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DDx - Differential diagnosis: pg 334

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Quick Reference: see page 2 for complete information

C4T, C3T, C2T: Current Therapy in Equine Medicine 4, 3 or 2 1997

E: Equine Medicine and Surgery, 3, Mansmann, 1982

EM&S: Equine Medicine & Surgery, Colahan, 1991

ER: Equine Reproduction, Mckinnon, 1993

12M: Large Animal Internal Medicine, Smith, 1997

IM: Large Animal Internal Medicine, Smith, 1990

J: The Practice of Large Animal Surgery, Jennings, 1984

LA-D: Large Animal Dermatology, Scott, 1988

LAS: Textbook of Large Animal Surgery, Oehme, 1988

M: Manual of Equine Practice, Rose & Hodgson, 1993.

M8k or Mk: The Merck Veterinary Manual, 8th or 7th ed., 1998, 1991 Pic: Color Atlas of Diseases & Disorder of the Horse, Knottenbelt, 1994

POP: the Horse, Herbert editor

PP/USA/C: Poisonous Plants of US & Canada, Kingsbury, 1964

PP/M, PP/O, PP/A: Poisonous Plants/Montana, Ok. or Al.

R: Veterinary Obstetrics & Genital Diseases, Roberts, 1986

R-Y: Current Therapy in Large Animal Theriogenology, Youngquist, 1997

S: Equine Surgery, Auer, 1992

T: Current Therapy in Theriogenology, 2: Marrow, 1986

T&W: Techniques in Large Animal Surgery, Turner, 1989

T&W-A: Equine Surgery, Advanced Techniques, McIlwraith, 1989

Tox: Clinical & Diagnostic Veterinary Toxicology, Osweiler, 1985

Normal Laboratory Values: Inside back cover

Guide to Equine Clinics

3rd edition

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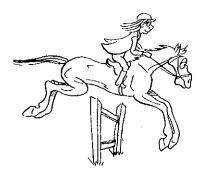




Volume 1: Equine Medicine

Volume 2: Lameness

Volume 3: Lameness diagnosis



to Jeannie Merek & Tony Corcoran



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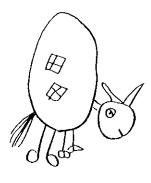


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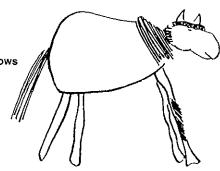
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3rd EDITION

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by Gina Pasquini: Horse w/ Windows



by Abbey Pasquini: Leg problem

Quick Reference

Each condition is keyed by abbreviation & page number to the commonly used reference books listed below

C4T, C3T, C2T: Current Therapy in Equine Medicine 4, 3 or 2; E. Robinson, W.B. Saunders Co., 1997, 1987, 1992

E: Equine Medicine and Surgery, 3rd edition, Mansmann, American Veterinary Pub., 1982

EM&S: Equine Medicine and Surgery, Colahan et al, American Veterinary Pub., Inc., 1991

ER: Equine Reproduction, A. McKinnon & J. Voss, Lea & Febiger, 1993

I2M, IM: Large Animal Internal Medicine, B.P. Smith, 2nd or 1st editions, Mosby, 1996 or 1990

J: The Practice of Large Animal Surgery, P.B. Jennings, W.B. Saunders, 1984

LA-D: Large Animal Dermatology, DW Scott, WB Saunders, 1988

LAS: *Textbook of Large Animal Surgery*, F. Oehme, Williams & Wilkins, 1988

M: Manual of Equine Practice, Rose & Hodgson, W.B. Saunders, 1993

M8k or Mk: The Merck Veterinary Manual, 8th or 7th edition, 1998

Pic: Color Atlas of Diseases and Disorder of the Horse. D.C. Knottenbelt, R.R. Pascoe, Wolf, 1994

POP: the Horse, your guide to equine health care, a magazine, K.S. Herbert editor

PP/USA/C: Poisonous Plants of the United States and Canada, Kingsbury, Prentice Hall, 1964

PP/M, PP/O, PP/A: Poisonous Plants/Montana, Oklahoma or Alabama, printed by the Extension Service of each state

R: Veterinary Obstetrics and Genital Dis-

eases, S.J. Roberts, Woodstock, 1986

R-Y: Current Therapy in Large Animal Theriogenology, R.S. Youngquist. WB Saunders, 1997

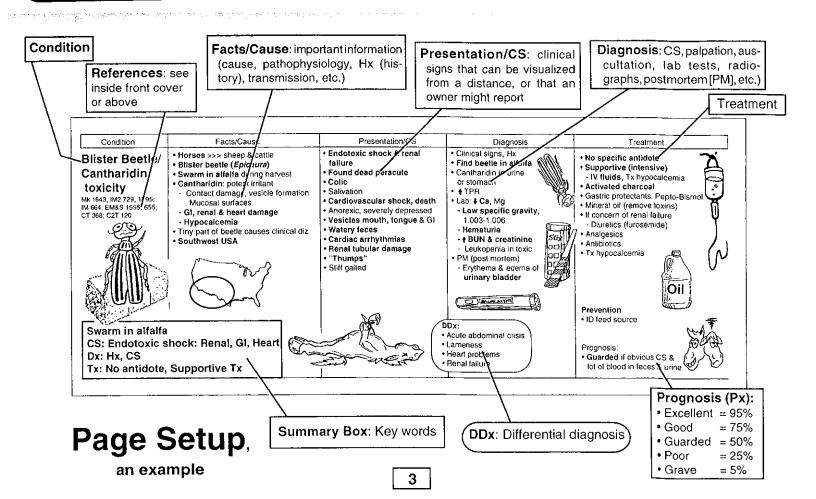
S: Equine Surgery, Auer, W.B. Saunders, 1992

T: Current Therapy in Theriogenology, 2: D. Marrow, W.B. Sounders Co., 1986

T&W: Techniques in Large Animal Surgery, Turner & McIlwraith, Lea & Febiger, 1989

T&W-A: Equine Surgery, Advanced Techniques, McIlwraith & Turner, Lea & Febiger, 1989

Tox: Clinical and Diagnostic Veterinary Toxicology, Osweiler et al, Kendal/Hunt Pub. Co., 1985



HE WAS OK YESTERDAY...THEN BINGO!

"Doc, I called you just as soon as I seen of Buck was sick. He's been a little poorly But he never missed a lick.

Last winter he got picky And wouldn't eat his grain So I gave him Doctor Bell's Tied garlic in his mane.

Then several months ago
When he started losin' weight
I give him Copenhagen
And a pound of catfish bait.

He come down with the splatters And all his hair fell out! So I fed him Larramycin And Mother's sauerkraut. Then last week after ridin' He got as stiff as pine! His navel needed smokin' So I used the turpentine.

He went plum down on Sunday. His kidneys, so I guessed. I doctored up his water And tied him facing west.

Last night I got to thinkin'.
You were here two years ago.
You gave him some concoction
For a cough and runny nose.

I wondered if your treatment, Which then improved his luck Had later turned against him And poisoned my 0l' Buck?

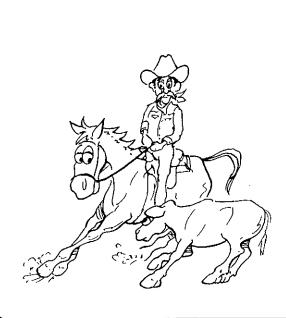
"Whattya think Doc?"

Baxter Black



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INTRODUCTION

The *Guide to Equine Clinics* is a quick reference guide for veterinarians and veterinary students. It should be especially helpful during the senior year in clinics.

Fashioned after Heidi Tschauner's *Senior Veterinary Student's Guide to Small Animal Clinics* and a direct continuation of The *Guide to Bovine Clinics*, it should help assess cases quickly until a more thorough reference can be located. This is not meant to replace reference texts, but to encourage the use of other references by keying each condition by page number to commonly used veterinary texts. During my veterinary education no teacher hinted how valuable *The Merck Manual* was for getting a handle on veterinary medicine. Board certified professors or those studying for board certification probably want the student to read the latest journal article on each condition. Admirable, but students don't have the time to search the journals and can carry *The Merck Manual*.

Susan Pasquini, my wife, typed her junior veterinary school class lectures on a portable computer. I then tightened them using the different veterinary texts, including The Merck Manual, Smith's Large Animal Internal Medicine, and Equine Medicine and Surgery. Susan would proof read and correct the charts as she studied for tests. This summer we have been revising & correcting for her senior year.

The key words in the first box provide a handle on each condition. Bold type allows skimming facts/causes, clinical signs, diagnosis and treatment. More in-depth information is given in light type and small type. Other texts keyed under the condition allow for quick references; while the cartoons hopefully add life and help page recognition. Differential diagnoses, the key to diagnosis, are highlighted in a rounded box for most conditions. The last chapter is lists of differential diagnoses for different syndromes to help you start making a rule out list. The index tries to list every name for the different conditions for convenience and quickness of use. The first page of each chapter has an alphabetical list of the conditions and their pages.

Many professors in the clinics expect you to remember everything presented in classes from semesters earlier. You won't! When in a panic about what a professor may ask, slide around a corner and quickly read through a condition in the guide. Short term memory may allow an intelligent answer to the professor in front of your peers. If the professor will allow, when a classmate is questioned about a condition; quickly find it and read along. This will give you an overall view and may raise questions you can ask.

The key to learning is reading as many texts and articles on each condition over and over as you take care of an animal with the condition. Time constraints make this impossible; and students often only have time to read about the cases assigned to them. The mini indexes at the start of each chapter allow quickly finding conditions. The outline format allows a quick review of each condition. At the minimum read the guide for each condition seen by all the students. The *Merck Manual* or *Manual of Equine Practice* by Rose and Hodgson are small texts that bring you to the next level. Then, when there is time, read the other texts referenced in the guide. Every day as long as the case is in the clinic skim the condition in the guide.

Hopefully this guide will open up the clinics for the lower classmen who are put off by trying to read an incomprehensible 200 page chart. Classes such as pathology, clinical pathology, parasitology, virology, bacteriology, neurology, etc. deal with conditions not fully discussed until later in medicine and surgery classes. Read about them in this guide to get an overview of facts, clinical signs, diagnosis and treatment. This should make these conditions less disconnected, thus, more meaningful and easier to learn. Go and see animals in the clinic. Examine an animal at your level of knowledge. Pretend you are the veterinarian in charge. Find in the animal's chart the physical exam findings and the differential diagnosis. Think about these. Check the diagnosis and look up the condition in this guide. Then see if you can detect any of the clinical signs. Hold the medicine being used and imagine yourself administering it. Guess if the animal will survive. Check on its progress over time, refreshing your memory with the guide and other texts if you have the time, over and over. Later in lectures, when a condition comes up, you will have a specific animal to relate it to.

This Guide is incomplete, some sections are good and others weak. We will keep working on it during our senior year. With help from other seniors and faculty members it may turn into the key to Equine Clinics. There are pages at the back of the guide to write any ideas, criticisms, praise, corrections, cartoons or new charts. When through with your senior year, please tear these out and send them for inclusion & credit to:

Susan Pasquini Sudz Publishing 1222 S. Hwy. 377 P.O. Box 1199 Pilot Point, Tx 76258 (817) 686-9208

Most of all, as you go through veterinary school, remember your elation on acceptance into veterinary school and your realization that you will one day be a veterinarian. Veterinary school seems to kill this excitement. Don't let it!

Chris Pasquini

Disclaimer: The authors do not assume any responsibility for any results obtained from the procedures, treatment, drugs, and dosages used; nor shall the authors be held liable for any misinformation or errors that may have been obtained by any persons or organization using this book.

Acknowledgment: Susan Pasquini's work makes up the heart of this book. Her revisions and corrections make up its complexion. Thanks goes to Dr. Kerstin Thorén-Tolling for the Clinical Pathology Chart. Pamela Pinchuk corrected the vaccination and parasite control pages. Rick Linkenheimer and Robert Diliberto created the basis of the Toxicology chapter. Alicia Emerson and Mark Sminkey edited the text. Thanks to Anne Couger, Oklahoma State Veterinary librarian, for her assistance. Special thanks to the authors of the books to which this guide is keyed. Hopefully the guide will encourage the student to use these more often.

I would like to thank Dr. James Brendenmehl from Oregon State University and the following teachers at Oklahoma State Veterinary School for the information and insight of their rounds that I have sat in on:

Dr. Pamela Pinchuk

Dr. Karen Copedge

Dr. David Stevens

Dr. Charles MacAllister

Dr. Mary Ann Guglick

Dr. Greg Pullin

Dr. Michael G. Collier

Dr. Henry Jann

I would like to thank the following students who helped edit this edition:

Robin Warden Lisa VanNieuwal Marilyn Wolgamott

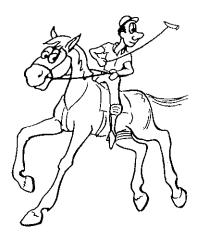
Margaret Mamula

Dawn Goldacker Cari Satterfield

Trent Stites

Ann Coffin

And lastly, I would like to thank the Students of Ross University who made my three years in paradise just that.



Welcome to the clinics. This should by far be your most exciting year in veterinary school. If it isn't, think about becoming a virology teacher. You have been given and remember (?) all the facts needed to function in this new world of hands on medicine. It can be terribly confusing. As you move from Equine medicine rotation to Theriogenology to Radiology to Small Animal Medicine you may lose track of your cases and may wonder what ever happened to "Amber", the mare with acute renal failure.

Notebooks: The major advice I can give you is get a pocket notebook (2.5x4") and a 5.5x8" notebook and write things down. Carry the pocket notebook everywhere and write down instructions the clinician gives you. Give up on trying to impress people with your phenomenal memory, clinicians won't care if you remember 99% of the things they told you, they will be upset if you forget to give a horse its life saving medicine. Write down things that you want to look up. Use the larger notebook for rounds, when, as a group, you talk about the horses everyone is treating. Below is a scheme that I am trying to iron out. Perfect it for your needs or idiosyncrasies or come up with your own scheme.

Number all the pages in the big notebook in the lower outside corner.

Table of contents: When you are waiting for something to happen in the clinic make a table of contents. On the first page, write page numbers along the left hand side, skipping a line between num-

bers. Continue this onto the next pages until all the pages in the notebook have two corresponding lines in your table of contents. As you fill out the pages in the notebook, name them after their contents and record them in your table of contents. This will be invaluable as you try to find vital information you wrote in the notebook last week.

Cases: Label the four pages following the table of contents "CASES". As cases are passed out, draw an opened box at the start of a line. Follow this with the case number, horse's name. Later when the diagnosis is made, write it on the same line in bold BLOCK LETTERS. This will obviously give you a list of all the cases seen. If you move to another rotation before the horse is discharged the open box will help you follow the case through (an important thing to do!). Ask the clinician in charge what happened or go look up the chart with the case number. Check the box off when the animal is discharged or necropsied. An open box indicates an unfinished learning experience.

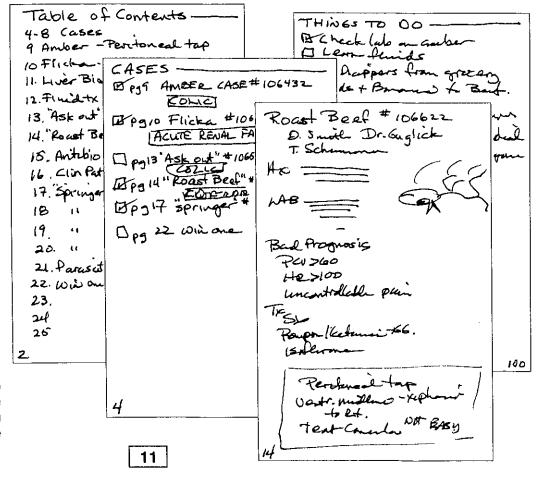
Information, confusion, drugs cases, etc.: The pages following the CASES pages are for the mass of information that will be coming towards you. For your own case give yourself enough pages to cover it (3 or 4 pages per case). For all cases write the case number, horse's name, owner's name, clinician's name and assigned student. Later write the diagnosis in bold BLOCK LETTERS enclosed in a box.

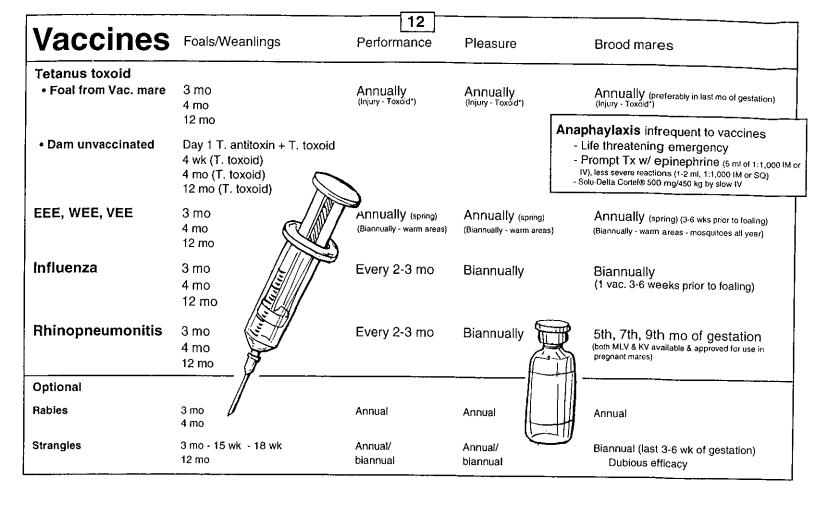
For all cases write down the history, signalment, etc. Be sure to write down all the instructions preceded by open boxes for you to check off as things get done. As you read up on the condition, jot down notes and organize your thinking. Write down things to ask the clinician. As lab data, radiology and other information comes in, write it down. If you run into another case's pages, put continued on an open page and continue on that page.

Miscellaneous: Fluid therapy, drugs, clinpath, etc. may need to be reviewed. Write down pertinent information for you to use in other cases and put page number in the table of contents.

Things to do! On the last page list the things to remember to do as they come up. Put a box on the left hand side and write down what task to do. When each task gets done, check it off.

Optional: Draw case horses, trying to characterize their clinical signs. These can be stick figures, but they will help you observe the horse and remember it in the future.





Minimum vaccination schedule

- For foal from immunized mare:
- -3, 4,12 mo: T. Toxoid, EEE, WEE, Influenza & Rhinopneumonitis
- · For foals from unimmunized mare
- Day 1, T. antitoxin + T. toxoid, 4 wk T. toxoid then like other foals
- Revaccinate
 - Annually with T. Toxoid, EEE & WEE
 - Performance every 2-3 mo w/ Influenza & Rhinopneumonitis
 - Pleasure biannually w/ Influenza & Rhinopneumonitis
 - Brood mares: during gestation
 - -- Annually w/ T. toxoid & EEE & WEE in spring
 - -- Biannually for Influenza
 - -- 5th, 7th, 9th mo of gestation for Rhinopneumonitis

Tetanus toxoid

- *Injuries or surgery: revaccinate w/ toxoid after injury or surgery if not vaccinated in last 6 mo
 - Tetanus antitoxin is contraindicated in horse previously immunized w/ tetanus toxoid because of possible serum hepatitis (pg. 88)
 - Telanus antitoxin has been associated wil fatal serum hepatitis, so restrict its use to horses that have never been vaccinated wil toxoid or those of unknown vaccination status who need immediate telanus protection
- · Injury or surgery to unvaccinated or unknown vaccination history
 - Tetanus toxoid & tetanus antitoxin in 2 separate injection sites & then toxoid
- Foals from unimmunized mares: give t. antitoxin + T. toxoid on day 1, revaccinate
 at 4 weeks and then put on same schedule as other foals
- Conveniently sold in 4 way combo w/ EEE, WEE & Influenza

Influenza

- For high risk foals (shows) primary series: 1 mo, 2 mo, 3 mo, then every 2-3 mo
- · If started before 3 mo, need 3 dose primary series
- Sold in 4 way combo w/ Tetanus toxoid, WEE & EEE

WEE & EEE

- · Warm areas biannual where mosquitoes are present all year
- · Annual revaccination prior to insect season in spring
- VEE not seen in USA, horses transported to potentially endemic areas (Texas, Calif., Cen. Amer., Mexico) should be vaccinated
- 4 way combo w/ Tetanus toxoid & Influenza

Rhinopneumonitis

- If start before 3 mo need 3 dose primary series
- Highly recommended for brood mares even though protection not 100%, sporadic abortions & rare abortion storms do occur in vaccinated mares
- Vaccination in face of abortion storm recomended
- Foals at high risk: revaccinate every 2-3 mo after 4 mo, for life

Rabies

- Use in endemic areas or where horses are near wild animals; it is a safe vaccine
 the diz is fatal, so use w/o hesitation
- . Do not used unapproved modified live vac.

Strangles

- Usefulness of vaccine questioned:
 - Outoreak of strangles have occurred in vaccinated horses
- Strangles vaccinations have local (injection reactions) & systemic side effects
- Therefore recommended only on farms where strangles is a persistent endemic problem or if transporting to such a farm
- Not routinely recommended in pleasure or performance horses in low-risk situations
- Must have a primary series of 2-3 injection at 3 wk intervals
- Use alternate injection sites (bec. of injection reactions)
- Performance horses previously unvaccinated: 2-3 primary vaccinations at 3 wk intervals before shipping
- M-protein extract preferred over whole cell

Other vaccines available: VEE, Potomac fever, Botulism, Eq. viral arteritis, Anthrax

> Taken from Equine Vaccination Guidlines, Pamela A. Pintchuk, DVM, 1993

Parasite control

Basic - assume all horses are parasitized

• Ivermectin every 2 months for life (no resistance noted yet)

Interval deworming program

- 6-10 week-old: first dose of anthelmintic for foals
- 8 week intervals for Ivermectin
- 4-6 week intervals for drugs other than ivermectin (recommended)
- Botation of drugs
 - Fast: change Rx every time
 - Slow: change every 1-2 years

Foals -	example program
(from C	T 52 by J.A. DiPietro)
0	harmania ()
2 mo	Ivermectin (nematodes)
4 mo	Oxibendazole (nematodes)
6 mo	Pyrantel pamoate (nematodes)
8 mo	Ivermectin (nematodes & bots)
10 mo	Pyrantel pamoate (nematodes)
12 mo	Ivermectin (nematodes & bots)

Adult -	- interval program (J.A. DiPietro)
Feb.	Pyrantel pamoate (nematodes)
April	Oxibendazole (nematodes)
June	Fenbendazole & piperazine
	(nematodes)
Aug.	Ivermectin (nematodes & bots)
Oct	Ivermectin (nematodes & bots)
Dec.	Pyrantel pamoate (nematodes)

- 1 control program recommendation is impossible because of the wide range of climatic, economic & management conditions across the USA
- Interval programs: determined by fecals taken 2 wks after deworming in 1st year of program or from past experience
 - A boticide should be given in bot season & 1 mo after first frost
 - Foals start at 8 wk-old bec. when ascarids first present

J - After 1 yr-old on adult program

- Strongyloides problem area treat foals w/ ivermectin or oxibendazole (15 mg/kg) at 3 wk-old
- Treat mares 12 hours postpartum (reduce S. westerii in milk & other nematodes)
- Deworm mare on same schedule as foal
- Use slow acting agents (pyrantel pamoate, ivermectin or benzimidazoles) for heavy ascarid infections to avoid possible impaction (not piperazine)
- Major problem w/ Benzimidazole resistent sm. strongyles

integrated or seasonal program (northern temperate zones) where there is a spring rise in number of adult strongyles. Cheaper & m/ prevent development of resistance

- Foals on interval program for first year
- Adults anthelmintic in spring
- Boticide at end of bot season, m/b also in middle of bot season

Seasonal program - Adults

May Oxibendazole (nematodes)
July Ivermectin (nematodes & bots)
Dec Ivermectin (nematodes & bots)



Continuous low-level feeding of pyrantel tartrate

- . 6-10 wk-old: first dose of anthelmintic for foals
- · Ivermectin at start of program
- Then continuous feeding of 2.64 mg/kg of pyrantel tartrate base in alfalfa pellet
- Administer a boticide to kill Gastrophilus during & at end of botfly season
 Ivermectin and organophosphates

Check efficacy of all programs with fecal tests periodically

Anthelmintic	Trade name	Stron Large		S. westerii	"Round"	Bot	Pinworms	Comments
Ivermectin	Eqvalan® Zimectrin®	+*	+	+	+	+	+	 Broadest spectrum, safe, no resistance noted Effective against mature, immature & migrating forms of "Round worms" (Parascaris equorum) Ineffective against tapeworms
Piperazine (PPZ)	Many (2)	+/-	+	6	+	-	-/+	 Fast acting so shouldn't use in foals w/ heavy ascario infections (m/ result in impaction) Added to BZDs for BZD resistent sm. strongyles
Pyrantel Pamoate	Strongid T Strongid P® Imathel®	+	+		+	•	-/+	Popular & effective 2 x dose for tapeworms
Organophosphate Trichlorion, TCF (Cor Dichlorvos (Equigardo	nbot®, Dyrex®)	-	-		+ +	++	+	Narrow margin of safety
Benzimidazoles (I Thiabendazole, TBZ (Equizol Mebendazole (Telmin®), Fen Oxfendazole (Benzelmin®)	e®), Cambendazole, CBZ (Camvet®	+	- /+		+	-	+	• BZD resistent sm. strongyles - emerging problem •+/-= ineffective for benzimidazole resistent strongyles effective for other small strongyles
Oxibendazole	Anthecide®, Equipar®	+	+	+	+	-	+	• Only Benzimidazola or Bro BZD attacking a single
Pro-BZD (Febantel)	Rintal®	+	-/+	த	+	-	+	 Only Benzimidazole or Pro-BZD effective against BZD resistent sm. strongyles
Benzimidazole com	bos							• Diporazina artichlada - 11 1 1 1 1 1
TBZ - PPZ	Equizole A®	+	+	NYI	+	_	_	Piperazine or trichlorfon added to thiobendazole increases offostivoness and instructional designs.
TBZ + TCF	Equizole B®	+	+	11 11		_	T .	increases effectiveness against resistent sm
MBZ + TCF	Telmin B®	•	•	- N. / /	•	7	7	strongyles

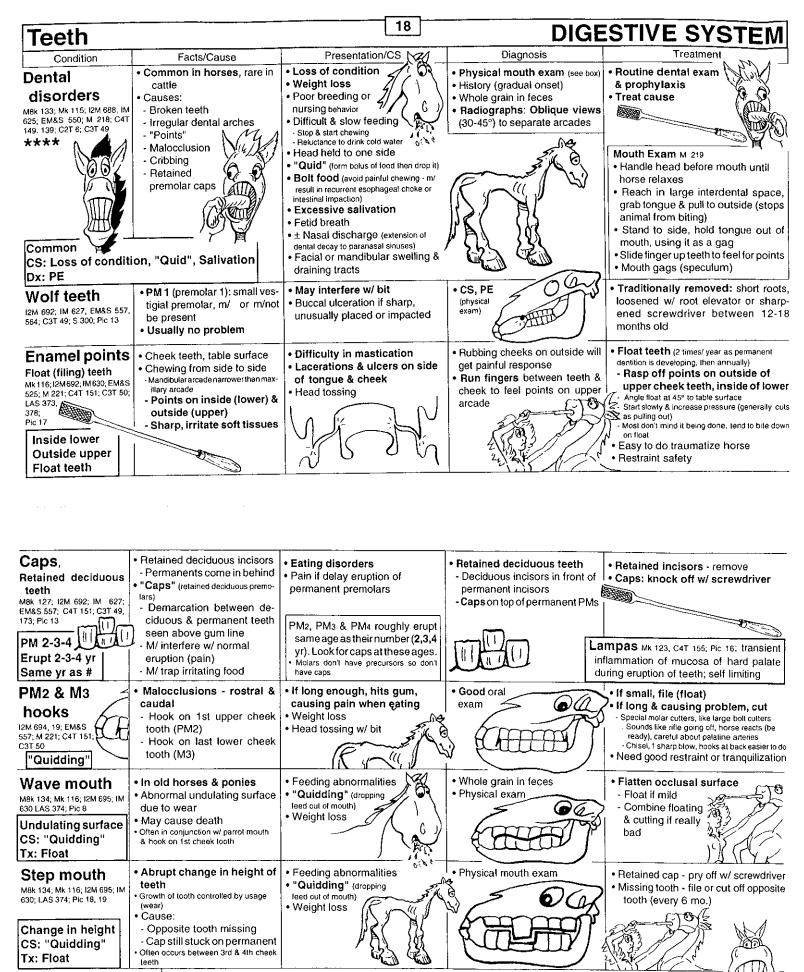
 ⁽⁺⁾ Indicates 90-100 % efficacy against non-migrating stages of nematodes at recommended doses, (-) indicates less then 90%



		5: 1	
Abscess, abdominal	65	Displacement - large color	
Abscess, tooth	20	Duodenitis-Prox. jejunitis	67
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			_
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Neonatal hepatic failure	86	Sand impaction	78
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Nephrosplenic entrapment	81	Septicemia	38
Neoplasia 28, 66,		Serum hepatitis	88
NSAIDs 27,	43	Strangulation, large intest.	
Oak, acorn toxicity	44	small intestine	68
Obstruction	64	Strongyles	36
Ovaro paint	78	Shear/step mouth	19
Pancreatitis	91		0-63
Parascaris 37,	65	Tapeworm	77
Parasites 24, 36, 37, 51,	65	_ '	3-20
Parrot mouth	20	Teratomas (dental)	20
Passive transfer	39	Tetracyclines	43
Peritonitis	53	Theiler's disease	88
Phenylbutazone toxicity	27	Toxic blue green algae	75
	288	Tyzzer's disease	87
Points.	18	Ulcers	26
Poisonous plants	45	Umbilical hernia	72
Potomac fever	43	Volvulus, large colon	80
Protein losing enteropathy	50	small intestine	69
Proximal enteritis	67	cecal	77
Ptyalism	23	colonic	81
Pyrrolizidine alkaloid	89	White foal diz	78
17		Wolf teeth	18



Sheer mouth

CONTRACTOR INCIA

I2M 695; IM 630; EM&S 562; Pic 11, 18

 Oblique occlusal surface to incisors when viewed from front • CS: Inefficient eaters; sling jaw from side to side when ridden • Tx: Level with saw & drumel 19

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Dental cysts. Dental teratomas. Dentigerous cysts, Heterotropic polydontia M8k 127; C4T 144, IM 630; · Dentigerous cysts arise from tooth germ tissue in the ear or temporal region

Occasionally in cranial vault or maxillary sinus

Lined by stratified squamous epithe-

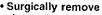


Seromucous discharge in area of ear

. Discharge from area of ear

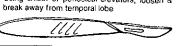
· Rads: see teeth attached to temporal bone

Occasionally may not see teeth



Cranium close so must be careful Dissect bluntly around capsule w/o

cyst Using chiset or periosteal elevators, loosen &



Parrot mouth, Sow mouth

EM&S 562; M214; T&W-A373;

M8k 126, 134; IM 630; EM&S 561; C3T 49; Pic 10

 Brachygnathia: parrot mouth or overshot, shortened lower jaw

- Most common deformity Prognathia: sow mouth, undershot, long lower jaw, less common

Inherited

 Malocclusion: parrot mouth becomes mouth

if no occlusion

Parrot mouth: if caught early enough, treated w/ braces to incisors to try to slow down growth of jaw

Abscessation of tooth roots. Periodontal diz

Apical, periapical, dental abscessation

Infundibular necrosis. Alveolar periostitis M8k 135; I2M 695; IM 628;

EM&S 558, C4 152: C3T 49: M 220; T&W-A 260: S 300; 380 Pic 21

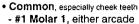
#1 - Molar 1

CS: PE. Rads

Tx: Repel

CS: Wt. loss, odor

"Cup



 Infundibulum, cup or dark spot. normal indentation in occlusal surfaces

 Teeth don't fall out because packed tightly together

Old loose teeth in aged horses should be removed, cause discomfort that leads to infection

Causes:

· Trauma (kicks)

· Broken or split tooth

· Any malocclusion: step mouth

Patent infundibulum

Difficulty in mastication

"Quidding" (dropping half chewed feed)

Only chew on one side

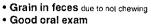
Lose weight & not eating as much · Swelling on ventral aspect of mandible,

or on side of face if first 2 cheek teeth, caudal 4 in maxillary sinus

#1 cause of maxillary sinusitis - Unilateral purulent nasal dis charge

Chronic: distortion of face, swelling w/n sinus

Tearing if pressure builds up on lacrimal



Smell, w/ rotten tooth first clue

Looseness (must be very rotten)

Dental pick into each tooth

- Patent infundibulum Periodontal diz starts w/ gingivitis

· Radiographs to confirm (best)

Obliques, 30° to horizontal,

centering beam in maxillary sinus

Air density between teeth roots & alveoli normal, location of periodontal membrane

- Destruction of lamina dura (side of alveoli) radiolucent area

Sequestra of dead dental tissue

- Fistulae



Remove tooth (see box pg 21)

· Pulled inside mouth (older horses)

- Pack w/ gauze

Repulsed into mouth (younger horses)

- Identify tooth by finger

- Trephine hole over its root

- Dental punch, knock out into mouth

Must be directly over tooth or will hammer out 2 teeth or m/ fracture maxilla or mandible

Flush out socket (chips of bone, tissue)

- After care (difficult part of procedure)

Pack off alveoli (so doesn't become impacted w/ feed) (see box)

· Float opposite tooth every 6 mo.

Often drainage as well as swelling, seec rusty area, if removed, see drainage tract

DDx:

. Eruption: swelling, but no drainage, normal

Tooth removal:

• Old, loose tooth (short root) - m/be extracted by hand or w/ small molar forceps

♦ Loose infected tooth (especially those split longitudinally)

Detach buccal & lingual gingiva from crown

- Occasionally m/be removed by rocking w/ a long-handled molar forceps

- Introduce fulcrum & lever tooth out

- If young horse m/be too long to move straight out between 2 arcades - May be able to push sideways by finger, or

- Cut off w/ tooth shears, then repeat & shear until tooth removed

Infected teeth that are not loose or too short to grip must be repulsed

Tooth repulsion:

· Maxillary (upper) cheek teeth

- PM2 & 3 in front of maxillary sinus

- Trephine directly above occlusal (table) surface of tooth (find w/ finger in mouth)

- Repel tooth w/ angled punch & mallet

PM4, M1 & M2 - located in maxillary sinus

5-8 vr-olds - trephine hole above caudal edge of tooth

- Over 8 yr-old trephine more & more towards center of table surface as age

- M3 - far back in caudal maxillary sinus, below orbit

- Trephine over frontal sinus (conchofrontal) - medial to medial canthus of eve

- Long, curved punch so can go through frontomaxillary opening & caudally

Mandibular cheek teeth

- P2 - trephine hole over center of table surface (any age)

- P3, P4, M1 & M2

- Trephine ventrolateral at jaw border (protects med. structures, good drainage)

- 5-8 yr-olds - progressively more caudal, so over caudal border of last 2 (M1, M2)

- > 8 yrs-old - trephine more over center of occlusal surface

- M3 (third molar)

- Trephine close to border of mandible through masseter muscle on a line from table of M3 to farthest point of angle of jaw

- Careful, mandible thin at this point

After care:

Pulled tooth

Pack alveolus w/ gauze, or

- Pack w/ dental wax (softens in hot water, then hardens)

Granulation squeezes wax out

Methylmethacrylate m/ be used to pack

• Repulsed tooth (see box)

- Pack off alveolus (so doesn't become impacted w/ feed)

4x4 gauzes rolled into circle

- Tie w/ umbilical tape 3' long, leaving long tags

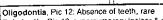
- 1 tag through trephine hole, 1 out of mouth, pull gauze into tooth socket

- Tie two tag arms together

Change every other day

- Takes 3-4 wk to granulate in, must always maintain





Polydontia, Pic 12: supernumerary incisor & molars Sand mouth, Pic 19: incisors worn down to gum line in area with lot of sand - *Florida



Jaw & Esophagus 22 DIGESTIVE SYSTEM Condition Presentation/CS Diagnosis Treatment Jaw fractures Trauma Difficulty eating Usually not difficult Surgical correction · Most commonly: interdental 12M696; C4T159; EM&S1461; Malocciusion 00000 Bone plates used (miss roots of teeth) area of mandible Intermedullary pins passed through Difficult eating Prognosis: - Wire teeth together, drill at base of toolh Tx: Immobilize Depends on mobility, if both sides to get through Use various combinations to wire together Oral Tumors M8k 196; 12M 696 • Rare in Eq. Melanomas unstable, need lot to stabilize of jaw Aluminum rod bent around, then wire it to teeth Palate closes from rostral Cleft palate, · Nurses, then stops Oral exam. Euthanatize if gross defect to caudal Milk comes out If short, pharyngostomy, (soft palate) M/b difficult if just in soft pal-**Palatoschisis** Defect always at back M8K 125; Mk 109, C4T 387; nose when If severe do mandibular symphysicotomy for better visualization EM&S 544; M 213; S 453; LAS M/b genetic component head is down 388, 344; S 453; Dissection in along muscles below Defect at back Dies of starvation Pic 9, 93: tongue, opening bottom jaw Soft palate pulled together DDx: POP 90-1/98 if not corrected CS: Milk out of nose Choke (pg. 23) after "freshen" edges Tx: Euthanize or Sx (cutting off edges of cleft) 1° - vesicular stomatitis (report diz), horse pox, candidiasis, pseudomonas, Gingivitis, If hard palate Rhodococcus, trauma grass, blister beetle toxicity; 2° due to periodontal diz, Incision made right against teeth on oral side Cut to bone (palatine a. just away from teeth) Stomatitis phenylbutazone (PBZ) toxicity, photosensitization, uremia, Mg toxicity Incise edge of cleft M8k 196; IM 740; M 215; C4T • CS: Reluctance to eat (ulceration, swelling & redness), depression, salivation Flap of mucosa & periosteum 387, 158; EM&S 547, 1707; Pic • DDx: Lampas, Neoplasia, Dental problems, Foot & mouth diz (reportable, horse resistant) slid together to close gap 25, 27; Pop 14-7/98, 8-7/97 End up w/ 2 large layers of Tx: Lavage mouth (povidone-iodine), ± Fluids, Tx 1° diz exposed bone next to teeth · Constriction of esophagus Esophageal · Recurrent esophageal feed Hx: Recurrent choke • Dietary: Slurry of pelleted Causes: impaction Contrast esophagram feed & roughage stricture - Prolonged choke Dysphagia Indirect bougienage: Esophagoscopy I2M 195; M 638; C3T 181; S - Trauma to neck dilate stenotic region - Reflux esophagitis Esophageal surgery Dietary management: - Esophageal surgeries - Long term slurry of pelleted feed & roughage (AVOID!) "Complete" pellets & no roughage - Squamous cell carcinoma Council owner m/ lead to GI problems (e.g., Ig. colon impaction or enteritis) Wound contraction Indirect bougienage (safe, nonsurgical method to dilate stenotic region) Earlier done the better, Fast animal, Daily sessions of dilation Esophageal surgery Recurrent choke Hi potential for complications, but m/ be necessary Tx: Slurry food, Bougienage - Esophagomyotomy, esophagoplasty & patch grafting Anxiety & distress (pawing ground, • Hx. CS Choke. Most commonly diagnosed Tends to resolve spontaneously esophageal disorder of the get up & down) Palpate, usually large, soft mass, not as saliva softens. Esophageal Arched neck easy to palpate horse few hours to several days obstruction Nasogastric tube to locate, m/ pull Retching · Causes: Don't be hasty M8k 194; Mk 173; I2M 701; IM Profuse salivating back plug of feed - Greedily eating dry grain Tranquilize 636; EM&S 583; C3T 175; C2T Survey radiographs Regurgitation of food & - Confine & give water, but no feed (swells) or pelleted feed 12; M 223; T&W-A 264; S 310;

LAS 395; Pic 34, 35



- (defective teeth) & young (erupting teeth)
- During recovery from anesthesia
- Exhausted animals
- · Occasionally medicinal boluses, ears of corn, potatoes or bolus of hay
- M/get strictures chronically

Sequelae:

Aspiration pneumonia

Greedy - dry food CS: Salivation, anxiety Dx: CS, PE

Esophagostomy last resort!

- Improper mastication by old

Necrosis of esophagus

Tx: Stomach tube

saliva through nostrils, bilateral (milk in toals)

Coughing

Drink water & sputter

Anorexia, depression

 Stomach tube down & gently lavage \/ (pumping & siphoning) (careful of aspiration pneumonia)

Heavy sedation: Rompun/Torbugesic, Detomidine/Torbugesic, lowering of head for ventral drainage & to avoid aspiration

- Never use mineral oil or dioctyl Na sulfosuccinate (DSS) (softener) as

too easy to aspirate

Very small amounts of water only

 Large bore stomach tube w/ cuff in esophagus & smaller lavage tube placed inside for more aggressive lavage (fluid passes back through larger tube, bypassing larvnx)

More aggressively pumping & siphoning

- Endotracheal tube into larynx, w/ cuff inflated - Under general anesthesia, head down so water drains out

Esophagostomy last resort!

Milk back toward pharynx

If really damaged must incise & remove, put stomach tube in & feed through tube

- Complication: esophageal fistula & stricture Ptyalism (saliva production, caused by pain in mouth, pharynx or esophagus; heavy metals, slaframine, swallowing

- Mucocele: swelling of parotid duct; Sialoliths: Ca carbonate - cause blockage

• 1° Salivary tumors rare (benign adenomas or adenocarcinomas [excise]; Adenocarcinomas m/ metastasize to lymph nodes) 2° metastatic tumors (malignant melanoma in old gray horses &

- M/b spontaneous recovery

Stomach tube & gently lavage

- Never use mineral oil

 More aggressively lavage under general anesthesia

Esophagostomy last resort, complication: esophageal fistula & stricture

 NSAIDs to control pain & inflammation (Banamine®, PBZ/phenylbulazone)

Broad spectrum ABs (if aspiration)

 Evaluate esophageal mucosa after choke is resolved:

- If minimal mucosal damage, return to normal feeding

Irritation &/or ulceration

. Withhold food for 24 -72 hours

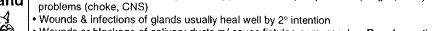
. Nonirritating, moist feed (slurry of pelleted feed)

- Free choice water





M 216; C4T 161, S 306; Pic 29



Wounds or blockage of salivary ducts m/ cause fistulae or mucoceles: Ranula: cystic dilatation in mouth

squamous cell carcinoma)

Gastrointestinal Diseases

24

DIGESTIVE SYSTEM

Condition

Gastritis: Gastric catarrh

Mk 193; EM&S 604



Facts/Cause

- Uncommon in the horse
- . Manifestation of other dizs Causes:
- - Irritating substances (see box)
 - Overeating, grain overload
 - Sudden diet changes
- Ingestion of hot, frozen, moldy, or spoiled feed
- Ingestion of sand or FB (foreign body)
- Cribbing
- Parasites (see below)
- Reflux from small intestine (associated w/small intestinal obstruction, material refluxed into stomach, can't vomit so gastritis)
- Carcinoma, delaying emptying

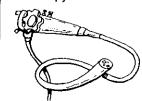
Uncommon

CS: Upset stomach, laminitis

Presentation/CS

- CS may be masked by 1° disease
- Drooling, excess salivation
- Don't eat
- Colicy (subacute)
- Feces dark or tarry if hemorrhage
- Habronema ulceration m/b inapparent clinically
- Gastric dilatation & pylorospasm
- Temp elevation
- Severe colic
- Pulse fast & weak
- Profuse sweating
- Retching uncommon
- ± Vomit
- Sitting dog stance
- Chronically
- Weight loss, debilitation
- Intermittent colic as apposed to acute colic
- Sequelae:
- Laminitis accompanies or follows gastritis
- Gastric rupture

 History, CS Endoscopy



Irritable substances

- Blister Beetles (pg. 47)
- · Coarse feed (foals)
- Lead paint (pg. 259)
- Arsenic
- Mercury (from "Blistering" limbs) Mercurial counter-irritants (horse eats off bandage)
- DSS

Remove cause

- Restricted, easily digested diet (bran gruels or mashes & green feed or
- Mineral oil (laxative if ingested spoiled or irritant feeds)
- Protectant, Pepto-Bismol®, Kaolin®
- Stomach tube (relieves gas or fluid distention)
- Gastric lavage (for chemical gastrilis or impaction, difficult with solids)
- H2 blockers
- Torbugesic® (butorphanol) (pain)
- Spasmolytics
- IV fluids for shock
- Gastrotomy, severe
- impaction or dilation When CS abate
- slowly back on feed



Stomach parasites - Gastritis: Clinical signs rare; infection common

Trichostrongylus | • Trichostrongylus axei infection

Small stomach worms M8k 205; Mk 196; EM&S 602;



- Small stomach worm
- Same as in cattle
- Adults small & slender (8 mm)

Small stomach worm CS: Gastritis, wt. loss Dx: Eggs like strongyles

Tx: Ivermectin

- Chronic catarrhal gastritis
- Weight loss



- Difficult because eggs similar to strongyle eggs
- Fecal culture, ID larvae
- · Post mortem:
- Nodular areas of thickened mucosa, small erosions & ulceration

 Ivermectin routinely deworm every 8 weeks



Bots, Gastrophilus

M8k 200; Mk 194; EM&S 601; M241, 227, C2T 323, 697; POP



· Adult fly lives 2 weeks

- Gastrophilus intestinalis (common bot fly)
- · G. nasales
- · G. haemorrhoidalis
- (nose or lip bot) Lay eggs in summer months
- Annoyance of flies
- Attachment to stomach mucosa - Ulceration & erosions
- Asymptomatic Mild gastritis
- · Stomatitis & pain on eating
- Larvae in feces
- Infection often
- assumed in fall



Assume infection of most horses in temperate areas by end of summer

- Anthelmintic Tx in late fall or early winter after frost kills flies (See pg 12)
- M/ need treatment at beginning of bot season
- Ivermectin or organophosphates (trichlorfon, dichlorvos)



worms M8k 201; Mk 195; EM&S 602; M 241; C2T 323; C3T 679; Pop Θ

Dx: Assume in fall Tx: Ivermectin

CS: Mild

Stomach parasite

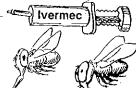
- Stomach worm Habronema muscae
- H. microstoma Draschia (H.) megastoma
- Adults worms 6-25 mm • "Summer sores": larvae m/ invade skin wounds
- Eye: conjunctivitis
- · Catarrhal gastritis w/ heavy mucus production when large numbers
- Draschia spp. most severe
- Tumorlike enlargements (4") in stomach wall
- Rarely rupture (if do, fatal peritonitis) Other habronema spp.
- asymptomatic 'Summer sores" migrate to

wounds (see pg. 284) Conjunctivitis if migrate to eye (see pg 295)

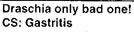


Gastric lavage m/ find eggs or larvae

Anthelmintics & management Ivermectin every 8 weeks



- Horse ingests flies/larvae Draschia (Habronema) occur in tumorlike
- Others free on stomach mucosa



Dx: Difficult Tx: Ivermectin



when in mouth

Bot eggs on hair

Life cycle

- Adult flies live 2 weeks
- Eggs glued to hair (G. intestinalis forelegs & shoulder; G. nasalis - submandibular area; G. haemorrhoidalis - lips)
- Larvae hatch, enter mouth & embed in tongue or mucous membrane for 1 month
- Pass to stomach, attaching to cardiac or pyloric portion (G. nasalis - proximal small intestine) 8 months
- Overwinter in stomach Pass in feces, pupate for 3-5 weeks

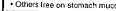




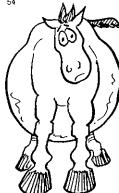
Life cycle

 Eggs or larvae ingested by larvae of house or stable flies (intermediate host)

swellings on stomach wall



dilatation. Reflux gastritis, Gastric impaction



Extensive ulceration of squamous mucosa (margo plicatus dorsally)

Draschia (Habronema) megastoma, weakening wall (rarely ruptures)



Grain overload CS: Gastric reflux - Violent colic - Laminitis Nasogastric tube

Rupture - Ingesta in Abdomen - Euthanasia

- Laminitis, check digital pulse



- of ingesta (impaction) or fluid (reflux) Abdominocentesis
- Rupture: ingesta present (repeat bec. if Dx, euthanasia)
- CBC w/ rupture: severe leukopenia w/ degenerative left shift

RUPTURE:

· Euthanasia (even if Dx early because stomach not accessible to surgery)



Gastric rupture:

- Classic history of acute pain suddenly relieved
- . In 20 min starts sweating
- Rapid progression to shock & death
- Mucous membranes purple; extremities very cold
- · Universally fatal w/ massive contamination of peritoneum
- Successful repair of seromuscular tears w/o contamination

Stenosis or obstruction of cardia

- Phytobezoar (FB), gastric ulcers
- CS: Ptyalism, retching & regurgitation
- Dx: Contrast X-ray
- Tx: Surgery





Pyloric stenosis

• Rare in horse; Congenital pyloric stenosis rare; 2º to gastroduodenal ulcers or fibrotic mass in pylorus

I2M 717; M 225; Pic 43

32 Diarrhea in Foals DIGESTIVE SYSTEM Condition Facts/Cause Presentation/CS Diagnosis Very common < 6 mos Diarrhea Diarrhea in · History: age related, FPT, ABs Fluid therapy (see box) Potential for systemic diz (neo- Depression Colic Intestinal protectants foals nate > adults) leading to death Dehydration · Blood cultures (bacteremia) absorbents M8k 246; Mk 184, J2M 391, 723; Mild & transient in nature, usually Colic · Often bacteremia associated Systemic ABs IM 343; C4T 631; C3T 449; M 235 w/ FPT (see box) FPT (failure of passive transfer) W/ Abdominal distention (if bacterial) (see box) **** Blood cultures first bacteremia & diarrhea < 7 d-old diarrhea w/ depression or Dehydration Broad spectrum ABs loss of suckle reflex, immediate Metabolic acidosis Monitor renal function because of dehydration evaluate for FPT (pg 39) w/ nephrotoxic drugs Prerenal azotemia • Hypoglycemia significant (no fat to compensate for not eating) Which m/ Bacteremia often associated w/ cause death FPT (failure of passive transfer) < 7 days-old</p> Depression & loss of suckle Colosti reflex +diarrhea Small & large intestinal diz Prevention: Causes see pg 337 Needs immediate evaluation No crowding • Failure of passive transfer (FPT) (pg 39 Separate age groups Foal heat (pg 35) Sanitation & hygiene • Nutritional (pg 38) - Eliminate contamination - Milk (lactase defc) Adequate colostrum!!! Milk replacers - Concentrates Parasites (S. westerii, large & small strongyles, Cryptosporidia) (pg 36) Viruses - Rota, corona (pg 38) Bacterial Salmonellosis, Clostridium perfringens, Very common, FPT Rhodococcus equi, C. difficile, Campylobacter B. fragilis, CS: Diarrhea Mechanical obstructions (FB, volvulus, intussusception, Dx: Hx, CS, FPT Tx: Fluids, ABs FB (foreign body) Antibiotics (pg 43) Prevention: Colostrum

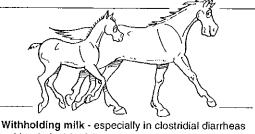
Treatment - Neonatal Diarrhea

Fluid Therapy (IV or oral)

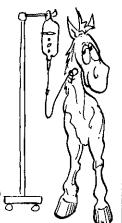
- Lactated Ringers (LR) (bec. most have metab. acidosis)
- Hypokalemia (→ potassium) add KCl to fluids (15-29 mEg/L) (milk has K+ so only if not suckling)
- Glucose if not nursing (add 1-2% dextrose to fluids in those of concern)
- Bicarb (1.3%) if Lactated Ringers can't handle acidosis (give slowly & separate from Lactated Ringers due to binding effeC3T of Ca)
- 3-5% dehydration use 3 mEq HCO3
- 5-8% use 5 mEq HCO3
- 8-10% use 7 mEq HCO3
- 1.3% gm/dl is isotonic HCO3 = 13 g/100 ml
- Must be cautious or will "knock" pH the opposite way

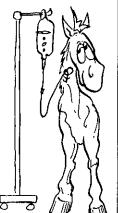
Plasma therapy (Expensive, 450 ml costs \$100 so have plasma donors)

- Hypoproteinemia 2-4 L (< 2 g/dL albumin) in FPT
- Monitoring amount needed based on PCV & total protein. 50-100 ml/kg/d maintenance



- Muzzle foal 8-12 hours
- · Milk out mare
- . Oral fluids by stomach tube (call replacers, for short periods bec. insufficient energy)





33

Intestinal protectants/absorbents

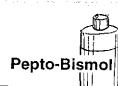
- Bismuth subsalicylate (Pepto-Bismol®) (neutralizes bacterial toxins & antisecretory effeC3T [antiprostaglandin])
 - Use 24 hr & stop if diarrhea persists, makes feces
- dark in color, making feces occult blood falsely positive
- Give 1 liter on call (have owner give 4 oz. [120 mls] QID) (if in hospital 1 liter QID)
- Kaolin + pectin (Kaopectate®)

Flunixin meglumine (Banamine®) - anti-endotoxemic drug, interfering w/ cyclo-oxygenase

 Anti-endotoxemic dose: 0.25 mg/kg, lower than analgesic dose

Antibiotics

- Indications
 - Septicemia or toxemia
 - FPT (Failure of passive transfer)
 - Immuno-compromised
- Prophylactic
- Contraindicated if cannot recognize diz cause
- Disadvantage: disrupts normal gastric flora, allowing pathogens to "set up house"
- Blood cultures first
- Broad spectrum antibiotics
- Monitor renal function because of dehydration w/ nephrotoxic drugs





35

Self limiting

Looks like yeast

Resistant to environment

(up to 9 months)

more investigation

Eimeria leukarti - commonly found in

feces of foals 30-125 days old

- Unlikely to cause diarrhea

Arabian foals w/ combined

immunodeficient diz

Self limiting

Looks like yeast

Presentation/CS

Condition

Nutritional diarrheas

M8k 247; Mk 184; I2M 393; IM 345: C3T 449

Self limiting

Facts/Cause Over-ingestion of milk (see box)

- Ingestion of foreign material
- · Mild & transient, except if systemic Signs (systemic CS, usually infectious causes) - Large quantity of coarse feedstuff (irritation)
- . Generally by 4-5 weeks mature enough to handle these feedstuffs better
- Coprophagic, ingested parasites, irritates mucosa

Parasiles a problem for longer period than feedstuff

- Orphan foals introduce to milk slowly (20-25% body wt., fed at 1-2 hr intervals)
- Milk replacers not readily available, best is nurse mare, or goat
 - . Teach to drink out of bucket or to suckle dairy goat (also provides company)

• Lactase deficiency (temporary): CHO intolerance - assoc. with viral diz (lactase produced in mucosal cells)

00 POAL 3 MILL POWER

so milk into colon, osmotic diarrhea)

 Self limiting, no Tx if transient Lact-Aid® to milk prior to feed-

ing (esp. if viral diarrhea) (in animals being force fed due to not eating)

Over-ingestion of milk (mare separated & rejoined, e.g., foal heal mating) (overwhelms intestinal ability to absorb,

Septicemia in foals

Mk 308; IM 309; C4T 595; C3T 435; C2T 222; M 287; EM&S 159, 1812; Pic 69; Pop35-1/98

- Very important in foals
- 1st week of life (1-5 days)
- Mostly generalized multiple organ system; some localized
- Failure of passive transfer (FPT) (most important predisposing factor)
- Overcrowding & poor management
 Hx of abortions, death of foals, dystocia, premature foal
 - Bacteria:
 - E .coli
 - Actinobacillus equuli
 - B-hemolytic Strep.
 - Klebsiella
 - Salmonella

- Systemic diz acute
- Behavioral CS
- Lethargy, weakness, loss of suckle reflex, inability to stand
- Meningitis
- Pneumonia
- Uveitis
- Acute lameness (joint ill), swollen joints
- ♦ HR. RR
- Seizures paddling, rigidity & opisthotonus
- Progresses to coma
- Diarrhea w/ or w/o colic terminally
- Blood cultures (take before ABTx) - May take 5-6 d to grow TTW (transtracheal wash) because of

Hx & CS in conjunction w/:

- high incidence of pneumonia
- CITE test: immunoglobulin titers Leukogram: early normal, terminally neu-
- tropenia w/ f bands & toxic changes
- ★ Fibrinogen m/b > 500 mg/dl
- Hypoglycemia

· Hx, CS

hourly

Lactase deficiency

orally & check

glucose levels

Administer lactose

- Temperature no help: normal, ↑ or ↓ · Auscultation no help - even in pneumonia because abnormal sounds m/ not be heard, unlike adult
- Rads: chest: atelectasis, † density

- Broad spectrum ABs
- Na or K Penicillin IV
- + Gentamicin as wait for cultures - 6-7 d to 2 wks, dep. on clinical response
- Plasma & fluids (immunoglob. deficiency, septic shock & metabolic acidosis, hypoglycemia)
- Diazepam (Valium®) for seizures

Prognosis:

- Poor, mortality high w/Tx
- · Recovery: polyantnritis, chronic debilitation



FPT, Mortality hi w/ Tx CS: Systemic, Behavior

Dx: Hx, CS, Cultures, TTW, CITE test

Tx: ABs, Fluids

E. coli Mk 184; IM 344

- Not an important cause of diarrhea in foals
- Associated w/ septicemia in FTP (failure of passive transfer) - Seed multiple organ systems
- Diarrhea can occur if animal becomes septicemic
- #1 blood culture isolate from septicemic foats

DDx of systemic diz

- Neonatal maladjustment syn. (pg 270) Combined immunodeficiency (pg 303)
 Autoim, hemolytic anemia (pg 141)
- Congenital white muscle diz
- Uroperitoneum (pg 154)
- Retained meconium (pg 83) Equine herpes intec



Failure of passive transfer, **FPT**

Mk 1519; IM 1610; C4T 581. 582; C3T 422, C2T 210; EM&S



- Born w/out circulating immunoglobulins
- · Colostrum (first milk) contains high % of immunoglobulin (passive transfer)
- Failure #1 cause of mortality in first week
- Intestinal absorption limited to first 12-18 hr of life (see box)
- Specialized intestinal epithelial cells
- Absorb Large proteins intact
- (immunoglobulins) Cells replaced in 36 hr
- 18 hr is a better cut off for good colostrum intake
- Stress or steroids m/ cause premature closure of specialized cells
- Peak immunoglobulin levels 12-24 hr after intake, protection against pathogens Prevention:

- Diarrhea & respiratory diz
- Mare udder distended (foal not suckling)
- Bacteremia:
- Depression
- Weakness
- Rapid or labored breathing
- Diarrhea
- Anorexia
- Injected sclera
- Survivors of bacteremia, sequelae:
- "Joint ill"
- Meningitis
- Panophthalmitis (inflammation of all structures of eye)

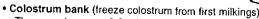


· Hx, CS

- Can't be determined by PE Laboratory evaluation (see box)
- CITE test: measure immunoglobulins
- If less than 24 hr feed colostrum
- 1 L w/in first 8-12 hr of birth
- > 24 hr: plasma or serum transfusion (20-40 ml/kg IV)
- If diseased, not as successful

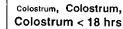
Prevention:

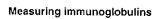
- Most feed colostrum automatically w/o testing
- . Make sure suckle in first 6 hours of life
- Leaving foal w/ dam gets better absorption
- More difficult to monitor if nursing mother
- If not, force feeding w/ bottle is preferable over stomach tube (better absorption)
- 2 liters of colostrum in first 4 hr after birth



- Thaw or microwave & feed

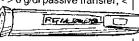
Colostrometer: measures specific gravity (immunoglobulin levels) of colostrum, < 1.050 associated w/ low antibody concentrations

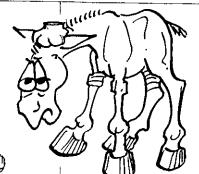


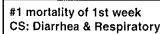


- Normal: 12-1500 mg/dl immunoglobulins Failure of passive transfer = < 200 mg/dl
- Partial failure = 200-400 mg/dl
- Field tests done at 12 hrs old (6 hr to get immunoglobulins into blood) - if failure, give colostrum
- Zn sulfate turbidity test field test, 1 hr, cheap
- CITE test, relatively expensive
- Sodium sulfite turbidity test field test (1 hr) looking for turbidity
- Single radioimmune diffusion test takes 24 hr, so not a field test (the standard test) Refractometer (total serum protein) - > 6 g/dl passive transfer. <

5 inadequate, 5-6 questionable







Dx: Hx, CS, Iq Tx: Colostrum or Plasma Prevention: Colostrum, Fluids

The same of the sa

DIGESTIVE SYSTEM

Condition

Adult diarrhea

M8k 241; Mk 184; I2M 723; IM 117, 659; EM&S 649: M 235

 Passage of fecal material w/ ♦ water content

 Poorly understood in horses · Often treated symptomatically

Facts/Cause

· Chronic diarrhea m/be associated w/ many dizs

• #1: Salmonellosis

· Colitis: almost exclusively large intestine (small intestine diarrhea compensated by large intestine, absorbs excess water, stomach diz almost never causes diarrhea in adults)

Stress diarrhea - change in diet or overfeeding

 Endotoxemia frequently accompanies diarrhea (mucosal damage) Chronic malabsorption (mucosal

Often cause not determined

DDx see pg 337

Presentation/CS

· Acute, peracute or chronic • Perfuse watery diarrhea

Often foul smelling

Dehydrated

- Injected vasculature of mucous membranes

- Prolonged capillary refill time

Peracute picture w/o diarrhea

- Very low volume of diarrhea w/ no systemic signs

Often die w/in 12 hr or sometimes 1-2 hr w/ no diarrhea

#1 cause is Salmonella

Chronic: diarrhea > 1 month (pg 46)

· Many cases - cause not determined

Diagnosis

History:

- Change in diet, deworming program

- Acute or chronic

• PE (see box)

- Dehydration

Purplish to brick-red mucous membrane

• Lab:

- Initially ♦ WBCs & PMNs (Salmonellosis & other acute causes)

- Toxic changes

- Hypokalemia

- Hyponatremia

- Hypocalcemic

- Metabolic acidosis (total CO2 or bicarb or blood gases)

- Prerenal azotemia w/ BUN nor

 Multiple fecals for Salmonella on all horses w/ diarrhea

 Potomac horse fever areas Paired acute & convalescent blood samples IFA for antibodies to Ehrlichia

Chronic diarrhea > 1 mo

Often perplexing diagnostic problem (see pg 46)



Fluids 1° importance - IV initially

Plasma (3-10 LIV initially for hypoproteine-

Nutrition

 NSAIDs (Flunixin meglumine [Banamine®] for effect on endotoxins)

- Ehrlichia - tetracyclines IV

- Salmonellosis - detrimental?

- Septic, neutropenic horse - broad spectrum ABs, justified (prevent bacteremia)



Prevention:

No crowding

Separate age groups

Sanitation & hygiene

- Eliminate contamination



#1 Salmonellosis CS: Watery feces

Dx: Often undiagnosed, Multiple fecals

Tx: Fluids - Fluids - Fluids!

Diagnosing adult diarrhea

- Change in diet, deworming program

- Acute or chronic

- Single or multiple cases

- Concurrent disease

- Medication

- Antibiotics (lincomycin, tetracyclines & erythromycin implicated), discontinue if diarrhea develops

- Exposure to toxins

PE (physical exam)

Vital signs (often normal in chronic diarrhea)

Bectal exam

- Note any weight loss

- Hydration (skin turgor, gum moisture, capillary refill time)

- Septicemic CS (injected sclera, conjunctiva, muc. membr., capillary refill time)

 Toxemia &/or dehydration often accompanies acute colitis, Salmonellosis, ehrlichiosis & many other acute diseases

Cardiovascular system (HR, pulse, capillary refill time)

- Laminitis CS (tameness, digital pulse, temp. of foot)

Fecal exam

Gross inspection - blood

- Microscopic exam - ova & protozoa

- Multiple cultures for Salmonella & Campylobacter if onset acute &/or febrile, or fibrin & mucin in feces

- Fecal occult blood

Blood

, ↑ PP/ total solids (dehydration)

. † fibrinogen indicates inflammation

, ↓ TPP: protein losing enteropathy

, WBCs, differential & morphology



- Acid-base values (Chem Micro CO2 device) if acute & dehydration or toxic, not for diagnosis, but for supportive therapy

· Serum -Chemistries

. Electrolytes (Na, Cl, K & Ca), nonspecific

. BUN: prerenal azotemia common w/ dehydration, recheck after hydrated

. Albumin/globulin values: hypoalbuminemia seen in chronic protein-losing (granulomatous bowel diz, oure toxicity, acute colitis)

IgA, IgM, IgG levels; immunodeficient in chronic diarrhea

. Paired sera for Ehrlichia risticii titer in acute diarrhea w/ fever

Paracentesis: chronic cases

Cytology, protein & fibrinogen (to rule out peritonitis, tumors & mesenteric abscesses) creatine, blood pH, or total CO2

Function & absorption tests:

 Oral glucose or xylose small intestine absorption tests - chronic especially w/ hypoalbuminemia; if indicates malabsorption, biopsy gut or evaluate Tx w/ antheimintic after 60 d

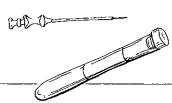
 Liverfunction tests, BSP half life if hypoalbuminemia or elev. liver enzymes (in enterocolitis elev. liver enzymes secondary)

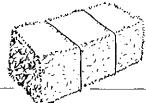
• Rectal biopsy: microscopic & FA for immune mediated disease . Culture for Salmonella

Response to Tx in undiagnosed chronic diarrhea

- Put on grass hay

- Stop "bute" if suspected



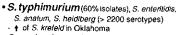




Condition

Salmonellosis

M8k 241, 120; Mk 184, I2M 723; IM 660; EM&S 643; M 235: Pic 69



Facts/Cause

- Severity depends on # of org., virility
- Transmission:
- From environ. (fecal/oral, feed & water)
- Passive & active carriers (aerosols) 1-5% normal horses asymptomatic carriers Shed for weeks, so isolate others
- Predisposing factors
- Stress: in 2-4 d see Salmonellosis
- . Esp. surgical stress (horse clinic prblm.) . Overtraining, deworming, hot weather, transport, food deprivation, changes in feed
- Antibiotics changes in GI flora
- Pathophysiology:
- Inflammation of bowel wall
- Organism invades GI mucosa (ileum, colon & cecum) Multiples intracellularly
- Disrupts normal absorption, secretory mechanisms in crypts
- Loss of protein into GI lumen
- Endotoxin into gen. circulation, passes to regional lymph nodes
- 10 d, septicemia & diarrhea, unlikely to live (even w/Tx)

Presentation/CS

- 1) Asymptomatic carriers
- Organisms in GI, active or passive shedders W/ disruption of GI m/ develop diz
- 2) Chronic diarrhea

4 clinical syndromes

- Septicemia: most commonly occurring in foals
- > 10 d of septicemia & diarrhea = death
- 4) Acute colitis peracute
- Most common syndrome
- May die w/n 12 hr
- Sudden onset of pyrexia (fever)
- Off feed
- Prox. enteritis w/ gastric reflux
- Colic (abd. pain w/ or w/o diarrhea)
- Profuse diarrhea
- Improve in 7-10 d w/ Tx

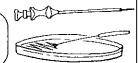


DDx:

 Acute obstruction (pg 64), nonresponsive to analgesic therapy - mistaken & taken to surgery, culture shows positive for Salmonella

Diagnosis

- · Hx, CS Fecal cultures (5 negative before Dx Salmonella free)
- Difficult to grow, 20 gr. of feces needed Clinical isolate organisms when feces starts to firm up
- Special enrichment broth, tetrathionate & selinite agar plate
- Rectal biopsy more sensitive than fecal in picking up Salmonella Do both if think it's being shed into envir.
- PM (post mortem)
- Catarrhal enteritis
- Necrotic hemorrhagic lining (distal small intestine, cecum & large colon)
- Lymph nodes enlarged & bulge on cut surface
- Adhesions of large colon (tre mendous damage, get leakage to serosal surface)
- · Lab:
- Neutropenia w/toxic degenerative left shift ∔ in Ña, K, Cl
- Metabolic acidosis
- 4 in protein due to loss of albumin
- Prerenal azotemia in creatinine
- · TPP: m/be up, down, or normal



Treatment

- Fluids: 1° importance - IV initially
- Plasma (3-10 L IV initially for hypoproteinemia TPP < 4 g/di)
- Nutrition
- NSAIDs (Banamine® [Flunixin meglumine) for effect on endotoxins & pain)
- DMSQ IV, antiinflam., antibact. & O2 free-radical-scavenger
- ABs m/b detrimental
- AB administration often precedes diz. doesn't appear to alter duration of diz, rapid resistance to ABs
- Septic, neutropenic horse-broad spectrum ABs justified (prevents bacteremia)



Potomac horse fever, Ehrlichiosis.

