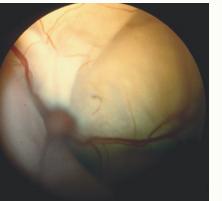
GOLD MEDALLION LESIONS



RETINAL HEOMORRHAGES



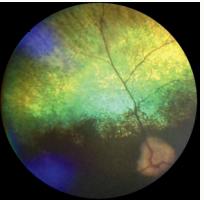
BULLOUS RETINOPATHY



TORTUOUS RETNAL VESSELS



PROGRESSIVE RETINAL ATROPHY



NORMAL HUSKY RETINA



IMMATURE CATARACT



MATURE CATARACT



HYPERMATURE CATARACT



ANTERIOR LENS LUXATION



LENTICULAR SCLEROSIS



Lesions of the uvea, retina and lens

Definitions



UVEAL CYSTS



HYPOPYON



HYPHEMA



ASTEROID HYALOSIS

Fluid filled pigmented cysts that arise from the pupil margin. They are most often benign. In Golden Retrievers may indicate pigmentary uveitis.

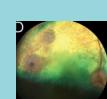
Inflammatory cells in the anterior chamber of the eye.

Red blood cells in the anterior chamber of the eye.

Sparkling calcium lipid complexes in the vitreous body. An aging change. Does not seem to change vision and is not treated.



GRANULOMATOUS UVEITIS



GOLD MEDALLION LESIONS

Keratic precipitates form from immune complexes depositing on the inside surface of the cornea. Can be seen in cats with FIP.

Hyperreflective "gold medallion" lesions in the retina are characteristic for previous distemper infection causing retinal inflammation.



RETINAL HEOMORRHAGES



BULLOUS RETINOPATHY



TORTUOUS RETINAL VESSELS



PROGRESSIVE RETINAL ATROPHY

When the retinal vessels become more twisty due to higher vascular pressure. Is the first sign of hypertensive retinopathy.

When the retinal vessels become more twisty due to higher vascular pressure. Is the first sign of hypertensive retinopathy.



NORMAL HUSKY RETINA



IMMATURE CATARACT

Opacification of the entire lens which also prevents any tapetal reflection.

A hypermature cataract involves the entire lens but has sparkly clear zones of resorption.

Blue irises, heterochromia or merle coat color is associated with variable to complete lack of pigment. This allows the choroidal vessels to show through.

Opacification of 15-100% of the lens that still allows for a tapetal reflection.



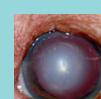
MATURE CATARACT



HYPERMATURE CATARACT



ANTERIOR LENS LUXATION



LENTICULAR SCLEROSIS

Opacification of the entire lens which also prevents any tapetal reflection.

A hypermature cataract involves the entire lens but has sparkly clear zones of resorption.

Dislocation of the lens into the anterior chamber. Can impede aqueous flow and cause glaucoma if not treated.

Cloudy central discoloration of the lens that is a normal aging change. It occurs in middle age as a result of lens fibers packing together over time.



Eyelid Lesions



Improve your detection

BLEPHARITIS



Eyelid inflammation
Often times allergy or immune mediated. Usually treated with oral prednisone and oral antibiotics to treat the secondary staph infection.

MEIBOMIAN GLAND ADENOMA



Age related benign eyelid tumors of the glandular tissue of the eyelid. Seen most commonly in dogs. The most common eyelid tumor in the dog.

AGENESIS



Eyelid agenesis is seen in some cats as a congenital defect of the eyelid. This usually affects the upper and outer portion of the eyelid.

DISTICHIA



Extra eyelashes that grow downwards through the meibomian gland openings. These lashes can rub and irritate the eye surface. They can be plucked or corrected with cryosurgery.

ENTROPION



Rolling of the eyelid inward. Here there is lower eyelid entropion. This is often a breed specific disease based on conformation. Can also be due to scarring, nerve damage, infection, weight loss.

ECTROPION



Rolling of the lower eyelid away from the eye. Can make the dog more susceptible to conjunctivitis and corneal inflammation.

TRICHIASIS



Misdirected eyelashes that cause them to rub on the eye surface. Seen most commonly in dogs than in cats.

CHALAZION



A chronic obstruction of the meibomian gland causing inspissated glandular secretions and a focal swelling. A stye is a more acute infection of the meibomian gland.



Canine Corneal Ulcer

Vet Helpful Notes
Canine Feline

4

STAIN TYPES

Superficial

Superficial Ulcer



Floor only
sharp
borders

Indolent Ulcer



Floor only
indistinct
borders

Deep

Stromal Ulcer



Walls and
floor
take up
stain

Descemetocoele

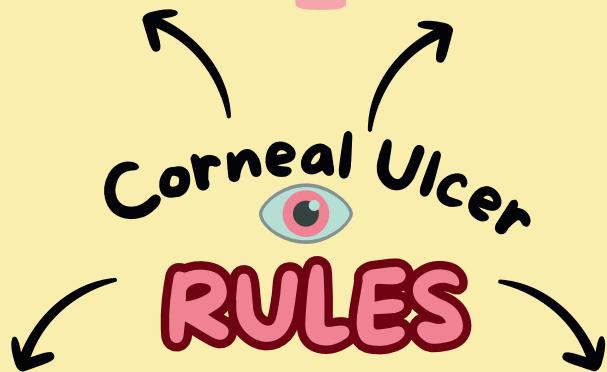


Walls
only
take up
stain

The most critical treatment for an ulcer is removal of the primary cause



If an ulcer has not healed in 7 days, change your diagnosis, not your antibiotic



Infected ulcers are green and deep or green and melty

Corneal epithelium migrates 1mm/day so a simple ulcer should heal within 7 days



Canine infected ulcer



strategies

1. Perform cytology

to see if you have rods or cocci
gm + or gm - is helpful too

GRAM + 
staph
strep

GRAM - 
Pseudomonas

Most Common Isolates in Stromal Infections

1. Perform cytology

Use a small corneal brush

Rub the edge of the ulcer

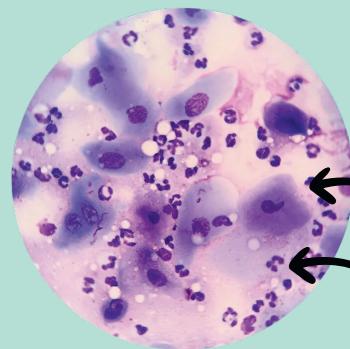


Use a small corneal brush.

DON'T rub on a descemetocoele or perforated cornea



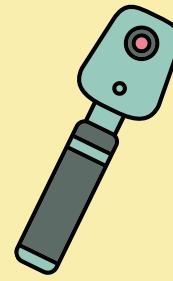
GENTLY Rub at the EDGE of the ulcer to get the best sample.



Even if you don't find bacteria, neutrophilic inflammation suggests infection of the cornea



Canine infected ulcer



It's always best to refer! But start treatment to the best of your ability

Antibiotic drops q 2 hours is cytotoxic to the eye, so only use at this frequency for 24- 48 hours



Infected Ulcer



RULES

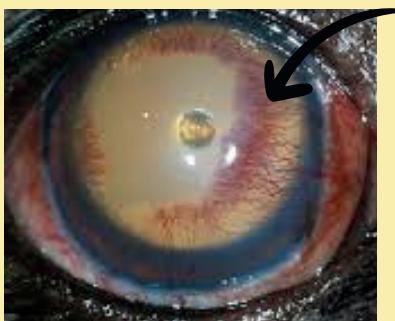
Serum applied to non melting ulcers or a sequestrum is not helping

Infection is assumed IF there is:

- Stromal loss
or
- Milky infiltrates
or
- Hypopyon!

How to monitor therapeutic Response

You are



monitor the corneal vascularization. It should be steadily (at ~1 mm per day) moving towards then into the ulcer bed



Canine melting ulcer

Vet Helpful Notes
Canine Feline

Apply serum q 2 hours and recheck the ulcer in 24 hours; you can add amnion TID



serum



Draw off serum with a sterile eye dropper. Replace q 3 days. Keep in fridge.



Serum

Serum has natural anti-collagenases to reduce the "melting"

- Collect from a donor with healthy eye and push through a 0.22 micron syringe filter if you want to keep it longer (5 days vs 3 days)
- Use heterologous serum (serum NOT from the patient)
- Melting is caused by the patient's own immune reaction, so you don't want to use the patient's own serum
- Can be from a different species!

While some ophthalmologists trust amnion to replace serum, right now it's not proven to be a complete substitute.



Amnion

This is Vetrix EyeQ commercial amniotic membrane drops. Use TID



Amnion

Amnion is commercially prepared drops derived from placental membranes.

It is a natural biologic scaffold.

It also has high concentrations of natural anti-inflammatory components that reduce inflammation and promote re-epithelialization of the cornea **QUICKLY**.

melting ulcer

serum +/- Amnion

culture and sensitivity
widen antibiotic coverage
frequency q 2 hrs for 24-48 hours
oral doxycycline
oral pain reliever
Refer!





Corneal Ulcer



Vet Helpful Notes
Canine ♥ Feline

Non Trauma Causes



ectopic cilia



trichiasis



distichia



foreign body



dry eye



eyelid tumor



herpes virus



lagophthalmus



entropion



Corneal Ulcer



Vet Helpful Notes
Canine & Feline

Non Trauma Causes

ectopic cilia



Ectopic cilia are one or several hairs that grow abnormally through the conjunctiva and come into contact with the surface of the eye

trichiasis



Trichiasis are hairs that originate in a normal location on the skin around the eye but are oriented in an abnormal direction

distichia



Distichiasis are extra eyelashes, which come out in the wrong place on the lid margin and rub on the eye.

foreign body



Foreign material that lodges in the cornea

dry eye



Also known as Keratoconjunctivitis Sicca (KCS), is decreased or inadequate tear production which irritates the eye.

eyelid tumor



Meibomian masses are the most common eyelid tumor of middle-aged and older dogs and are benign. Some growths can rub and irritate the cornea.



Corneal Ulcer



Non Trauma Causes

herpes virus



Herpesvirus causes superficial or surface ulcers on the cornea in cats

lagophthalmus



Lagophthalmos is an inability to fully close the eyelids and protect the cornea from drying and trauma

entropion



Entropion is when the eyelid rolls inward. This inward rolling often causes the hair on the surface of the eyelid to rub on the cornea causing irritation

Canine common medications

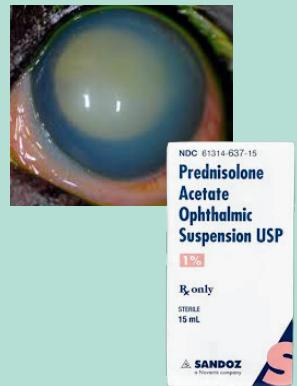
Endothelial disease



Glaucoma



Uveitis



Cataracts



Corneal ulcer



Melting



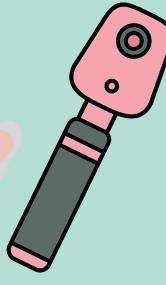
KCS



Pannus



Canine



Cataracts



- As cataracts mature, they can induce low grade inflammation.
- Ophthalmologist frequently recommended daily NSAID drops like Ketorolac when cataracts mature.
- This can help prevent uveitis and secondary glaucoma.



Pannus



- Chronic inflammation of the corneal epithelium and anterior stroma. Typically bilateral and results in neovascularization, pigmentation, and opacification.
- Treated with immune modulating drops like cyclosporine or tacrolimus. Sometimes 0.1% dexamethasone is added to cyclosporine
- Frequently seen in Shepherds, Greyhounds and Dachshunds. UV light exacerbates the condition.

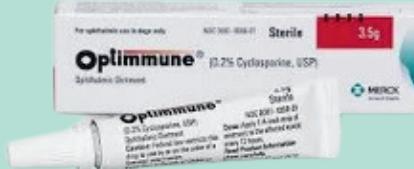


Canine



Vet Helpful Notes
Canine Feline

KCS



- Tear Film deficiency that causes the cornea to become inflamed and irritated from lack of lubrication



- Most common cause is an immune mediated attack of the lacrimal tissue.



- Optimmune is a commercially available ointment with low dose cyclosporine is frequently the first line treatment option for mild to moderate KCS ($>5\text{mm/min}$). Often move up in strength to 2% cyclosporine or use tacrolimus



melting ulcer



- Melting ulcer is keratomelacia. A deeper ulcer with infection "melting" the cornea



- Vetrix EyeQ is a commercially available option you can use for melting ulcers. It uses amnion to form a "biologic scaffold"



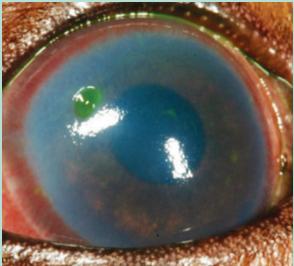
- Serum is the traditional method to combat melting ulcers. It is simply serum taken from a healthy donor pet's blood sample. Other options include acetylcysteine



Canine

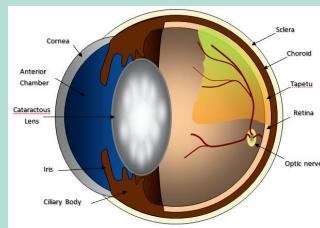


corneal ulcer



- A defect on the surface of the cornea. Fluorescein stain is absorbed by the damaged cornea.
- A simple corneal ulcer should be treated with a broad spectrum topical antibiotic. Reserve antibiotics like fluoroquinolones for complicated ulcers with infection present!

Uveitis



- Inflammation in the uveal tract: iris, ciliary body and the vascular "tunic" the choroid vascular layer of the eye. The BROWN part of the diagram above.
- Prednisolone is a potent steroid to help reduce inflammation in the eye
- There are MANY causes of uveitis ranging from cataract induced inflammation, glaucoma, cancer, infections etc.



Canine



Vet Helpful Notes
Canine Feline



endothelial degeneration



- Impaired Na/K+ pumps so the cornea becomes water-logged and cloudy-blue.
- The fluid filled cornea weakens the corneal integrity and puts dogs at risk for fluid filled blisters and corneal ulcers.
- Muro 128 is a salt solution that helps prevent further water accumulation. Unfortunately, it does not make the cornea more clear.
- Dogs at risk: Boston Terrier, English Springer Spaniel, Chihuahua, Boxer and Dachshund



Glucoma



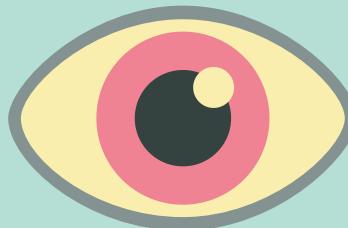
- Impaired fluid drainage from the eye causing high pressures in the eye (Pressure >30 mmHg)
- Latanoprost is a prostaglandin analog that causes a miotic pupil to **INCREASE** the drainage angle. The other common drugs used to address glaucoma include dorzolamide +/- timolol and mannitol IV
- Common breeds: Cocker Spaniel, Basset Hound, Chow Chow, Shar Pei, Shiba Inu, Beagle, Boston Terrier