Stress (Sx) - Carriers

Dx: 5 fecals, biopsy

CS: Diarrhea

Tx: #1 fluids

Ehrlichia colitis. Equine monocytic ehrlichiosis

M8k 243; Mk 185, I2M 724 1640; IM 660; C3T 92; C2T 250; EM&S 651; M 235



River valleys - Summer - Ehrlichia

CS: #1 Laminitis - #2 Diarrhea

Tx: Fluids, Oxytetracycline

- Reported in Potomac River Valley
- 1° river valleys (more in the North)
- Seasonal: July & August (May Nov.)
- · Transmission unknown
- Multiple vectors (Dermacenter ticks?)
- Noncontagious, occurs sporadically, nonhorse to horse transmission
- Endemic to certain farms
- · Ehrlichia risticii
- Predilection for monocytes
- Incubation period 10 d
- Mortality rates between 20-35%
- 1° cause of death due to laminitis, not enterocolitis

- Clinical syndrome similar to Salmonella
- Acute onset of depression
- 107° F (important for Tx)
- Off feed
- 24-72 hrs: Diarrhea, perfuse, watery, not as fetid as Salmonella
- Colic preceding, during, or following diar-
- Often resolves w/in 1-10 d w/out specific therapy, but supportive w/ IV fluids
- Laminitis 3-5 d after diarrhea
- 1° cause of death due to laminitis, not enterocolitis
- Sequelae: - Ileus, toxemia, DIC

- 50% of cases undiagnosed
- Hx & CS
- River valley location Serologic titers
- · Indirect immunofluorescence antibody test TOC
- 4-fold rise in titer
- Vaccine makes Dx difficult due to titer ↑& those vaccine still can develop diz
- Absolute peripheral monocytosis (DDx from Salm.)
- Leukopenia (as in Salm.)
- PM (post mortem)
- ID organism in monocytes of GI (not in peripheral blood) & in enterocytes (intestinal epithelial cells) of mucosa of lg. intest. (colon & cecum)
- No severe tissue damage
- EM: find damage to enterocytes associated most commonly w/ disruption of secretory mechanisms

In crypt area

Fluids

DOC: oxytetracycline, 7-10 d. IV sid effective w/in 24 hr after temp, elev (prevents progression of diz); m/not be showing signs of diarrhea at this time so hard to recognize unless taking temp.

- Once develop enterocolitis tends to follow course, drug not effective
- Oxytetracycline may predispose to Sal monella if carrier
- Supportive care similar to any cause of enterocolitis
- Recurrence common



Prevention:

- Vaccine bacterin administered prio
- to hi season 2 shots 3 wk apart Short lived, protected for 4 months
- Revaccinated yearly
- Not completely efficacious
- Durino outbreak, vaccine not effective - 3-4 weeks post vaccine to develop
- Incidence of diz decr. since vaccine. but keep getting new areas popping up



NSAIDs

IM2 725; IM 661

 Excessive doses of nonsteroidal anti-inflammatory drugs (NSAIDs) associated w/ diarrhea secondary to development of hypoproteinemia & cecal & colonic edema. NSAIDs inhibit prostaglandin synthesis, disrupting mucosal blood flow & resulting in hypoproteinemia & septicemia. Many are slow to respond to Tx (long intensive care)

Lincomycin Mk 1446, I2M 725; IM 661; C4T 666

Dx: Hx, Monocytosis

An antibiotic

· Contraindicated in horse because severe, m/b fatal colitis may develop

Tetracyclines Mk 1438; 12M 725, IM 661 • M/ cause severe & even fatal diarrhea in horse, especially if severely stressed or critically ill

Trimethoprim-Sulfa, Erythromycin & Penicillin IM2 725; IM 661 • Associated w/ mild-to-moderate diarrhea in small

percentage of horses treated



Condition Chronic

diarrhea I2M 728, IM 663; C2T 100; M





Dx: \$ - ?

Facts/Cause

Most frustrating to Dx & Tx

- Diarrhea > 1 month
- Disease of large colon
- 3 groups of chronic diarrhea
- Chronic inflammation
 Histologic changes of colon
- 2) Physiologic disruption
- No morphologic changes in colon Abnormal volatile fatty acid synthesis &/or absorption from cellulose
- Disorder of other systems besides GI, small % (CHF & hepatic diz)

Presentation/CS

- Diarrhea > 1 month Hydration m/b OK if drinking keeps up w/ loss
- Moderate weight loss
- ± Signs of septicemia (injected, congested or hyperemic mucous membranes
 - Feces soft, slightly watery, m/b intermittent

Hx. CS (> 1 mo)

Attempt to differentiate inflammation from physiologic causes

Diagnosis

- Work up m/ be extensive & expensive - M/ never Dx cause (idiopathic)
- CBC for chronic inflammatory signs
- Chronic inflammatory anemia (RBCs & PCV)
 WBC normal to moderately
- Fibrin normal to 1
- Normal CBC doesn't rule out inflam. (m/be localized or mild)
- Peritoneal fluid analysis
- ± † protein &/or WBC (indicates inflammation), often inflammation of colon - values normal
- Serum chemistry
- Varies severe to mild chem, abnormalities (hyponatremia, hypochloremia, azotemia & metab. acidosis)
- Total serum protein usually decr. due to leakage from damaged colonic capillaries (hypoalbuminemia)
- + hepatic enzymes indicate hepatic diz (SDH, GGT, AST & serum bile acids)
- Feces:
- Salmonella m/ need 15 fecal cultures to get a positive
- Rotavirus in weanlings (ELISA)
- Rectal biopsy & culture for Salmonella

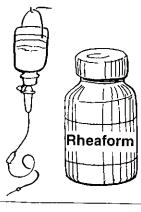
OFTEN THE ABOVE DOESN'T TELL CAUSE! (idiopathic)

- Exploratory laparotomy
- Masses or abscesses
- Biopsy of colon & cecum & mesenteric lymph nodes for histopath, & culture of Salmonella
- No abnormalities except water in large colon (idiopathic)

Treatment

- Empirical (from experience) Inflammation often
- untreatable (lymphosarcoma, granulomatous enteritis)
- Chronic Salmonella generally doesn't respond to antibiotics)
- · Chronic parasitism anthelmintics
- Sand: Metamucil®
- Bismuth subsalicylate effective in some cases (inhibits prostaglandins)
- Rheaform® (lodochlorhydroxyguin) for some cases of maldigestion of cellulose (mechanism unknown). If effective, must be continued or diarrhea resumes

Empirical Tx



Diarrhea > 1 mo Often idiopathic

Tx: ? - Empirical

DDx:

- Idiopathic (unknown) most common (pg 47)
- Infectious agents
- Chronic Salmonellosis (pg 48)
- Chronic parasitism Strongylus vulgaris, sm. strongyles
- Rhodococcus equi infection (pg 48) - Rotavirus in weanling foals (pg 38)
- Noninfectious inflammation
- Infiltrative disorders
 - . Granulomatous enteritis
- Lymphosarcoma (pg 30, 51)
- Sand enteropathy (pg 49)
- Physiologic disruption Noninflammatory disorders
- Abnormal fermentation of cellulose
- Small % disorder of another system besides GI
- Congestive heart failure 2° hepatic fibrosis (pg 125)
- Chronic hepatic diz (inflam., fibrosis or fatty infiltration) (pg 90)
 - Chronic hepatopathies & cholelithiasis

Idiopathic chronic diarrhea I2M 728, IM 663

• Most common cause of chronic diarrhea

- Stress prior to onset of CS
- . M/b due to ↑ volatile fatty acids in large intestine so # fermentation (theories)

- Fecal cocktail Fresh feces from normal horse, or from freshly dead animal that has not been ill)
- Mixed w/ warm water, buffered w/ bicarb Via stomach tube
- Warn owner of possible more severe diarrhea if Salmonella in cocktail feces



- Hx of stress prior to onset of CS
- PM: no gross histologic abnormalities
- No lab abnormalities
- Animal can compensate for loss of electrolytes
- No changes in CBC
- Exploratory laparotomy
- No abnormalities except water in large colon
- Edema in lamina propria. no specific cause



(which may exacerbate problem) resolves while on drug, once withdrawn, recurs Eliminating grain in diet - in

Rheaform® lodochlorhydroxyquin

- some cases diarrhea will resolve Alter environment (stress) () }
- Nonspecific
- Fecal cocktail (see box)
- Corticosteroids (Dexamethasone & prednisolone due to the theory that it may be hypersensitivity reaction to feedstuff

Prognosis:



- Routine deworming every 8 weeks (grazing horses assumed to
- be infected) see pg 14 Foals: 6-10 wks old
- Treat all horses on pasture
- Anthelmintic resistance of cyathostomes an emerging problem, see pg. 14
- Resistance to benzimidazoles except oxibendazole
- Ivermectin, Pyrantel pamoate (Strongid T®), oxibendazole & piperazine effective
- Periodic fecals to check
- Corticosteroids



Small strongyles,

Dx: Hx, CS

Px: Poor

#1 Chronic diarrhea, Stress

Tx: Rheaform, Fecal cocktail

Cyathostomiasis Mk 196; IM 1517; EM&S 649

Strongylus

Blood worms.

Sclerostomes,

Dx: Difficult

Red worms

Palisade worms.

Large strongyles

CS: Colic, diarrhea Tx: Ivermectin

vulgaris



· Chronic diarrhea due to digestion of

Cyathostomes

- larval stages in Spring Encyst in gut wall
- Rapidly leave gut in spring
- Rehydrate themselves w/ oral solutions
- Large strongyles see pg. 75
- enteric artery & its branches causing parasitic thrombosis & arteritis leading to: Infarction & ischemia to segments



Colic

. S. vulgaris migrate to cranial mes-Gangrenous enteritis

Chronic diarrhea

Normal appetite

Mild weight loss

Performance poor

. M/ resolve by itself

Severe weight loss

responsive diarrhea

Not extreme fluid loss

• M/b severe non-

Hypoproteinemia

Ventral edema

- Intestinal stasis, torsion or intussusception & rupture
- Acute or chronic diarrhea
- Mild infections
- Dullness, progressive weight loss, intermittent colic
- Due to larvae in GI tract
- Intermittent signs of colic
- Hypoproteinemia

47



+ Hx, CS

Strongyles in

feces, L4 in feces,

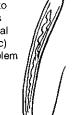
L4 on rectal sleeve

- Ova in feces
- Fecal culture of larvae to DDx different strongyles
- Rectal palpation of cranial mesenteric artery (if colic)
- · Suspected parasite problem









DIGESTIVE SYSTE

Rhodococcus equi

I2M 728; IM, 511, 344, 663



salmonellosis

Consider in all cases of diarrhea CS: Diarrhea, Weight loss, Death Dx: Difficult, 15 fecal cultures

Tx: ABs useless, Fluids, Rheaform

Chronic

Mk 184; I2M 723;

IM 663

Facts/Cause Chronic diarrhea in foals 1-4 months, uncommon in adult

- 1º affects lungs pneumonia
- Transmission: oral
- · Endemic to certain farms
- Damages intestinal villi
- Granulomatous & abscesses in small & m/b large intestine

Presentation/CS

- Asymptomatic Acute
- Abdominal pain in acute phase
- Diarrhea
- Chronic (necrosis & ulceration)
- Weight loss
- Diarrhea intermittent (flare ups of bacteria)
- Respiratory signs

of asymptomatic & symptomatic animals) Usually done by rule out . History of respiratory diz . Low on rule outs for chronic

Diagnosis

Difficult to Dx (isolated from feces



ABs (respiratory diz) - Erythromycin & Rifampin



Treatment

Prognosis

 Respiratory diz responsive to Tx; GI diz less so

Symptomatic

- ABs don't respond
- Oral fluids adequate (as apposed to those needing fluid therapy)

Rheaform® (iodochlorhydroxyquinone), slows diarrhea, chronic cases, may resolve while being used, reappears w/ stop. Slows down feces passage, incr absorption of toxins & m/ cause acute worsening. Stop gap measure





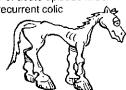
CS: Chronic diarrhea, Resp. diz

Dx: Difficult, Hx, CS

Tx: Erythromycin & Rifampin

- Important in adults & young Tremendous economic losses Weanlings > neonates
- Chronic diarrhea, adults >>> young
- Septicemia to endotoxemia, esp. FPT Multiple organs systems
- Small & large intestine (damages mucosa) Hard to disinfect area
- Adult w/out CS often source (carriers)
- S. typhimurium, public health
- Acute
- Bacteremia & septicemia w/out diarrhea m/b - Diarrhea (fetid smelling), blood & fibrin in
- Depressed
- Colic or abdominal pain
- Dehydration
- Bacteremia: lungs, joint, vertebral bodies, renal, uveitis or CNS CS
- Chronic diarrhea: adults > young
 - Diarrhea mild to moderate
 - Weight loss
 - History of acute episode m/be
- Dx of recurrent colic

Death



 Even more difficult to Dx than acute Salmonellosis

- 15 cultures to get positive m/b Shed intermittently (for normal diarrhea needed 5 negative cultures to rule out) Rectal swabs or biopsy less effective
- Blood cultures
- Hypoglycemic
- Loss of electrolytes into intestinal tract, 15 K* defc, metab. acidosis, + PCV (dehydration) & total protein or hypoprotein (if no immune response)
- Neutropenia toxic granulation of PMNs (not pathognomonic)



Prognosis: Poor Prevention: No vaccine

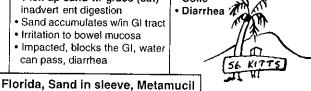
Sand enteropathy



• Pick up sand w/ grass (eat) inadvert ent digestion

- Sand accumulates w/in GI tract
- · Irritation to bowel mucosa
- · Impacted, blocks the GI, water can pass, diarrhea

Colic



 Hist. of sandy environment Sand in feces

- Feces + water in rectal sleeve
- . Sand settles in fingers



w/ sand Hemicellulose product via nasogastric tube or added to feed

Metamucil® (Psyllium)

hydrophilia mucilloid):

laxative, binds



Chronic liver disease

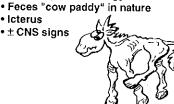


 Signs compatible to hepatopathv

- Icterus seen in sclera
- Hepatitis & hepatoencephalopathy (acute)

· Generally thin, lethargy

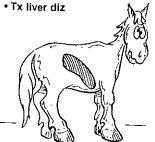
- Icterus
- ± CNS signs



•

Albumin m/b in chronic,

- in acute normal due to long half life
- Prolonged BSP
- Prolonged PT, PTT
- Abnormal liver biopsy
- Thin, poor condition
- D-xylose or glucose absorption tests (but results misleading, suggesting GI diz bec. of malabsorption - instead have altered blood flow)



Fungal-related diarrhea

- Very rare, Aspergillus fumigatus, a normal inhabitant of GI tract
- Problem when excess ABs use
- Diarrhea, generally mild & persistent in nature

Excess ABs



Abdominocentesis

Eimeria - problem in young horses primarily



See pg 53

- Inflammation of peritoneal serosa
- Exudate of serum, fibrin & prot. into peritoneal cavity
- Exudate can disrupt GI motility, resulting in diarrhea
- M/b diarrhea or normal fecal output
- Chronic or intermittent colic
- Depression, anorexia
- Weight loss
- Absent intestinal sounds (ileus)
- Mild dehydration
- Chronic tocalized
- Debilitating, but no pain or temp.

- # Fibrinogen & PMNs Cytology Rectal exam - Distended intestines

Tx underlying diz

Fluid, plasma (CO)

Broad spectrum ABs

Prognosis:

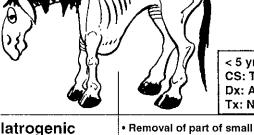
Chronic diarrhea w/ chronic peritonitis, responds poorly to

49



bowel

resection



intestine

effect

Removal of part of colon has no

< 5 yrs, Malabsorption CS: Thin, Edema, Diarrhea Dx: Absorptive tests, Rectal

Tx: No Tx

> 60% removal of bowel

- Get weight loss

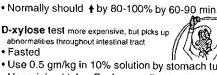
- Abnormal xylose absorption test

Can become malabsorption & hypoproteinemia

51

INCOME DE LA CONTRACTION DEL CONTRACTION DE LA C

- Ventral edema
- Thin



absorbed so misses problems further along GI

Fasted 18-24 hours

90 & 120 min

- Use 0.5 gm/kg in 10% solution by stomach tube
- Heparinized tube. Peak normally 60-120 min. after administration (checking plasma)

Glucose absorption test, cheaper than D-xylose, but rapidly

Feed glucose, 1 gm/kg in water sol. by stomach tube

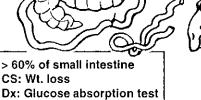
· Blood samples in grey top tubes. Samples 0, 30, 60,

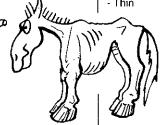
D-xylose

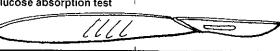
· Flat curve suggests malabsorption of small intestine, some problems in interpretation

· Hx, CS

Glucose absorption test







DIGESTIVE SYSTEM

Condition

C3T 225, C2T 81

Endotoxemia Mk 184; I2M 733; IM 668;

Facts/Cause

- Endotoxin from gram neg. org. Protective mechanisms of body normally keeps endotoxin out of blood
 - Mucosa barrier to endotoxins
- Kupffer cells (liver macrophages) remove if toxins get through mucosa
- Causes (see box)
- Any disruption of GI mucosa can result in absorption of toxins
- Local or systemic gram neg. infec.
- Endotoxins in general circula-
- Inadequate tissue perfusion
- Multisystem organ failure
- Cardiovascular collapse
- Acute renal failure
- Ileus
- Complication:
- Laminitis if get endotoxic for any reason
- Early, late & end stages (see box)

Bacteremia- bacteria in blood Endotoxemia - endotoxins in blood Septicemia - both in blood

Gram (-) organisms

CS: Multiple system failures, Laminitis

Dx: CS

Tx: Empirical Tx

Presentation/CS

- ↑ HR (tachycardia), ↑ RR · Initially depressed, then convulse,
- then coma Pyrexia in late (not end stage in which they
- reach a point where they can't thermoregulate so hypothermic
- Mucous membranes from pink to purple to brown
- Toxic line (over upper incisors) Colic
- Diarrhea
- Dehydration, anorexia
- Laminitis



Causes of Endotoxemia

Any disruption of GI mucosa can result in absorption of toxins

- Strangulating & nonstrangulating obstruction (pg 64, 68)
- Parasitism (pg 24, 36)
- Enteritis
- Colitis
- Grain overload (pg 29)
- Local/systemic gram neg. infec.
- Pleuritis (pg 118)
- Pneumonia (pg 118)
- Metritis (pg 183)
- Mastitis (pg 241)
- Septicemia
- Colitis (Salmonellosis) (pg 34, 42)

 CS mostly: Toxic line, +HR, RR; Gum color

- Lab:
- Neutropenia & margination w/ toxic changes, immature PMNs - myelocytes
- Glucose varies depending on stage
- Liver dysfunction can shown in liver enzymes, + BUN & creatinine



Early stage

- Hypoxemia PaQ2 low Hyperpnea (fast breathing)

- WBCs (Leukopenia: margination of PMNs)
- Thrombocytopenia
- † PCV, hemoconcentration
- † Glucose
- · Respiratory alkalosis

Late stage (reversal of early)

- + WBCs (Leukocytosis
- Glucose
 Metabolic acidosis that overrides res-
- piratory alkalosis Hepatic dysfunction, † liver enzymes
- Coagulation disorders developing, pyrexia, hypotensive, weak pulse

Empirical, unfortunately

- Underlying diz so treat other
- systems - ABs for gram - organisms. removal of foci of infection, if ischemic
- intestinal diz, correction Fluids: + blood volume (prevent or correct shock) (hypo-perfusion, hypovolemia) Underlying dizprocess cor-
- rect acid/base abn. Maintain or tolood flow
- Dopamine: † cerebral, coronary, mesenteric & renal blood flow Anti-endotoxins, not common, to neutral-
- NSAIDs (flunixin meglumine) to combat production of prostagtandins & endot-
- DMSO, scavenge toxic oxygen radicals
- Anti-pruritic
- Anti-inflammatory



- Endotoxic shock DIC (disseminated intravascular
- coagulation) Multi-organ failure
- Renal failure
- Acute respiratory distress syndrome
- Heart may fail CHF & simultaneously comatose
- due to lack of glucose to brain Once reach this point, death

Peritonitis

Mk 146; I2M 742; IM 674, 784, 124; EM&S 670; M 243 C4T 206; C3T 236, 285; C2T 79; Pic 89

Inflammation of peritoneal serosa

- Acute or chronic
- Diffuse > local (in horse usually diffuse because of inability to wall off)
- Exudate of serum, fibrin & protein into peritoneal cavity
- Exudate can disrupt GI motility, resulting in diarrhea
- Bacterial infection:
- Streptococcus equi &

Rhodococcus (Corynebacterium) equi,

- Streptococcus zooepidemicus

 Anaerobes, including Bacteroides fragilis;
- Omentum or mesentery m/localize a septic peritonitis (less so than cattle)

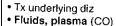
ACUTF:

- Severe colic rigid, "splint" abdomen, lie
- Ileus (# borborygmi)
- Anorexia, depression, lethargy
- Occasionally diarrhea, low volume,
- PERACUTE (intestinal rupture)
- Toxemia
- Hypovolemic shock, circulatory failure
- Death m/b 4-24 hrs
- · CHRONIC:
- ± Diarrhea or normal fecal output
- Chronic or intermittent colic
- Depression, anorexia
- Weight loss
- or absent intestinal sounds (ileus)

- · History, especially if postop · Abdominocentesis (difficult to
- interpret if postop) ↑ PMNs, macrophages (bacte-
- ria-free or phagocytized) & proteins
- Cytology: determine chronicity
- Gram stain bacteria Creatinine (suspect uroperi-
- toneum) - # Fibrinogen & PMNs: suggests chronic infection in horses & catlle, + PMNs w/ acute & chronic

infection, unlike dogs (+ w/ endotox-

- emia) Lab:
- Acute leukopenia w/ toxic left shift, neutropenia
- **♦** PCV (dehydration)
- Rectal exam:



- Long term broad spec. ABs (started, change if C&S indicates) (amineglycoside w/ pen., Metronidazole for Bacteroides)
- NSAIDs for endotoxemia & colic
- Exploratory Sx once stabilized, to repair defect
- Peritoneal lavage (gallons of sterile saline in & siphoned off) Lose protein, m/b hypoproteinemic
- Standing lavage in flank & out ventral Withhold food until condition
- improves
- Heparin to + adhesion formation Feed hi protein/hi CHO diet



Tx: Long-term ABs, lavage

Px: Guarded

- - Pyrexia (elev. temp.)
- watery feces, or constipation

- · Perforation of other infected organs (prostatic or hepatic abscess, pyometra)

• GI perforation or surgical contamination

- · Penetrating abdominal wounds Chemical insult (urine or bile)
- Neoplasia, visceral ischemia (transmural bacterial migration) Parasitic: verminous arteritis (s. vulgaris), larval migration (S. edentatus), perforating lesions (ascarids, tapeworm)



"Dilution is Solution to Pollution"

Prognosis:

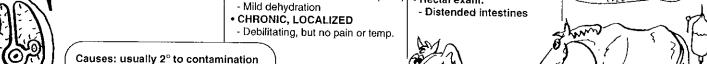
Septic peritonitis frequently fatal despite intensive Tx

Chronic diarrhea w/ chronic peritonitis, poor



2° to Contamination CS: Colic, ± death





DIGESTIVE SYSTE

Colic

M8k 165; Mk 168; IM 124, 680; I2M 749; EM&S 514; E 524; C4T 174; 190; M 206; S 348;



Facts/Cause Manifestation of abd. pain

- Visceral abdominal pain
- (pushing on abdomen doesn't cause pain unless touching effected organ) - Parietal abdominal pain: peritonitis
- . External palpation elicits pain
- Basic causes
- 1. Distention of gut w/ fluid, gas or ingesta
- 2. Pulling on root of mesentery
- 3. Ischemia or infarction
- Horses: low threshold of pain, mild indigestion causes pain
- If severe shock
- Infarctive diz (volvulus, torsion, thromboembolism)
- Severe visceral distention (extreme flatulence, impaction or dilation)
- Severe, unrelenting pain potentially life threatening
- 90% uncomplicated & respond to analgesic Tx

Presentation/CS Signs of pain variable,

- acute, chronic, or intermittent
- Restlessness
- Lying down & getting up - Rolling
- Sweating
- Kicking at abdomen
- Sudden drop to ground in pain
- Flank watching
- Abnormal posturing
- Self inflicted trauma
- Anorexia & depression
- # HR w/ weak pulse
- . ★ Capillary refill time
- Cold extremities
- Mucous membranes
- Bright red (vasodilation) followed by dark red (vasoconstriction) if cardiovascular in-
- Shock if severe

Diagnosis

- History (Hx) - Insurance
- 10 P's (see pg 56):
- Physical exam (attitude, teeth breath)
- Pain severity: continuous & progressive or intermittent
- Pulse: > 60 beats/min.; pass nasogastric tube . Digital pulse - laminitis
 - Perfusion mucous membranes,
- refill time, temp, extremities Peristalsis: absent; ominous
- Percussion: "ping" for gas
- Pass nasogastric tube
- Palpation per rectum
- Peritoneal tap
- Protein: normal < 2.5 g/dl Cells: normal betw. 5-10,000
- Bact, in WBCs or free
- Plant material w/ WBCs: rupture euthanasia
- PCV & TPR
- PCV ≤ 45% PCV: > 85% survival > 60% PCV: 25% survival

COLIC SURGERY OR MEDICINE? approach

depending on value of horse, if Sx not an option, treat medically or euthanize (insurance?)

Medical Tx of Colic

- Analgesia Xylazine DOC, Banamine®
- Decompression nasogastric tube
- Fluid therapy, oral or IV, usually both
- Gallon of mineral oil: need pump on nasogastric tube
- NO phenothiazine tranquilizers, no atropine
- Colic Surgery

- Do NOT do unnecessary laparotomies

- Earlier the decision for surgery, the better for recovery
 - Stabilize before surgery, pre-op fluids, ABs Ampicillin DOC, Banamine® Anesthesia: Xylazine/ketamine comb. w/ glycerol glycolate Isoflurane Dors, recumbency, ventral midline incision
- Work through all of the intestinal tract
- Decompress intestine
- Correct problem (Pelvic flexure enterotomy,
- Small intestine resection & anastomosis) Post op:
- . Incidence of infection high
- . Abdominal bandaging
- . Fast 24 hr post op, started back on small, amount of hay
- . Intestinal motility stimulators (drugs), metoclopramide



Abdominal pain CS: Sequela - Shock Dx: 10 P's Sx or Rx Tx?

90% Respond to analgesics

CS requiring surgery

- · Severe abd. pain poorly or nonresponsive to analgesics
- Discolored peritoneal fluid w/ prot., RBCs & WBCs
- Rectal exam: obstruction &/or displacement of viscera
- Progressive deterioration of cardiovascular status w/ abd. pain & uncertain Dx
- · Significant gastric reflux
- · Recurrent abdominal pain w/ uncertain cause

Prognosis:

- Colic: excellent, 90% respond to analgesics
- Surgery: Guarded to poor
- 50% recovery from colic surgery
- HR of 100, survival about 30%
- PCV > 60%, 25% survival

CAUSES - COLIC pg 337 or IM 127, 128

COMMON CAUSES:

- GI common causes
- Gas distention of intest., cecum or colon (pg 63)
- Hypermotility & intestinal spasms (pg 63)
- Feed impaction, constipation (pg 77, 78)
- Meconium impaction (newborn) (pg 83)
- Gastric ulcers (foal) (pg 26)

• Extra - GI

- Mesenteric abscess (pg 65)
- Ovarian tumor, abscess, or hematoma (pg 188)
- Parturition (pg 224)
- Acute hepatitis or hepatic lipidosis (pg 88)
- Diaphragmatic hernia (pg 71)
- Ruptured bladder (foal) (pg 155)
- Urinary tract, urolithiasis (pg 152)
- Uterine torsions (og 232)

• TOXIC CAUSES:

- Blister beetle (pg 45) - Warfarin (pg 143)
- Herbicides
- Lead (pg 269)
- Phenylbutazone or other NSAIDs (pg 27)
- Poisonous plants (pg 313-332)

LESS & UNCOMMON CAUSES

(See pg 336 or IM 127, 128)

From Large Animal Internal Medicine, B.P. Smith, Mosby Co., pg. 127-128

DIAGNOSIS (expanded) History (Hx)

- Nature & duration (continuous, severe colic worse than intermittent mild colic)
- Progression of pain
- . Medication (given by owner or another vet that could mask pain)
- Evaluate cardiovascular status before giving any drug
- Initiating factors: changes in diet, water consumption, exercise or weather; but m/ have no apparent initiating factors
- Deworming history
- Past episodes of colic, history of colic on farm?
- Breeding hist, (uterine torsions (late gestation), colonic impaction [postpartum], scrotal or inguinal hernias [stud])
- Feces: when last passed?
- Value of horse to owner, esp. if surgery possible
- Insurance: notify insurance carrier if surgery is considered, need permission



10 P's: Physical exam, Pain, Pulse, Perfusion, Peristalsis, Percussion, Palp. per rectum, Pass nasogastric tube, PCV & TPR & Peritoneal tap

1. Physical exam

- Attitude of horse
- . Fever &/or CNS depression rather than colic need medical Tx, notsurgical usually
- Abrasions, rolling, or standing calmly
- Xylazine, better if not used (sometimes so painful, hard
- Sweating

and the control of th

Abdominal distention

- Adult distended flank Ig. intest, disorder causing obstruction
- & distention Foal - distention w/ small or large intestinal lesions
- TPR Rectal temp.
- Check teeth for points (poor mastication m/ predispose
- to intestinal impaction) Foul breath - implies anorectic or spontaneously refluxing gastric contents (suspect a serious disorder)
- 2. Pain- severity; continuous & progressive, or intermittent

- 3. Pulse
- Pulse (rate & quality [strong, fair, poor]) 0 - Pulse rate > 52 beats/min of fair or poor quality =
- hypovolemia - If pulse > 60 beats/min pass nasogastric tube
- immediately Digital - check at pasterns & monitor due to associated between abdominal disorder & laminitis, endotoxemia causing both

4. Perfusion

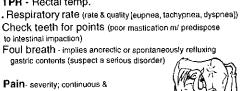
- Open mouth & check color
- of mucous membranes, capillary refill time . Pink, pale (peripheral vasoconstriction)
- . Light blue due to cyanosis - Purple due to pre-capillary sphincter failure, pooling of
- deoxygenated blood, shock due to strangulation - Feel temp, of extremities (ears & dist, limbs); Shock: ice cold

Hypovolemia &/or poor perfusion:

- Pulse rate > 52 beats/min of fair or poor quality
- Prolonged capillary refill time (> 2 sec)
- Poor skin turgor (dehydration) Cool extremities
- Need for fluid therapy



Rompur















Root of

Base of

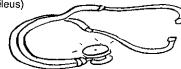
cecum

Urinary bladder

Colic

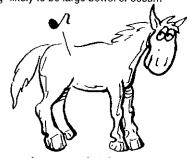
Mk 168; I2M 749; IM 680; EM&S 515; C3T 201; S Colic - diagnosis (cont.)

- 5. Peristalsis/auscultation: split the paralumbar fossa on each side into dorsal & ventral parts & listen for gurgling or silence
- Borborygmi (intestinal sounds)
- Present? a favorable sign (low-pitched progressive sounds in all 4 quadrants)
- . Absent? ominous, suggests absence of motility



6. Percussion: flick abdomen, detect gas distended organs

- Small bowel distention unlikely to hear
- "Ping" likely to be large bowel or cecum



7. Passage of nasogastric tube

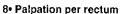
- Indicated for most, if not all colics
- If pulse > 60 beats/min pass immediately
- (can relieve pain & prevent stomach rupture)
- Stomach fluid (gastric reflux, siphoning)

. Eval. for pH

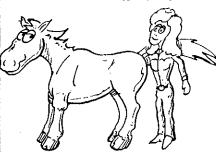
.. pH > 5 - alkaline, small intestinal contents (obstruction & back up) pH < 5 acid, stomach contents

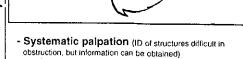
Evaluate for color

- .. Dark brown or coffee grounds Suggests gastric or duodenal hemorrhage
- .. Brown & foul often in prox. enteritis (duodenitis/jejunitis)
- .. Foul odor assoc. w/ presence & of blood, bile, or prolonged stasis



- Used w/ PE & lab. to make presumptive Dx
- -Only caudal abdominal accessible, 60% beyond
- . Pelvic flexure impaction & nephrosplenic entrapment are among few that can be diagnosed rectally
- Easy to tear rectum (see box)
- Feces (hard & dry & mucous, or diarrhea)
- . Look for sand (feces & water in rectal sleeve, sand settles out in finders)
- . Mucous & no feces suggests prolonged duration





. Left kidney, then across to spleen & nephrosplenic ligament

Head of

Pelvic flexure

Inquinal ring

Sm. colon

. Distended bowel, large or small intestine? 1 or many loops?

- Displaced? Impacted?
- .. Distended loops always abnormal
- .. Small colon (fecal balls & bands & sacculations)
- .. Cecum (medial band) & ventral large colon (bands)
- .. Try to find pelvic flexure (no bands felt, but has 1)

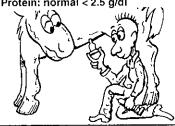
. Gas or ingesta distention

- .. Gas & fluid: springs back following pressure
- .. Ingesta: doughy & indents to pressure . Impacted - colon distended, heavy & hard to move
- . Dehydration colon contracted & sacculations
- . Palpate intestinal wall thickness, thickened indicates tissue edema (m/b strangulation needing Sx)
- . Pain during palpation m/ help locate
- . Inguinal rings
- . Urinary bladder

9. Peritoneal tap - EDTA & Sterile tube

- Color: normally clear, straw-colored serous & doesn't coagulate
 - . Turbid: ## of WBCs & protein
- , Pink or red: free hemoglobin or hemorrhage
- . Darker brown or green w/ fetid odor: bowel rupture suggested (do culture to confirm)

Protein: normal < 2.5 g/dl



- . > 2.5 g/dl: 1st sign of abnormality, inflammation or vascular occlusion & leakage of fluid & protein
- Fibrinogen >10 mg/dl: suggests acute inflammation or blood contamination
- Cells (WBCs): normal between 5-10,000
 - >10,000: progressive vascular damage & diapedesis into abdomen
 - ↑WBCs & protein w/o ↑RBCs; abdominal abscesses or peritonitis

- Differential WBC count

- . Acute infection.: # neutrophils
- . Chronic: +total WBCs + hi # of mononuclear
- Bacteria in WBCs or free: indicates ischemia & release of bacteria & endotoxins (poor Px), need to differentiate peritoneal fluid from intestinal fluid (needle Into intestine)
- . Peritoneal fluid: phagocytized tracteria w/in
- . Intestine: numerous mixed bacteria, plant ma terial & few WBCs
- Plant material w/ WBCs: ruoture = euthanasia

10. PCV & TPP (packed cell volume & total plasma protein)

- Hydration status (PCV = dehydra-
- . PCV ≤ 45% 85% survival
- > 60% 25% survival
- . Serial measurements useful for response to fluid therapy
- WBCs in blood helps detect nonsurgical

- Electrolyte changes

- . Happens quickly, usually not severe changes, metabolic alkalosis or factic acidosis
- . Fluid therapy usually corrects

PROCEDURES:

- PERITONEAL TAP: - Ventral midline, caudal to xiphoid & to right
- of midline (to avoid spleen), lowest point of abdomen - Teat cannula through nick in skin or 18-g 1.5" needle
- . Peritoneum tents so push cannula through
- Anticoagulant tube (EDTA) for cytologic exam
- Sterile tube (w/o anticoagulants) for visual inspection & culture
- Wait patiently, drops will eventually appear

PASSAGE OF NASOGASTRIC TUBE:

- Indicated for most if not all colics
- If pulse > 60 beats/min pass immediately (can relieve pain & prevent stomach rupture)
- Check for stomach fluid (gastric reflux) by siphoning
- Large diameter tube, if trouble getting it in, use xylazine
- Not easy: must work to siphon off GI contents, add water. negative pressure & move around
- Grain in stomach will continue to plug tube, keep pumping in water & keep trying

PALPATION PER RECTUM:

- Easy to tear rectum, so take necessary precautions
 - . Restraint (nose twitch, etc.)
- . Tranquilize if necessary (Xylazine 0.3-0.5 mg/kg IV, can add butorphanol 0.01-0.02 mg/kg IV)
- Tenesmus topical anesthetic into rectum (20 ml of lidocaine) or epidural
- Lots of lubrication on arm (many dehydrated), be gentle, don't fight peristaltic waves
- Palpate backwards: sigmoid loop (long) mesocolon in front of pelvic inlet), so go in as lar as possible initially & palpate backwards



DIGESTIVE SYSTEM

Bankrine Grailles

Colic

Mk 168; IM 680; 12M 749; EM&S 515; C4T 179, 182; 179, 1 C3T 201

COLIC SURGERY OR MEDICINE?

- Approach depending on value of horse
- If Sx not an option, treat medically or euthanize
- Ask about insurance, must have permission to do Sx or to euthanize
- Cardinal CS requiring surgery (all m/ not be present)
- Severe abdominal pain poorly or nonresponsive to analgesics
- Discolored peritoneal fluid containing incr. prot., RBCs & WBCs
- Rectal exam: obstruction &/or displacement of viscera
- · Other factors indicating surgery
- Progressive deterioration of cardiovascular status, w/ abdominal pain & uncertain Dx
- Significant gastric reflux
- Recurrent abdominal pain w/ uncertain etiology
- · Ali cardinal signs m/not be present so make decision on a number of factors
 - History & signalment

 - . Duration & severity of abd. pain &/or sweating ... Usually intest. tympany, spastic colic or gastric dilation improve in 2-
 - If uncertain do serial peritoneal fluid taps as a diagnostic aid
- Do NOT do unnecessary laparotomies
- . Earlier the decision for surgery the better for recovery
- Perform before bowel necrosis & marked changes in peritoneal fluid
- . Large intestine greater need for quick surgery than small because:
- .. Peritoneal changes later than in small
- .. Surgical resection harder
- Often need to go to Sx before final Dx can be made
- Many diagnoses not confirmed until exploratory laparotomy







Medical Tx of Colic: 1) analgesia, 2) decompression 3) fluid therapy

- 1. Analgesia as soon as clinical exam is finished
- Xylazine (Rompun®) DOC most potent
- . Monitor, take away feed (muzzle), nasogastric tube
- 20-30 min before wears off, interferes a little w/ GI motility
 Dosage typical for sedation 300 mg/1000 lb horse IV
- Banamine® (NSAIDs) onset slower, duration much longer, TID or BID, as w/ phenyibutazone get in vein, longer acting & m/ cover up some CS, so diagnosis first Anti-endotoxic effect also

2. Decompression

- Nasogastric tube stand to the side to do this!
- Decompress cecum w/ trochar, not very safe only if going to surgery or if transporting 2-3 hours to hospital
- 3. Fluids: oral or IV, usually both
- Oral fluids cheaper, via nasogastric tube
- . 1-2 gallons every hour
- . Not if refluxing
- Gallon of mineral oil need pump on nasogastric tube
- . If get it on the skin, wash off as it will cause scalding
- DSS (dioctyl sodium sulfosuccinate) osmotically active surface acting agent) pulls fluid into impaction, 5% solution, 8 ounces in 4-6 L of water
 - . Need a hydrated patient
- IV fluids use 14 gauge or larger catheter at least 4 inches, can secure w/ super glue Fastest way to soften impaction is IV fluids if \$\$\$ is no problem
- . Lactated Ringer's (Fluid of Choice), but saline is OK if that is only one available
- .. Give at least distilled water, tap water will lyse RBCs massive hemoglobinuria (red)
- .. Needs to be put in sterilely
- .. Cover w/ Betadyne® ointment .. Bandage
- Change every couple of days, must care for jugular veins or will get thromboembotism
- .. Altach extension tubing so you don't have to handle the catheter Use large 5 liter bags of fluids
- Avoid peripheral vasodilators: vasoconstriction in shock maintains atrial pressure to vital organs. Vasodilatation m/ cause catastrophic fall in blood pressure
- . NO phenothiazine tranquilizers

Prognosis EM&S 517; C3T 206

- · Based on degree of pain, rectal exam findings, presence of gastric reflux & peritoneal fluid tap
- 50% recover from colic surgery
- Resection & anastomosis, 75% die Must tell owners what chance this animal has to survive
- · Duration: continuous uncontrollable pain negative
- · Guarded to poor prognosis if:
- Systolic blood pressure < 100 mm Hg or weak to impalpable pulse
- HR > 80 bpm
- . HR of 100, survival about 30%
- . HR 120, purple mucous membranes, colons distended Euthanize (waste of money to do Sx)
- Cyanotic membranes bad sign
- PCV > 60% bad
- Profound leukopenia
- Blood lactate conc. > 8 mmol/L
- Elev. BUN > 42 mg/dl
- Blood pH < 7.28 concern
 - < 7.00 death eminent (normal 7.32 -7.44)</p>
- ♣ Survival rates associated w/:
- Fibrin degradation products > 20 µg/ml

Bad Px

• PCV > 60%

pain

Uncontrollable

- Prolonged PTT > 1.25
- antithrombin III < 85% control
- + platelets < 100,000 นไ
- Bad rectal findings
- Distended small intestine (strangulation most common, so resection) • HR > 80 bpm
- Large bowel; severe gas distension is negative sign (probably twisted & obstruction)
- Grittiness or crepitation bad sign (ruptured)

Colic Surgery EM&S 518; CT 201; S 355, 210; LAS 437

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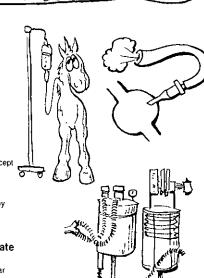
- Surgery for colic, the earlier the better
- Decompress intestine
- Correct problem

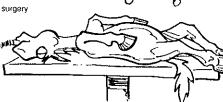
Stabilize before surgery

- · Anesthesia major concern because m/b in shock
- · Do not waste excessive time, many instances surgery before completely stabilized
- Pre-op fluids hydration & acid/base balance & electrolyte imbalances corrected
- Antibiotics Ampicillin DOC (pen gets anaerobes, except
 - Bacteroides fragilis, not gram neg.)
 Metronidazole (Flagyl®) for anaerobes, gentamicin nephrotoxic
- Sx will be clean-contaminated or contaminated if spillage
- Banamine® (flunixin meglumine) for endotoxins released by worms & opening blockage

Anesthesia - ability to ventilate a horse a must in colic Sx

- Xylazine/ketamine combined w/ glycerol glycolate (centrally acting neuromuscular blocker) pre op. Then:
- Isoflurane (excreted through lungs 75% & least cardiovascular Halothane more tikely to potentiate arrhythmias due to epinephrine
- release & direct affect on arterial blood press. & depth of anesthesia Clipping done prior to induction
- Intubate in sternal recumbency (before going to surgery - Often has full stomach, may get reflux)
- Surgery can be fairly long: 3-4 hours
- Complication of myositis paralysis of limb, looking like radial n. paralysis - positioning & weight
- Fast surgery to minimize anesthetic signs
- Keep light (keep blood pressure up) Support w/fluids, well padded or floating on air bags
- Dorsal recumbency



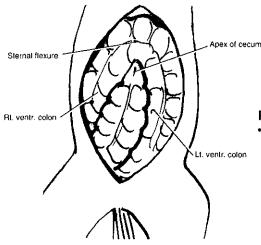


Exploratory laparotomy S 356

Ventral midline incision: less hemorrhagic, umbilicus to xiphoid cartilage

Open abdomen & note what you see

- . Apex of cecum on ventral midline
- · Ventral colon surrounds the apex
- . Abdominal cavity:
- Look for ingesta (bad Px)
- Distended loops of intestine ballooning out incision,
- . Decompress w/ needle & tubing to continue exploration



Ventral Incision - Structures seen

Small colon Transverse colon Lt. dors. colon Ceccum Rectum Ceccum Rt. ventr. colon Rt. dors. colon

Preliminary exploration:

- Gently explore abdomen by gently sweeping viscera w/ hand
- Cause of colic m/b immediately apparent
- Look for:
- Turgid loops of bowl
- 2. Hard masses
- 3. Tight mesenteric bands
- Roughened peritoneal surfaces & fibrin adhesions
- Root of mesentery & connection of cecum & colon for volvulus

Deeper exploration: cecum is reference point

- If cause not apparent, systematically explore abdomen
- · Apex of cecum: on ventral midline
- Ventral colon surrounds the apex (right ventral, sternal flexure, left ventral)
- Note 4 bands & sacculations of ventral colon & cecum
- Locate cecocolic lig. between lateral band of cecum & right ventral colon

Find small intestine

- Ileocecal ligament: pull apex of cecum up & locate the ligament connected to its dorsal band
- Ileum follow ileocecal fold from cecum to ileum, or reach down left side of base of cecum to blindly find
- Jejunum: trace small intestine forward (pull out & replace, go along entire length, point thumb in direction your going to keep track)
 Ascending duodenum becomes jejunum at duodenojejunal flexure
- Ascending duodenum located by its connection to small colon, duodenocolic ligament on left side of cecum (can't be exteriorized)
- Duodenocolic ligament: connects ascending duodenum to the small colon
- Descending duodenum: reach between the base of the cecum and the right body wall dorsally (only tubular structure located here) (can't be exteriorized)
- Pylorus: found by following descending duodenum cranially (can't be exteriorized)
- Liver: located against right body wall & touching base of cecum
- Right kidney: nestled in the renal impression of the caudate lobe of the liver dorsally
- Epiploic foramen (opening between peritoneal cavity & omental bursa)
- Pass your hand between the descending duodenum & right body wall to the liver & right kidney
- If no bowel in this area, no need to check epiploic foramen
- To enter epiploic foramen: pass finger under the caudate lobe of liver forward into epiploic foramen between portal vein (ventrally) & caudal vena cava (dorsally)

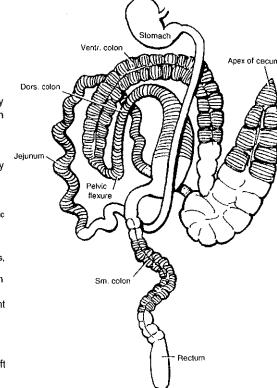
Find large intestine by returning to cecum

- Cecum: palpate up body to base in right paralumbar fossa
- Body of cecum: along right abdominal wall from base to apex (can be partially exteriorized)

- Base of cecum in right paralumbar fossa anchored to dorsal abdomen (can't be exteriorized)
- Ventral colon: around apex & body of cecum (4 bands)
- Cecocolic lig. pull up cecum & see connection between lateral band & right ventral colon
- Pelvic flexure: found on left side near pelvic inlet, junction between ventral & dorsal colon

Pull out pelvic flexure very carefully or may tear, place on drape between horse's thighs or on a special table

- Dorsal colon (ID left dorsal, diaphragmatic flexure & right dorsal parts)
- Left dorsal colon is small w/1 band in mesentery
- Note right dorsal colon's stomach-like expansion (3 bands, but no noticeable sacculations)
- Mesocoton connects ventral & dorsal colon
 Contains blood supply (colic branch to ventral colon & right colic
 artery to dorsal colon)
- Connects to dorsal abdomen at root of mesentery
- Small colon (descending colon): located to left of base of cecum heading into pelvic cavity (2 bands, sacculations & fecal balls) (part can be exteriorized)
- Transverse coton: connects right dorsal colon w/ small colon
 - Transverses abdomen from right to left in front of cranial mesenteric artery
- Spleen: on left side attached to right kidney & against right body wall
- Nephrosplenic ligament: palpated in dorsal left side (find spleen & follow to ligament dorsally)

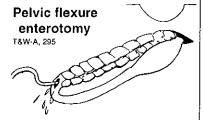


Structures that can be exteriorized through a ventral midline incision (shaded)

Surgical team of several surgeons preferred, as colic surgery is very tiring

Surgeries (T&W-A 268, 383; LAS 443) Decompression surgery

- · Distended bowel (jejunum, cecum or large colon)
- · Punctured w/ needle through purse string suture, tie as remove needle 12-14-gauge needle attached to rubber tubing (through strong bands of large intestine)
- Don't do multiple enterotomies to decompress. end up w/ adhesions
- Do decompression in sections & milk to one opening



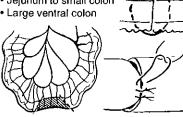
- Opened to empty out large colons (dirty, special table used))
- · Incision toward ventral or dorsal colon, not in pelvic flexure
- · Empty colon: Garden hose up enterotomy. May take up to hour of flushing (sand) to get all out. If enteroliths, must milk down to incision site. Closure of colon by direct apposition

- · Wash & put GI back in correct position
- Other enterotomies also done: jejunum, cecum, large & small colons

End to end anastomosis

T&W-A 306; S 337

- Jejunum to jejunum
- Jejunum to small colon



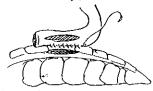
Small intestine - resection & anastomosis T&W-A 298

- M/ remove roughly half of small intestine in horse
- If ≥60% get small bowel syndrome
- About 50-60 feet of small intestine
- Check viability
- Responds to pinch w/ peristalsis
- Pink color indicates viability
- Inject w/ fluorescein & see if fluoresces (not as reliable in horse as dog)
- Dopplers used, oxygen tension, etc. Procedure - resection & anastomosis
- Occlude lumen by making tiny hole in. mesentery & tying a penrose drain tight enough to occlude (3-4" back from proposed cut)
- Angle cut across bowel
- Antimesenteric side of necrotic piece longer

than mesenteric side so all remaining bowel retains blood supply

- Angle important so large opening to help prevent constriction of lumen when healing

 Direct appositional closure (can't invert or even)
- bowel as it shrinks lumen)
- . Take full thickness, but coming out at edge of mucosa (simple continuous, adhesions at knot of suturing)
- Close mesentery (prevent internal hernias)



Side to side anastomosis

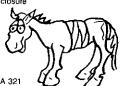
T&W-A 318; S 337

- Used to unite unequal sizes of bowel (adjacent segments of lg. colon w/short mesocolon, jejunocecostomy, cecocolostomy, colocolostomy)
- Parker-Kerr oversew stumps
- 2 layer hand suturing technique usually
- · Jejunocecal anastomosis (jejunocecostomy)
- If ileum must be resected (can't anastomose jejunum to ileum because get tremendous thickening & blockage)
- Cut off ileum, leaving 6-8" blind stump due to inability to exteriorize (connected to base of cecum)
- Closed w/ a Parker-Kerr
- Anastomose end to side jejunum to dorsal band of cecum as far towards base as possible

Clinical signs

Closure of abdomen

- Large suture material, #2 Vicry!
- Near far/far near suture pattern (knot in
- Sometimes do simple interrupted Speed is of essence
- Important to lavage between layers
- Then SQ & skin closure



Postop T&W-A 321

- Incidence of infection high, present wi temp, elev. after a few days, then may have diarrhea Look closely at incision for pus (drain)
- Abdominal bandaging (Elasticon® right on skin, w/ sterile pad on incision, otherwise get tot
- of edema) Often need at least a week of intensive care to get
- them through, ileus, diarrhea, etc. Monitored by auscultation
- Fast 24 hr post op, depending on motility
- Started back on sm. amounts of hay, no grain, or let graze for a bit, monitor, IV

fluids 3-4 days, follow 10 P's

- Intestinal motility stimulators (drugs)
- Metoclopramide better drug, if given too quickly causes CNS side effects, ataxic, excitable Usually added to IV & given slowly
- Neostigmine parasympathomimetic Rule out K & Ca problems 1st, make sure efectro
- These drugs can be dangerous as they make the
- bowel contract
- . If enterolith, will contract over it, causing damage

Treatment

Many spontaneously recover

If don't spontaneously recover

- Rompun® (Xylazine) suppresses

intestinal motility & an analgesic

No atropine as antispasmotic

because m/ cause adynamic ileus

Mineral oil (helpful if irritation

of bowel is the cause)

- Dipyrone (antispasmotic, since spasms

Condition Spastic colic Mk 168; IM 680; EM&S 541; M

#1 Colic? **Spasms**

Intestinal tympany

Flatulent colic

Mk 168: IM 696: EM&S 542: M



- Probably most common cause of
- colic

Facts/Cause

Spasm & hypermotility of intest, tract

- Pain caused by spasms of gut
- Hyperexcitable horses predisposed
- Causes unknown, implicated: Autonomic nervous system
- imbalances
- Irritation of gut by parasites, enteritis & moldy feed
- S. vulgaris
- Rarely life threatening
- Spontaneous recovery common
- · Accumulation of gas in large colon or cecum
- 1° tympany
- Microbial fermentation of lush grass, grains or pelleted feeds
- 2° tympany
- Obstruction of colon or cecum
- Mechanical:
- .. Fecoliths or enteroliths in sm. colon
- .. Large colon displacement
- . Functional
- .. Adynamic ileus
- · Flatulent colic: tympanic horse that passes large amount of gas
- No obstruction synonymous w/ 1° tympany

- No systemic deterioration Frequent passage of feces
- Spontaneous recovery common

Presentation/CS

Loud gassy intestinal sounds

· Acute onset, lasting several minutes

(m/b heard standing next to horse)

• Intermittent abdominal pain

- Patchy sweating on neck

 Diarrhea suggests onset of enteritis

- Moderate to severe abd. pain

- Little systemic deterioration

Severe distention of cecum &

- Dyspnea (pressure on diaphragm)

- Systemic deterioration

. Usually intermittent

• 1° tympany

- Flatulence

• 2° tympany

large colon

- Severe bloat

- Bloat

early

- Pulse rate elevated Rectal exam - normal

borborygmi (frequently)



Diagnosis

· Auscultation: loud rumbling

- Must DDx 1° from 2°
- 1° tympany
- Flatulence
- Auscultation: gas sounds & loud borborygmi
- Rectal: distended colon & cecum
- Little systemic deterioration
- 2° tympany
- No feces passed



- DDx 1° from 2° tympany
- 1° tympany

Oil

in 10-60 min

cause pain)

- Analgesics: dipyrone, Banamine® (flunixin meglumine),
- Rompun® (xylazine), butorphanol Mineral oil via nasogastric tube
- . Not if gastric distention (gas, fluid or ingesta)
- Antifermentative Rx (questionable efficacy)
- Walking to promote flatus
- No need for trocharization if flatulence
- 2° tympany
 - Usually require surgery, see cecal & colonic causes of colic

Postpartum COlic E 1361

See REPRO pg. 240; Common, normal postpartum pain caused by uterine contractions that persist past foaling

1° OK - 2° not

 DDx from severe colic: Inversion of tip of uterine horn, rupture of vessel in broad ligament, trauma to large colon, uterine rupture

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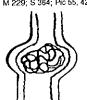


DIGESTIVE SYSTEM

Condition

Simple obstruction small intestine

M8k23 160, 173; Mk 100; I2M 755; IM 685; EM&S 606; C2T 47; M 229; S 364; Pic 55, 42,



 Obstruction w/o strangulation, uncommon, most obstruction are w/ strangulations

Facts/Cause

- Secreting: small intestine usually absorptive, w/ obstruction becomes a secreting organ
- Intestinal distention (from saliva, fluids from pancreas, stomach & ingesta)
- Dehydration
- Mucosal barrier intact initially
- Loss of barrier w/ time (distention causes necrosis of mucous membrane) . Endotoxin absorption
- Partial or complete obstruction
- Proximal or distal location
- . Proximal rapid fluid into stomach, sequesters HCI

Partial obstruction

- Slow onset

DDx from

Colic

- Slow dehydration
- Mild pain (anorexia, lethargy, pawing)

Presentation/CS

- Complete obstruction
- Acute, sometimes violent colic
- Severe hypovolemia (dehydration)
- Proximal vs distal obstruction
- Proximal more intense

Anterior enteritis pg 67

Complete obstruction

- Reflux in stomach

'Summer colds"

Respiratory signs if lung

Thin & potbellied

· Poor hair coat

- Severe pain

migration

- Stomach rupture if not treated

 DDx from anterior enteritis first for Tx

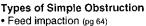
Diagnosis

- Proximal obstruction
- Metabolic alkalosis (sequestration of HCI)
- Protonged m/b metabolic acidosis due to hypovolemic shock Distal obstruction
- Normal electrolytes until hypovolemic shock then metab, acidosis
- Peritoneal fluid normal · Rectal exam - distended

loops

 Medical for partial obstruction & stable condition

- IV Fluids may soften things up
- Mineral oil (laxative) (1 gal./1000 lbs.) , Not if refluxing into stomach
- Banamine®
- Surgery if complete obstruction, deteriorating condition or if peritoneal evidence of intestinal



- · Ascarid impaction (pg 65)
- Muscular hypertrophy of ileum (pg 65)
- · Pedunculated lipoma (pg 67) · Abdominal abscesses (pg 65)
- · Adhesions (pg 66)
- Neoplasia (pg 66)
- Ileus (pg 66)
- History: just dewormed Ascarids in feces (m/b no eggs)
- Gastric distention, can't rectally palpate foals
- Alkaline fluid in stomach (reflux)
- Dehydration (* PCV, TPP)
- Abdominocentesis: monitor for intestinal damage
- Tx to see if problem stops

Potbellied - "Spaghetti"

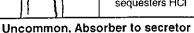
- · Mineral oil if incomplete obstruction
- Fluid

 Enterotomy if complete obstruction & no response to mineral oil



Prevention

- Deworm at 8 wk-old, repeat at 6-8 wk intervals until yearling
- Piperazine or any anthelmintic



CS: Dehydration, Pain

DDx: from anterior enteritis Tx: Partial: Medical; Complete: Surgery

Ascarid

impaction, **Ascariasis** M8k 174, 202; Mk 202; I2M 756; IM 685; EM&S

617; E 1411;

J 606

Parascaris equorum

- Foals & yearlings, rare in adults
- Anthelmintics that paralyze worms (piperazine, organophosphates, pyrantel) in heavily parasitized foals lead to obstruction or rupture
- Obstruction:
- complete or partial
- Migrate through



Sequela: intestinal or gastric rupture

- Feed/Foreign body impaction: 12M 756 Large >> small intestine, distal ileal impaction observed, Cause unknown, Hi fiber diet contributes
- Ileal impaction, Muscular hypertrophy of ileum

M8k 174; IM 686; I2M 756; EM&S 623; E 566; M 229; J 608; S 371; Pic 55, 60

- Thickening of distal end of ileum before it enters cecum (ileocecal valve)
- Get dilatation, if total, backs up to stomach
- . M/ go from partial to complete

Associated w/ mucosal lesions

- S. vulgaris migration
- Intussusception
- Neurogenic stenosis w/ prolonged closure of ileocecal valve
- Idiopathic

 Incomplete to complete obstruction CS



- No peritoneal fluid changes due to just thickening
- No necrosis of tissue
- Stenotic
- Laparotomy to confirm
- Ileomyotomy, † luminal diameter.
- Side to side anastomosis to bypass lesion



Thickened walled CS: Obstruction

Dx & Tx: Exploratory ileomyotomy

 Abdominal abscesses

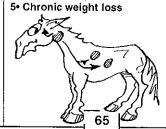
IM 687; I2M 757; EM&S 674; E 576; M 229

- · Strep. equi (bastard strangles) Strep. zooepidemicus, Corynebacterium, Salmonella, E. coli
- · Mesentery most common site · Also liver, spleen, kidney, umbilious or uterus
- 5 yr-old
- Routes of infection:
- Respiratory infections spread via lymphatics, umbilical infections. hematogenous, foreign body penetration & parasite migration



- Bastard strangles, Mesentery CS: Fever from where? Colic Dx: Rectal, exploratory
- Tx: Medical or Sx

- 1. Fever of unknown origin
- 2. Intermittent/prolonged colic small amount of leakage from abscess nonresponsive to Tx, depressed, off feed, mild • in RR/HR, intermittent fever, variable GI signs
- 3. Severe pain nonresponsive to analgesic Tx, go to Sx, abscess pressing on mesentery or causing obstruction
- 4. Toxemia rupture of abscess



Control of the contro

- · Hx, CS
- Rectal palpation
- Peritoneal masses (most cases)
- Laboratory
- Neutrophilia, left shift
- **† fibrinogen** (> 500 mg/dl) - Abd. fluid - exudate - will vary
- . Cultures generally negative Abdominal rads in small horses/loals -
- difficult to see unless gas cap is present Ultrasound
- Exploratory laparotomy (most diagnostic)

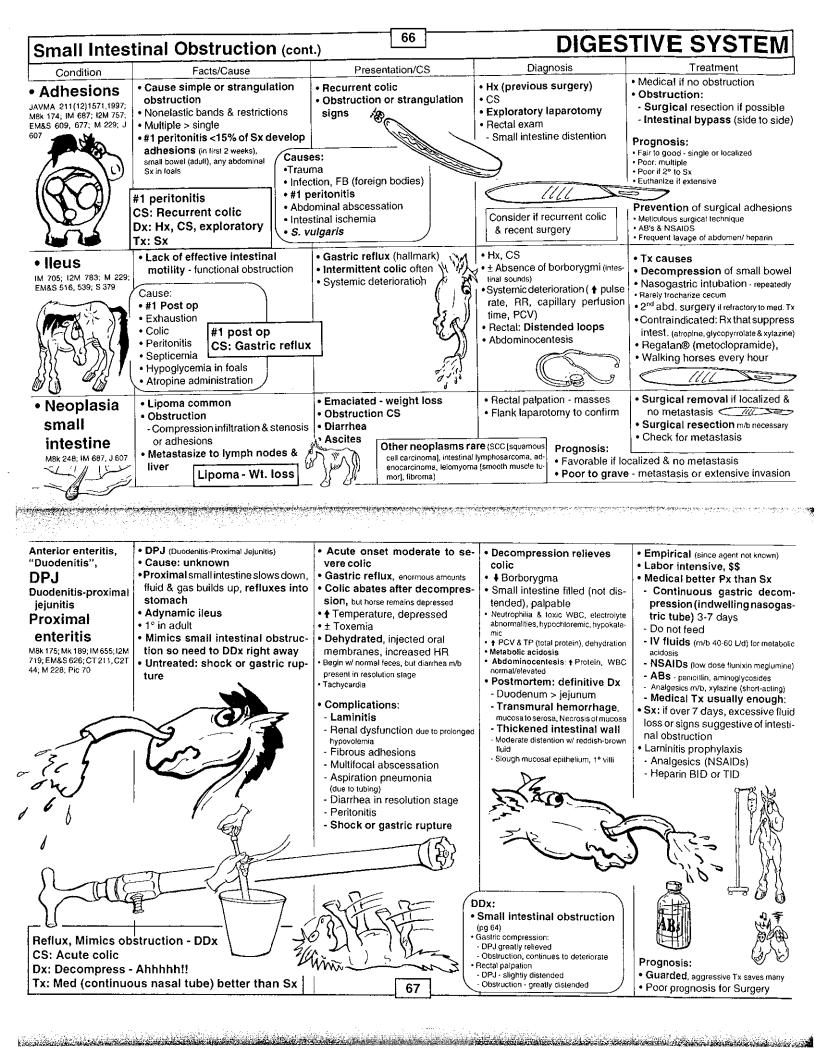
Laparoscop



- Prognosis: Good after surgery
- Conservative - Long term ABs (weeks.- months)
- Analgesic NSAIDs for endotoxemia
- Surgery if significant bowel involvement
- · Remove if possible
- Drain (marsupialization or through skin)
- Bypass surgery if obstructed bowel



Prognosis: · Good prognosis



adults, distention in foals

or metabolic acidosis)

Dehydration

Sand It was best rate of rate of the was read of withing a Mountaged of term Often Books com it

♠ RR (pain, distention, endotoxemia &/

69

Older horses

vascular supply

- Strangulation

• M/ twist around intestine & occlude

Surgery

grave Px, do not delay Sx > 4 hrs

· Silent abdomen: ominous

Rectal: distended small in-

Abdominocentesis-rapid changes - Cloudy & turbid - TP > 3 g/dl

↑ Celi count > 5000 - 100,000

Free bacteria & plant

· Lab - Metabolic acidosis

· Dark red conjunctiva

testinal loops

material

- Decompress intestine

. Resection & anastomosis

Varies dep. on extent of damage

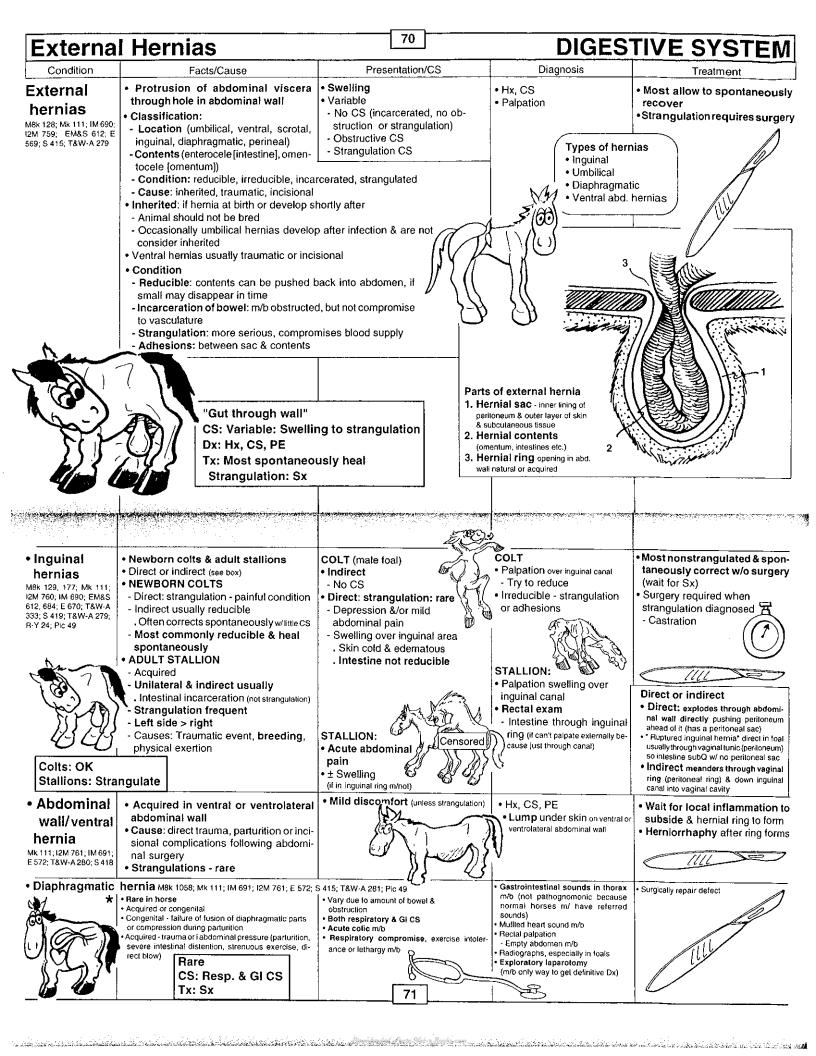
Guarded if resection required

(89% died in 1 study, sounds grave to me!)

of nonviable sections

- Correct problem

Prognosis:



#2 congenital lesion

Dx: Hx, CS, PE

Internal

hernias

Pic 48: T&W-A 274

M8k 176; IM 689; 12M 759; E

569; M 229; S 421, 368, 373;

CS: Most asymptomatic

Tx: Time (most heal spontaneously)

nial sac

Types

Tx: Sx resection & anastomosis

ligament)

hole & gets trapped

Epiploic foramen

- 'Vest-over-pants" closure of abd. wall (causes more tension)
- Preplace overlapping horizontal mattress sutures
- Draw all sutures tight & individually tie sutures
- Free overlapping edge sutured to fascia w/ a continuous pattern
- Close subQ w/ continuous pattern

Similar to other strangulations

Clinical course - acute rapid

Prolonged capillary refill time

Dehydration & hemoconcentration

(fluid into intestine + lack of intake)

• + RR (pain, distention, endotoxemia

Severe unrelenting pain

& severe

· Internal often causes strangulation/intestinal

External often doesn't cause strangulation

• **† HR** > 60 bpm

&/or metabolic acidosis)

Close skin w/ interrupted pattern (nonabsorbable silk, nylon, polypropylene, polyester, Vetafi®)



- Nasogastric tube
 - Enterogastric reflux: relieves large amount (> 5 L)
- Rectal exam:
- Distended small intestinal loops
- Epiploic m/be too far cranial
- Thick intestinal walls (edema)
- Abdominocentesis:
- -Cloudy to serosanguinous & turbid (normally clear yellow).
- **TP** > 3 g/dl

Epiploic entrapment

- + cell count (normal
- < 5,000) > 5000 100,000
- Free bacteria & plant material indicates intestinal rupture & poor Px

Prognosis:

balanced

Surgery:

- · Varies dep. on extent of damage
- Guarded if resection required

 Stabilize before surgery Intense fluid therapy Correct acid/base & electrolyte imbalances

Decompress intestine

Correct problem

viable sections

Don't waste excessive time, many

instances surgery before completely

Resection & anastomoses of non-

 Epiploic entrapment

Strangulation

CS: Colic

 Epiploic foramen (Foramen of Winslow) - Opening betw. peritoneal cavily & omental bursa

Displacement of intestine through a

normal or pathologic foramen w/in ab-

dominal cavity w/out presence of her-

Simple or strangulation obstruction

Mesenteric defects, acquired or con-

genital (gastrosplenic, cecocolic, broad

Congenital fibrous bands or adhesions

· Incarceration - intestine goes through

obstruction

Internal vs External

- Location rt. dorsal abdomen
- Beneath caudate lobe of liver
- Betw. caud. vena cava & portal vein
- Old horse more susceptible
- Hepatic atrophy + size of foramen
- Gastrosplenic · Gastrosplenic ligament: between left
 - greater curvature of stomach & spleen Distal jejunum & ileum most commonly
 - incarcerated
- · Mesodiverticular bands

ligament

entrapment

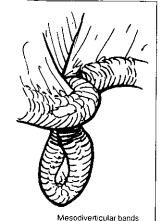
- Connective tissue that forms between mesentery & antimesenteric border of small intestine
- Forms a passage for a possible internal hernia





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Nonstrangulating infarction, Strongylus vulgaris, Blood worms,

Palisade worms. Sclerostomes, Red worms

M8k 202, 166, 171, Mk 202; 12M 762; IM 692; E 578; EM&S 538, 679; C2T 70; \$ 375, 399

- Cause:
- Strongylus vulgaris
- Shock
- S. vulgaris (blood worm)
- Verminous arteritis
- Thrombus in walls of cranial mesenteric or ileocolic arteries (larvae inside wall)
- History: recently moved to high parasite area
- Pathophysiology:

CS: Colic

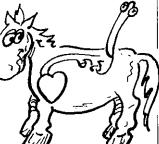
Dx: Difficult

Tx: Ivermectin

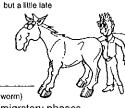
- Emboli or vasoactive substances from thrombus cause obstruction or constriction of vessels
- Ischemia (lack or oxygen) resulting in infarction of intestine

Strongylus vulgaris, Cran. mesenteric a.

- Obstruction variable CS
- Fever
- Depression
- Colic (abdominal pain)
- Acute or chronic



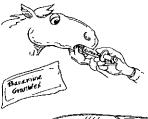
- Dx difficult -presumptive
- Often suspected on history, exam, CS & peritoneal fluid
- Difficult w/o exploratory
- Hx often recently moved to hi parasite area
- · Rectal exam inconclusive
- Enlarged mesenteric a.,
 m/ b chronic inactive lesion
- ecal m/b CS before patent Peritoneal tap
 - M/b hi # of cells (variable)
- Exploratory definitive Dx (don't want to do if don't have to)
- Postmortem: definitive Dx



S. vulgaris (blood worm)

- · Diz during all 3 migratory phases
- Initial migration through intestinal wall
- 4th stage larvae migrate through vascular system to cranial mesenteric a.
- 5th stage larvae migrate back through mucosa into lumen of intest

- Mild CS & suspected
- S. vulgaris
- Ivermectin every 2 mo for life see pg 14
- Banamine® (flunixin meglumine) reduce inflammation & endotoxins Aspirin m/b for anticoagulant (not proven)
- Surgery
- Resection & anastomosis



Prognosis:

- Depending on severity & extent of necrosis
- Poor: if surgery & extensive resection
- Good: mild colic episode

 Effective anthelmintic program & farm management

· CS: GI - Vomiting, colic & constipation or diarrhea, Lameness, Gangrenous ergotism of extremities, Convulsive ergotism Tx: Remove contaminated grain, Supportive Tx, Supplemental feeding, Antibiotics, Pain control

Toxic blue -

green algae

Ergotism, Ergot

- See TOX pg 324, Ruminants more sensitive than monogastrics
- CS: Acute death, colic, bloody, diarrhea, dyspnea, ± CNS: seizures, photosensitization, hepatic diz
- Tx: No specific antidote, Often animal dead or dying, before Tx, Supportive care, Activated charcoal & oi
- Px: Poor, die in 24 hrs



See TOX pg 330; Parasitic fungi, Claviceps of rye, oats, wheat & Kentucky blue grass, Paspalum or Dallis grass)





- Saline cathartics (Epsom salts)
- Often sufficient for 1° impaction of short duration
- Risk of cecal rupture & poor response to medical Tx
- Immediate surgery
- - Cecocolic anastomosis
 - . Easiest w/ stapler due to being unable to exteriorize
 - . Cecum atrophies to some degree

Cecal volvulus

IM 698; I2M 768; T&W-A 290; S



• 1° rare

Post op sequela

. Usually 2° to large colon volvulus

 ≥ 270° obstruction of blood vessels & tissue damage

2° to ig. bowei volvulus CS: Colic

Dx: Ping

Acute colic

rition or 2° to cecal

leading to impaction

motility disruption

Balloted on right side

Cecal perforation (\$ 388): Uniformly

fatal diz; idiopathic in brood mares near partu-

Systemic shock



• "Pinging" in right flank

Rectal exam

ENCE TO

Large, distended, often edematous cecum

- Laparotomy
- Decompression
- Correction of cecal torsion

Tx: Sx Anaplocephala perfolata (most common): ileocecal junction, cecum

· Paranaplocephala mamillana · duodenum & jejunum

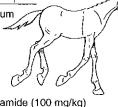
· Anaplocephala magna - sm. intest, cecum

· Young more than old horses

Not proven to cause clinical disease, implicated in

n sa manana na manana na manana m

Tx: Pyrantel pamoate (2 x normal dose) or







M8k 205; Mk 204; IM 689, 692; I2M 1697; EM&S 618;

Tapeworm

infection

Life cycle indirect through orbatid mite

intussusception of small intestine & small intestinal impaction

Niclosamide (100 mg/kg)

CONSERVATIVE - aggressive

- Oral fluids (if no reflux), IV

• Gallon of mineral oil (laxative)

DSS (anionic surfactant) (hi doses &

Saline cathartics (Mg sulfate or

Epsom salts) retain water by osmotic

properties, thus softening impaction

Need adequate hydration

SURGERY: often burst when try surgery

Epsom salt

Be aggressive in medical therapy

by stomach tube every 12-24 hours

prolonged Tx results in diarrhea)

Fluid, lots, 2 large bore catheters

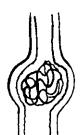
in both jugular veins - soften up

Treatment

Impaction large colon

Condition

M8k 177; IM 698; I2M 768; E 561; C2T 53; M 230, S 390; T&W-A 283; Pic 61



• #1 cause of colic

 Simple obstruction much more common than strangulation obstruction

Facts/Cause

- #1 Feed impaction of large intestine due to decrease in diameter size at pelvic flexure & transverse colon. Cecum & small colon also become impacted
- lleocolic impaction due to hypertrophy of ilium or ileocecal infussusception
- No reflux
- Predisposing factors
- Coarse feed
- Poor dentition
- Dehydration
- (common when water turned off)
- Cold weather
- → Water intake

#1 Colic, Feed impaction Sites: Pelvic flexure, Transverse

colon, lleocolic opening

CS: Colic, ¥ Feces Dx: Hx, CS, Rectal

Tx: Aggressive Conservative Tx

White foal diz, Lethal white

foal,

lfeocolonic aganglionosis (IA) M8k 130; Pic 48

. Completely white foal w/ blue eyes

- "Overo" paint sire & dam
- · Autosomal recessive trait
- · Born dead or weak w/ colic

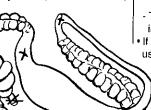
White Overo paint foal, GI defects, Euthanize

Mild to moderate pain

- ♣ Fecal output
- · Feces hard, dry & often mucous covered

Presentation/SC

- Progressive anorexia due to no dietary intake
- Dehydration, if no water intake



· CS, History (Hx)

- · Rectal exam:
- Firm ingesta-filled colon

Diagnosis

- . Pelvic flexure (junction between sacculated ventral & nonsacculated dorsal colons)
- M/not be able to find; usually just forward of pelvic inlet on left side of abdomen
- Transverse colon rectal exam inconclusive
- If can't make Dx by rectal exam use CS & rule out other causes



Impaction sites - common sites

1. Pelvic flexure (most common)

- 2. Transverse colon (junction w/ large right dorsal colon)
- 3. Ileocecal opening (hypertrophy, intussusception)
- Prognosis: · If reach early can shift quickly · If late, pelvic flexure will rot



- Unrelenting pain
- † Peritoneal nucleated cells &/or protein
- Colonic displacement

No Tx: Euthanasia

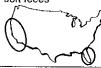
 Check for any colored hair, may change prognosis

Sand impactions

Mk 187; IM 698; I2M 768; E 547, 565; M 232; C2T

- 55; \$ 393; T&W-A 284; Pic 62
- Sandy regions (in environment & hay) Florida, California · Some horses have a "sand tooth"
- · Fine sand accumulates in ventral colon
- Course sand in dorsal colon, transverse colon
- · Fills right dorsal colon (dried out ingestion here so filters out)
- Fluid & gas can flow through, present w/ soft feces

Regional, Obstruction Dx: Feces in glove Tx: Metamucil®



· Similar to ingesta impaction. but also: • Diarrhea

· Appear normal at birth

Acute abdominal

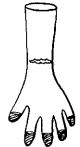
M/ be recumbent

No meconium

pain

passed

Develop colic & die on 2nd day



· Hx. CS

- Rectal exam
- Sand in colon (if in dist, rt. dors, or transverse colon, difficult to Dx)

. History: Overo parents

No meconium on rectal exam

PM: diffuse colonic hypoplasia

· Histopath.: absence of submu-

minal ileum, cecum & colon

cosal & myenteric ganglia in ter-

Signalment, CS

- Feces in glove & fill w/ water
- Sand settle out in fingers (know normal amount of sand in horses of area)
- M/b able to shift medically
- Metamusil® by nasogastric tube (see box)
- Surgical indications
- Unrelenting pain, Incr. peritoneal nucleated cells &/or protein, Colonic displacement

Prevention:

- In sandy areas monitor feeding
- Feed off ground

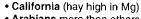


Metamucil® (Psyllium hydrophilia mucilloid) by nasogastric tube

- Lubricates & binds sand to move it out
- Forms get when mixed w/ water so have tube place in before mixing
 After initial dose mix dry w/ feed (prolonged use)

Enteroliths/ **Fecoliths**

Mk2 179; IM 699; I2M 769; E 564, C3T 223; C2T 68; S 392; T&W-A 285; Pic 63



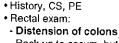
- Arabians more than others 5-10 years (rarely < 4 yr)
- · Enteroliths: magnesium ammo-
- nium (mineral concretions); starts from a nidus (broken tooth, stone, string, hair, FB, silicon dioxide [flint-like stone]); Calif. hay
- Fecoliths: concretion of fecal material
- Can get as big as a soccer ball Single (smooth) or multiple (squared from rubbing (tetrahedral)
- Obstruction at junction of right dorsal & transverse colon (diameter change). Right dorsal colon > transverse colon, small colon
- -intermittent due to swelling & falling back eventually blocks

- · Pain variable depending on extent of distention
- Feces, scant liquid feces, if partial obstruction



Mineral concretions Obstruction transverse colon CS: **♦** Feces

Tx: Sx Remove



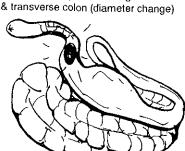
- Back up to cecum, but all in normal position
- Peritoneal tap normal until pressure necrosis of intestine



Conservative

- Apple cider vinegar
- Remove surgically
- If stuck in small colon remove from there Otherwise move back to small colon to take out. watch for squared sides (multiple)

Obstruction at junction of right dorsal



Foreign body (FB) obstruction

- Usually in small colon or transverse colon
- · Young horses eating bedding, rope, fence material, rubber
- Fecoliths



IM 699: I2M 769: E 563:

T&W-A 284; S 401

Similar to enterolith

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Prognosis: poor

surgically correct

& motility returns)

If catch w/n 2 hours can

If later than 4 hours poor

 Px depends on amount of devitalization (watch to see if colonic vasculature, color

Summer

Strangulation

Sx survival low

CS: Obstruction, Shock

Colonic intussusception (IM 773) • Rare

Twists 360°, lots of strangulation,

return to thorax

endotoxemia, no venous

DIGESTIVE SYSTEM

Rectal tears

Condition

M8k 151; Mk 134, EM&S 664;C3T 232;







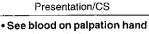


#1 reason for law suits (eq)

- latrogenic: during rectal exam

Facts/Cause

- Males > females, Arabians
- Take precautions for rectals
- Good restraint
- . Stocks ideally Hold against wall, if right handed - wall on left,
- stand at side Nose twitches
- Xylazine if necessary
- Lots of lubrication
- Take out feces & repeat lube
- Don't fight peristaltic waves
- Peritoneal pouches 8" from anus
- Most tears at 12", so into peritoneal cavity (G4) (see box) or into dorsal mesorectum separated from peritoneal cavity by thin connecting peritoneum (G3)
- Dorsal tears most common (splits over knuckle w/ peristaltic wave)



- If undetected when tears
- Sweat (horse & vet!)
- Uncomfortable
- Start straining
- Grade 4: rapidly progressive
- Progressive elevated HR
- Progressive colic

Stop straining:

each mixed together)

lasting than lidocaine)

- Endotoxemia/shock Grade 3: similar to grade 4, but slower progression

· Propantheline bromide

Acepromazine, xylazine or detomide

. Lidocaine lubricant enema (25-50 ml of

• Epidural: Rompun® (xylazine) (longer

Diagnosis Feel rectum tearing when hand in rectum

- Physically examine tear
- Heavily sedate animal
- Epidural
- Access depth of tear using bare fingers
- Peritoneal tap
- Massive response, huge peritonitis
- Grade 4 feces

- Grade 1: heal by 2° intention
- ABs heal on their own. Swelling of 2°, may become Grade 3 tear
- Grade 4: cannot be treated, Px is terrible
- Grade 3: Emergency
- Tell owner immediately!!!
- Prepare to ship to surgical facility
- . Stop straining (see box)
- . Carefully evacuate rectum
- . Check tear (see Dx)
- . Pack rectum w/ Betadyne® (povidone iodine) soaked cotton
- . Start on broad spectrum ABs Procaine penicillin G + gentamicin or m/b metronidazole (Flagyl®)
- Banamine® to counteract toxemia
- Ship to surgical facility (refer)
- Surgical facility Various Tx
 - Temporary colostomy allows torn part to heal, stopping feces from entering tear
- Best to prevent them!



#1 Lawsuit, G3 (into mesorectum) CS: Blood, Sweating

Dx: PE

Tx: Emerg., Refer for Sx

GRADES

- Grade 1 (G1): only through mucosa & submucosa
- · Grade 2 (G2): muscle layers only, mucosa intact
- · Grade 3 (G3): through mucosa, submucosa & muscle layers into space between layers of mesorectum, serosa only separation from peritoneal cavity (most common tear), Fecal balls jammed up between layers of mesorectum
- Grade 4 (G4): full thickness into peritoneal cavity
- 1. Temporary colostomy allows torn part to heat, stopping feces from entering Sx through flank, not easy, lot narrower (18 ribs) than cow, smaller paralumbar fossa & thick
- · Exteriorize loop of small colon, cran. to lear
- Suture colon to muscle wall
- Open colon & suture cut to edge of skin Fecal balls come out the side of animal
- Disadvantage doesn't completely rest torn colon some feces pass
- 2. Cut small colon
- Bring 1 end out colostomy & suture to wall
- Close off the other distal end of colon
- Allowing distal end to rest from peristalsis
- Problem: distal portion becomes atrophied during healing period
- 3. Piece of fenestrated plastic pushed into small colon proximal to tear, fenestrations used to suture sleeve to colon, hope fecal balls will go

Rectal prolapse

M8k 150; Mk 133; IM 835: I2M 911: EM&S 666; C2T 73; E 576; S 411; LAS 444; Pic

- · Bowel turns out of anus, external intussusception-like
- Straining causes prolapse
- Prolapse = strains more, so w/ time worsens more prolapses
- Mares > males

Causes - Prolapse · Straining, tenesmus

- Constipation
- Diarrhea
- Parasitism
- Chronic cough
- Urethral obstruction
- Advanced pregnancy
- Prolapsed, strains more, so

- Type 1: initially & acutely
- Red doughnut of mucosa most common
- Type 2 & 3: longer, more tissue hanging out (complete protapse)
- Type 2: complete prolapse
- Type 3: complete w/ invagination of colon, much longer than type 2
- Type 4: intussusception of rectum &/or small colon
- Double-layered: 2 entrances, a trench around the prolapse
- Straining, tenesmus
- W/ time worsens
- Desiccated, edematous (due to strangulation (venous return), dries &
- More comes out due to gravity

Prolapse

- Differentiate Type 1-3 from Type 4
- Probe between prolapsed mass & inner rectal wall
- . Type 1-3 can't pass probe much past anus . Type 4 passes freely

Clean tissue

- · Decide if viable
- · Return if healthy prolapse, caught early
- Epidural to stop straining
- Massage, warm, wet, squeezing edema out
- Purse string sutures to close down anus (animals w/ soft feces, pigs especially) Doesn't work on horses
- Necrotic, usually exposed mucosa (outer layer)
- Resect & remove
- . Circumferential incision around base & another at end
- Connecting w/ a horizontal incision
- Peel off outer layer down to submucosa (do not remove submucosa)
- Remove everything that is necrotic
- End edge must be closed to base end
- Skewer w/ long needles to hold in place
- Take off only the mucosa, NOT submucosa Usually it is not full thickness necrotic



CS: Straining, Desiccates w/ time Tx: Epidural return or Sx

Epidural to stop straining

- Between sacrum & 1st caudal vertebra. 1st movable spot - wag tail
- Must be right on midline, 45° horizontal; go through ligamentum flavum (yellow ligament)
 - Negative pressure, put drops on end of needle (fill hub
- · When in epidural space drop disappears, put on more & it also should disappear
- 7 ml of lidocaine, if too much, ataxic
- Xylazine 100 mg in 7-8 mls saline for 1000 lb Takes 40 min onset, lasts longer & doesn't make





Meconium retention

IM 335: I2M 380: E

• The 1st feces of life, usually passed by 12-48 hrs; Retained if not passed by 12 hours w/ attempts to pass #1 cause of abdominal discomfort in newborn; colts > fillies (smaller pelvis)

• Dx: Digital exam, CS, Dehydration, Prerenal azotemia, Stress WBCs

• Tx: Stool softeners, Laxatives, Analgesics, Soapy water enema (DSS), Fleet enemas Sx intervention in refractory cases 83

Atresia ani, anal atresia 8 413

· Rare; Imperforate anus

. Dx: visually . Tx: Surgical correction possible





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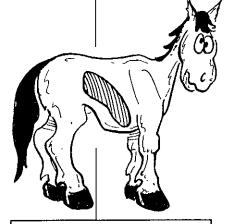
DIGESTIVE SYSTEM

Condition Liver

disease M8k 220, 130; Mk 138; IM 837; I2M 913; EM&S 692; E 634; C4T 214; C3T 353; M 245; Pic 79 Liver m/b diseased long before it fails to function

Facts/Cause

- CS not seen in early stages of liver diz
- Loss of 80% of liver before regeneration & recovery impossible
- Remarkable ability to regenerate



No pathognomonic CS of liver diz CS: Icterus, Weight loss, CNS Dx: GGT, ALP, SDH, BA Sedate

Presentation/CS

- No pathognomonic CS for liver diz
- No CS of liver diz consistently present . Most signs related to failure of liver function (except pain)
- · Icterus (common in acute, not always in chronic)
- Also seen in anorexic or fasting horses
- Hemolysis can also cause + bilirubin
- Failure of uptake, conjugation or excretion of bilirubin
- Wt. loss common, but not specific for chronic diz
- Diarrhea possible in chronic liver diz
- Dermatitis (hepatic photosensitization) due to phyllogrythrin (normally excreted in bile) accumulat-
- Pruritus sometimes (mechanism unknown)
- Hemorrhage terminally (clotting factors made
- Pharyngeal or taryngeal collapse w/ loud stertorous inspiratory noises & dyspnea in some (especially ponies) (mechanism unknown)
- Hepatoencephalopathy
- Behavioral changes: docile animal becomes aggressive, aggressive becomes docile
- Depression, incoordination, aimless walking, head pressing, yawning
- Multiple causes: low blood glucose levels, * ammonia, altered plasma/ amino acid ratio

Liver biopsy site

• Right 14th ICS (intercostal space) intersection w/ line from tuber coxae to point of shoulder

MAN

Diagnosis

- History (Hx), CS
- - Slightly ♦ blood glucose
 - Ammonia (4x) - # BUN (urease needed)
 - Terminally ♦ serum albumin
 - Enzymes:
 - . GGT fairly specific for liver diz
 - . ALP in chronic, also in bone, intesline, placenta & macrophages
 - SDH, LDH & GDH
 - .. In acute diz, normal or
 - in chronic
 - . SDH: active hepatocellular necrosis
- Excretion tests
 - . Bilirubin: ↑ indicates liver diz, bile blockage, hemolysis, or fasting horse
 - Bile acids: > 15 µm/l indicates hepatic diz, cholestasis or portal systemic shunting
- BSP, normal < 3.5 min Liver biopsy: safe & simple, but

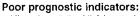
avoid if liver abscesses suspected

Treatment

- fx for liver failure
- 1st sedate (xylazine) 10% glucose IV
- Blood ammonia (# # of bacteria producing ammonia)
- Nasogastric tube
- Mild laxative
- Lactulose® Vit K
- Limit CHO
- Correct any acidosis slowly
- Slow 5-10% dextrose drip
- Dietary management
- Small meals 4-6 x/d
- Vit. B1, folic acid & Vit. K1 weekly

Vit B.

- · Fresh plasma transfusions
- · Steroids: if not infectious
- · Protect from sun when grazing
- Colchicine
- Antibiotics: avoid those metabolized by liver such as tetracycline & chloramphenicol



- Albumin < 2.5 g/dl &/or ↑ globulin level
- Prothrombin time > 30% of normal

- . Greatly . GGT & ALP w/ normal or . SDH or LDI
- BSP half life > 8 min.
- Marked fibrosis

Grave w/ pyrrolizidine alkaloid toxicosis, mitotic inhibition

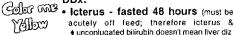
Diagnosis of liver diz:

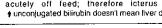
- History
- · Lab:
- Slightly # blood glucose (gluconeogenesis)
- Ammonia (4x) (urease of liver needed to convert)
- ↑ of ammonia doesn't correlate to level of CNS signs
- → BUN (urease needed)
- Terminally \$ serum albumin (liver makes albumin, long half life)
- Enzymes:
 - . + GGT in chronic diz fairly specific (gamma glutamyl transferase)
 - .. Found in biliary tract (cholestatic instead of hepatocellular)
 - .. Also in pancreas, lungs & kidney (renal diz, not † because excreted in kidney)
 - . ALP in chronic (alkaline phosphatase)
 - . Also in bone, intestine, placenta & macrophages
 - , SDH, LDH & GDH (sorbitol dehydrogenase, lactate dehydrogenase & glutamate dehydrogenase)

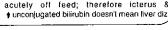
Meller

- .. In acute diz; normal or I in chronic
- .. SDH, liver specific, good indicator of active hepatocellular necrosis
- Excretion tests (checks liver's excretory function)
- Bilirubin: from heme, mainly from RBCs, unconjugated bilirubin is converted by liver to conjugated which is secreted by bile system into intestine where it is converted into urobilinoger
- . Elevation indicates liver diz, bile blockage, hemolysis, or fasting horse
- . Liver diz mostly unconjugated (indirect reacting)

- .. Direct to lotal ratio usually < 0.3
 . Bile blockage or intrahepatic cholestasis
 ... † Conjugated (direct reacting) & unconjugated
 - Direct to total ratio > 0.3
- Bile acids synthesized by liver from cholesterol & excreted in bile
- . Bite acids > 15 um/l indicates hepatic diz, cholestasis or portal systemic shunting
- BSP (sulfabromophthalein) dye, clearance (halftime) used in ig. animals more than retention test. Inject IV & blood samples taken 5 times in 12 min . Normal < 3.5 min
- Liver biopsy safe & simple
- Do not do if liver abscesses suspected
- Diffuse or zonal lesions seen in most toxic, infections
- & metabolic fiver diz usually Dx by biopsy
- Easily miss focal lesions abscesses, granulomas, neoplasia & liver flukes

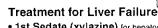












- 1st Sedate (xylazine) for hepatoencephalopathy Diazepam, chloralhydrate or barbiturates
- 10% glucose IV (if low blood glucose)
- # Blood ammonia (# # of bact, producing ammonia)
- Nasogastric tube
- . Mild laxative (mineral oil) + neomycin or factulose
- . Neomycin (may induce diarrhea, so not commonly used)
- . Lactulose® (acidity in GI to incr. amount of ammonia converted to ammonium, therefore not absorbable)
- Limit CHO, low protein diet to minimize ammonia
- Correct any acidosis slowly (exacerbates hepatoencephalopathy)
- Slow 5-10% dextrose drip (decr. hepatic work load)
- · Dietary management
- Small meals 4-6 x/d, beet pulp, cracked corn, molasses
- Force feed mixed paste if not eating (by rehydrating pellets & nasogastric tube)
- Dextrose to water (ready source of energy) - IV feeding expensive. Amino acid supplementation
- Vit. B1 folic acid & Vit. K1 weekly
- Fresh plasm transfusions for clotting abnormalities & m/b Vit. K
- Corticosteroids: if suspect acute hepatitis not due to infectious agent, & in chronic w/ unknown cause (prednisolone doesn't require hepatic transformation)
- Grazing, protect from sun
- Colchicine used empirically in chronic liver cirrhosis to ♣ fibrosis, but may cause laminitis
- Antibiotics: w/ liver biopsy & positive results of bacteria Culture & sensitivity should be done
- Avoids those metabolized by the liver, such as erythromycin, tetracycline, chloramphenicol































85

DIGESTIVE SYSTEM

Condition Neonatal toxic

hepatic failure, Iron toxicity. Hepatic failure in

foals M8k 232; Mk 138; IM 848; I2M 926.



CS: Hepatoencephalopathy

Tx: Supportive • Px: Poor

Dx: Hx, CS, Lab, PM

Facts/Cause

- Iron fumarate toxicity - When given to foals before nursing
- If given after colostrum consumed, toxicity because of protection of glutathione in colostrum
- Fed w/ lactobacillus products containing ferrous fumarate
- Products taken off market

 CS in 2-5 days (rarely when older) Hepatoencephalopathy

Presentation/CS

- Seizures
- Marked depression
- Ataxia, aimless wandering
- Head pressing
- Icterus, unless die peracutely
- Comatose in hrs & death, regardless of Tx



before colostrum, 2-5 d Lab:

History of iron administered

- Hi serum bilirubin

- (conjugated & unconjugated)
- Hi ammonia
- Prolonged prothrombin time Serum liver enzymes not always diagnostic
- SDH (sorbitol dehydrogenase) normal or slightly GGT (gamma glutarnyl transferase) m/b hi in normal foals

Diagnosis/Differential Dy

Serum glucose frequently low Tremendous 1 in BUN, PCV m/b normal

PM (postmortem)

- Small liver
- Red-brown areas of necrosis (massive hepatocellular necrosis)
- Alzheimer Type II astrocytes in brain
- Fibrosis of areas not necrosed

Treatment

- Supportive Tx
- IV dextrose temporary improvement



Prognosis:

- Poor: most don't recover w/ Tx
- If develop liver diz w/o liver failure usually
- Rarely liver failure w/ severe fibrosis several months after birth

DDx:

Stix

- Perinatal herpes infec.
- Tyzzer's diz (> 8 ds) (pg 87)
- Bacteremia (Actinobacillus equuli)
- Congenital anomalies
- Atresia of bite ducts
- Portocaval shunts
- Hepatotoxicities (pg 86, 89)

Mycotoxins -**Hepatotoxin**

M8k 2076; IM 853; EM&S



Aflatoxin & Rubratoxin

- Toxic metabolites of molds
- · Grow on feed (certain grains, corn, peanuts & cottonseed)
- Diz may be as short as 5 d, if less severely affected will survive w/ supplemental therapy

Toxic metabolites of molds in feeds

Product off market, Fe before colostrum

CS: Liver failure Dx: Analysis of feed Tx: Unsuccessful

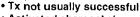


- Icterus Weiaht loss
- Diarrhea
- Dermatitis
- Hepatoencephalopath
- Acute liver insufficiency



Analysis of feeds for mycotoxin conc.

- M/ have eaten it weeks-months before
- Liver biopsy:
- Microscopic biliary hyperplasia
- Fatty changes
- Central lobular fat infiltration & hepatic necrosis



- Activated charcoal slurry orally & oxytetracycline IM for 5 d m/ reduce losses if given under 24 hr after ingestion
- Variable responses to Tx. depending on amount of destruction



Nearly 100% fatal

IV antibiotics (tetracyclines, p

50% dextrose IV

Fluid therapy



Tyzzer's Diz, Baçillus piliformis infection,

Dead foal diz

Mk 152; IM 848, 378; I2M 925; EM&S 698; E 640; C4T 218; C3T 442; C2T 110, 242;



CS: Jaundice, Dead

Dx: Difficult, Stain liver

Tx/Px: nearly 100% fatal

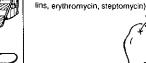
"Dead foal diz", Bacillus piliformis

· Bacillus piliformis (motile, filamentous, gram (-), spore forming, obligate intracellular bacteria)

- Fatal diz of foals 7-40 days (also fatal in lab animals)
- Focal bacterial hepatitis
- · Mares m/b carriers
- Sporadic, generally only one foal affected
- . Most found dead or in
- Depression
- Anorexia
- Recumbency
- ± ↑ Temp. (102 106° F)
- . Marked jaundice usually • Convulsions (hypoglycemia)
- Coma terminally



- . Definitive Dx difficult, CS nonspecific
- Jaundice if live long enough
- · Lab nonspecific:
- Marked fof liver enzymes SDH, LDH, ALP, AST, GGT
- Marked ★ bilirubin (mostly indirect)
- Profound hypoglycemia
- (< 20 mg/dl terminally)
- PM (postmortem) - Marked jaundice
- Huge liver (hepatomegaly)
- Pale, diffuse foci of necrosis (1-2 mm)
- Colltis, lymphoid necrosis & focal myocardial necrosis
 Histo.: multifocal areas of necrosis, bile duct proliferation
- Definitive Dx:
- Giemsa or silver stained liver sections
- . Long, slender bacillus, alone, in stacks &/or bundles in hepatocytes



םם:



Grave: nearly 100% death

Prevention:

- Sporadic, so not indicated
- If confirmed case, monitor all foals for first 40 d of life
- Subsequent foals of a mare that have had a Tyzzer's foal monitored carefully



neoplasia

IM 867; I2M 945; Pic 88

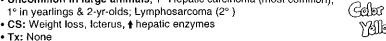
Hepatic

Infection or toxic damage to foal's liver; EHV (equine herpes virus) - hepatocyte necrosis

CS: Usually aborted or weak w/ CS or other system

• Dx: Liver lesions (Hepatocyte necrosis, Intranuclear inclusion bodies in hepatocytes)

• Uncommon in large animals; 1° Hepatic carcinoma (most common);



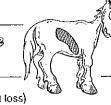




Liver abscesses

IM 858: I2M 935

- More common in cattle; common incidental fining in horses on necropsy
- CS: Similar to other abdominal abscesses (intermittent colic, intermittent fever, weight loss)
- Dx: CS; Can't be palpated per rectum
- Tx: Rarely drained to outside or removed; Long term ABs (penicillin or ampicillin m/b in combined w/ rifampin or metronidazole)





Downloaded from Vet-e Books.com

Poisonous plants in hay

Dx: Hx, CS, PM (fibrosis)

CS: Liver failure, CNS, icterus

Tx: Euthanasia, Tx HF; Px: poor to grave

Other plantsFungal hepatotoxins

Tansy ragwort (S. jacobea)

- Flowers" composite, showy, vellow

-Leaves: deeply irregular

Description

gara digangan biyan da andar oo ka dibi kada dhabihada da dada 🕮 🕾 barabada daran 200 kala barah ka barah da ana da ana

Prognosis:

· Poor to grave: due to

tremendous amount of fibrosis

enzyme activity to help w/ prognosis

Mildly affected cases: use serial liver biopsy &

DIGESTIVE SYSTEM

Chronic active hepatitis, Idiopathic¢

IM 849; I2M 927; EM&S 701; C4T 221; C2T 112; M247; Pic



Substantial inflammatory process w/in liver

- Cause unknown (idiopathic)
- Bacterial infection from portal vein or up bile duct
- Sporadic occurrence

(class wg Yallow

Depression

- Progressive weight loss
- Intermittent fever
- Icterus
- Intermittent CNS signs
 - Disorientation, not full blown hepatoencephalopathy



Diagnosis/Differential Dx

- History, CS
- Nonspecific signs
- Liver biopsy
- Bacterial culture & sensitivity
- Fibrosis
- Laboratory
- Elevated liver enzymes
- BSP prolonged
- Postmortem: 6
- Inflammatory cell infiltrate (mononuclear in some cases, PMNs in others)

arab

 $\Box\Box$

 $\Box\Box$

- Loss of hepatocytes
- Fibrosis
- Focal granulomas
- Nodular hyperplasia (trying to regenerate)
- Biliary hyperplasia if cholangiohepatitis



- Pyrrolizidine alkaloid toxicity (pg 89)
- Rile stones
- Abdominal abscesses
- Other chronic wasting diz (pg 48-49)

Treatment

- Supportive & similar to other chronic hepatic problems
- Treat for liver failure
- 10% glucose IV (if low blood glucose)
- **♦ Blood ammonia** -nasogastric tube mineral oil + neomycin (but kills GI flora) or lactulose (acid GI ammonia to ammonium)
- J- Slow 5-10% dextrose drip
 - Dietary management
 - Small meals 4-6 x/d (beet pulp, cracked corn, molasses) V | | | | | | | | | . Force feed mixed paste if not eati
 - IV feeding \$. Vit. B₁, folic acid & Vit. K₁ weekly
 - Steroids in lymphocytic \ plasmocytic hepatic infiltrate
 - Increases appetite, stabilizes cell mem branes, reduces inflammation & connective tissue formation
 - Antibiotics if bacterial
 - cholangiohepatitis (culture & sensitivity) Trimethoprim sulfa (gram neg.)

Prognosis:

 Poor, depends on amount of fibrosis





Cholangitis

Mk 138; IM 868; I2M 948 C3T 263

· Rare inflammation of bile system associated w/ chronic active liver diz Horses have no gall bladders

Idiopathic

Dx: Biospy

Rare inflam, of bile system

CS: Liver failure Dx: # GGT & AP

CS: Intermittent CNS signs

Tx: Tx for liver failure

Tx: ABs

- Weight loss
- · Variable icterus
- Variable
- changes in appetite



- SDH, GGT & AST + if active hepatocellular damage, but variable
 - If both GGT & AP elevated suspect c h o
- langitis or cholelithiasis Liver bioosy
- Endoscopic culture of bile duct



rognosis: Guarded to poor if fibrosis marked

Trimethoprim-sulfamethoxazole

Bile stones, Cholelithiasis

Mk 138; IM 867; 12M 947; M 247; EM&S 701; C4T 216; C3T 259; Pic 85

Bile stones CS: Colic, CNS Dx: Ultrasound Tx: ABs

Lipidosis

217, 543; C2T 114;

M 2 'B; Pic 83

Hyperlipemia,

Mk 138; IM 862; I2M 940; C4T

· Calculi (bile stones) in bile duct Horse has no gall bladder
 Choledocholithiasis: presence of calculi in

- Cause (debatable): infection, inflammation of biliary tree, bile stasis, alteration of bile composition, ascarid eggs
- Cholangitis
- · Hepatocellular diz: if multiple small or single large stones obstructs bile outflow

Prolonged negative energy bal-

Liver diz between acute & chronic

1° ponies, adult horses in association

Fat mobilized from adipose tissue, free fatty

. Fat accumulates in kidneys, heart, etc

w/ azotemia (inhibits uptake of FFA)

· Similar to fatty liver syndrome in cattle

Fat accumulation in liver &

triolycerides in plasma

ance - Starvation

Pathophysiológy:

acids to liver





Lethargy, duliness

Hyperlipidemia

- Diarrhea, fever

Comatose

· Severely depressed

- Duliness & lethargy

Nervous excitability

± Death w/in 10 ds of CS

Chronic, intermittent colic. mild in nature

- CNS signs: sudden depression
- Anorexia



Dx is difficult - CS (icterus, colic, CNS) Ultrasound - dilaled bile ducts

- Chronic, hepatomegaly
- Echogenic stone in bile duct Exploratory, palpate bile ducts
- Laboratory:

Hx. CS

(lipids)

 Dramatic ★ in cholestatic enzymes. especially GGT

White or yellow opacity to plasma

↑ triglycerides in blood

+ Serum hepatic enzymes

- Prolonged BSP clearance time

Postmortem: fatty infiltration of

liver & kidney (pale, swollen & greasy)

(> 500 mg/dl)

- # Bilirubin

- Metabolic acidosis

- Azotemia in horses

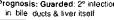
† Hepatocellular enzymes BSP normal unless complete obstruction



Many untreated because asymptomatic &/or not diagnosed - ABs (because associated w/

- infection) Trimethoprimsulfonamide, penicillin w/ gentamicir
- ± Fluids & plasma
- Surgical attempted in limited # of cases Manually crush stone & push into duodenum
- (difficult to do w/o causing additional inflam.) Anatomically difficult area to reach

Prognosis: Guarded: 2º intection in bile ducts & liver itself

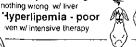


Tx 1° diz: pregnancy (induce), lactating (take foal off), parasites Correct negative energy balance

- Hyperlipidemia, feed alfalfa gruels
- Hyperlipemia (severe)
- Fluids for dehydration, LR for acidosis Slow IV drip of glucose († release of insulin, if too fast † acidosis)
- Insulin + glucose or galactose
- on alternate days Heparin, alters lipoprotein lipase activity to † uptake into tissues (hemorrhage
- m/b, check clotting profiles) Tx until plasma clear (< 400 mg/dl)



Hyperlipidemia: good



Hyperlipidemia: less severe than hyperlipemia, < 500 mg/dl triglycerides

Ponies, Neg. energy, Fatty liver

CS: HF, Diarrhea Dx: Lipids in blood Tx: Tx 1° diz

- Off feed
- Lactating
- 2° to other diz

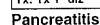
Causes:

- Parasitized

Starvation

- Late pregnancy





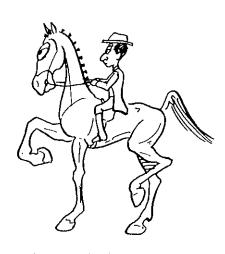
Mk 121; IM 871; I2M 950; E 643; C2T 46; Pic 90

- Rare in horse; Etiology unknown, pancreatic duct obstruction, duodenal inflammation, parasitic migration)strongylus or parascaris CS of inflammation of exocrine pancreas: colic, shock, tachycardia, gastric dilation w/ reflux; Chronic: hyperkeratotic dermatitis - rare
- Dx: Serum amvlase > 700 lU/L
- Tx: Symptomatically: gastric decompression (nasogastric tube) & shock therapy (fluid & electrolytes, m/b Ca)

91



Pancreatic Diz Rare in horses



Abscessation in foals 113 Rhodococcus equi 113 S. zooepidemicus 114 bacterial pneumonia 115 Anaphylaxis 117 ANTU poisoning 97 Arytenoid chondritis 104 Aspiration pneumonia 116 Bacterial pneumonia, adult 115 foal 112 Bastard strangles 96 Bleeders 101 Broken wind 120 Chondritis, arytenoid cartilages 104 Cleft palate 22 COPD (Chronic obstructive pulmonary diz) 120 Cough DDx 341 Cyanosis DDx 241 Diaphragmatic hernia 71 Dictyocaulus 116 Dorsal displacement of soft palate 106 EHV-1 111 EIPH (Exercise induced pulmonary hemorrhage) 101 Epiglottic entrapment 107 **Epistaxis** 95 DDx 340 Equine adenovirus 110 Equine arteritis 110 Equine rhinovirus 110 Ethmoid hematoma 94

110

101

EVA (equine viral arteritis)

hemorrhages

Exercise induced pulmonary

RESPIRATORY SYSTEM

Follicular hyperplasia	95	Pneumothorax	121
Fungal rhinitis	95	Prematurity	121
Granulomatous pneumonia	113	Pulmonary edema	117
Guttural pouch disease	98	Respiratory distress syndrome	
Guttural pouch emphysema	99	Recurrent laryngeal neuropati	hy 104
Guttural pouch mycosis	100	Retropharyngeal abscess	97
Guttural pouch tympany	98	Rhinopneumonitis	111, 215
H ₂ S	97	Rhinovirus	110
Heaves	120	Rhodococcus equi	113
Herpes virus-1	111, 215, 254	Roaring	104
Immaturity (foals)	121	Rhinitis	94
Influenza 1	109	Sinusitis	103
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Laryngeal hemiplegia	104	Smoke	97, 314
Laryngopalatal subluxation	106	Snots	111, 215
Laryngitis	104	Strangles	96
Lung worm	116	Strep. equi	96
Lymphoid hyperplasia	95	Strep, zooepidemicus	114
Nasal discharge	94	Stridor	241
DDx	340	Subepiglottic cyst	107
Neonatal respiratory stress syn	drome 121	Toxicology	97
Paranasal sinuses	102	Tracheal stenosis	107
PCP	97	Tracheitis	107
Pharyngitis	95	Tuberculosis	115
Pleuritis	118	Viral respiratory diseases	108
Pleural effusion DDx	344	Zn3P2	97
Pleuropneumonia	118		* •
Pneumocystis	114		
Pneumonia	112-116		

Condition Facts/Cause Presentation/CS CS of nasal problems: Nasal discharge, respiratory noise & dyspnea usually Nasal cavity

1° viral or bacterial diz: other CS that go with bacteria or viral diz

Foreign bodies: CS: often malodorous discharge, Dx: endoscope & X-rays, Tx: Forceps - removal

• Fungal infection: rare, Cryptococcus, gen. bilaterally in nares; seen in SW, devastating due to meningitis; Rhinosporidia causes rhinitis • Fungal granulomatous polyps in nasal cavity & turbinates, Dx: CS, endoscope, smears & cultures, histo., Tx: surgical removal, Amphotericin B, ketoconazole

• Nasal septum: usually congenital disorders, relatively rare; cystic degeneration, abscesses, neoplasms, CS: M/ cause obstruction

- Tx: Resection of nasal septum

 Wry nose, squify mouth: congenital, CS: deviated nasal septum, severe malocclusion of incisors, No Tx, Px: depends on degree of distortion

Nasal polyps: Dx: endoscope, X-rays • Tx: surgical removal

Pedunculated alar fold - CS: Respiratory noise, Tx: bilat. resection of alar fold

 Atheroma - Sebaceous cyst in false nostril, young horses, swelling, painless, doesn't obstruct airflow, Tx: Cosmetic surgical removal

Amyloidosis: nodular or plaque-like deposits of glycoprotein in upper respiratory tract in response to continued immunological stimulus, Tx: Stop stimulus, Sx

Nasal discharge, Rhinitis

• CS, not a diz

Diagnosis

· Dx: CS, Endoscope

• Tx: 1° cause

Common causes: See pg 340



Ethmoid hematoma,

problems

239, Pop 22-7/98

M8k 1096; Mk 426; IM 56; I2M 56; E 738; EM&S 392; M

150; C3T 270; S 435; T&W-A

Progressive ethmoidal hematoma, Hemorrhagic nasal polyps

M8k 1098; IM 557; I2M 618; E 741; M 147; C3T 274; S 440; POP 71-7/97

 Slowly expanding angiomatous masses

- Mucosa lining ethmoid conchae

Cause unknown

Uncommon condition

• Generally > 8 yr

. Thoroughbred, Arabian



Uncommon

CS: Intermittent epistaxis

Dx: Endoscope Tx: Sx ablation

Intermittent epistaxis

- Uni- or bilateral

Blood-tinged to mucopurulent

- Rarely latal epistaxis (as in guttural mycosis)

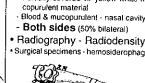
 Abnormal respiratory noise if occludes nasal passages

· Coughing, choking

· Excessive salivation (ptyalism)

• A Respiratory effort





Endoscope

follicles

Radiographs

Ultrasound

Biopsy

. Hx (history), CS (clinical signs)

• Endoscope

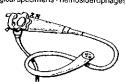
- In ventral meatus & viewed w/ tip up Color: deep red-purple, yellow-brown

- Irregular, rounded surface

Partially covered w/ yellow-white mu-

Radiography - Radiodensity

Surgical specimens - hemosiderophages



pharyngeal lympho-nodular

Microbial cultures hard to eval-

uate (always contaminated)

• Rhinitis (pg 94)

• Laryngitis (pg 104)

· Guttural pouch diz

Neopłasia (biopsy)(pg 98)

DDx:

Surgical ablation

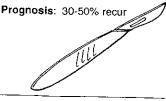
· Sx curettage - bleeding (pack nose w/ gauze)

Cryosurgery extirpation, minimal blood loss, impractical for lg. hematomas

Treatment

Laser (photoablation) minimum bleeding, but requires multiple procedures for large

. Can be done in standing horse



Warfarin/dicoumerol

Moldy sweet clover

Pyrrolizidine alkaloids

Epistaxis



 Presence of blood at external nares from nasal cavity, nasopharynx, guttural pouches or lungs (unilateral or bilateral)

Hemoptysis: blood out of mouth; rare from lungs in horse because long soft palate with epiglottis over it closes off mouth

Blood from lung hemorrhage often not seen because swallowed, if seen associated w/ strenuous activity

 Dx: Hx (uni- or bilateral; exercise, etc.), Physical exam, CBC, Feces for occult blood, Endoscopic exam, Transtracheal wash, Radiographs of lungs, Biopsy

- Cough

Common causes: See pg 341 Toxicity

Ethmoid hematoma (pg 95)

Nasal polyps (pg 94)

Fungal granulomas

· Trauma from nasogastric or

Guttural pouch mycosis

· EIPH (rarely see epistaxis)(pg 101)

CS, not a disease

Pharyngitis/ chronic pharyngitis,

Pharyngeal lymphoid hyperplasia,

Chronic lymphoid follicular hyperplasia, Follicular pharyngitis, Follikelkatarrh

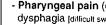
M8k 1095; Mk 719; IM 546; I2M 607; E 747; EM&S 748; M 153; S 446

• Inflam. of pharyngeal tissues

- · Not a specific diz, but a response to other dizs
- Sequel to bacterial & viral respiratory diz
 Local physical, chem. &/or allergic causes
- Acute & chronic forms

Chronic follicular hyperplasia

- Lymphoid hyperplasia (most common cause of chronic pharyngitis)
- Associated w/ chronic cough Associated w/ guttural pouch emphysema
- Diz of young (more prominent lymphoid follicles than older horses)
- So important to know age
- Normal follicles m/b mistaken for pathology (young)
- Usually spontaneously disappears at 3 yr



ACUTE pharyngitis

- Pharyngeal pain (odynophagia, dysphagia [difficult swallowing])
- Nasal discharge (bilateral)
- Regional lymphadenopathy (submandibular, retropharyngeal Inn.)
- Respiratory noise (often inspiratory)
- Pharyngeal swelling

CHRONIC pharyngitis

- Similar to acute cough, pain
- Swelling, narrowed airways

- · Associated w/ other infections
- Streptococcus spp. (Strep. equi)
- Herpesvirus (EHV-1 & EHV-2)
- Parainfluenza
- Influenza
- Rhinopneumonitis
- Irritation by nasogastric tube
- Trauma (endoscope or nasogastric tube)
- Abscesses caused by Strep, equi (Strangles)

 FB (foreign body)(horses less commonly than ruminants)
- Retropharyngeal Inn. rupture into pharynx

- Tumors of nasal cavity
- endoscope
- Thrombocytopenia
- Purpura hemorrhagica

Mild pharyngitis - none - Chronic - hyperplasia of Symptomatic:

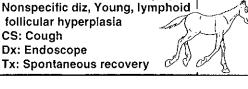
- NSAIDs to ♦ pharyngeal pain
- Soft feed (green grass)
- Fluids if dehydrated
- ABs for 2° infections
- Topical preparation (ABs, anti-inflam. drug, glycerine (spray through intranasal catheter TID, commonly used, efficacy un-

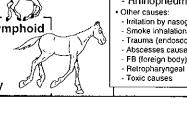
Chronic lymphoid hyperplasia:

- Tends to resolve w/ age
- Empirical & palliative
- Rest 4-8 wk (often returns when start retraining)
- If kept in training sulfa compounds & throat preps. Penicillin & broad spec. A8s
- Cautery if doesn't improve (obliterates reactive tissue) . Trichloroacetic acid, electrocautery, or
- freezing w/ liquid nitrogen Syrupy expectorants on tongue for cough
- Routine immunization

Prevention:

- Frequent immunization (pg 12)
- Every 60 d, influenza & rhinopneumonitis





96

RESPIRATORY SYSTEM

Condition

Strep. equi

infection,

lymph node

abscessation

Retropharyngeal

M8k 1090; Mk 744; IM 1639, 580;

I2M 1639, 580; E 734; EM&S

674, 434; Pop 11-5/98, 32-7/98

Facts/Cause S. equi: gram-positive cocci w/lg Strangles, Distemper,

Named because untreated horses sound like strangling (swelling of lymph nodes closes off phar-

 See retropharyngeal lymph node abscessation pg. 97

1-5 yr-olds predisposed (all horses susceptible)

Worldwide, contagious

Direct contact

- Morbidity 30-100%

- Mortality 9-10% . 2º pnéumonia

± Spread to guttural pouch, sinuses & to rest of body

"Bastard strangles" (see box)

Presentation/CS

 Sudden onset of fever 2-6 d after exposure

Upper respiratory tract catarrh

 Swelling & abscess of Inn. of upper neck (lymphadenopathy)

- Submandibular & retropharyngeal lymph nn.

Depression, anorexia

Sequelae & complications

Often results in death

· Guttural pouch empyema

Purpura hemorrhagica

 Suppurative necrotic bronchopneumonia Aspiration of pus or metastatic spread

Low incidence

- Difficult to Tx

Myocarditis

 May stand w/ neck stretched · Reluctance to swallow

 Nasal discharge (serous, then mucopurulent)

 Rupture of abscess onto skin eventually

 Preceded by epilation & oozing of serum

• "Bastard strangles": metastasis of S. equi to other organs, com-

monly to lung, mesentery, liver, spleen, kidney & brain, see GI pg 65

Diagnosis

 History & clinical signs Abscesses + High fever

Firm at 1st, then fluctuate as lique faction & suppuration develops

Culture Strep. equi S. zopepidemicus gulckly invades causing confusing bact. Dx



Treatment

Early CS (pyrexia & depression)

- Hi doses of Procaine pen G, DOC

Stops further development

· Lymph node abscessation

- Encourage abscess to rupture (hot packs)

. Isolate

. Aspirate swollen lymph nodes

. Lance ripe abscess ventrally

- ABs contraindicated (prolong diz by halting ripening of abscesses)

Advanced strangles

Prolonged lever, anorexia, depression, lethargy or dyspnea in some horses

Systemic penicillin

- Tracheostomy (rare)

Just exposed to strangles

- Penicillin m/ prevent diz

Tx Complications

Metastatic abscessation to thoracid

or peritoneal cavity - "Bastard strangles"

- Purpura hemorrhagica - systemic pen & corticosteroids



Control:

 Commercial vaccines not completely Successful & cause injection abscesses

· Isolate new horses

structures

 If nasal discharge: culture for S, equi & keep isolated until eliminated

Relieve respiratory distress

- Temporary tracheotomy if needed

- Surgical drainage often necessary

- Ventral approach recommended over

dors, or Viborg's triangle because less vital

- Broad spec. ABs immediately

Submandibular & retropharyngeal Inn.

Dx: Hx, CS

"Bastard strangles"

lymph node

abscesses

IM 538; I2M 59

CS: Swelling, nasal discharge

Tx: Abscesses - rupture - heal

Retropharyngeal

Medial & lateral retropharyngeallymph nodes

Latera; chain along fateral side of guttural pouch Medial chain over dorsolateral pharynx &

under caudovental guttural pouch

- #1 Strangles/Strep. equi (from contact) or trauma

Strep, zogepidemicus, Corvnebacterium pseudotuberculosis & Actinobacillus spp. - Extension of gultural pouch infections In area of vagus (CN 10), glossopharyn-

geal (9), hypoglossal (12), spinal accessory (11) & sympathetic trunk

#1 Strangles - Press on pharynx

CS: Difficult swallowing & breathing

Excessive salivation

- Dysphagia (difficult swallowing) Odynophagia (painful swallowing) Both due to Cranial nerves 9 & 10

Dyspnea

Painful "throat-latch" swelling

Purulent nasal discharge

Nasal or oral regurgitation

Extension of head & neck

• Weight loss

Cranial nerve involvement



Hx, CS

· Endoscope: reduced size or collapse of lumen of pharynx

Lateral radiographs

- Soft tissue mass between pharynx & guttural pouch (pharyngeal wall appears enlarged) m/ contain gas

M/b compression of larynx & trachea

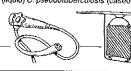
 Percutaneous needle aspiration of purulent material

Ultrasound

Lab - inflammatory response

- Leukocytosis, neutropenia

Postmortem: Abscess - S. equi (liquid) C. pseudotuberculosis (caseous)



(procaine pen.); first take C&S (culture & sensitivity)

NSAIDs to reduce



Cellulitis (pg 143) · Guttural pouch empyema or tympany (pg 98)

Parotiditis

Lymphadenopathy

 Neoplasia Hematoma

Toxicology



• See TOX pg 314; Pressure wood treatment, Horses like to lick "leaky" wood, † O2 demand, Irritating to skin & resp. tract • CS: Gasping • Dx: Rapid rigor mortis • Tx: No specific therapy, Fluids to flush kidney

Dx: Endoscope, rads

Tx: Sx drainage - ABs

 See TOX pg 314; Toxic gas, "Rotten egg" smell, Agitation of liquid manure holding pits, Irritant to eyes & respiratory system • CS: Pulmonary edema, Hyperpnea, Apnea, Asphyxia • Tx: Fresh air, Artificial respiration

Smog, SO₂

• See TOX pg 314; Smog, Sulfur oxides + H2SO4, Eye irritation & salivation, emphysema, respiratory distress • Tx: No specific Tx

Smoke

• See TOX pg 314; Barn fires: alveolar damage, interstitial edema, hypoxia & 2° bronchopneumonia

• CS: Oral burns, Respiratory problems (cough, stridor, tachypnea) • Tx: Patent airway, O2 therapy, Bronchodilators

• See TOX 315; Rare: Use declining, ↑ permeability of pulmonary capillaries, Strong emetic

• CS: Pulmonary edema "drowns in own fluid", Death w/o convulsions • Tx: No specific Tx, emetics early

• See TOX pg 315; Kilrat®, Goph-rid®, Release of phosphine gas on contact w/ water (more rapid at low pH), Odor of rotted fish, Emetic • CS: Exposure, rapid death, dyspnea, Garlic smell to stomach • Tx: No specific Tx, Gastric lavage, Symptomatic







Guttural Pouch Diseases

RESPIRATORY SYSTEM

Guttural pouch

M8k 1098; Mk 739; IM 549; 12M 610; EM&S 402; E 743; C3T 275, S 480; Pop 26-7/98

Condition

Guttural

pouch

tympany

M8k 1099; Mk 740; IM 549;

12M 610; EM&S 404; E 744; M 150; S 481; LAS

344; Pic 94; Pop 26-7/98

· Guttural pouches are unique to the horse

Air-filled diverticulum of auditory tube betw. pharynx & middle ear

- Paired pouches betw. atlas & pharynx

- Divided by stylohyoid bone into large medial & small lateral compartments

- Median septum: mucous membranes of 2 pouches on midline

- Structures crossing dorsocaudal aspect of medial pouch

. Cranial nerves 7, 9, 10, 11 & 12

. Cranial sympathetic trunk

Facts/Cause

· Unilateral >> bilateral disten-

Cause unknown (idiopathic)

M/b congenital redundancy of mucosal

fold (plica salpingopharyngea) causing

pharyngeal opening of auditory tube to

- Fillies more than colts

tion of guttural pouch

. Internal carotid artery

- Lateral pouch crossed by external carotid artery

- Pharyngeal opening of auditory tube, thus of guttural pouch

. Located in lateral walls of nasopharynx below pharyngeal recess

. Mucosal fold (plica salpingopharyngea) helps make up opening

Not sterile, normally contains Strep. zooepidemicus



Infrequent · Retropharyngeal swelling,

- Nonpainful, soft, fluctuant Foals from birth to 1 1/2 vr

Mild cases just swelling

eral if large enough

• Severe swelling (press on pharynx)

Presentation/CS

- Variable respiratory distress

- Extension of head & neck

- Dysphagia (difficult swallowing) - Unilateral, can appear bilat-

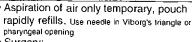
Diagnosis CS: Swelling, retropharyngeat space

Unilateral or bilateral Deflation by catheterization through

pharyngeal opening or percutaneous needle aspiration & ext. compression

Radiographs

• Endoscope - pharyngeal opening



Treatment

 $HHll_{th}$

- Fenestration of median septum (through pharyngeal opening). Ineffective if bilateral

Sx excision of redundant tissue of pharyngeal opening of auditory tube (plica salpingopharyngea)





Good if surgery successful



Foals - Uncommon CS: Nonpainful swelling

Dx: CS, Scope, rads Tx: Sx - release air

DDx:

Guttural pouch empyema (pg 99)

· Retropharyngeal abscesses or cellulitis (pg 97)

Strangles (pg 96)



Guttural pouch empyema

M8k 1098; Mk 739; IM 550; I2M 611; EM&S 406; E 744; C3T 279; M 148; S 482; LAS 342; Pop 26-7/

- Purulent material in guttural pouch
- Infection through pharyngeal opening of auditory tube or lymphatic spread
- 2° to chronic, localized from generalized ascending respiratory infection
- Usually unilateral
- Strangles (Strep. equi) common extension
- Rupture of retropharyngeal abscess

Intermittent nasal discharge

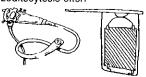
- Worsens when head lowered - Unilateral or bilateral because
- pharyngeal opening of auditory tube behind nasal septum
- Non-odorous, white or opaque
- Lymphadenitis

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- Parotid swelling & pain
- Difficult swallowing (dysphagia) due to pharyngeal compression
- Difficult breathing (dyspnea)
- Chondroids (hard concretions of inspissated pus)

CS: Chronic nonresponsive nasal discharge

- Radiographs standing lateral
- Fluid line of radiodense masses (chondroids)
- Oblique rads to tell which pouch
- Endoscope through pharyngeal opening
- Aspiration of fluid C&S (culture & sensitivity), microbial analysis (B hemolytic streptococcus)
- Leukocytosis often



- Need to lower head for drainage Medical or surgical depending on duration & nature of diz

Tx complicated by poor drainage because

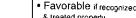
pharyngeal opening above floor of pouch (rostrodors.)

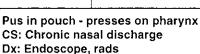
- Medical:
- Parenteral ABs, relapse common
- Daily lavage w/ saline AB solution through Al pipette or Chamber's catheter
- 500 ml, lower head to prevent aspiration
- Irrigation w/ indwelling catheter m/ cause severe inflam, changes Daily catheters beller
- Course of Tx protracted
- Surgical drainage if medical Tx ineffective
- Chondroids: Sx always indicated



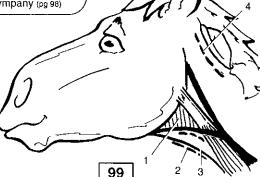
· Favorable if recognized early & treated property

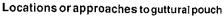




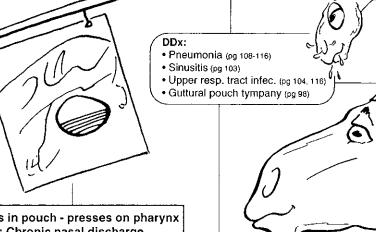


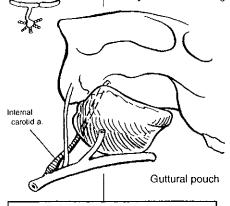
Tx: ABs/ lavage, Chondroids - Sx





- •1. Viborg's triangle: bound by tendon of sternocephalicus m., linguofacial vein & ramus or mandible
- Whitehouse: ventral midline incision, split sternothyrohyoideus muscles
- Modified Whitehouse: skin incision ventr. to linguofacial vein, blunt dissect along larynx to guttural pouch
- Hyovertebrotomy: incise ventral to wing of



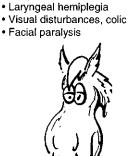


Fungal, Roof of med. compartment CN 9-12 & Int. carotid a.

CS: Epistaxis - Dysphagia

Dx: Endoscope

Tx: Sx - tie off int. carotid



Horner's syndrome (sympathetic



Topical therapy, endoscope

Fungicidal & lungistatic Fix, topical enzymes & organic iodine compounds Variable success

Treatment

Parenteral ABs, corlicosteroids, thiabendazole, ketoconazole & iodine compounds of questionable efficacy

- Tx protracted so danger of fatal epistaxis during therapy

Surgical - best prognosis

-Balloon-tipped catheter into distal internal carotid a. combined w/ proximal ligation of internal carotid a.

Closes off artery over guttural pouch

Stops blood entering from common carotid or Circle of Willis

[[[

DDx:

• Epistaxis (pg 95)

- EIPH (Exer.-induced pulm. hemorrhage) (pg 101)

- Ethmoid hematoma (pg 94)

Guttural pouch or pharyngeal neoplasia - Tracheobronchial foreign bodies

Dysphagia

- Fxs of hyoid apparatus

Pharyngeal/guttural pouch fistula
Cleft palate (pg 22)

Esophagilis

Pharyngeal paralysis

- Foreign body entrapment in mouth or esophagus - Pharyngeal neoplasia

Lead poisoning (pg 269)

Bacteria, viral, & mycotic CNS infections



"Bleeders". **Exercise** induced pulmonary hemorrhage, EIPH.