The Eye a window inside



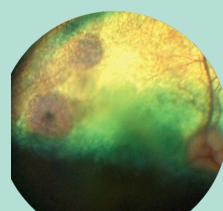
Vet Helpful Notes
Canine ♥ Feline



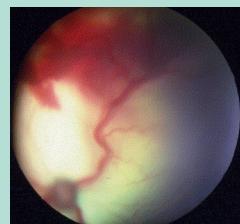
Lymphoma



Distemper



Hypertension



FeLV



Herpes



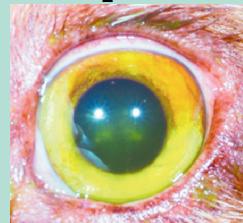
Toxo



ITP



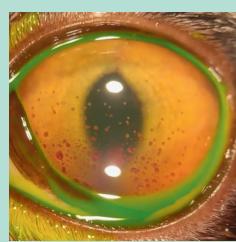
Lepto



Histo



FIP



Diabetes

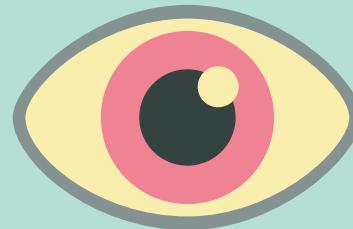


T1-T3





The Eye a window inside

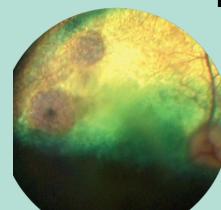


Lymphoma



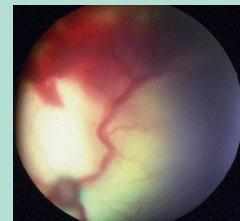
Eye mass

Distemper



Gold Medallion Lesions

Hypertension



retinal tortuosity
and hemorrhages

FeLV



Reverse D pupil

ITP



Hyphema

FIP



Granulomatous uveitis

Herpes



Dendritic ulcer



Fluffy infiltrates

Histo



conjunctivitis,
chorioretinitis

Lepto



Icterus



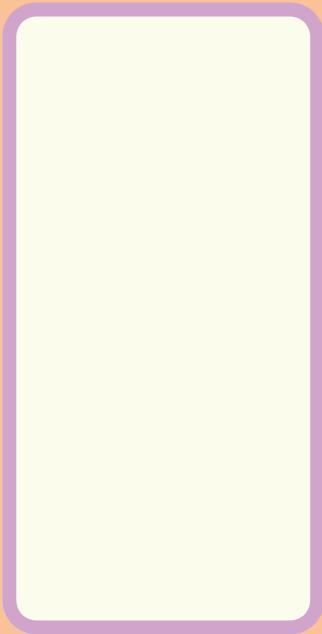
Horner's syndrome

Diabetes

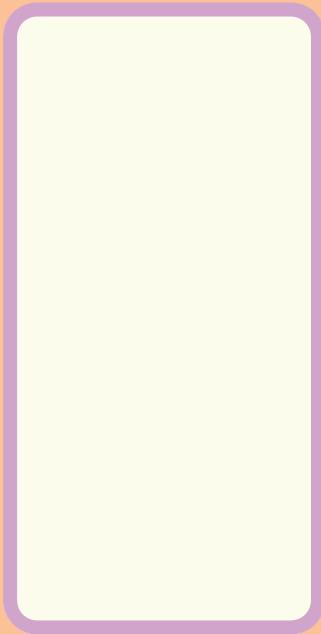


Cataracts

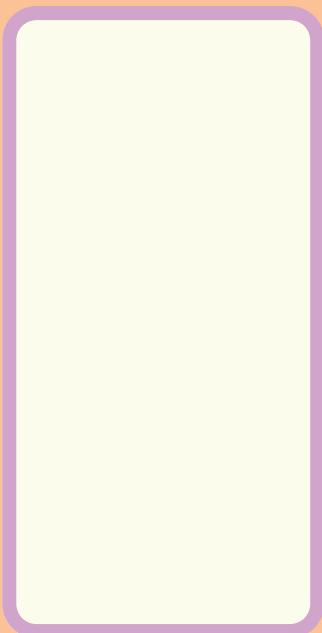
Medication



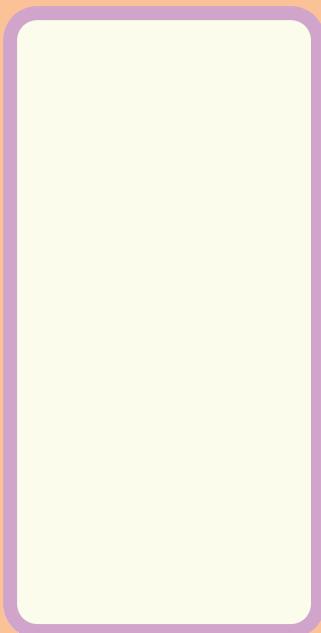
Medication

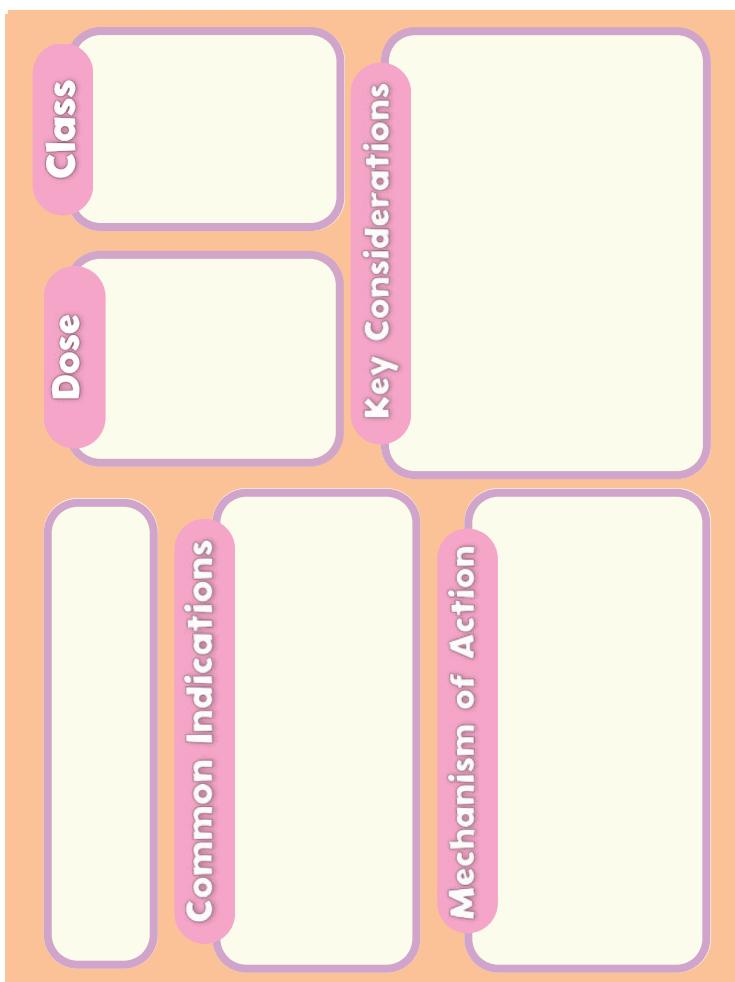
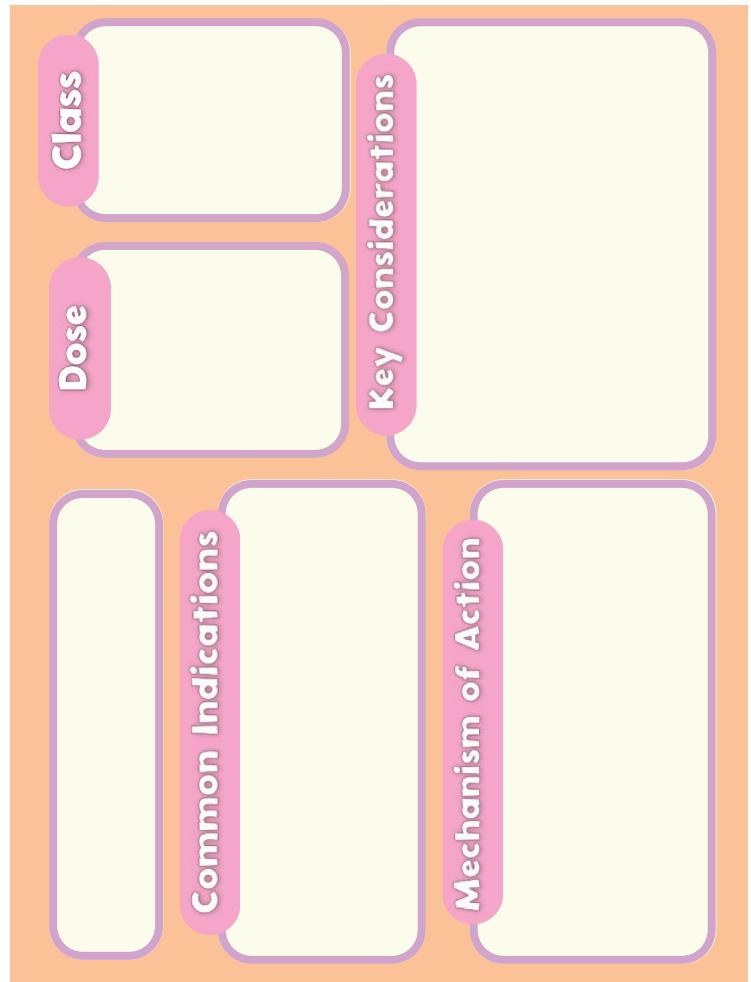
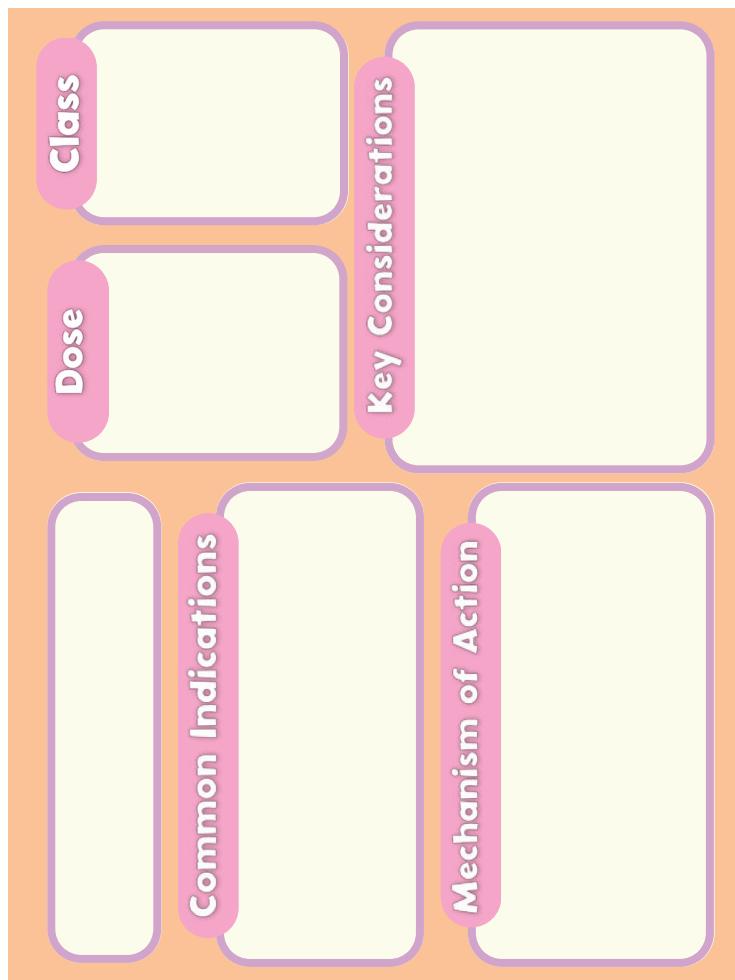
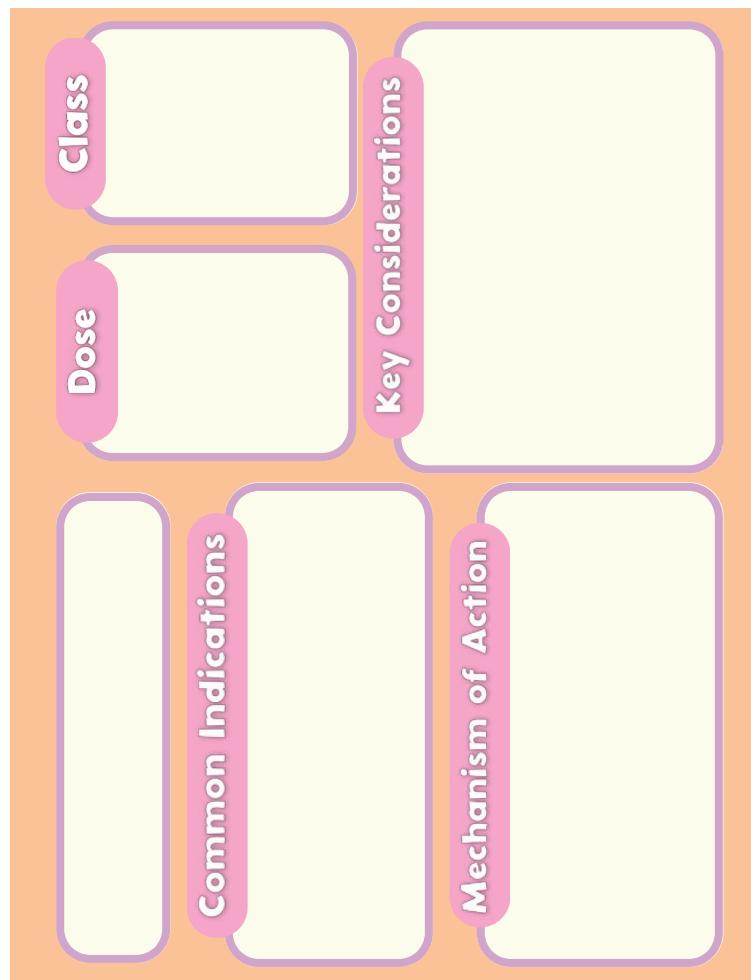


Medication

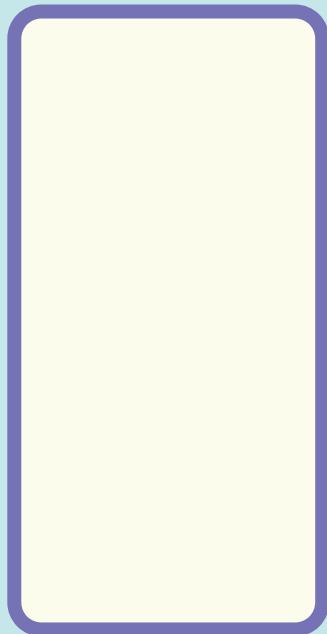


Medication

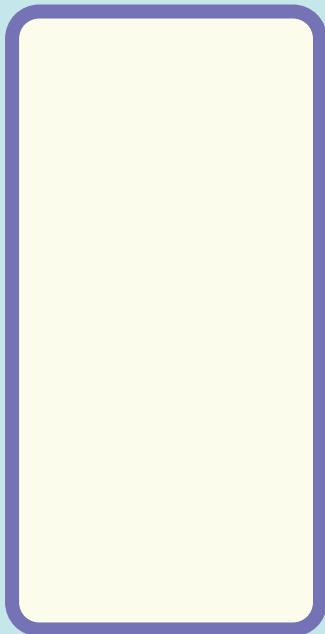




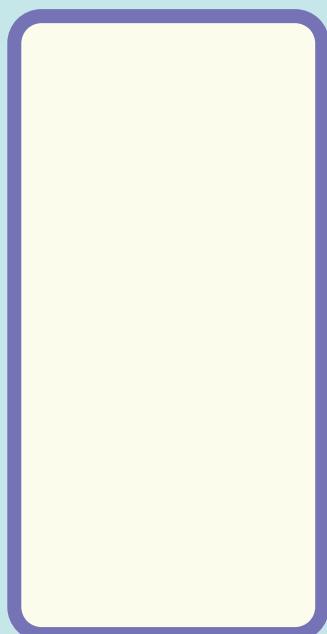
Medication



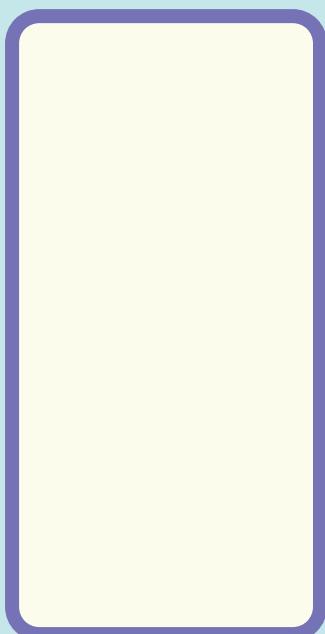
Medication

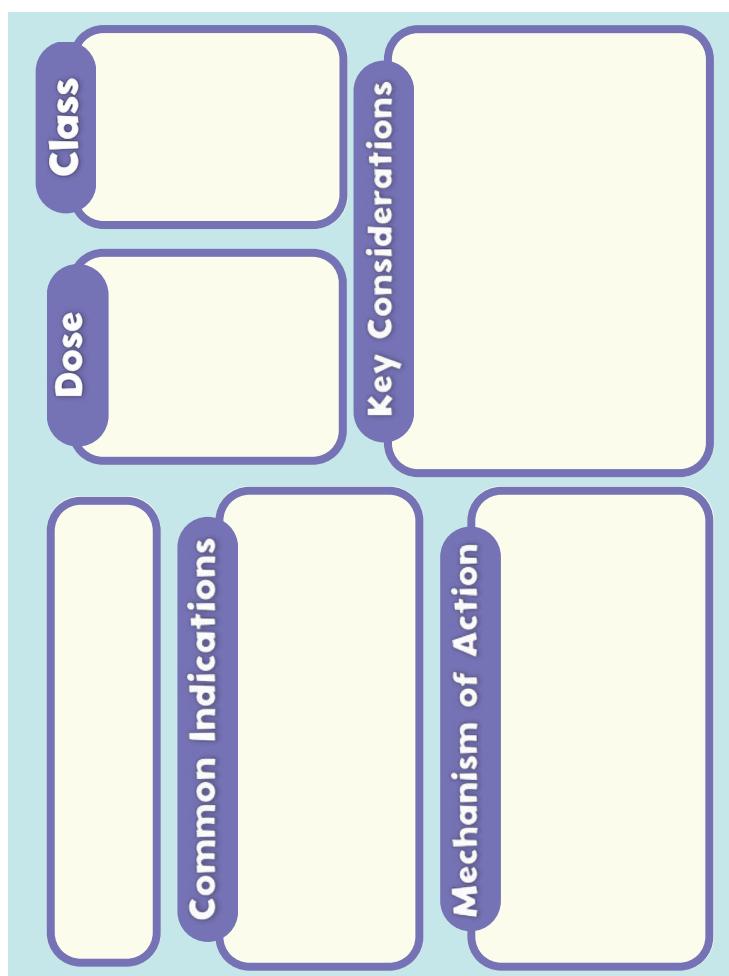
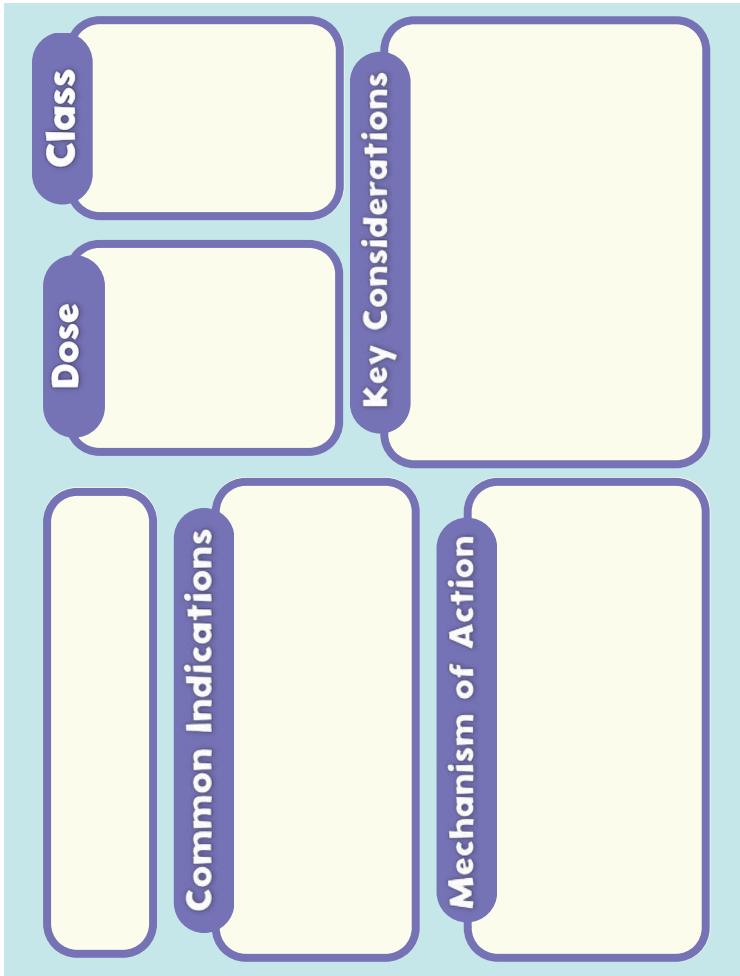
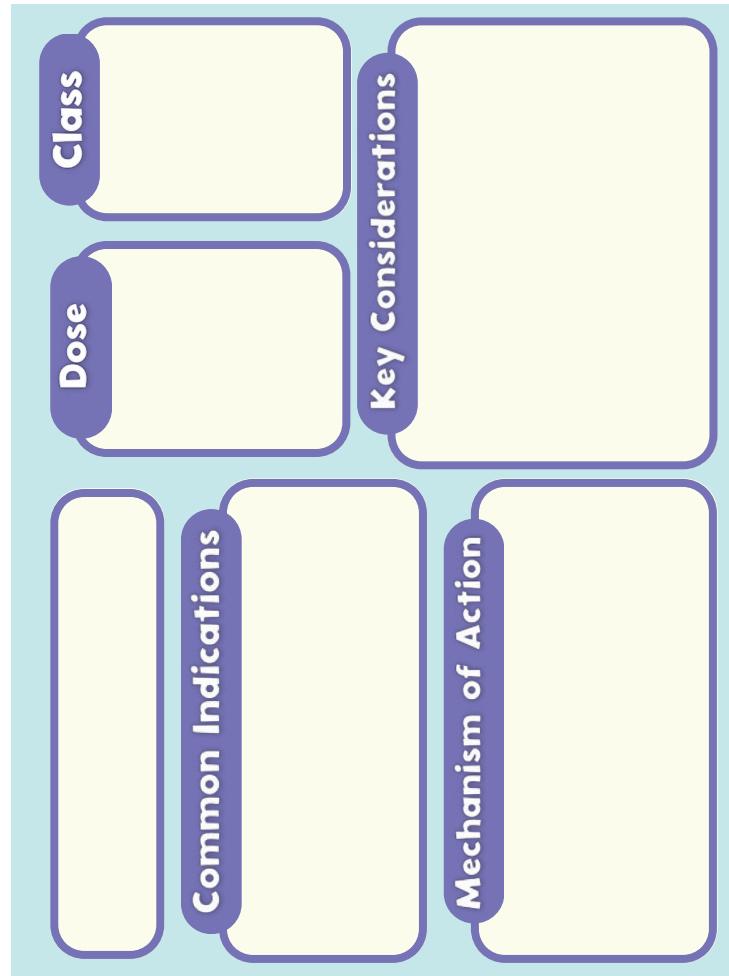
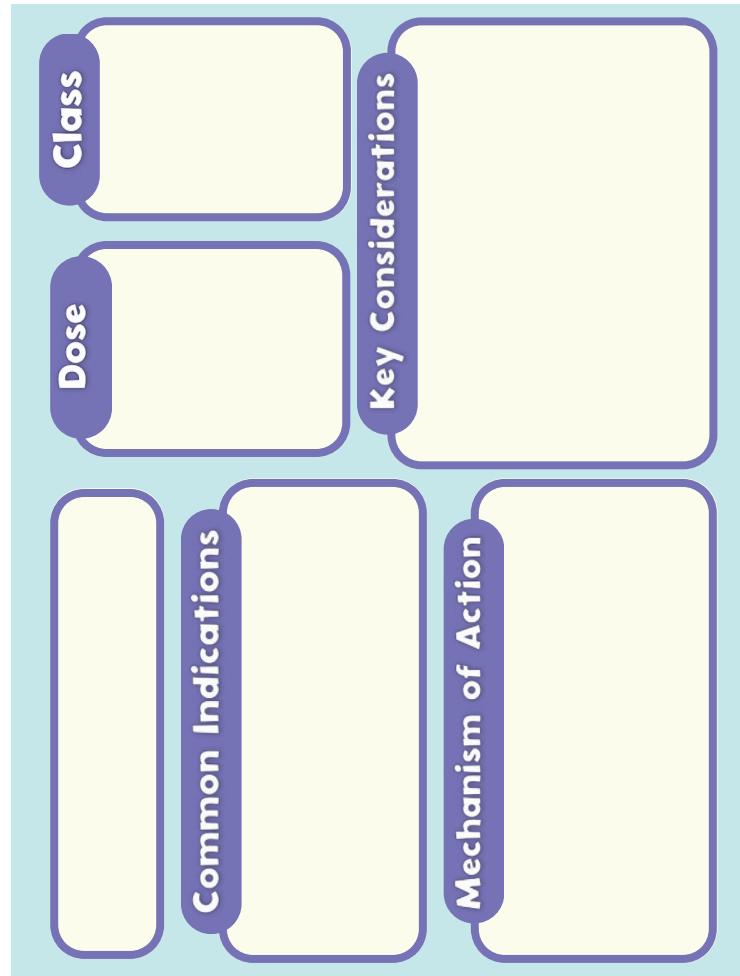


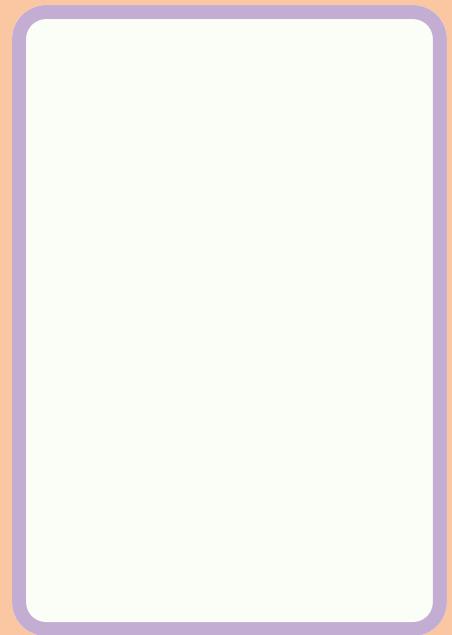
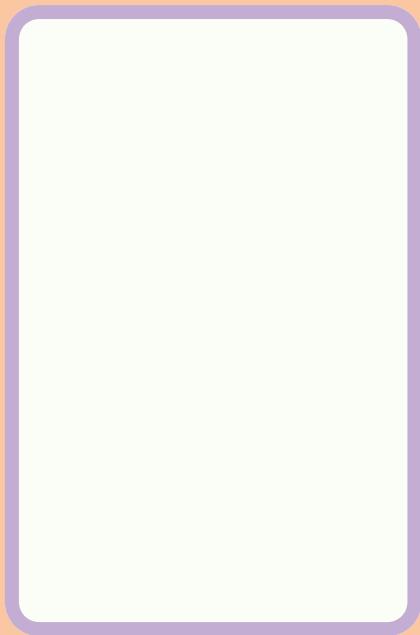
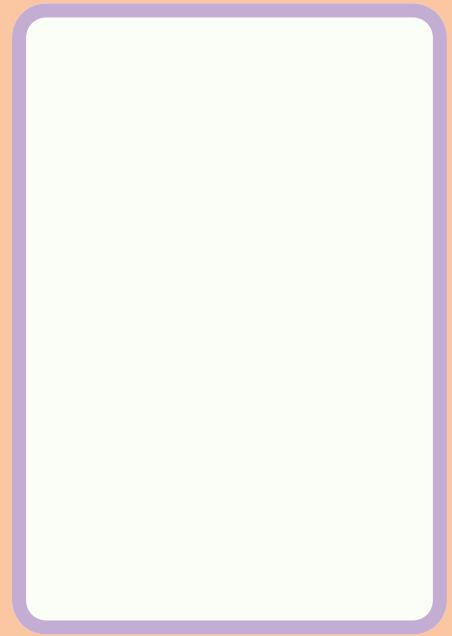
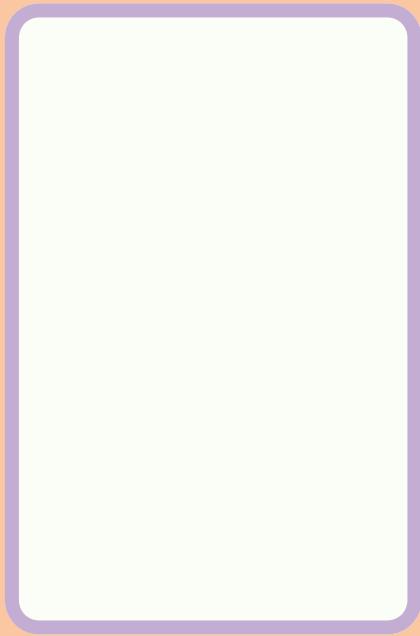
Medication



Medication







Function

Key Points



Notes

Function

Key Points



Notes

Function

Key Points



Notes

Function

Key Points



Notes



VET HELPFUL NOTES

VETERINARY ECG FLASH CARDS



VetHelpful.com
Canine Feline



P wave with no subsequent QRS complex



In second deg node. The AV

In 2nd degree also called t

In 2nd deg a more ser

In high-g

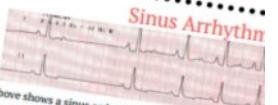
In th

beet

2nd degree AV Block



Sinus Arrhythmia



The ecg above shows a sinus arrhythmia with a rate of 100bpm. The sinus beats. This rhythm normalized with an atropine. 15 minutes later to de

Right Bundle Branch



looks like a scary VPC rhythm

Heart Arrhythmia Classification

Grade	Ventricular Arrhythmia
0	No ventricular arrhythmias
I	Unifocal, infrequent VPCs (<30/hour)
II	Unifocal, infrequent VPCs (>30/hour)
III	Multifocal VPCs
IVa	Ventricular couplets
IVb	Ventricular triplets or nonsustained ventricular tachycardia
V	R-on-T

Arrhythmias due to faulty conduction in the AV node

Partial or complete blockage of electrical activity at the AV node is AV Block (Types 1-3).
Junctional escape beats are started at the AV node.

Supraventricular arrhythmias

SVT (Supraventricular tachycardia)
Atrial Fibrillation

Ventricular Arrhythmias

Junctional escape beats arise from a high ventricular location
V Tach
Left and Right BBB
VPC's: monomorphic, (all look the same, so they come from the same hyper-exciteable focus in the ventricle)
polymorphic (VPC's are differently shaped, meaning they come from different locations in the ventricle= more sinister arrhythmia)
fusion, bigeminy, trigeminy

ECG Interpretation

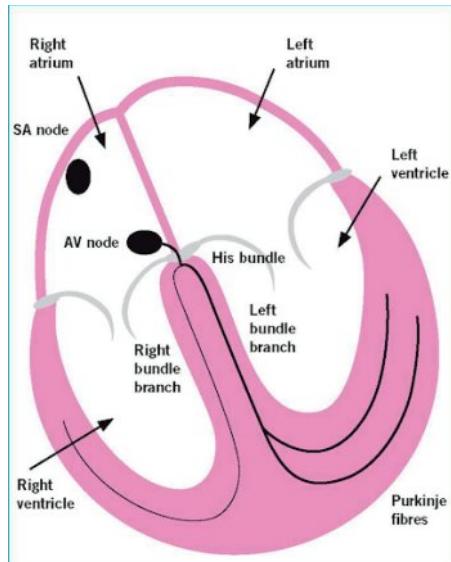
Normal heart rate ranges

Adult dog	70-160 beats/minute
Giant breeds	60-140 beats/minute
Toy breeds	70-180 beats/minute
Puppies	70-220 beats/minute
Adult cats	120-240 beats/minute

Six basic questions to help with electrocardiogram interpretation

- What is the heart rate (slow, normal, fast)?
- What is the rhythm (regular, regularly irregular, irregular)?
- Is there a QRS complex for every P wave?
- Is there a P wave for every QRS complex?
- Are they consistently and reasonably related?
- What is the morphology of the QRS complex (narrow and upright = supraventricular or wide and bizarre= ventricular)?

Heart Conduction

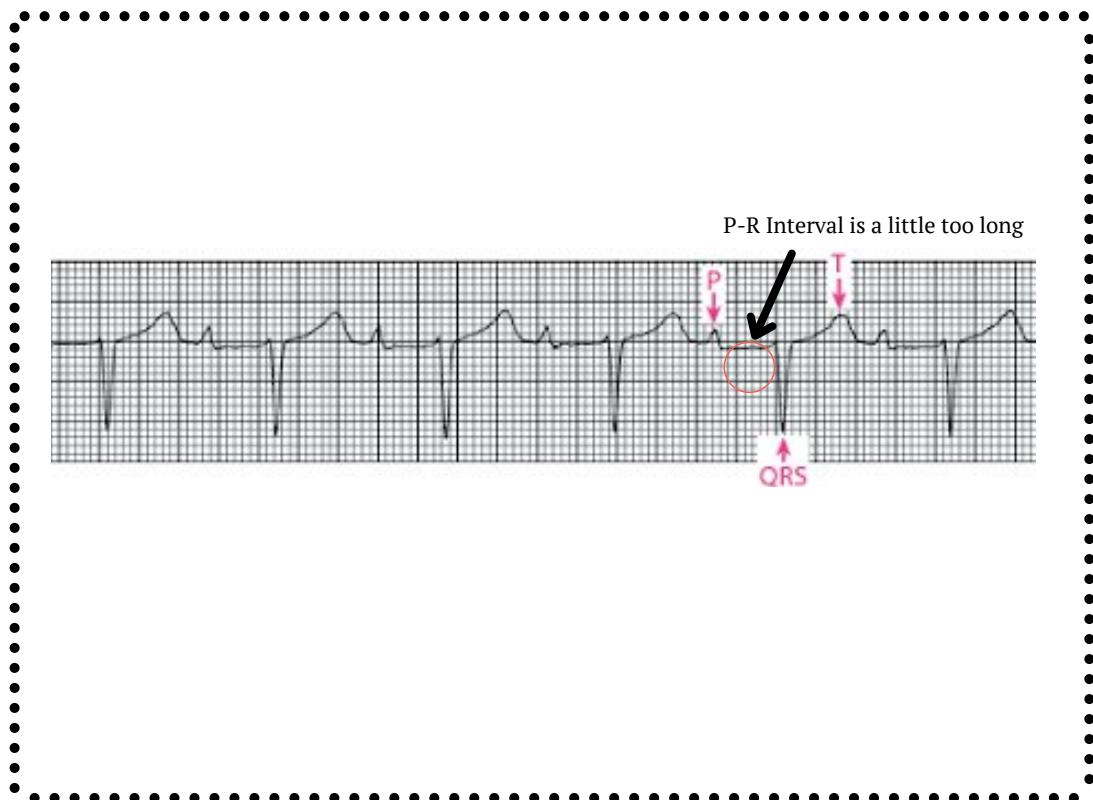


Conduction starts at the SA node in the right atrium and propagates to the AV node, down through the bundle of His and finally to the Purkinje fibers of the ventricles.

Partial or complete blockage of electrical activity at the AV node is a form of AV Block (Types 1-3).

Junctional escape beats are started at the AV node.

Premature ventricular complexes occur at various locations in the ventricles.



First degree AV Block

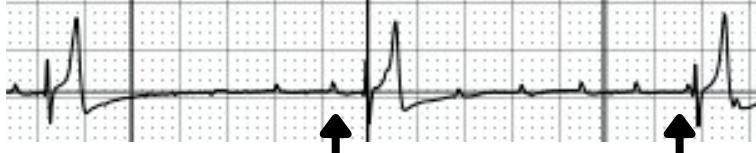
P-R Interval is a little too long



- Atrioventricular (AV) block describes an abnormality affecting conduction of the signal from the atria once it reaches the AV node. The block occurs at the level of the AV node or the bundle of His and is graded as first, second and third degree AV block
- 1st degree: In 1st degree AV block, conduction across the AV node or the bundle of His is merely slowed - the P-R interval is thus prolonged. The normal P-R interval is less than 0.14 seconds in a dog and 0.08 seconds in a cat.
- First degree block is usually benign and caused by increased vagal tone or drugs like beta blockers. It is usually reversible and does not typically require treatment.

Third degree AV Block

P waves fire along independently from the QRS complexes. P waves are unassociated from the QRS complexes. Short arrows show P waves in different positions in front of the QRS complexes.



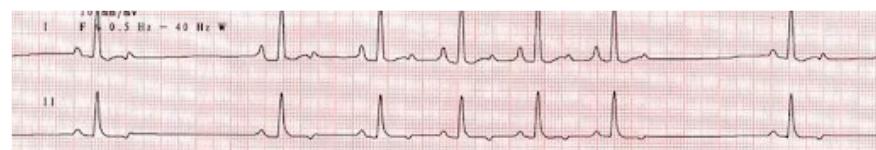
In third degree AV block all impulses are blocked. The result is a heart rate that is too slow (bradycardia) because it is generated entirely by the ventricles which have a slow rate of firing.

Fibrosis/degeneration of the AV nodes is the most common cause of AV block. Inflammation, electrolyte disturbances or toxins/medications can also interfere with normal heart conduction at the AV node.

An escape rhythm will start firing from the ventricles if there is a complete block at the AV node. The escape rhythm is much slower than the rate of firing coming from the SA node or AV nodes. **Cats with 3rd degree AV block have HR's of 80-130bpm. Dogs have a HR of 20-60bpm.**

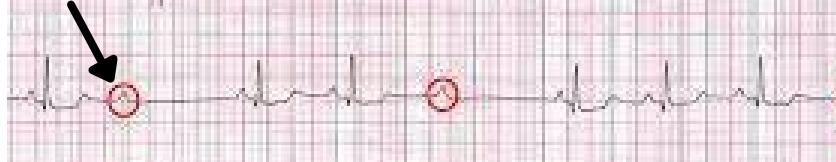
Dogs with third degree AV block have signs of lethargy , exercise intolerance and collapse. The recommended treatment is to place a pacemaker. For **cats** with advanced AV block, the most common symptoms are **labored breathing and collapse**. This may look like **seizure** activity. However, interestingly about 1/3rd of cats will have no symptoms at all when 3rd degree block is recognized.

P wave with no subsequent
QRS complex



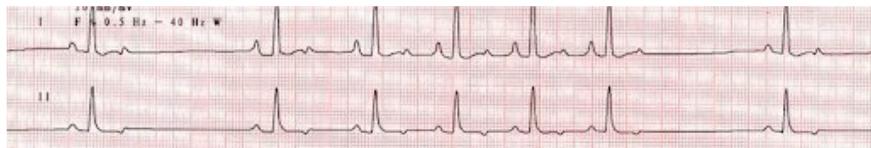
2nd degree AV Block

- P wave with no subsequent QRS complex

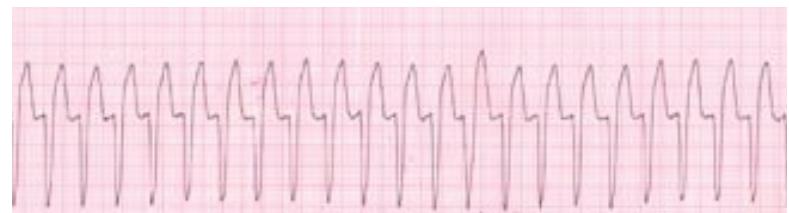
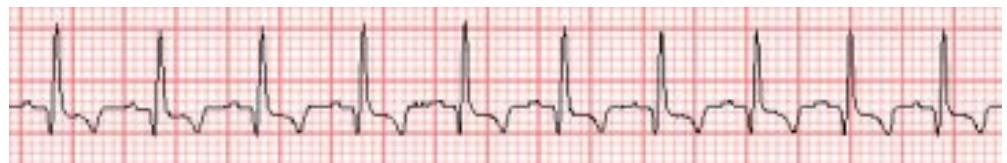


- In second degree AV block some impulses are conducted normally, while others are “blocked” at the AV node. The AV node is located at the junction between the atria and ventricles.
- In **2nd degree Mobitz type I AV block**, the **P-R interval is variable** and indicates high vagal tone (this is also called the Wenckebach phenomenon).
- In **2nd degree Mobitz type II block**, the **P-R interval is elongated but there is no variation**. This indicates a **more serious** conduction disorder.
- In high-grade second-degree AV block more impulses are blocked than are conducted.

Sinus Arrhythmia



- The ecg above shows a sinus arrhythmia with a rate of 100bpm for central beats and 60bpm for the other sinus beats. This **rhythm normalized** with an **atropine** response challenge, indicating a **sinus arrhythmia**.
- The atropine response test is performed by administering 0.04 mg/kg of atropine IV and repeating an ECG 15 minutes later to determine if the sinus bradycardia was abolished.
- An atropine response challenge can rule out a conduction disturbance. It is used to determine if there is **high vagal tone** influencing the rate of firing of the AV node to cause sinus **bradycardia** or periods of sinus arrest from sick sinus syndrome. Disease within the SA node can cause it not to discharge an impulse in a timely fashion. If the impulse is not initiated within 8 seconds, a dog will faint/collapse.
- Sick sinus syndrome is most common in West Highland White Terriers, Daschunds, Miniature schnauzers, Boxers and cocker spaniels.



Sinus Tachycardia

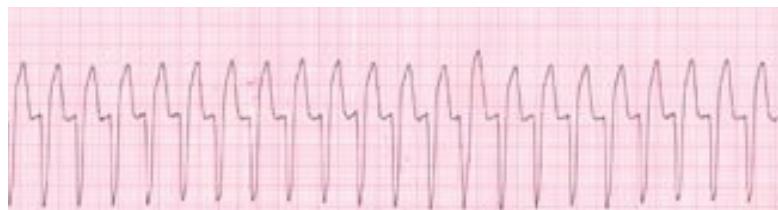


HR 180, can see p wave and t wave deflections

- **Sinus Tachycardia** is a very common rhythm disturbance in small animals due to **stress and excitement**. Unrelenting sinus tachycardia may be due to CHF and high sympathetic drive. Animals in **pain** or with **fevers** may have persistent sinus tachycardia.

- The heart rates with sinus tachycardia are typically **slower than in SVT**. Also, **P and T waves** are **easy to distinguish** in sinus tachycardia.

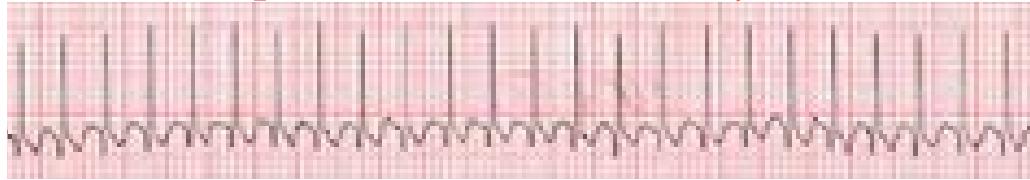
Ventricular Tachycardia



- Ventricular tachycardia significantly **decreases cardiac output** and can worsen congestive heart failure (CHF) and contribute to weakness or collapse. Sustained v-tach lasting >30 seconds is an indicator of medium to high risk for sudden death (due to development of ventricular fibrillation).
- Treatment of ventricular tachycardia indicated in an awake animal with clinical signs resulting from the arrhythmia (weakness, worsening CHF, collapse, or syncope) and in animals with sustained (> 30 seconds) and rapid (> 180-200 beats per minute) ventricular tachycardia.
- **Lidocaine is the drug of choice**, given first as a bolus and often followed up by a constant rate infusion of the drug.

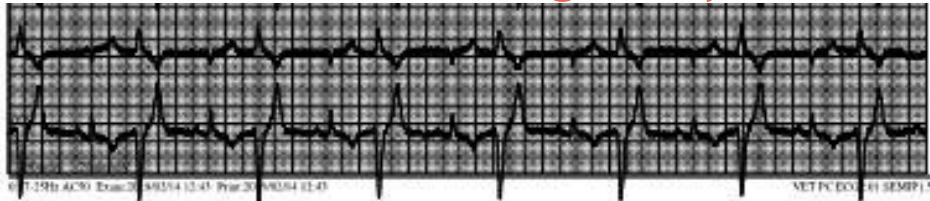


Supraventricular Tachycardia

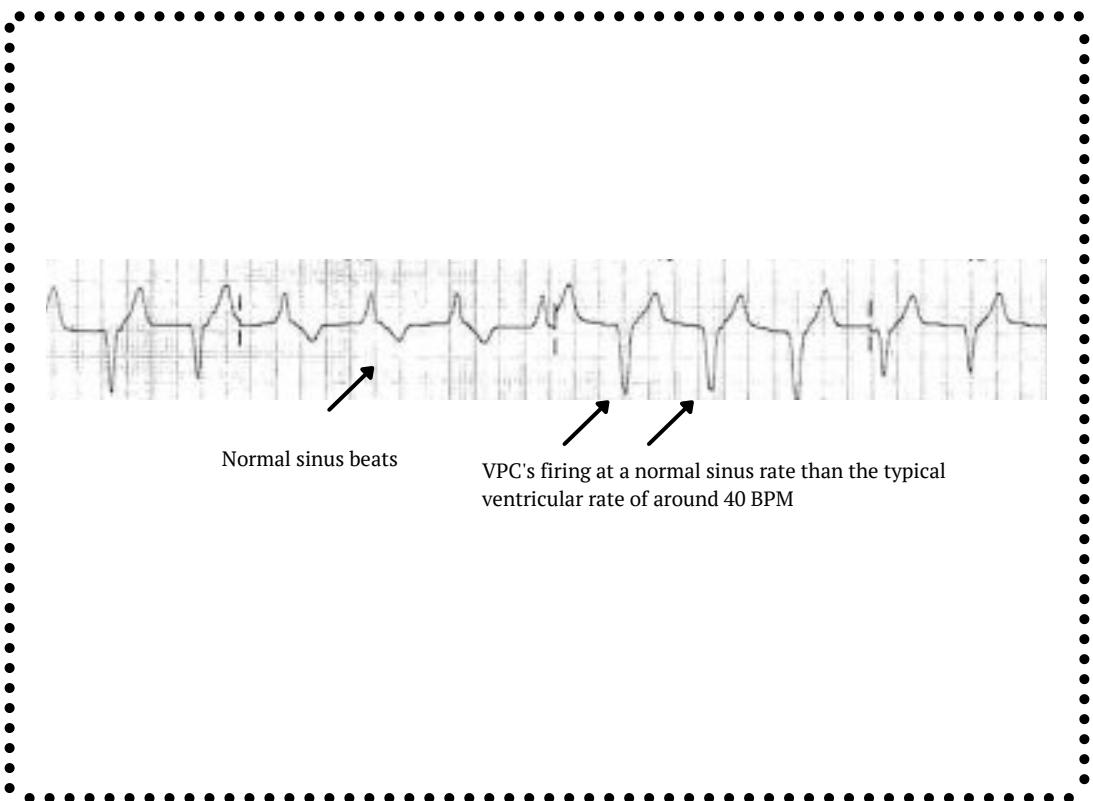
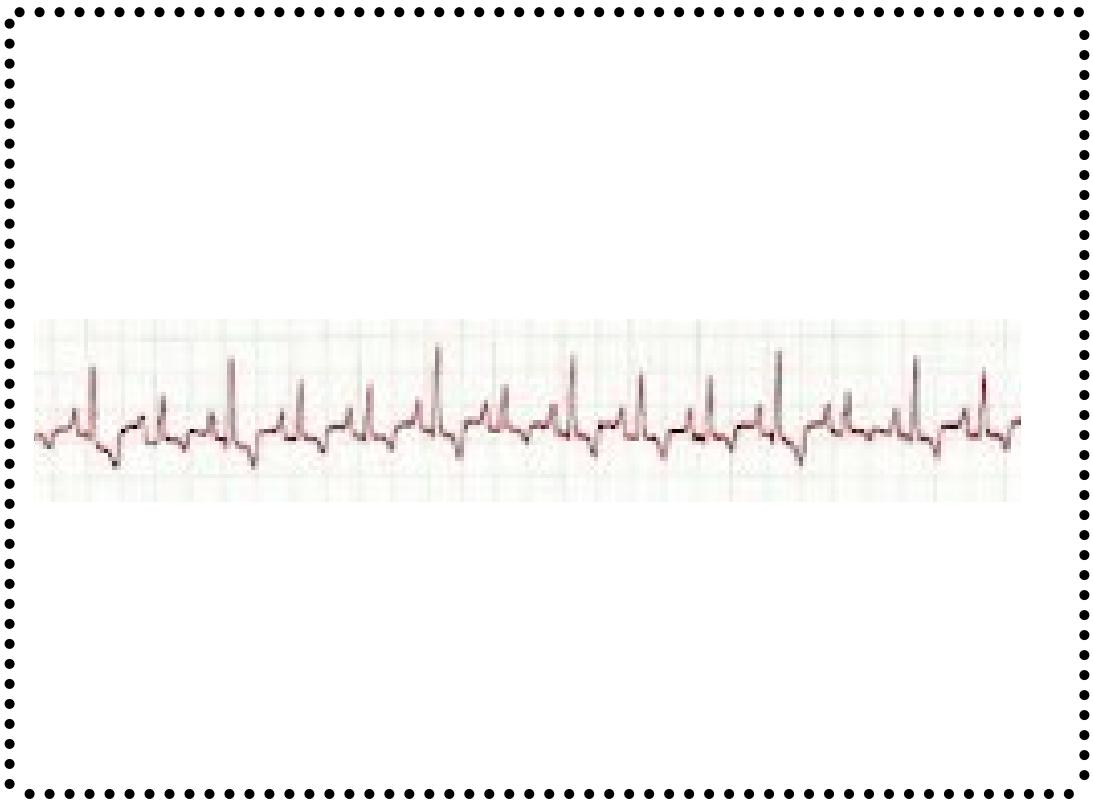


- Sustained supraventricular tachycardia (SVT) can be a life-threatening rhythm disturbance in the dog.
- This rhythm disturbance occurs most often in animals with **primary noncardiac diseases**. SVT is distinguished from Sinus Tachycardia most often by it's **higher heart rate** and because the **P and T waves cannot be distinguished**.
- **Drug therapy** is appropriate for animals with rapid supraventricular arrhythmias (**rates above 260 beats/minute in the cat or above 220 beats/minute in the dog**) as sustained tachycardia can lead to heart failure, weakness or syncope. In cases where emergency treatment is required, **IV diltiazem** can be administered in an initial 0.25 mg/kg IV bolus over 2 minutes. Subsequent 0.25 mg/kg boluses can be repeated at 15 minute intervals until conversion occurs or to a maximum dose of 0.75 mg/kg
- Causes of SVT include atrial stretch (endocardiosis, DCM), hypoxemia, renal, or GI (especially pancreatic) disease, ventricular pre-excitation syndromes or heartworm disease.