"Bobbling", "Choking",

"Gurgling M8k 1093; Mk 738; (M 603; I2M 603; EM&S 451; C4T 441; C3T 335; M 162; Pop 20-9/97



· Bleeding from lungs because of

"Bleeders": misnomer because bleeding from lungs through nose, not from nose

 Big economic concern for owners of performance horses ("don't finish race")

- Race tract terms: "bobbling", "choking" & "gurgling"

- Flat racers (Thoroughbreds, Quarterhorses & Appaloosas) freq. betw. 43-75% EIPH

- Highest percentage in Arabians - Epistaxis low % of EIPH

- Possibly all Thoroughbreds experience EIPH

- ↑ W/ ↑ in distance & intensity of races (sprinters highest incidence)

- 🛊 W/ age (ongoing lung damage, accumula tive effects)

Pathogenesis not understood:

Follows exercise at speed, mechanical effort of lungs

 Previous pulmonary damage, dust & respiratory diz

- Pulmonary damage over time

- Bleed from dorsal caud, lung lobes

- Once start bleeding, continue w/ subsequent working

• Epistaxis after strenuous exercise (race)

- Only in 10% of EIPH horses

- Dyspnea, i RR & effort

Poor (race) performance

Slowing or stopping toward end of race Difficult, labored or abnormal breathing

Excessive swallowing after a race (swallowing blood)

Coughing after exercise (m/b clearing blood)

Exercise intolerance

 "Cool out" slowly from exercise (RR, prolonged peripheral vasodilation & sweating)



· Hx, CS

• Endoscope

- Blood in lower airways just after exercise (30-60 min. after exercise; by 6 hours, blood is cleared)

TTW (transtracheal wash)

- Hemosiderophages

(macrophages containing hemosiderin) Sano's trichrome or Perl's stains

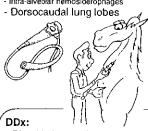
- Harsh bronchial tones over trachea due to blood & in some causes over bronchi in caud, area

Laboratory: usually normal

hemogram & chemistries
• Rads: dorsocaudal lung fields

Postmortem:

- Intra-alveolar hemosiderophages



· Blood in lower airways

- Bleeding lung abscess

- Neoplastic mass

- Foreign body Nasal trauma

- Nasal turbinate necrosis - Ethmoid hematoma (pg 94)

- Guttural pouch mycosis (pg 100)

Airway foreign bodies

Neoplasia

 Tx difficult because of chronic nature plus little understanding of causes

 Environment free of dust & infectious diz (impossible at racetrack)

Enforced rest (3-6 mo) helps some,

most continue when return to training

Lasix® (furosemide) allowed at

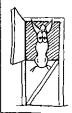
some state racetracks

Diuretic, mechanism unknown

- Efficacy not proven
- Reduces bleeding, doesn't prevent EIPH

- Must prove bleeder 1st (endoscopy, TTW evaluation by board certified clinical pathologist)

Diuretic helps clear system of illegal drugs





· Exercise intolerance

- Many conditions (see pg. 348)

Excessive swallowing

· Inappropriate breathing

Diffuse pulmonary hemorrhage

Subpleural or parenchymal hematoma

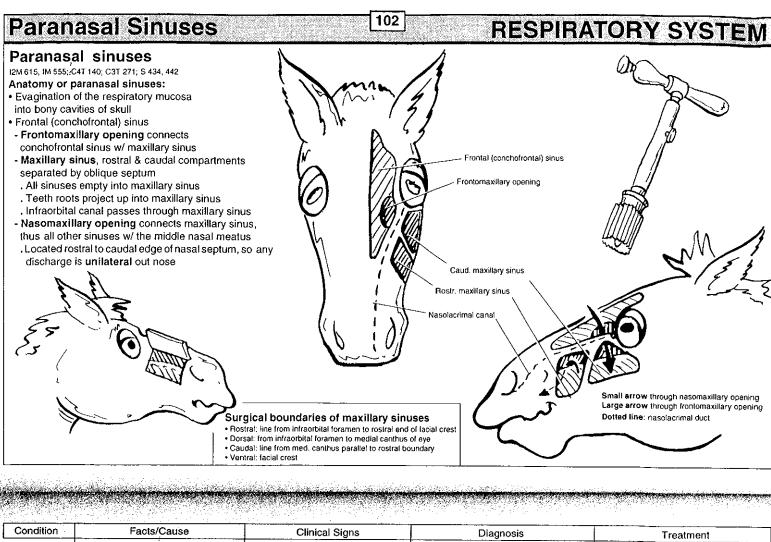
- Focal or diffuse pulmonary diz

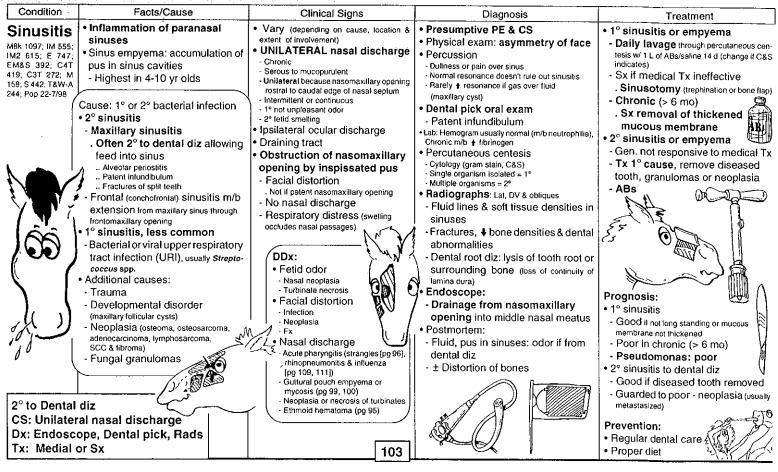
- Neoplasia

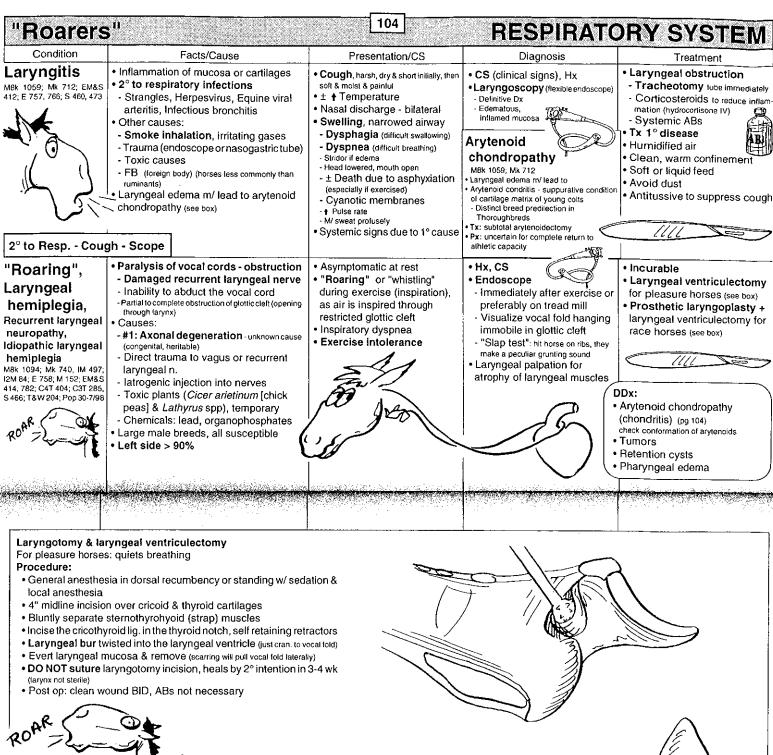
Lungs bleeds w/ exercise CS: Bleeding w/ exercise - \$

Dx: Endoscope, TTW Tx: Rest, Lasix











Pathophysiology:

- Permanent paresis or paralysis of vocal cord & arytenoid cartilage
 Degeneration of the recurrent laryngeal nerve
- Lt. more commonly affected (longer, wraps around aortic
- Paralysis of intrinsic laryngeal mm., except lat. cricoarytenoid - Dorsal cricoarytenoid m. can't abduct the vocal cord to open glottic cleft
- "Roaring" caused by vocal cord & arytenoid cartilage hanging in glottic cleft & decreasing inspiratory air flow, no resistance

Laryngeal prosthesis and laryngeal ventriculectomy For race horses to help performance

Procedure: general anesthesia, right lateral recumbency

- 5" incision over larynx parallel & ventral to the external jugular & maxillary veins
- Dissect to the caudal edge of the dorsal part of the cricoid cartilage
- Place a mattress suture through the caudal edge of the cricoid cartilage near dorsal midline
- · Bluntly dissect between cricopharyngeal & thyropharyngeal muscles to locate the muscular process of arytenoid cartilage
- Drill a hole through the vocal process w/ a 14 or 16 gauge needle
- · Reach under the cricopharyngeal m. & pull suture through the hole in muscular process
- Tie the suture tight (mimics cricoarylenoideus dorsalis m. abducting the vocal corditaterally)
- · Suture the skin incision
- Do a laryngeal ventriculectomy as outlined above



Epiglottic entrapment (pg 107)

(outline of epiglottis not obscured)

Presentation/CS

Exercise intolerance

Dorsal displacement

of soft palate

Dorsal displacement of the soft palate. Laryngopalatal

I2M 367; C4T 415; EM&S 400; M 155; T&W 221; POP

subluxation



 Normally epiglottis overlaps dorsal side of soft palate

- · Pathological displacement:
- No predisposing factors in roughly 1/3 of cases
- Forced contraction of sternothyrohyoid & omohyoid mm. pull larynx caudally
- Numerous dizs change stability of larynx & soft palate/pharyngeal arch
- . Exercise despite painful disorders
- . Caudal retraction of larynx during exercise
- Epiglottic hypoplasia
- . Rostral displacement of palatopharyngeal arch
- Pharyngeal lymphoid hyperplasia
- Laryngeal chondroma & chondritis
- Subepiglottic cysts
- . Epiglottic entrapment
- Laryngeal hemiplegia

so they are forced to breath through their mouth

Normal position of soft palate

- Tx other diz conditions first Limit ability to dislocate larynx if
- no predisposing diz - Tongue tie helps restrict caudal

Treatment

- retraction of larynx
- Excision of portions of free border of soft palate
- . Doesn't prevent displacement, but eliminates part of soft palate that gets sucked into larynx once displaced
- Sternothyromyectomy (see box)



Prognosis:

 Value of Sx is of controversial success rate

Sternothyromyectomy

- · Standing, sedated & local in form of a "U-block" or
- Dorsal recumbency (anesthetized & intubated)
- Procedure:

Diagnosis

Epiglottis not visualized,

covered by soft palate

Endoscope

. Normally the epiglottis overlaps dorsal side soft palate

of caudal edge of soft palate onto pharynx) form a seal around the larynx

Caudal edge of soft palate & palatopharyngeal folds (extensions

Dorsal displacement is normal during deglutition (swallowing)

Can be induced in all horses if external nostrils are closed off.

- 5-6" ventral midline incision rostral to cricoid cartilage
- Bluntly dissect between sternohyoid muscles Undermine sternohyoid & omonyoid
- Cut out a 4" portion of both muscles (transect through sternohyoid & omohyoid until close to external jugular v.
- Repeat on opposite side
- Place a Penrose drain & suture to SQ & skin Postop:
- Remove Penrose drain in 6 d Remove sutures in 12-14 d
- Begin training when sulures removed



Epiglottis normally dorsal to soft palate

CS: Tiring

Dx: Endoscope: no epiglottis

Tx: Tx other diz, Sx

Epiglottic entrapment

EM&S 421; E 764; C3T 281; M 146; S 462; Pop 30-7/98

Dx: Endoscope

Tx: Sx

Subepiglottic tissue

CS: Exercise intolerance

- Displacement of subepiglottic tissue over epiglottis
- Normally subepiglottic tissue is folded accordion tashion to allow free movement of epiglottis
- Most common in Standardbreds & Thoroughbreds



eating DDx:

 Ulceration of epiglottic apex Dors, displacement of soft palate (pg 106) (outline of epiglottis can't

Exercise intolerance

Abnormal resp. noise during

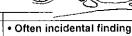
fast work (inspir. or expir.)

Uneven respiratory pattern

Coughing at exercise or after

- Surgical removal or transection of entrapping tissue
- Short surgery if experienced so just IV anesthesia, less experienced use general Dorsal recumbency Ventral laryngotomy approach (midline over larynx, separate strap mm.)
- Approach through cricothyroid ligament (filling caudal thyroid notch)
- Grasp free edge of entrapping tissue & retract epiglottis into laryngeal lumen (if intermittent entrapment, tissue m/ not be over epigloitis, then grasp tissue lateral to epiglottis)
- V-shaped incision or crescentic incision (controversy on how much to remove) Subepiglottic tissue & taryngeal incision allowed to heal by 2° intention
- Laser surgery in future

- Withhold food to prevent contamination of wound Phenylbutazone & prophylactic ABs for several days
- Stall rest up to 45 d
- Endoscope before return to training Complication of surgery
- Removing too much m/ result in dorsal displacement of soft palate (horse worse after surgery than before) Expected if concurrent problem before surgery
- Remove too little, recurrence of entrapment



- Chronic cough
- Dyspnea during exercise Stridor
- Poor performance
 - 107

- Endoscopic exam definitive
- Obscures normal serrated margin of epiglottis & freq. its dors, vascular pattern
- Epiglottis not obscured (thicker & blunted at apex)
- Free edge of entrapping tissue seen over laryngeal surface of epiglottis
- M/ only entrap lateral margins of epiglottis Ulcerations of entrapping tissue
- Oral endoscope if concurrent dorsal displacement of soft palpate
- Lateral radiograph: shows thick ness, length & shape of epiglottis
- Measure of length of epiglottis
- From tip to thyroid cartilage (normal 8.76-9.35 cm)
- Short length poorer post surgical Px (< 7.5 cm)



Prognosis:

 Depends on preoperative complication (palatal or epiglottal anomalies)

Surgical removal or transection

of entrapping tissue (see box)

Length of epiglottis < 7.5 poor

Cause - unclear

- Happens during swallowing Assoc. w/ palatine & epiglottic
- defects (hypoplasia of soft palpate, persistent laryngopalatine distocation, cleft palate, hypoplasia of epiglottis & epiglottic deformities)
- Congenital & acquired forms



Subepiglottic cyst

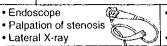
CT 281; M 156; T&W 225; S 461

- · Relatively uncommon
- CS: respiratory obstruction
- DX: Endoscope

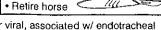
Endoscope

· Lateral X-ray

• Tx: Surgical removal



- Refer for surgery (difficult & complications)



Tracheitis E 770 • Bacterial or viral, associated w/ endotracheal tube, tracheostomy or smoke inhalation





12M 367; C4T 424; EM&S 424; E 728; M 156; S 488



· Acquired - direct trauma

- Retire horse





CS: Cough, Nasal discharge Exercise intolerance Dx: Difficult - CS & PE

Tx: Rest, ABs for 2° bact.

- IgM & IgG in lungs, enhances PMN & macrophage phagocytosis of virus
 - Immunization or exposure stim. production of these antibodies

• ★ Temp (biphasic, usually missed)

Lethargic, depressed &

Nonproductive hacking

cough characteristic of influenza

Serous or mucoid nasal dis-

charge, unlike other 1° viral causes

No lymph node involvement

· Uncomplicated cases, heal w/

Complicated - 2° bacterial in-

fection (those having no rest, no de-

fense mechanisms of respiratory tract,

- Limb edema (severe & significant -

stock up due to no exercise when

previously used to lots of exercise)

immunocompromised & foals) - Mucopurulent discharge

- Productive cough

- CS of pneumonia Uncommon complications

- Myocarditis

- Myositis

Re-exposure - anamnestic response

103-106° F

Weakness

in few weeks

partially off feed

History of explosive outbreak

Prognosis:

Excellent in

uncomplicated cases

Stall rest for 3 weeks w/ hand walking

Well ventilated stall, protected from

NSAIDs, use to decrease lever so animal

Antibiotics minimum 7 d (controversial)

NSAIDs

- Usually put on prophylactically

Isolation of affected horses

weather, keep down dust

continues to eat & drink

- Important to foals

Vaccine (see pg 12)

dam not vaccinated)

Multiple killed vaccines

after 4 month vaccine

- However, CS very similar to other viral infec.
- Acute & convalescent antibody titers (3-4 weeks apart compared)
 - 4 fold or greater incr. signif
- M/not see if previously vaccinated
- Not usually done, recommended if many animals to provide preventative med. such as vaccinations
- Viral isolation from pasal swabs early, first 48-72 hours, not usually done: \$, time)



- Rhinopneumonitis (pg 111)
- Eq. viral arteritis (pg 110)
- Other resp. infec. (pg 112-115

months for life

- Pleasure horses: Biannually

- Performance horses: every 2-3

Revaccinate at 12 months

If backyard & no new animals, don't need to revaccinate beyond 2-3 years of age Manufact, recommends annual revac., not

- Initial vac at 3 month, (at 1 month if

Booster in 3-4 weeks (weanling)

if started before 3 months need 3 dose primary series
If at risk revaccinate every 2-3 months for life

- sufficient for young & performance horses
- Brood mares: Biannually
- If pregnant 1 month prior to fealing

Influenza 1; Equine influenza

M8k 1084: Mk 737: IM 525: 12M 585, 1636; EM&S 380; E 732; C4T 446; C3T 316; Pop 17-7/97

- · Most common respiratory virus
- Myxovirus w/ 2 antigen types
- No cross species infection
- Subject to antigenic "drift"
- inactivated by most common detergents Epidemiology:
- Oubreaks: spreads rapidly through group of susceptible animals
- IP (Incubation) 1-3 days
- Throughout herd in 7-10 days
- 1-3 yr-olds, all ages susceptible
- Most severe in unexposed previously or unvaccinated
- Shed virus 1-8 days
- M/b carriers w/ no CS
- M/b vaccinated & shed w/o CS
- Generally restricted to URT (upper respiratory tract)
- Foals 1° LRT (lower resp. tract),
- bronchopneumonia, pneumonitis



Pathogenesis:

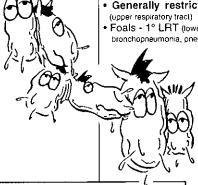
- Inhalation &/or direct contact w/ nasal secretions

Invades respiratory epithelial cells, causing hyperemia, edema & desquamation of epithelial cells of the nose, pharynx, larynx & trachea

Superficial erosion & loss of ciliary clearance mechanisms (potential to develop lower respiratory tract diz)

Repair time up to 3 weeks (re-epithelialization)

Must be rested or develop complications

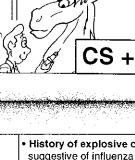


Explosive outbreaks CS: URT - Nasal discharge

Dx: Hx

Tx: Isolate, Rest, ABs, NSAIDs

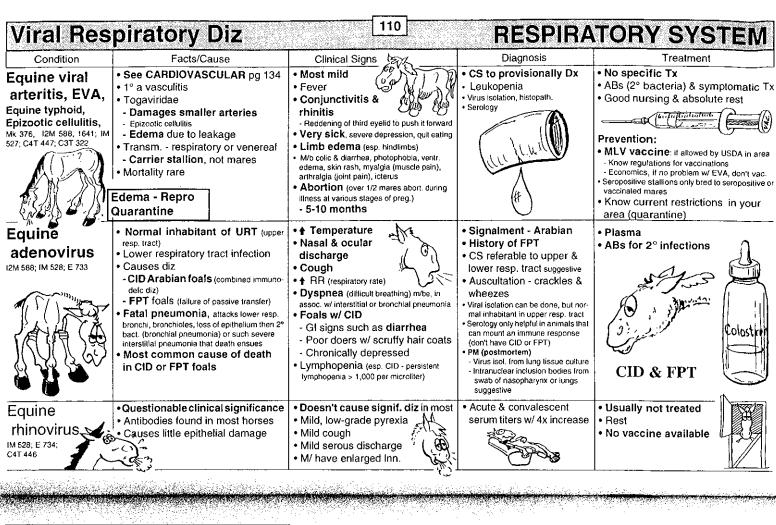
Vaccinate











"Snots" EHV-1, Rhinopneumonitis, Herpesvirus 1, Equine viral

rhinopneumonitis, Eq. abortion virus M8k 1081; Mk 734, IM 526; I2M 587, 1638; E 733, C4T 444; C3T 319; Pop 28-9/97



- 4 separate syndromes, resp, abortions, neonatal & CNS diz
- Herpes virus 1, 2 serotypes, Type 1 causes abortion, CNS & resp. diz, Type 2 resp.
- Transmission:
- Inhalation direct or indirect from nasal discharge, aborted fetuses, placenta
- IP (incubation period) 3-7 d
- Carrier animals recrudescence
- Introduced to farm by new horses
- Rapid spread, in susceptible horses
- High morbidity, low mortality
- Annual outbreaks in foals (inhorse) areas); herd immunity determines if episode occurs
- Pathophysiology:
- Multiply in epithelium of nasal, pharyngeal & lonsil areas, damaging epithelium T-lymphocytes transmit virus through body
- Respiratory problems in foals
- Weanling (maternal ABs waning)
- Stress groups moved so can't hear moms
- Starts as upper then moves to lower in foals
- Asymptomatic in mares
- Abortion storms weeks to months later
- . Most common infectious cause (1/4 of all Dx abortions)
- Future breeding unaffected
- Immunity on recovery not

permanent

Rhinopneumonitis

CS: "Snots" - foals, Abortion storms

Dx: Hx, CS, Serology Tx: Rest, Vaccination

• Foals

- "Snots" runny (copious), serous nasal discharge (purulent if 2° bact) Conjunctivitis m/b
- Malaise, inappetence, edematous mandibular &/or retropharyngeal Inn., constipation followed by diarrhea
- ± Dry cough (self limiting, wk 10 d), m/ have dry cough for longer (3-4 wk)
- 2° bacterial infection common
- . Mucopurulent nasal discharge Appetite good, except in foals, poor due to more systemically affected, more depressed Lymph node involvement, submandibular & retropharyngeal Inn. enlargement
- Biphasic temp. 102-106°F
- Mares asymptomatic
 - Abortion 4-5 mo later
 - Last trimester (7-11 mo)
- · Neonatal death die in a few days M/b normal at birth then weak, resp. distress, irretractable diarrhea to death
- Interstitial pneumonia
- CNS (myeloencephalopathy) some Mild ataxia & posterior paresis to posterior paralysis & recumbency
- Most frequent in mares shortly after foaling during an abortion outbreak (m/b also in barren mares, males, foals, etc.)
- Can redevelop diz w/in 4-5 mo following infection

Hx (history), CS

- "Snots": can't be DDx from other viral diz by CS
- Virus isolation from nasopharyngeal swabs & citrated blood early in course of diz (rarely done)
- Serology of acute & convalescent sera (more commonly done)
- Leukopenia (neutropenia & (vmphopenia)
- Abortion: Hx of storm of
- "snots" on farm 4-5 mo prior



DDx: "Snots"

- Eq. viral arteritis (pg 110)

- Other viral infec.
- Eq. Influenza (pg 109), CS similar so can't distinguish, although Heroes gen, is milder
- S. eaui

Prognosis:

- "Snots": self limiting
 - Immunity lasts couple of years, not permanent
- Future breeding unaffected

"Snots"

 Self limiting (no \$ advantage of treating alth

Monitor

- Antibiotics empirically for 2° bact., if mucopurulent discharge or lung involvement
- Similar to influenza
- Stall rest for 3 wks, to heal resp. epith.
- NSAIDs, though masks CS, esp. bact. infec.
- Isolate infected animals
- Disinfect environment

Abortion Isolate

Vaccinate all







Prevention:

Isolate new horses 3-4 weeks

- Reduce stress (latent carriers)
- · Segregate breeding & training horses
- Keep at farm for 3 weeks after resp. or abortion outbreak
- Vaccine see pg 13, almost mandatory due to constant movement of horses
 - Give to all horses on premises, not just mares
 - MLV (Rhinomune®) only lessening CS
 - Immunity for only 2-4 mo
 - Foals 3, 4, & 12 months
- Foals at risk booster every 3-4 mo to maturity
- Adults
- Performance: 2-3 mo boosters
- Pleasure: biannually - Broad mares: Vac 5th, 7th & 9th months of gestation
- . Vac. in abortion storm (recommended) Both MLV & killed virus vaccines available & approved for use in pregnant mares



Rhodococcus *egui* lung, Abscessation in foals. Granulomatous pneumonia,

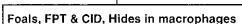
M8k 1087; Mk 743, I2M 580; IM 511; E736; EM&S 436; C2T 230; M 291

"Rattles"

- Gram positive pleomorphic rod In soil & manure of adults
- Very resistant, disinfect w/ phenol
- Hides in macrophages
- Strong immune defense to fight
- FPT & CID Arabs more susceptible
- Endemic to some areas. Continue to cause problems vr after vr
- . # of organism in environment ↑ in Spring
- . Affects 2-6 month-old (maternal antibod-
- ies waning) - Summer (incidence in hot, dusty, dry part)
- Route unknown: Aerosolization or ingestion & possible hematogenous spread from GI, parasitic arvae may carry it to lungs
- Suppurative pyogranulomatous pneumonia

- Abdominal "tucking" on inspiration
- Fever
- Nasal discharge
- Coughing
- ± Insidious onset & 1st noticed when advanced diz
- Diarrhea (if ulcerative colitis)(usually no GI CS w/o pneumonia)
- Other diz manifestations
- Enterocolitis
- Arthritis
- Hepatic or more commonly splenic abscesses
- Vertebral abscesses
- Hypopion (pus in anterior eye





CS: Pneumonia

Dx: Chinese letters, Rads: abscesses

Tx: DOC Erythromycin + Rifampin 4-10 wk





113

FPT

CID

Auscultation

- Adventitial sounds initially
- Some severe diz w/ no sounds
- Lab: ↑ WBC & Fibrinogen
- Radiology:
- Alveolar pattern (alveoli filled) m/b air bronchograms
- Nodular & cavitary lesions
- (abscesses) - Diffuse suppurative bronchial pneumonia
- Enlarged Inn
- TTW (transtracheal wash)
- Chinese letter organisms (pleomorphic rods) on gram stain highly suggestive
- Large number of PMNs
- Gram stain 1st to choose AB &
- start before culture & sensitivity results In past associated w/ high mor-



- 4-10 weeks \$ (always use together or
- get resistant strains) - Erythromycin IV, then orally - may cause
- severe diarrhea (discontinue)
- Pen & aminoglycoside combo (less effec-tive, not > 7 d w/o checking for nephrotoxicosis), used when can't tolerate erythromycin
- Trimethoprim-Sulfa (not as effective) Stop using antibiotics based on
- CS, rads & blood values
- Stall rested & hand walked
- Isolate, although present in soil, so less
- important than w/ S. zooepidemicus
- · Expense of Tx & then chronic poor doers



Prevention: Remove manure

- Change location
- Disinfect stalls w/ phenol
- # dust (water down)
- Endemic farms monitor foals (RR, temp & auscultation) every 2-3 d during 1st 4 mo
- Adequate deworming
 Vaccinate for viral resp. dizs

Prognosis:

- Most foals recover w/ early Tx
- Based on ★ of fibrinogen & age
- Younger the foal, higher mortality & longer Tx

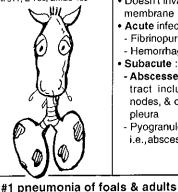
Presentation/CS

Condition Strep.

Facts/Cause adults zooepidemicus

Pneumonia Foals

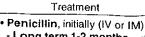
IM 511: E 736; EM&S 433



- #1 pneumonia in neonates &
- Septicemia in lung & 1° bact, pneumonia
- Adults 2° to resp. viral infec.
- · Normal inhabitant of upper respiratory tract
- · Doesn't invade intact mucous membrane
- Acute infection develops:
- Fibrinopurulent pneumonia
- Hemorrhage in some cases
- Subacute :
- Abscesses in lower respiratory tract including lungs, lymph nodes, & occasionally visceral
- Pyogranulomatous pneumonia i.e., abscesses

· Similar to adults

- Lethargy & anorexia
- Fever
- ♦ RR (resp. rate) or effort
- · Nasal discharge
- Coughing
- Exercise intolerance
- Diagnosis · Hx, CS
- TTW (transtracheal wash)
- ID gram positive cocci
- Culture
- Radiographs in field
- Bronchopneumonia
- Abscesses
- Lab: hematologically typical of bacterial infection. except in those septicemic, in which neutropenic & toxic changes



- Long term 1-2 months, well beyond duration of CS
- Monitoring discontinued 24 48 hr & reculture & base Tx on WBC, fibrinogen values back to normal, & rads
- Trimethoprim-Sulfa (orally) if IM muscle prblms w/ pen
- Restrict exercise (hand walk or small paddock)

Control:

- Isolate infected animals
- No vaccine
- Proper care of umbilious
- Adequate colostrum, etc.





Pneumocystis pneumonia IM 511; EM&S 441

CS: Coughing - Exercise intolerance

Dx: Gr. + cocci - Abscesses Tx: Pen - long term 1-2 mo

Pneumocystis carinii

- Usually a nonpathogenic opportunist
- · Problem in immunocompromised foals (FPT or CID)
- · Causes severe interstitial pneumonia
- Occasionally seen in foals w/ concurrent lower respiratory diz
- · Always associated w/ another infection



CID & FPT

Bacterial pneumonia Adults

IM 505; I2M 566; C4T 449; E



- · Lower Resp. Diz mostly bacterial usually 2° to viral resp. diz)
- #1 Strep. zooepidemicus (B-hemolytic)
- Normal inhabitant of mucous membranes, skin & tonsillar tissue
- S. equi (less common)
- 2° Gram negative complication of Strep.
- Pasteurella spp (most common), Klebsiella spp, E. coli, Bordetella bronchioseptica
- Anaerobe complication recently: Bacteroides fragilis & B. melanino-
- Not normal inhabitant so consider significant if cultured
- Pathophysiology:
- ·Overwhelming or compromising normal defense mech.
- . Cilia clearance, phagocytic cells, mucous membr. barrier
- Stress (transport, weather, training)
- Mixed infections synergy
- Aspiration

- Similar to foals
- Fever (m/b intermittent, take multiple to see fever spike)
- 🛊 RR (resp. rate) (resting rate sensitive parameter)
- Nasal discharge
- Coughing (m/ or m/not be spontaneous, induced by tracheal palpation)
- Exercise intolerance
- Chronic weight loss
- Enlarged lymph nodes



DDx:

- Infectious
- Viral (usually upper)
- Fungal & parasitic (less common)
- Noninfectious
- COPD (chronic obstructive pulmonary diz) (pg 120)
- 1º neoplasms (rare in horse)
- Metastatic neoplasms (renal or gastric carcinoma or
- Thoracic lymphosarcoma

· Hx, CS

PE (physical exam)

Auscultation

- Early - harshness &

intense expiratory sounds When expiratory as loud as inspiratory sounds = significant airway diz)

- Advanced: end inspiratory crackles (suggests transient atelectasis &/or + secretions)
- Pleural rubs (extend to pleura) - If ventral thorax quiet - subpleural
- abscessation or pleural effusion
- Percussion: fluid line if pleural effusion



- + WBCs w/ neutrophilia (often)
- ↑ Fibrinogen (500-1000 mg/di) (often inflam, hepatocytes stim, to produce)
- . Hi levels indicate chronic pneumonia or
- TPP + wk- mo due to chronic antigenic stim.
- Rads Dx, Px & progressive evaluation
- Cranioventral opacity
- Abscesses multiple opacities - Horizontal line - free fluid in pleural space
- Uitrasound
- Pleural fluid & fibrin w/in fluid
- TTW (transtracheal wash) culture, gram stain & sensitivity (EDTA tube for cytology & syringe for aerobic & anaerobic cultures)
- Degenerative PMNs w/ engulfed bacteria
- Thoracocentesis (if pleural fluid present)
- Drainage if interfering w/ respiration Cytology, culture (include anaerobic) & sensit
- Foul odor suggests anaerobes
- Necropsy: Consolidation (cranioventral), Adhesions, Necrotic debris in airways

Tx same as foals

- Vigorous antibiotic therapy (culture & sensitivity important)
- Penicillin initially best (IV or IM) Long term 1-2 mo, usually well
- beyond duration of signs Monitoring - discontinued on basis of
- WBC counts
- Fibrinogen values back to normal Rads-resolution of pneumonia (rad CS last longer than diz)
- Temporarily stop ABs for 24-48 hr & re-culture, if WBC count & fibrinogen return to normal & rads resolving stop
- Trimethoprim-Sulfa (orally) switch from IM pen in very young if pain & long term muscular problems
- Thoracocentesis drainage (if pleural fluid interlering w/ resp.)





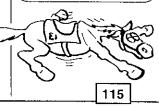
#1 Strep. zooepidemicus, Similar to foals

CS: Abscesses - Cough - Nasal discharge Dx: Hx, CS, PE, TTW

Tx: Penicillin long term 1-2 mo

Tuberculosis IM2 594

Rare in horses, especially in USA







Shock organs of horse - Lunas

Allergic reaction to allergen

initiated by nonallergenic mech.,

Both reactions cause release of

chemical mediators (histamines,

Anaphylactoid reactions -

chemical or physical stimuli

kinins)

Large colon

Diarrhea

Life threatening

Epinephrine IV

 Severe respiratory distress, recumbency, convulsion & death

- Pulmonary edema

- Emphysema

- Edema & hemorrhage of large intestine

- Laminitis

intervals if necessary

Cortícosteroids

 Methylprednisolone Na succinate (1-2 mg/ kg IV or IM) or

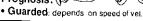
Dexamelhasone (0.25 - 1 mg/kg IV or IM)

Antihistamine

- Diphenhydramine (0.25 - 1 mg/kg IV or IM)



Prognosis:







Pleuropneumonia

RESPIRATORY SYSTEM

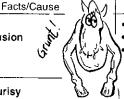
Condition

Pleuropneumonia, Pleuritis. **Pleurisy**

M8k 1086; Mk 741; IM 516; I2M 577; EM&S 459; E 774, C4T 453; C3T 327



 Pleuritis & pleural effusion



Cause: Pleurisy

- · Most commonly w/ pneumonia orlung abscessation
- General anesthesia & recumbency due to underlying pneumonia
- Viral respiratory infections
- · Uncommon causes include trauma, perforating wounds or open chest wounds
- Esophageal perforations
- · Less commonly, EIA, mycoplasma, Norcardia
- · Pneumonia, w/ or w/o lung abscessation
- 90% Strep. zooepidemicus
- E. coli, Pasteurella spp, Actinobacillus spp, Klebsiella pneumoniae
- Majority mixed infec. aerobes & anaerobes
- Anaerobes: Bacteroides fragilis
- Seen after stress
 - Transport, shows, races
 - More common in Thoroughbred & Standardbred race horses

Pain - "Grunts" - S. zooepidemicus

CS: Putrid breath - Anaerobes

Dx: Auscult/Percuss - Thoracocentesis

Tx: Long term ABs - Metronidazole/anaerobes

Px: Guarded

Presentation/CS

- · Similar to cattle
- · Resp. distress (pleural effusion) Acutely painful
 - "Grunts", intercostal muscle spasm
 - Abduct elbows, "catch" to inspiration
 - Guarded shallow respiration Reluctant to lie down or move around
- Guarded cough, appear anxious
- Non-specifically off feed
- Nasal discharge m/b, mucopurulent, often bilateral
- ± Foul smell if anaerobes
- Fever, anorexia, depression
- Stiff gait
- Colic
- CHRONIC:
- Continued depression, anorexia
- Not painful, nerve fibers in parietal pleura destroyed as fluid builds up
- Weight loss
- Sternal or limb edema 2° to fluidin pleural cavity & peritoneal cavity (protein loss), & due to blockage of lymphatics



Diagnosis

. Putrid breath if anaerobic infec.

• CS - resp. distress & pain

No lung sounds ventrally:

- Dull resonance ventrally,

- Horizontal fluid line

US: picks up small amount of fluid

sensitivity (include anaerobic)
. Gram stain for tentative bacteria

Cloudiness: # # of WBCs

1600 - 300,000 cells/Mt

Protein in pleural fluid > 3g/dl Hematology usually nonspecific

Anaerobes in 46% of cases

- Foul odor suggests anaerobes

- WBC count - normal < 10,000/µl

. Pleuropneumonia WBCs betw.

auscullation:

Percussion:

oormal dorsally

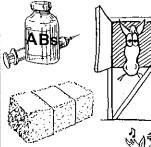
Thoracocentesis

Cytology, culture &

- Long term Systemic ABs
- Empirically until C&S, Brd. spectrum ABs Follow IV or IM w/ oral ABs

Treatment

- Anaerobic pleuropneumonia Metronidazole/combo Tx
- Pleural drainage (see box)
- Anti-inflammatory Rx for pain &
- ♣ productivity (Banamine®, PBZ)
- Corticosteroids controversial (Contraindicated?)
- Rest
- Adequate diet
- Encourage eating because of prolonged course





- · Guarded in all cases
- Poor anaerobes
- Poor survival rate foul odor to peritoneal fluid or breath
- Pleural fluid cell count & prot. no prognostic value

Diagnosis:

- CS respiratory distress & pain
- Pleximeter (spoon) to percuss chest for pain
- · Putrid breath if anaerobic infection
- Auscultation
- Absence of lung sounds ventrally (or only bronchotracheal sounds)
- Normal dorsal lung sounds
- M/ or m/not be adventitial sounds, underlying pneumonia, lung compressed dorsal,
- can be due to lung consolidation Heart sounds louder & wider area than normal due to fluids
- Pleural friction rubs acutely only

Percussion

- Dull resonance ventrally, & norm, lung resonance dorsally unless abscess against body wall
- Horizontal fluid line
- Ultrasound
- Picks up small amount of fluid missed by auscultation,
- percussion & radiographs
- Hypoechoic & anechoic fluid
- Abscesses if contiguous at lung surface - Pulmonary consolidation & atelectasis
- Thoracocentesis EDTA + sterile tube
- Cytology, C&S (culture & sensitivity) include anaerobic
- . Gram stain for tentative bact.
- . Anaerobes in 46% of cases (routinely use anaerobic transport media, do not refrigerate/kills)
- Visualize normally clear & yellow
- . Cloudiness: ## of WBCs
- , Foul odor suggest anaerobes (no odor doesn't rule out [R/O] anaerobes, compare to breath odor)
- WBC count norm. < 10,000/μl
- . Pleuropneumonia WBCs betw. 1600-300,000 cells/ut
- Protein in pleural fluid > 3 g/dl
- Hematology usually nonspecific
- Chronic: anemia & elev. TPP > 8 g/dl WBCs not always elevated
- Fibrinogen usually elevated, indicates inflammation

















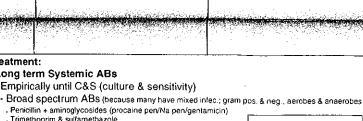
Thoracocentesis:

- · Ultrasound to guide
- EDTA (anticoagulant tube) + sterile tube (routinely use anaerobic transport media, do NOT refrigerate, cold kills)



- · Use cannula, chest tubes or thoracotomy
- · Thoracocentesis w/ cannula easy
- · Indwelling chest tube continued pleural effusions
- 1 way flutter valve so air doesn't get in
- Aseptic placement & maintenance can leave for wks
- Remove when nonfunctional
- Heparinization after drainage to maintain patency
- Open drainage if chest drain insufficient
- Open chest & treat as a draining abscess





Metronidazole combos

Bacteroides fragilis resistant to penicillin Metronidazole: effective, but not against aerobes so always combo therapy

- Pleural drainage (to drain or not to drain?)
- If thick pus, drain w/ chest tube
- No pus, but gram positive & WBC count 1. drainage recommended

Empirically until C&S (culture & sensitivity)

- Follow IV or IM w/ oral ABs (after condition is stable)

Penicillin + aminoglycosides (procaine pen/Na pen/gentamicin)

. Empirically (from experience) bec. hard to do culture & sensitivity



Treatment:

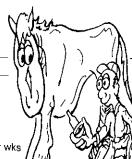
Long term Systemic ABs

Chloramphenicol

Trimethoorim & sulfamethazole

- Anaerobic pleuropneumonia

- . 6th or 7th ICS at level of costochondral junction



Pneumothorax

Mk 635, 708; IM 531; I2M 591; EM&S 457

*

Rare, Trauma CS: Dyspnea Tx: Relieve

 Rare in horses · Causes:

- #1 trauma (out of starting gates) Rib fxs., puncture of trachea, rib punctures lung Rupture of emphysematous lung bullae

- Rupture of esophagus

Types of pneumothorax

Closed: air trapped in thorax Open: air out of thorax through wound

Tension pneumothorax

. Flap of tissue allows air in

thorax, but stops escape On PE m/not find open chest

wound, m/ have fx rib that penetrated parietal pleura

- Air in both sides

2 sides of thorax communicate

• ± External wound

 SQ emphyse Dyspnea

Tachypnea

Tx: Dust free - Steroids - Bronchodilators

± Cyanosis

 • Movement of chest wall



- Absence of lung sounds dorsally (lung partially collapsed)

- Hyperresonance over area of pneumothorax Rads diagnostic, not usually done

· Aspiration of air from thorax

Ultrasound (SQ emphysema m/

prevent use)

SQ emphysema + dysonea, suspect pneumothorax because SQ emphysema doesn'i cause dyspnea

Prognosis:

Good if seals & air removed

 Poor if air leaking from pulmonary or esophageal lesions

· Hx of doing well for couple of days, then respiratory stress & diarrhea

♣ RR (respiratory rate)

Auscultation

- No adventitial sound

- Loud bronchial tones

Nonfetid diarrhea

Postmortem

- White focal lesions on liver

- Intranuclear inclusion bodies present in multiple cells throughout body

- Hyaline membranes rare

Serum titer in foal prior to suckling, if positive, mother had it



· Relieve pneumothorax & Tx_cause

Telanus antitoxin

Broad spectrum ABs

· Open "sucking" wounds

- Closed & chest tube

Extremely dyspnic

Get negative pressure by putting tube in dorsal & aspirate air off pleural cavity, or Sterile needle dorsal to tubing into bottle of water (suction apparatus)

If not severely distressed:

- Leave alone & air will be resorbed

• If Sx necessary - stabilize horse 1st & place chest tubes

Recurring pneumothorax

- Leave tubes in place for continuous drainage (indwelling chest catheter & Heimlich chest drainage valve)

Neonatal respiratory distress syndromes,

RDS. **Immaturity**

IM 327; I2M 363, 372; C4T 604, 609; EM&S 429; M 290

· Complex of diseases: 1) Birth asphyxia in associated w/

dystocia, placentitis, C-sections

Meconium aspiration

3) Prematurity (less than 320 d) or dysmaturity (320 ds or older, but immature

- No surfactant: + surface tension - alveoli collapse, inability to expand lungs

 2° to placentitis (E. coli, Strep. zooepidemicus)

5) Utero acquired rhinopneumonitis (Herpes virus 1)

No surfactant

CS: Dyspnea, Diarrhea & Death

Dx: Hx

Tx: Euthanasia

Foals born normally or weak at birth, then become more sick

w/n week

Respir. stress & diarrhea - Poor suckle reflex, depressed

- Generally die w/n 1-2 wk

2º bronchopneumonia



 Therapy unrewarding · Intensive nursing

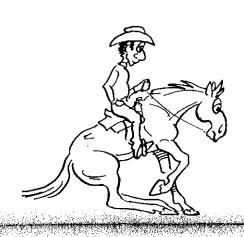
Antibiotics to prevent 2° infections

 Usually get adequate colostrum, but tend to stop suckling so IV fluids indicated

Usually euthanasia



Prognosis: Poor



CIRCULATORY SYSTEM

Anemia	134	Embolism	127	PDA	130
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Elevated PCV DDx	350	PCV DDx	350		

Pericarditis

M8k 91: Mk 52; IM 476; I2M 532; EM&S 330; E 681; C3T 402; C2T 171

**

Facts/Cause

- . Uncommon (more common in cattle)
- Fluid into sac (pericardial)
- . Generally 2° to pleuritis
- #1 cause Idiopathic, not uncommon No bacteria, nonsuppurative w/ pericardial effusion transudative in nature, not relative
- Heart failure due to compression
 - Sudden death
- · Pathophysiology:
- Inflammation of pericardium
- Accumulation of fluid &/or exudate in pericardial cavity
- -Compression of heart w/ decr. ability to pump Compensates by vasoconstriction, + HR, Na retention
- . Circulatory collapse if fail to maintain cardiac oulput
- Fibrin constrictive
- Types:
- Effusive (removal of fluid helps)
- Constrictive (fluid removal doesn't help)

- Both

Uncommon, Effusive or Constrictive

CS: Edema, jugular pulse Dx: Auscultation, US, Tap

Tx: Px - Poor

Presentation/CS

- Nonspecific: fever, anorexia, depression, weight loss
- Peripheral edema
- Jugular venous distention & pulsation
- + HR & RR
- Colic
- Syncopic episodes
- Congested mucous membranes Cvanotic
- · CHF (congestive heart failure)



Causes: pericarditis

- #1 Idiopathic, not uncommon
- · Trauma (ingestion of penetrating FB, external punctures)
- Hematogenous spread (septicemia) Strep. zooepidemicus > S. equi
- Extension of infection from lungs or pleura (pleuritis)
- Viral infection: parainfluenza & equine arteritis
- Neoplasia

Diagnosis

- Clinical signs Tachvcardia
- ★ Capillary refill time
- · Arterial pulse weak
- Auscultation
- Absence ventral (lung sounds
- Louder than normal dorsal
- Muffled heart sounds
- . No splashing, washing machine murmur as in cattle (no gas producing organisms in sac)
- Pericardial friction rubs
- Ascites
- Lab: CBC not specific, fibrinogen often elevated - Chem. usually normal, except m albumin often & # globulin
- Dehydration († PCV, TPP, BUN & creatinine)
- + AST, CK, LDH (myocardial isoenzymes)
- Rads not sensitive ECG: + QRS, S-T segment elevated or slurring
- Ultrasound confirms pericardial effusion, note changes of heart movement, thickened myocardium (chronic) Echo-free space in pericardial space M/b echo dense fibrin
- Pericardiocentesis
- Lt, 5th ICS 1-4" above electanor
- Incr protein > 2.5 g/dl - Bacteria & viral culture m/b neg
- Pleural & peritoneal fluid (if any) moderate transudate
- PM (postmortem)
- Distended pericardial sac (serosanguinous or urine colored, foamy fluid)
- Fibrin & pleural effusion
- CS of congestive heart failure (CHF)

Treatment

- Tx unrewarding
- EFFUSIVE
- Pericardiocentesis w/orw/o lavage
- · Local infusion of antibiotics
- CONSTRICTIVE
- Pericardectomy TOC (Tx of choice)
- Prolonged surgery, expensive & risky
- ABs (gram negative & pos. & anaerobes) - Initial gram stain or empirically Culture & sensitivity (C&S)
- **NSAIDs**
- Steroids if negative bacteria culture
- Contraindicated diuretics
- (improve ventral edema, but reduce venous return further compromising heart failure)



Prognosis:

Poor, in many cases no Tx; & even it intensive Tx, end up euthanized



 Routine vaccine for respiratory viruses

heart failure, CHF

Right sided CHF, fluid backs out to the periphery

- (pulse should only go 1/3rd of the way up the neck)
- Ascites (abdominal fluid)
- Splitting of 2nd heart sound, pulmonic & aortic valves not closing synchronously due to dilation of right



Left sided CHF: fluid backs up into lunas

- Poor peripheral perfusions
- · Pulmonary edema w/ crackles on auscultation



Often see bilateral heart failure in horses & cattle

pulmonary hypertension

DDx:

- Right heart failure (pg 125)
- Bacterial endocarditis (pg 132)
- Right AV insufficiency
- Cardiomyopathies (pg 126)
- Pericarditis (pg 124) Left side failure (pg 125)
- Pleuritis or pleural
- effusions (pg 118) Congenital pulmonic valve stenosis
- Cardiac neoplasm (pg 127)

Myocarditis

Fluid backup:

Mk 50; IM 472; I2M 527; EM&S 320; E 688; C3T 393



Uncommon

Dx: Rarely Dx

Tx: Digoxin, rest

CS: Sudden death

Uncommon

Lt. side: into lungs - dyspnea

Rt. side: into body - edema, ascites, pulsation

- Inflammation of myocardial wall
- Cause Myocarditis · Bacteria from septicemia, pericarditis, endocarditis
- Staph. aureus, Strep. equi, Clostridium, Mycobacterium · Virus: Eq. influenza, Eq. viral
- arter-itis (EVA), Eq. infectious anemia (EIA) Parasite (strongyloides,
- onchocerciasis) migration Thromboembolic diz from bacterial,



- Variable or unnoticed
- 1° diz m/ mask vague heart signs (Strangles)
- Tachycardia
- Febrile
- Muscle pain (myalgia)
- · Reluctance to move
- Exercise intolerance
- · Congestive heart failure CS
- Peripheral edema • Sudden death after exercise
- . M/ lead to idiopathic dilated cardiomyopathy

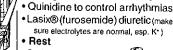


 Rarely diagnosed, mild vague signs of heart involvement

- 1° cause masks heart signs • 🕈 AST, CK, LDH (aspartate amino transferase, creatine kinase, lactate dehydrogenase, which are also # in
- hepatic & skeletal muscle problems)



- Masked heart signs
- Lameness
- Heart signs
- Cardiac neoplasia (pg 127)



sure electrolytes are normal, esp. K*) • Rest Control complications

Dysrhythmias

CHE

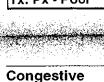
- Shock



Px: Good, if no CHF Guarded to poor with CHF

- Prevention Vaccination for bacteria





325, 521; EM&S 271, 273; E

M8k 81; Mk 46; IM 100; I2M

· Peripheral edema: brisket, submandibular, limbs · Jugular pulsation, jugular distension

 Respiratory dyspnea - Prominent S3 (third heart sound) - Tachycardia Pleural effusions

> Cor pulmonale (EM&S 273): Right heart failure due to underlying chronic respiratory failure due to progressive

> > Treat 1° agent • 1° Digoxin, positive inotropic agent

PM: M/b no gross lesions



Septicemia (pg 35)
 Respiratory diz (pg 93)

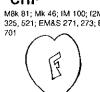
 CHF (pg 125) Cardiomyopathy (pg 126)



Colic (pg 54)

Endocarditis (pg 132)

& viruses Parasite control



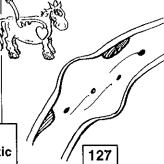
Thrombosis -**Embolism**

M8k 99; IM 483; I2M 538; EM&S 284, 287; E 685; C4T 267; C3T 383, 406, 510; C2T 173



- #1 Catheterization
- Trauma, venous stasis
- Thrombophlebitis: inflam, of vein associated w/ a thrombus
- Embolism: foreign material carried in bloodstream
- Frequently arise from thrombus
- Parasitic arteritis most common cause in horse (aorta & cranial mesenteric a.) S. vulgaris
- Aorticioiliac thrombosis in heavily exercised horses
- Bacterial endocarditis, thrombophlebitis, omphalophlebitis
- Arteriosclerosis: thickening of arterial wall
- Cause: Strongylus vulgaris

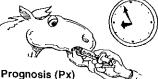
- Occurs 12-24 hr after removal of catheter
- Thrombosis of terminal aorta or
- iliac arteries Vague hindlimb lameness, exer-
- cise intolerance & poor performance, heavy sweating, except for hindlimbs, cold hind-
- Emboli: cranial mesenteric a. Chronic episodes of colic



- Positive blood culture
- Thrombosis of terminal aorta or iliac aa
- Rectal palpation of enlarged, firm terminal aorta
- Radiographs:
 - Aneurysm: soft tissue extensions of vessels
- Ultrasound
- · Postmortem:
- S. vulgaris in cranial mesenteric a. or aortic wall
- Dilation of vessel (aneurysm)



- Surgical removal of thrombus rarely attempted, except for jugular vein
- Anthelmintic for S.vulgaris

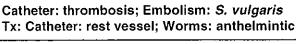


Aneurysms: guarded to grave

Thrombus: guarded

Prevention

- IV catheters: asepsis & replacement when phlebitis occurs
- Silicone rubber or polyurethane catheters for prolonged use
- Septic or endotoxic patients:
- . Aspirin (17 mg/kg every other day)
- . Low dose heparin (30 U/kg SQ BID)
- Parasite control for S. vulgaris (see pg 14)



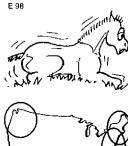
CIRCULATORY SYSTEM

Stix

Vit. E / Selenium

deficiency diz. Nutritional myodegeneration, NMD, White Muscle diz,

Enzootic muscular dystrophy Mk 538, IM 1352; I2M 1513, 1881;



Vit. E/Se defc. in foals 1°

• Rapidly growing 2 wk to 4 mo

Facts/Cause

- · White muscle diz uncommon in adults
- Mechanism not understood
- Selenium (Se) or Vit. E defc (antioxidants, protect from free radicals)
- PUFA, polyunsaturated fatty acids in diet (produce free radicals) - lead to Vit. E defc, especially in calves
- 2 syndromes of degeneration
- Cardiac form: pulmonary edema
- Skeletal muscle form (more common)

Se deficient soils (NE, NW USA, E Seaboard)

- Acid soils
- Hi sulphur inhibits Se uptake
- Vit. E defc poor quality hay, straw or root crops
- Congenital: death 2-3 ds old
- Delayed type: 2 wk 6 mo



 Sudden death, handling/exercise brings on signs, or death ⊕ - Weak

- SKELETAL FORM
- Slower onset
- Muscular weakness
- Trembling
- Recumbent (unable to rise)
- Stiffness in gait, BAR
- -Swollen, hard supporting mm. (gastrocnemius, semitendinosus, semimembranosus, biceps femoris, lumbar, gluteal & neck muscles)
- Dyspnea if diaphragm & intercostal muscles affected)
- CARDIAC FORM
- Sudden onset - Sudden agonal death, or
- Depression
- Dyspnea (foamy nasal discharge)
- Profound weakness, recumbency
- Rapid, irregular heart beat

Anorexia in hrs

• ★ RR, HR

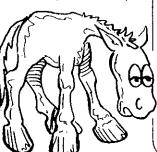
Mild colic, diarrhea

• Sweating (Intermittent perfuse)

· Progressive respir. distress

in 8-15 hr, recurs prior to death

Short course, death in 24 hr. despite Tx



DDx:

Sudden death

· Hx: CS: foals, calves

- Sudden death

- Vit. E. Selenium

not supplemented

tissue biopsy (lab)

- Myoglobinuria

Postmortem:

- Se levels in whole blood or

- ↑ CPK in 1000's IU/L, AST, LDH

- Glutathione peroxidase levels

- Pale muscles, linear pale areas in

Mvocardium: necrosis in cardiac

skeletal mm., symmetrical

areas even if no cardiac diz

& lambs

• I ah·

- Enterotoxemia, pneumonia (pg 112-116), toxemia Cardiotoxic plants
- Lasalocid, gossypol, Cassia occidentalis, Phalaris spp Copper deliciencies (pg 129)
- Excessive molybdenum/sulfates
- Lymphosarcoma, fibrosarcoma
- Stiffness in gait, BAR
- Spinal cord compression, trauma (pg 236) Cerebellar diz (pg 251)
- Meningitis (pg 258)
- Polyarthritis
- Neurotoxins (organophosphates)
- Tetanus (pg 241) Clostridial myositis

- 1 mg of Se/d
- Vit. E/Se to asymptomatic animals
- Vit. E/Se to affected, every 2 wk, not to exceed 4 doses
- Vit. E/Se: birth in defc areas
- Selenium toxicity- follow manufacturer's suggestion
- Use E-SE® made for horses (not bovine, too potent)

Prognosis (Px):

- Cardiac form: grave
- Skeletal muscle form: better

Prevention

- Supplementation to pregnant & lactating mares in defc areas
- Periodic blood (or tissue) tests for animals at risk



Some myopathies respond only to Se or Vit. E, some to both

Foals, Skeletal > cardiac forms CS: MM: Weak, trembling; Heart:

Dx: Hx, CS, Lab, PM: Pale muscles

Tx: Vit. E / Se defc

Monensin toxicity,

Mk 472; IM 1639; I2M 1917; E 198; EM&S 321; C4T 665; C3T 366

lonophore toxicity

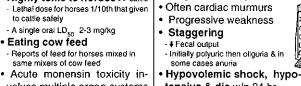
- Coccidostat in feedstuff for growing cattle
- Dilated cardiomyopathy
- Myocardial necrosis
- · Not seen often, but fatal
- . Highly toxic to horses >> cattle Lethal dose for horses 1/10th that given to cattle safely
- Reports of feed for horses mixed in
- volves multiple organ systems

Other ionophores

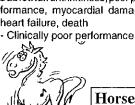
Lasalocid

Narasin

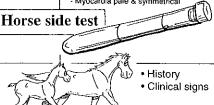
Salmonycin



- tensive & die w/n 24 hr If survive get 2° consequences
 - that lead to demise
 - Survive acute, CS of CHF in 2-5
 - Drop dead in training
 - · Sublethal: unthriftiness, poor performance, myocardial damage, heart failure, death



- History (Hx), Clinical signs (CS)
- Auscultation
- f or f GI sounds
- Muffled heart sounds
- Arrhythmias
- Lab: Hemoglobinuria
- ↑ PCV & TP, Hemoconcentration
- + Urine SG
- ★ BUN/creatinine
- + K⁺, then returns to normal
- ♣ Serum P having to do w/ marked increase in P clearance
- Urine pH acidic due to by-products of monensin breakdown
- + Unconjugated bilirubin (RBC injury)
- "Horse-side" test: heparinized tube of blood, darkens rapidly • ECG:
- Depression of "t" waves which coincides w/ hypocalcemia
- ST segment deviation (myocardial damage suggestion)
- Postmortem
- Hemopericardium, epicardial hemorrhage, edema around coronary aa. Pleural effusion & abdominal effusion
- Myocardia pale & symmetrical

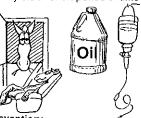




- Tx intensively
- Empty intestinal tract to 4 absorption
- Avoid parasympathomimetic drugs due to potential for cardiac arrhythmias
- Mineral oil, activated charcoal
- IV fluids, LR safest (hypotensive), K* supplementation, hypokalemic phase, indicated initially, however can't monitor K* levels
- If diz associated w/ renal failure, caution
- No digoxin, even though arrhythmias, m/ kill monensin-poisoned cardiac cells, causing further harm

Digoxin

- Mainly rest
- Many die in short period of time



Prevention:

- Clean silo between mixing feeds
- Settles from bag to bag so watch feeding horses "cow feed" w/ this additive
- Now using Lasalocid® instead
- Copper (injectable or dietary)

Prevention:



Mk 1237; IM 1104, 166, 1192, 1127; C4T 392

Copper

Hypocuprosis

deficiency.

CS: Dilated cardiomyopathy

Dx: Hx, CS, "Horse-side" test

Tx: Rest, Oil, Fluids, No digoxin

Cow feed, Coccidiostat, Horses >> Cattle

Cu deficiency & bleeding in aged parturient mares

Apparent relationship between

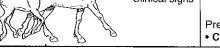
 Bleeding in aged parturient mares

± Loss of

pigmentation

129

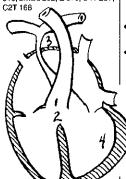
to the property of the contract of the contrac





Congenital Heart Defects 130 CIRCULATORY SYSTEM Condition Facts/Cause Diagnosis Presentation/CS Treatment VSD. Ventricular . Hx (foal), CS #1 congenital heart defect Asymptomatic if small None Auscultation Occurs singly or w/ other defects defect septal defect - Holosystolic murmur (harsh, Moderate w/large defects M8k 72, 63; Mk 37; IM 459; I2M 512; E 678; EM&S 262, 315; C4T Failure of the interventricular septum No Tx - Poor growth, lethargy to close (usually high up) Heard on both sides of thorax (PMI 255; C3T 409; C2T 167 - Allows left to right shunting (left ventricle - Dyspnea generally rt. side, because shunt directed to right) - Exercise intolerance - Palpable cardiac thrill Pressure overload - pulmonary hy-- CS of CHF - Splitting of 2nd heart sound pertension, dilation of right ventricle Angiocardiography - definitive Dx Px (prognosis) & left atrium #1 heart defect Postmortem Good: small defect - m/b Cause unknown - VSD asymptomatic for life CS: Asymptomatic to tiring - Lung congestion Poor: moderate to large defects Dx: Holosystolic murmur - Right ventral hypertrophy Intensity of murmur not cor-Tx: None related w/ size PDA, • Hx, CS · Ductus arteriosus: fetal connection be-CS depends on size Surgical correction in tween pulmonary trunk & aorta (shunts blood from Auscultation · Asymptomatic if small Patent ductus neonates (before right side presright ventricle to aorta, bypassing the lungs) Continuous "washing machine" CS of CHF sure builds up, if pressure in right ≥ left Closes w/in 4 days of birth normally to ("machinery") murmur (hi pitched, alterarteriosus Cyanosis to caudal body if will blow out lungs when Sx close form ligamentum arteriosum nating in intensity) in systole & diastole (aortic press >> pulmonic during both) shunt) M8k 68, 63; Mk 35; IM 461; (2M switches from lt.-rt. shunt If patent, results in left to right 515; E 676; EM&S 262; C4T 256; to rt.-lt. shunt shunt (aortic press >> than pulmonary) - PMI: left 3rd-4th ICS at level of shoul-C2T 169 . Overload of rt. ventricle & pulmonary arteries der (heard on both sides) Right ventricle fails or dilates & hypertrophies Later murmur during systole & finally none (to raise pressure) when right & left sides are of equal pressure Pulmonary congestion & hypertension Angiocardiography definitive Dx Right to left shunt when pulmonic circulation Radiology pressure ≥ systemic circulation pressure - Cardiomegaly (not specific) Occurs by itself or w/ other defects - Pulmonary overcirculation (not specific) ECG no consistent pattern Lt to rt shunt PM (postmortem) CS: Asymptomatic to cyanosis PDA (patent ductus arteriosus) Lung & chamber changes dep. on size of shunt Dx: Washing machine murmur Px (prognosis) (cardiomegaly, left & right ventricular hypertrophy, Tx: Correct early Insufficient data pulmonary congestion & edema) Tetralogy of Multiple congenital heart defects Cyanosis (mucous membranes Hx, CS None - Overriding aorta of mouth, nose & vagina) Auscultation: Fallot Slow growth (owner's com-- VSD (ventricular septal defect) - Loud holosystolic murmur M8k 72, 63; Mk 38; IM 462; I2M - Pulmonic stenosis (It. 4-6th ICS) 516; EM&S 262; E 679; C4T 257; Exercise intolerance No Tx - Right ventricle hypertrophy (due to - Gallop rhythm w/ exercise, early C2T 168 Dyspnea pulmonic stenosis) systolic ejection click Collapse Ventricular pressures equalize, Palpable thrill associated w/ murmur

131



- because VSD usually large
- More common in calves than foals

Multiple defects

CS: Cyanosis, slow growth

Dx: Holosystolic murmur

Tx: No Tx

- · ECG: generally nonspecific, right axis deviation
- Ultrasound
- Right ventricular hypertrophy
- Equal sized ventricular lumens
- Overriding aorta
- VSD (ventricular septal defect)
- Cardiac catheterization: equal ventricular pressure
- PM (postmortem)
 - Round apex, hypertrophy of right & left atria, right ventricle & ventricle septum
- Overriding sorts
- VSD (ventricular septal defect)
- Pulmonic stenosis

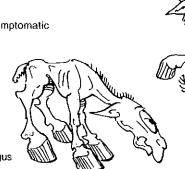


Px (Prognosis)

- Poor w/ cyanosis, exercise intolerance or poor growth
- Intensity of murmur not correlated w/ severity

Other Congenital heart defects MBk 71; Mk 34; IM 463; 12M 518; C4T 257; EM&S 262; E 676

- Atrial septal defects (ASD): most commonly patent foramen ovale, common in calves, often asymptomatic
- Pulmonic stenosis: uncommon by itself, in foals w/ VSD CS murmur & cyanosis variable
- Tricuspid valve atresia: rare in foals w/ other defects, cyanosis, murmur
- Ventricular hypoplasia: foals & calves, usually associated w/ early death
- Aortic anomalies: persistent rt. aortic arch & double aortic arches CS: esophageal obstruction
- Persistent right aortic arch: forms a ring around the esophagus with the pulmonary trunk, ligamentum arteriosum & base of heart, constriction of the esophagus





CIRCULATORY SYSTEM

Condition

Facts/Cause

· Usually acquired, common

Older horses

- Bacteremia

Cause: unknown

Presentation/CS

Diagnosis · Hx (older) CS • #1 Cardiac murmur

musical murmurs

arterial pulse

Endocardial diz. **Bacterial**

endocarditis, Vegetative endocarditis,

Valvular heart diz M8k 62: Mk 49: IM 465; I2M 520; EM&S 302, E 684; C3T 399

 Subclinical bacteremia . Septic thrombus - 2° to pyemia, mastitis or prostatitis

Corynebacterium pyogenes)

. Streptococcus most common

. Actinomyces pyogenes (formerly

- latrogenic jugular sticks
- Migrating strongyles (no proof)
- Vegetation on AV valves Layers of fibrin, blood cells, bacteria & necrotic tissue on endocardium of valves
- Aortic valve > left (mitral) AV > Right (tricuspid) AV, uncommon pulmonic
- Insufficiency murmurs (valves don't close & blood leaks backwards)
- PMI over 4th ICS at shoulder
- Disseminate sepsis: seeding of other systems, lungs, kidneys &
- Can progress to CHF

• Exercise intolerance

- · Weight loss
- CHF signs
- Jugular distention
- SQ edema
- Ascites
- · Intermittent fever, anorexia • Disseminated sepsis, CS to other organ systems
- Pyelonephritis
- Pneumonia (adventitial sounds, ♣ bronchial tones)
- Lameness, hematuria or pyuria



 Ventricular dysfunction. - Thickening of valve, ragged or cystic

- Holodiastolic, decrescendo,

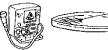
Intensity doesn't correlate to severity

Water-hammer or bounding

Ultrasound (B-mode, 2 dimensional)

- Chamber enlargement

- Pos. blood culture, bacteremia, Cultures not always positive - Ideally blood cultures collected at temp
- elevation, before peaking Monitoring temperature necessary, 5 cultures taken, usually 10 min. apart
- PM (postmortem)
- Vegetations on valves, shaggy, changes of nodules, fibrous thickening, fenestrations or holes through valves
- Lab: Leukocytosis
- · ECG: not reliable
- · Radiographs: nonspecific



Vegetation valves: growth stops valves from completely closing, blood backs up through them - murmur of insufficiency [leaky], in horse aortic valve most commonly, then It. AV (mitral), rt. AV (bicuspid) valve rarely pulmonic (Cow - tricuspid most commonly)

Long term antibiotics \$\$

Fibrous tissue of vegetation m/ protect

Treatment

- from antibiotics
- Initially for gram positive
 Culture & sensitivity (C&S)
- . Gram negative: pen + aminoglycosides
- Rifampin in combo w/ another improves short term outlank
- Aspirin (to prevent platelet adhesions)
- Lasix® (Furosemide): diuretics for CHF
- Digoxin to improve contractility if CHF



Px (prognosis):

- Guarded to grave
- Frequently sudden death
- Chronic poor doers

- Endocarditis of other causes
- · Degeneration of valves
- Viral
- Inflammation
- Trauma
- Rupture of rt. chordae tendinae
- Cardiomyopathies (pg 126)

Atrial fibrillation

Valvular diz: Aortic,

Dx: Murmur, US, Culture

Tx: Long term ABs • Px: Poor

CS: Tiring, CHF, Disseminate sepsis

M8k 80, 63; Mk 45; IM 485; I2M 541; EM&S 241; E 691; C4T 240, 244, 253; C3T 387

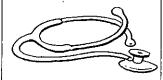


Common in horses

- Irregular heart beat
- Rapid uncoordinated atrial contractions (ECG - "f" waves) . Resulting in insufficient filling of ventricles
- Irregular spaced ventricular beats No 4th heart sound
- Idiopathic, not related to heart diz
- Generally not 2° to heart diz (if underlying heart diz, Px is worse)
- Not associated w/ GI diz as in cattle
- Geldinas
- . Benign condition usually

Exercise intolerance, #1 presenting complaint

- Exercise induced epistaxis (nose bleeding)
- Underlying respiratory diz
- Weakness, syncope (fainting)
- CHF (congestive heart failure)



 Sinus dysrhythmia (4th heart sound) · Ventricular & atrial ectopic beats

(regular rhythm)

Auscultation:

- No 4th heart sound
- Murmurs in less than 50%
- HR slow, normal or elevated
- Resting < 50 bpm generally - Exercise m/b 240 bpm
- Generally no pulse rate deficit unless hi HR
- ECG to Dx
- -Undulating "f" waves replace
- "P" waves Irregular cardiac rhythm w/ no underlying
- regularity
- Irregular R-R interval
- QRS irregularly spaced
 Ultrasound to detect underlying heart diz
- Generally no underlying heart diz
- Abnormal echocardiographic dimensions suggest heart diz

- Benign atrial fibrillation
- Quinidine sulfate orally Check ECG in 2 hr
- Every 2 hr maximum of 6 doses/day Repeat on day 2 if not converted
- Rest 4-6 wk best, but usually not done
- High heart rate
- Digoxin 4-5 d to slow HR to < 60 bpm
- Then quinidine therapy
- CHF or underlying heart diz
- Try quinidine, but conversion low, usually revert



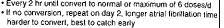


Prognosis:

- Good benign atrial fibrillation
- Guarded to grave: > 60 bpm or CS of CHF

Quinidine Quinidine sulfate orally (1 g/100 lb) Negative inotrope, causes systemic hypotension, ventricular response rate

- M/b bad side effects
- Use w/ caution (w/ CHF risky)
 Monitor w/ PE, auscultation & ECG
- Normal acid base balance & hydration before Tx
- Check if side effects & do EKG in 2 hours
- Side effects: colic, diarrhea, ventricular rhythm, supraver tricular dysrhythmia or laminitis discontinue if side effects
- Every 2 hr until convert to normal or maximum of 6 doses/d



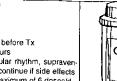


"f" waves, benign

CS: Tiring, epistaxis Dx: Auscultation, ECG

Tx: Quinidine







CIRCULATORY SYSTEM Anemia 134 Condition Facts/Causes Presentation/CS Diagnosis CS due to inadequate O2 to • † O2-carrying capacity of blood History: Anemia Treat underlying cause Pathophysiology of causes - Deworming body tissue M8k 8; IM 423; f2M 423, 1213; 1. Blood loss • ♦ HR (tachycardia) - Diet & access to pasture C4T 273; EM&S 1802; M 331 - Housing 2. Hemolysis (†Ē⊄⊅◊◊◊↔◊◊◊ de-• ★ RR (tachypnea) - Drug history struction) + Exercise tolerance (tiring) - Immune status (last Coggin's test) 3. Inadequate erythrocyte prod. Depression (bone marrow) Travel history (babesiosis) Pale mucous membranes Types Past illness (chronic anemia) Regenerative or responsive anemias - Mare history (isoerythrolysis) Blood loss & hemolysis - bone mar-- Exposure to new horses row responds by # erythropoiesis PE (physical exam) Nonregenerative: inadequate RBC Color of mucous membrane . Icterus in horses associated w/ hemolysis, fasting or cholestatic liver production in bone marrow - Epistaxis (chronic blood loss) MCV (mean corpuscular volume); reflects the - Respiratory or GI diz (any chronic inflammatory diz (abdominal abscess, size of RBCs pneumonia, or lymphosarcoma) will cause mild to moderate nonregenerative anemia) PCV X 10 MCV (fl) = $\overline{RBC \text{ count (millions/}\mu\text{l})}$ CBC - PCV reduced - Pink plasma (IV hemolysis) - # MCV indicates regen, anemía - MCV > 60 fl (rare) suggests regenerative anemia (immature RBCs larger than mature RBCs) Heinz bodies: suggests toxicosis by phenothiazine, **♦ MCV** (microcytosis) indicates red maple leaves, or wild onlons iron deficiency Agglutination suggests immune-mediated anemia . Coggin's & Coomb's tests in adults . Newborn: hemolytic cross-matching w/ mare **WBCs** . † PMNs (neutrophilia), † globulin (hyperglobulinemia) &/or † fibrin Blood loss, hemolysis or **♦** production (hyperfibrinogenemia) suggest chronic infection Hypoproteinemia: indicates blood-loss anemia or underlying diz causing PCV unstable - Spleen stores CS: Tiring blood loss (granulomatous bowel diz or intest. lymphosarcoma) Bone marrow analysis: necessary to adequately characterize Dx: Hx, PE, CBC, Bone marrow, Tests anemia as regenerative or nonregenerative Tx: Tx cause Diagnosis (cont. below) Horse RBCs have different characteristics than other species Diagnosis (cont.) Urine analysis 1. Unstable PCV · Spleen stores up to 1/3rd of RBCs - Occult blood: hemoglobinuria (from IV hemolysis) or Adrenergic stimulation releases splenic stores, incr. PCV by as much as 50% myoglobinuria (myopathy) Exercise, excitement or blood loss cause adrenergic stimulation • Fecal occult blood: indicates GI loss of blood (gastric ulcers - Therefore must evaluate PCV serially under dif. levels of excitement [foals] or gastric SCC [squamous cell carcinoma]) Can't use PCV to estimate magnitude of blood loss in hemorrhage for Serum iron & total iron-binding capacity (TIBC) at least 24 hr because of possible splenic release - Low serum iron & high TIBC - iron deficiency 2. Rouleaux formation: normal alignment of RBCs like stacked coins - Low serum iron & norm. TIBC - chronic anemia Gives a false high RBC sedimentation rate • Coggin's test positive indicates EIA (eq. infectious anemia) Must be differentiated from auto-agglutination 3. Icteric plasma: normal in horse (hyperbilirubinemia increases yellow color) . Coombs' test: positive indicates immune-mediated anemia (eg. infectious anemia, autoimmune hemolytic anemia, drug-induced 4. No peripheral signs or regeneration Only mature RBCs are released into circulation from bone marrow hemolytic anemia, neonatal isoerythrolysis) Polychromasia (reticulocytes), macrocytosis extremely rare 5. Howel-Jolly bodies: normally found in blood Doesn't indicate a responsive anemia as in other species Aα DDx - Anemia (large print indicates common causes, small print less common) Blood loss Hemolysis Inadequate RBC production - Neonatal isoerythrolysis (pg 139) - Intestinal parasites (strongyloides) (pg 36) Abdominal abscess or other chronic - Ectoparasites (lice (pg 272), ticks) - EIA (equine infectious anemia) (pg 138) abscessation - Gastric ulcers (pg 26) Chronic pneumonia/pleuritis (pg 118) - Red maple leaf toxicosis (pg 141) - Equine purpura hemorrhagica (pg 140)

- Equine ehrlichiosis (pg 135) Onion poisoning (pg 141)
 Autoimmune hemolytic anemia

· Babesiosis (piroplasmosis) (pg 144)

Incomplete blood transfusion

Clostridial infections

135

and the second programmer of the programmer of the second programmer and the second programmer a

- Equine ehrlichiosis (pg 135)

- Lymphosarcoma (pg 30)

(glomerulonephritis) (pg 148) Radiation loxicosis Idiopathic aplastic anemia

- Myelogenous leukemia

- Chronic renal failure

Eq. viral arteritis (pg 134)

- Immune-mediated thrombocytopenia

- Equine purpura hemorrhagica (pg 140)

Hemophilia A or other congenital factor defc. Guttural pouch mycosis (pg 100)

Warfarin poisoning (pg 143)

- Gastric SCC (squamous cell carcinoma) (pg 28)

DIC (disseminated intravascular coagulation) (pg 142)

Moldy sweet clover toxicosis (pg 143)

Presentation/CS

Swamp fever

M8k 499; Mk 28; IM 1073-4, 1401; I2M 1204, 1223; C4T 280; T 703, 707; EM&S 1804, 1837; Pop 14-1/98, 90-7/97 Rarely see clinically because controlled through testing

Facts/Cause

Mechanical transmission of blood of infectious horse

- Needles, blood sucking/ flies (deer, stable)

In utero

- Colostrum (foals sucking infectious mare)

Infected pregnant mares generally abort (but rare because test & euthanize)

Retrovirus: lifetime infection (whether show CS or not)

Horses, mules & donkeys

Stress exacerbates CS

 "Hot zone" (92% of EIA+ horses around Gulf of Mexico & Mississippi River)

· Intermittent fever

 Depression, progressive weakness

Weight loss

Edema (pitting)

Icterus

± Melena or epistaxis

Mucosal hemorrhages & petechiation (ocular, vulvar & ventral aspect of tongue)

Usually inapparent

. ± Acutely fatal hemolytic anemia

· Periodic flare-ups of CS + CNS

· Ataxia, weakness (encephalomyelitis, meningitis)

 Abortions any stage of gestation. especially during febrile episode Or, give birth to normal, live, free of infection foal

Diagnosis · Hx: wt. loss + periodic fever: suspect EIA

• I ab:

 Progressive or transitory anemia

♣ PCV & platelet count (thrombocytopenia)

- # Monocytes

- # Bilirubin

Coggin's test (agar immunodiffusion) for serum antibodies 95% effective

- Foals nursing infected dams temporarily positive (up to 6 mo. due to colostrum) Recently infected horses negative for 1 wk

45 d until antibodies form

If aged, will be positive sometimes

- State labs run test

Viral isolation impossible PM (postmortem)

Splenomegaly, hepatomegaly

Deposition of antibody/antigen in kidney (glomerulonephritis) incidental finding

0

No lesions in aborted fetus

Rare because of test, Infected for life CS: Recurrent fever, Wt. loss, Icterus

Dx: Coggin's test

Tx: Euthanasia positives



Short IP (incubation period)

• Multiplies in macrophages, stim. production of T & B lymphocytes

· Complement fixation antibodies & attach to RBCs

Both intra- & extravascular hemolysis

Treatment

Euthanize positives

No vaccination or specific Tx

No Tx clears the carrier state

· Isolate when diagnosed & keep isolated for life if don't euthanize





Control:

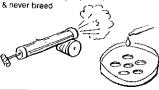
Stable fly control

 Foals from infected mares isolated until Dx free of infectious by disappearance of maternal antibodies (up to 6 mo)

 Tested for transport across state lines & at racetracks

Once positive can't transport

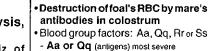
Tested every 6 months If positive, euthanize (depends on state), or put in quarantine for life & never breed



Neonatal isoerythrolysis, NI.