Ventricular Bigeminy



- **Bigeminy** is a **single ectopic (irregular) ventricular heartbeat** following each regular heart beat. In many cases it causes **mild or no symptoms**.
- **Nonfrequent ventricular extrasystoles are not always treated.** For example, slow idioventricular rhythms ,ventricular arrhythmias with a proven extra-cardiac cause. Address hydration status, acid-bases status, and provide oxygen if needed first (except with very fast or potentially lethal rhythms).
- In addition to exposure to drugs or toxins, other extra-cardiac causes of arrhythmias include: Hypovolemia, Acidosis, Alkalosis Electrolyte alterations, Hypoxia, Anemia, Uremia, Gastric torsion, Pancreatitis, Hypoglycemia, Splenic torsion, rupture, tumors Fever, hyperthermia, Sepsis etc.

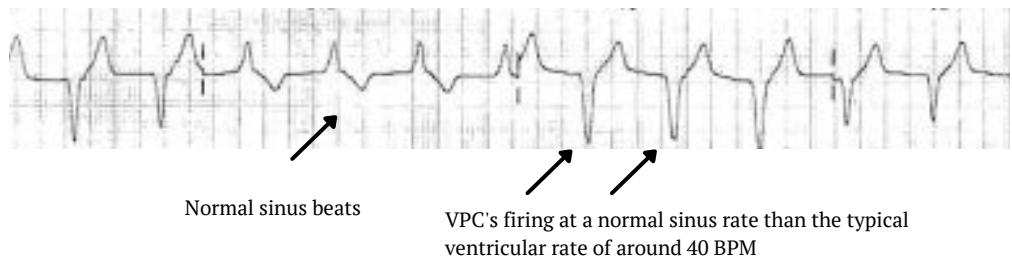


Electrical Alternans

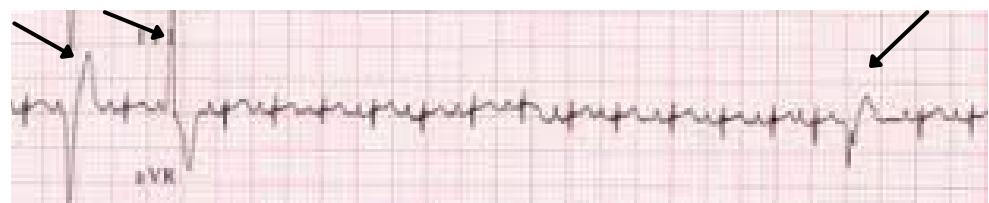


- Electrical alternans is characterized by an **alternating size of the QRS complex** and/or occasionally the T wave, which can change from beat to beat. This ECG sign generally suggests a large volume of pericardial effusion is causing the heart to move back and forth inside the pericardium. Sinus tachycardia is common with cardiac tamponade (pressure of the fluid around the heart). Low voltage (shorter) QRS complexes are frequent as well.
- Pericardial effusion is most commonly found in Golden retrievers and other medium to large breed dogs. The mean age for dogs with idiopathic pericardial effusion was 7 years and for neoplastic effusions was 9 years.
- The most common cause of pericardial effusion is neoplastic. The most common neoplastic causes are: hemangiosarcoma at the R atrial region and secondly chemodectomas at the heart base.

Idioventricular Rhythm



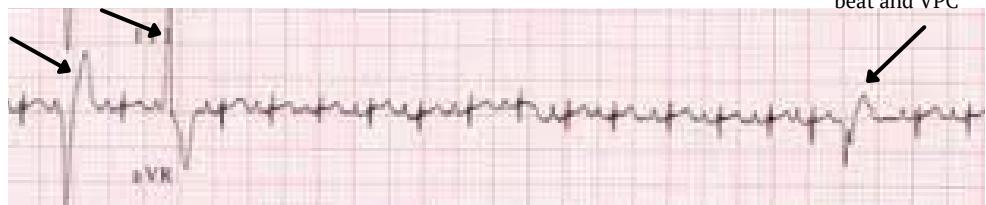
- This is a ventricular arrhythmia that occurs in **systemically ill animals** (e.g., vehicular trauma, splenic disease, GDV) at a slow rate less than 160 beats per minute. It is called an accelerated idioventricular rhythm. This rhythm **rarely poses a clinically significant risk** and usually does not create problems during anesthesia. These arrhythmias are often less frequent during anesthesia than they are in the awake animals.
- No direct anti-arrhythmic therapy is indicated if all of the following criteria apply: HR and blood pressure are within normal ranges and there are no clinical signs of hypotension are apparent (weakness, syncope) and no other premature ventricular ectopic beats are occurring.
- These ventricular beats are not premature.** They are depolarizing similar to the normal sinus rate.



Polymorphic VPC's

Two different types of VPC's

Fusion beat: simultaneous sinus beat and VPC



This ECG shows several wide bizarre complexes: VPCs. They are differently shaped (polymorphic), indicating they are complexes arising from more than one excitable foci on the ventricle. Having more than one excitable focus for VPC formation raises the concern for progression to a malignant arrhythmia.

Grading arrhythmia severity to determine if therapy is indicated:

1. **Hemodynamic alterations** are present secondary to the arrhythmia. Clinical signs include: syncope, weakness, collapse, hypotension, exercise intolerance, respiratory effort, restlessness, worsening of heart failure.
2. **Pairs or triplets of VPCs.** When there are polymorphic VPCs, there is an increased risk of progression to ventricular fibrillation.
3. **Sustained/fast ventricular tachycardia (>180-200 bpm)**
4. **R-on-T phenomenon** (R wave falling very close to the T wave of the preceding QRS complex)

Right Bundle Branch Block

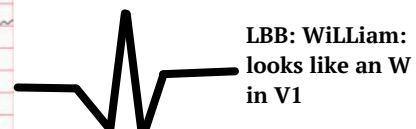


RBB: MoRRow:
looks like an M
in V1

Right bundle branch block looks like a scary VPC rhythm, but is actually a **harmless rhythm** caused by delayed electrical impulses through the right ventricle. The delayed impulses through the right ventricle causes the wave form to look more wide and bizarre. However, the **distinguishing feature** is that this wave form has a **p wave**.



In contrast to a
Left Bundle
Branch Block seen
here.



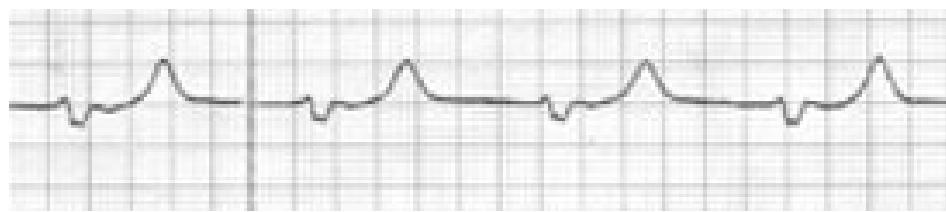
LBB: WiLLiam:
looks like an W
in V1



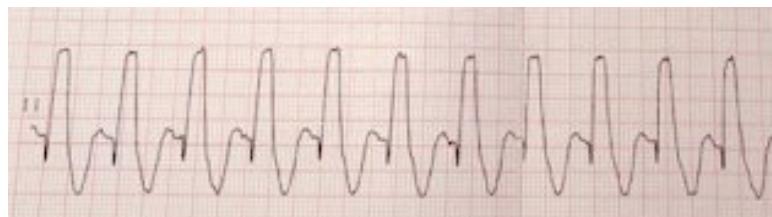
RBB



← V Tach



Left Bundle Branch Block



LBB: WiLLiam :
looks like a W in
V1



- Left bundle branch block, in contrast to a right bundle branch block, is a more serious wave form and indicates heart disease is present. Like a RBB, the LBB is delayed electrical conduction through the purkinge/HIS fiber system on the left side of the heart. It has the **opposite deflection as the RBB** (see below) and also **has a p wave** to distinguish it from Ventricular tachycardia.

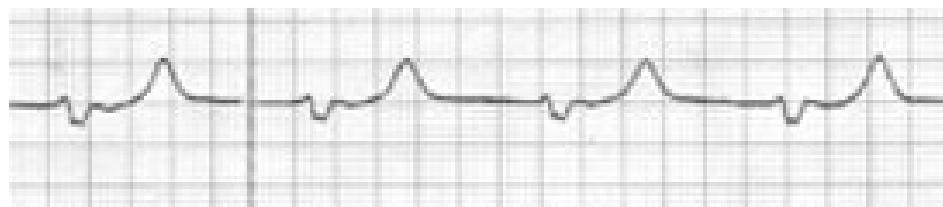
RBB



← V Tach



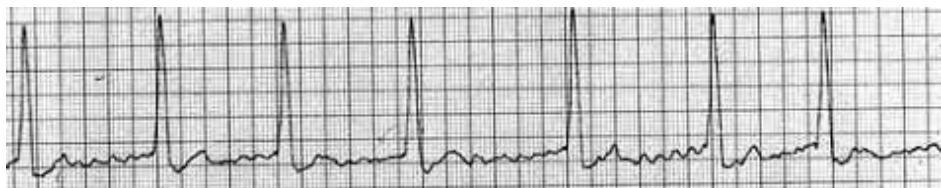
Hyperkalemia



- The ECG above shows a **lack of p waves**, a **widened QRS** and a **tall T wave** characteristic for severe hyperkalemia. Hyperkalemia is one of the most common findings in cats with urinary obstruction and, if severe, can be life-threatening and lead to a bradycardia and possible death.

- ECG changes in mild hyperkalemia include increased amplitude and narrowing of the T wave and shortening of the QT interval. Moderate hyperkalemia causes prolongation of the PR interval and widening of the QRS.

- As hyperkalemia progresses, P waves decrease in amplitude, become wide, and eventually disappear. Bradycardia may be observed, although is less pronounced in cats. The QRS may merge with the T wave creating a sine wave appearance.



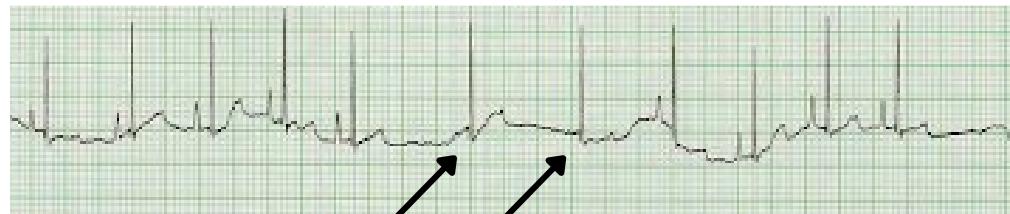
Atrial Fibrillation

F wave: bumpy baseline, lack of identifiable p waves



- Electrocardiography (ECG) is required to definitively diagnose AF. This is determined by a **lack of identifiable P waves** (an irregular bumpy baseline called an F wave) and an irregular ventricular rate characterized by a **variable R-to-R interval**. Usually, the QRS complexes are narrow and predominately upright in lead II and generally the **HR is fast**.
- A high percentage of Doberman pinschers affected with Dilated Cardiomyopathy (DCM) present with atrial fibrillation. Atrial fibrillation may develop before any other evidence of underlying myocardial disease (chamber enlargement or systolic dysfunction). These dogs should be carefully followed for the development of DCM.
- DCM in the Doberman pinschers is a very malignant form of DCM compared to other breeds. Death usually occurs due to heart failure or sudden death within 6 months of diagnosis.

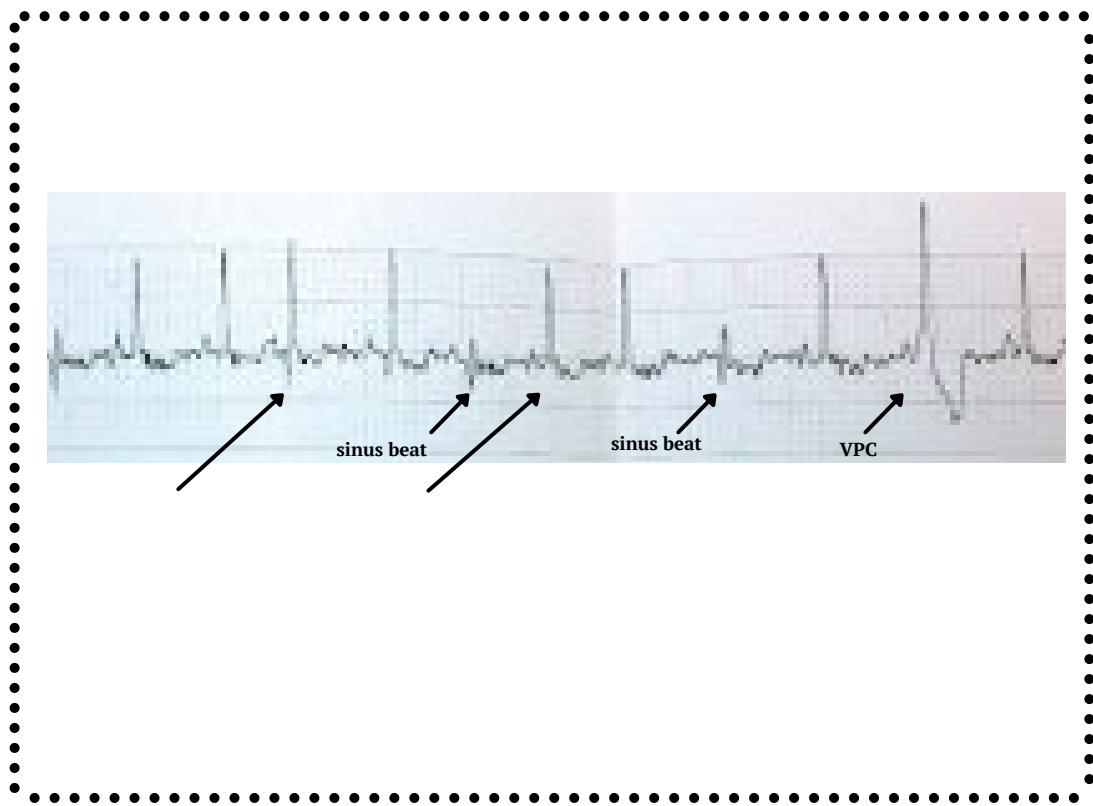
Junctional Escape Beat



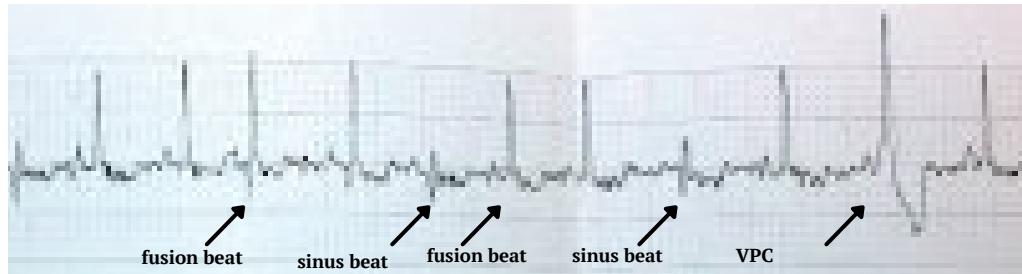
Junctional beats. Look like more normal complexes without the preceding p wave

This is a dog post-op from a GDV surgery who has a couple of junctional escape beats (the two central tall beats with no p wave preceding). Junctional beats arise from the junction between the atria and the ventricles at the Bundle of His. They have a more supraventricular look, but unlike a **supraventricular beat**, they **don't have a p wave** (because they arise from below the atria).

This rarely requires treatment.

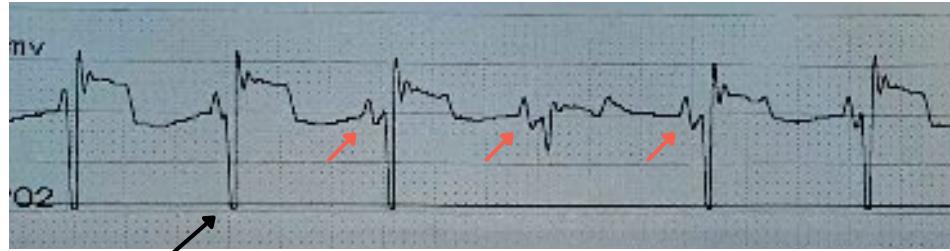


Fusion Beats



Fusion beats are a combination of a normal sinus beat merged with a VPC.

The **fusion beats** are the **intermediate** sized waveform. They indicate more VPC's are happening in this patient than immediately meets the eye. On first glance it may appear that there is only one VPC present. This Boxer dog should have a holter monitor investigation.



VPC's firing at same rate
as sinus beats

→ Red arrows show sinus beats. Can see p waves of
sinus beats landing very close to the VPC's and firing
at the same relative rate as the VPC's. Both VPC's
and sinus beats are firing independently.

Isoarrhythmic Dissociation



VPC's firing at same rate as sinus beats

→ Red arrows show sinus beats. Can see p waves of sinus beats landing very close to the VPC's and firing at the same relative rate as the VPC's. Both VPC's and sinus beats are firing independently.

Atria and ventricles are beating at the same rate by their independent pacemakers **but are not associated with each other**. Happens a lot in cats, and is **typically a benign rhythm**. Can be abolished by accelerating the rhythm with atropine. This rhythm rarely causes hemodynamic compromise.

It is **not 3rd degree AV block** because both the atrial pacemaker and the **ventricular or junctional pacemaker are firing at a relatively normal and similar rate**. It is not really understood why isoarrhythmic dissociation occurs. It can happen in healthy cats.

Isoarrhythmic Dissociation



VPC's firing at same rate as sinus beats

→ Red arrows show sinus beats. Can see p waves of sinus beats landing very close to the VPC's and firing at the same relative rate as the VPC's. Both VPC's and sinus beats are firing independently.