Hemolytic diz of newborn

M8k 16; Mk 22; IM 1619; I2M 423; C4T 592; EM&S 1804;



Facts:

Mares Abs in colostrum lyses foals RBCs

CS: 24-36 hrs, icteric & anemia

Dx: Anemia, Tests

Tx: Rest, Fluids

1% of horses, but not all show CS

- Multiparous mares (previously sensitized) higher titers than 1st preg.

1st pregnancy possible if transplacental contamination of foals

- High incidence in A+ Arabians

Select donors A & Q negative

Variable onset of CS, depending on amount of colostrum & antigen type (types A & Q the worst)



. Foals normal at birth

 CS 24-36 hrs after suckle (colostrum)

Lethargic & weak

- Icteric, especially sclera

- Pale mucous membranes

- ★ HR, RR due to anemia

- Labored breathing (hypoxia)

- Progressively weak & recumbent

Often yawn (need O2), shiver

Afebrile

- Seizures w/ severe anemia

 Milder syndrome w/ anti-R or anti-S, more tolerant



- + RBCs (

- # Hemoglobin

+Total bilirubin up to 20 mg/dl (mainly unconjugated) due to RBC destruction (hypokalemic if not suckling)

Hemoglobinuria, hemoglobinemia

Lytic &/or agglutination tests

- Antibodies in colostrum or mare's serum against toal's RBCs - Coomb's test (direct antiquobulin test)

Detect antibodies attached to foal's R8Cs

Indirect antiglobulin tests Detect antibodies in mare's serum 8 attached to RBCs

Postmortem

Pale &/or icteric

Splenomegaly

Severe anemia lesions (centrolobular hepatic necrosis) Withholding milk questionable when Dx at 24 hrs (colostrum depleted & foal's absorption diminished)

Minimize stress

◆ Exercise (rest)

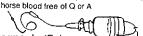
Generalized support

- IV fluids for diuresis of hemoglobin load on kidney, acid base balance IV dextrose

 Transfusions if severe anemia (PCV 10-15%) not if above 15%, just

Stop gap measure as cells don't last long

Also sensitizes foal to luture transfusions Mare's RBCs washed; transfused or other



Prognosis (Px)

 Varies depends on quantity of antibodies ingested, type of antibodies, severity of anemia, rapidity of CS

Peracute m/ die

 Others, support & transfusion if PCV continues to fall; may save



1. Mare negative for factor (Aa) considered at risk

Sensitization of mare to antigen (Aa+) & production of antibodies (anti-Aa)

- Exposure during previous pregnancy Blood transfusion

- Transplacental contamination of foal's RBCs 3. Foal w/ antigen Aa*

- Inherited from stallion Aa* 4. Ingestion of colostrum w/ Anti-Aa antibodies =

RBC hemolysis or agglutination

Antibodies (anti-Aa) attach to antigen (Aa+) on foal's RBCs Spleen (reticuloendothelial system) removal or lyzed (intravascular) by complement (agglutination or lysis)

Anemia if can't compensate

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Detect mares at risk: blood type for negative Aa & Qq

Breed negative Aa or Qq mares to negative Aa or Qq stallions

Problem: not many negative stallions & hard to find Tests to ID foals & mares

If unknown or incompatible mating

- Screen "at risk" mares 30 d before foaling for

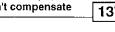
antigens to foal RBCs ACD anticoagulated sample to lab

Repeat closer to parturition if results equivocal

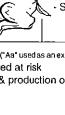
- If positive give alternate source of colostrum

. Muzzle loal & keep w/ mare

. 36-48 hrs allow to suckle from mare (colostrum gone & specialized epithelial of foal to absorb antibodies lost by 19-24 hr)



















Presentation/CS

CIRCULATORY SYSTEM

Condition

Purpura hemorrhagica

Mk 43; IM 1073; I2M 1203; C4T 293: EM&S 1836





 Arthrus Type 3 reaction, similar to serum sickness

- 1-3 wk after initial S. equi illness

Facts/Causes

- Excess antigens (presumably Strep. protein)
- Ag/Ab bind into small complexes (because of Ag excess)
- Small complexes remain soluble & are not filtered by reticuloendothelial system
- Complement taken up by Ag/ AB complexes
- Immune complexes cause vascular endothelial damage (vasculitis & thrombosis)
- Result in edema & purpura (purplish discoloration)

Normally antibodies in excess & form large complexes w/ antigens which are filtered by the reticuloendothelial system

Cause: sequel to another diz

- #1 Strep. equi infec. (strangles)
- Influenza

Ag/Ab/C complexes = vascular damage

Dx: Earlier Strangles or bacterin

Tx: 2X ABs, NSAIDS, Steroids

- Chronic suppurating wounds
- Vaccination w/ S. equi Bacterin

• Edema

- Urticarial wheals (elevated patches of skin) progressing to:
- Sharply demarcated edema of muzzle, eye, head, belly & limbs (hot, dependent)
- Purpura (petechiation, ecchymoses of mucous membranes usually more extensive than in EIA
- Muscle soreness due to swelling, don't move
- Pain associated w/ edema
- Stridor & dyspnea (due to edema around nares & pulmonary edema)
- Diarrhea or colic due to edema & hemorrhage of gut
- ± Fever, anorexia & depression, depending on whether animal still has S. equi
- Diz lasts 1-2 wk, 50% recover
- Death due to asphyxia, anemia or toxemia of 2° infection
- Chronically (wks later)
 - Weight loss
- Skin sloughing (edema compromises vasculature)
- ± Mild renal diz (due to immune complex deposition)
- Sequelae:

- Sloughing, laminitis, cellulitis, pneumonia & diarrhea common, prolong recovery

 Hx: recent strangles or bacterin · CS: Edema, purpura

Diagnosis

- Lab
- Mild to moderate anemia or hemoconcentration if plasma loss > BBC loss
- Compliment
- Normal platelet counts
- Normal body profiles, neutrophilia
- † gamma globulins & fibrinogen due to S. equi
- PM (posimortem)
- Marked edema: airways, intest.
- Hemorrhage: sparse or extensive
- Focal muscle ischemia (pale) or hemorrhage (dark)
- Deep abscesses or polygenic cellulitis E. eaui





- EVA (pg 134)
- Ehrlichia equi (pg 135

Tx to:

- Remove Aa
- 2. Reduce immune response

Treatment

- 3. Reduce vessel inflammation 4. Supportive care
- ABs to clear circulating bacterial protein
- 2 X dose of pen., streptomycin, tetracycline, oxyletracycline, Triple sulla any abscesses
- NSAIDs for inflammation & analgesia (Bute & Banamine®)
- Steroids for hypersensitivity vasculitis to reduce edema (controversial) (dexamelhasone, prednisolone) m/b 4 wi Cover w/ ABs because of 2° sepsis
- Tracheostomy if asphyxia eminent
- Blood transfusion if anemia severe
- Bandaging limbs
- Good nursing
- Lasix® to ↓ edema after well hydrated
- Some cases refractory to Tx, death from 2° septic processes
- Elevate feed & water to muzzle level will eat more readily





Px (prognosis):

 Good w/ early aggressive therapy Prevention: avoid exposure of previously sensitized horses to S. equi antigen

Red maple leaf toxicity, Heinz body hemolytic

CS: Edema & purpura

M8k 2116, 2122; IM 1098; I2M 1227; EM&S 1806; C4T 650, 278; C3T 375; C2T 679

anemia

- Oxidizing agents causing acute hemolytic anemia
- Phenothiazine (piperazine, [de wormer] or methylene blue)
- Plants: See Toxic plants: 325,
- Onions: wild or domestic
- . Brassica family (rape, kale)
- . Red maple; wilted leaves of (Ace. rubrum) (fresh leaves less toxic) 1º in NE US, seasonal severe in Fall when leaves falling off & wilting
- Specific substance not identified
- Heinz bodies formed by oxidative denaturing of hemoglobin in RBC
- Removed by spleen (reticuloen-dothelial system) RBCs w/ Heinz bodies less deformible, oxidized & broken down (ex-
- travascular hemolysis)
 Also change tonicity so some intravascular hemolysis
- Methemoglobin (chocolate color) Also produced w/ Heinz bodies
- by Red maple toxicity Oxidative change of hemo-
- globin iron to nonfunctional ferric state) Can't carry O2, resulting in
- brown color of peripheral blood & mucous membranes

- · Weakness, depression, anorexia
 - Exercise intolerance
- Icteric mucous membranes
- Brownish colored mucous membranesw/ Red maple toxicity (methemoglobinemia)
- No fever
- + HR & RR



Acute event - 2 syndromes

- Type 1
- Moderate to severe anemia w/ large # of Heinz bodies present
- Progressive † in MCV as animal responds to anemia present
- No methemoglobinemia or -uria
- Most will live
- PCV also
- Type 2: more severe
- Severe anemia
- Methemoglobinemia, -uria: brown discoloration
- Tend to die
- Few to no Heinz bodies, fell they have already been destroyed. Also have hemo-globinuria. Suggests more severe hemolytic crisis
- Sequela:

Hemoglobin nephrosis



- E1A (pg 138)
- Purpura hemorrhagica (pg 140)

Purpura: disorder characterized by

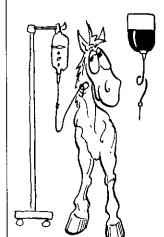
purplish discoloration of mucus mem-

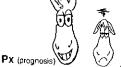
branes caused by hemorrhage

- Hx of exposure (drugs or leaves)
- Lab
- Anemia (varying degrees)
- Heinz bodies in all, round to oval to serrated refractile granules
- Located near cell margin or protruding Crystal violet or new methylene blue
- stains to unfixed blood smears No hematologic evidence of regenerative anemia in horse (because RBCs mature (not reticulocytes)
- when leave bone marrow normally) Hemoglobinemia & hemoglobinuria (profound RBC destruction)
- ↑ Serum bilirubin (indirect)
- Modest to marked
 ◆ BUN & creatinine (reflect risk of hemoglobin nephrosis)
- Methemoglobinemia w/ red maple toxicity



- Remove source of toxicity Supportive:
- Blood transfusion if PCV < 10 12%
- IV fluid if evidence of renal damage Cathartic to empty GI to rid toxins





Good if modest anemia

 Poor if renal damage (but can be saved)

Grave w/ methemoglobinemia

Oxidation = hemolysis CS: Anemia, icterus, tiring

Dx: CS, Hx, Heinz bodies, Tx: Support, Fluids





139

Condition

DIC, Disseminated intravascular coagulation

Mk 24; IM 1077; I2M 1207; EM&S 1838; E 404; C3T 504; C2T 306; M 332



to abnormal homeostasis) RBCs are damaged passing through damaged arterioles & removed by endoreticular system Causes:

(see box)

infarction)

cosa

cause colic

 Septic processes (salmonellosis, metritis)

Neoplasia

 Acute Gl disorders (strangufation, acute enteritis, protein losing enteropathy, embolic infarction)

Facts/Cause

Dizs causing vasculitis, activate

platelets & clotting mechanism

- Acute Gl disorders (strangulation, acute

enteritis, protein losing enteropathy, embolic

. Horse: prevalent in acute GI disorders that

Spectrum from diffuse thrombosis

- Ischemic organ failure to severe

hemorrhagic diathesis (predisposition

. Initiating factor for laminitis

. Endotoxins absorbed through damaged mu-

Never a 1° disease entity

Renal diz

· Hemolytic anemia

• EIA

Thrombotic purpura

· Strongyloides migration

Presentation/CS

Variable - depending on 1° diz

 Rarely overt hemorrhage Thrombosis & ischemic organ failure

Renal failure: common

- Oliguria, azotemia (↑ BUN)

Depression & ileus

Colic: due to GI microthrombosis

Occult fecal blood, rarely melena · Rarely respiratory (microvascular thrombosis) - Tachypnea, & hypoxia

CNS: microvascular thrombosis

Delirium, convulsions, coma

• ± Acute laminitis: digital ischemia

"Blown" jugulars: thrombosis of major veins

Catheters, & needles

· Petechial or ecchymotic hemorrhages (depletion of clotting factors)

Life threatening hemorrhage very rare Trauma or Sx m/ induce uncontrollable hemorrhage

 Chronic, compensated DIC - Few or no CS

M/ become clinically obvious DIC due to stress, concurrent diz

Diagnosis

 Systemic CS · Lab: no test definitive Dx

- Strongly suggestive

. Thrombocytopenia

. < 100,000/ml platelets suspect DIC Mild to moderate prolongation

of PT &/or APTT time Findings often NOT helpful

Occult blood in feces

Treatment

Treat 1° disorder

 Supportive Tx to combat shock & maintain tissue perfusion - IV fluids

ABs for septic conditions

Sx for strangulating intestinal

obstructions

Banamine® (flunixin meglumine) IV, q8h) for endoloxins

Corticosteroids not indicated. may worsen

Life threatening hemorrhage rare (fresh plasma, if platelet concentrates < 50,000/µl) Heparin - highly controversial



Septicemia

• Warfarin toxicosis (pg 143)

 Moldy sweet clover toxicosis (pg 143)

· Inherited coagulation abnormalities



Px (prognosis)

Poor if doesn't coagulate

Depends on underlying diz

DIC = "Death Is Coming" 2° to other dizs

CS: Multiple systems Dx: < 100,000 platelets

Tx: Tx 1° diz, support



Iron-defc anemia

IM 1103: I2M 1232: EM&S 1809; E 412

· Less common in horse than other species

• Due to chronic blood loss: parasitism, bleeding, GI lesions or hemostatic defects · Dietary deficiency seldom a cause even in neonates

•Tx: Correct chronic blood loss; Iron cacodylate only safe preparation for horses, avoid injectable iron dextran (for pigs) bec. can cause anaphylaxis

Moldy sweat clover toxicosis

Mk 1682, 1733; IM 1081; I2M 1884; PP/Mt 40; EM&S 1838; C3T 371; C2T 325; PP/O 18



(Warfarin,

coumarins)

Melilotus spp.

 Moidy sweet clover hay (especially small bails) or silage

 Dicoumerol synthesized from coumarin by molds

Winter on moldy hay, Palatable

- Grazing fields not dangerous

 #1 in cattle, all animals susceptible · Consumption over long time

Source

Tx of navicular diz in horses

Rodenticides

· Mechanism of action

Competitive inhibition of Vit. K

Vit. K needed for production of clotting factors (II, VII, IX & X)

- Thrombin formation depressed

Rapidly absorbed from GI

Potentiated by Vit. K deficiency in diet

Concurrent protein bound therapy (phenylbutazone & chloral hydrate)

thrombocytopenia **Epistaxis**

petechiae) in mucous membranes

Hemorrhagic diathesis (predisposi-

Swellings (shoulders, thighs, neck,

back & chest) - due to hematoma formation

Hemorrhage where body traumatized

(brisket, tuber coxae, carpi) hematoma

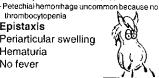
Ecchymoses (small hemorrhagic spot. >

tion to abnormal homeostasis)

Periarticular swelling

Hematuria

No fever



Prevention:

tion of hay & silage

Discard moldy portions

Limit access to rodenticides or plants

Limit other drugs (especially Bute)

Careful monitoring Warfarin Tx in horses

Hepatic diz strong contraindicated for Tx

Daily monitoring PT until dose doubles PT time
 Check PT 2 x wk during therapy

Exposure

Hemorrhages

Prolonged PT (lateralso APTT)

· No other abnormalities of clotting profile (normal platelet count, plasma fibrinogen)

Anemia

• Hypoproteinemia

No fever

Easily by careful forage preparation &/ or inspec

· Chemical analysis for dicoumerol - feed, blood & liver (absence doesn't rule out)

Remove source

Stop warfarin if PT exceeds 2x therapeutics value Tx

Remove plants

Vit. K1 (IM) every 6 hrs until PT normal Not K3 since poor action & nephrotoxic in horses

Fresh plasma (control hemorrhage by providing clotting factors)

 Whole blood transfusion (if life threat) ening anemia)



Px (prognosis):

Good if early Dx & prompt Vit. Kt

DDx:

• DIC (thrombocytopenia) (pg 142)

Mycotoxicosis

Septicemia (fever) (pg 35) Liver failure (pg 84)



Autoimmune hemolytic anemia, AIHA

M8k 14; Mk 21, 248; IM 1096; I2M 1225; E 408; C4T 279; C3T 495: C2T 295



Cats. horses & dogs

 Anti-erythrocyte antibodies against horse's own RBCs

Causes:

 1° idiopathic, or More commonly 2° to:

Purpura hemorrhagica

Antibodies complex w/ antigens on RBCs causing destruction & removal

- Lymphosarcoma, esp. GI form - Chronic bacterial infection

- Rarely procaine penicillin

- EIA, piroplasmosis of ehrlichiosis

Variable

 Typical of 1° problem + hemolytic anemia

Vit Kı

Hemolytic anemia CS

- Depression

- Pale mucous membranes

- Variable icterus

- + HR & RR

- Variable to intermittent fever

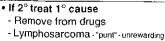
Anemia variable

PCV < 10% is life threatening - Horse only species that doesn't show regenerative hematological

signs · Direct Coomb's test for

antibodies Response to corticosteroid Tx





Steroids contraindicated in viremia & EtA so get neg. Coggin's test 1st 1° Autoimmune hemolytic anemia

- Systemic steroids if neg. Coggin's

. Dexamethasone (parenterally 3-5 d. gradually decrease over 7-14 d If no response in 5-7 d reevaluate cause

of bone marrow suppression Once controlled: oral prednisolone

Quite environment

Nutrition (Vit supplements) Blood translusion of little value











Condition

Ehrlichiosis

Mk 375; JM 1073, 1115; J2M 1204; EM&S 1806, 1837; C4T 282; C2T



Necrotizing vasculitis

Causes:

• 2° to:

· 1° (idiopathic)

Infections

- Neoplasia

Rickettsia - Tick - N. Calif. CS: Undulating fever & edema Dx: Inclusion bodies - PMNs Tx: Oxytetracycline

Platelet count < 100,000

Rule out (R/O) DIC right away

- Drug administration (penicil-

digoxin, sulfas & erythromycin)

Immunologic disorders

(EIA, lymphosarcoma & auto-

immune hemolytic anemia)

lin, heparin, quinidine, thiazides,

Facts/Cause

• Rickettsia Ehrlichia equi

Necrotizing vasculitis in

horses, donkeys & mules

Seasonal in N. Calif

Severity age related

Transmission: tick suspected

- Late fall, winter & spring ;

Presentation/CS

< 1 yr m/ only have fever</p>

 1-3 yr old: fever, depression, mild limb edema & ataxia

Horses > 3 vr

- Undulating fever (102-106°F for 6-12 d)

Depressed, partial anorexia Mucosal petechiation

Icterus

Reluctant to move

- Edema of all legs (more in hind

Progressively worse in 1st 3-7 d w/gradual resolution in 2-3 wk if untreated

Vary w/ underling diz process

Petechial hemorrhages w/ or

w/o ecchymotic hemorrhage

Oral, nasal &/or vaginal mucosa

- Epistaxis, melena, hyphema

- Spontaneous hemorrhage unusual

Prolonged bleeding from

wounds or injection sites

Hematoma formation

w/ trauma (< 40,000 ul)

unless < 10 000/ut

· Hemorrhagic diathesis

Diagnosis

- Hx. CS
- Mild to moderate leukopenia

- Mild to moderate anemia

- Inclusion bodies in neutrophils w/in 3-4 ds of CS

. Giemsa stain: blue-gray

DDx:

• Lab

- EIA (pg 138)
- Purpura hemorrhagica (pg 140)
- Chronic liver diz (pg 84)
- Viral encephalitis (pg 256)
- Viral arteritis (pg 134)

• Hx, CS

- Lab
- Prolonged bleeding time & abnormal clot retraction
- No effect on clotting time or plasma fibrinogen
- Response to Tx supports Dx

Treatment

- Oxytetracycline, 7-14 d (prompt remission of fever & CS w/in 1 d)
- Recovery usually w/in 10 d
- Penicillin, chloramphenicol & streptomycin have no inhibitory effect
- Corticosteroids m/b for severe edema & ataxia

Oxytetracycline

Px (prognosis)

- Good
- Recovery gives immunity for 2 y & no carrier
- Unexplained case
- Stop all other drugs, if Rx 🖑 necessary replace w/ most-dissimilar Rx
- Dexamethasone (IV or IM) 10-15 d Usually platelets ★ in 4-7 d
- Prednisolone (IM q12h) for cases refractory to Dex
- Prognosis: Good



Steroids

Other causes of thrombocytopenia

♣ Platelet production

Myelogenous neoplasia (granulocytic leukemia, myelomonocytic leukemia, monocytic leukemia, eosinophilic myeloproliferative disorders Phenylbutazone loxicity, rarely (pg 27)

Hypoplastic anemia

2. Abnormal sequestration (spieen)

3. Shortened platelet life span #1 cause (destruction or consumption)

- DIC (pg 140), Vasculitis

Immunemediated thrombocytopenia, **IMTP**

M8k 44: Mk 56, 428: IM 1075: I2M 1205; E 403; CT 507; C2T 310; M



Platelets < 100,000 CS: Bleeding

Dx: Hx, CS, Lab, Tx Tx: Stop Rx, Steroids

Anemia of

chronic diz

I2M 1233; C4T 283; EM&S 1808

- · #1 anemia of horses Mild to moderate nonregenerative anemia
- Causes
- Neopiasia
- Chronic inflammatory or infectious process (pleuritis & internal abdominal abscessation)

Most mild

- Fever (105°F)

(pinkeye)

- Cough

- Disturbance of iron metabolism





Hemolytic syndrome

· Near terminal event in acute or chronic liver failure, Sudden & rapidly progressive

· Vary from subclinical to severe

- Poor appetite, Stiff hindlimbs

reddening of 3rd eyelid, push forward

- Conjunctivitis & rhinitis -

· Very sick, severe depression

- Limb edema (esp. hind limbs)

- Ventral edema of eyelids, ventral

abdomen (prepuce, scrotum, mammae)

Intense icterus sclera & conjunctiva (reddish orange), hemoglobinemia & hemoglobinuria, "burr cells" (RBCs)

• Px highly unfavorable - nearly all die

related to liver failure IM 1102; I2M 1231, 1233; E 410

Equine viral

arteritis, EVA, Equine typhoid, Epizootic cellulitis, Pinkeye

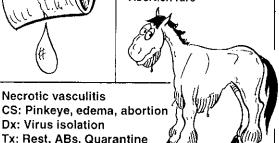
Mk 376; IM 527, 1401,1397; I2M 1204; EM&S 1836; E 734; C4T 294: C2T 313



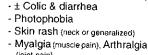
Prevention: vaccine

Togavirus

- small arteries
- Inhalation (aerosol), or
- Venereal (semen, vaginal discharge) Feces
- Carrier stallion, not mares
- Abortion rare



- · Diz not commonly diagnosed
- Damages smaller arteries - Panvasculitis of entire cardiovascular system
- Edema: leakage due to necrosis of
- Transmission



- Icterus
- Papules on upper muzzle
- Abortion (see pg 216)

- CS to provisionally Dx
- · Virus isolation, serology, histopathology
 - Nasopharyngeal & conjunctival swabs, citrated blood samples & semen
- Serology difficult to interpret; asymptomatic animals can have titers
- · Lab: only leukopenia

Abortion: (pg 216)

 Abortion during or following illness



DDx:

- EHV-1 (abortion mos later, PM - inclusion bodies)
- Eq. influenza (abortion rare) (pg 109)
- Purpura hemorrhagica (pg 140)

- No specific Tx
- Antibiotics & symptomatic therapy

Tx underlying cause & anemia

Good nursing & absolute rest



Reportable in some states



Prevention: 🖳 MLV vaccine, closely regulated,

- Know current USDA restrictions in your area Must be seronegative to vaccinate
- Given annually
- Economics, if no problem w/ EVA, don't vaccinate
- Seropositive stallions bred only to seropositive or vaccinated mares
- Don't ship mares to positive stallions
- Semen culture all positive stallions for shedding

CIRCULATORY SYSTEM Anemia Facts/Cause Presentation/CS Condition Fever 102-107° F • Hx (area) CS: fever, anemia, • Babesiacidal Rx Babesiosis. · S. USA, endemic in SE Florida Malaise & anorexia jaundice & hemoglobinuria - Bernil® (diminazene diaceturate) . Not a big problem in USA Piroplasmosis, Hemolytic anemia, rapid Tick infested area Fluids . Babesia equi (most pathogenic) · Hemoglobinuria, dark yellow-Tick fever, - Hemoglobinuria & death • Organisms in Geimsa stained · Imidocarb® (protozoocide) elimi- B. caballi (persistent fever & anemia) colored urine RBCs of thin blood smear nates carrier state Texas fever, • Tick, Dermacenter nitens in USA Death PM (postmortem) Red water - B. equi different from other Babesia, has Depression, anorexia, incoordi-- Jaundice M8k 23; Mk 71; IM 1088; I2M 1217; an excerythrocytic (lymphocytic) stage nation, lacrimation, nasal dis-- Enlarged spleen C4T 281; EM&S 1806 · Chronic carriers once infected charge, swelling of eyelids, frequent lying down Px (prognosis) Recovery rule if Tx'd early No vaccine Tick life cycle - 3 weeks DDx: Female ingests parasites in a blood meal, these are passed · Ehrlichiosis (pg 142) transovarially to larval progeny. The tick drops off the animal & lays eggs. The eggs develop into larvae which attach to a newhost Tick, Dermacenter, protozoa EIA (pg 134) Controlling endemic areas: CS: Fever, anemia & complete their life cycle. • Liver failure (pg 186) Parasites in tick saliva enter bloodstream to RBCs, merozoites break out of RBCs to infect others Control ticks Hemolytic anemias Dx: Hx, CS, Organism - Range fires, reduction of tick pop. Animals infected on new pastures w/ infected tick larva (pg 142, 143) Tx: Bernil® Anaplasmosis M8k 21, 12M 1214: in ruminants not in horses CS of depression, recurrent fever, No effective Tx Depression Myeloproliferative Abnormal proliferation of Intermittent fever petechial hemorrhages bone marrow elements Petechial & ecchymotic diz None effective allure of normal hemorrhages Thrombocytopenia Anemia BBC Neutropenia Nonregenerative anemia Thrombocyles (platelet) Weight loss, lymphadenopathy I2M 1244: WBC production Abnormal cells in circulation C4T 295, 283 splenomegaly, edema Poor performance Neoplastic infiltrate of bone marrow Often young < 5 yr-old Prognosis: Poor Bone marrow - Fever, Hemorrhage, Anemia (WBCs, Platelets, RBCs) • External: Hemorrhage, . Hx, CS of hemorrhage Blood in external hemorrhage Stop hemorrhage Trauma (laceration, splenic rupture, Occult in internal bleeding - External - suture or pressure bandages - Internal (m/ not attempt if animal is poor - Anemia Blood loss anemia, reticulopericarditis) - Hypoproteinemia Acute massive loss = - Surgery (castration) Hypovolemic surgical risk because cause often not found) hypovolemic shock - Abdomínocentesis & - Erosion of carotid artery by Tx hypovolemic shock (see box) shock - Tachycardia thoracocentesis guttural pouch mycosis PCV < 12% consider whole blood Mk 26; IM 1068; I2M 1198; E 402; - Tachypnea Bone marrow responds in 5 d transfusions (see box) Internal: EM&S 1807; C4T 276, 290; C3T Peripheral signs or regenera- Cold extremities - Rupture of middle uterine a. 492, 517; C2T 300; Pop 71-7/98 - Muscle weakness tion in horses minimum (no - Rupture of aneurysm of crabasophilic stippling as in cow) - Cardiovascular collapse nial mesenteric artery Lab: - Renal damage Splenic rupture Acutely PCV & TP normal Anemia (declines in 24 hr due to mobilization of No icterus extracellular fluid to maintain volume) RBC (polychromasia, basophilic slippling, Howell-Jolly bodies & nucleated RBC w/in 4 d) Tx hypovolemic shock (\$) C4T 290; C3T 517 - IV 40-80 ml/kg sodium containing crystalloid solution (even though dilutes PCV) - Give more than blood loss because distributes to intracellular space Whole blood transfusion (\$ expensive & rarely done) - Only a temporary measure because RBCs survive only 4 d 1st transfusion usually tolerated 2nd not, due to trillions of blood types & incompatibility - Replace 20-40% of blood loss to allow bone marrow time to respond Monitor for adverse reaction (20 ml/kg/hr) (tachypnea tachycardia, dyspnea, muscle fasciculations or sudden collapse: slow down **External & internal** infusion or disconlinue steroids or epinephrine if anaphylaxis [.2 to .5 mls IV (1: 1,000 dilution)]) CS: Hypovolemic shock, anemia, no icterus Dx: Hx, CS, Lab Tx: Stop bleeding, Tx shock

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URINARY SYSTEM

Acute renal failure (ARF)	148	Oliguria - DĐx	343
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Acute Renal Failure (ARF)

Tubular necrosis (most)

common cause of ARF) - due

to 2 broad categories - Ischemia (vasomotor

nephropathy)

Facts/Cause Presentation/CS

. CS fairly nonspecific • CS of 1° diz (i.e., diarrhea,

septic shock, myositis, colic)

• ± Abdominal pain, mild colic

Oliguric (diminished urine secre-

tion; anuria very uncommon in large

- Hemolysis (may also be result

Complications to RF

Depression & anorexia

nonspecific

Dehydration

animals)

- Diarrhea

- Laminitis

of renal failure)

Endotoxemia

- History, CS
 - · Rectal palpation: enlarged left kidney (can't reach right)
- 4 Creatinine azotemia
- Hyponatremia, hypochloremia
- Low/normal K* due to restricted oral intake if polyuria; slightly ∱ if oliguric Often hypercalcemia

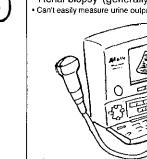
Diagnosis

- Low specific gravity (< 1.020) (isosthenuria)
- Microscopic hematuria
- Urinary casts, granular or leukocylic, pigment casts w/ RBCs,
- or hyaline (noncellular due to mucoprotein in urine) Ultrasound (US): enlarged or abnormally-shaped kidneys, w/ abnormal consistency to renal parenchyma
- Contrast studies only in small ponies & foals PM (postmortem)
- Maybe normal on gross exam, heavy, cut surface bulges
- Perirenal edema
- Histopathology
- . Tubular necrosis
- . Casts in tubules

Interstitial edema, interstitial cellular infiltration, vascular thrombosis

Renal biopsy (generally not done due to risk of hemorrhage)

Can't easily measure urine output. GFR





URINARY SYSTEM





Condition

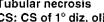
Acute renal

M8k 1136; IM 872; I2M 953;

failure,

ARF

Tubular necrosis CS: CS of 1° diz, oliguria



Dx: Lab, Creatinine, US, PM Tx 1° - Fluids





- Aminoglycosides (pg 157) - One of the most common causes of tubular nephrosis
- NSAIDs (nonsteroidal anti-inflammatory drugs) (pg 157)
- · Heavy metals (pg 156) · Rare

Causes of ARF

- Hg (mercury), Arsenic, Cadmium, Lead
- Oxalate-containing plants (pg 156)
- Pigweed, sorrel, dock, rhubarb
- Chlorinated hydrocarbons (See TOX pg 312)
- Pigment nephropathy myositis
- Nephrosis associated w/ large amounts of myoglobin or hemoglobin
- , Myoglobin myositis, severe tying up episode, or several milder episodes . Hemolysis less likely - DIC
- Vasomotor nephropathy
- Any condition predisposing to hypotension &/or release of endogenous pressor agents
- . Acute blood loss, marked hemoconcentration, dehydration, septic shock, thromboembolic episodes, severe intravascular volume deficits
- Tubular necrosis (predominant lesion)
- Acute glomerulopathy (rare)
- Acute interstitial nephritis (sporadic)

Treatment of ARF

- Tx predisposing cause
- Initial
- Fluids: correct volume deficiencies, electrolyte & acid-base abnormalities
- . Monitor serum Na, CI, K, & HCO3 IONS daily & correct
- . Na & CI IONS low in polyuric ARF
- . K usually normal or low in ARF so don't need to lower it
- HCO3 often normal in ARF NaHCO3 to correct metabolic acidosis by BW kg x .03 x base deficit mEq HCO3
- Determine if oliguric or polyuric after volume & electrolyte correction
 - If oliguric monitor volume changes by:
 - Daily body weight & PCV/TP
 - . Central venous pressure (manometer in jugular vein)
 - . Fluid needed = % dehydration x body weight
- Unless oliguric or anuric replace fluids over 4-6 hrs
- If oliguric slowly & w/ diuretic therapy
- Oliguric diuretic therapy to † urine production († renal bloodflow, † GFR) after fluid replacement therapy, done early, after few days doesn't work well, nor if tremendous number of casts from kidney
 - Mannitol (20% IV) (expensive)
- Lasix® (furosemide) (1 mg/kg/2 hr)
- Dopamine in 5% dextrose solution w/ furosemide in anuric or severe oliquric ARF
- Must intensely monitor to insure that blood pressure doesn't † during infusion (cardiac
- Normal urine flow (nonoliguric) more likely than oliguria after initial fluid therapy
- 40-80 ml/kg/day until marked

 in serum creatinine, then
- 10-20 ml/kg/d until creatinine normal & horse is eating & drinking
- Check creatinine 2-4 d after fluid therapy is stopped
- Hypertension after diuretic therapy acepromazine (.02 mg/kg)
- Avoid prostaglandin inhibitors (being used if diarrhea & endotoxemia), can use small amount of Banamine®
- Xylazine IV for pain if hydration & blood pressure normal
- · Peritoneal dialysis used in foals, especially beneficial if ruptured bladder or nephrotoxic renal failure, helping to bring electrolytes back to normal



- Good if cause removed & oliguria resolved (& no 2° complications)
- Reverse hemodynamic causes & renal failure will resolve
- Renal failure > 1 d or perirenal edema or kidney enlargement, Px goes down
- · Px monitored daily by checking creatinine



or uveitis

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IM 371: I2M 972, 328, 757.

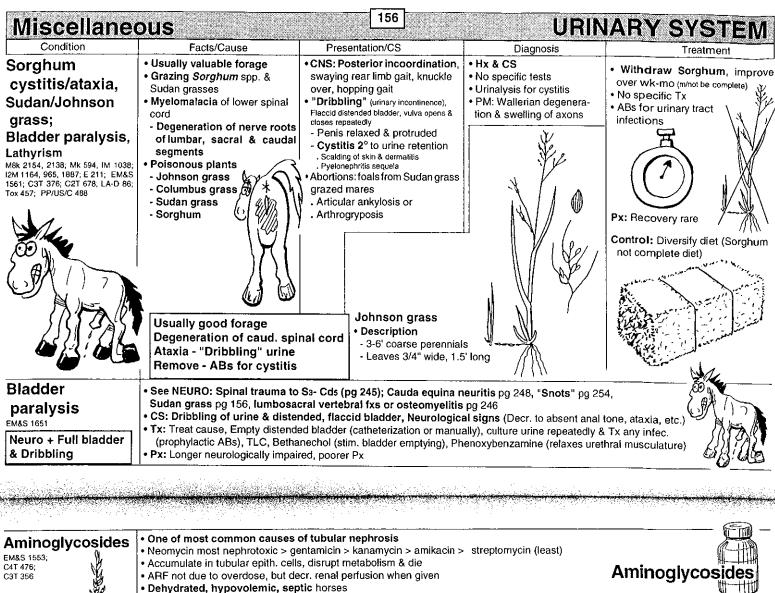
C4T 602; M 288

bladder)

155

Associated w/ cystitis

Bethanechol (parasympathomimetic)



Oxalate



• See TOX pg 328; Halogeton, Greasewood, 1º cattle, Rare - horse, West, Insoluble pcpt. w/ Ca, Vascular necrosis & renal tubular blockage

CS: Colic, Weakness, Frequent urination, Crystals in urine

Tx: Hopeless once CS; Fluids, Ca gluconate

Ethylene Glycol, Antifreeze

• See TOX pg 317; Rare in horse Sweet tasting alcohol, form insoluble Ca oxalate in renal tubules

CS: Hind limb ataxia, salivation, depressed sensorium, nystagmus, seizures,
 Dx: Azotemia,
 creatine, oxalate crystals in kidney

• Tx: Early: 20% ethanol (50 ml/hr), activated charcoal, NaHCO3 IV, Replace fluids



NSAIDs Phenylbutazone Toxicity



• See GI, pg 27; NSAIDs, widely used & misused, overdose; damage to GI mucosa, kidney (acute tubular necrosis from abnormal amounts of drugs, more commonly subacute or chronic interstitial nephritis from prolonged drug use); No problems if proper dosage & hydrated animal, Not a common diz process

CS: GI & kidney, laminitis sequela, renal damage (azotemia)

Tx: Stop NSAIDs, symptomatic • Px: Guarded in severe cases

Blister beetle toxicity

• See GI pg 45; Horses >>> sheep & cattle, Cantharidin. Swarm in alfalfa, Southwest

• CS: Endotoxic shock = death 2 d, renal failure & ulceration of GI mucosa, colic

• Tx: No antidote, supportive • Px: guarded if CS

Heavy metals



• See TOX pg 313; Rare, Hg (mercury), Arsenic, Cadmium, Lead

· CS: GI signs (incr. salivation, oral erosions, colic, hemorrhagic diarrhea); CS of uremia (depression, seizures, oliguria)

Dx: Tubular necrosis, azotemia, isosthenuria, enzymuria, & electrolyte imbalances

Tx: Dimercaprol, 1 lb of activated charcoal orally, ARF Tx

Phosphorus

• See TOX pg 315; Garlic odor, Corrosive & hepatotoxic, Protoplasmic poison

• CS: Biphasic, GI irritation; Latent phase; 2nd phase, Depression, Hepatic & renal failure - death, oliguria, icterus & bleeding tendencies

• Dx: Biphasic CS, † liver enzymes, † BUN; PM: enlarged liver, icterus, GI irritation, hemorrhage & necrosis; Histo: liver & renal lesions Tx: Emergency, Symptomatic Tx, Mineral oil, activated charcoal, No fat in diet for 3-4 d (absorps phosphorus) • Px: Grave

Dogbane, Indian hemp See TOX pg 327; Resin containing plant

CS: Abnormalities of urinary system, Crystals in urine

Tx: Fluids to dilute, Diuresis







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Breeding soundness exam (BSE)

C4T 505; T654; R623; R-Y179; EM&S 949; ER196; E1305, EM&S 949;

Be very thorough & detailed

- · Keep thorough records
- Society for Theriogenology has standardized forms
- When BSE performed
- If doesn't settle in 3 cycles
- Routinely required of barren mares at end of breeding season
- Before each breeding season
- Before buying a broodmare or maiden mare
- Often asked for after purchase when problem is suspected

General history, m/not be a lot available, especially if young

- · Helps direct you toward physical exam
- . Age is important
- ->15 yrs, fertility falls off, esp. if producing foals on yearly basis
- Older maiden harder to breed than young maiden
- · General health of horse Bad teeth? Parasitism? Chronic laminitis, etc. not good candidate for carrying foal
- Medical history:
- Abdominal surgery, uninsurable bias against mare, especially if bowel has been opened, adhesions & good chance it will colic again, w/ growing fetus, adhesions cause discomfort, lay down & roll, recolic, etc.
- Palpate midline for scars (steel staples)
- Worming & vaccination history
- Drug therapy, especially steroids
- Infectious diseases:
- CEM (contag. eq. metritis) or eq. viral arteritis (EVA)
- . Can't take horse on farm quarantined
- Mare's purpose (show, racing)

- Training &/or anabolic steroids
- Time required for body to shift gears into being a brood mare if
- If bought in Jan., will not necessarily be cycling
- Flushing effect: change from poor rations to high quality increases chance of getting pregnant
- Overweight problem, may reflect hypothyroid condition, get weight off & if necessary, measure T3/T4 w/ a TSH stimulation test Supplement w/ feed additive
- Herd problems viral arteritis, etc., management

Brief stallion history

- . Number of mares bred
- . Number conceived & foaled
- · Historical evidence of mares infected by stallion
- · Number settled per cycle

Breeding history

- · Last year's breeding record if possible
- · Last foaling date: number & dates of foalings
- · Age at 1st heat
- · Heat & breeding dates
- Number of live offspring Abnormal foalings
- Abnormal pregnancies
- Hx of early embryonic death (EED) or abortions & possible causes
- · Intervals between heats
- · Previous year's breeding cycle pattern
- Number of breedings required for last conception
- · Method to tease & breed mare (Al, pasture or natural hand
- Barren or not
- Length of time barren

- History of services (All or natural to what stallion)
- Prior possible exposure to venereal diz (CEM or infected stallion)
- Hx of uterine infections & treatment
- · Endometrial biopsy report
- · Genetic or hereditary background of family infertility

Physical exam:

- Physical condition
- Excessively heavy or thin
- Hirsutism (abnormal hairiness). long winter hair coat normally lost at time of estrus, be suspicious of pituitary disorder causing anestrus
- · Some breed 2 year-olds too early
- Presence of disease
- Body systems: teeth, eyes, locomotor, circulatory, respiratory & nervous systems
- Laminitis (rotated P3, reluctant to stand for breeding)
- Hypertrophic pulmonary osteopathy assoc. w/ ovarian tumors

Visual exam of genital organs

- Vulvar discharge
- Check for predisposition to pneumovagina or need for Caslick's operation
- Apposition & tone of vulva
- Vulvar conformation (normal: vertical; poor: dorsocranisi w/ anus cranial to vulva)
- > 70% of vulvar openings below the pelvic floor
- Gently pull vulva apart & listen for aspiration of air
- Vulvar or perineal scars or tumors
- Mammary glands for mastitis, abscessation, neoplasia or injury

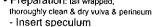
Rectal exam - carefully

- · Preparation: tail wrapped & tied out of way, use lots of lubrication
- " Calibrate one's hand": so can use as measurements while palpating (e.g. 1st joint of index finger 1")
- Uterus 1st
- Palpate for pregnancy or twin conceptus
- . Bulging & thinning of uterine horn near bifurcation (vesicle) for
- if pregnant discontinue palpation; if nonpregnant continue · Uterine horns for size, shape, tone, thickness, width
- Abnormalities or irregularities (adhesions, large endometrial cysls, uterine dilations, edema, atrophy, excessive small size, mucometra or ovometra)
- · Cervix: width, length & tone for where in cycle
- · Uterine tubes: seldom palpated in mare
- Ovaries
- Small & firm (< 35 mm/1.5") in anestrus, doubles in estrus
- Presence & size of follicles (follicle > 35 mm/1.5" ovulation soon)
- Ovulation fossa
- Abnormalities (parovarian cysts, adhesions, tumors, hema-Iomas, varicosities, etc.)
- Bony pelvis, palpated for abnormalities that m/ interfere w/ parturition (knocked down hip, asymmetrical, palpate pelvic canal for

Ultrasound exam (per rectum)

- · Must be a routine part of an equine repro exam
- Uterus
- Twins
- Pathology: endometritis, cysts, abscess, tumors
- Pregnancy, vesicle can detect earlier than 15 days of gestation
- Ovaries
- Follicles > 1.5" (35 mm) imminent ovulation, depending on the time of year





- . Transverse fold of hymen at vaginovestibular junction m/ cause resistance, expand w/ constant pressure of speculum
- . 1st cervix for color & tone (before air affects it)
 - . Exudate
- Vaginal wall for inflammation or congestion, lacerations, scars, stenosis or tumors, varicosities
- . Pale, pink, red or inflamed; moist or dry & charac-
- Withdraw speculum & evaluate vestibular area

Culture: (pg 172) if not bred recently, uterine culture before contaminating by palpation per vaginum

- Should be taken
- Especially if uterine discharge observed
- Protected swab (sterile sleeve or speculum)
- Uterine culture is superior to cervical culture . Better results during estrus
- Culture urethra & clitoral fossa & sinuses (for Klebsiella & CEM)
- Repeated cultures often needed

Cytologic exam & sensitivity

- Absolutely necessary
- Smears from uterine culture swab or flush'
- Presence of PMNs indicates infection

Manual palpation per vagina

Digitally palpate cervix opening

- Indicates where in cycle by how many fingers can - Cervical competency, adhesions, scarring or stenosis
- Fiberoptic or endoscopic exam Cervix & uterus: adhesions, cysts, @o tumors, fluid accum., fibrosis, etc.

Uterine biopsy (pg 174)

- Most valuable tool for predicting ability to carry to full term (especially in older mares), cost justified
- Doesn't tell if mare can get pregnant
- Valuable indication of present & past diz conditions
- Amount of scar tissue present determines ability to carry to term
- PMNs characteristic of active acute inflammation
- Experienced pathologist to interpret, takes time
- Kenney categories 1-3 see pg 174

Endocrine assays

- Progestins for persistent CL associated w/ prolonged anestrus
- ECG (equine chorionic gonadotropin [formerly PMSG]) is secreted by endometrial cups which are formed at 35 d of gestation (poor means to differentiate EED [early
- embryonic death) (hi ECG) from failure to conceive) Testosterone, Progestin, Inhibition for GCT

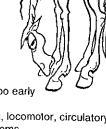
Chromosome analysis or cytogenetic study (ER 258): Some infertile maiden mares

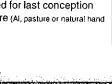
Evaluate managerial factors & competence of breeding farm personnel

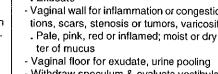
> Note: Not all of the above will be used for every mare, it depends on reason for exam, stage of cycle, etc.











Repeat Breeder

REPRODUCTIVE SYSTEM

Anestrus

IM 242; I2M 1523, 247; E 1321; T 663,

· Anestrus: lack of estrus

· CS, not a diz

- M/b normal physiological phenomenon, a sign of diz, or indicate poor heat detection

Pregnancy #1 cause of anestrus - rule it out 1st

Causes - Anestrus

· Normal physiologic phenomena

Pregnancy

- Seasonal anestrus/Transitional - fall & winter (mare long day breeder)

Inadequate heat detection

Psycological problems

Cycling, but not showing estrus

. Nervous mares

. Mares w/ foals

Nutrition - poor

- Weight loss

- Fat mares

Chronic debilitating diz

Congential anomalies Gonadal dysgenesis (uncommon)

Persistent corpora lutea

Spontaneous

Fetal death after endometrial cups start secreting ECG

1-4 mo of anestrus during breeding season

Uterine pathology

- Pyometra if prevents PGF release

Pituitary tumor

Ovarian tumor

· Fescue toxicity (fungus - Acremonium coenophia)



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Diagnosis:

• Reproductive & general Hx

Thorough breeding soundness exam (BSE)

Evaluate estrus detection program

• Rule out (R/O) pregnancy 1st

Animal side milk or serum progesterone test kits





Causes of Anestrus IM 243, 1367; 12M 246, 1525

Pregnancy

Seasonal (pg 164)

• Inadequate heat detection/ Unobserved estrus (pg 169)

• Transitional (pg 164)

• Prolonged estrus ("split estrus") (pg 165)

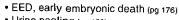
• Persistent corpus luteum (pg 166)

· Silent heat (pg 166)

Nymphomania (pg 168)

Nutritional infertility (pg 168)

Twinning (pg 176)



Urine pooling (pg 186)

Windsucker (pg 177)

Granulosa theca cell tumor (pg 188)

Ovarian hypoplasia/dysgenesis (pg 190)

Lymphangiectasis of uterus (pg 191)

Chronic diz

• Pituitary tumors (p 298)

Weight loss

Fescue toxicity



Repeat breeder

IM 244; I2M 249;



Definition: mare bred during 3 or more successive periods w/o becoming pregnant

 Management of repeat breeders is frustrating & expensive - If expensive mare, still economically sound to breed

Pathogenesis:

- FF: failure of fertilization - either male or female

. Multiple females affected check stallion Individual female affected check female

- EED: early embryonic death

5-8% lose pregnancy in first 4 mos. of gestation

DDx FF from EED

• FF no affected on interestrus interval

 EED prolongs interestrus interval (intermediate length to multiples of normal cycle lengths); after maternal recognition of pregnancy at 14-16 d

Diagnosis:

Evaluate heat detection

· Evaluate breeding technique

· If herd problem: evaluate stallion

- Physical condition

- Semen quality

- Libido & ability to mount

- CEM, if other factors ruled out (R/O)

- Al (artificial insemination) technique

. Semen quality

Evaluate thawing, transportation, timing & deposition techniques

Errors in heat detection & timing of breeding

. Question & observe

. Milk or serum progesterone (detect heat)

· Individual problems: check mare

- Body condition - poor nutrition

- Reproductive exam

. Conformation of vulva - pneumovagina & endometritis

Urine pooling - FF (spermicidal), EED

Repeat breeders = FF or EED



Common causes

- Transitional season (pg 164)

- Windsucker (pg 177)

- Endometritis/fibrosis (pg 183)

- Twins (pg 176)

Poor Al timing

Poor heat detection (pg 169)

EED (early embryonic death) (pg 176) - Uterine lymphagectasis (pg 191)

· Less common causes

- Urine pooling (pg 186)

- Rectovaginal fisula (pg 236)

- Malnutrition (pg 168)

- Metritis (pg 183)

- Pyometra (pg 183)

- Heat stress Poor semen quality

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Uncommon causes

- CEM (contagious eq. metritis) (pg179)

Gonadal dysgenesis (pg 190) Neoplasia, uterine or cervical (pg 190)

Ovarian neoplasia (pg 188)

Paraovarian cysl (pg 190)
 Oophoritis

- Salpingitis (pg 190)

lodine delc

- Phosphorus defc Zearalenone toxicity

- Intersexuality

(open cervix, cervicitis)

.. Cervical canal occlusion - FF

.. Postpartum metritis - pus & debris

FF, failure of fertilization

.. Endometritis & minimum pus - EED, not FF

Uterinm culture (pg 172)

- Cytological smear

- Endometrial biopsy (pg 174)

- Embryo flushing to DDx FF from

. Collecting degenerating embryo - EED

. Failure to collect either doesn't indicate oviduct blockage as in cow

- Ultrasound detection of pregnancy

. Loss of embryo after detection w/ ultrasound confirms EED

- Hormonal assays

. ECG (equine chorionic gonadotropin, formerly pregnant mares serum gonadotropin

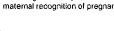
.. Detects if pregnant for 40 ds at least (endometrial cups produce ECG after 40 d of gestation to 120-150 d, even if

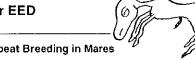
felus dies) Can confirm EED, not FF

. Progesterone levels can't confirm pregnancy, but low levels early in expected pregnancy can confirm nonpregnant (< 1 ng/ml)

Prognosis: often treatment unrewarding







164 REPRODUCTIVE SYSTEM Estrus - Infertility Condition Facts/Cause Presentation/CS Diagnosis Treatment · History: Winter season Seasonal · Normal Fall-Winter anestrus Failure to respond to Spontaneously resolve w/ • Normal in temperate latitudes · No response to stallion teasing stallion teasing day length anestrus Anestrus also normal during - No signs of estrus · Rectal palpation IM 1366; I2M 1523; R 591; - Uterus thin-walled & flaccid pregnancy R-Y 71; ER 145; E 1321; - Ovaries small, firm, inactive C4T 525; C3T 635 Speculum: DDx: - Vagina dry, pale w/o much Pregnancy MUCUS (difficult to insert glass speculum) Pyometra (pg 183) - Cervix flaccid, usually tight (but m/b Prolonged diestrus partially or fully dilated Prognosis: Excellent Undernutrition Rule out (R/O) causes of anestrus Gonadal dysgenesis (pg 190) Normal - Winter & pregnancy Dx: Hx, Rectal Artificially Iength of light Tx: Resolves w/ 1 light or time brings on seasonal estrus sooner Regular teasing & examining genital At beginning & end of Only consistency of estrus Transitional Time of year: early Spring seasonal anestrus (transition beis mare's inconsistency organs & breeding will get some, but Rectal palpation period, tween anestrus & estrus or estrus & anestrus) - Anestrus or prolonged estrus Ovarian changes most must wait until physiological Vernal transition Late Fall & Spring - Erratic follicular growth breeding time arrives ↑ in size of ovaries days before Inconsistency of mare's cycle is - Ovulation or anovulation Goal: hasten regular physiological 12M 1523; R 592 R-Y 71, 167; follicles can be palpated concern to vets & owners - Other cyclic & reproductive breeding season . Usually associated w/ loss of Prolonged estrus included (see 1. Keep mares in good health irregularities winter coat Not overly fat Good nutrition to cause gradual pg 65 below) From firm to rubbery weight gain m/ hasten estrus 2. Regular frequent teasing w/ stallion

Consistently inconsistent mares **CS: Anestrus**

Dx: Hx & Palpation Tx: Artificial light



3. Artificial light - gradually # light to 15-16 hr/d to initiate ovarian activity, most satisfactory means of hastening breeding season

inconsistent results (see box)

Prolonged estrus, "Split" estrus, Seasonal prolonged estrus

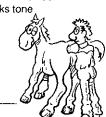
IM 1363; R 593; R-Y 71, 169; C3T 634; M 257



- Type of transitional period problem
- Prolonged periods of estrus
- At end of transitional period or start of spring breeding season
- Natural & common (up to 90% of maidens & barren mares)
- 8-30 days of estrus (average 18 d) may be > 30 days
- Physiology:
- Graafian follicles develop & secrete enough estrogen to cause CS of estrus
- Estrogen low until last follicular wave, absence of progesterone
- Follicles regress (atresia) in-stead of ovulate
- Eventually normal cycle begins (w/ lime 1 follicle ovulates later in response to + LH (luteinizing hormone))
- 'Split" estrus
- Uncommon condition
- In & out of estrus
- M/b related to stress, improper teasing or transportation

- Mating w/o pregnancy early in breeding season
- Responds to teasing
- Receptive to stallion
- . Allow stallion to mount for 8-30 days, m/b ≥ 30 days
- No pregnancy
- Prolonged receptivity ends & normal ovulation occurs
- "Split" estrus
- In & out of receptivity to

- Hx: Mating w/o pregnancy early in breeding season
- Rectal palpation
- Small & firm ovaries
- Uterus lacks tone



affect breeding season **GnRH** During last follicular wave

(see box)

- . HCG (human chorionic gonadotropin)

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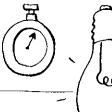
• Wait a month (will start to evulate) then

· Hasten start of breeding season

- Artificial light - 15-16 hr/day will

cause prolonged estrus earlier in year & not

. Progesterone for period of time





- Granulosa-theca cell tumors (pg 188)
- Exogenous steroid Tx
- Nymphomania (pg 158)

Hasten start of breeding season ER 179; E 1326

- Artificial light will cause prolonged estrus earlier in year & will not effect breeding season
- Gradually fight to 15-16 hr/day to initiate ovarian activity 200 watt incandescent bulb in box stall started in mid November or early December to get ovulation by February 60-90 d between 16 hrs of light & start of physiologic breeding
- Most satisfactory means of hastening breeding season
- Hormonal methods: limited & inconsistent results
- $Altrenogest @ \hbox{ (synthetic progestin) or ally for 15dm/ hastenestrus w/in 4-5d after Tx stops,} \\$ only during last follicular wave
- Progesterone, shortens transitional period, 100 mg in oil IM for 7-10 days
- . Normal estrus 3-5 days after Tx stopped, only during last follicular wave
- Normal cycles thereafter w/ 50% conception on 1st service
- HCG (human chorionic gonadotropin) causes ovulation of follicles > 3.5 cm w/in 48 hr Reduces duration of estrus & extra services by the stallion, only during last follicular wave
- GnRH (genadetropin-releasing hormone) more reliable than others . Repeated small doses (not single dose) m/ induce follocular development & ovulation
- Start of breeding season, natural & common CS: Breeding w/o pregnancy Tx: Wait a month & breed

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Hormonal methods: fimited &

0 19:		166	REPRODU	CTIVE SYSTEM
Condition	Facts/Cause	Presentation/CS	Diagnosis	Treatment
Persistent CL, Persistent corpus luteum, Prolonged diestrus M 1370; 12M 1526; R 594; R-Y 68; ER 149; E 1328; EM&S 1027; AT 530; C3T 633 *** Progesterin blocks es CS: Anestrus during Dx: Hx, CS, rectal, US Tx: 2 PGF shots	strus, No PGF breeding season	Anestrus during breeding season 1-3 months of anestrus (nonreceptivity) (average 60 days) Nonreceptive to stallion Anestrus after estrus DDx: Pregnancy (eliminate by rectal palpation & ultrasound) Inadequate teasing (pg 169) Granulosa-theca cell tumors Pyometra (pg 183) Emaciation Gonadal dysgenesis (pg 190) Embryonic death (pg 176) Resorption	Hx, CS No response to teasing Rectal palpation: Cervix closed & high on vaginal wall, tight & dry ↑ Uterine tone Follicles on ovaries (ovulation common) confusing because follicles aye should be in heat, but rejects stallion No CL palpated (because buried in ovary in horses) Laboratory: ↑ Progesterone (similar to diestrus & pregnancy) Elevated plasma protein Ultrasound (US)	• 2 PGF2a injection if 4-5 days old CL • Luteolizes persistent CL so ovilates promptly • Douching w/ sterile saline + ABs is not effective as prostaglandins • Uterine curettage is of questionable value • Estrogen of no value (prolongs Cl PGF2a injection if 4-5 days old CL • 10-15 mg of PGF2alpha (Lutalyse®) • 250-500 µg of fluprostenol (Equimate®) • Luteolizes persistent CL • Ovulation after Tx from 1 or 2-9 d • 4+ cm follicle, ovulate promptly • 2/3/ds ovulate 3-7 days (5 days average) • 1/3rd large follicles regresses & ovulation in • 6-12 days (average 9 days) • CL < 4-5 days old refractory to PGF2a • 2nd dose 7-10 days later
'Silent heat", Psychological anestrum, Anestrus due to abnormal behavior, Bubestrum, Covert heat PM 1527; R 595; ER 193; E 1347	No estrus signs (nonreceptive to stallion), but normal estrus cycle "Lactational anestrus" misnomer Nervous foaling mares, don't breed when foal is by side (still cycle) Not true lactational anestrus as seen in sows, ewes & cows Shy maiden mares "Impersonal", quiet, undemonstrative mares Inadequate teasing fails to show estrus signs in cycling mares Management	No external signs of estrus, or only occasionally When they show signs, they ovulate	Hx: foaling mare, no CS of estrus Genital exam Cycling mares: Follicles on ovaries Vaginal exam show cycling Edematous cervix	Frequent persistent teasing Paddock mare next to penned stallion Pasture breeding to quiet stallion Careful repeated rectal & vaginal exams + Al Twitch & hobble mares as approaching ovulation Often "break down" & accept stallion & show external signs of estrus Try different stallion (some mares pick Restrict foal suckling to 4, 1 hour periods/d (m/ work) Estrogen of no value
The state of the s	and the second s			at faster vieter is des seen vieter is des seen a n en
pathological conditions	Pathological conditions affecting uterus & ovaries, or systemic disease	Failure to exhibit signs of estrus on teasing	Do not use anabolic stero on mares for breeding	ids
Anestrus - pathological conditions 1595; ER 148; EM&S 1028 • Pyometra			• Do not use anabolic stero	ids • Tx pyometra
pathological conditions 595; ER 148; EM&S 1028	uterus & ovaries, or systemic disease • Failure of endometrial cells to	estrus on teasing	• Do not use anabolic stero on mares for breeding • Persistent CL	
pathological conditions 595; ER 148; EM&S 1028 • Pyometra • Hysterectomy &	uterus & ovaries, or systemic disease • Failure of endometrial cells to	• Anestrus	Do not use anabolic stero on mares for breeding Persistent CL (can't palpate) No ovaries 1 ovary large Opposite ovary small, firm & inactive	
• Hysterectomy & ovariectomy • Granulosa-theca	• Failure of endometrial cells to produce prostaglandins • Most common tumor	Anestrus Anestrus Either anestrus, stallion-like behavior or inter-	Do not use anabolic stero on mares for breeding Persistent CL (can't palpate) No ovaries 1 ovary large Opposite ovary small, firm &	• Tx pyometra
• Pyometra • Hysterectomy & ovariectomy • Granulosa-theca cell tumors	Failure of endometrial cells to produce prostaglandins Most common tumor of ovaries	Anestrus Anestrus Either anestrus, stallion-like behavior or intermittent estrus behavior	Do not use anabolic stero on mares for breeding Persistent CL (can't palpate) No ovaries 1 ovary large Opposite ovary small, firm & inactive Occasionally bilateral	• Tx pyometra • Remove diseased ovary • No Tx • Correct underlying problem Take
pathological conditions 595; ER 148; EM&S 1028 • Pyometra • Hysterectomy & ovariectomy • Granulosa-theca cell tumors • Gonadal dysgenesis	• Failure of endometrial cells to produce prostaglandins • Most common tumor of ovaries • Chromosomal abnormalities • Parasitism, pituitary tumors, old debilitated mares w/ poor teeth, chronic debilitating diz, underfed & over-	Anestrus Anestrus Either anestrus, stallion-like behavior or intermittent estrus behavior Anestrus	Do not use anabolic sterol on mares for breeding Persistent CL (can't palpate) No ovaries 1 ovary large Opposite ovary small, firm & inactive Occasionally bilateral Cytogenetic studies Sm., hard & inactive; uterus	Remove diseased ovary No Tx Correct underlying problem

REPRODUCTIVE SYSTEM

Condition Nymphomania

Neurotic mares

IM 1364; I2M

1526; F 596

Facts/Cause

 Wanton estrus behavior (abnormal & undestrable)

- During estrus (associated w/ developing Graafian follicle)
- Diestrus behavior normal



CS: Wanton, Dangerous

Hx, CS, Don't palpate



Rare

- Psychosis
- Mild nymphomania
- Exaggerated behavioral estrus during

Presentation/CS

- estrus, abating during diestrus
- Severe nymphomania Squirt urine
- Tail switching
- Posturing
- Squealing when rear quarters
- groomed or touched Dangerous, aggression lowards horses & people



Dangerous to palpate

No genital organ abnormalities Cycle normally



Treatment

- Euthanasia for dangerous mares Progesterone Tx generally poor results progesterone blocks estrus &
- ovulation in normal mares) Dexamethasone for show mares (CS reported to disappear from 1-5 d after Tx)
- Ovariectomy · Mild cases respond
- Severe do not respond, euthanize

DDx (all eliminated by history, season & PE)

- Seasonal prolonged estrus (pg 165)
- Exogenous steroid Tx
- Granulosa-lheca cell tumors (pg 188)

Nutritional infertility

Tx: Destroy!