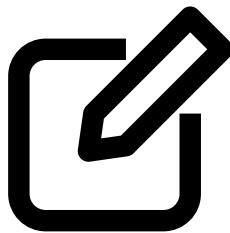
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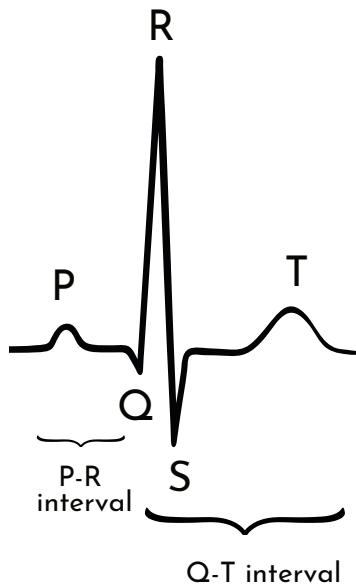
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Vet Helpful Notes

ECG Interpretation Work Book





P wave: depolarization/contraction of the atria

R wave: (QRS complex) Ventricular depolarization and contraction

T wave: repolarization of the ventricles

Conduction starts at the SA node in the right atrium and propagates to the AV node, down through the bundle of His, and finally to the Purkinje fibers of the ventricles.

Partial or complete blockage of electrical activity at the AV node is a form of AV Block (Types 1-3).

Junctional escape beats are started at the AV node or bundle of His.

Premature ventricular complexes occur from various locations in the ventricles.

A ventricular escape rhythm usually 30-50 bpm in dogs and 60-100 in cats

Junctional escape rhythms are faster in dogs, usually 60-80 bpm

Vocabulary



Sinus: originating from the SA node

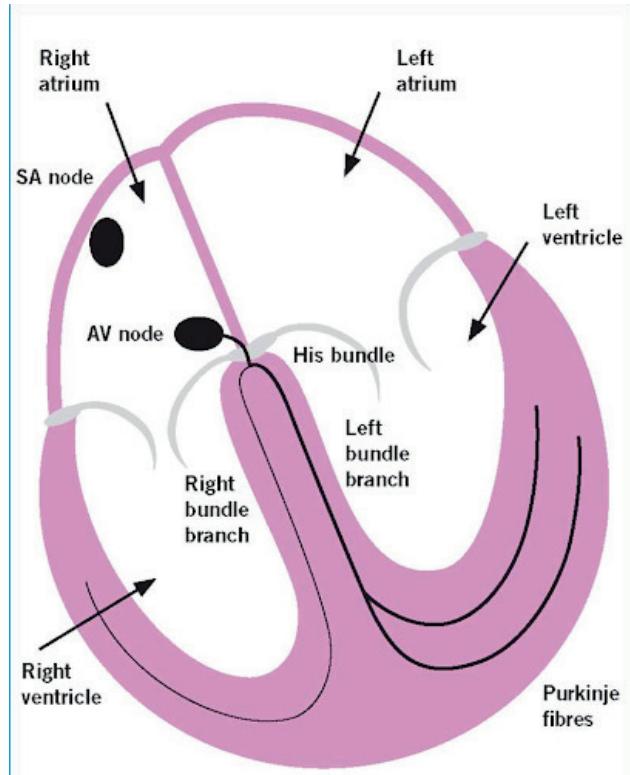
Supraventricular: coming from above the AV node, the QRS has a normal shape

Ventricular: coming from the ventricle, the QRS has an abnormal shape and is "wide and bizarre"

Premature: occurring early

Paroxysmal: sudden and occasional

Escape: occurring after a delay



ECG questions to ask:

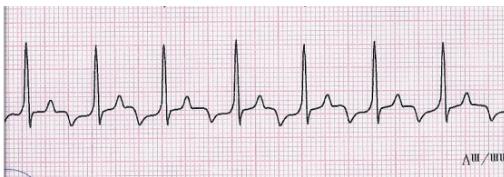
Is there a p for every QRS and a QRS for every p wave?

For example, in Type II and III AV Block you will not have a QRS for every p wave



Is the rhythm regular or irregular?

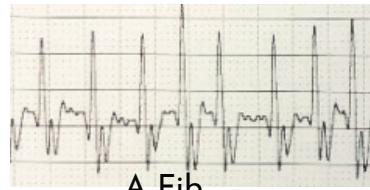
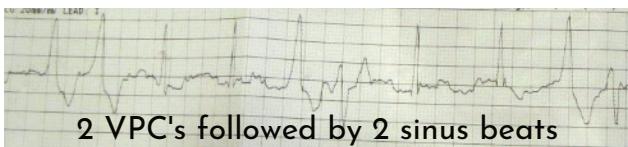
For example, a sinus tachycardia that is beating very regularly. All beats may be spaced apart regularly.



If the rhythm is irregular, is it regularly irregular or irregularly irregular?

For example, you can have an irregular rhythm with VPC's happening close together, but occurring at regular intervals- such as in bigeminy.

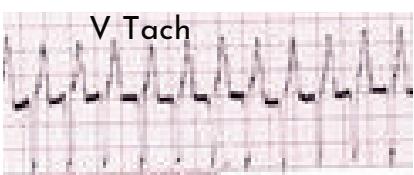
An example of an irregularly irregular ECG rhythm is atrial fibrillation



Is the rhythm too fast or too slow?

Too fast = tachycardia Too slow = bradycardia

A very common cause of bradycardia is elevated vagal tone or 3rd degree AV Block



Do the QRS complexes look narrow or large, wide and bizarre?

Narrow QRS complexes originate from above the ventricles.

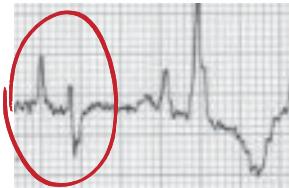
Wide and bizarre complexes originate from the ventricles.



although these look narrow here, when you see the other type of complex, you know these are actually wide and bizarre and much bigger. Therefore, they are ventricular.

normal sinus beats. They are narrow and smaller than the other wave forms

Evaluating Common Arrhythmias



Normal Heart rate

Narrow QRS



Regular rhythm



Irregular rhythm

Wide QRS



Regular rhythm

Normal sinus rhythm

Normal sinus arrhythmia

1st or 2nd deg AV Block

Accelerated idioventricular rhythm
RBB or LBB sinus rhythm

Increased Heart rate

Narrow QRS



Regular rhythm



Irregular rhythm

Wide QRS



Regular rhythm

Sinus Tachycardia

Supraventricular Tachycardia (SVT)

Atrial flutter

Atrial fibrillation

Ventricular Tachycardia (VTach)

RBB or LBB sinus tachycardia

Slow Heart rate

Narrow QRS



Regular rhythm



Wide QRS



Regular rhythm

Sinus bradycardia

Junctional escape rhythm

Ventricular Escape Rhythm



If you have a patient under anesthesia and see this come up on your monitor.
The rhythm is very SLOW.

- Is there a p wave for every QRS?
- Is there a QRS for every p wave?
- Do the p waves seem associated or disassociated from the QRS complexes?
- What is this rhythm?
- What is the treatment for this rhythm?



If you have a patient under anesthesia and see this come up on your monitor. The rhythm is very SLOW.

- Is there a p wave for every QRS? NO
- Is there a QRS for every p wave? NO
- Do the p waves seem associated or disassociated from the QRS complexes?

They seem disassociated from the QRS complexes. In other words, the p waves march along at a pretty regular rate, but irrespective of where the QRS complexes are showing up.

- What is this rhythm?

This is 3rd Degree AV Block

3rd degree AV block means there is no communication between the atria and the ventricles. So, the heart is basically contracting at a slow ventricular escape rate. The ventricular escape rate in dogs is very slow at 2-40 bpm. In cats, it's a little higher at 60-80 bpm and sometimes higher.

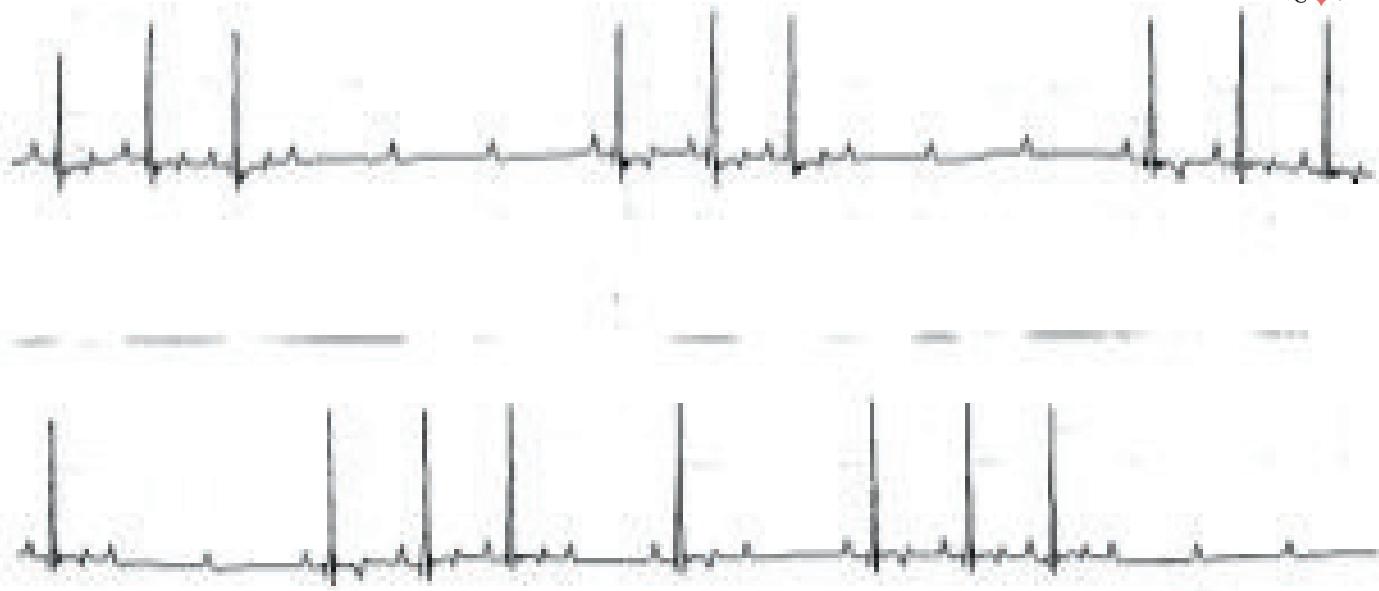
FACT dogs with 3rd degree block will often have syncope (fainting spells)

But, cats, being cats--- are typically ASYMPTOMATIC. That is presumed to be because of the intrinsically higher HR set by the ventricular pacemaker (bundle of HIS) and because of their sedentary nature.

If you hear a very slow heart rate, even without an arrhythmia, you might want to check an ECG just to see.

- What is the treatment for this rhythm?

The treatment for 3rd degree AV block is a pacemaker. A pacemaker is an implanted device that consists of a small generator implanted under the skin and is attached to a pacing lead that travels to the ventricle and is embedded there. The pacemaker can sense if the heart is firing at a correct rate. It steps in to trigger a heartbeat if there is too long of a delay.

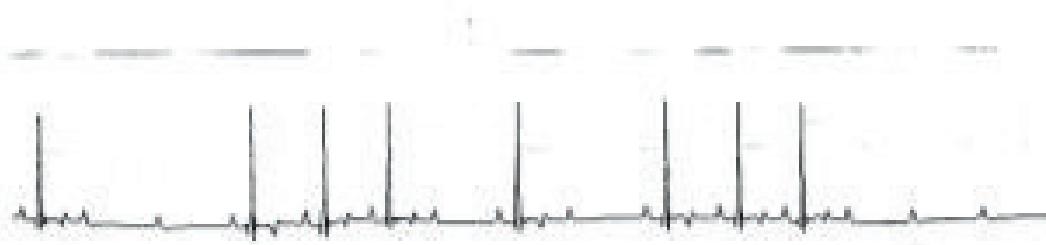


Is there a p wave for every QRS?

Is there a QRS for every p wave?

What is this rhythm?

What is Vagal Tone and how can this play a role in this arrhythmia?



Is there a p wave for every QRS?

YAH

Is there a QRS for every p wave?

Nope. In fact, there are multiple p waves without a QRS. This means, the electrical conduction from the atria is being blocked at the AV node before it can signal the ventricles to contract (and produce a QRS complex). P waves that produce a delayed QRS complex (ie an elongated P-R interval) is FIRST degree AV Block.

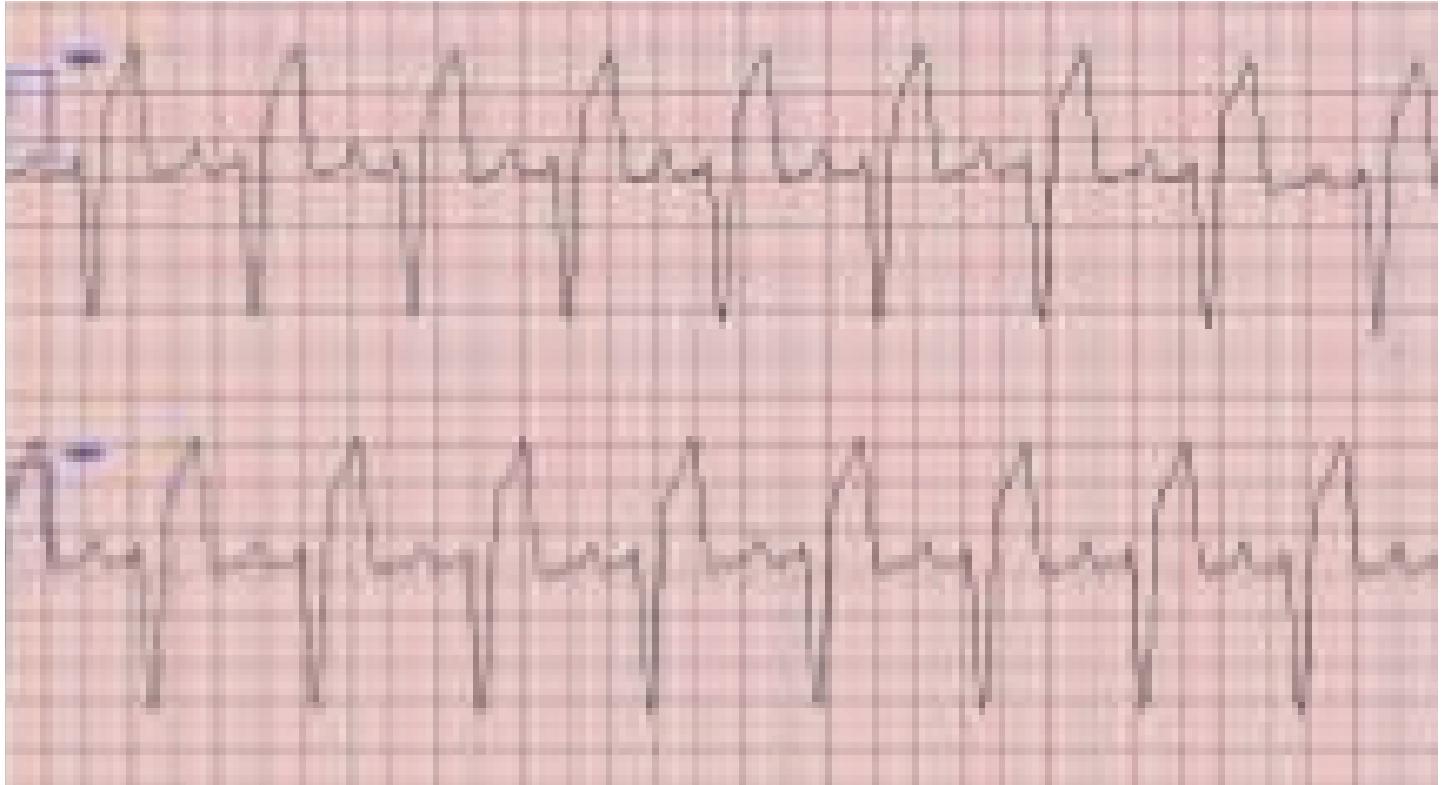
What is this rhythm? SECOND degree AV Block.

P waves that are sometimes blocked and there are NO associated QRS complexes is evidence of a SECOND degree AV Block. Second degree AV block might be benign. It can be associated with increased vagal tone. Sedation drugs can increase vagal tone for example. This why 2nd degree AV block can be seen in some sedated/anesthetized patients.

What is Vagal Tone? It is the parasympathetic enervation to the heart. The parasympathetic nervous system controls "REST and DIGEST". When there is increased vagal tone, it acts directly on the SA node (where the electrical activity of the heart starts). High vagal tone SLOWS down the heart rate (REST) and also has other effects on the body to promote a resting system (digestion etc.).

To check if the 2nd degree block is just due to high vagal tone, you can do an atropine response challenge. If you use atropine, it raises the heart rate and blocks the vagal tone to the heart. The 2nd degree block should resolve in this case.

P waves which NEVER communicate through the AV node and are NEVER associated with QRS complexes is THIRD degree AV Block. This is NOT GOOD. If you're basically relying on the last pacemaker in the ventricles to keep the heart beating. This ventricular escape rhythm is the slowest of the pacemakers (SA node is the original pacemaker, then there is a pacemaker in the AV node and the purkinge fibers of the ventricles).

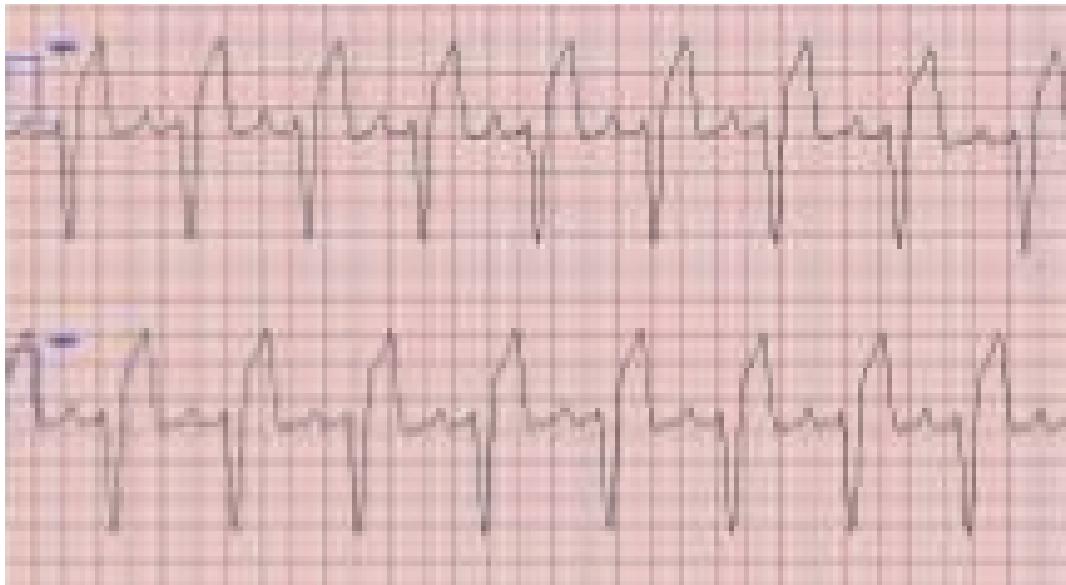


This dog has a normal heart rate but this rhythm looks very funky/scary. I know the ECG looks blurry. Just squint and lemme know these answers!

Is this rhythm regular or irregular? (not the wave forms, are the wave forms happening in a regular or irregular fashion?)

Is there a p wave for every QRS? Is there a QRS for every p wave?

Can you guess the rhythm diagnosis?



This dog has a normal heart rate but this rhythm looks very funky/scary. I know the ECG looks blurry. Just squint and lemme know these answers!

Is this rhythm regular or irregular? (not the wave forms, are the wave forms happening in a regular or irregular fashion?)

This looks like a regular rhythm

Is there a p wave for every QRS? Is there a QRS for every p wave?

YAH all around

Can you guess the rhythm diagnosis?

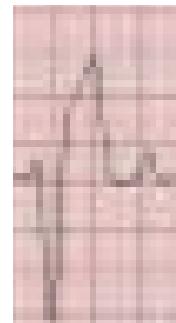
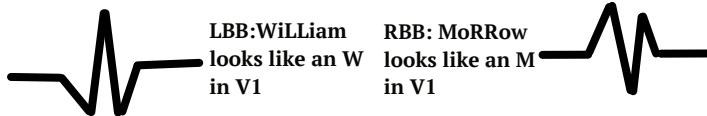
This is a RIGHT BUNDLE BRANCH BLOCK. The QRS complexes are wide and bizarre. But what's in front of every QRS? A P WAVE. So, the p wave is initiating the electrical activity. It's just moving through the purkinje fibers in the right ventricle very slowly, compared to the left ventricle. This wide bizarre complex looks sort of like an "M" with a prominent downward deflection.

What treatment is needed?

RBB are generally considered benign. No treatment is needed.

LBB is a concerning arrhythmia.

"William Morrow"



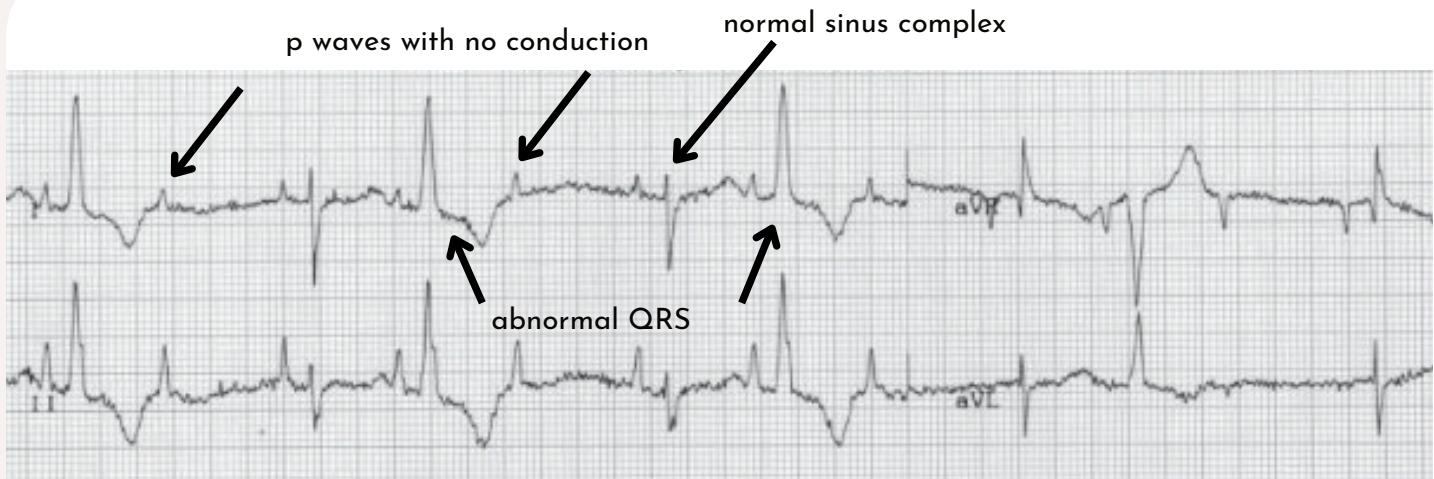


Point out the normal sinus complexes. Then point out the abnormal complexes.

Is there a p wave for every QRS?

Is there a QRS for every p wave?

Can you say what's going on in this strip?



ECG strip from a 16 yo cat

Point out the normal sinus complexes. Then point out the abnormal complexes.



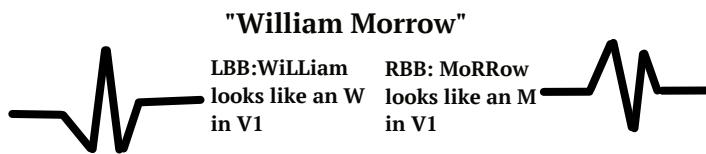
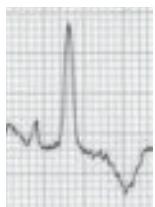
Is there a p wave for every QRS? YAH

Is there a QRS for every p wave. Not always. That's 2nd degree block

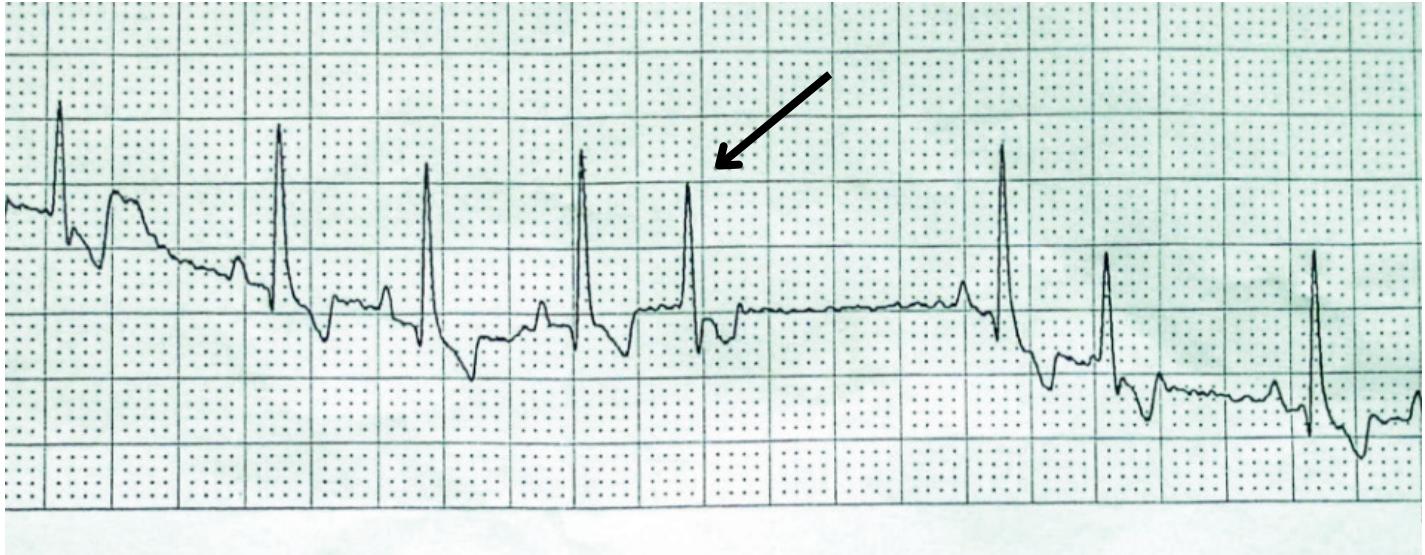
Can you say what's going on in this strip?

Because there IS a p wave in front of the ventricular looking beat, that means this is a bundle branch block. BUT, this is a LEFT bundle branch block. A LBB looks more like a W with a tall middle deflection.

A LBB is delayed conduction through the purkinje fibers of the left ventricle. There's also some 2nd degree AV Block.



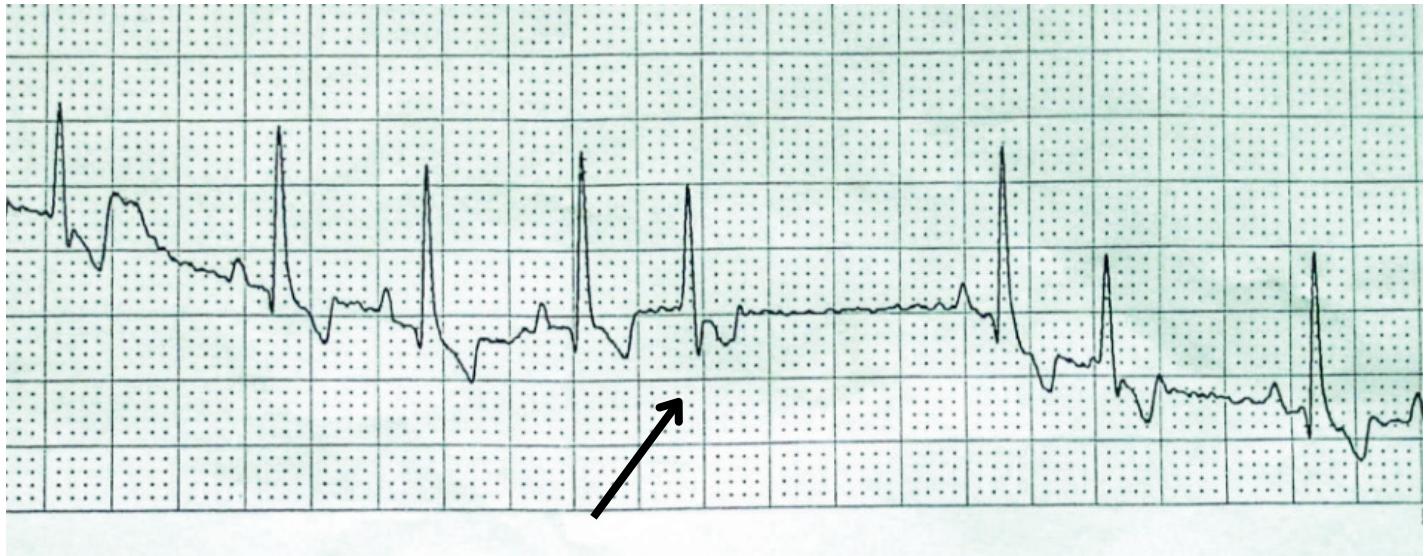
So this ECG shows 2nd degree AV block AND a left bundle branch block. The rate of firing of the p waves is 200 bpm, so an atropine challenge is NOT necessary. (This is not 2nd degree block due to high vagal tone). There is likely degenerative disease of the heart's electrical conduction system. If it deteriorates further, a pacemaker might be needed.



What type of complex is the arrow pointing towards?

Is there a p wave for every QRS? A QRS for every p wave?

Is this beat occurring prematurely?



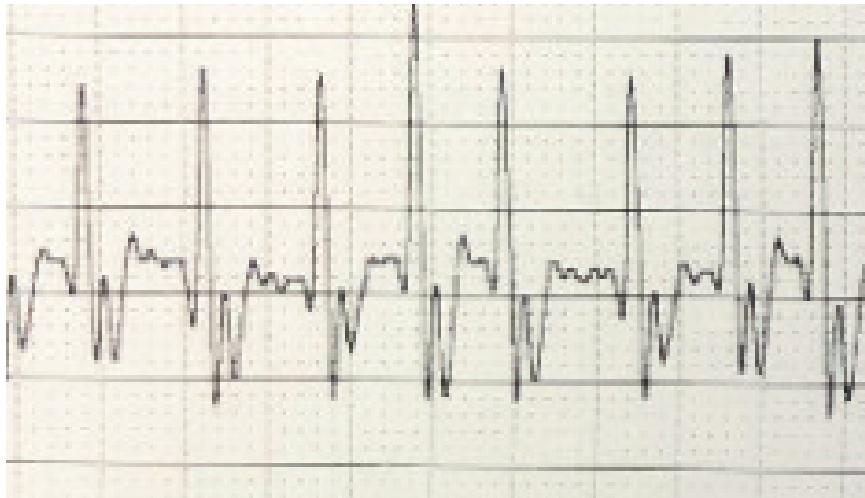
What type of complex is the arrow pointing towards?

A junctional premature beat (or a beat arising from the AV node). There is no p wave, but the complex looks supraventricular.

An atrial premature contraction looks like a normal sinus beat, but the p wave is unusual- it can be taller, upside down or wider . Because this looks like a normal supraventricular beat, but has no p wave , we know it's not derived from the atria. It's also not wide and bizarre which would mean its not derived from the ventricles. It's just at the junction between the atria and the ventricles. There is a QRS for every p wave, but not a p wave for every QRS.

This beat is premature (you can see it pops up too close to the last complex compared to the normal sinus beats). It is occurring in a 9 year old SF boxer with an enlarged left atrium.

This stretched out atrium is leading to some abnormal electrical activity.



Is the rhythm regular or irregular?

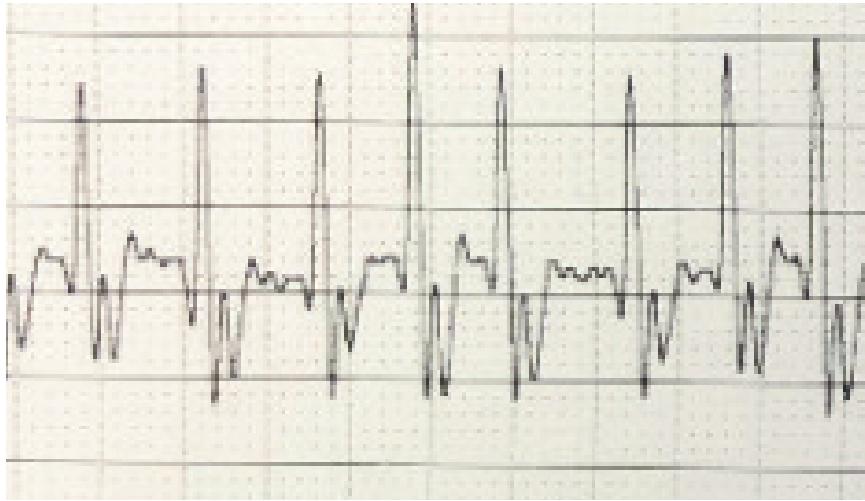
If the rhythm is irregular is it regularly irregular? Or is it irregularly irregular?

Do you see p -waves for every QRS?

Can you name this rhythm?

HINT: This is a fast HR. The patient has an audible arrhythmia that sounds like "shoes in the dryer".

How do you treat this rhythm? What is a common disease that causes this rhythm?



Is the rhythm regular or irregular?

The R-R interval varies, so the rhythm is irregular

If the rhythm is irregular is it regularly irregular? Or is it irregularly irregular?

It is irregularly irregular

Do you see p-waves for every QRS? It's hard to distinguish the p waves clearly- the baseline looks bumpy

Can you name this rhythm?

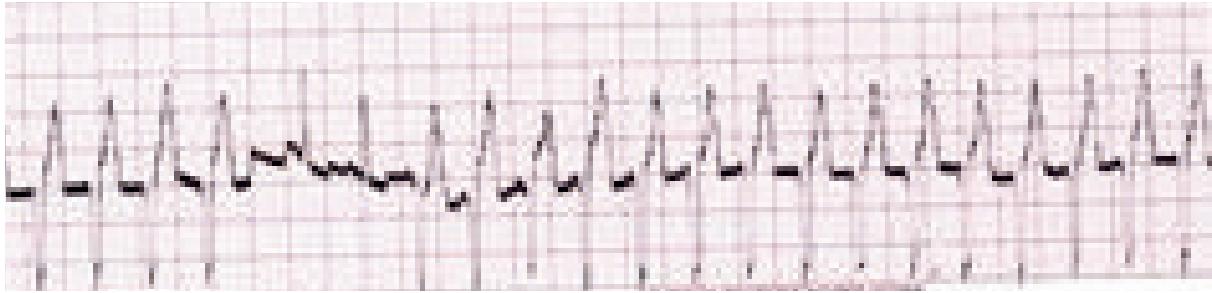
This is atrial fibrillation

HINT: This is a fast HR. The patient has an audible arrhythmia that sounds like "shoes in the dryer".

How do you treat this rhythm? What is a common disease that causes this rhythm?

You treat A fib by slowing down the HR with betablockers, diltiazem or digoxin.

Dilated cardiomyopathy is a common condition that causes A fib because the conduction is altered when the heart wall muscle becomes stretched and thin.

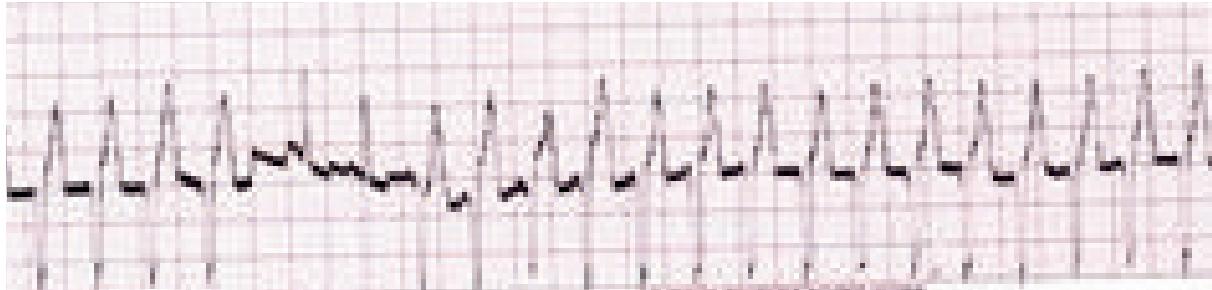


Sorry you have to squint on this one. But, you can tell right away there are two types of rhythms here. This is a very fast rhythm.

What are the two smaller complexes? Do they originate from the atria or the ventricles?

What are the rest of the complexes? Do they originate from the atria or the ventricles?

What rhythm is this? Do you need to treat this rhythm? If so, how?
HINT: they are very fast. The patient has evidence of poor perfusion: low BP, pallor.



Sorry you have to squint on this one. But, you can tell right away there are two types of rhythms here. This is a very fast rhythm.

What are the two smaller complexes? Do they originate from the atria or the ventricles?

These two complexes are sinus beats that originate from the atria.

What are the rest of the complexes? Do they originate from the atria or the ventricles?

The rest of the complexes are ventricular in origin, premature ventricular beats.

What rhythm is this? Do you need to treat this rhythm? If so, how?

HINT: they are very fast. The patient has evidence of poor perfusion: low BP, pallor.

Yes, rhythm is V tach (ventricular tachycardia). The rhythm broke for a couple of beats and you can see the normal sinus complexes. It went right into V Tach again. The treatment of choice is IV lidocaine.



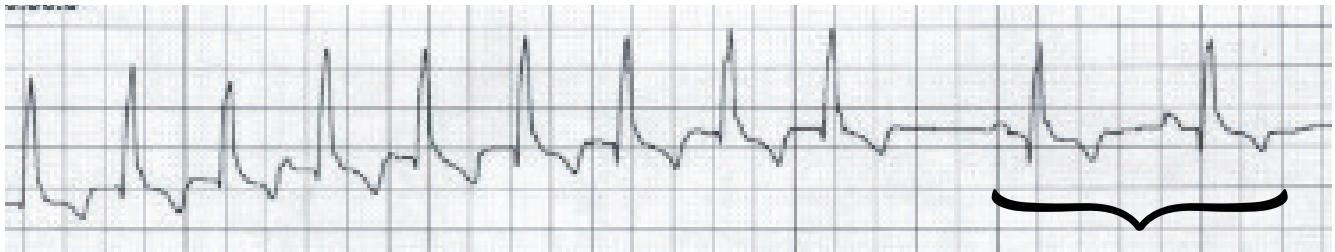
If this is a normal sinus rhythm (NSR), what is the run of complexes preceding the NSR here?

HINT:

Is the rhythm faster or slower than the NSR?

Can you make out p waves before every QRS?

Are the complexes originating from the atria or the ventricles?



If this is a normal sinus rhythm, what is the run of complexes preceding the NSR here?

HINT:

Is the rhythm faster or slower than the NSR?

This rhythm is faster than the sinus beats following this run of beats. The R-R interval preceding the NSR is smaller, therefore the rhythm is faster. It is SVT (supraventricular tachycardia)

Can you make out p waves before every QRS?

It's hard to see the p waves before every complex. In fact, the t waves almost look like they could be a p wave in the faster set of sinus beats.

Are the complexes originating from the atria or the ventricles? These are originating from the atria. They have a similar size/shape as the sinus beats.

This rhythm diagnosis is a run of supraventricular tachycardia. It starts and then abruptly stops. It would be a good idea to place a Holter monitor to continuously evaluate an ECG tracing to see how many times in a day there are runs of SVT. If there are a lot of episodes, then diltiazem would be a good choice to treat this patient.



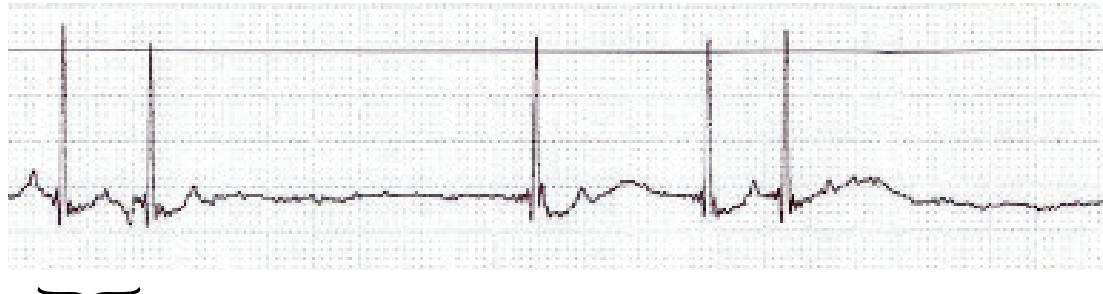
Two normal sinus beats followed by a LONG pause

What are the two beats that occur right after the long pause?

IE, where do they originate from? At the atria or the ventricles?

What's missing from the complex? Is the rate of these two beats faster or slower than the two preceding normal beats?

What is the diagnosis? HINT this is a Schnauzer.



Two normal sinus beats followed by a LONG pause

What are the two beats that occur right after the long pause?

These are junctional escape beats. They look like a supraventricular beat without a p wave.

IE, where do they originate from? At the atria or the ventricles?

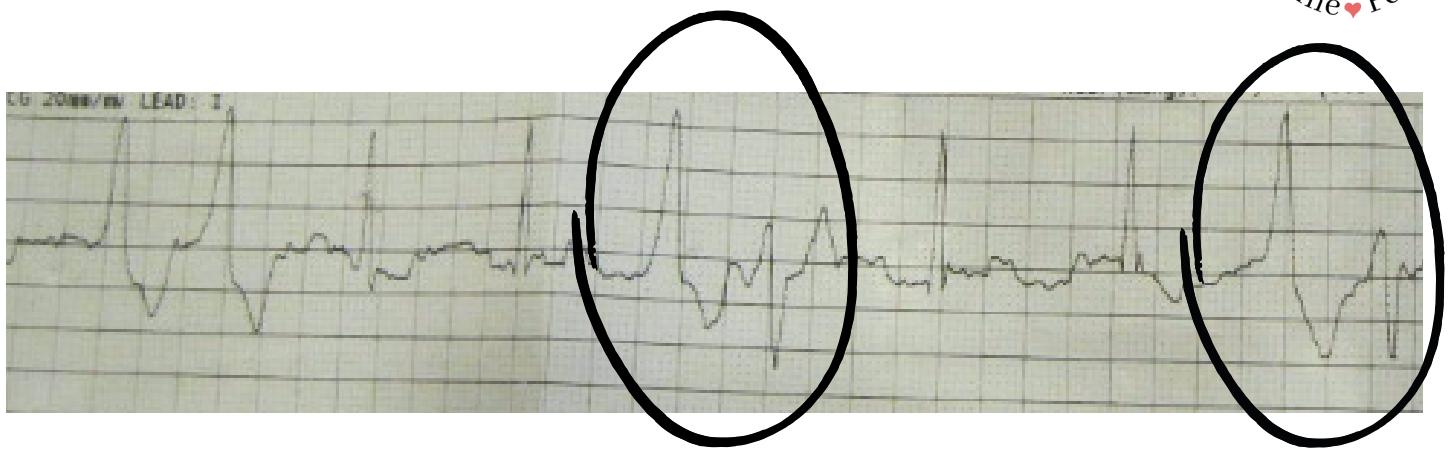
These originate in between the atria and the ventricles, at the junction. They are "junctional beats".