. Thin mares & those losing weight rapidly usually fail to ovulate normally

 Poor nutrition 1st month after ovulation early embryonic death & fetal resorption - Poor nutrition after 35 d pregnant, maintain normal foals

· Overly obese mares on lush pasture reported to be poor breeders

Foals of mares on hi nutrition m/ have scours 1st 1-2 weeks of life

- Monitor foal intake & milk out mare manually

 Racing mares in training m/ naii i "deep" anestrus (stress, overwork, lack of energy)



 Breeding season: positive energy balance so gain weight slowly (barren, foaling & lactating mares)

- Maintenance ration in only moderate body condition 1st 8 months of gestation

- 9th month - modest 1 in nutrition & protein until foaling

♦ Foal heat conception

- ♠ Feed last few wks of gestation
- Rapidly nutrition (grain?) after foaling
- High energy ration 1 mo, or more after breeding
- Prevent early fetal death & resorption
- High plane of nutrition 1st mo. of pregnancy

Obese mares

- Diet in winter months to normal weight
- Place on slowly rising plane of nutrition & positive energy balance
- Racing mares in training: give time to equilibrate

. Hx, CS: not pregnant

Poor heat detection,

Unobserved

Silent estrus

IM 1370; I2M 1527

estrus,

#1 cause of infertility

· Management defect in detecting estrus

- Apathy or ignorance of signs by observers
- Use of low libido stallion
- Inappropriate teasing methods
- Mare problem
 - 10% reject 1st stallion, but display signs for 2nd stallion
- Some mares are indifferent to teasing (covert estrus)
- . Know mare to tell when to artificially inseminate

· Mares not pregnant

· Unobserved same signs as silent or covert estrus

Signs of estrus

- #1 standing quietly w/ tail raised (to allow mounting)
- "Winking"
- "Squatting"

- Rejection

- Ears back Squeating

Frequent urination

Signs of anestrus

- · Pressing against teasing bar
- Look for follicles Progesterone levels
 - 14 d of diestrus (normal due to secretion of CL)

Sequential palpation of ovaries

- Falls (< 1 ng/ml) 7 d surrounding estrus (normal)
- If normal, indicate inadequate observation

· Improve teasing management Review procedure & retrain observers

- Check stallion's libido
- Tease w/ different stallion
- Sequential palpation of ovaries
- Reproductive records
- PGF2alpha occasionally to predict when in estrus

Prevention:

 Adequate management of teasing & observation



#1 cause of infertility, Management

CS: Not pregnant

Dx: Palpate for follicles, Progesterone Tx: improve heat detection & teasing

- Kicking & striking DDx:

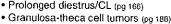
• Silent or covert heat (pg 166)

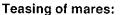
• Prolonged diestrus/CL (pg 166)

• Pyometra (pg 173)

Undernutrition (pg 168)

Gonadal dysgenesis (pg 190)





• Maiden & barren mares

Start teasing early in breeding season

Tease daily on alternate days until confirmed pregnancy

 Foaling mares: Tease 4-6 d after foaling to detect 1st postpartum heat ("foal heat"

Group teasing used: timorous mares m/ not approach or m/ be driven away from stallion

Foaling mares - apprehensive for foals

Individual teasing: good for shy mares
- Teasing chule & foal box good for lactating mares

- Good for close to individual breeding

169



Solid barrier between mare & stallion (so mare doesn't kick stallion)

Head to head introduction (stimulates normal greeting

. Stallion "muzzles mare" (discourage mare from biting)

.. Observe mare's response, CS of heat

- Stallion approaches mare's perineum

Infertility - Infections REPRODUCTIVE SYSTEM Condition Facts/Cause Presentation/CS Diagnosis Treatment Uterus normally sterile Infertility Genital · To establish Dx of infection · Caslick's operation (#1) • Discharge from genital tract Vagina, vestibule & vulva non-Clinical signs of diz + Sexual rest m/b enough infection sterile - Soiled perineum & tail • ± intrauterine infusion (see box) - Culture organisms A 599, 601; T 730; ER 381 - Many organisms normal inhabitants - Matted hair - Inflammatory cytology If mucopurulent material of mares & stallions Vulva m/ gap exposing - Uterine biopsy - Remove by douching & Positive cultures does't mean acmucous membrane - Ultrasound (fluids during diestrus) siphoning tive infection unless clinical or · Perineum m/b sunken ± Stilbesterol or estradiol (IM) · Speculum exam histopathic signs of diz Mild exercise m/ help involution Exudate Positive cultures - Mucosal color of vagina & cervix Oxytocin - 80-100% 1-2 days after service or



- 2. Normal or pathological parturition
- 3. Copulation

few days after foaling

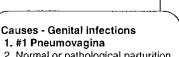
. Usually clear by 6-10 days (± chroni-

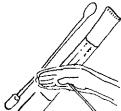
cally infected, especially if pneumovagina)

Barren mares >>> foaling mares

>>maiden mares (lowest)

4. latrogenic (hand & instruments)





- Conformation of cervix
- Routine culture & sensitivity of suspicious & infected mares & stallions
- Culture mare & stallion
- Protected culture swab (Tiegland®)
 - . Smear a sterile glass slide
- . Amies transport medium
- Exudate in vagina to lab
- Pre-ejaculatory fluid to lab
- Cytological or biopsy & histo exam
- Endometritis
- Organisms in lumen of vagina, endometrium or endometrial glands
- If + culture & no histological evidence of diz, consider culture contaminated
- Ultrasound (fluids during diestrus)



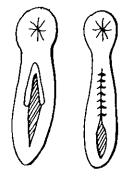
Parenteral ABs of little value

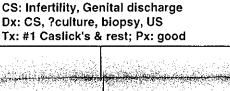
After treatment:

 Bacteriological & cytological or histopathological exams to see when to breed

Prevention:

 Hygiene during breeding & foaling of major importance!!!





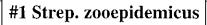
1 S. zooepidemicus, #1 "Windsuckers"



Infectious agents - endometritis & infertility

- Klebsiella aerogenes (pneumoniae) (pg 181)
- · Pseudomonas aeruginosa (pg 181)
- · Staph. aureus
- Enterobacter agglomerans
- Taylorella equigenitalis (CEM) (pg 179)
- Miscellaneous organisms: Corynebacterium equi, Actinobacillus (Shigella) equuli, Aspergillus fumigatus, Candida albicans (pg. 181), Mucor spp., other Strep., Staph., Salmonella spp., Mycoplasm, etc.

• #1 Strep. zooepidemicus (genitalium) (pg 180) E. coli (pg 181)



Prognosis:

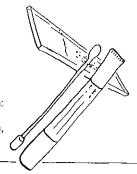
- . Good: usually respond to Tx in 1-2 months
- · Acute infection usually spontaneously clears unless pneumovagina
- . Sooner diagnosis & treated the better the prognosis
- · Chronic severe cases m/ cause sterility
- · Pseud. aeroginosa, E. coli & Klebsiella difficult to impossible to Tx
- . Strep. & CEM easy to Tx
- · Obstructive pyometra poor or hopeless
- · Urovagina: guarded to poor
- · Older barren mares w/ Hx of abortions, pyometra & infertility: poor

Intrauterine infusion (lavage)

- Frequent douching w/r
- Mild antiseptic solution (weak Betadyne® or Novalsan®) Avoid too irritating sol (10% Lugot's solution)
- Mild chlorine sol. (50 ppm), warm physiological saline sol. alone
- Physiological saline solution + ABs (pen., streptomycin, tetracycline, nitrofurazone, polymixin B &/or neomycin combo)
- · After C&S (culture & sensitivity) use proper ABs in oil or saline
- Streptococcus or Gram + organsims
- . Penicillin, nitrofurazone, broad spectrum ABs (terramycin, oxytetracycline, aureomycin, chlortetracycline, tetracycline & ampicillin)
- Klebsiella, E. coli, Pseud. aeruginosa
- . Streptomycin, neomycin, chloramphenicol, polymixin B sulfate, gentamicin & amikacin
- Pump or gravity feed 50-250 ml into uterine lumen
- 2-5 x at 24-48 hour intervals
- Preferred at estrus when natural uterine defence mechanisms working
- Clitoridectomy may be necessary for Klebsiella & CEM

Interpretation of smears & cultures

- · Slide smears Gram stain
- Determine type of bacteria & numbers
- Presence of PMNs (indication of degree of endometritis)
- C&S (culture & sensitivity) by vet gives quick information
- · Culture by lab will be more accurate
- · Possible contamination always to be suspected
- Pure colonies of 1 organism in large numbers w/ evidence of endometritis (PMNs) more significant than:
- Mixed cultures w/ no PMNs (insignificant)
- If culture positive & no histological evidence of infection, consider swab contaminated
- Often histologic evidence, but negative swab



Culture & Cytology

REPRODUCTIVE SYSTEM

Uterine cultures & cytology

T 719; R-Y 87; E 1312, EM&S 957; M 257

- Bacteria ≠ endometritis: recovery of bacteria doesn't diagnose endometritis because organisms are mostly commensals & can easily contaminate culture
- · Not an aid in diagnosing or confirming inflammation
- · Used to suggest a cause of an already confirmed inflammation
- · Maiden horses culture?
- · Post breeding all mares showed positive cultures, so no use culturing then or after foaling, infection for 10 davs

Technique:

- Culture endometrium not cervix which doesn't reflect uterus
- Culture at anytime (estrus or diestrus), if affecting fertility, will be there whenever. Many people believe in culturing during estrus over diestrus as it easier at estrus because open, however also more resistent at this time due to estrogen
- Interpretation: must be significant (not if +1, occasional colony, to +4, look at cytology results). Type of organism also
- Pathogenic organisms: Strep. zooepidemicus 65% E. coli, proteus (30%), Pseudomonas aeruginosa, Klebsiella pneumoniae
- Documented inflammation: Staph. spp., Corynebacterium, Proteus spp. should be considered patho-
- Contaminations: alpha hemolytic Strep., Enterobacter spp & Staph. epideridis, rarely cause
- Repeated recovery to confirm role: Candida spp. & Aspergillus (fungus)
- . Often develop secondarily to antibiotic therapy
- . Potential for severe irreversible damage

Significance of culture

- 1. Greater than 48 hours post breeding 🦓
- 2. Repeat recovery during one estrus
- 3. Recovery during successive heats
- 4. Several organisms at one time, usually means contamination, coliforms from rectum (poor conformation)
- Nature of organism

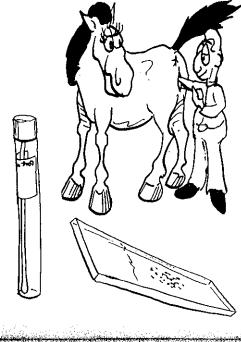




- Cell types
- Mostly epithelial cells (tend to clump more as approach
- Occasion PMN normal, < 1%
- . 69-70% = endometritis
- Lymphocytes infrequent
- RBCs if traumatic
- Spermatozoa, usually degenerating
- Mucous (diestrus stringy; estrus a lot more)
- 1 swab for culture (bacteriology)
- 1 swab for cytology (cell type present [scraping])
- Normal < 1% inflammatory cells
- If great # PMNs have a problem

Cytology over culture

- If culture positive & cytology negative, go with
- If culture negative & cytology full of inflammatory cells: have problem





· Easiest during estrus when cervix relaxed & dilated

- Preparation
- Well restrained & in clean envir.
- Sterile gloves for each mare
- Tail wrapped
- Surgically scrub external genitalia (Nolvasan® or Betadyne®) & dried
- Protected culture swab (Tiegland®)

- Prepare all swabs in turn:

- . Remove cap while still in plastic envelope . Pull swab back a little in tube
- Pencil mark handle of swab next to end of tube so know when protected on removal from mare

Swab clitoral fossa

- Smear a sterile glass slide
- Break tip of swab into Amies transport medium

- Put on a new plastic sleeve

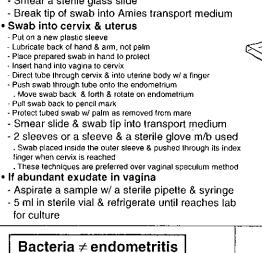
- Insert hand into vagina to cervix

- · 2 sleeves or a sleeve & a sterile glove m/b used Swab placed inside the outer sleeve & pushed through its index

· If abundant exudate in vagina

Cytology over culture

- Aspirate a sample w/ a sterile pipette & syringe
- 5 ml in sterile vial & refrigerate until reaches lab





Uterine Biopsy

REPRODUCTIVE SYSTEM

Severe

Uterine fibrosis,

Condition

Barren mares

T 723, 656; ER 225; R-Y 89; E 1313; EM&S 960; M 253; S 751



· Fibrosis: when widespread becomes main limiting factor in carrying to term

Facts/Cause

. Endometrial categories 1-3:

- Predicts ability to carry foal to term: most valuable tool for predicting (especially in older mares)

Barren 1 year w/ moderate fibrosis twice as fertile as those barren for 2 or more years Widespread changes much worse than scat-ered changes in ability to carry to term

Doesn't say if mare can get pregnant

Infertility

- Barren mares: 3 or more good breedings
- EED (early embryonic death)

Presentation/CS

· Repeat breeders

• Hx, CS

- Uterine biopsy
- Fibrosis categories 1-3 C1 is normal
- C3 is nearly hopeless to carry to term

Diagnosis

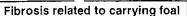
 Don't buy Category 3: recommend against for broad mare

Treatment

 Category 3: not sterile, so if valuable it m/b worth:

 Breeding to get on an average one foal in 10 yrs

 Minimum contamination of breeding, greatly improves foaling rate for mares w/ fibrosis



CS: Barren

Dx: Biopsy: catogories 1-3

Tx: Don't buy C 3

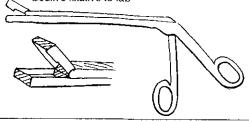
Foaling performance (produced toals) Kenney catagories

Category 1 - 70-92% Category 2 - 50-67%

Category 3 - 4-10%



- Procedure (see box below) Swab for culture before biopsy
- Biopsy uterine bifurcation
- Don't do if pregnant because will abort
- Bouin's fixative to lab



Uterine biopsy

T 723, 656; ER 225; E 1313; EM&S 960; M 253; S 751

- Uterine biopsy: valuable & easy
- Predicts ability to carry foal to term: most valuable tool for predicting (especially in older mares)
- Fibrosis: when widespread becomes main limiting factor in carrying to term
- . Categories 1-3 of fibrosis: used to predict
- .. 1 is normal
- .. 3 is nearly hopeless to carry to term
- Doesn't say if mare can get pregnant

Indications for biospy

- 1. Barren mares: 3 or more good breedings
- 2. Mares with EED (early embryonic death)
- Repeat breeders
- 4. Clinical endometritis or pyometra
- 5. Behavioral anestrus in breeding season June July
- Mares requiring genital Sx (prognostic indicator)
- 7. Fertility exam

Contraindication for biospy

1. Pregnancy (will abort due to dilation of cervix)

Endometrial (Kenney) categories 1-3

Kenney: amount of fibrosis/scar tissue present determines scale, thus determines ability to carry to term. Not a reversible condition; lining of uterus must have a lot

- of glandular tissue
- Category 1: No detectable changes or only slight inflammatory changes which are sparse & scattered, no hypoplasia, no atrophy

Category 2: Slight to moderate changes

- Diffuse & scattered changes, cellular infiltrates, scattered librotic foci w/ inflammation, scattered periglandular fibrosis, nest or clumping of glands 3- 5.5 mm in 4 or more fields, widespread lymphatic lacunae Atrophy
- Category 3: Widespread changes
- Widespread, diffuse, severe inflammatory changes Deep atrophy in breeding season Not sterile, but low foaling rate < 10%

- Periglandular dilation, glands m/ have cells w/in them
- Some places have no glandular tissue
- Scar tissue isolates glands into clusters

Interpretations for categories

- Done by expert (lab)
- Parts of evaluation
- Adequate sample ≥ 1 linear cm
- 2. Histological stage of estrus (endometrial epithelium type
- 3. Inflammatory (cellular) response, type & no. of cells present
- 4. Degree & extent of endometrial degeneration (fibrosis lymphatic lacunae & cystic glands, size, # clumping of endometrial glands)

Normal endometrium & estrus cycle

- Anestrus: cuboidal epithelium (atrophy) w/ straight glands
 Transitional period (in Spring & Fall) still atrophied though
- may have follicles that don't ovulate so biopsied for repeat breeder (need to wait until come into true breeding season)
- Estrus: columnar epithelium & torturous glands Margination of PMNs in capillaries, but don't migrate through stroma

Pathological changes of endometrium

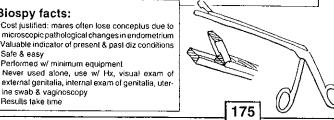
- Inflammation:
- Acute: margination in capillaries & migrate through stroma Chronic: mononuclear cells, plasma cells, eosinophils & mast cells
- More common to get "no growth swabs" w/ indications of inflammation
- Fibrosis: permanent as apposed to inflammation When widespread becomes main limiting factor in carry-
- Glands tend to dilate w/ scartissue, become occluded; get periglandular fibrosis More widespread, regardless of severity, lower the
- chance to carry to term
- Lymphatic lacuna: dilated lymphatic vessels
 Endometrial cysts: coalesced lymphatic lacunae
- Reduce abilily to carry to term
- DDx from surgical edema
- Lymphatic lacunae look like cracks in slide or lakes, no
- Endometrial cysts: only small ones found w/ biopsy
- M/b associated w/ EED (early embryonic death)
- Endometrial glands, size, #, clumping, activity very important, support of early conceptus, endometrial milk. Arrangement of glands indicates amount of scartissue present

Biospy facts:

- Cost justified: mares often lose conceptus due to microscopic pathological changes in endormetrium
 Valuable indicator of present & past diz conditions

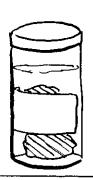
Performed w/ minimum equipment

ine swab & vaginoscopy Results take time



Uterine biopsy procedure (see box)

- Preparation: empty rectum, detailed rectal palpation of genital organs
- Bandage tail & wash & dry vulva & perineum
- Swab for culture before biopsy (avoid contamination)
- Because inflammatory changes most common pathology of endometrium.
- Interpret culture & biopsy together Guarded swab (preferably double guarded to avoid contamination)
- Use hand instead of speculum to open vagina (contamination) . See swab technique 1 site sufficient in absence of specific pathology
- Essentially representative of whole endometium
- Biopsy uterine bifurcation
- Palpate entire uterus for uniformity (texture)
- Abnormal areas also biopsied
- Any stage of estrus because of ease of dilating cervix
 - . Don't do if pregnant because will abort . Endometrial architecture varies w/ time of season
- .. Inexperienced vets biopsy at diestrus when full luteal effect to
- avoid misinterpretation Write down stage of estrus & physical findings at time
- Introduce biopsy through cervix, into body of uterus as done for swab (guarded or double guarded w/ gloved hand & inside rectal sleeve)
 - Other hand in reclum to hold wall of uterine horn against jaws of forceps so
- can take adequate sample
 . Sharply pull to complete biospy
 . Don't take bile through entire wall, so be careful
- No sensation in lining of uterus Instruments as long as possible in large mares Forceps area large enough to get adequate sample, must go from mucosal surface through
- mucosa muscularis Tissue preparation
- Bouin's solution 2-24 hr, 1 part tissue to 10 parts sol.: dislodge tissue from biopsy w/ line hypodermic needle, Bouin's better than formalin. 70% ethyl alcohol or 10% formalin
- Routine histopathologic procedure tained w/ hematoxylin-eosin





external genitalia, internal exam of genitalia, uter-

- Dystocia

- Severe infection & metritis

intromission

- Remove stitches in 10 days

Single umbilical tape stitch at bottom for added strength

Dx: Hx, Tilted vulva

Tx: Caslick's + Rest

Infertility Infection from

normal &

pathological

parturition

Characterized

- Dystocia
- Dead or diseased foal
- Retained placenta
- Severe trauma
- Infertility due to:
 - Incomplete or delayed involution
- Uterine inertia
- All mare's genital tracts infected during or just after foaling
- Infections enters as mare rises & vagina & uterus balloons w/ air

- Normally gone by 9th or 10th days



Hygiene, Hygiene, **HYGEINE!!!**

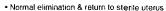
Prevention:

- ♦ incidence of infection
- Careful washing & disinfecting perineal region before foaling

REPRODUCTIVE SYSTEM

- Sterile bandage on tail
- Foal on sterile heavy rubber sheet
- Temporarily closing vulva w/ skin clips after foaling





- "Leukocyte tide": massive invasion of WBCs in endometrium & lumen of uterus
- Phagocylize infective organisms
- # Blood supply
- Mucous production
 Relaxation of cervix
- All of above happens any time in estrus w/ uterine infections
- Results in release of prostaglandins, luteolysis of corpus luteum
- Prompt onset of estrus
- Shortened estrous cycle

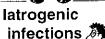
Infection from copulation



Complication:

- Pneumovagina which prevents normal elimination of infec. in vagina no matter what the hygiene (semen always has some organisms)
- Normal protective mechanisms prevent establishment of the majority of these infections







 Improperly sterilized instruments (speculum, douching equipment, gloves) Artificial insemination

Hygiene, Hygiene, Hygiene!!!

Prevention:

Mare

- Tail wrap
- Thorough cleaning of perineal region & buttocks (soap & water then wiped dry w/ cotton)

Stallion

- Wash penis & prepuce w/ soap & water & thoroughly rinse before & after service
- If mild antiseptic used, rinse w/ clear water & dry before service
- Betadyne® (povidone iodine), Nolvasan® (chlorhexadine) or quaternary ammonium compound
- Excessive frequency m/ incr. Pseudomonas, Klebsiella & E. coli (resistant to antiseptic & ABs), especially in stallion which is washed more often)
- Breeding area clean, free of dust & protected from wind

Diagnosis

History (import, contact), CS

Mare, from clitoral sinuses &

fossae, cervix if possible & any

Stallion: swab of prepuce, pe-

nis, urethral fossa & sinus, dis-

tal urethra & pre-ejaculatory

Amies w/ charcoal (or Sluart's

media), refrigerated at 4° C, to lab in

± Blood tests in 20-30 d after

breeding to allow antibodies to

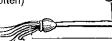
Passive hemagglutination &

Bacterial culture:

exudate

fluid

ELISA



Condition

Contagious equine metritis, CEM

Mk 693; IM 1397, 1414; 12M 1561; EM&S 1034; E 1338; R-Y 182; C3T 757; M 260; Pop 14-5/





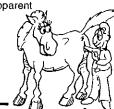
Facts/Cause

- Diz of infertility No clinical CEM in USA
- · Federal regulation restricts importation of horses 2 yrs old from countries w/ CEM (most of Europe & Japan)
- 1975 1st case in France, 1977 to US; economic
- Highly contagious venereal diz
- · Reportable to state vet in most areas
- Taylorella equigenitalis (originally
- Transmission: coitus, hygiene of breeding



Presentation/CS

- · Infertility, fail to conceive
- Endometritis
- Profuse, sticky, mucopurulent, vulvar discharge
- 2-6 d after breeding - M/b asymptomatic
- Conception rate low (due to salpingitis, infection of mesosalpinx) (If get pregnant, very early abortions)
- Fertility regained when subsides
- Abortion uncommon
- No CS of systemic diz
- · Stallion carrier, clinically inapparent



CEM like organism: found in a donkey jack in Ca. in 1997 & 1 in Ken. in 1998 & other horses on the farms of these Jacks since then.

produce diz? carrier mares & stallions?

Considered a subspecies of CEM for now since little is known about it: cultures like CEM, contagious? can it

· Send tissue to lab for culture Location of organisms

 Stallion: resides in smegma of prepuce. urethral fossa & surface of penis

Clitoridectomy: remove clitoris · Xylazine sedation & restraint

Sinusectomy & cliteridectomy

· Suture or leave open to heal

· Local anesthetic around & under clitoris

- · Mares: clitoral fossa
- . Foals exposed at birth m/ retain infection until breeding age

Treatment

- Eradicated from USA but CEMlike organism & possibility to import
- Report CS of CEM or known exposure - quarantine diz
- Not difficult to treat, susceptible
 - to most antibiotics & disinfectants - Chlorhexidine (Nolvasan®) & nitrofurazone cream to mares & stallions
 - Systemic antibiotics unnecessary
 - Uterine lavage (weak Nolvasan®) Some remove clitoris to eliminate nidus
 - of infection (see box)
 - Free carrier stallions of organism, get rid of smegma ("bean" in urethral sinus) (regular cleaning w/ chlorhexidine [Nolvasan@) & nitrofurazone cream)



Prevention of CEM & CEM-like:

- Isolate & test all horses that have been off the property
- 3 swabs > 6 d apart of breeding mares or stallion & 1st mare bred tested in 20-30 d
- Strict hygiene during breeding (disinfect equipment)
- Artificial insemination

Contagious, Eliminated in USA, New CEM type organism? CS: Vulvar discharge, infertility (not abortion)

Dx: Culture clitoral fossa, prepuce, penis

Tx: Easily treated, Sinusectomy & clitoridectomy

180 REPRODUCTIVE SYSTEM Infectious Agents of Infertility Condition Facts/Cause Presentation/CS Diagnosis Treatment • CS of diz 205 Infertility #1 cause of infectious infertility Streptococcus Caslick's if pneumovagina Discharge from genital tract Common cause of abortion & foal Speculum Sexual rest zooepidemicus - Soiled perineum & tail infections Mucosa: congested & ABs in oil or saline lavage IM 1382; I2M 1535; R 601; ER 382 Common inhabitant of external geni-- Matted hair inflammed - Penicillin, nitrofurazone, broad Vulva m/ gap, exposing . Moist, blood vessels prominent & talia of mares & stallions spectrum (terramycin, oxytetracycline, Normally no diz unless resistance mucous membrane aureomycin, chlortetracycline, tetracycline & ampicillin) Cervix: brick red instead of pink of mucosa lowered Perineum m/b sunken . Edematous, os relaxed & open 50-250 ml into uterine lumen 100% of mares contaminated for Thin, atrophic vulvar lips Cloudy mucopurulent exudate - 2-5 x at 24-48 hr intervals 3-6 days following parturition to yellow-white pus in vagina Preferred at estrus when natural uterine - Normally eliminated by 6-10 days . M/b none or just thin exudate from defence mechanical working - Others not cleared until 2nd estrus Most evident at estrus due to - Others chronically infected hypersecretion of mucus (especially if pneumovágina) C&S (culture & sensitivity) Conception possible in mild infections Uterine biopsy - But likely terminated by abortion, DDx acute & chronic difficult EED (early embryonic death), or a Breeding Hx diseased foal Older more likely chronic Pseudomonas aeruginosa infections yometra - uterus dilated Mild or acute cases difficult because m/ follow changes not pronounced Factors lowering resistence Prognosis: Usually respond to Parturition Pneumovagina Tx in 1-2 mo Acute infection usually spontaneously clears Breeding unless pneumovagina Old age Sooner Dx & Tx'ed, better Px · Lowered resistance Chronic severe cases m/ cause sterility · Wear & tear of endometrium Pseud. aeroginosa, E. coli & Klebsiella Prevention: #1 infectious infertility difficult to impossible to Tx Health certificate on mare required by some stallion owners Strep. & CEM easy to Tx Obstructive pyometra: Poor or hopeless Common cause of abortion - Cultural exam of cervix &/or uterus Dx: CS, Speculum, C&S, biopsy Urovagina: Guarded to poor - Interpretation difficult Older barren mares w/ Hx of abortions Clinical & histo exams m/b desirable Tx: Calsicks & rest, lavage pyometra & infertility: Poor Klebsiella pneumoniae var genitalium Infertility Speculum Klebsiella Caslick's if pneumovagina (formerly Freidlander's bacillus, encapsulated genitalium, "Viscid rod" infection) Thick, viscid, tenacious, Vaginal & cervical mucosa - Sexual rest pneumoniae, slimy exudate containing dull reddish-brown Uterine lavage: ABs in oil or saline · Less common than Strep. flocculi Often 100's of ml of exudate in "Viscid rod" - Streptomycin, neomycin, chloramzooepidemicus phenicol, polymixin B sulfate, genta-- Dull-grey to yellowish-white uterus · Gram negative encapsulated rod micin & amikacin infection - Estrus: profuse exudate Rectal exam: uterus enlarged & Transmission by coitus, instruments, 50-250 ml into uterine lumen - Soil perineum & tail, matted walls thickened IM 1382; I2M 1535; R 605; ER 2-5 x at 24-48 hr intervals vet's hands Preferred at estrum hair • More difficult to Tx than Strep, or CEM Clitoridectomy · Sterility more severe than Strep. m/b necessary ± Carrier in clitoral fossa (like CEM) Px (prognosis) Common contamination of saw dust Predisposing factors Chronic severe cases m/ cause • Other infections # inflam. response - Age of mare sterility Adhesions in cervix = pyometra - Length of infection Pseudo. aeruginosa, E. coli & Klebsiella - Time of estrus cycle difficult to impossible to Tx Obstructive pyometra: poor or hopeless - Frequency of service Older barren mares w/ Hx of problems: poor Milky white exudate + flocculent **Pseudomonas** Pseudomonas aeruginosa Infertility Tx same as Kiebsiella, except material Stallions to mares Exudate no clitoridectomy infection Turns green, watery-grey or Older mares more likely to have CS IM 1382; I2M 1535; R 606 yellow-green on exposure to · Sporadic outbreaks by more pathogenic strains Px: Same as Klebsiella (see box) C&S (culture & sensitivity) . M/b found on culture of cervix Infertility E. coli Consider that m/b contaminant Consider possibility as IM 1382; I2M 1535; R 606 contaminant · Candida albicans, fungus Culture Infertility Nystatin (5 d) Candida · Usually just contamination Usually asymptomatic · Biopsy for serious endometritis Amphotericin B

M/b serious - endometritis

- Organism w/in uterine lumen

or endometrium or

endometrial glands

· Chlotrimazole uterine infusion

every 3rd d for 3-4 treatments (human

product - suppository inserted into uterus)

- Rapidly eliminated spontaneously

Serious infection following prolonged

ABs Tx of vagina & uterus

infection

Uterine infection

Endometritis, Metritis. Pyometra

IM 1382, I2M 1535; T71 718, 730; R 607; R-Y 161, 179; EM&S 1033; 1036,1065; E 1332, 1340; M 262; C4T 517



- Barriers to infec. of endometrial cavity (uterus) Vulva & vestibular sphincters

Facts/Cause

- & cervix

 Parturition breaches all borders, also service, Al, examination, or defect in a barrier (pneumova-
- Bacteria usually transient & eliminated during puerperium (period of confinement after labor)
- Estrogen i phagocytosis (PMNs & monocytes)
 Prostaglandins cause luteolysis of CL
- Prompt estrus w/ short estrous cycle
- Involution of uterus
- Normally occurs by 10-15 d (before that can't be safely retracted) - Fluid abnormal after 14-18 d
- Involution & repair in 40-50 d
- Predisposing factors
- Abortion
- Concurrent systemic diz
- Malnutrition
- Dystocia & manipulation

Causes:

- "Windsucking"
- Abnormal parturition
- Service
- Gross contamination



• B-hemolytic Streptococci (S. zooepidemicus &

· E. coli, Pseudomonas aeruginosa & Klebsiella

Organism

S. equisimilis)

pneumoniae

Presentation/CS

Infertility after mating

· To establish Dx of infection

- 1. Clinical signs or diz
- 2. Culture organisms
- 3. Uterine biopsy
- Speculum exam
- Exudate
- Mucosal color of vagina & cervix
- Conformation of cervix
- Routine C&S (culture & sensitivity) of suspicious & infected mares & stallions

Diagnosis

- Culture mare
- Protected culture swab (Tiegland®)
- Smear a sterile glass slide
- . Amies transport medium
- Exudate in vagina to lab Cytology or biopsy & histology exam
- Endometritis
- Organisms in lumen of vagina, endometrium or endometrial glands
- ·if culture positive & no histo, evidence of diz, consider culture contaminated

Prevention: Hygiene during breeding & foaling of major importance!

Treatment

+ Sexual rest m/b enough

Uterine lavage (remove fluids &

Physical removal of uterine contents

Stilbesterol or estradiol (IM m/b useful)

Mild exercise m/ help involution
 Parenteral ABs of fittle value unless

Bacteriological & cytological

or histopathological exams to

Caslick's operation (#1)

m/b critical, douching &

see when to breed

toxic products)

siphoning

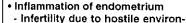
systemic diz

After Tx:



"Windsucking"

Endometritis



ment to sperm & egg

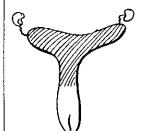
- 100% have some degree of inflammation after service or parturition
- Mild/severe, acute/subacute/chronic
- Normally healthy mare
- Usually clears in 3 or 4-15 d
- If 2° infection (Pseudomonas) m/b several estrous cycles to clear - Cleared by defense mech. of uterus
- Older barren mares
- Defense mechanism defective
- . Few PMNs & phagocytosis of bact.
- . M/ not clear chronic infection

 Asymptomatic usually clears spontaneously

• CEM - Taylorella equigenitalis · Contaminants - Alpha hemolytic Strep,

Proteus, Citrobacter spp.

- Infertility after mating (temporary or chronic)
- Intermittent or continous vaginal discharge



Young healthy mare

- Speculum: edema & relaxation of cervix Flocculent pus in cranial vagina
- Rectal exam: varying uterine distention & turgidity & edema
- Older barren mare
- Speculum: no noticable changes in cervix . Creamy colored exudate in uterine lumen
- Rectal palpation
- No enlargement of uterus

vagina (closed or open) %

- Palpate fluid (lochia or pus in uterus)

- Abscesses of uterine glands

- Friable & swollen uterus (be careful)

Cultures: don't support or deny Dx (contamination

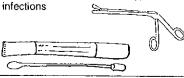
- Degenerative & proliferative changes

- Varying destruction of endometrium

Rectal palpation

problem) Biopsy: uterus

- Culture less than 50% positive
- ± Cytology & histopathology to detect



Speculum: m or m/not be pus in cranial

See above

See above



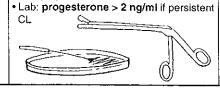
Metritis: inflamation of mucosa. muscularis & endometrium

- Pyometra: accumulation of pus in uterus
- Pathophysiology:

100% after service, Usually cleared

- Mild to severe metritis: permanent pathological
- changes to uterine wall (severe)
 Pyometra rare: fluid abnormat in uterus after 14-
- . Mucoid to inspissated or cheesy . Opened or closed (cervical adhesions)
- M/b associated w/ persistent Ct (endometrium doesn't release PGs)
- M/b incurably sterile

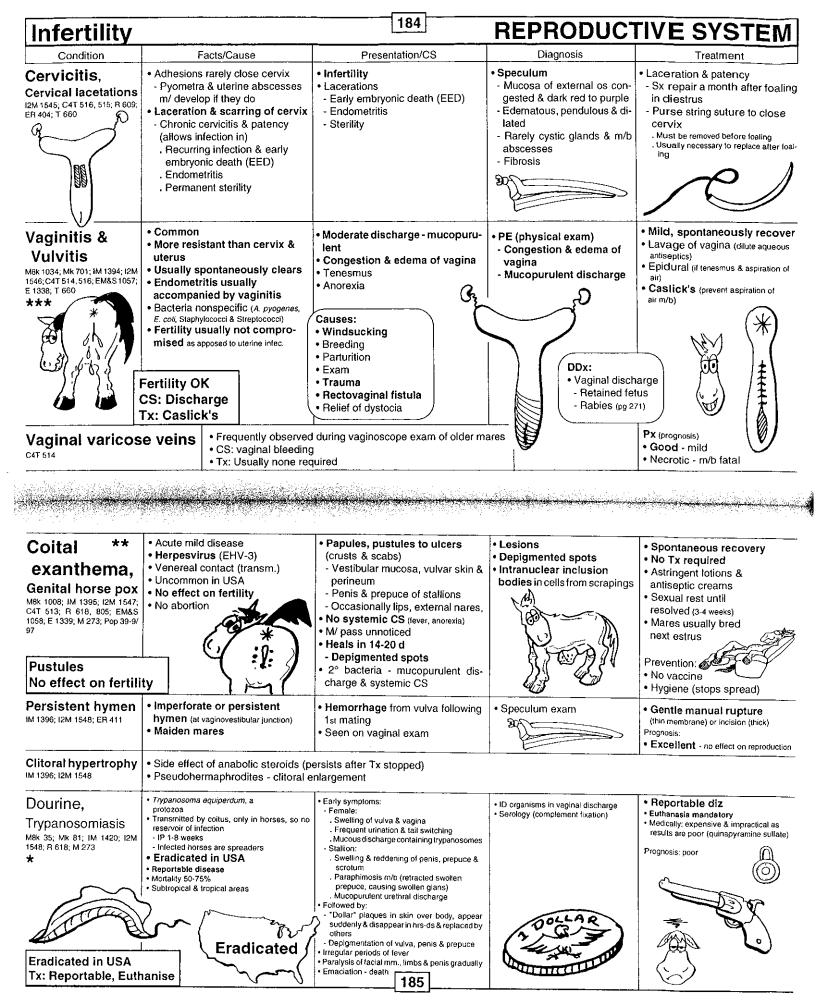
- Metritis
- Discharge from genital tract (several gallons m/b)
- Septic metritis
- . Fever, depression
- . Anorexia
- . Laminitis
- Pyometra
- Few clinical signs
- Anestrus main complaint (persistent CL)
- ± Vaginal discharge
- Sequelae:
- Peritonitis
- Laminitis

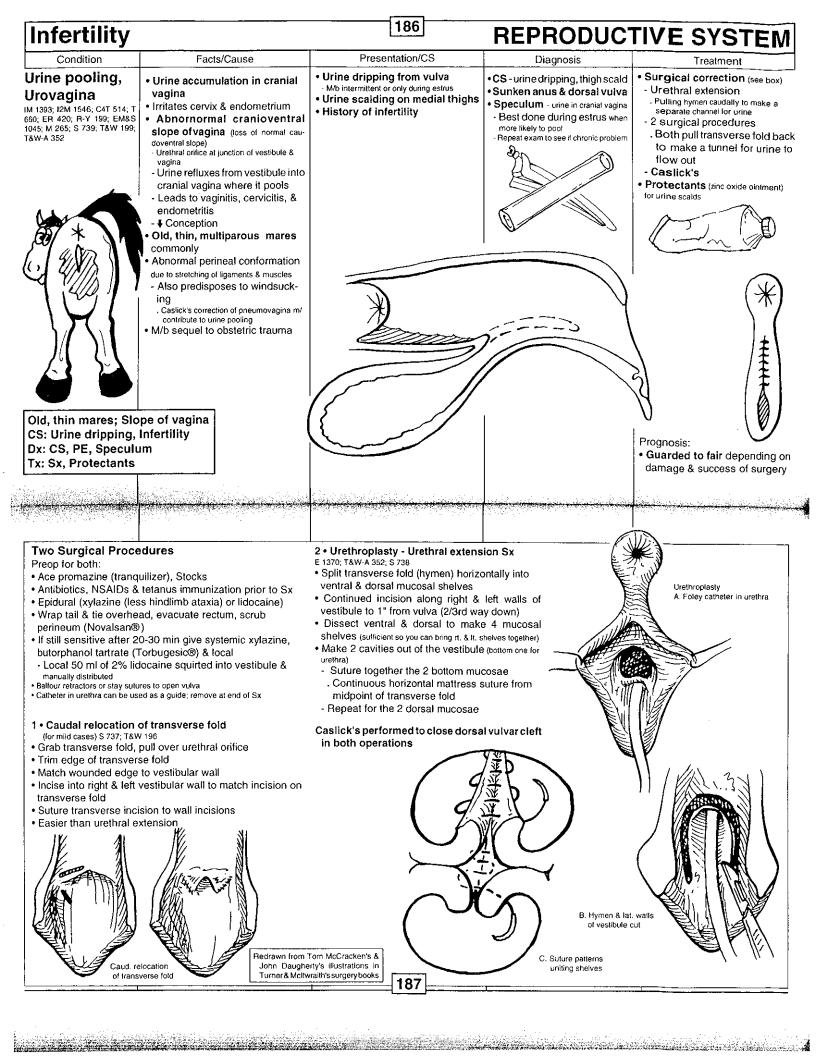






Infertility after mating





188 REPRODUCTIVE SYSTEM Infertility Condition Facts/Cause Presentation/CS Diagnosis Treatment • #1 ovarian tumor, commonly Granulosa Sexual behavioral changes Rectal palpation Surgical removal TOC for all Anestrus or irregular/persistent - 1 large multicystic ovary ovarian tumors theca cell estrus (nymphomania) . Unilateral & benign usually (m/b - Atrophy of other ovary tumor, · Virilism (acts like stallion, heavy malignant) History, CS Adults >>> young neck, mounts) Peripheral testosterone usu-Ovarian neoplasms Secretes steroids which suppress Infertility until removed ally > 50 pg/ml (normal in nonpreg-Prognosis: IM 1375; I2M 1531; R 620; R-Y gonadotrophin secretion so follicles don't 170; ER 399; EM&S 1025; E Abdominal discomfort nant < 50). Pregnant mares also > 50, so · Good: most return to normal develop in norm, ovary, atrophies 1341; T 658, 769; M 264 fertility by next breeding sea-- Won't cycle until tumor removed *** -> 100 pg/ml assoc w/ virilism son after Sx. Some may take up to 1 Originates from follicular cell type Ultrasound M/ have loaled & mare doesn't cycle, anestrus · Causes endocrine disturbances, DDx: estrogen, progesterone, testosterone Other ovarian neoplasms: Other ovarian neoplasms Other ovarian tumors (see box) Arrhenoblastoma - originates from endothelial cell type Ovarian hematomas Endocrine disturbances (estrogen & testosterone produced) Ovarian abscesses Behavior: virilizing (stallion behavior) - infertility Stallion behavior Cystadenoma #1 Ovarian tumor From surface epithelium & primary sex cords Excessive anabolic steroids Nonsteroid secreting Cycle normally w/ normal fertility, m/b pregnant, just space occupying CS: Anestrus or nympho, Virilism, Infertile Testicular feminization Dx: Rectal, > Testosterone · Normal pregnant mares Ultrasound shows no stroma Pseudohemaphroditism Tx: Sx remove. Px: good Teratoma: uncommon & difficult to diagnose Excessive hemorrhage follow- Rectal palpation None, slowly regresses Ovarian · None, normal estrous cycle - Does not alter cyclic activity ing ovulation - Continue to cycle 1 enlarged ovary, 1 norm. hematoma May be up to 12" in diameter Found on palpation Feels similar to ovarian tumor No effect on reproduction IM 1376: I2M 1531: C4T 529: ER No effect on reproduction No behavioral · Regresses, tumor enlarges 401; R-Y 171; EM&S 1024; T DDx: Hemorrhage confined because Lab normal changes 659; M 263; Pop 71-7/98 ovulation inside ovary Ultrasound Ovarian tumor May occur more common in dairy cattle Paleation Cystic follicles * None: doesn't effect estrus or fertility Aspiration, surgical removal Doesn't alter cyclic activity Ultrasound M8k 1005; C4T 531 M/ interfere w/ movement of ova into uterine tube Ovaritis R 609 Very rare in mare

Ovariectomy, Colpotomy



· Surgical procedure, not a disease · Indications:

- Temperamental mares, espesially in working horses, people prefer steady per-

Elective ovariectomy

- Pathology, granulosa cell tumor (nymphomania)
- 1) Ventral abdomen approach
- 2) Flank approach
- 3) Vagina (colpotomy) approach
- Standing (flank or vagina) requires a tractable mare because must remain still during procedure
- Ecraseur also required as ligation very difficult to impossible in vaginal approach
- Ovary must be < 4" for vaginal & 5" for flank
- Cost of standing less than abdominal because of general anesthesia
- Not if reproductive tract infection or introduce into the peritoneal cavity
- Stocks almost essential
- Recovery time . Colpotomy 2 wk . Grid flank 3 wk
- Incision flank 4-6 wk Ventral midline 6-12 wk
- Vaginal (colpotomy)

Colpotomy most common method

- Complication: ileus post op
- Colic secondary to ileus
- Mineral oil prior to surgery, to remove fecal balls which feel like ovaries
- Hold off feed 24-48 hours
- · Restrain in stocks to minimize movement
- Xylazine (0.8 1.0 mg/kg)
- Caudal epidural- 5 ml mepivacaine (Carbocaine-V®)
- Twitch before vaginal incision & maintain throughout surgery Clean out vagina w/ dilute Betadine®
- Pre-op antibiotics
- · Stab incise through dorsal right or left hand wall of vagina, 1 inch from cervix
- Bluntly open stab incision w/ fingers until hand in peritoneal cavity
- Locate ovaries by following uterine horns (prevents taking out a fecal ball, bad mistake!)
- Infuse mepivacaine around ovary (sponge & polyethylene tube)

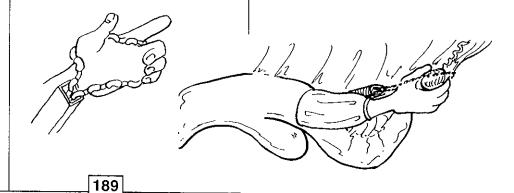
- Ecraseur over hand into abdomen (done completely blind)
 - Twitch horse

Oil

- Grasp ovary & close chain over ovarian pedicle (mesovarium)
- Controversy over slowly or quickly tightening chain to remove
- Don't suture vaginal incision (heals by 2nd intention) Postop care:
- Cross ties post surgery for 1 wk (controversial,
- but may prevent evisceration while getting up) Stall rest for 5 days & m/b controlled walking
- Feed 5 hr after surgery
- Light riding in 7 days, full training 14 days

Standing flank approach:

- Stocks, xylazine, morphine & twitch used
- Grid incision or vertical incision approach used
- Ecraseur or ligation on ovarian pedicle



Temperamental mares

Through vagina, flank or ventral abdomen



drainage



- "Jelly" uterus (flaccid, edematous, thick, doughy or spongy uterine wall) Submucosal cyst m/ bulge into uterine

DDx

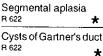
 Pregnancy (remain static. amnionic vessel enlarges)

- Prostaglandins if Hi progesterone from persistent CL

Prognosis: poor

since part of aging process





CS: Sterility

· Rare (uterus)

Cyst of remnant of mesonephric or Wolffian duct in floor of vagina

Tx: Massage, infusion & breed. Px: poor

Lymph stasis, Older mares - normal

Hermaphroditism

Dx: Rectal, US, biopsy

Occasionally described

Mare w/ large protruding clitoris & irregular sexual symptoms

Pseudohemaphroditism

R 623

- Male (XY) genotype
- Looks like female
- Acts like male
- Androgen insensitivity (no androgen receptors) so male sexual organs don't develop
- Female external genitalia & mammary gland
- Displays stallion behavior



- Cytogenetic detection
- Rectal palpation:
- Only tubular organ: vagina, no cervix or uterine horns
- Gonads in ovarian position, but are testicles
- -Nomale accessory sex glands
- No Tx
- Cytogenetic detection to save expense of trying to get pregnant, since sterile



Prognosis:

· Hopeless for repro - sterile



Male (XY) genotype, Female genitalia

Cs: Sterility

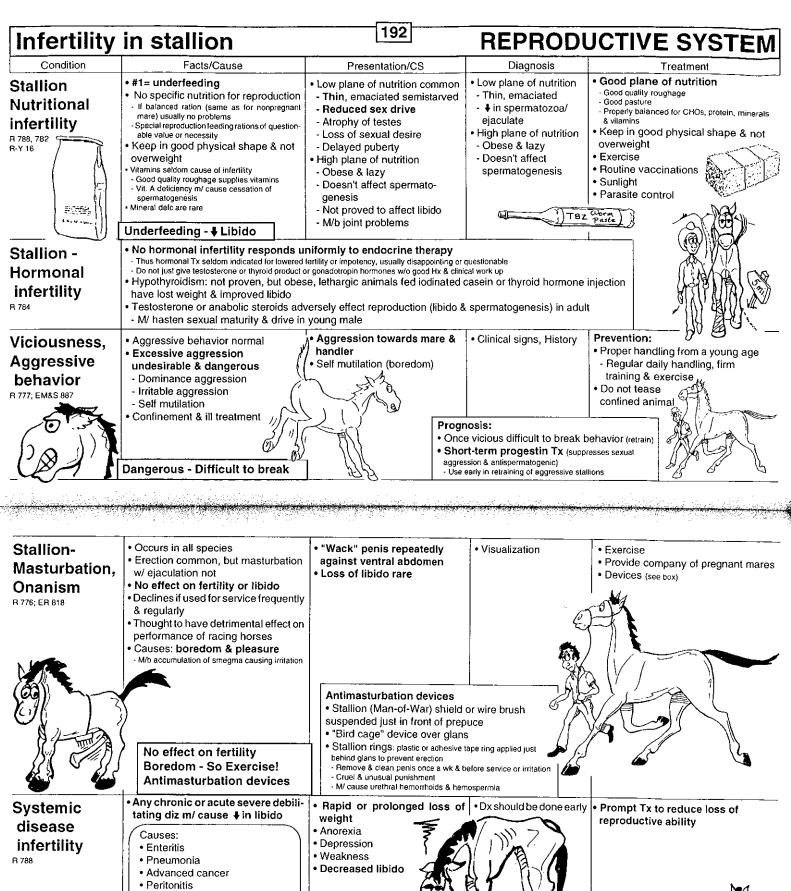
Dx: Cytogenetic detection

Tx: None, Px: hopeless for repro

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Stallion behavior, but mare genitalia Prolonged Tx w/ anabolic steroids

Granulosa cell tumors



Libido Tx cause

- Anaplasmosis
- · Parasitism, pediculosis, mange
- Tuberculosis
- Paratuberculosis





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Age infertility Young & very old m/ have reduced libido due to complete lack of sexual drive

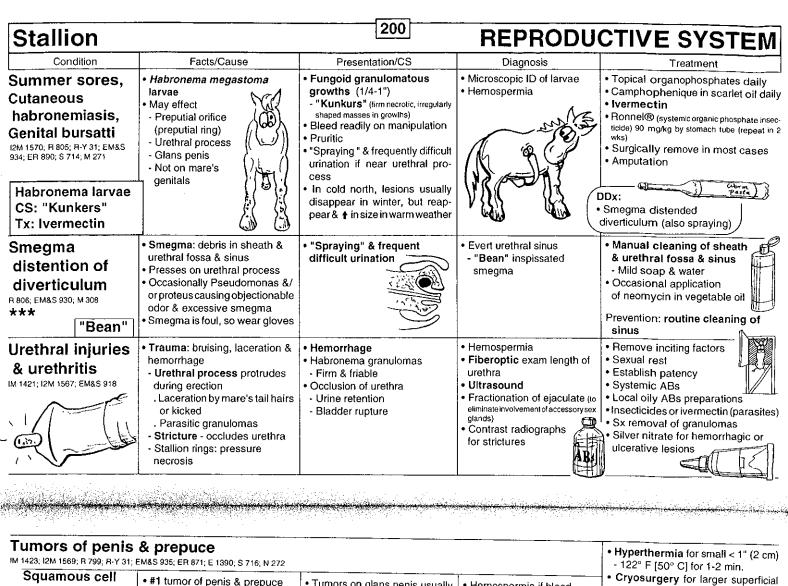
R 789

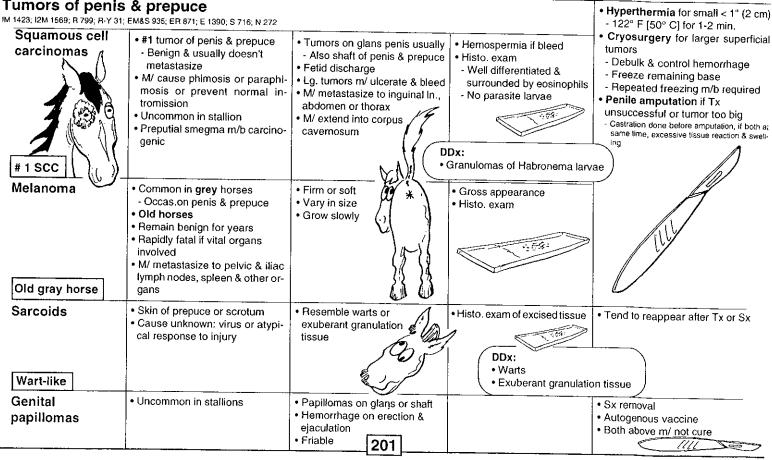
194 Infertility - Stallion REPRODUCTIVE SYSTEM Condition Facts/Cause Presentation/CS Diagnosis Treatment Lack of desire & inability to copulate · Breeding history Patience & understanding Impotency, No intromission . Thorough physical exam Potency (ability to erect, mount & Long periods or sexual rest - Indifference to mare, or Psychic, ejaculate) - Excessive aggression to Careful & repeated (allow to forget) Impotentia coeundi IM 1432; 12M 1575; R 786-789; R-Y 20 Proper nutrition observation during coitus · Impotency: mare or handler Sufficient exercise - Seen in all species including man Determine capacity of Tx chronic diz states (parasitism) - Varies from complete dysfunction stallion Don't overuse to slowness to exhibit libido, mount Prolonged sexual rest if overused & ejaculate Make coitus a pleasurable experience m/ help slow breeder (see box) Causes of impotency Hormonal Tx of questionable value . Genetically lower sex drive (testosterone) Traumatic/negative experience lodinated casein feed to obese. w/ copulation lethargic males m/ help (if hypothyroid) • Excessive discipline or Diazepam (Valium®) 10 min chastisement during copulation before breeding m/b effective (# anxiety) Excessive breeding Sexual inhibition (management) Turn stallion on to coitus - Inadequate socialization, · Training, handling & management important to libido Prognosis: isolation or Socialize young w/ mares Impotency guarded to poor All male groups Careful, patient, quiet training (artificial vagina) M/b permanent in young stallion . Homosexual behavior common Teach them to associate sex w/ pleasure not pain for better results Proper restraint & handling of service so stallion has confidence · Older usually respond to sexual rest . Intimidated weak males Proper footing & room Decrease confidence Psychic behavior: Encourage & compliment (feed the ego) · Female behavior is uncomplicated (willingness to Problem stallions: be mounted) Change breeding site m/b positive or negative Stallion behavior more complicated (identifica-Sexually desirable mares in strong estrus tion, courting, teasing, producing an erection & Allow stallion to pick mare of his fancy complicated mechanics of copulation) Frequent changes of mounts Controlled by CNS & testosterone Longer periods of "teasing" . Don't mess w/ minds, sensitive male sexual Some won't copulate w/ well scrubbed & tail bandaged mares Artificial vagina in hands of experienced handler Negative sexual experience Little correlation betw. blood levels or testoster-Estrus mare standing next to phantom one, libido & sperm quality or quantity Patience - Make sex fun! Run in pasture w/ quiet nonpregnant female (sex therapist) Psychogenic disorders Intromission w/o ejaculation | Intromission, but no Provide ideal breeding environment **Ejaculatory** - Environmental factors - Won't "flag" tail ejaculation - Patience & encouragement dysfunction - Abusive handling - No "pulsations" of penile No spermatozoa in "tail-Drugs: urethra (palpate) - Breeding accidents end" sample of ejaculate - Alpha-adrenergic agonists - Sexual overuse - Not content & relaxed after collected as stallion dis-L-Norepinephrine 15 min before breed- Damage to dorsal nerve of penis service mounts ing (induce contraction of muscles around ductus deferens & accessory sex glands) Insufficient penile friction No "belling" or "flowering" of B adrenergic antagonists glans following erection . Carzolol 10 min before (necessary for ejaculation) Frustration breeding . Contraindicated in chronic obstructive pulmonary diz No "flowering" - Patience Full erection before intromission • Inability to readily enter mare Inability to enter mare Help direct penis into vulva before Premature - "Flowering" or "belling" of glans "Flowering" or belling" of "flowering" erection making penis too large to enter Lubricate vulva glans R 807 mare "Belling" before mounting Help enter before "flowering" Slowness of · Usually acquired vice Improper training, rough treatment or accidents during service breeding - Leads to impotency A 777 195

196 REPRODUCTIVE SYSTEM Stallion Facts/Cause Treatment Condition Presentation Diagnosis Hematoma Reduce edema Mare kicking erect penis during History & CS Trauma to Hydrotherapy Swelling service penis, - Massage Rare in stallion; common in bull · Pressure wraps "Broken" Results - Judicious use of diuretics - Hematoma (bleeding outside tunica penis - Sling close to body albuoinea) M 647, 740; R 775; R-Y 29; Sexual rest Swelling EM&S 929; ER 880, 887 , Paraphimosis - Laceration - Rupture of penis Prevention: Properly supervised service · Make sure mare is in estrus, ready to receive Mare's kick - Supervise · Mare restrained properly (hobbles, etc.) CS: Hematoma, swelling . Good footing for stallion & mare Tx: Reduce edema · Breeding hobbles or Al in nervous &/or violent mares Hemorrhage from prepuce & penis • Rare • Causes: Tumors, Laceration, Bleeding from outside the funica albuginea (unlike bull) R 775; S 709 • Most recover w/o Tx • Local Tx • Sexual rest • Remove tumors Associated w/ RBCs in ejaculate Hemospermia · RBCs in ejaculate Conservative or infertility Cause: surgical Tx of problem I2M 1570: FI 776: R-Y 18, 33: Visual EM&S 939; ER 864 - Penile lesions or fistulas Sexual rest Endoscope Urethritis · Radiographs of - Urethral stricture genital organs - Vascular lesions in mucosa (stallion rings, lacerations or habronemiasisof RBCs in ejaculate urethral process, strongyle larvae or infection of accessory glands & urethral calculi) Inability to protrude the penis Phimosis · Hygiene, possibly pathological bacteria Keep numbers down, do not sterilize or more IM 1420; I2M 1566; R 793; R-Causes - Phimosis Y 30; EM&S 935; ER 888 pathological bacteria m/ take over Clean w/ mild soap, not antiseptics Psychic impotency • #1 Small preputial orifice Tx 1° cause Clean preputial cavity daily w/ hydrogen peroxide - Sequel to injury & scarring (cicatrix formation) · Oily AB preparations infused - Melanomas or Habronema granulomas Broad spectrum ABs systemically Rarely congenital Can't protrude penis Surgical correction Inability to withdraw penis into **Paraphimosis** Traumatized penis History Prompt Tx to reduce swelling & edema & return prepuce - Swollen, pendulous penis · Clinical signs IM 1421; I2M 1566, 1688; M to prepuce 740; R 802; R-Y 30; EM&S Results in: Chronic ulcerative Purse string suture in preputial orifice if penis can be 920; ER 888 balanoposthitis develops - Edema, swelling & - Balanoposthitis if not relieved Support penis in sheath w/in 2 hrs (stallion support) . Prevent gravitational edema & chronic prolapse Phenothiazine-derived tranquil-Surface of penis dry, thickened & inelastic Cold packs & pressure to reduce swelling & allow izers: relaxation of smooth replacement of penis muscles & engorgement of cor-Paralysis (tranquilizers) - Emollient AB ointment of excoriations (supf. wounds) pus cavernosum w/ blood - Flaccid penis Acepromazine - Steroids if inflammatory edema Blood m/coagulate in spaces Causes - Paraphimosis of corpus cavernosum - Systemic ABs to control bacterial infections · Trauma, swelling of prepuce - Exercise to reduce edema Can't fully erect - Isolate from mares Stenotic preputial orifice Often doesn't recover Tumor Paralysis: Paralysis or penis - Slow injection of benzotropine mesylate - Spinal diz or trauma immediately after paralysis due to phenothiazine tranquilizers reported to cause detumescence (flaccidity) Infec. diz (rhinopneumonitis, rabies, - If chronic then same treatment as paraphimosis Phenothiazine-derived Amputation of paralyzed penis (phallectomy) tranquilizers Surgical retraction (Bolz technique) (phallopexy) Sexual rest Prognosis: Good: if swelling & edema resolve rapidly & penis returns to prepuce in few days Can't withdraw penis Guarded to grave: if chronic Priapism R-Y 32: persis-Phenothiazine tranquilizers Stallion that can't fully erect, m/b able tent erection w/o sexual Tx: Return to prepuce to ejaculate if penis placed in vagina arousal

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198 REPRODUCTIVE SYSTEM **Stallion** Condition Facts/Cause Presentation/CS Diagnosis Treatment Clinical signs Sexual rest Inflammation of the glans · Stenosis of preputial orifice Balanopenis (balanitis) & prepuce · Adhesions between penis & posthitis (posthitis) prepuce IM 1422; I2M 1568; R 803 Occasionally in stallion, Pain commonly in bull, ram & dog Copulation avoided Rarely due to bacteria · Early symptom of Dourine (not seen in USA) Herpes virus described (formerly Inflammation of glans & prepuce Spontaneous recovery 10-14 d Papules, pustules to ulcers · Acute mild diz Coital Lesions Herpesvirus (EHV-3) - Vestibular mucosa, vulvar No Tx required · Depigmented spots exanthema, Astringent lotions & antiseptic creams · Venereal contact (transm.) skin · Intranuclear inclusion Genital horse - Balanoposthitis Sexual rest until resolved (3-4 wk) Venereal balanitis & vulvitis bodies in cells from scrapings - Occas. lips, ext. nares Mares usually bred next estrus Uncommon in USA pox No effect on fertility No systemic CS (no fever or IM 1395, 12M 1547; R 618, anorexia) No abortion 805, M 648; Pop 31-9/97 Depigmented spots 2° bacterial infection Discharge & systemic CS Prevention: Papules - Depigmentation No vaccine Spontaneous recovery · Hygiene (stop spread) • Reportable Dourine, Trypanosoma equipedum Swelling of penis, prepuce & · ID organisms in vaginal discharge Transmitted by coitus · Serology (complement fixation) Euthanasia mandatory scrotum Trypanosomiasis - Only in horses, so no reservoir of infec. Medical Tx "Dollar" plaques n Mk 81; IM 1420; I2M 1565; R Expensive & impractical as Eradicated in USA Emaciation - death results are poor · Reportable diz. Eradicated . Quinapyramine sulfate Mortality 50-75% Prognosis: Poor Stallion - Stallion's external genitalia har- Bacterial balanoposthitis rare Culture prepuce, penis & CEM & Strep. zooepidemicus bor pathalogic bacteria, fungi Clinically inapparent in stalurethral fossa & sinus Carrier of - Free carrier stallions & yeasts lion usually Single isolation of Taylorella . Get rid of smegma (bean in urethral - Bacterial balanoposthitis bacteria equigenitalis Dx for CEM Mare vulvar discharge uncommon Mk 693; IM 1397, 1414, 1422; Infertility . Amies w/ charcoal Regular cleaning w/ chlorhexidine - May affect sperm motility C4T 579; M 786; R-Y 21 Others: serial isolation of large (Novalsan®) & nitrofurazone cream #1 problem: passed to mare numbers in relatively pure Strict hygiene during breeding during coitus cultures Artificial insemination less infections - Infertility of mare Strep zooepidemicus Pseudomonas aeruginosa & - #1 infectious infertility in mare Klebsiella spp - Abortion & foal infections Thorough washing of penis & pre- CEM (contagious eq. metritis) puce, including urethral fossa & sinuses Diz of infertility (fail to concieve) No clinical CEM in USA lodine based surgical scrub daily for 1-2 wks . Thoroughly rinse w/ tap water + dilute - Reportable diz - quarantine hydrochloric acid - Not difficult to treat - Taylorella equigenitalis . Dry penis 1% silver sulfadiazine cream Klebsiella pneumoniae Breed mares artificially Less common than *Strep*. More difficult to Tx than Strep or CEM Sterility more severe than Strep. . Semen mixed w/ ABs containing semen extender (Polymyxin-8, {1000 U/ Pseudomonas aeruginosa ml], reagent grade gentamicin [100 mcg/ml], amikacin sulfate [100 mcg/ml]) Buffer both gentamicin & amikacin w/ 8.4% Na - Hard to Tx E. coli, usually a contaminant if bicarbonate before adding to extender cultured Natural service (Thoroughbreds) Censored Candida albicans . Infuse AB extender into uterus Usually just contamination immediately before service Serious infection following prolonged AB Tx of vagina & Candida - Nystatin (500,000 IU/d for 5 d) Amphotericin B Clotrimazole uterine infusion (400-600 mg) every 3rd d for 3-4 treatments (human prod-Strep. zooepidemicus & CEM uct - suppository inserted into uterus) CS: Inaparent Dx: Culture Tx: Hygein, Al 199





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Testicle		202	REPRODU	CTIVE SYSTEM
Condition	Facts/Cause	Presentation/CS	Diagnosis	Treatment
Testicular aplasia/ hypoplasia 41426; 12M 1571; EM&S 900, 05	Aplasia (complete absence of 1 or both testicles): Rare Hypoplasia Unilateral or bilateral Both scrotal or abd. testicles Cause of hypoplasia Transplacental infection or intoxication Hormonal insufficiency Zinc defc Impaired testicular descent Vascular disturbances Abnormal karyotype Adult: prolonged exogenous	Hypoplastic: small testicle Normal 3 yr old scrotal width 3" (8 cm) ** ** ** ** ** ** ** ** *	Scrotal width < 3" Ejaculate Azoospermia Low concentration of sperm Numerous morphological defects	No successful Tx Manage breeding stallion Base number of mares bred on number of normal motile sperme ejaculate None Testicular agenesis Anorchism
> 3" scrotum	steroids			EM&S 894 • Rare in stallions
esticular degeneration 1 1426; 12M 1571; R 826; M8S 901, 906; ER 855; S 685	Causes: • ↑ Temperature · Cryptorchid, ectopic testes, inguinal hernias · Systemic dizs cause temporary infertility due to hi temp (EIA, Pneumonia, Strangles) · Prolonged hi environment temp + hi humidity • Torsion of testis • Salmonella abortus equi & "epizootic" celluliitis due to	Testicles: - Atrophic, softer & small - Chronic: firm due to fibrosis	Physical exam (PE) Semen evaluation (see box) 50 to 60 days for sperm to reach ejaculate Testicular biopsy only as final recourse M/b hemorrhage, degeneration & necrosis Semen evaluation: 50 to 60 d for sperm to re Take repeated samples of	over long time
Semen evaluation	arteritis & influenza viruses • Ehrlichiosis • Piroplasmosis & Dourine	DDx: • Testicular I (hard to DE	Normal semen: 5-159	1/2 abnormal heads, tails & middle pieces
esticular neoplasia 1427; I2M 1572; R 833; R-Y ; EM&S 903; S 681	1° uncommon in large animals Seminoma #1 primary neoplasia (arise from germinal epithelium) Dermoid cysts or teratomas (in cryptorchid testes) Large cysts w/ hair, cartilage & bone Sertoli cell or sustentacular cell tumors rare Interstitial cell (Leydig cell) tumors occasionally (don't produce hormones usu.) Extremely aggressive horses & vicious Castration reverses behavior Lipomas uncommon	Fertility: only slight reduced conception rate to severe infertility & sterility Enlarged testicle	Semen evaluation M/b w/in normal limits EReasonable pregnancy rates	Surgical removal Wait until end of breeding season semen quality OK Bilateral: wait until semen quality falto negate use in breeding, then move
	May not affect semen	(IIII
Cute orchitis 1427; I2M 1572; EM&S 902; 1881	Inflammation of testicles Causes: Infection Trauma Strep. zooepidemicus commonly	Hot, swollen, painful testicle Refuse to mate "Hopping gait" Chronic: testicular atrophy & fibrosis Sterility	Hx, CS Ejaculate - WBCs - Motility & - ↑ Morphlogical abnormalities	Sexual rest imperative Close confinement - physical rest ABs IV (C&S) 1-2 wks past resolution of pain & swelling Glucocorticoids & NSAIDs to reduce inflammation Ice packs (sling under testes)
		12/2		The state of the s

204 REPRODUCTIVE SYSTEM Stallion Condition Facts/Cause Presentation/CS Diagnosis Treatment Manual correction (not always possible) Palpation of tail of Asymptomatic if < 180° Excessively long mesorchium Torsion of Recurrence likely predisposes Severe torsion epididymis out of place spermatic Sx correction Twisting during a race - Acute pain Should be caudal - "Hopping" gait NSAIDs Hot/testicle cord Analgesics - Abdominal discomfort IM 1428; I2M 1573; - # HR & RR R 828; R-Y 26; EM&S 914; - Unilateral swelling & edema of scrotum Inflammation of testicular artery • Causes: - ↑ Testicular temp - Strongyle larvae - Painful testicle - Eq. arteritis virus - Soft & friable testicle "Bag of worms" w/in Abnormally distended & Nonpainful "bag of worms" w/in Thrombosis: unilateral castration Varicocele (transect spermatic cord proximal to thrombus) spermatic cord tortuous veins of pampiniform spermatic cord IM 1429; I2M 1573; EM&S 915: S 681 - Backflow & stasis in veins Causes disturbances in thermoregulatory mechanism (counter current heat exchange belw. hot artery & cool veins keeping testicle temp down) ABs (invitro sensitivity) · Infertility (due to obstruction) Palpate of epididymis esp.tail · Inflammation of epididymis **Epididymitis** 1-2 wk after inflammatory - Induration (process of hardening) IM 1429; 12M 1573R 841; EM&S 909 Cause: cells gone from semen · Enlargement of tail of epididymis Spermatic granulomas, abscesses & - Infection or trauma No cure if obstructive enlargement Adhesions to vaginal tunics - 1° or Unilat. to save valuable Culture - 2° to orchitis or infec. of Chronic abscesses, periorchitis & breeding animals Ejaculate - abnormal sperm accessory sex glands fibrosis If vas deferens & seminal - Bilateral obstruction: azoospermia Granulomas if sperm escape Tail most commonly involved vesicles have no infection · Strep. zooepidemicus Unilateral castration Prognosis: Long recovery period Obstruction often develops Repeated cultures of semen for Poor: obstruction usually occurs preventing sperm from leaving elimination of infection Bilateral: hopeless Tail of epididymis • Obstruction • Px: Poor · Unilateral: remove affected testicle & epididymis Associated w/ orchitis, epididymi- Low fertility infection of Rectal exam Relieve blockage by massage of tis or seminal vesiculitis Failure to ejaculate - Enlarged ampullae ampullae & sexual stimulation ductus · Strep zooepidemicus Semen exam Regular collection of semen m/ Usually unilateral deferens. Massage ampullae per rectum prevent recurrence Ampuliae followed by prolonged sexual Empiracle blockade of ß receptors & Occluded stimulation & semen collection m/ Blocking of ductus deferens Urinary bladder stimulation of alpha receptorsm/cause result in ejaculation Hi concentration of semen, ejaculation

ampulla

M 649; C4T 580; R 845

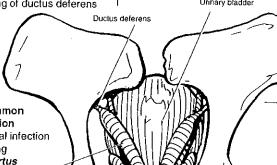
Low fertility

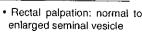
Seminal

vesiculitis

IM 1430; I2M 1574; C4T 579;

R 847; EM&S 916; ER 861; E





often in "strings" or plugs"

M/b WBCs & bacteria M/b clots of pus & low

- Painful

motility

- Semen: m/ contain PMNs & **RRCs**
- Culture:
- Sheath, penis, urethral fossa, preejaculatory fluid, urethra (before &after ejaculation)
- Seminal vesicle effluent (manually expressed, sterile urethral catheter positioned at the colliculs

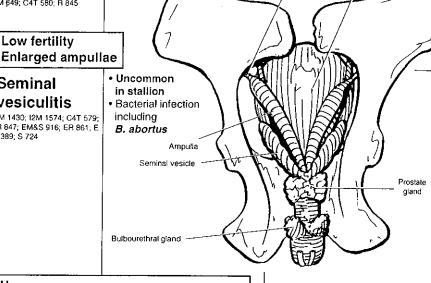


- Broad spectrum ABs (culture invitro
- sensitivity) 2-4 weeks Tx failures occur
- Erythromycin, trimethoprim (both hi pKa & fall soluble)
- Not aminoglycosides (low pKa & not fat soluble)



Prognosis:

- Mild case m/ spontaneously recover in 2-3 months
- Fair to poor depending on causative agent



Uncommon

Painful, refuse to breed

Tx: ABs. Px: M/ spontaneously recover

- Refusal to cover
- M/b unable to ejaculate

 Rare in all animals except dog Prostatitis R 851

206 **Cryptorchid** REPRODUCTIVE SYSTEM Diagnosis Treatment Condition Facts/Cause Presentation/CS Surgical removal indicated Testicle missing from • Testicle(s) not in scrotum . 1 or both testicles missing Cryptorchid, - Malpractice to remove just de- Deep palpation over inguinal ring from scrotum scrotum High m/ or m/not reveal an inguinal crypscended testicle Types of cryptorchids: Suspected only 1 testicle torchid, not abdominal. Stand by shoulder - Always remove retained 1 first - "High flanker" or inguinal crypremoved flankers • Rectal palpation 80% effective - 5-6" skin incision over supf. ring - "Ridgeling" or "Rig"; hard torchid if testes through inquinal M8K 986; I2M 1571; IM Ductus deferens into vaginal ring/ - Bluntly dissect to supf. inguinal ring canal, but not in scrotum to handle 1589, 1426; EqS 722; M270; inquinal canal): inquinal cryptorchid Inguinal cryptorchid at supf. inguinal ring Owner thinks its castrated - Descended epididymis into T 185, 748; T&W 185; S - If ductus deferens not into wall then Open vaginal tunic & remove testicle. 677, 691 inguinal canal but testes in ab- "Superficial" inguinal cryptorchid (inabdominal domen - Rectals may be impossible due to side canal) Forceps through inguinal canal, grasp & - Complete abdominal: testes & epididymis inside abdomen withdraw peritoneum & vaginal lunic fractious horse Most unilateral, left > right - Open vaginal tunic Suspect malpractice (cryptnotremoved) Grasp gubernaculum & pull tail of epididymis & then If right sided cryptorchid, 60% chance inguinal testicle out canal & remove testicle Look for scars on scrotum - 1 out of 10 abdominal cryptorchid "Deep" abd. cryptorchid (not inside canal) - Blood testosterone levels · Normally readily palpated in scrotum by 30 days of birth - Push hand through inguinal canal or internal abd. . 5000 units of HCG, then 1 hr later • If in inquinal canal m/ descend up to 2 years old (4years?) oblique muscle take another sample Locale ampulla at dors, aspect of bladder & trace to . Thought to be genetic tail of epididymis . Tremendous rise if any testicular Gubernaculum Suture supf. inguinal ring if damaged tissue present - Estrogen levels Postop: . Geldings < 50 picogr/ml - Tetanus immunization - Examine internal vaginal ring in 24 . Cryptorchids > 400 picogr/ml . Stallions very high hours, if no adhesions or viscera in ring discharge horse Descent of testicle in fetus: migrates from kidneys via gubernaculum into scrolum Gubernaculum attached to tail of epididymis (future ligament of tail of epididymis) & by future proper ligament of testicle to testis • Inguinal canal; passage through abdominal wall. Inguinal rings tighten up in first week of life. - Deep inguinal ring: bounded by internal abdominal oblique m., rectus abdominis m. & inguinal lig. (free edge of external abdominal oblique m.) - Superficial inguinal ring: slit in aponeurosis of external abdominal oblique m. • Vaginal ring: opening where peritoneum evaginates through inguinal canal to become vaginal tunic.