What's missing from the complex? Is the rate of these two beats faster or slower than the two preceding normal beats?

What's missing is the p wave. The rate is slower between the two junctional beats sandwiched between the normal sinus beats. This is because they are junctional "escape" beats that arise from the AV node of the bundle of His. They take over to initiate a beat when there has been too long a delay from the SA node. Their rate of firing is slower than the AV node but faster than a ventricular escape rhythm.

What is the diagnosis? HINT this is a Schnauzer.

This is Sick Sinus Syndrome. It is characterized by a "brady-tachy" (slow-fast) tracing. You see long pauses and then initiation of the rhythm by the junctional escape complexes. Following these two junctional escape beats are spurts of normal or premature atrial beats. These dogs eventually develop clinical symptoms of exercise intolerance, lethargy and syncope (fainting). Ultimately a pacemaker will be recommended for this patient.



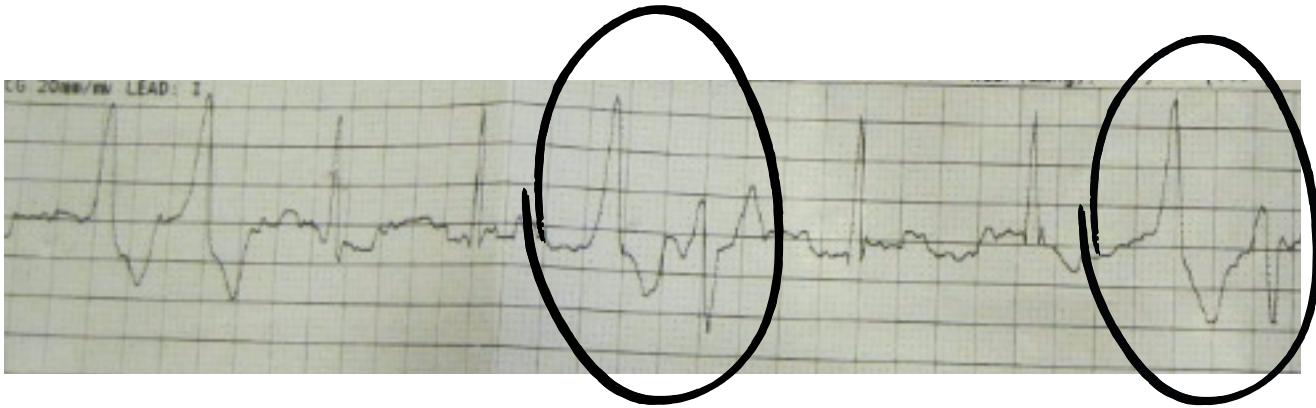
What types of complexes are happening between the circled beats?

Are they ventricular or supraventricular?

What do the paired circled complexes look like? Are they ventricular or supraventricular?

Do the two complexes that are within each circle look the same or different than each other?

Do you know the name for these paired beats that look different from each other but occur repeatedly?



What types of complexes are happening between the circled beats?

In between the circled beats are normal sinus complexes.

Are the circled beats ventricular or supraventricular?

The circled beats are both ventricular. They look wide and bizarre compared to the normal sinus beats. This rhythm strip is showing multifocal VPC's. These are VPC's that look different. (ie one big and one smaller VPC in this case).

Do the two complexes that are within each circle look the same or different than each other?

They look different than each other.

Do you know the name for these paired beats that look different from each other but occur repeatedly at regular intervals?

This is called bigeminy. ("Bi" means two). There is one normal beat followed by an abnormal beat. In this case there is a normal beat followed by a pair of VPC's. Multifocal VPC's mean that there are VPCs that arise from different parts of the ventricle and therefore look different from each other. Multifocal VPC's indicate a more concerning arrhythmia is present. Investigation and treatment is warranted.

Antiarrhythmics

Some Block Potassium Channels

Class I: Sodium (SOME)

Class II: Beta Blocker (BLOCK)

Class III: Potassium Channel Blocker (POTASSIUM)

Class IV: Calcium Channel Blocker (CHANNELS)

(Class V: Work by unknown mechanism)

Class I: Lidocaine, Mexilitine

Class II: Atenolol, Propranolol

Class III: Sotolol, Amiodorone

Class IV: Diltiazem

Class V: Digoxin

Lidocaine/Mexilitine
Ventricular Arrhythmia

Atenolol/Propranolol
V tach, Feline HCM

Sotolol, amiodorone
A fib, Vtach

Diltiazem
SVT

Digoxin
A fib

Medical Uses

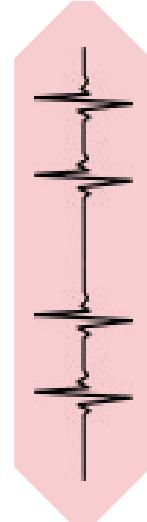
Lidocaine: blocks Na channel to prolong the action potential and slow the HR.

Atenolol: Beta 1 blocker that causes the heart to beat slower and less forcefully

Sotolol, amiodorone: Prolongs refractory period of action potential.

Diltiazem: Prevents calcium influx into cardiac cells to inhibit cardiac contractions.

Digoxin: slows AV node.



Lidocaine

Class I antiarrhythmic

Na channel blocker

Xylocaine

- Used most often in cardiac disease for animals in V Tach. Given IV
- Can also be used as a local, regional and topical anesthetic.

Considerations

- Combines with inactive fast Na channels to inhibit recovery and repolarization.
- Only effective IV due to high first pass effect orally
- Contraindicated in SA, AV block, caution liver disease and use in cats



Mexilitine

Class I antiarrhythmic

Na⁺ channel blocker

Mexitil

- Oral antiarrhythmic with similar effects as lidocaine
- Used to treat V tach and PVC's
- Often used with sotalol in dogs
- Needs to be given 3 times a day

Considerations

- Extreme caution 2nd or 3rd degree AV block
- Caution dogs with MDR1 mutation
- GI distress, (give with food), dizziness



Digoxin

Cardiac glycoside

Class V

Lanoxin

- Used primarily for heart rate control
- in A fib
- Found naturally in Foxglove
- Often combined with diltiazem
- Inhibits Na/K pump on cardiac muscle causing refractory AV node

Considerations

- Extreme caution with kidney disease and heart failure
- Can easily be over-dosed cause toxicity, must monitor blood levels
- Contraindicated in cats with HCM



Atenolol

Class II antiarrhythmic

Beta blocker Beta 1 selective

Tenormin

- Primarily used for ventricular hypertrophy and tachyarrhythmia in small animals
- Fairly Beta 1 selective so safe for animals with asthma

Considerations

- Contraindicated with bradycardia
- Caution with CHF, CKD
- Can cause lethargy, hypotension, diarrhea



Diltiazem

Class IV antiarrhythmic

Ca⁺ channel blocker

Cardizem, Dilacor

- Used most often to treat SVT and A fib
- To control A fib is used alone or with digoxin
- Inhibits cardiac muscle contractions by preventing calcium entry into cardiac myocytes

Considerations

- Contraindications: hypotension, AV block.
- Can cause GI side effects: vomiting, diarrhea, anorexia



Sotalol

Class III antiarrhythmic

Non selective Beta Blocker and K⁺ channel blocker

Betapace

- Used to treat V tach
- Most often used in boxers with arrhythmogenic cardiomyopathy to suppress ventricular ectopy

Considerations

- May cause fatigue/dizziness, nausea and vomiting
- Can be tried in patients refractory to lidocaine and procainamide
- Beta blocking activity is 30% that of propranolol



Propranolol

Class II antiarrhythmic

Beta blocker

Inderal

- Used for acute treatment of APC's, VPC's, SVT
- Can be used to treat thyroid storm in cats

Considerations

- Contraindicated with CHF unless secondary to tachyarrhythmia
- Adverse effects bradycardia, lethargy, hypotension, bronchoconstriction
- Best if used IV; has poor oral bioavailability-so other oral beta blockers are preferred

Amiodorone

Class III antiarrhythmic

K⁺ channel blocker mostly some Na⁺ channel blocker and beta blocking properties

Pacerone

- Used for refractory V tach and prevent recurrence of A fib
- Because of the high possibility of side effects, typically only used for refractory V tach

Considerations

- Adverse effects: hepatopathy, immune dyscrasias, skin reactions
- Possible to have substantial side effects

Atropine

Parasympatholytic

Anticholinergic, antidote, Tx bradycardia

atropine sulfate

- Often used as part of pre-anesthetic protocol to reduce respiratory secretions
- Used during CPR
- Used for bradycardia, SA arrest
- Used to treat organophosphate and carbamate toxicity, muscarinic mushroom toxicity by reducing SLUD signs

Considerations

- ileus, obstructive GI disease: can reduce GI motility
- Contraindicated with tachycardia, narrow angle glaucoma, HCM

Blood Pressure Reduction

Vasodilators

Nitroprusside
Sildenafil
Pimobendan
Hydralazine

Calcium Channel Blocker

Amlodipine

Ace Inhibitors and ARBs

Enanlapril
Benazepril
Telmisartan

Alpha 1 Receptor Blockers

Prazosin
Phenoxybenzamine

Pimobendan

Ionodilator

Indicated for both preclinical heart disease and CHF

Vetmedin

- It's uses improves survival from CHF and chronic valvular disease when used with furosemide vs furosemide and enalapril
- Increases time to first onset of CHF when incorporated into therapy
- Extends survival when given to pre-clinical DCM Dobermanns

Considerations

- Inhibits PDE-III to increase calcium sensitivity and improve cardiac contractions Also has vasodilatory effects.



Sildenafil

Vasodilator and Phosphodiesterase inhibitor

Treats pulmonary hypertension

Viagra

- Used primarily to treat pulmonary hypertension which results in syncope and right heart failure in dogs
- Marketed as a generic, Revatio, for pulmonary hypertension and as a brand name, Viagra, for erectile dysfunction in people

Considerations

- Don't give to patients on organic nitrates like nitroglycerin
- Caution hypotension, fluid depletion, severe left ventricular outflow obstruction



Phenoxybenzamine

Alpha adrenergic antagonist: ends in -osin, ine

Non selective alpha blocker

Dibenzyline

- Used mostly in small animal medicine to relax the urethra when there is urethral obstruction
- Also used for pheochromocytoma for vasodilation

Considerations

- Adverse effects: hypotension, rebound hypertension, increased IOP, tachycardia
- Slow onset of action: can take a few days

Nitroprusside

Vasodilator

Can only be given IV

Nitropress

- Potent systemic vasodilator
- Used primarily to treat acute left sided heart failure
- Do not confuse with nitroglycerin (sound alike issue)

Considerations

- Blood pressure must be continuously monitored
- Can cause severe hypotension and cyanide toxicosis

Hydralazine

Vasodilator

Interferes with calcium movement

May decrease cardiac output and cause reflex tachycardia

Apresoline

- Treat systemic hypertension
- Reduce high blood pressure by vasodilation.
- Used primarily for CHF treatment for valvular heart disease

Considerations

- Not used as commonly as other vasodilators like Ace inhibitors.
- Caution: severe renal disease, intracerebral bleeding, auto-immune disease
- Often used with beta-blockers to combat reflex tachycardia

Prazosin

Alpha 1 receptor blocker

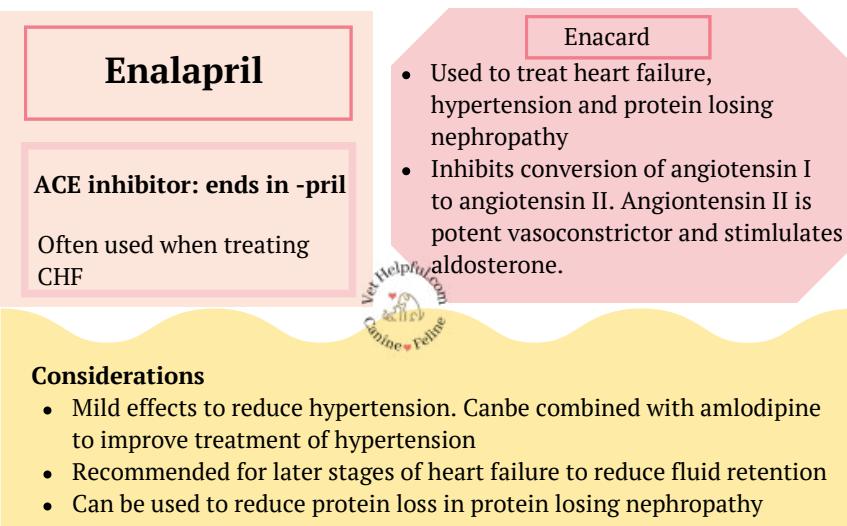
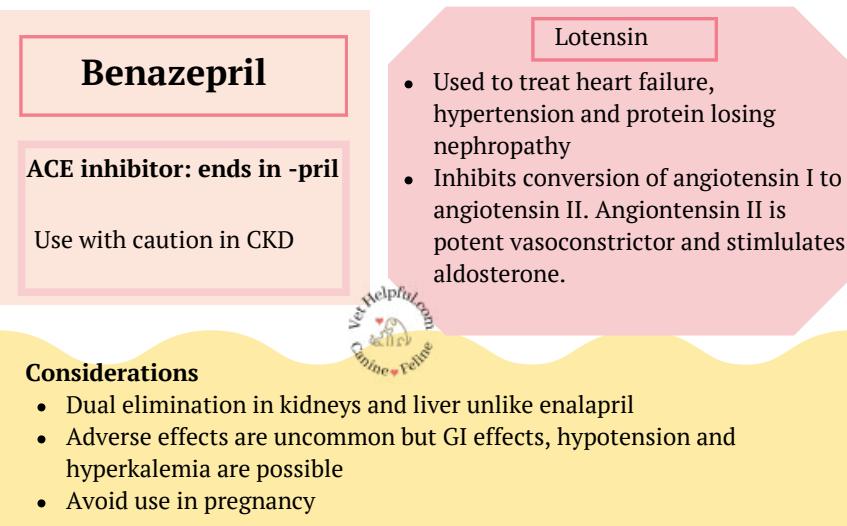
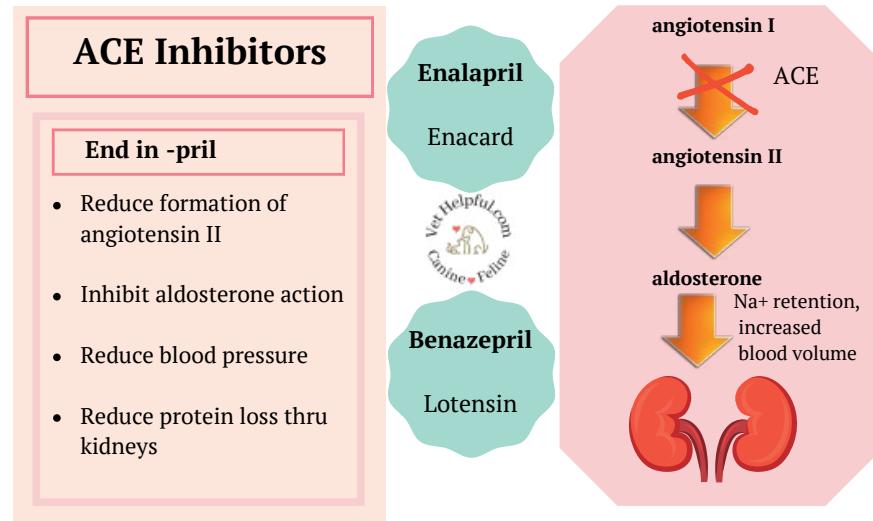
Hypotension, dizziness, lethargy and GI effects

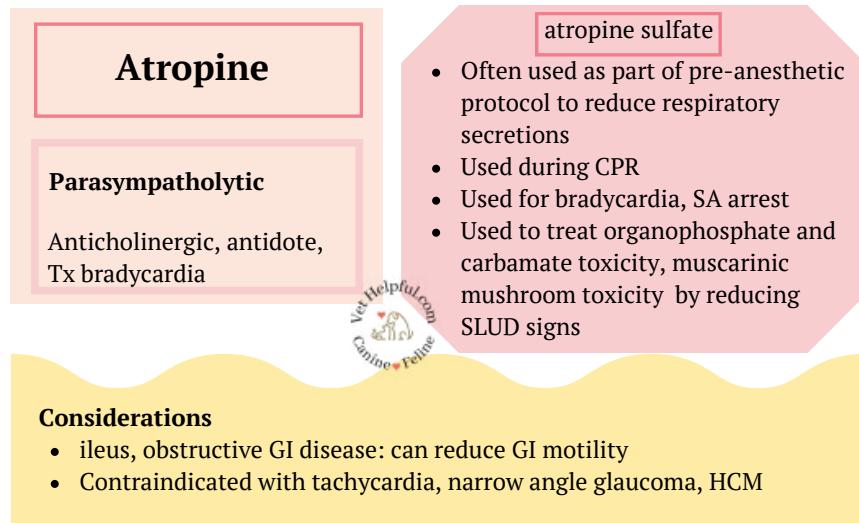
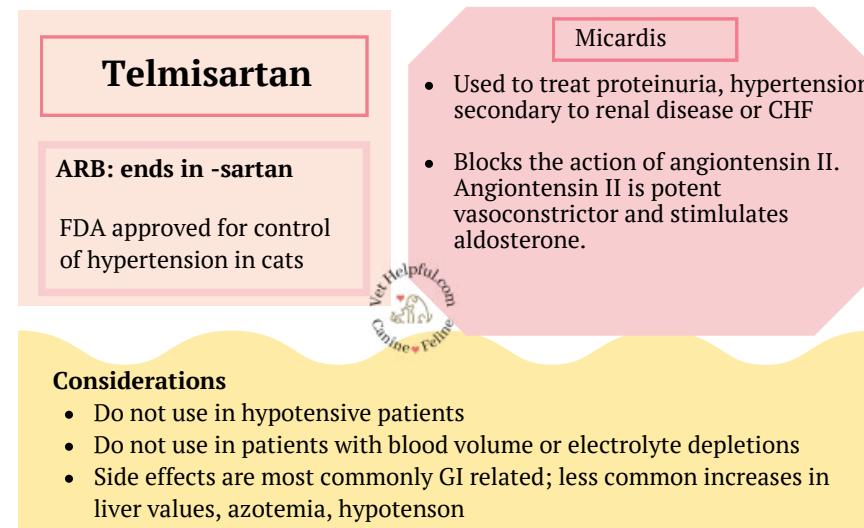
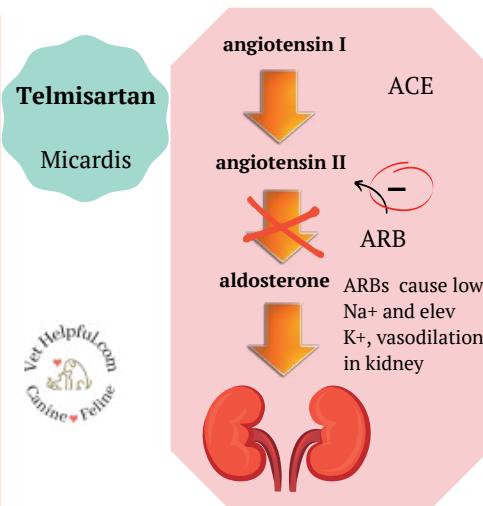
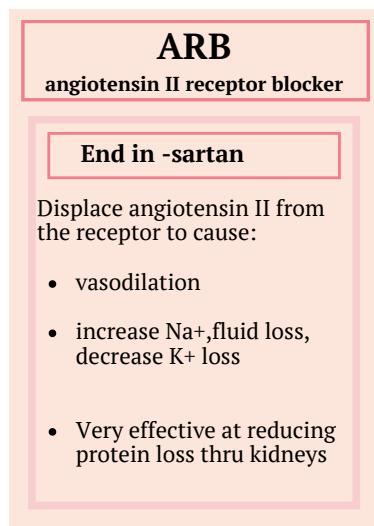
Minipress

- Used commonly to reduce feline urethral sphincter tone in urethral obstruction
- Alpha 1 blockade relaxes smooth muscle

Considerations

- Most commonly used in cats to treat functional urethral obstruction
- Can be used in dogs for reflex dyssemia
- May also be used to treat CHF, systemic hypertension or pulmonary hypertension in dogs





Amlodipine

Calcium Channel Blocker

May decrease cardiac output

Norvasc

- Treat systemic hypertension
- Decreases calcium influx in cardiac and vascular smooth muscle: Vasodilation

Considerations

- Drug of choice for hypertension in cats
- Adverse effects: hypotension, bradycardia and in dogs can cause gingival hyperplasia which can resolve after discontinuing the medication.