Surgery for cryptorchid, be prepared to go into abdomen, may still be at kidney

- Dorsal recumbency, tilled table w/ affected side little higher, encouraging bowel to fall away
- 5-6" skin incision made over superficial inguinal ring
- Bluntly dissect down to superficial inguinal ring w/ fingers
- Large branches of external pudendal vein in region, avoid traumatizing them

Inguinal cryptorchid located in inquinal canal

Open parietal vaginal tunic & remove testicle

Descended epididymis into canal

Hook epididymis, pull testicle out & remove

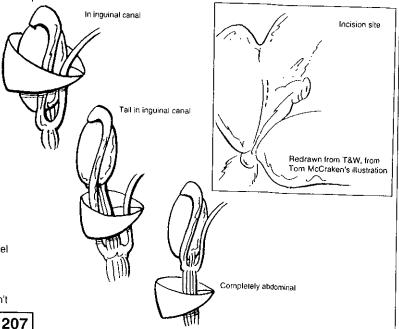
- "Superficial" abdominal cryptorchid: if not in inguinal canal
- Push forceps through inguinal canal & grasp peritoneum & vaginal tunic under it
- Withdraw forceps, holding peritoneum, vaginal tunic & gubernaculum inside vaginal tunic (excessive traction will rip vaginal tunic)
- Palpate cord-like gubernaculum w/in vaginal tunic by rolling between finger & thumb
- Nick vaginal tunic (parietal layer) w/ Metzenbaum scissors
- Grasp gubernaculum w/ Ochsner forceps
- Place traction on gubernaculum to pull tail of epididymis through canal
- Hook epididymis w/ finger & gently pull testicle through, manual dilation of inguinal canal m/o necessary
- Positively ID testicle, compare to tail of epididymis
- Emasculate testicle, if can't be retracted sufficiently to emasculate, ligate cord
- ± Pack w/ gauze bandage over ring if inguinal canal enlarged by surgery & fear of herniation to allow swelling time to obliterate inguinal canal
- Suture skin w/ heavy polymerized caprolactam (Vetafil®) in continuous pattern or w/ simple interrupted sutures w/ long ends
- Rectal palpation of vaginal ring (peritoneum over inguinal canal) next day for bowel or adhesions in ring
- Remove gauze packs & skin sutures & discharge horse
- "Deep" abdominal cryptorchid or if other reasons forceps retrieval doesn't WOrk (accidental rupture of vaginal tunic, vaginal ring or medial wall of inguinal canal,

loss of vital landmarks or previously unsuccessful Sx)

CONSISTS Of an Outer parietal layer & a visceral layer adhering to structures of the spermatic cord and testicle/

Vaginal tunic: evagination of peritoneum out inguinal canal around the spermatic cord & testicle; it

- Digital exploration inside of internal inguinal ring to locate gubernaculum, epididymis or testicle
- If fail to find, manual exploration of abd. w/ hand m/b necessary
- . Push hand through inguinal canal or internal abdominal oblique muscle (thin on medial wall of inguinal
- Locate ampulla of ductus deferens at dors, aspect of bladder & trace it cranially to the tall of epididymis (if can't find then m/b no testicle)
- Close external inguinal ring if damaged, w/ interrupted structures (Dexon® [2] polyglycolic acid] or Vicryi®)



Castration

208

REPRODUCTIVE SYSTEM

Castration. Orchiectomy

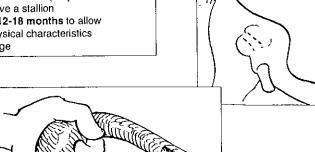
T&W 177; LAS 464; J 1092; E 1390; S 687; M 208; T 745

Procedure:

- · Recumbent under general anesthesia
- Anesthesia
- . Guafenesin w/ thiamylal sodium, xylazine followed by ketamine
- . Rapid induction w/ thiamylal sodium or thiopental sodium alone
- Cast w/ left side down for right handed operator
- Tie upper hind leg cranially
- Prepare surgical site, clipping or shaving not necessary
- Standing animal w/ local anesthetic
 Tranquilize or sedate
- Stand on left hand side just behind the front limb, facing rear end (right handed operator)
- Local infiltration of skin & SQ 1 cm from median raphe (10 mls)
- Infiltrate spermatic cord w/ 18-20 gauge needle
- Some owners demand a standing castration, sensible you m/ talk them out of it or send them to another veterinarian in the next county
- "Open" castration (cut parietal vaginal tunic before emasculator)
- Separate scrotal skin incisions 1/3" (1 cm) on either side of median raphe
- Incise through skin the length of the testicle, leaving parietal vaginal tunic intact
- Squeeze testicle in vaginal tunic out incision
- Grasp testicle in left hand & strip SQ tissue from vaginal tunic w/ a gauze sponge
- Cut through parietal vaginal tunic (open castration) & hook finger in tunic to maintain tension & continue incision proximally
- Digitally break through mesorchium (connecting vaginal tunic)



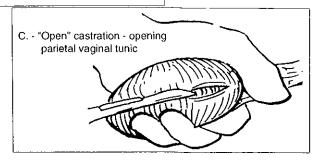
- · Help manage a horse when in company of other horses
- When undesirable to have a stallion
- Generally not done until 12-18 months to allow development of some physical characteristics
- Maybe done at a later age



B. - Stripping SQ tissue from intact parietal vaginal tunic

Redrawn from Tom McCracken's illustrations in Turner & McIlwraith's surgery book

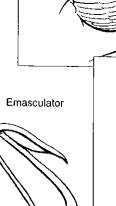
A - Incision sites

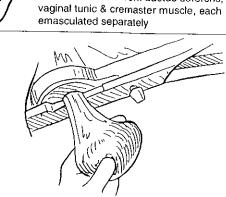


- Separate testicular vessels from ductus deferens, vaginal tunic & cremaster muscle
- Cut ductus deferens, vaginal tunic & cremaster muscle (emasculator, for short period)
- Then emasculate vessels
- "Nut to nut" of emasculators, make sure cutting edge is distal to crushing edge
- . Place forceps proximally on spermatic cord as a safeguard if problems develop
- . Maintain crush for 1-2 minutes
- Digitally enlarge skin incision for drainage
- "Closed" castration (parietal vaginal tunic not cut before emasculator)
- Parietal vaginal tunic dissected, but not opened
- Emasculation of entire spermatic cord
- Greater chance of inadequate crush of testicular artery bec. more structures between jaws of emasculators
 - . Restrict this technique to horses w/ small testicles

Postoperative:

- Tetanus immunization
- Antibiotics usually not needed
- Close observation for several hours & limited exercise
- Periodic observation during first week
- Forcible exercise BID until healed
- Separate from mares (1 wk) so no pregnancies
- . Complications UNCOMMON but may be life threatening
- Hemorrhage, minor OK for several hours, if beyond 24 hrs m/ require surgery
- Prolapse of abdominal contents through inguinal ring (usually first few hours) . Replace bowel in scrotum, temporarily suture, fluids & ABs & refer to equine surgical facility
- Edema of scrotum common third or fourth day, usually due to inadequate exercise
- Acute wound infections & septicemia
- Cirrhous cord: chronic infection due to poor technique, inadequate exercise, or poor drainage
- Hydrocele in vaginal tunic due to inadequate resection
- "Proud cut" (see box)





"Open" castration: separating

testicular vessels from ductus deferens.

E. "Closed" castration: do not open parietal vaginal tunic, emasculate entire spermatic cord

"Proud cut"

- Persistent masculine behavior after castration
- "Proud cut": part of epididymis not removed during castration, also "cut proud'

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Maybe just a psychological problem



209

loxoid

Estrous cycle T 670; ER 473, 495; R-Y 64, 166; EM&S 982; E 1321; M 257; Pop 35-1/98

- Seasonal polyestrous (polycyclic)
- Jan/Feb-Aug/Sept. (occurs during particular season)
- Depends on length of daylight
- Begins when daylight +16 hr
- . Normally April or May
- . Feb. 15th, desirable start because want to birth on Jan 1st/artificial birthday (345 d gestation)
- . Artificial light started in mid Dec. to start cycling in Feb
- Transitional anestrus at end & beginning of season w/ little follicular activity occurring
- 21-22 d cycle (13-34 d) longer in early spring & late fall
- Number of cycles † in spring
- Peak in May-June, shortest cycles
 Minimum cycles in winter
- Estrus, receptivity of 7 days (4-8 d)
- Very irregular, need close attention
- Ovulation occurs 24-48 hours before end of estrus

Diestrus 15 days (6-25 d)



· When ovulation & breeding occurs

- Sperm leaves uterus in 15 min & into oviduct, survives long time in oviduct, in uterus 1 hr survival rate Once reach oviduct, propelled by swimming & contractions in oviduct
- . Go to site of fertilization within 6 hr (isthmus)
- Sperm survive 2-4 day, so want sperm in oviduct when ovulates
- Ova can be fertilized between 2-12 hours

Fertilization:

- Fertilized egg enters uterus 5-6th day
- . Lives off uterine milk (from uterine gland)
- Conceptus attaches on 14th day
- . In 1 horn just to side of bifurcation, usually in horn that did not contain the last pregnancy
 .. Every other year mares - 1 horn normal & supports a pregnancy,
- other horn abnormal (scar tissue) & won't support
- . "Maternal recognition of pregnancy" caused by attachment
- .. Prevents normal involution of corpus luteum by preventing luteinizing prostaglandin from en-

Events of pregnancy

- Initial corpus luteum maintains preg. for 80-90 d
- . CL secretes progesterones to maintain pg
- At 40 d surge of 2° follicles ovulations that help CL maintain preg.
- Endometrial cups (cells of fetal membranes invading the
- . 40 d of gestation produce ECG (equine gonadotropin). formerly PMSG (pregnant mare serum gonadotrophin)

- . Production lasts 120 days
- After 120 d fetal progesterone produced by fetal bone marrow (do not need ovaries to support preg.)
- Hippomanes or "colts tongues": semisolid, rubberlike, irregular, flattened masses commonly found floating in the aliantoic
- Cervical star: region of choricallantois in region of the internal opening of the cervix, devoid of villi

"Artificial birth date": Jan. 1st for all foals born that year

- Want to foal New Year's Day
- Gestation 345 days, so breed 15th of Feb.
- Economics of vet practice: get in foal early
- Of all horses bred, 50% will have live foals

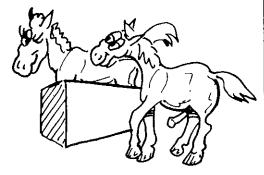
Gestation lengths

- Stallion to mare 340 d (foal) Thoroughbred 345 d (foal) · Mare to Jack 355 d (mule)
- Stallion to Jennie 350 d (hennie) Jack to Jennie 365 d (foal)
- Some have been recorded up to 375 d

Pregnancy diagnosis

ER 501; T 675; E 1317; EM&S 979

Early & accuracy important because of short breeding season



- Presumptive Dx of pregnancy
- 1. Absence of signs of estrum 18-22d after service
- 2. Teasing 18-22 d following service causes no visual signs of estrus
- Some show no signs of estrum whether conceived or not
- Tease regularly by stallion or aggressive or testosterone-primed
- Every 1-2 d to determine end of estrus
- Tease for 18-21 d (m/b 40) to see if conception has occurred
- 10-15% of mares will have signs of estrum 1st several months of pregnancy, usually weak & short, lasting a few days Due to normal development of follicles for 1st 90 d of gestation
- NOTE: postestral bleeding not seen in mares as it is in cattle 3. Pear shape of abdomen > 5 mo. - unreliable
- 4. External signs at end of gestation
- Distention of teats w/ colostrum: last 3-4 d of preg.
- "Waxing": 4-48 hr before foaling
- Vulva enlarged, flaccid & edematous prior to foaling



Rectal exam (see pg 212)

- Earliest & accurate Dx (experienced vet)
- Vesicle 20-30 d ventral bulge & thinning to side of uterine bifurcation
- Palpation of fetus through rectal wall: 90-120 d
- Ovaries of little value in pregnancy diagnosis



Biological tests for pregnancy

- MIP (Mare Immunological Pregnancy test)(pg 213)
- Accurate between 40-120 days of gestation
- Practical & almost as accurate as rectal exam
- Plasma & milk progesterone values

Ultrasound per rectum - diagnostic (see pg 213)

• 15-50 day diagnoses pregnancy, 95% accuracy

DDx - Pregnancy

- Endometrial cyst or lymph lacunae
- Distended bladder from 70-110 d
- Pneumovagina (pg 177)
- Enlarged or distended colon 90-120 d
- Pyometra assoc. w/endometritis & focal cystic degeneration
- Tumors of uterus & ovaries: uncommon
- Fetal maceration: uncommon (pg 221)
- Twin pregnancy: common (pg 176, 214).

Pregnancy Diagnosis - Days

20-30 d Rectal palpation of vesicle Palpable fetus 90-120 d Location of ovaries & > 7 mo. enlarged uterine artery

40-120 d MIP test (95%) 16-17 d Plasma & milk progesterone

values - 90%

Ultrasound per rectum (95%)

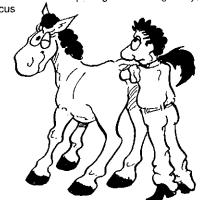


15-50 d



Rectal exam - Pregnancy Dx

- Earliest & most accurate Dx (experienced vet)
- Chorionic or blastodermic vesicle 20-30 d
- Ventral bulge & thinning to side of uterine bifurcation
- 60-90 d water-filled balloon extending into body of uterus
- Nonpregnant completely involuted horn
- Pliable, soft, flat & flaccid
- . Palpation of fetus through rectal wall: 90-120 d
- Ballot horn, difficult after 5-7 months
 Ballot horn & feel small, heavy, submerged, floating object
- Deep bodied, older mares difficult to palpate after 5-7 months
- After 7 mo.: palpate enlarged, whirring uterine artery or note ovaries low (8-10" below lumbar vertebrae)
- Ovaries of little value in pregnancy diagnosis
- Corpus luteum (CL) only palpable for few ds-wk after ovulation (deep
- Follicles continue to develop for 40-150 d after conception
- · Speculum if pregnant: cervix: small, w/ closed, puckered external opening covered w/ gummy, sticky



Rectal palpation - procedure

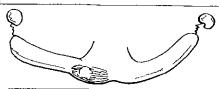
· Restraint more important than in cow

(nose twitch, tail twitch [forced firmly dorsally & cranially), elevate foreleg, breeding hobbles)

- Tranquilizers or sedation
- Handler & yet on left (near side)
- Stocks w/ rear rope low (mare m/ drop rear end) - Palpate around a stall partition
- Bandage tail & hold out of way (so tail hairs not dragged in)
- Liberal lubrication of rectum & rectal sleeve
- Rectum tighter & drier than cow's, bucket of soapy water applied w/ sponge - K-Y jelly or cheap 500 gm of Na carboxymethyl cellulose (Hercules®) in 8 gal. of water & 100 ml of Novalsan® (chlorhexidine)
- Easy to fatally tear rectum in horse (90% result in death): lawsuit Remove hand when peristalsis occurs (peristalsis stronger than in cow)
- Penetrate anus w/ a finger, then "cone" fingers - Exert ventral pressure when removing manure to milk out urine & pus which mi
- Pass hand as far forward & pull rectum back
- Gas distends rectum like drum, grab a peristaltic ring & pull back to release pressure at anus
- 1st locate bony pelvis
- Locate cervix: sweep pelvic brim
 - Pass arm forward to about the middle of your forearm
- Sweep fingers over floor of pelvis near the pelvic brim to locate cervix (longitudinal lump on midline)
- Option for inexperienced locate ovary 1st and follow down horns to cervix . Fibrous bean-shaped 4-6" (10-20 cm) cranial to pubis 2-4" (5-10 cm) ventral to lumbar vertebrae in nonpregnant mare
- Easier to reach left ovary w/ right hand & vice versa
- . Pass hand down proper ligament to horn of uterus & follow horn down to cervix
- Palpate bifurcation of uterus for vesicle (thinning of uterus)
- Pass palm of hand foreword from cervix while exerting ventral pressure & side to side motion (get impression of size & content of uterus) Cup uterine horns w/ fingers on ventral surface
- Sweep hand across front of both uterine horns to detect ventral enlargement with thin wall at junction of 1 horn & body

Rectal findings - pregnancy Dx

- Cervix firm & contracted
- Pregnant horn firm & tubular
- Thinning of uterine wall near bifurcation
 - Chorionic or blastodermic vesicle can be palpated between 20-30 d
- Nonpregnant, completely involuted horn
- Pliable, soft, flat & flaccid
- Horn changes
- 3-4 mo: horn lies on abdominal floor
- > 3 mo: impossible to retract heavy uterine
- 5-6 mo: uterus well forward & tension on broad ligament
- . Ovary 8-10" (20-25 cm) below lumbar vertebrae



Times to rectally palpate

- Experienced vet 30-40 ds of pregnancy
- Less experienced 40-50 ds
- Highly skilled 20-30 ds
- Easier in maiden mares or primgravidae & barren nonfoaling mares
- Harder in those conceiving on foal estrum when uterus not completely involuted
- Repeated early palpation didn't incr. abortion rate
- Do not directly palpate the amnionic vesicle or manually slip the membranes as in the cow

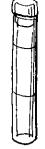
Biological Tests for Pregnancy

- MIP test (Mare Immunological Pregnancy test)
 - Gonadotrophic hormone (pregnant mare serum gonadotropin [PMSG] or equine chorionic gonadotropin [ECG] from endome-
- Accurate between 40-120 d of gestation
- Practical & almost as accurate as rectal exam-
- Indicated in nervous or vicious mares, small ponies (small rectum) or for inexperienced vets
- False negatives if serum taken at < 38 d or > 120-150 d
- False neg. if early embryonic death (EED) between 40-120 d (endometrial cups keep secreting)
- Procedure: 10 ml blood from jugular vein into sterile tube, allow to clot at room temperature. Remove serum & refrigerate. Do not over heat sample.
- Hemagglutination-inhibition test
- Highly accurate > 90%
- PMS gonadotropins inhibit agglutination of horse RBCs coated w/ PMS gonadotropin in presence of PMS gonadotropin antiserum

Plasma & milk progesterone values

- High levels of plasma progesterone 16-17 d after mare has gone out of estrus:
- . 90% accuracy to Dx pregnancy
- . Persistent CL w/out pregnancy common & gives a false positive
- Low levels or progesterone nonpregnancy
- Radioimmunoassav
- Help determine stage of estrus & Dx learly pregnacy
- However several days required
- Assess when out of estrus, good when no teaser stallion available or poorestrus behavior



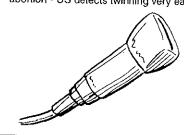


Early embryonic death (EED) common

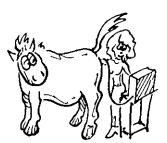
•2-20% between pregnancy diagnosis & 110 d Peaks at 25 & 30 d

Ultrasound, US 7 679

- Per rectum: diagnostic not harmful
- Visualizing the uterus through the rectum
- •15-50 d Dx pregnancy, 95% accuracy
- Repeat exam at 5-10 d intervals (reduces error, see vesicle grow)
- Don't attempt before 14 days
- 21 d: white echo of embryo in conceptus, migrates to more dors, position
- At 30 d: "blinking" heart beat = alive
- Diagnosis errors:
- Ovarian follicle (too close to ends of uterine horns)
- Endometrial cysts or lymphatic lacunae (older mare)
- Cystic structures (re-examine in 5 days, cysts don't change)
- Fetal death in 15% between 15-50 days
- Twinning (disastrous), most end in abortion - US detects twinning very early







REPRODUCTIVE SYSTEM

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Abortion

M8k 992: Mk 1142: I2M 252, 1549: T 705; R-Y 113; C4T 534; E 1347; EM&S 1066; M 26



Expulsion of dead or nonviable fetus

- 50% up to mid-gestation (160 d), EED (early embryonic death) (see pg 276)
- 3-4%(?) past mid-gestation
- · Lucky to Dx 1 in 10 causes of abortion, most causes unknown

• Rhinopneumonitis (herpes-1) #1 cause

- · Called infertility (pg 176) when unnoticed abortion between 20-90 d of gestation, most common time
- Older mares (over 18), higher abortion rate

· If fetus retained - autolyses (opaque cornea, soft mushy organs, gelatinous blood-tinged SQ & placenta

DDx (see pg 342 for complete list)

Twinning (pg 176)

- EHV-1 (Rhinopneumonitis)
 Uterine infec., Bact. or mycotic (ascending)
- Salmonellosis, Streptococcus zooepidei
- CEM, Mycotic abortions (Aspergillosis)
- Eq. viral arteritis (pg 216)
- Mares over 18 yr Malnourished mare (belw. 25-31 d)
- Management problem. (rough preg. exam < 40 d)

Diagnosis

• EHV-1 (Rhinopneumonilis) (pg 216)

- Strep. zooepidemicus (pg 180)

- CEM (contag. eq. metritis) (pg 179)

• EVA (Eq. viral arteritis) (pg 216)

Salmonellosis (pg 221)

· Bacterial & mycotic

• Infertile mares

DDx:

Stress (transport, working)

Ultrasound

Tx: Bacterial & mycotic Good breeding hygiene

- Tx genital diz before breeding

- Castick's to prevent pneumovagina ("windsucking")



Lab - send in blood sample & chilled aborted fetus + placenta - Dx 50-60%

Condition

Twinning

M8k 996; T 651, 705; EM&S 1059; E 129, 1319, 1349; M 267; ER 532; R-Y 137; C4T 539, 545; C3T 657



Disastrous to pregnancy

Tx: None highly successful

CS: Asymptomatic or EED or abortion

20-30% of all abortions

Disastrous to pregnancy

 Occurs commonly, usually natural abortion

- If not interrupted, 95% abort at 6-7 months of gestation

Facts/Cause

- 20-30% of all abortions
- Twins rarely produced alive
- Usually 1 in each horn, 1 small & healthy, 1 mummified or blob of tissue

1• Do nothing & keep evaluating for 16-18 d (6 d intervals) - If one does not grow, terminate it (don't allow it to reach 35-45 d when it is difficult to remove one w/out

Treatment expanded

< 35 days (golf ball-sized); once

Presentation/CS

EED (early embryonic death) or abortion

Asymptomatic

diagnosed, choices:

- Mycotic abortions (pg 217) jeopardizing the other Squeeze manually or surgically (causes trauma to uterine lining so give antiprostaglan-

dins [Bute or Banamine®] & progesterone for a few days to enhance pregnancy), Reexamine in a few days. Usually lose both, then re-breed next estrus Dilate cervix & douche uterus to cause abortion; re-breed next estrus

3. PGF2 alpha IM for luteolysis & breed next estrus

> 35 days (baseball-sized): if aborted, mare doesn't cycle for several months (endometrial cups producing ECG prevent estrus) Allow to go to term

Progesterone supplementation in last half of gestation m/ help

Treatment

NO Tx satisfactory (see box)

< 35 d m/ terminate one &</p> hope other goes to term or

Terminate both & re-breed at next estrus (dilate & douche)

> 35 d if terminate doesn't cycle for months

Supplement w/ progesterone

& hope goes to term

Prevention:

Repeat rectals of "twin-prone" mares & breed when only one mature follicle

If two mature tollicles:

Breed next estrus, or

Tap one follicle per vagina, or - Breed 12-18 hr after 1st follicle

has ovulated Consider embryo transfer in "twin-prone" mares

"Snots", EHV-1,

Dx: US

Rhinopneumonitis, Herpesvirus 1, Equine viral rhinopneumonitis,

Equine abortion virus

M8k 996, 1081; Mk 734, 526; IM 1398: I2M 1549: C4T 536: T 703 706, 711; R-Y 186; EM&S 1069; Pop 28-9/97



Tx: Time, Prevention

Vaccination

Respiratory virus - young & old

- #1 Diagnosed abortion in late pregnancy
- Abortion storms (last trimester)
- Maiden mares, Sporadic - Economic importance of diz is abortion, not Respiratory or CNS
- #1 infectious cause of abortion (1/4 of all diag. abortions)
- Herpes virus similar to IBR in cattle - Persistent virus in environment
- Contagious: annual outbreaks in foals (respiratory)
 Herd immunity determines episodes
- Transmission:
- Direct or indirect from nasal discharge, aborted fetuses, placenta or placental fluid
- Carrier animals always present
- Intro. to farm by newly acquired - Fetal infection 4-5 mo IP (crosses placenta,
- fetus immuno-incompetent, dies) Future breeding unaffected
- · Immunity on recovery not permanent

1° Respiratory virus - contagious

- · Foals (annual outbreaks)
- "Snots" runny nose (copious)
- Conjunctivitis
- Malaise, inappetence, edematous mandibular &/or retropharyngeal lymph nodes, constipation followed by diarrhea
- Dry cough in entire group
- Self limiting, wk 10 d
- M/ have dry cough for longer period 2° bacteria infections common mucopurulent
- nasal discharge
- Mares: asymptomatic
- Slight fever lasting a day ("didn't eat grain")
- Abortion storm 4-5 mo later Last trimester (7-11 mo)
- Neonatal death in a few d
 - M/b normal at birth then weak, respiratory distress, intractable diarrhea to death
- CNS (myeloencephalopathy) some animals Mild ataxia & posterior paresis to posterior paralysis & recumbency
 - Most frequent in mares shortly after foaling during an abortion outbreak (m/b also in barren mares, males, foals, etc.)

- "Snots": can't be DDx from other viral diz by CS
- Virus isolation nasopharyngeal swabs & citrated blood
- Serology of acute & convalescent sera
- Leukopenia (neutropenia & tymphopenia)

Abortion:

- . Hx of storm of "snots" on farm 4-5 months previously
- PM (postmortem) Fetus
- Amnionic fluid opaque (normally clear)
- Icteric
- Thoracic cavity yellow fluid
- Liver & lungs, white foci of necrosis
- Intranuclear inclusion bodies - diagnostic
- Pulmonary edema
- Histopathology: liver, lung, adrenal & lymphoreticular tissue
- Virus isolation & viral antigens in fetal tissue (lung, liver, adrenal & lymphoreticular)
- FA (fluorescent antibodies)
- · Fetal serology (serology of mare of little value)

"Snots":

 Self limiting (no economic advantage of treating all)

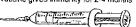
 Monitor: antibiotics for 2° bacteria, if temp. 102-3° F or mucopurulent discharge or pulmonary involvement

Abortion:

- Isolate
- Determine cause

Prevention

- Isolate new horses 3-4 wk
- Reduce stress (latent carriers)
- Segregate breeding & training horses
- Keep at farm for 3 weeks after resp. or abortion outbreak
- Vaccine see pg 12 (not 100%, almost mandatory due to constant movement of horses), give to all horses, not just
- MLV for resp. form, only lessening CS Foals 3, 4 & 12 mo
- Adults every 2-3 mo for performance horses & biannually for pleasure
- Brood mare, both KV & MLV aoproved for use in pregnant mares
- . Vaccinate 5th, 7th & 9th mo of gestation . Vaccinate in abortion
- storm (recommended) - Vaccine gives immunity for 2-4 months



- Immunity last couple of years, not permanent

215



"Snots" - self limiting

Abortion: Future breeding NOT affected



- Eq. influenza

DDx: "Snots"

 Other viral infec. - Eq viral arteritis

S. equi

#1 Cause of Abortion, Last Trimester CS: "Snots" then abortion storms Dx: Hx

DDx: Abortion

EVA (Eq. viral arteritis) (pg 216)

· Bacterial & mycotic

Twinning (pg 214)

Salmonellosis (pg 221) Strep. zooepidemicus (pg 180)

CEM (contag. eq. metritis) (pg 179) Mycotic abortions (pg 217)

Condition

Equine viral arteritis, EVA,

Equine typhoid, Epizootic cellulitis

M8k 997, 500; Mk 376, IM 527, 1401, 1397; I2M 1552; C4T 537; T 703, 706; R-Y 191; EM&S 1070; E 1347; Pop 4-2/98



EIA,

Equine

infectious

Swamp Fever

M8k 499; Mk 27;

IM 1074, 1401;

12M 1552; C4T

536: T 703, 707

anemia.

• See CIRC pg 143

- Togaviridae
- Damages smaller arteries . Edema due to leakage

Facts/Cause

- Transm. resp. or venereal
- Carrier stallion, NOT mares
- Abortion rare



Presentation/CS

Fever & leukopenia

Most mild

 Conjunctivitis (pinkeye) & Rhinitis Very sick, severe depression, quit eating

· Limb edema (esp. hindlimbs)

- ± Colic & diarrhea, photophobia, ventral edema, skin rash, pain in muscles joints, icterus
- Abortion: of those aborting, over 1/2 abort during illness
- 5-10 months of gestation

Rhino., EHV-1 (abortion months

later; PM - inclusion bodies) (pg 215)

Purpura hemorrhagica (pg 140)

Eq. influenza (abortion rare) (pg 109)



illness

of specimens

Postmortem

Virus isolation, serology, histopathology (nasopharyngeal & conjunctival swabs, cit rated blood samples & semen) Abortion:

Abortion during or following

Virus isolation, serology, histo-

pathology (placental & fetal fluids & other

- Few labs work w/ it, so ask about collection

- Fetus: no specific signs

Myocardial arteritis

fever: suspect EIA

until antibodies develop

for serum antibodies

positive

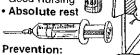
- No inclusion bodies

Diagnosis

 ABs & symptomatic therapy Good nursing

No specific Tx

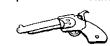




Treatment

Prevention:

- MLV vaccine closely regulated
- Economics, if no problem w/ EVA, don't vaccinate
- Seropositive stallions only bred to seropositive or vaccinated mares
- Don't ship mares to positive stallions
- All positive stallions, semen culture for shedding (m/ shed for only a couple of months, then never again)
- Euthanize positives or isolate for life



- No vaccination or specific Tx Stable fly control
- No Tx clears the carrier state
- · Isolate foals from infected mares until Dx free of infec. by disappearance of maternal antibodies
- Once positive can't transport
- Tested every 6 mo; if positive, put in quarantine or euthanized

If aged, will be positive occasionally

Nasal & Ocular discharge, edema Abortion during or following illness

- See CIRC pg 136
- Rarely see because controlled through testing, so:
- Rare cause of abortions
- Infected mares generally abort Transmission: Hematogenous, needles, bloodsucking flies
- Intrautero
- Retrovirus: lifetime infection

Rare cause controlled

CS: Intermittent fever + Wt. loss

Dx: Coggin's Test

Yx: Euthanize of isolate for life



- Intermittent fever Depression
- Progressive weakness Weight loss

• EIA (pg 216)

DDx:

- Progressive or transitory anemia Usually inapparent, but m/ cause death
- Abortions any stage of gestation, especially during
- febrile episode - Don't abort unless very ill
- · May give birth to normal, live free-of-infection foal





- Autolyzed fetus frequently

Culture & histopath, of fetal

tissues (especially abdominal contents)

FA of impression smears or sections of pla-

Serology, many labs don't do Salm. serology

Difficult to get titer from mare - no bacterial

- Placentitis or inflamed fetal

History of wt. loss & periodic

Coggin's test (agar immunodiffusion)

- Foals nursing infected dams temporarily

Recently infected horses negative for 1 wk



Retained placenta

PM (postmortem)

cental or fetal tissue

tissues

Salmonella abortion

M8k 997: Mk 184; IM 1413, 247, 819; I2M 1560; R-Y 183

Late gestation, PH CS: Diarrhea, RP Hygiene

Streptococcal abortion

M8k 997; IM 1413; I2M 1560; T 701, 705; R-Y 183; E 1348

- Turn of century S. abortus-equi, only 2
- Now associated w/ S. typhimurium or other nonhost specific spp.
- Contagious outbreaks recognized in Wash., Ill. recently; not in other
- Similar to intestinal form of Salm.
- · Carriers feces, milk, or placental & uterine fluids
- Public health/PH (handle infected tissue w/ care) (zoonosis)

Strep. zooepidemicus

- #1 bacterial abortion

· Normal flora of genitalia

Ascending infection up

(tolerated in low numbers)

Vulvar abnormalities

predispose ("windsuckers")

E. coli, or Klebsiella.

cervix

#1 Abortion bacteria, normal flora

Tx: Intrauterine Abs, Caslick's

- Abortions Late in gestation
- Retained placenta (RP) Diarrhea
- Fever
- Vaginal discharge (uterine infec.) · Metritis rare, fatal complication



- Sporadic abortions
- Pyometra
- Asymptomatic





titer usually

- Placentitis (concentrated around cervical star)
- Fetal inflammation
- Histopathology
- Culture from fetus
- Isolated from most parturient mares (so not diagnostic)



Prevention:

- Mares w/recurrent infections should be bred at first estrus after Tx
- Artificial insemination safer
- Caslick's for vulvar abnormalities

Mycotic abortions. Mycotic placentitis

M8k 997; IM 1416, 1398; I2M 1563; C4T 537; T 701; R-Y 193; E 1349

Late in gestation Leathery placenta

- Aspergillus fumigatus
- #1 fungal abortion in mare & COW (Mucor, Allescheria, Coccidioides, Histoplasma, Candida, Cryptococcus)
- Sporadic Abortion in later 1/2 of preg. (often near term)
- 1 or 2 animals in herd Winter more common
- 5-30% of infectious abortions
- Has recurred in subsequent preg.
- Transmission through cervix usually
- Abortion late in gestation



- Abortion late in gestation Postmortem:
 - 6 - Thick, leathery placenta (placentitis), especially chorio allantois (maternal side)
 - Fetal bronchopneumonia
 - Fungal culture of placenta
 - Histopath., tissue smear or Section (morphology or IF staining)

Prevention:

- Carriers cultured
- Control w/ hygiene & avoid carrier animals
- Isolate aborting mares
- · Remove fetus, placenta & contaminated material from premises (handle carefully, zoonosis)



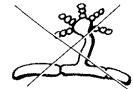
- · Future fertilitym/b affected depending on bacteria, uterine infections also
- Easy to treat, most ABs - IM penicillin
- Intrauterine infusions daily

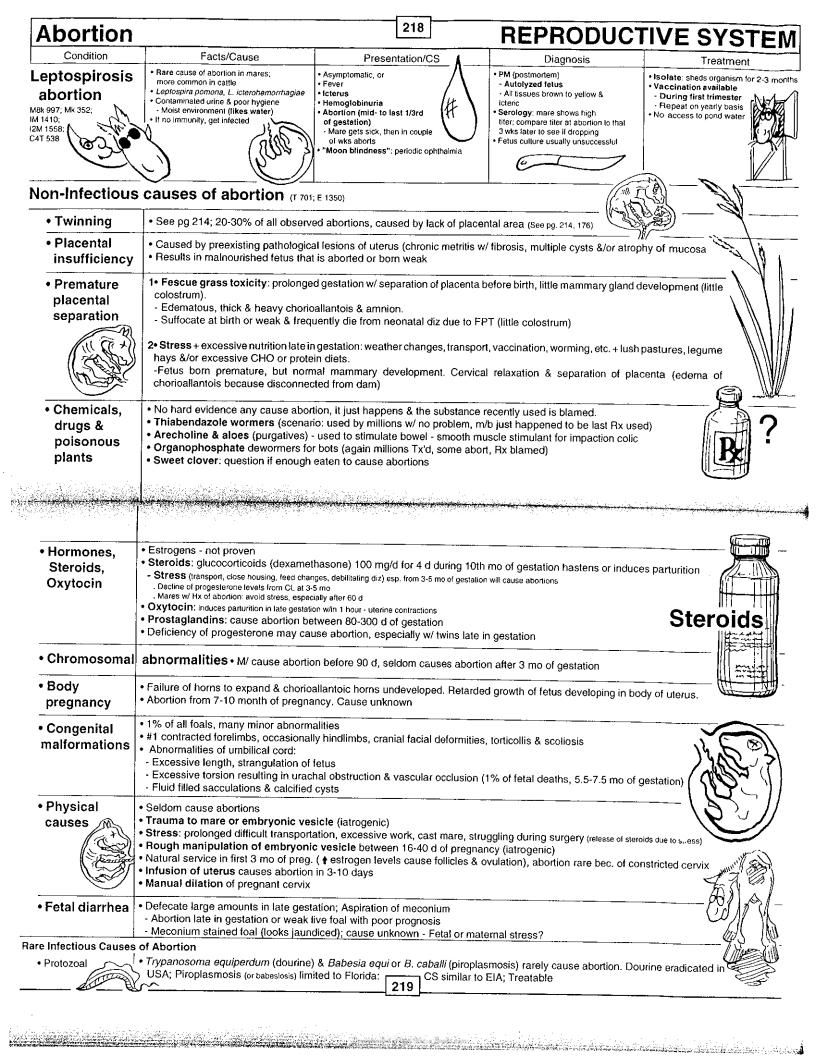


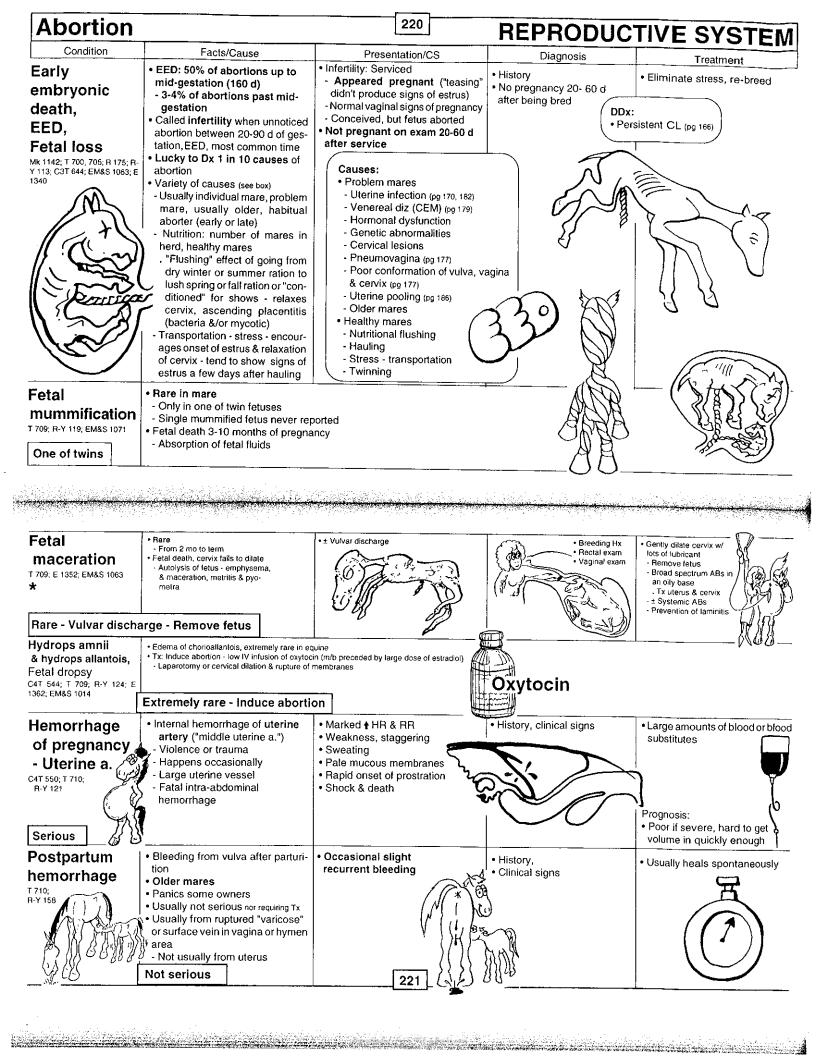


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· Reduce exposure to fungus







Pregnancy Termination

REPRODUCTIVE SYSTEM

Pregnancy termination

T 715; R-Y 177; E 1351

- Indications
- Mismating
- Purchased pregnant mare w/ unwanted fetus
- Dx of twin pregnancy
- Research
- Can interrupt (terminate) at any stage, the earlier the better



Repositol

Interruption Times & Methods			
1-3 d	Mega dose estradiol		
6-45 d	Prostaglandins or flushing		
	Saline infusion		
80 - 150 d	Cervical dilatation		
	Ovariectomy		
	Manual removal or induce		
340 d	Oxytocin (for live foal)		

- 1-3 days: Mega dose estradiol injections
- Breed w/in last 12-72 hr or still in estrus
- Repositol® (diethylstilbestrol)
- (1 injection of 100-200 mg) or

Estradiol (several injections of 10-20 mg every 36-48 hrs)

- Relaxes cervix
- Probably will prevent pregnancy
- Should come into estrus, if doesn't or is pregnant:
- 6-45 days: Prostaglandins or flushing after breeding or end of estrus

- Let mare go out of estrus for 6 days as need mature CL
- Interrupts corpus luteum, stops progesterone production which terminates pregnancy
- . Prostaglandins IM (easiest).
- CL must be mature (6 d), PGF2 alpha (2.5-5.0 mg tM at 12 hr intervals) or Prostaglandin Fluprostenoi® (potent PgF2a analog, 2 mg IM)
- Infusion of uterus w/ 1 liter of saline irritates lining of uterus to release prostaglanding
- 3-4 d return to estrus
 - Observe for next onset of estrus Failure: if 10 -12 d not returned to estrus, uncommon to fail
 - Repeat treatment (14 d interval between Tx)

• 35 - 80 days: Saline infusion

- Equine fetus attached, endometrial cups forming after 35 days
- No PGF2 alpha once endometrial cups formed because PMSG (pregnant mare serum gonadotropin) protects accessory corpus luteae formed
- Saline infusion, 500-2000 ml (add 2 million IU of Na penicillin
- to flushing solution)

 Pre-Tx w/ 10 mg of estradiol 24 hrs before flushing to cause cervical relaxation
- Estradiol not necessary usually because cervix dilates spontaneously w/in 4 hrs of infusion
- Expulsion usually w/in 24 hrs (m/ occur immediately)
- If longer than 24 hrs, repeat flushing
- . Repeat every 24 hrs until terminated

• 80 - 150 d: Cervical dilation

- Penetrate cervical seal w/ 1 finger & gently dilate cervix for 10-20 min
- Ruptures allantochorion
- Gently remove fetus manually Pre-Tx w/ estradiol 24 hrs earlier
- will facilitate by relaxation of cervix

- Saline not used after 90 days!
- (kills, but fetus too large to pass easily)

 If not manually removed after cervical dilation, abortion usually occurs in 2-7 d (more reliable to manually remove)
- < 4 mo: Ovariectomy will usually cause abortion in 4-6 days, but permanent sterility
- > 4 mo: Manual removal (if large) or induce labor w/ oxytocin

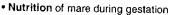
Induced parturition

- C4T 546, 548; R-Y 144; EM&S 997, E 1355
- 340 d: Induce parturition for live foal
- Oxytocin (60-100 IU)
- induces parturition in late pregnancy w/in 1 hr causes uterine contractions
- Strict criteria to give oxytocin:
- 1. At least 340 d gestation
- 2. Need milk in udder
- 3. Need cervix starting to relax (2 fingers easily introduced into uterus)
- If have all 3, then safe to induce
- Do NOT do this as a convenience
- Don't need to prime mare with estrogen as in other species

Advise against very late abortions

- Don't dilate if want to retain pregnancy they will
- 120 d before next estrus once endometrial. cups have formed (too late for early foal)

Late Gestation Care



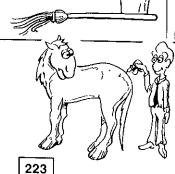
- Hay usually fed free choice during entire gestation & supplemented as energy demands increase
- Maintenance diet up to last third of gestation (so don't lose weight)
- Last 1/3rd of gestation: increase energy
- . Sweet feeds of corn & molasses
- . Can't eat enough hay to maintain weight in this stage of gestation
- Last 5-6 wk of gestation: greatest use of energy due to starting lactation
- Observation a must
- Dewormed 30 d prior to due date
- Vaccinations:
- Boosters, tetanus, influenza, encephalitis (up to you)
- Rhinopneumonitis (2 forms: one resp., one abortion)
- . Pneumabort® a killed virus safe during preg. . Schedule on bottle, but 5th, 7th & 9th mos of gestation (period when virus can cross placenta & kill fetus)
- Hypocalcemia in only hard working horses, not so much in milk production, i.e., brood mares

Exam of parturient mare

C4T 547; IM 253; T 694

- Proper restraint (see box)
- Varies w/ temperament of mare
- . Twitch, sideline or hobbles m/ help
- No stocks! Mares attempt to lay down when stimulated by manipulation of genitals
- Chemical restraint m/b necessary
- Acepromazine or Xylazine/ketamine

- IV or general anesthesia for longer periods
- Epidural anesthesia to relieve pain of dystocia & stop abdominal straining
- . Allows thorough exam, repulsion & mutation of
- Clean environment w/ good footing
- . Bandage tail & tie to halter (prevents injury to tail if mare
- Sanitation, sanitation, sanitation!!! when entering genital tract
 - Surgically scrub buttocks & perineal region & operator's hands
- Adequate lubrication:
- Methylcellulose only lasts a short time
- Petroleum preferred for mutation & fetolomy Frequent reapplication necessary
- Examination of birth canal thorough!
- Previous trauma
- Adequacy of relaxation & size
- Presentation, position & posture of fetus
- Determine if fetus is alive
- Form a plan for delivery & immediate postpartum care



Foaling:

- Clean quiet area to which mare has adapted
- Pasture is better than stall, but usually can't observe in pasture
- Caslick's suture must be removed within week or two, but if necessary just before foaling to prevent tearing





Chemical restraint:

- Acepromazine maleate (0.04-0.08 mg/kg IV)
- Xylazine (Rompun®) m/ still kick
- Xylazine/ketamine: Xylazine (1.0 mg/kg IV) tollowed by ketamine (2.0 mg/kg IV) muscle relaxation & analgesia for short periods (10-20 min)
- IV or general anesthesia for longer periods
- · Epidural (see box below)

Epidural anesthesia to relieve plain of dystocia & stop

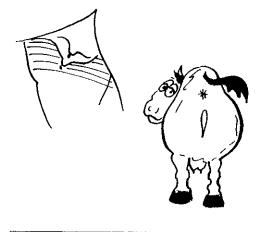
- Allows thorough exam & repulsion & mutation of fetus
 - 18 gauge, 3" spinal needle w/ stylet
 - Enter epidural space between 1st & 2nd caudal vertebrae
 - 2% lidocaine (6-8 ml for 1000# mare [450 kg]) will relieve straining w/out causing collapse of rear end



Foaling - Warning Signs

IM 253; Pop 45-2/98

- 1. Large bellies (DDx from those that had lot of babies or hay belly)
- 2. Fetal movement when drinking cold water or eating
- 3. Mammary gland development
 - Fills 3 weeks before expected date (most noticeable for 2nd or more foals)
 - Last day before foaling, udder tense, teats distended
 - Foaling day: "waxing": drying of milk secretion on udder (usually will foal that evening)
- Night foaling: most, usually between 7:00 PM 1:00 AM, isolate themselves to foal (tremendous control over time
- Put mare in foaling stall 2-3 wks prior (acclimatize)



Three Stages of Parturition

C4T 547; R-Y 68, 141; EM&S 994, E 1352; T 693

Stage 1: Positioning of fetus, aligning it w/ birth

- · Fetus extends head & forelimbs & rotates into dorsosacral position
- CS: Vary from mild discomfort (restlessness, nervousness, uterine contractions) to sweating & marked colic
 - 2-4 hr for cervical dilation & end of 1st stage
 - . M/b delayed for hours to days
 - . Normal delivery m/b preceded by several bouts of false labor
 - . 80% foal in evening after dark to midnight

Stage 2: Delivery of foal

- "Water breaks" to aid expulsion of foal
- Explosive in mare, usually 20 min to deliver
- Rupture of chorioallantois as mare generally lays down, putting pressure on it, yellow to chocolate fluid
- Foal enters birth canal in slightly dorsoiliac position
- . Shoulders enter successively so hoof of one forelimb at level of fetlock of other
- . Head lies between carpal joints
- Amnionic membrane protrudes out vulva w/in 5 min of "water breaking"
- Abdominal straining begins shortly after 2nd stage starts, very strong contractions
- Placenta starts to separate
- 30-45 min maximum time for foal to be born &
- Placenta separates rapidly from endometrium, culting off

oxygen supply to foal

- If not delivered in 45 min, death of fetus from asphyxiation

Stage 3: Expulsion of fetal membranes

- 30 min 3 hours
- Myometrium continues to contract to expel membranes & involute uterus
- Mare lies down & acts uneasy
- Usually membranes pass easily
- > 3-6 hr considered retained placenta (RP)
- RP starts to deteriorate & rot; m/ absorb breakdown products, develops toxic septicemia or circulation disturbances in feet due to histamine absorption
- . Postpartum laminitis, most notably in draft horses
- . Potentially a dangerous situation (more dangerous
- Normal vaginal discharge for about 7 days, usually darkchocolate



Eutocia - Normal parturition

C4T 547; EM&S 994, E 1352

- Good news! Foaling generally normal in mare (as apposed to cow)
- Try to leave mare alone and let her give natural birth
- Foal born wrapped in amnion
- Then wiggles to rupture amnion
- Clear amnion from nostrils
- Foal born attached to umbilicus, pulsating to transfer blood to foal
- Don't try to remove umbilious from foal
- . Movement of foal will separate it normally
- . Do NOT ligate cord in domestic animals due to contamination
- . Umbilical cord will dry up
- . If tears abruptly, then it is a problem to deal with
- Dip naval in 2% tincture of iodine to disinfect area
- Naval III: ascending infection up umbilical cord
- . 2-3 days after birth foal becomes very sick (systemic diz often settles in joints, hence name "joint ill"). Very enlarged, fluid-filled joints
- Relatively long cord, very twisted usually
- . Occasional twisting to occlusion, but rare
- . Blamed for stillborns



Dystocia C4T 552; R-Y 146; E 1356, EM&S 1015; M 261

TRUE EMERGENCY in MARE

- 45 min
- Delivery in which something goes wrong
- When either of 1st or 2 stages of parturition are prolonged or not progressive
- Need veterinary assistance immediately if any abnormalities of parturition
- More urgent than other species because when foal breaks free, placenta does also, no air supply
- Examination of birth canal & fetus indicated
- Most dystocias fetal in origin
- Few maternal (herniated & no abdominal push, overwt. mare & no exercise, tendency to give up, exhaust early)
 - Oversized fetus, rare in mare (big problem in cattle)
- Cleanliness & lubrication necessary
- If contaminate uterus, may save foal, but destroy breeding possibility of dam
- Need lots of lubrication: if long time, lose all fluids, not unusual to put lots of lubrication back into uterus
- Methylcellulose in mare (cheap & water soluble)
- Treatment of dystocia
- 1• Mutation: reposition fetus
- 2. Traction: use to deliver, best for dam & foal

If can't mutate enough to use traction, can:

- 3. C-section: deliver live baby
- 4. Fetotomy: cut in pieces & deliver pieces

Causes of Dystocia

- Common causes
- Malpresentation (pg 228)
- Malposition (pg 228)
- Maiposture (pg 228)
- Torticollis
- Arthrogryposis
- Less common causes
- Pre-term parturition
- Abortion (pg 214)
- Twinning (pg 214)
- Vaginal/vulvar obstruction
- (tumor, callus, hematoma, abscess)
- Pelvic deformities (fracture, injury)
- Uncommon causes
- Fetopelvic disproportion
- Uterine torsion (pg 232)
- Uterine dorsoretroflexion - Uterine inertia
- Vaginal prolapse
- Ruptured prepublic tendon (pg 233)
- Abdominal ventr. hernias
 Prolonged gestation
- Induction of parturition (pg 222)
- Premature separation of chorioallantois from endometrium
 Congenital defects

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- Hydrops of fetal membranes (pg 221)

Most dystocias fetal in origin

Obstetric Operations

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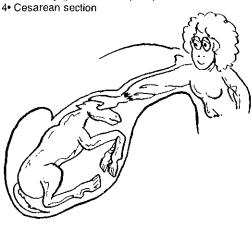
REPRODUCTIVE SYSTEM

Obstetric operations

C4T 555; T 693

Obstetric operations for resolution of dystocia:

- 1. Mutation of abnormal presentation, position or posture
- 2. Forced extraction
- 3. Fetotomy (partial or complete)



1 • Mutation T 694; R-Y 147

- Normal presentation; anterior presentation, dorsosacral or slightly dorsoilial position w/ head, neck & forelimbs extended
- Other presentations, positions & postures first

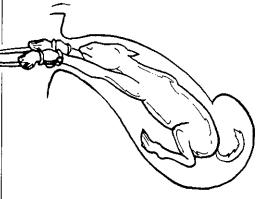
corrected by mutation if possible

- Obstruction of fetal head, thorax or pelvis less common than in cow
- "Hip-lock" uncommon in equine so rotation while extracting not necessary
- Oversized fetus uncommon
- Do NOT get arm between fetus & pelvis or m/break your arm
- Preparation:
- Well lubricated birth canal & fetus
- Epidural to stop straining
- Mare standing preferable (unless long time)
- . Most not prone to kick when in foal, generally patient
- . Want to lay down
- . If fractious, be careful
- Never get between horse & hard spot (wall, etc.)
- Work in open area
- Extreme care not to rupture uterus
- Do NOT attempt if uterine wall tightly contracted around fetus

Procedure:

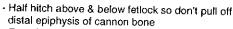
- 1st: repulse fetus out of mare's pelvis into uterine cavity (more room)
- Attempt to rotate fetus into a dorsosacral position
- Malposture of fetal extremities (most common cause of dystocia in mare)
 - . Repulse as far cranially as possible
 - . Flexion of extremity
 - .. Repulse proximal end
 - .. Rotate middle portion laterally

- .. Traction applied to distal end
- .. By operator if room for both hands, or
- .. By assistant pulling on obstetric chain or snare
- .. Protect birth canal from trauma
 - ... Cup fetal hoof w/ hand
- If longer than 15 min use alternate form of delivery - cesarean section

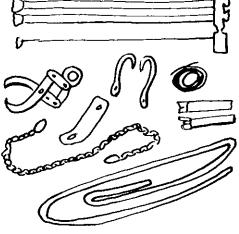


2 • Forced extraction (Traction) C4T 559; R-Y 150; T 695

- Mutate fetus in dorsosacral position w/ head, neck & forelimbs extended
- Place traction snares or chains (OB)

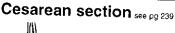


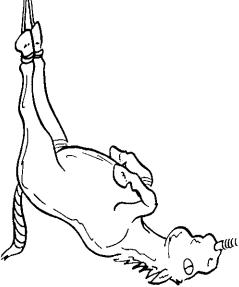
- Eye of snare on dorsal aspect
- Only direct head w/ traction devices, do not pull w/ them (head bones not as well developed as in the cow); don't use eye
- Moderated amount of traction (only 2 or 3 people pulling)
- Do not use mechanical aids!
- Simultaneous traction w/ abdominal press of mare
- Want foal to bend over pelvis
- Should be easy
- If can't do rapidly, reexamine fetus & correct impediment; do a C-section



3 • Fetotomy T 695; R-Y 150; M 261

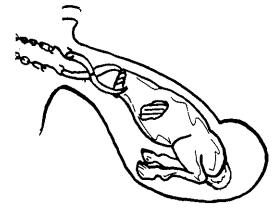
- Reduction of fetal size by amputation, partial or complete
- Requires expertise
- Partial fetotomy usually enough, followed by
- extraction of the remains of fetus
- Restraint: epidural anesthesia/general anesthesia
- Nonirritating lubricants
- If done w/in 12 hr of onset of labor, Px of mare's survival good





Sequelae to fetotomy

- Retained placenta
- Metritis
- Peritonitis
- Laminitis
- Vaginal & cervical laceration
- Delayed uterine involution
- Nerve paralysis (facial & radial)



Orientation of Fetus

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REPRODUCTIVE SYSTEM

Orientation of fetus: presentation, position & posture C4T 555

- Presentation: relationship of long axis of fetus to dam, & part of fetus approaching pelvis
- Longitudinal presentation: fetal spine parallel to dam's spine
- . Anterior longitudinal: head at cervix, normal in mare, either way in cow
- . Posterior longitudinal: tail towards pelvis (breech)
- Transverse presentation: fetal spine perpendicular to mare's spine
- . Ventral or dorsal: depends on side facing pelvis
- Position: relationship of dorsum of fetus to the four quadrants of mare's pelvis: sacrum, left or right, ileal or pubis
- Dorsopubic (upside down)
- Dorsoilial (left or right)
- Dorsosacral (or slightly dorsoilial) preferred
- Posture: relationship of fetal extremities to its body
- Normal: both front feet at level of head w/ hind feet trailing behind
- Problem w/ abnormal posture: takes up more pelvic room in the dam

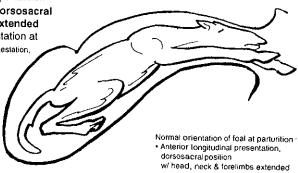
Hydrocephalus

- Dystocia due to fetal monsters
 Not common in mare
- Correction
- If bones soft incise soft part of head & release fluid
- If bones hard partial fetotomy of enlarged part of head

Normal orientation of foal at parturition

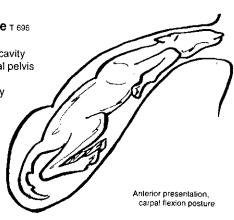
Anterior longitudinal presentation, dorsosacral position w/ head, neck & forelimbs extended

 Only 1 in 500 foals in posterior presentation at delivery (50% in posterior presentation in early gestation, rotate by 6.5 months)



Abnormal Orientations of Foal Anterior presentation, carpal flexion posture T 696

- Unilateral or bilateral
- "Engaged carpal flexion": head & flexed limb(s) in pelvic cavity
 Disengaged carpal flexion" flexed limb cran. to maternal pelvis
- Correction
- Repulse affected limb & fetus cranially into uterine cavity
- Grasp metacarpus just above the fetlock
- Lift limb dorsally, flexing elbow & shoulder
- Lift fetlock above the mare's pubis
- Cup hoof & pull into pelvic inlet
- Snare around fetlock m/ be useful, lift carpus & pull
- "Engaged" m/ deliver, if enough room, w/o repulsion
- Traction on head, extended limb & flexed limb & repulsion of shoulder (extend it)
- · Dead fetus: amputate just distal to carpus
- · If not corrected rapidly & fetus alive: C-section indicated



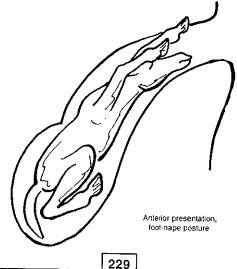
Anterior presentation, shoulder flexed posture

Anterior presentation, shoulder flexed posture

- Forelimb lies alongside or under fetal abdomen
- Unilateral or bilateral, engaged or disengaged
- Correction:
- Attempt to turn into a carpal flexed posture
- . Grasp radius & pull toward pelvis
- . Difficult because of long limbs
- . Traction snare m/b helpful, but difficult to position
- Once in carpal flexed posture, mutate
- Forced extraction w/o correction only if fetus small or pelvis large (lubrication & forced extraction of extended limb & head), expect fetal trauma
- Fetotomy
- Amputate head 1st
- Incise over dorsal border of scapula
 - Place gigli wire in incision & around limb, between scapula & thorax
- In bilateral cases m/ have to only remove 1 limb

Anterior presentation, foot-nape posture

- 1 or both limbs lie on top of head
- Sequelae: rupture of vagina, rectovaginal fistula, 3rd
 degree perineal laceration
- Immediately correct this posture
- Correction: lift fetal head & repel, while assistant pulls downward & laterally on snares attached to fetlocks
 Forced extraction
- If hooves penetrated vaginal or rectal walls, attempt to replace in vagina prior to correction of posture
- Rectovaginal fistula: incise mare's perineum to make 3rd degree perineal laceration
- Fetotomy of limbs in vagina or rectum; amputation of head m/b necessary to reach limbs



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Anterior presentation, shoulder-elbow flexion posture

- Relatively rare
- Shoulder & elbow flexed, head lies next to fetlock instead of carpus
- Correction
- Repel head while pulling on limb
- Fetotomy usually not necessary

 Anterior presentation, "dog-sitting"

Anterior presentation, "dog-sitting" posture

- (ventrovertical presentation)
- Hip joints flexed
- Cranial part of fetus delivered OK, but stops after thorax emerges
- Forced extraction doesn't work & m/ drive hind hooves through uterus
- Correction difficult: rotate fetus to dislodge digits
- Try to repel hindlimbs (long arm needed)
- Fetotomy:
 - Cut fetus transversely behind last rib
 - Deliver hindquarters or cut in half longitudinally

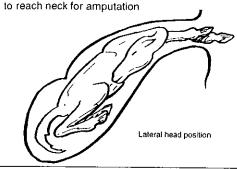
Abnormal Orientation of Fetus

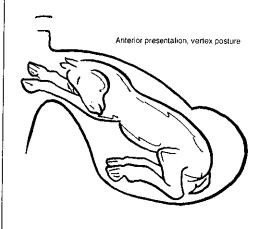
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REPRODUCTIVE SYSTEM

Anterior presentation, lateral dead posture

- · Common cause of dystocia in mare
- DDx from "wryneck" (congenital curvature of cervical vertebrae)
- Correction difficult because of long neck
- If small m/ extract w/o correction, but unlikely
- Repel into uterus
- Lay mare on side w/ fetal head uppermost
- Grasp fetal mouth, move head toward midline of
- Grasp muzzle or lower jaw or orbital grooves w/ 1 hand & press on side of head w/ palm of other hand & pull head into pelvic cavity or
- Mandibular snare (soft rope) pulled while pushing w/ other hand on head
- Only use moderate traction
- Partial fetotomy
- Indicated if "wryneck", fetal head beyond reach or can't be rapidly corrected
- Amputate head & neck at point of deviation & remove
- If small pelvis, amputate limb opposite neck flexure



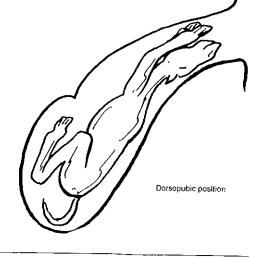


Anterior presentation, nape, vertex & breast-head posture

- Vertex posture: bridge of muzzle against mare's pubis w/ poll into pelvic cavity
- Nape posture: displaced further than vertex w/ nape of neck in pelvis
- Breast-head posture: muzzle against sternum
- Correction: traction on mandibular snare (soft rope) around lower jaw, while
- Repel poll
- Spontaneous delivery of nape posture possible if mare normal to large in size
- Fetotomy if fetus dead
- · C-section if fetus living & can't rapidly reposition

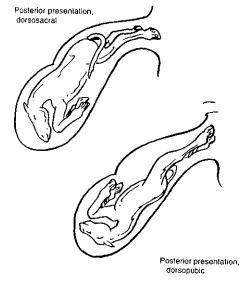
Dorsoilial or dorsopubic position

- Failure of normal rotation to dorsosacral position
- Unless small, must reposition before delivery
- Differentiate from uterine torsion
- Correction
- Place obstetric chains on both fetal limbs
- Secure head w/ mandibular snare or blunt eye
- Grasp humerus near shoulder & lift upward, while:
- Assistant applies traction on dorsal limb downward & medially
- Fetus must be freely movable or m/ produce uterine torsion



Posterior presentation. dorsosacra!