Blood Pressure Support

Norepinephrine
Dobutamine

Alpha Blockers

End in: -osin, -ine

Non-selective alpha blockers
Phenoxybenzamine

Selective alpha-1 blockers
Prazosin

Selective alpha-2 blockers
Mirtazapine
Yohimbine

Alpha Receptors

Alpha-1

Alpha-2

Alpha Agonists cause

- Blood vessel constriction
- Pupil dilation
- Urethral sphincter tone
- Analgesia
- Anxiolytic
- Sedative

Dobutamine

Sympathomimetic

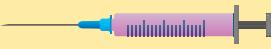
**Synthetic catecholamine
that activates B1 receptors
on the heart**

Dobutrex

- Causes dose dependent increases in cardiac contractility and ejection fraction to increase blood pressure
- Has a very short half life so must be given as a CRI

Considerations

- Can cause arrhythmias at high doses
- May be ineffective in patients who recently received a beta blocker



Norepinephrine

**Sympathomimetic and
alpha adrenergic agonist**

**Used to manage
hypotension**

Levoron

- Acts on alpha 1 and alpha 2 receptors to cause vasoconstriction
- Naturally occurring catecholamine that stimulates the sympathetic nervous system

Considerations

- Can cause peripheral and visceral vasoconstriction which may reduce blood flow to major organs
- Used to treat hypotension unresponsive to fluid resuscitation



Diuretics

Furosemide

Spironolactone

Hydrochlorothiazide

Furosemide

Loop diuretic

Most commonly used diuretic for CHF

Lasix

- Blocks the $\text{Na}^+/\text{K}^+/\text{2Cl}^-$ transporter on the loop of henle to inhibit 25% of NaCl reabsorption and promote diuresis.
- May cause pre-renal dehydration but this can often be corrected.
- Can cause electrolyte imbalances

Considerations

- Do not use with cardiac tamponade as it can worsen cardiac output
- Do not use with an aminoglycoside as dehydration can promote kidney injury



Spironolactone

Aldosterone antagonist

Potassium sparing diuretic

Aldactone

- Used as potassium sparing diuretic and adjunctive treatment for heart failure or ascites
- Should not be substituted for furosemide in CHF
- Competitively inhibits aldosterone at the distal tubule



Considerations

- Contraindications: hyperkalemia, addison's, acute or significant kidney disease
- Use precautions when handling: hazardous drug- use gloves

Hydrochlorothiazide

Thiazide diuretic

Used for nephrogenic DI, Ca Ox urolith prevention, and diuretic for CHF

Microzide

- Enhances excretion of Na^+ , Cl^- and water at DCT of kidney to enhance diuresis
- Decreased calcium excretion so this helps with reduction of Ca Ox stone
- Has anti-hypertensive effects



Considerations

- Caution severe kidney disease, electrolyte imbalances
- Caution Diabetes mellitus, SLE
- Do not use with hepatic impairment or hypercalcemia



Blunt Sharp Scissors



Used to cut suture. Less expensive than Mayo or Metzenbaum scissors which should only be used to cut tissue.

Mayo Scissors



Used to cut connective tissue, muscle, fascia

Tenotomy Scissors



Used for delicate surgery

Metzenbaum Scissors



Used for fine dissection and cutting; they have a long handle to blade ratio

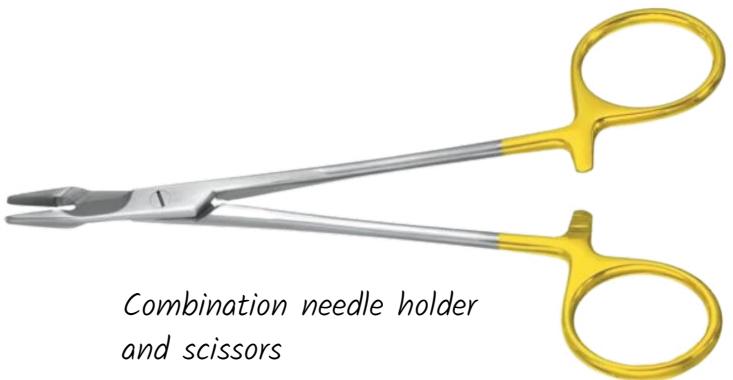


Lister Bandage Scissors



*Used for cutting into
bandage layers without
harming tissues underneath*

Olsen Hegar Needle Holder



*Combination needle holder
and scissors*

Mayo Hegar Needle Holder

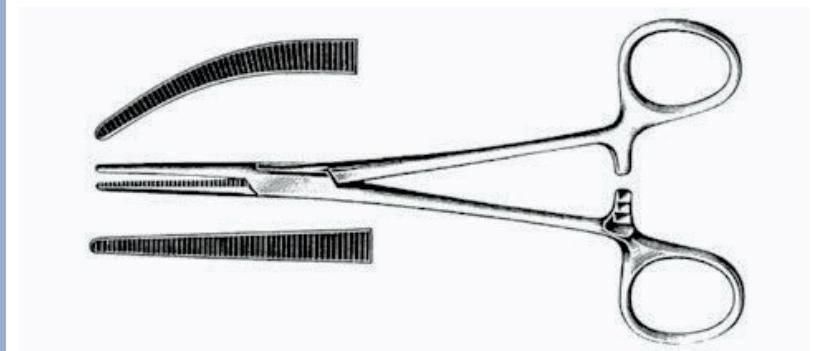


*Mayo Hegar needle holders have
NO scissors*

Iris scissors



*Originally developed
for ophthalmic surgery*



Debakey Vascular Clamp



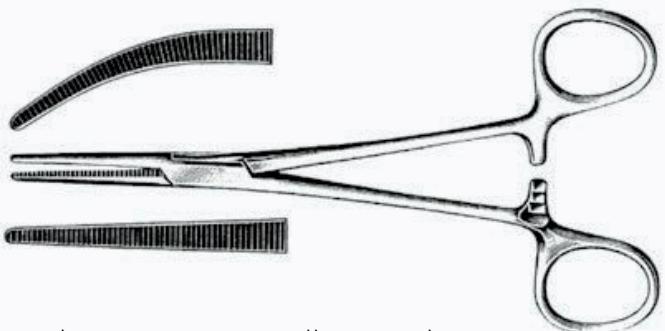
to gently clamp a vessel to control hemorrhage

Halsted Mosquito forceps



Used to compress smaller vessels

Crile Forceps

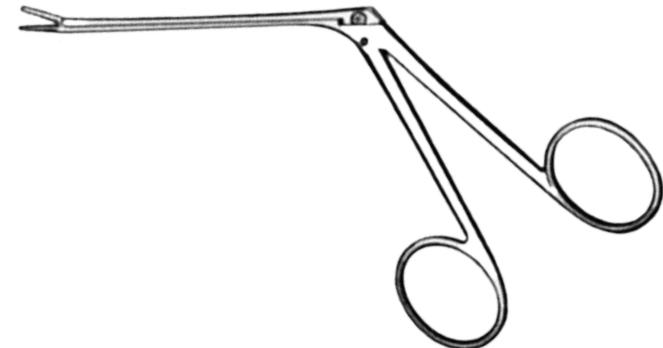
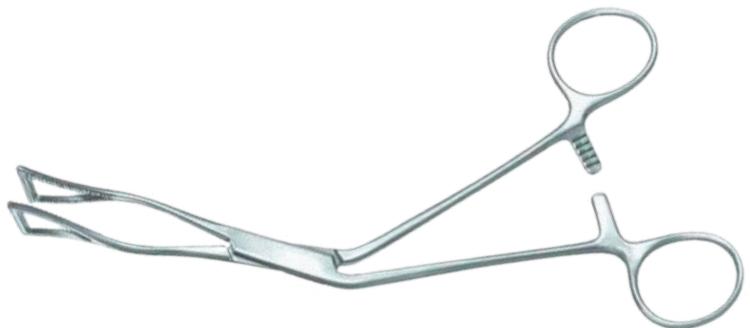


*Used to compress smaller vessels.
Has a fully serrated jaw, compared to Kelly*

Kelly Forceps



*Used to compress smaller vessels.
Longer clamping jaws than mosquito forceps. Does not have a fully serrated jaw like Crile Forceps*

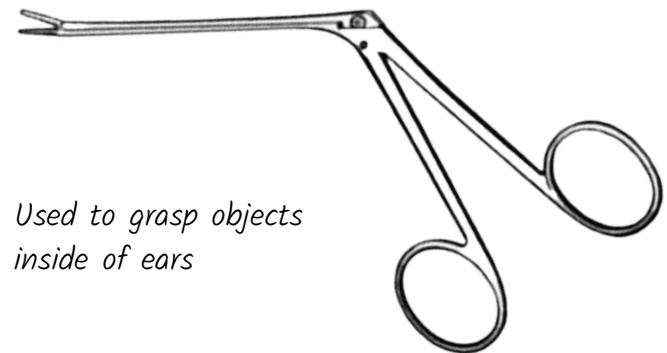


Satinsky Vascular Clamp



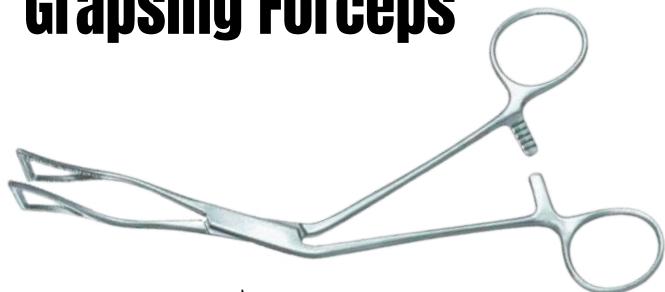
Specific for cardio-thoracic surgery to clamp specific vessels

Alligator Clamp



Used to grasp objects inside of ears

Lovelace Lung Grasping Forceps



Triangular grasping area distributes the force over delicate tissues like the lung

Doyen Intestinal Clamp



For atraumatic clamping of bowel



11-Blade



*Pointed blade for stab incisions.
Often used to open the linea alba*

10-Blade



*Large, cutting edge; traditional
blade for surgery*

15-Blade

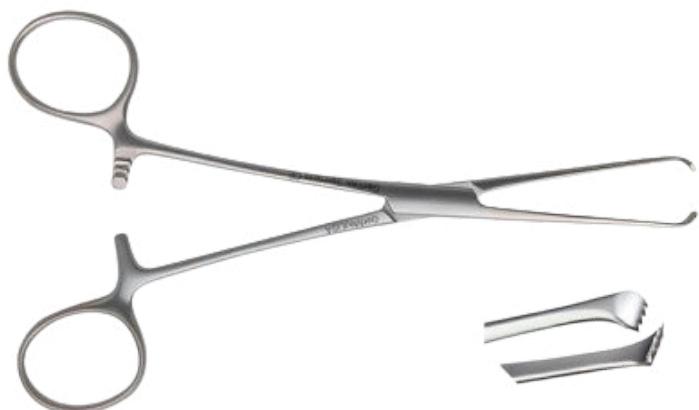
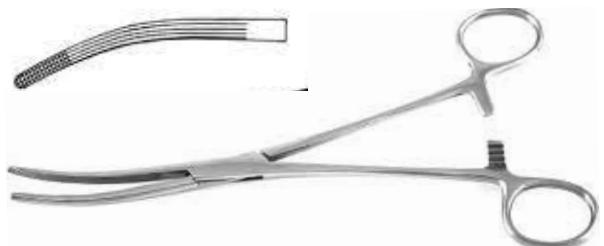


Short, cutting blade: for more precise cutting

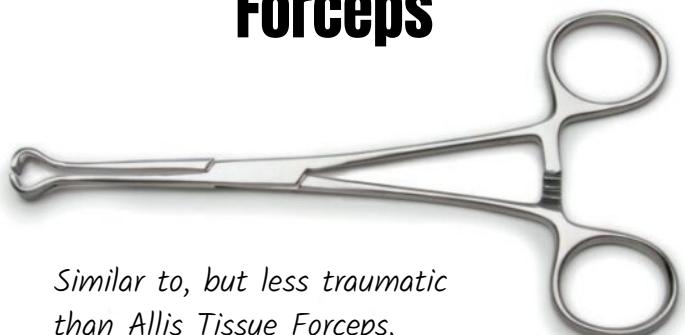
12-Blade



*Curved blade with sharp edge on the
inside of curve. Useful for dental
surgery, removal of periosteum*



Babcock Tissue Forceps



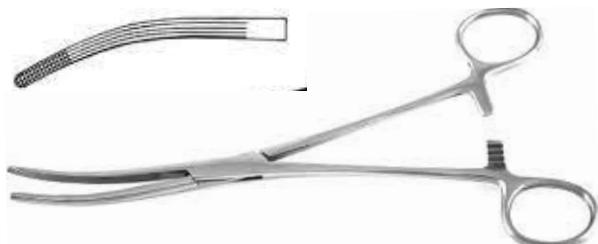
Similar to, but less traumatic than Allis Tissue Forceps.
Rounded flat ends for grasping delicate intestinal tissues

Rochester Pean Forceps



Fully serrated jaws to clamp off larger vessels

Rochester- Carmalt Forceps

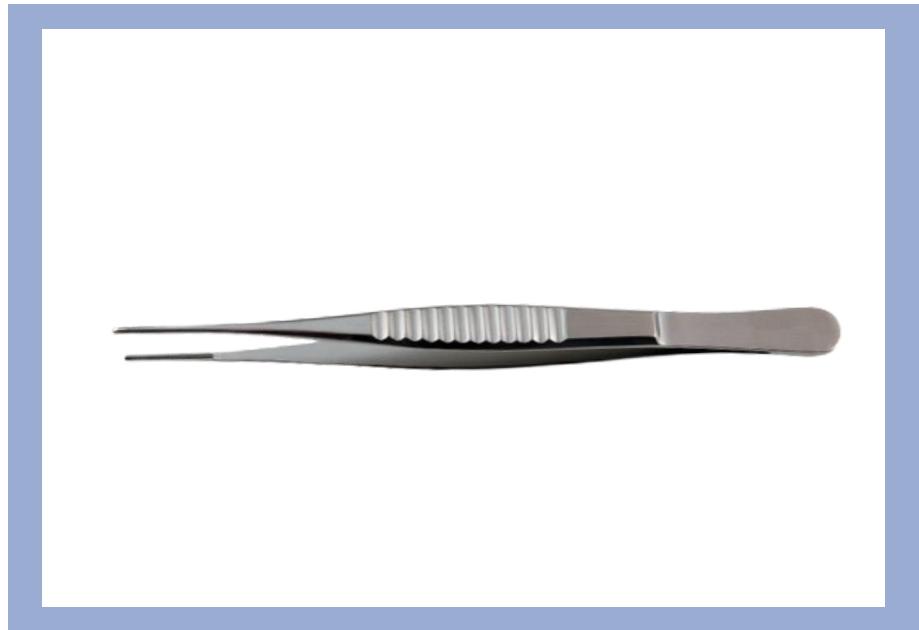


"Stars and Stripes" hemostats: cross-hatch at the end and longitudinal stripes along proximal jaws. Used to clamp tissues, ligate pedicles

Allis Tissue Forceps



For lifting, grasping dense or slippery tissue. Has a row of sharp teeth on each end



Brown Adson Forceps



*Most standard forceps
used in general surgery*

Debakey Adson Forceps



Most delicate thumb forceps for surgery

Adson Tissue Forceps



*Forceps with delicate tips to manipulate
delicate tissue. Has a wide thumb grip*

Debakey Tissue Forceps



*Atraumatic forceps used in
vascular surgeries*



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Russian Thumb Forceps



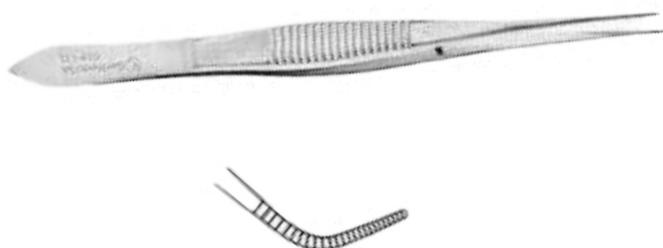
Heavier thumb forceps for more aggressive tissue handling

Potts Smith Tissue Forceps



Standard for ENT surgery

Dressing Forceps



Good for holding gauze for dressing wounds

Snook Spay Hook



Primarily used to locate the ovary during a spay surgery

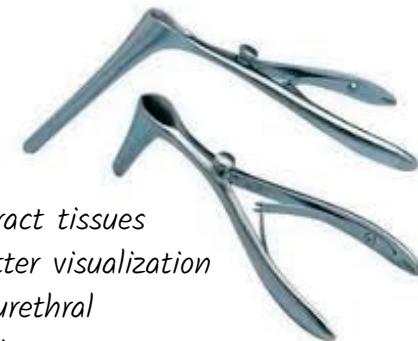


Malleable Ribbon Retractor



Commonly used during orbital procedures to retract orbital fat. Can be bent/adjusted as needed

Small Animal Vaginal Speculum



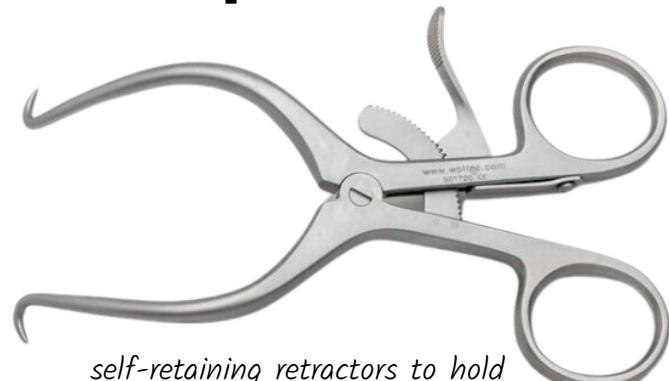
Used to retract tissues to allow better visualization for female urethral catheterization

Barraquer Wire Speculum



Primarily used to retract eyelids during ophthalmic surgery

Gelpi Retractors



self-retaining retractors to hold back tissues of a deeper incision for the surgeon



Hohmann Retractor



Used to move and protect tissues during orthopedic surgeries

Senn Miller Retractor



A double ended retractor used to retract tissues during surgery. Has a curved, fork-like end.

Army Navy Retractor



Used to retract shallow incisions: skin, muscle, fat

Beckman Weitlaner Retractors



Self-retaining retractors to hold open shallow incisions such as in spinal surgeries



Balfour Abdominal Retractors



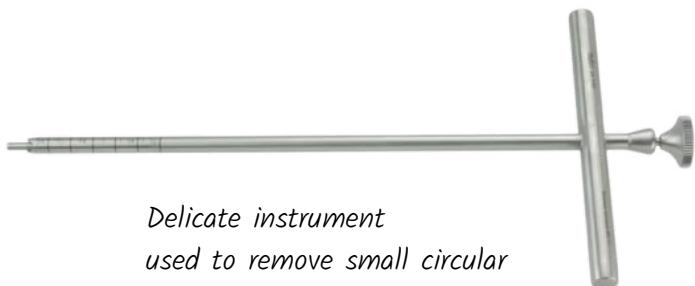
Self-retracting retractor used during abdominal procedures to hold open the abdomen. Has 7 to 10 inch spread

Cystotomy/Gall Bladder Spoon/Scoop



Used to scoop out stones from hollow organs like the urinary/gall bladder

Michele Trephine



Delicate instrument used to remove small circular pieces of bone to gain access to certain areas such as the sinuses

Poole Suction Tube



Designed to evacuate pooled blood and fluid during deep abdominal procedures



Volkmann Bone Curette



Commonly used during orthopedic procedures to scrape away debris and fibrous material from bone

Wire Cutters



Used to cut wires and pins during orthopedic surgeries

Kern Forceps



Used to hold bone plates or bone fragments during orthopedic surgery

Adson Cranial Rongeur



Used to grasp and trim and remove bone during neurosurgical procedures



Bone Cutter



Jaws the right size and angle for certain bone cutting procedures

Lorna Edna Towel Clamp



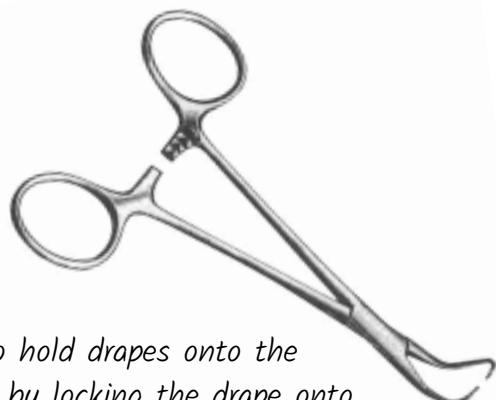
Atraumatic towel clamp used to secure equipment to drapes such as suction tubing

Jones Towel Clamp



Light weight clamp ideal for delicate procedures

Backhaus Towel Clamp



Used to hold drapes onto the patient by locking the drape onto the skin



Bard Parker Scalpel Handle #3



Used to attach blades # 10, 11, 12, 15

Wire Cutter Scissors



Used to cut stainless steel wire

Littauer Suture Scissors



*Used to hook suture loops to
remove stitches*

Vet Helpful Notes

VETERINARY SURGICAL INSTRUMENTS
FLASHCARD PACK- OVER 50 CARDS SHIPPED

VetHelpful.com
Canine  Feline 