- OB chains half hitch above & below fetlock
- Forced extraction (worry about compression of umbilicus as goes through birth canal)
- Little time to get out once compressed

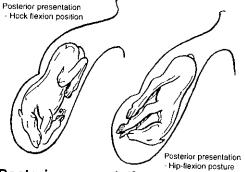


Posterior presentation, dorsopubic

- Correction: rotated to dorsosacral presentation OB chains - half hitch above, half hitch below
- Forced extraction

Posterior presentation, hock flexed posture

- Unilateral or bilateral, engaged or disengaged
- Posterior presentation w/ hindlimbs flexed at tarsus
- Correction: Grasp lateral aspect of metatarsus & displace hockupward & forward
- Pull hoof into pelvis
- Or snare on fetlock, pulling while operator repulses foal forward
- Amputation distal to hock if can't be repositioned

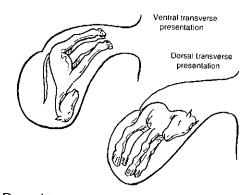


Posterior presentation. hip-flexed posture

- Posterior presentation w/ flexion of hip
- Rear limbs under body of fetus
- Correction difficult: mare in lateral recumbency, affected limb uppermost. Attempt to mutate hip-flexion to hock-flexion which is then further mutated as above
- Cesarean section
- Percutaneous amputation of affected limb(s)

Ventral transverse presentation

- Fetal spine perpendicular to dam's, 4 feet toward
- DDx from twin pregnancy
- M/b complicated by rotation of uterus
- Consider cesarean section as correction difficult
- Correction unlikely to be successful
- Convert to posterior longitudinal presentation, dorsosacral position Repulsion rotation
- Amputate forelimbs (m/b necessary to amputate hindlimb below hock to reach forelimbs)
- Repel forepart of fetus & apply traction & rotalion to hindlimbs



Dorsal transverse presentation

- Less common than ventral transverse, either transverse presentation rare 0.1% of all pregnancies
- Fetal spine perpendicular to dam's spine, w/ legs toward dam's head
- Cesarean section best bet
- Mutate into posterior dorsosacral presentation
- Repel forepart while hindparts extracted & rotated
- Mulation & fetolomy
 - Attempt to saw around lumbar areas & deliver cranial & caudal parts of fetus

Rupture of prepubic tendon,

Prepubic desmorrhexis H 229; R-Y 122; T 709; E 1352; S 418

Extensive unilateral

ventral hernias

C4T 543; R 229

Abnormal

C4T 543; T 699

pelvis

in light mares

Less common in cow because of subpublic tendon which mare doesn't

Associated w/ excessive uterine weight or twinning or hydrops amnii

 Last 2 months of gestation

M/b associated w/ trauma

 Often preceded by ventral edema (udder to xiphoid)

- 4-6" thick, tense edema

- Move slowly, refuse to lie down

Rupture

- Severe distress

- Pain - sweating

- ♦ RR, weak pulse, m/b internal hemorrhage

± Shock

- Collapse & death m/b w/in min to hours

History

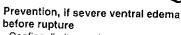
Clinical signs (CS)

• Rupture:

- Elective cesarean section near term

 Repeated vaginal exam & induced parturition w/ extraction when cervix dilates

 Euthanasia of mare following delivery often indicated



Confine, limit exercise

Avoid bulky feed

- Light, laxative concentrate feed

- Canvas girdle w/ tightening straps

Watch closely for parturition to assist

 Induce parturition late in gestation w/ oxytocin & assist delivery

Prognosis:

 Poor in all cases, can't apply abdominal pressure during 2nd stage of parturition

 If survive usually euthanized (usefulness gone & unsightly)

Draft mares last 2 mo of gestation CS: Ventral edema then rupture Dx: Hx, CS Tx: Euthanize; Px: poor

· Rare in mare, rupture of abdominal tunic

 Late in gestation Treat like ruptured prepubic tendon

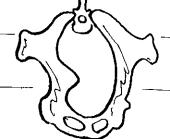
· Fractures or exostoses of pelvis · Stenosis of birth canal, rare

· Elective cesarean section if diagnosed before labor

Vaginal Entrance of urinary bladder into uterus cystocele

- Eversion through urethra or prolapse through rupture in vagina

Replace bladder & suture vaginal wound before extraction of fetus



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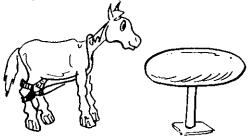
REPRODUCTIVE SYSTEM

Artificial insemination

C4T 574; R-Y 36; T 652, 640; E 1393; Pop 19-2/98

Semen collection - jump mare or phantom

- · Use jump mare exhibiting estrus
- Small, stocky mare
- Ovariectomized so they will allow covering at will
- M/ show signs of estrus because of rise in production of adrenal estrogens
- Wrap tail & clean perineal region
- Adequately restrain mare (twitch, breeding harness or breeding chute, California hobbles [allow walking] w/ quick release knots)



- Phantom mare or dummy
- Most stallions can be trained to mount a phantom mare
- Texas phantoms: 2 oil drums welded together & mounted on steel pipe
- . Pad w/ several layers of plastic foam & covered w/ reinforced plastic, canvas or leather
- Stand estrus mare next to phantom or cover its rear

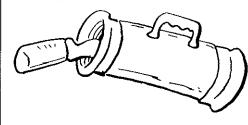
end w/ urine from an estrus mare

 Let young stallion watch an expert stallion mount phantom

Artificial vagina (AV), Colorado model,

Missouri model or Japanese model

- · Ideal method for collecting
- All consist of hollow tube lined w/ rubber that can be filled w/ water
- Stallions show individual idiosyncrasies, some like one model better than another; some like them tight, some loose



Procedure: collection

- · Fill artificial vagina w/ warm water
- Proper temperature (same as mare's body temp.)
- Penis should penetrate lubricated AV w/o excessive resistance
- Lubricate inside of AV w/ sterile nonspermicidal lubricant just prior to use
- Warm collection bottle to body temp. & attach to AV just before collection

Preparation of stallion

- Show collecting area, then bring in when shows some interest
- Expose stallion to jump mare or phantom until erection attained
- Wash penis of smegma (cotton or paper towels (water alone or w/ mild, nonirritating soap)
- Rinse thoroughly if soap used (harmful to sperm)
- Usually stallions permit this once accustomed & find it stimulating
- Collection area large enough & with good footing
- Restrain erect stallion for a short period prior to mounting (heighten excitement)
- Allow stallion w/ erection to mount phantom or estrus mare
- Stallion handler & collector on left side of stallion
- Direct penis gently to left side of mare's or phantom's buttocks & into artificial vagina (AV)
- Hold AV w/ distal end slightly elevated (like normal vagina)
- Once thrusting begins, palpate ventral base of penis to detect urethral pulsations that occur at ejaculation
- Lower AV during ejaculation so semen enters collection bottle
- Keep AV in place until stallion dismounts (ensure collection of complete ejaculate)
- Immediately drain water from AV to allow remaining semen into collecting bottle
- After dismount, rinse penis w/ warm water to remove lubricant
- Aspirate gel portion of semen, if unfiltered
- If filtered, remove filter

Maintain temperature of collection bottle

- Evaluate semen
- If stallion fails to ejaculate, lead him away from mare immediately after dismounting (so he doesn't kick her in frustration)
- Encourage another erection & allow to approach mare or phantom & attempt another collection
- If second collection fails, check temp. & pressure in AV (usually start w/ a slack AV & † pressure w/ bicycle pump)
- Several attempts m/b necessary to get ejaculation
- If unsuccessful w/in 1 hr, postpone until later to avoid frustrating stallion
- Second collection
- Give stallion time to recover (handler smoke a cigarette)
- Wait 1 hour

Morphology

- Phase-contrast microscopy of semen fixed in buffered formal saline (preferred method, eliminates artifacts of stained smears)
- Microscopy of stained smears
- Total sperm output =Volume x Concentration
- Total number of progressively motile sperm = Volume x Concentration x # PMS

Semen extender

- Antibiotic-treated extenders recommended
- Semen normally contaminated w/ bacteria
- Extenders don't improve fertility
- Prepackaged dry powder mix or makeup your own

Insemination

- Inseminate mares w/ minimum of 100 500 million progressively motile, morphologically normal spermatozoa
- Deposit semen into uterine body w/ clean plastic insemination pipette (disposable pipettes recommended)
- Inseminate at day 2 or 3 of estrus
- Inseminate every other day until ovulation occurs

Most breed registries require freshly collected semen for artificial insemination

- Transport of semen is therefore limited
- Frozen semen thus is also limited, but good conception rates have been reported



Semen evaluation (E 1385)

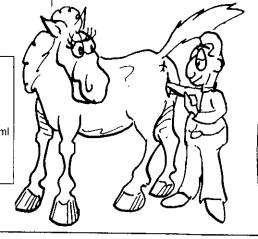
- Maintain all supplies to evaluate semen at 37° C
- . Volume: record total volume of gel-free semen
- Concentration
- Hemocytometer or spectrophotometer (properly calibrated)
- Motility
- Determine soon after collection
- Place a drop of semen on a warm slide
- Cover w/ warm cover slip
- Estimate % of progressively motile spermatozoa (PMS) under a microscope
- Dilute highly conc. samples w/ warm saline or extender to make a better estimate

Semen Extender Preparation C4T 575
Powdered milk (Sanalac®) 2.4 gm

Glucose 4.9 gm Sodium bicarbonate (7.5%) 2 ml

Sodium bicarbonate (7.5%) 2 mł
Gentamicin sulfate 100 mg
Distilled or deionized water qs to 100 ml

- Maintain extender at 37° C
- Add semen to extender 1:1 1:3 ratio



REPRODUCTIVE SYSTEM

Rectovaginal tears, Perineal lacerations

Condition

C4T 512, 550; R-Y 199; T 760; E 1308; EM&S 1049; M 265; T&W-A 356; J 1125; S 739, 743



· Perineal trauma (of body wall at pelvic outlet)

- Most wounds in vestibule wall

. Most not deep, heal rapidly & don't cause problems (1st degree)

Facts/Cause

- Deep wounds m/ lead to abscesses
- Common in unattended foaling
- Leg of foal forced dorsally, generally into constricted area of hymen (slight constriction at vagina/vestibular junction)
- Foal's hoof lacerates vestibular wall, or penetrates vestibular &/or rectal wall
- . If foal's foot pushed out of rectum & then back, get rectovaginal fistula
- . If foal foot in rectum as born, splits out perineum, ends up w/ cloaca
- Generally caudal to peritoneal pouches so doesn't enter peritoneal cavity (no fatal peritonitis)

Classification of perineal lacerations

1st degree - superficial, involving dors.

2nd degree - entire wall of vestibule &

3rd degree: vestibule, perineal body.

Rectovestibular fistula: hole betw.

mucosa of vestibule

rectum & anal sphincter

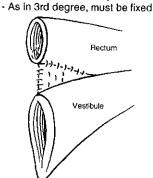
vestibule & rectum

perineal body

 1st degree: most common, usually heals rapidly w/o problems

Presentation/CS

- · 2nd, 3rd & fistula
- Acutely hemorrhage, have owner stuff
- a sheet into defect (hemostasis) until you arrive - Mare straining, due to imitation & trauma, causing damage; watch for this in first 48-96 hrs
- 2nd degree: injures restrictor vestibuli muscle
- . Epithelialization instead of "bridge"; leaving a
- large defect in perineal body Results in sunken appearance to dorsal vulva
- . Predisposes to pneumovagina & fecal material into vestibule
- 3rd degree:
- Mare hopelessly sterile until treated & repaired (feces in genital tract)
- Rectovestibular fistula: hole through vestibule, perineal body



Surgery

 Usually wait - 4-6 week before Sx for inflammation to subside

Treatment

- Epidural or NSAIDs to make more comfortable
- Cover w/ antibiotics
- ± Some debridement initially
- 3rd & 4th degree: lose next breeding season, therefore most wait until foal is weaned to do Sx

1st degree laceration

 Caslick's operation (see pneumovagina pg 177)

2nd degree laceration Sx

- M/b repaired immediately or delayed
- Breed mare before elective Sx
- Reconstruct perineal body (restrictor vestibuli muscle)
- Standing in stocks Local anesthetic &/or epidural
- Tail wrapped & tied overhead in case ataxia from epidural
- Incise triangular area around laceration to vaginovestibular junction
- Elevate mucous membrane
- Suture triangular field from cranial to caudal
- Oppose raw triangular suture dorsal to suture line w/ simple interrupted sutures
- Skin closure: vertical mattress, interrupted pattern
- Place deep mattress sutures across reconstructed perineal body
- · Aftercare:
- Tetanus immunization
- Systemic ABs for 3 d
- Remove sutures in 10 d



3rd degree laceration Sx (3rd & 4th degree Sx difficult to do or understand) Reconstruct separation of vagina & rectum

- Preop:
- Soften feces (pelleted feed, or grain, bran, alfalfa mixture for 5 d preceding operation)
- Hold off feed 24 hr; Remove feces & clean out rectum & perineal area
- Standing in stocks, tail wrapped & tied overhead in case ataxia from epidural
 Epidural (xylazine 100 mg in saline, lasts long time, 4 hr) Tranquilize
- Retractors (Balfours) or retention sutures used to open up vulva
- · First stage: repair rectovaginal shelf
- Remove scar tissue up to the intact rectovaginal shelf
- This separates intact vestibular & rectal mucosa
- Incise into intact rectovaginal shelf to connect wall defects
- Dissect dorsally & ventrally along cut, separating rectal & vestibular mucosa & submucosa from walls
 - . Results in 4 flaps of tissue (2 rectal & 2 vestibular) that can be opposed w/o tension
- 6 bite suture pattern (#4 Vetali®) (see surgery texts for 4 bite pattern)
- 2nd line of sutures (continuous) through inverted mucosal flap
- 2nd stage: repair perineal defect between vulva & anus
- Closes anal sphincter
- Done at same time or wait for shelf to heal (1 mo)
- Similar to repair of 2nd degree laceration (above)

Rectovaginal fistula: 2 methods

- •1: Split perineal body transversely from perineum to 1-2" cranial to fistula
- Suture the rectal defect w/o entering the lumen of the rectum
- Then suture the vaginal defect in the same manner
- Close space between rectal & vaginal tissue w/ simple-interrupted
- Close skin of perineum
- 2: Convert to 3rd degree tear by cutting out to perineum & repaired as a 3rd degree laceration - Lose support of perineal body & have to incise the anal sphincter, only advantage is good

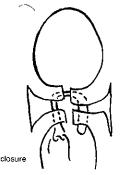
- Keep loose feces w/ bran, grain, alfalfa diet & mineral oil
- Painful passing faces so resist as long as possible, constipated then strain (danger of tearing)
- DSS to take in more water
- NSAIDs, postop monitoring of feces coming through
- If doesn't defecate, gentle enemas, don't want to palpate

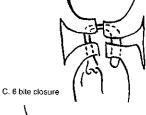
Tetanus immunization

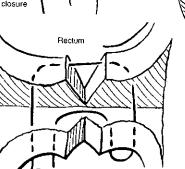
- Systemic antibiotics for 3-5 d • Remove sutures in 10-14 d
- Difficult to manage

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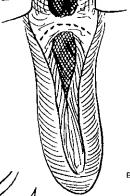
Tons of complications, mostly related to pain in mare not passing leces

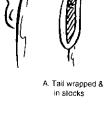






Vagina/ vestibule





B. Incise through walls &

D. 6 bite suture pattern (#4 Vetafil®)

- . 1. Through right vestibular flap from inside vestibule
- 2. Not full thickness of right rectal flap, then across
- 3. Into edge of left rectal flap & out ventrally
- 4. Through left vestibular flap
- 5. Back through left vestibular flap & across
- 6. Return to start, through right vestibular flap from opposite direction
- Tie suture everts vestibular mucosa
- Check that rectal sutures do not penetrate rectum!

Cesarean section, C-section

CS: Serious sequelae

Tx: Oxytocin, infusion

Dx: Check "Dutch britches'

E 1373; T 695; S 756

Dystocia: prolonged 1st or 2nd stage of parturition

- 1st stage: contraction of uterus
- Normally only 1-4 hrs
- Foal should rotate into dorsosacral position w/ head & front legs toward birth canal

eat fetal membranes

- Complete dilation at end of stage
- Breech or transverse presentations m/ result in never reaching 2nd stage
- 2nd stage: fetus enters birth canal & stim. forceful abdominal contractions, causing birth
- Normally last 20 min
- Most dystocias occur in this stage
- 3rd stage: expulsion of fetal membranes & involution of uterus, very rapid usually
- Indication for C-section
- Transverse presentation (especially draft horses, infrequent in light horses)
- Large fetus
- Small birth canal
- Uncorrectable positions due to ankylosis of limbs or vertebrae
- Neoplasia, masses, pelvic fxs
- Hydrocephalus or other fetal deformities
- · Correction of dystocias
- 1. Mutation (moving foal into correct position)
- 2. Fetotomy
- 3. Cesarean section
- Rapid decision to do C-section after evaluation under epidural anesthesia & tranquilization essential
- Fetotomies should only be performed on dead, emphysematous, or deformed foals
- . Avoid fetotomies requiring 2 or 3 cuts (mare less tolerant to long fetotomies than cow)

C-section procedure

Squeeze fluid back into retained portion

Usually expelled in 15-30 min

- Anesthesia: keep as light as possible as all cross placenta

Contraction causes fluid to come into exposed placenta

- (U) into abdominal cavity (prevent adhesions)
- (hind feet if anterior presentation, forefeet if posterior presentation [caud])
- (keep fetal debris or blood from abd.)
- far from major blood vessels (long enough so doesn't tear as foal is removed)

- Leave umbilical cord for several minutes before clamping & severing (pulsations sending
- Remove placenta if easy; if not leave in
- Oxytocin (20-40 IU/mare IM) (involution)
- Run whip stitch (simple continuous) along edge
- 2nd layer, inverting (Cushing's) w/ buried
- 3rd layer abdominal closure
- (linea alba, SQ & skin

- Properly pad, position & maintain blood pressure
- Dorsal recumbency w/ legs lifted upwards (well padded)
- Linea alba incision

- Exteriorize feet & put OB chains above &
- Assistant pulls foal from uterus
- blood to fetus)
- & just separate from edges of incision
- of incision, just binding edge to compress edges & shut down bleeding
- Lavage abdomen w/ saline & suction (remove blood & debris)
- Antibiotics directly in abdomen unnecessary

- Some lay w/ GG, paralyzed & awake, then local Must have facilities at stud farm
- Caudal ventral midline approach most common
- Incise skin from mammary gland to cranial to umbilicus
- Pour sterile saline w/ heparin (20,000 50,000
- Locate pregnant horn by finding feet
- Pack off uterus w/ wel towels or laparotomy pads
- Incise uterus over greater curvature
- below fetlock



- Broad spectrum ABs 5-7 d

Prognosis:

Good, but reduced if Tx

delayed, or infection

- Banamine® (ffunixin meglumine) for endotoxemia given while placenta still retained
- Tetanus prophylaxis
- Heparin SQ 40 IU/kg TID
- Oxytocin (20-40 IU) IM every 2 hrs
- Controlled walking 2 wk
- 2 wk in small paddock
- Not bred by natural cover for 8 wk

Prognosis:

 Depends on how soon done





Uterine Prolapse

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REPRODUCTIVE SYSTEM

Uterine prolapse

M8k 1032; Mk 700; IM 1389; I2M 1542; C4T 549; E 1361; EM&S



Facts/Cause

- · Rare in mare, common in dairy cow & sow Invagination of the uterus & protrusion from the vulva
- Uterine eversion: invagination of uterine horn not protruding from vulva

Rare in mare

Px: Good

- Cause unknown
 Associated w/ dystocia or retained placenta M/ follow foaling
- Partial eversion of a horn + pain & straining
 Removal of RP (retained placenta)
- Clinical Signs Protrusion of uterus from vulva
- Edematous & enlarged tissue in few hrs
- Contaminated tissue (m/b lacerated & trauma-

Replacing uterus

Mild presurgical scrub of uterus

ointments into edematous tissue

- Clenbuteros (reported to relax uterus)

If fresh, easy; alternately massage dors. & ventr. aspect to move cervical end in, then invert prolapse

After time harder (if enlarged & edematous) Vigorously & carefully massage emollient

- Straining, abdominal pain
 Restlessness
- Anorexia, † pulse & RR
- CS transient usually unless shock



 Protruding uterus Restrain animal (prevent trauma)

- Clean & protect uterus
- Stand mare w/ hindquarters higher than forequarters

Treatment

- Replace uterus (see box)
- Oxytocin (stimulate myometrial con-traction) after uterus replaced
- Antibiotics
- Amputation if severely traumatized or if



Px (prognosis)

Favorable - generally, if serious damage



Equine

M8k 1032; IM 1064;



· Much lower incidence than in cow for obvious reasons

 Most commonly a few weeks after foaling

CS & Dx: Protrusion of uterus

Tx: Replace - Oxytocin - ABs

- · Mammary swelling, heat, pain Ventral edema
- · Depression & anorexia
- · Mild hindlimb lameness

Culture & sensitivity (C&S)



ABs from culture & sensitivity

- Before results trimethoprimsulfonamide (5 mg/kg orally bid) or penicillin (20,000 IU/kg IM bid) & gentamicin sulfate (2 mg/kg IV or IM tid)
- Frequent milking
- Hot packs



Prognosis:

 Good: improve in 3-5 days, clinically normal in 1 week

mastitis

Less than cow

CS: Swelling, heat, edema

Dx: C&S

Tx: ABs; Px: Good

Tumors of udder • Extremely rare in mare

Postpartum colic

Uterine

1033; IM 253

rupture



C4T550; R-Y159; E1361; EM&S

Dystocia - Uterine torsion

Tx: Repair & Tx peritonitis

CS: Pospartum colic

Common

torsion

of uterus

(peritonitis)

 Normal postpartum pain caused by uterine contractions that persist past foaling

Inversion of tips of uterus : abnor-

Normal after parturition

Sequela to dystocia or uterine

Usually on dorsal aspect of body

- Foal feet through wall

Partial or complete tears

DDx from more serious colic

- Normal discomfort Occasional rolling
- Inversion of tips of uterus
- Severe pain



M/b asymptomatic until peritonitis

Severe hemorrhagic shock & death

develops hours after parturition

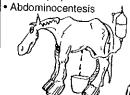
Hx (pospartum), CS

 Normal pospartum pain can be controlled

- Differentiates it from more serious colic & inversion of

DDx: Severe colic:

- Inversion of tip of uterine horn
- Rupture of vessel in broad lig.
- Trauma to large colon
- Uterine rupture
- Suspect in any case of
- postpartum colic
- Rectal palpation
- Uterine palpation



Book of the second of the seco

• Hx, CS

Hx of exposure

 Normal, spontaneously recover



 Inversion of tip, manually correct through uterine lumen

Repair uterine tear - Blind suturing through birth

- canal or flank laparotomy Systemic ABs
- NSAIDs
- Tx peritonitis (pg 53)
- Peritoneal lavage (Ig. volumes of sterile saline & ABs)
- Followed by drainage



- Remove source
- Supportive care
- Check hay for contamination



'Summer syndrome", Fescue toxicity

Dx: Hx, CS

M8k 2086, 2083; C4T 571, 84, 670; IM 1660; I2M 257; CT 369; PP/O 19

Tall fescue

CS: Repro problems Dx: Hx, exposure Tx: Remove & support

- Tall fescue (Festuca arundinacea)
- Usually good forage
- Claviceps-like fungus m/b



"Summer syndrome"

Postpartum colic

M/b vaginal hemorrhage

- Pyrexia, inappetence - Loss of body condition
- Poor hair quality
- Compromised repro function
- . Agalactia
- .Thickened placentae
- . Prolonged gestation

occasionally horses

Weak foals at birth Fescue foot in cattle & sheep,



- Perennial grass
- 3-4' tall
- Broad leaves





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Lesion localization - Spinal cord

12M 165; C4T 314; S 538

UMN & LMN CS: helps differentiate peripheral from central lesions; & if central, helps localize level of the lesion.

LOWER MOTOR NEURON (LMN): motor part of reflex arctomuscles or glands (peripheral nerves). LMN are spontaneously active w/out input of UMN

- LMN CS to area innervated by damaged peripheral n. or its cell body in CNS (not UMN CS because UMN requires intact LMNs)
- Flaccid paresis or paralysis ("timp as a dish rag")
- Tone:

 to absent (hypotonia atonia)
- Reflexes:

 to absent (hyporeflexia areflexia)
- Fast atrophy (neurogenic atrophy) W/in 1 week

UPPER MOTOR NEURON (UMN): CNS neuron that affects LMNs (peripheral or cranial nn.). Initiates & maintains conscious movements & provides tone to extensor muscles (posture). Excitatory UMNs (inactive until needed) & inhibitory UMNs (constantly keep LMN under control)

- UMN CS caudal to damage in CNS, degree of signs depends on amount of damage to spinal cord (white &/or gray matter). Loss of the ability to initiate voluntary motor activity, & possibly uncontrolled hyperactivity of LMNs due to ↓ inhibition
- Tone normal to 1
- Reflexes normal or 1 (normo- or hyperreflexia)
- Spastic paresis or paralysis
- Slow disuse atrophy
- Extensors facilitated (extended limb (s))



SHIFF-SHERRINGTON SYNDROME: hyperextension of the forelimbs with lesions to the thoracic spinal cord

(removal of ascending inhibition) (bad prognostic sign, serious spinal cord lesion). Rare in large animals.

Proprioception damage: usually 1st CS in spinal cord damage compression. For localization, interpreted the same as LMN/UMN

- Ataxia
- · Postural deficits (wide base stance, knuckle over)
- Delayed initiation of movement

Superficial pain loss: lost at the same time as motor function loss. If superficial pain perceived, so will be deep pain. (A withdrawal reflex, DOES NOT require perception)

Deep pain: first to show pain & last to disappear (first to show, last to go). Loss of deep pain is a bad prognostic sign. Evaluated only when superficial pain is absent

Cutaneous trunci reflex or panniculus reflex: a normal twitching of the cutaneous trunci muscle to stimuli. Sensory fibers from lateral wall dermatomes pass craniodorsally to thoracolumbar spinal cord segments (1 or 2 vertebrae cranially). Ascending sensory tracts extend up the spinal cord to lateral thoracic nerve (C8) innervating the cutaneous trunci muscle

 Panniculus absent 1 - 2 vertebrae caudal to spinal cord lesion

Hyperesthesia: abnormal increase in sensitivity. Spinal cord segment lesions cause a focal hyperesthesia to the dermatome

Diffuse or multifocal pain: often due to inflammation

Presentation/CS

Focal pain: often due to compression of the spinal cord or nerve roots

CS & spinal cord region damaged

C1-5: Cervical region

- · No LMN to all limbs
- UMN CS, proprioc. & pain defc.- all limbs

C6-T2: Cervicothoracic region

- LMN thoracic limb.
- UMN, propriocep. & pain defc.- p. limbs

T3-L3: Thoracic & thoracolumbar region*

- Normal thoracic limb (+/- Shiff-Sherrington)
- UMN CS, proprioc. & pain defc.- p. limbs
- Cutaneous trunci absent 1-2 vert. caudal

L4-S2: Lumbosacral region

- · Normal thoracic limbs
- · LMN signs pelvic limbs

S3-Cd5: Sacral region (cauda equina)

- UMN signs bladder, anus & urethra (flaccid paralysis of anus, no defecation)
- · Loss of sensation to tail, penis, vulva &
- · Distended, flaccid bladder, incontinence

Horner's Syndrome - possible in lesions of C1-T3 (see box below)

- * Lesions between C1-S2
- UMN pelvic region
- · Anal & tail tone normal
- Bladder m/b distended, urethralis m. normal

Condition Facts/Cause · Common sites: C1, T12, L5 Spinal trauma CS depend on site of trauma M8k 928; IM 1004; I2M 165, 1130; EM&S 799; C4T 10, 304; Foals: cervical vertebral Fx C3T 535; E 1176, 1233; M Severe 371; J 1021; S 549, 573 Adults: fractures, thoracolumbar area "Wobblers" often fall & must be Ddx following traumatic episode C5-T2(see above) T3- L3 DDx: OAAM (pg 247). · Herpes myeloencephalopathy (pg 254) • Degen, myeloencephalopathy (pg 249) · Protozoal myeloencephalopathy · Verminous myelopathy Cauda equina neuritis (pg 248) • Wobbler (pg 247) • Rabies (pg 271) · Congenital abnormalities · Spinal tumors (pg 246) Anal & tail tone normal Bladder m/b distended, urethralis m. normal · Sorghum cystitis/ataxia (pg 157) Stiff neck

Meningitis (pg 268)

Recumbent animal

LMN CS: level of lesion

UMN CS: behind level of lesion

Obturator n. paralysis (rare, pg 257)

• Hx, CS · Noncompressive - stiff neck Rads • Mild compression - ataxia (proprioception - 1st to go) - Recumbent - BAR (bright, alert & responsive) to depressed ± Phrenic n., paralysis of diaphragm - death - UMNs all 4 limbs (spastic) UMN - pelvic region Anal & tail tone normal Bladder m/b distended, urelhralis m. normal . LMN thoracic limb (flaccid) · UMN pelvic limb (spastic) UMN - pelvic region Anal & tail tone normal Bladder m/b distended, urethrafis m. normal T3- L3, Thoracic & thoracolumbar spine Dog sitting posture Sternal recumbency - pelvic limbs extended, not tucked up Normal thoracic limbs LMN signs pelvic limbs • UMN - pelvic region Anal & tail tone normal - Bladder m/b distended, urethralis m. normal L4-S2 Lumbosacral region LMN signs pelvic limbs UMN - pelvic region

Dx

Treatment Evaluate suffering (manage or euthanize from this) Manage pain: severe: Demerol® or morphine Evaluate repeatedly over 1st hrs for Px Euthanize if unable to rise after

several days or if suffering If bladder problem: empty distended bladder, culture urine repeatedly & Tx any intection If paralyzed rectum: empty rectum manually

 Most recover spontaneously. not influenced by drugs

 IV DMSO, in acutely affected, same dosage, every 12-24 hr (antiinflammatory, scavenges free radicals)

- Steroids: dexamethasone IM or IV
- Worry about laminitis, give early - Hydrocortisone, expensive, advantage is its short life
- ±Mannitol if sure of no hemorrhage, mixed in lactated Ringer's,
- saline or 5% dextrose, given slowly, expensive to manage these animals

Slings: if fx stable & animal stands w/ assistance







Prognosis figured from:

Repeated neurological exams (1st several hours) - not from radiographs, because pieces of fx likely to

be in different position than at time of injury Longer recumbent & neurologically impaired,

poorer prognosis

Horner's Syndrome: see pg 255 - spinal cord lesions in C1-T3 area & sympathetic ANS lesion between T1-3 gray malter & eye

In all species

- Miosis (loss of sympathetic to iris) - Enonhthalmos

- Ptosis

Excessive sweating on affected side (horse)

245

· Incontinence (atonia of urethral sphincter)

Normal limbs

 ↓ to absent anal tone

· Distended & flaccid bladder



Condition

abscess,

Osteomyelitis

M8k 923; IM 1009; I2M 1134; C4T 3; EM&S 804;

M 372; J 1013; E 1230

body

Facts/Cause Hematogenous infection Vertebral

 Neonates - septicemia Salmonella, Actinobacillus equuli. E. coli, Strep., Rhodococcus equi &

Klebsiella pneumoniae Adult w/ bacteremia

Brucella abortus &

Mycobacterium tuberculosis

Brucella, m/b extension from wounds in horses in same environment as cattle

Mycobacterium tuberculosis extension from granulomas present in lungs

Lumbar vertebrae commonly involved

Sequelae:

Compresses spinal cord

- Erodes through meninges (pachymeningitis)

Meningitis

Pathological fractures

Fistulous withers, draining tract from vertebral bodies externally

Presentation/CS Localized to vertebrae

· Occlusion of spinal cord

- Mimics spinal trauma (see pg 245)

↑ Temperature (helpful in Ddx)

- Sick, bacteremia, septicemia

- Pain, heat, swelling over site gen.

- Standing or recumbent - If standing often reluctant to move

due to pain

- Neurological signs (acute or chronic)

. UMN &/or LMN signs

. Paresis & ataxia

All 4 limbs or just hindlimbs, depending on vertebrae affected

Pachymeningitis:

Meningeal inflammation CS

Hyperesthesia

· Intermittent spastic muscle contractions

• "Weather vane" (head stiffly extended)

· Recurrent profuse sweating

of compression) PM (postmortem)

Uneven, deformed, soft vertebrae

Sequestration of necrotic bone

 Fistulous tract from abscess to subarachooid space occasionally

Diagnosis

- Hyperfibrinogenemia, neutrophilia, monocyto-

Epidural abscess most common, CSF normal
 Inside dura: CSF > 100 PMNs/dl,

CS of spinal cord problems

CBC: chronic inflammation

- Globulin ★ in adults, but ★ or ↓ in

neonates, depending on colostrum

- Bacteria m/b on gram stain smear

• Radiographs - plain films

- Osteomyelitis (random

hypolucency & sclerosis)

Myelogram (to locate site

in intervertebral joint)

Diskospondylitis (osteolysis

Brucellosis titer: rising or > 1:160 if spinal

Infradermal skin test for spinal tuberculosis

- LMN & UMN CS

Protien > 200 mg/dl

- Xanlhrochromia

brucellosis

Sick animal - fever

Treatment Long term ABs for 2-3 mo

Generally effective if early

Culture & sensitivity

Broad spectrum ABs if culture inconclusive or no culture

Amikacin or gentamicin + Potassium pen. G IV 1-2 wk

. Followed by trimethoprim-sulfonamide combination - for 2-3 mo.

Sx curetlage of necrotic bone & drainage of abscess recommended by some, but difficult because of inaccessibility of large







No universally successful

Rare recovery w/ short term

- Fusion of intervertebral joint

(bone graft or decompression & immo-

Dorsal decompressive laminectomies:

poor success & high complication prob-

corticosteroids & 6 mo rest

Surgical arthrodesis m/b

successful (for breeding animals)

Hematogenous, lumbar CS: UMN/LMN, sick, fever

Dx: CS, Rads Tx: Long term ABs

Spinal tumors

IM 1010: I2M 1136

Pathophysiology:

Hematogenous septic thrombi into metaphyseal arteries that enter the physes of vertebrae

Metaphyseal aa. & vv. communicate w/ ventral vertebral plexus

Ventral vertebral plexus drains into caud, vena cava & portal veir

 Ventral plexus has no valves so blood w/ bacteria m/ backup w + abdominal or pleural pressure

CS indistinguishable from spinal fractures, m/b insidious onset leading to paraplegia or tetraplegia

DDx: (no fever)

See Ddx for spinal trauma pg 245

Px (Prognosis): • Poor

treatment

bilization)

Wobbler syndrome, Equine sensory

ataxia, Cervical malformation-malarticulation,

Cervical stenotic myelopathy M8k 919, 902; Mk 593; IM 997, 1580; I2M 1121, 1127; EM&S 807; E 1214; C4T 306; C3T 532; C2T 355 M 365; S 551; J 1023

Stenosis - white matter -

Stenosis of cervical vertebral canal

Heredity not established

Young horses

2 types of stenosis

· Rare - no treatment

1. Static stenosis - C5-7.

2. Dynamic stenosis - C3-5

Pathophysiology:

- Focal compression of dorsal spinal cord (white matter diz) Demyelination myelopathy

- Proprioceptive loss (sensory ataxia)

- Motor generally normal; but m/b spastic or weak (compression of spinocerebellar tracts - UMN)

Pain & spinal reflexes generally unaffected

Pronounced hindlimb ataxia & paresis

🔖 🛨 Forelimbs spastic

"Clumbsy" ("Tin soldier" walk) hypometric (proprioception)

"Knuckling", spasticity, stumbling, scuffing of toes, incomplete limb protraction, circumduction, crossing over, interference. excessive sway of pelvis

Rarely - cutaneous desensitization & atrophy of

· Horner's syndrome & sweating of neck if severe OCD in some animals

DDx:

1. Static stenosis: cervical vertebral stenosis - C5-7

2. Dynamic stenosis: cervical vertebral instability - C3-5

Compression not relieved by extending head

Flex head - compresses vertebral canal

When extended, no radiographic sign

Narrowing of lumen due to excessive bone production (osteopetrosis)

Older: 1-3 yr-olds (6 mo - 10 yr)

Younger horses 6 mo - 2 yr

of compression

See DDx for spinal trauma pg 245

· History & CS "clumbsy"

PE (physical exam):

Pull tail laterally while walking (horse unable to compensate)

Turn sharply while trotting. exacerbates proprioceptive deficit

Placing tests: pull limbs away from midline, failure to replace limbs spontaneously Reluctance to move backwards

- Up or down slope: exaggerated ataxia

Radiographs:

- Neck: plain films nonflexed & flexed

- Static compression

Measure diameter of spinal canal & compare to normal horses

Chronic cases, remodeling m/ give normal measurements w/ CS

Dynamic compression

Subluxation on flexion of vertebral junctions Sometimes can't see w/o contrast material

Myelography for subtle lesions

eneral anesthesia & lateral recumbency

Clicking noises when flexing neck

± Palpate malformation of atlas,

- Metrizamide

Hx, CS: Arabian

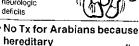
not progress some appear to improve, learn how to compen sate for

Prognosis:

Guarded

In some, diz does

neurologic deficits



hereditary





Radiology - definitive Dx

DDx:

Normal awkwardness at birth

See DDx for spinal trauma pg 245

OAAM.

Occipito-atlanto-axial malformation M8k 920, 902; Mk 593; IM 1016; I2M 1142; EM&S 797, E 1231, C4T 2; C3T

proprioceptive loss

Tx: ? Px: Guarded

CS: Clumbsy, knuckling

Dx: PE, Rads, Myelograph

531: M 370: S 566

Developmental defect

Arabian foals - inherited

Symmetric occipito-atlantal, atlanto-occipital fusion & other malformations

Quarter horses - nonfamilial

Morgans & Standardbreds

 Spinal cord compression - Lesion to UMN & conscious ± Dead at birth

Ataxic at birth or in few wks

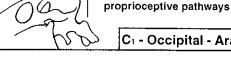
. Difficulty bending head to suckle "Weather vane" stance - neck

extended if symmetrical tesion Torticollis ("wryneck", twisted neck)

 Symmetric UMN CS (Cervical region) - #1 tetraplegia or tetraparesis

- Proprioceptive deficits (ataxia)

Hyperreflexia & hypertonia



C1 - Occipital - Arabians

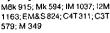
Condition

Equine

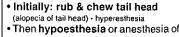
Facts/Cause Inflammation of nerve roots Cauda equina - Cauda equina particularly &

neuritis, polyneuritis

1163; EM&S 824; C4T311; C3T 579: M 349



- cranial nerves
- LMNs (lower motor neurons) flaccid paresis or paralysis
- Adult horses
- · Cause unknown (idiopathic)
- Maybe autoimmune (resembles Coonhound paralysis)
- Maybe hypersensitivity



effected area Progressive paresis, m/ terminate

Presentation/CS

- in paralysis of tail, bladder, rectum, anal sphincter
- Weak or paralyzed tail
- · Anus hypotonic or atonic & distended
- Fecal retention or incontinence
- Urinary incontinence (parasympathetic fibers in sacral nerves [pelvic n.], LMN type [atonic bladder, distended & easily expressed))
- Scalding of thighs due to dribbling
- Protruded, relaxed penis
- ♣ Sensation to perineum
- · Prepuce usually still sensitive (L2-4 roots)
- If lumbrosacral involvement
- Hindlimb flaccid, weak & ataxic (denervation atrophy m/ develop)
- All 4 limbs m/ be affected

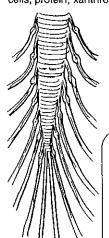


- Asymmetrical (different than cauda equina)
- Depends on nerves involved
- Atrophy of temporal & masseter muscles (motor branch of CrN 5) Facial paralysis (CrN 7/Facial n.)
- Keratitis & corneal ulcers (CrN7/Facial)
- · Head tilt & other CN deficits possible

Sequela: Urinary infections (due to incontinence)

Diagnosis CS: loss of sensation to peri-

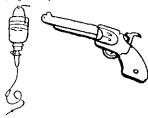
- neum, urinary incontinence EMG abnormalities (due to denervation)
- PM (postmortem): - Cauda equina thickened,
- discolored & covered w/ fibrous tissue
- Granulomatous inflam, of nerve roots Axonal degeneration & demyelination
- Wallerian degen, in spinal cord
- CSF tap shows † mononuclear cells, protein, xanthrochromia

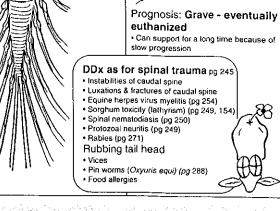


No therapy consistently effective

Treatment

- Steroids at inflammatory doses
- Supportive care
- Fluid therapy
- Manual evacuation of bladder & rectum
- Euthanasia (severity gradually progressive)





- CS: Toxic to CNS: Posterior incoordination.
- "Dribbling" (urinary incontinence)
 Cystitis 2° to urine retention
- Tx: Withdraw Sorghum, ABs (cystitis)
 Px: Recovery rare

 See URIN pg 156; Usually valuable forage, Grazing Sorghum spp. & Sudan grasses · Myelomalacia of lower spinal cord

Equine degenerative myeloencephalopathy,

M8k 921, 902; Mk 593; IM 1002; I2M 1128; EM&S 757; C4T 319; C3T 559; C2T 353; M 366

- · Common cause of ataxia in young horses
- Foals 6-8 mo (m/b born w/it)
- Degenerative, noncompressive spinal cord diz
- Demyelination of dorsal funiculi of cervical spinal cord & brain stem
- Loss of conscious proprioception (don't know where limbs are)
- Cause: unknown, implicated:
- Vit. E deficit suggested
- Confinement on dirt paddock
- Insecticides & wood preserva-
- Processed or pelleted feeds
- · Arabians, now in all breeds
- Familial predisposition reported

- Symmetrical ataxia
- Proprioceptive (sensory)
- Knuckling, stumbling, stabbing as limbs placed on ground, circumduction, abduction, interference, spas-
- ticity, abnormal limb protraction, hypermetria All 4 limbs; hindlimbs m/b more
- affected



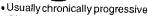
History, CS

- Postural placement tests
- Pull tail to side or push withers or tuber coxae (m/ stumble or fall)
- Turn sharply (slumble & can't lift inside forefoot)
- Laryngeal test
- Endoscope larynx & stap saddle area Normal contralateral arylenoid closes
- Radiographs, plain & contrast
- Spinal canal normal diameter
- CSF normal
- Vit. E levels of feed & individuals
- PM (postmortem)
- Fiber degeneration of ascending & descending spinal cord funiculi
- Large eosinophilic "spheroids" in gray matter



DDx as for spinal trauma Cervical stenotic myelopathy

(spinal canal stenotic)



- Signs are irreversible
- · Euthanasia, usually when become dangerous to self or others
- Supplement Vit. E if not severely affected & < 12 months
 - Supplement for life
- Sprinkle on conc. mixed w/ vegetable oil Maintenance 6,600-10,000 IU/kg/day
- Up to year to be neurologically normal

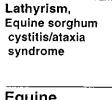


Prevention:

On farms w/ problem

Mares supplemented w/ Vit. E/Selenium to increased Vit. E in milk

- Mare w/ normal Vit. E status, will not show deficiency Check all animals on farm (subclinical)
- Change to hay w/ adequate Vit. E



Inflammation of nerve roots

Tx: Euthanasia. Px: grave

CS: LMNs, Numb rump, Dribbling

EDM



Compression - Vit. E defc ? CS: Ataxia, knuckling

Dx: Hx, CS Tx: Euthanasia







Condition

Facts/Cause #1 cause of multifocal, asym-

metric, progressive CNS diz

Can mimic any neurologic diz

- Lesion in brain & spinal cord

- Asymmetric loss of LMN (lower

Route of infection unknown

Organism randomly migrates

· Sarcocystis neurona

motor neurons) &/or UMN

Infectious, but not contagious diz

Presentation/CS

Diagnosis

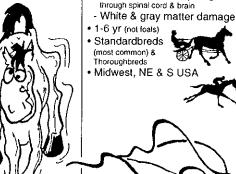
Treatment

Equine protozoal myeloencephalitis, EPM

M8k 934, 926; Mk 594; IM 939; I2M 1046; M 368; EM&S 757; E 1225; C4T 329; C3T 554; C2T 359; POP 43-1/98; 12-4/98, 9-5/







#1 multifocal, asymmetric neurological diz

CS: Ataxia, atrophy, down

Dx: CNS CS + Western blot (CSF) Tx: Timethoprimsulfa + Pyrimethamine

of parasites through CNS Multifocal, asymmetrical

progressive diz of CNS CS can mimic any neurologic diz

 Strongylus vulgaris, Micronema, Setaria, less commonly Habronema

·Similar to protozoal migration,

- Inflammation & necrosis of cau-

- Asymmetric loss of LMN (lower

Pathophysiology:

Organism randomly migrates through spinal cord & brain

Similar, but less common than EPM

Tx: Ivermectin + EPM Tx

Multifocal & asymmetric

Gait abnormality (peracute or acute)

 1or all 4 limbs depending on where it migrates

- Asymmetrical (because multifocal)

Ataxia, paresis & spasticity

. Knuckling, circumduction, crossing over, tetraparesis - areflexia, hyporeflexia (LMN) or hyperreflexia (UMN) depending on site of lesion

Muscle atrophy of individual muscle groups

Localized areas of sensory deficits "Strip sweating" localized areas (der

matomes, sympathetic white matter tracts)

 Cerebellar, brain stem (less common) or cerebral signs, cranial nerves Head tilt, facial paralysis, circling, nystagmus, dysphagia, blindness w/ or w/o abnormal pupillary reflexes

Untreated progressive to recumbency in 14 day- 6 months

#1 DDx for multifocal,

asymmetric neuro diz

 Hx (age), CS (asymmetric, multifocal ataxia & weakness)

 CNS CS + pos. Western blot of CSF highly suggestive

 Presumptive: treat & R/O (rule out) others

Western blot serological lest for CSF & serum, 50% of horses positive serum

CSF taps: normal or m/b + protein & + monocytes (pleocytosis)

Post multiple sections of spinal cord

- Multifocal & asymmetric

- Gross: gray-brown discoloration, w/hem orrhage, swelling, & liquefaction Histo: nonsuppurative inflammatory fo-

cal malacia & hemorrhage, perivascular culfing, gliosis, astrocytosis, neuronal necrosis & giller cell proliferation

Multinucleated giant cells

- Parasite in CNS definitive Dx. schizonts & merozoites at periphery of lesions, but m/not be demonstrated

 COMBO of antifoliate drugs - Trimethoprimsulfa (PO o12h4-

8 wk) +

 Pyrimethamine (Daraprem®. malaria drug) (PO q12h 3 d, then PO q24h 4-8 wk)

Blood count every 2 wk during therapy because m/ cause foliate defc. (leukopenia, thrombocytopenia & anemia; rare) Discontinue & give foliate

oliate supplement ?\$: potential toxicity in

NSAIDs

 No steroids (because of need for cell mediated immunity to control parasites)

 DMSO IV to inflammation in 5% dextrose - given without difficulty, but causes intravascular hemolysis so hemoglobinuria or hematuria

Variable positive or neg. response time Insurance require 6 wk before euthanize Monitor CBC every 10-14 d, folate

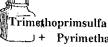
inhibitors, can get pancytopenia Marked platelet drop, cut back dose

Multiple B vitamin complex supplement

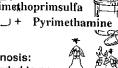
Stall rest

· Diclazuril & Toltrazuril: antiprotozoal diz. need testing

Euthanize those that don't respond



Prognosis: •Guarded to poor



Cerebrospinal nematodiasis.

Verminous myelitis/ myeloencephalitis

M8k 926, 940; Mk 594; IM 1011; I2M 1137; EM&S 751; E 1190, 1231; C3T 556; M 372



Occasional aberrant migration

but much less common

dal brainstern & spinal cord

motor neurons) &/or UMN

White & gray matter damage

Mimics protozoal myelitis

Gait abnormality (peracute or acute)

- 1or all 4 limbs depending on migration

- Asymmetrical

cranial nerves CS

 Ataxia, paresis & spasticity . Knuckling, circumduction, crossing over, tetraparesis - areflexia, hyporeflexia (LMN) or hyperreflexia (UMN), depending on site of lesion

Muscle atrophy of individual m. groups

Localized areas of sensory deficits Cerebellar, brain stem, cerebral, or

 Head tilt, facial paralysis, circling. nystagmus, dysphagia, blindness w/ or w/o abnormal pupillary reflexes



 Presumptive: 1° treat animal & rule out (R/O) others

Serologic tests not available

CSF taps

Xanthrochromia

DDx • SEE DDx for spinal trauma pg 245

If recent migration

. + RBCs, mononuclear inflammatory cells

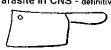
. Eosinophils (highly suggestive of aberrant migration)

PM multiple sections of spinal cord

- Multifocal & asymmetric Gross: gray-brown discoloration, w/

hemorrhage, swelling. & liquetaction Histo: nonsuppurative inflam., focal malacia & hemorrhage, perivascular cuffing, gliosis, astrocytosis, neuronal necrosis & gitter cell proliferation Multinucleated giant cells

Parasite in CNS - definitive Dx



DDx

SEE DDx for spinal trauma

• Ivermectin (PO once), repeat in 10-14 d m/b, more clinical studies on ivermectin

Thiabendazole, (10 x n o r m a l dose) SID for 2 d

Fenbendazole (Panacur®) at hi doses 3-5 d in a row

Setaria organophosphates &/or diethylcarbamazine

 NSAIDs: Banamine®, bute, aspirin or naproxen to # inflammation around parasite when it dies

No corticosteroids



Trimethoprimsulfa + Pyrimethamine (pg 250)



Prognosis:

Guarded: some respond

to Tx w/ anthelmintics

Some may improve & maintain some neurologic deficits

Setaria spp.

Common filarid parasite of cattle

· Aberrant migrations in horse

- Predilection for spinal cord • Tx: Diethylcarbamazine (single dose m/b effective) or organophosphates

Strongylus vulgaris

CS normal infestation

Colic

Gangrenous enteritis

 Acute or chronic diarrhea

Micronema deletrix

· Free living rhabditid nematode

 Penetrates skin of face, lips, gums & tongue

 Migrates through vascular system to CNS



Habronema infections

• Draschia (H.) megastoma (most

Tumorlike enlargements in stomach wall

Other habronema spp. asymptomatic - CNS migration of Draschia

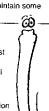
Pour-on insecticide before migration into CNS

Warm climates USA - by Aug.-Sept.

Cold climates - by Oct.

The second of the second company of the second second second second second second second second second second







Treatment - Prognosis

Hyperimmune serum, from horses vaccinated

Given before recumbent can reverse CS.

Proper disposal of carcasses

Very expensive \$1500 per 500 mis for foal

Antitoxin available, usually too late

Condition Botulism. "Shaker foals". "Forage

poisoning" M8k916; Mk328, IM1033; I2M 1159; EM&S 759; E 1242; C4T 327, 157; C3T



370, 542; M 374; Pic 26



C. botulism toxins CS: Rapidly fatal paralysis Dx: Difficult Tx: ?

paralysis

M8k 972, 917; I2M 1167;

· Ixodes holocyclus

· Foals & most other domestic species, most common in dog, sheep & cattle

Hypomagnesemia, Grass tetany: Affects only ruminants; tetanic spasms, convulsions

Clostridium botulinum type 8 & C

- Gram positive, anaerobic, spore-forming

Alkaline or neutral soil, decomposing tissue

- Prevents synthesis or release

- No proprioceptive deficits

Horses > susceptible > cattle

- Rapid & fatal (respiratory paralysis)

1) "Forage poisoning" ingestion

2) "Shaker foal diz" ingestion of spores, grow in GI (toxicoinfectious

Individual sporadic cases more

botulism) - 2 wk - 8 mo-old Mid-Atlantic states, exclusively

common than herd outoreaks

of preformed toxin, occurring 1° in adults

of ACh at NMJ (LMN)

. Loss of muscle tone

- Ubiquitous in soil

• Toxin

Facts/Cause Presentation/CS

> Progressive muscle weakness "Forage poisoning"

Gradual or rapid onset

Dyspnea

- Diffuse paresis - weak & trembling Recumbency

"Shaker foals"

- Paresis (stiff, stilted gait)

- "Shaking" (generalized m. tremors)

- Drool: difficulty chewing & swallowing

Tongue hanging out, not retractable - cardinal & early sign

Muscle tone

."Sleepy look", pytosis, loss of tone lo eyelids, ♦ in tail tone

. Mydriasis (para-ANS), slow pupillary response, ileus, constination & urine retention

· Inability to stand once down

Dyspnea

 Death - respiratory paralysis or pneumonia W/in 24-72 hrs



· Generalized flaccid paralysis (resembles botulism)



Diagnosis

. Difficult to Dx, because small amounts cause diz, improbable to demonstrate toxin in tissue or feed

- Tongue paralysis - early • Inject mice ("wasp-waisted"

death) - False negatives • Presumptive Dx: motorparalysis, if sus-pected, eliminate other causes

- Cl. organism in hay



DDx:

• Rabies (pg 271)

Septicemia

• Trauma

ID tick

· Nutritional myonecrosis

Hypoglycemia

Flaccid paralysis

· Esophageal obstruction

Prognosis:

for Botulism

Control:

Vermin control

"Shaker foals" mortality > 90%: grave

"Forage poisoning"

- Slow onset better than rapid

- If recover, takes weeks to months

🗦 • Remove tick, recover quickly

Prognosis:

Good after removal of tick

Tetanus,

Lockjaw

M8k 447, 929; Mk 330; IM 1023,1479; I2M 1150; EM&S 785; E 1209; C3T 540; M 385

Clostridium tetani

CS: "Saw horse", Dyspnea

Tx: Wounds - Antitoxin + Toxoid

Vaccinate - Vaccinate - Vaccinate

Dx: Presumptive - Hx, CS

· Clostridium tetani

- Spore in soil/feces

 World wide distribution Deep puncture wounds

(anaerobic environment needed)

- Necrotic tissue, cells lyse, releasing toxin

 Neurotoxin picked up by nerves to spinal cord (ascending tetanus) Excess toxin travels by lymph & blood to

brain (ascending tetanus) All species susceptible (horse & man most sensitive)

- IP 10 -14 days (week - weeks)

Toxin ascends nerves to spinal cord, causing ascending paralysis, if excess toxin in blood to

 Tetanospasmin, tentanolysin &

brain, ascending telanus

nonspasmogenic Blocks post synaptic inhibition to motor nerves.

causing hypertonia & snasms

· Initially intractable colic & vague stiffness

 Muscle spasms (masseter, neck, hindlimb & wound location)

· Then spasticity, general stiffness, & hyperasthesia

 Sound & tactile stimuli (elicit muscular spasms)

• "Lockjaw" (masseter m.)

· Prolapse of 3rd eyelid

· Erect ears, flare nostrils

• "Sneering" expression due to contraction of facial mm., retracted eyelids

"Pump handle" tail (stiff & extended)

· Excess salivation, quidding (drop food), dribble water (can't swallow)

"Saw horse" stance (extensor rigidity)

· Sweat profusely (horse only)

· Difficult walking, turning & backing up

· Recumbency, legs extended, ears parallel to back, unable to rise

Dyspnea (m/b aspiration pneumonia)

Lose consciousness & convulse

• Death due to respiratory paralysis or convulsions

 Survivors begin to show improvement in 2 wk, CS m/ persist for 1 month

2° Complications:

Laminitis

 Aspiration pneumonia, 2° to laryngeal & pharyngeal paralysis

 Tremendous myositis due to thrashing around it recover

Usually presumptive Dx

History

· CS muscular rigidity & hyperesthesia

- "Pump handle" tail

- "Saw horse" stance

- Erect ears

 No reliable clinical test for Dx (try to culture)

DDx:

Laminitis

Hypocalcemia

 Exertional rhabdomyolysis

Heat stroke

Pleuropneumonia

Vaccination:

Pregnant mares

- Annual toxoid booster 1-2 mo prior to foaling

· Foals from vaccinated dam

Toxoid to foals at 3, 4 & 12 mo

· Foals from unvaccinated dam

Tetanus antitoxin (1500 IU) day 1 + T. toxoid. then T. toxoid 4 wk, 4 & 12 mo

Annual revaccination for all horses

Tetanus prone wounds or surgery Unvaccinated horses or unsure

- Tetanus antitoxin + tetanus toxoid (mixed together)

2nd toxoid 1 mo later

Vaccinated horse

253

 Debridement of area, lavage & inject penicillin in wound area

 Muscle relaxation to control m. spasms acetylpromazine + 5% peniobarbital (cheap), or diazepam (\$) repeat multiple times

Quiet, dark stall, pack ears w/ cotton

• Good footing, deep bedding, m/b sling

High levels of systemic penicillin

- Minimum of 7 d IV 2-3 X normal dose Antitoxin (binds loose toxin to prevent binding

to nerves, once bound can't unbind) · IM, hi dosage, minimum 100 IU/kg, repeated

every 3-5 d (IM doesn't cross BBB)

Endothecal antitoxin (into cisterna magnum (subarachnoid space), under anesthesia) Remove 30 ml of CSF slowly, inject 50 ml (1000 IU/ml) toxoid. Success variable & many compli-

 Good nutrition: alfalfa pellets soaked in water containing electrolyte solution



If survive 7 days: fair to good

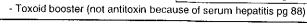














Condition

"Snots", Rhinopneumonitis, EHV-1, Myeloenceph-

alopathy, Herpesvirus 1, Viral rhinopneumonitis, Abortion virus M8k 924; Mk 593; IM 526; I2M 1015; EM&S 751; E 1228; C4T 333; C3T 556; M 367; Pop 3-9/



Facts/Cause

• See RESP pg 111 Herpes virus

- Transmission: Direct or indirect from nasal discharge, aborted feti, placenta or placental fluid
- Carrier animals always present Introduced to farm by newly acquired
- Persistent virus in environment Annual outbreaks in foals in horse areas
- Separate syndromes: Respiratory problem in foals, Abortion storms, Neonatal death or Myeloencephalopathy
- M/ have had resp. diz in the past
- CNS least common syndrome
- Vasculitis of arterioles, w/ 2° ischemic necrosis of spinal cord
- Affects white matter (almost exclusively)
- Lumbosacral area
- Unknown mechanism
- Arthrus hypersensitivity reaction Type 3 due to vasculitis (m/b)
- Neurotropic herpes strains (m/b)
- Most frequent in mares shortly after foaling during an abortion outbreak (m/balso in barren mares, males,

CNS least common syndrome CS: Ataxia (rear), incontinence, BAR

Dx: Hx, CSF

Tx: Steroid, Catheterize

Vaccination

Presentation/CS

- 1° Resp. virus (pg 111)
 Foats "Snots" runny (copious) nose Mares - asymptomatic
- Abortion 4-5 mo later
- Neonatal death in a few days
- Myeloencephalopathy some
- Incoordination & weakness in hind limbs, mild ataxia & posterior paresis to posterior paralysis & recumbency
- Rarely are forelimbs affected
- Usually remains standing
- ± Recumbency (bad sign), need help to get up, crawl around stalls due to not affecting front limbs
- Urinary bladder incontinence & distension
- Hypotonic tail

Prevention

- M/ or m/n be pyrexic which m/ or m/not be associated w/ respiratory signs
- No marked † temperature
- BAR, w/ good appetite

• Management (see pg. 111)

- Foals 3, 4 12 months-old

Vaccine see pg 12 (almost mandatory)

- Booster all horses every 2-3 months

Vaccine gives immunity for 2-4 months.

- No progression after 24 hrs

DDx:

 See DDx for spinal trauma pg 245

Diagnosis

. Hx of resp. diz, or abortion

CS symmetrical

incoor-

Lab: lymphopenia early due to viral

- Normal cell count (> 8/ml)

To confirm Dx: herpes virus titer, though

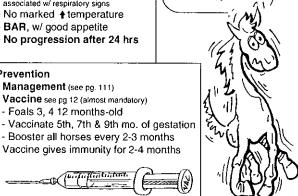
generally not done, CS & CSF enough

dination w/ incontinence

infection, but short lived

Protein

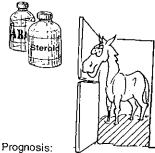
· CSF: Xanthrochromia



Steroids: Dexamethasone (hypersensitivity reaction) once w/in 24 hrs (IM or IV), Can

Treatment

- give after 24 hrs, but doesn't help & m/ cause - Stabilizes after 24-48 hrs
- Broad spectrum ABs to ofset dexamethasone& catherization of bladder
- Catheterize urinary bladder
- Keep on feet or myositis
- · If recumbent, assist to feet
- Slings used to get them off muscles
- Deep bedding if recumbent also helpful, ideally pack stall w/ shavings, wet down, then straw to give mat
- Feed & water placed in front of them



- Good if remain standing
- Guarded if recumbent > 2 d
- Quite a while to completely resolve
- Able to compensate for neurological deficits (ridden at low grade performance levels)

Horner's syndrome

IM 169, 980; I2M 1100, 169;



· A syndrome, not a disease Disruption of sympathetic

pathways to head (see box)

 Horse most susceptible of all livestock

Causes: Horner's -many

- Avulsion of brachial plexus
- Injection in neck hitting vagosympathetic trunk
- · Infections of guttural pouch
- Cervical abscesses
- Cervical tumors
- · Space occupying lesions of cranial thorax
- · Esophageal rupture
- Retrobulbar abscess
- Retrobulbar tumor

- Miosis (small pupil, same side, due to loss of sympathetic innervation)
- . Enophthalmos (sinking of eyeball)
- Ptosis (drooping of upper eyelid)
- · Excessive sweating on ipsilateral head & neck (seen only in horses)
- Regional hyperthermia
- Find location of damage
- Physical exam
- Endoscopic exam of guttural pouch
- Palpate jugular groove for swelling
- X-ray cervical vertebrae
- Check chest (auscultation, x-ray)
- Gait & proprioceptive responses

Tx depends on cause

- Injection damage: quickly infiltrate (inject) saline (perivascular) to dilute out & NSAIDs
- Tx mycotic infections

Prognosis:

 Neurological signs often irreversible

Sympathetic pathway:

- Descend from brainstem down neck to synapse in T1-T3 segments of spinal cord
- Preganglionic fibers pass over T1-T3 spinal nerves to sympathetic trunk in dorsal thorax
- Pass through stellate (cervicothoracic) & middle cervical ganglia to pass up neck in vagosympathetic trunk to synapse in the cranial cervical ganglion



Postganglionic fibers pass to sweat glands of head, dilator muscles of the iris, periorbital smooth muscles & periarteriolar musculature

Sympathetic disruption to head Miosis, Enophthalmos, Ptosis, Sweating

Guttural pouch mycosis, Damage to CrNs

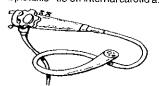
9, 10, 11 & 12 IM981; I2M 1101; EM&S 788, E 1206; J 1035

- See RESPIRATORY pg 100
- Mycotic infection of guttural pouch
- Grows on dorsomedial aspect of pouch over internal carotid a., . CrNs 9, 10, 11 & 12 & vagosympathetic trunk
- Eats through to artery & nerves & cause epistaxis & nerve dysfunction
- Extension of infection to parotid lymph node m/ cause lacial nerve damage (CrN 7)
- Epistaxis
- Nasal discharge
- Dysphagia (CN IX & X)
- Parotid pain (head extended & held close to ground)
- Horner's syndrome (sympathetic)
- · Facial paralysis (VII)
- · Sequelae:
- Aspiration pneumonia
- Extension vestibular diz

- Hx, CS
- Endoscopic exam of guttural pouch



- NO specific Tx of neurologic disorders
- Euthanasia
- Epistaxis tie off internal carotid a.



syndrome

travel over the roots of the brachial plexus & can be damaged w/ brachial plexus nerves

CS: miosis, ptosis, enophthalmia & sweating on head & neck (same side as lesion)

"Sweeney" Suprascapular paralysis

M8k 917; IM 1041; I2M 1169; E1238; EM&S 826; M 389; C3T 578; J 1019

Trauma to suprascapular n.

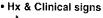
• Suprascapular n. crosses cranial border of scapula

· Common injury when yokes used to pull farm machinery, now w/ horse pulling contests

Trauma where nerve crosses scapula

· Acutely: lateral slipping of shoulder

 Chronic: prominent scapular spine due to marked atrophy of shoulder muscles (supraspinatus & infraspinatus mm.)

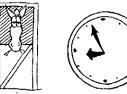




Time

Prevent 2° complications





Prognosis:

Poor if no improvement in 2 weeks

Radial n. paralysis

Tx: time

IM 1041; I2M 1169; E 1239; EM&S 828; M 389; AL 678: LAS 678



 Radial nerve: crosses axilla to dive into triceps brachii muscle & passes superficially on lateral side of arm under lateral head of triceps brachii m. (common injury site)

Innervates all extensor mm. of timb

Causes:

CS: Lateral slipping, prominent spine

 Direct trauma: kick, laceration, humeral fxs

Lateral recumbency w/o padding on lateral side of arm & shoulder (where it emerges under lateral head of triceps)

. Question if due to radial n. paralysis or compression of vasculature & ischemia to muscles

Danger of further injuries as horse recovers Overstretching of nerve during hyperextension "High radial nerve paralysis" injury above entrance into the triceps

- "Dropped elbow"

- Unable to bear weight on limb

Neurogenic atrophy of extensor mm. of limb

- CS of lower radial nerve injury

- Limb appears longer than opposite forelimb

Lower radial n. paralysis" injury near lateral elbow

"Knuckling over" (carpus, fellock & pastern joint)

Dragging foot = dorsal fetlock trauma

Can bear weight on limb

Loss of sensation to craniolateral forearm above the carpus

Hx, CS (clinical signs)

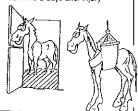
- "Dropped elbow"

- "Knuckling over"

Palpate for fxs of humerus

Radiology of humeral fxs

· EMG (electromyography) of exten sor mm. 5 days after injury



Conservative

- Time & TLC to see if function returns

 Horse can learn to "flip" distal limb so foot lands correctly ("lower radial n. paralysis")

Reduce inflam.: Dexamelhasone 3-5 days

Phenylbutazone IV & ice packs first 24 hours

NSAIDs (Banamine®, bute) or Demerol® for pain

Deep bedding, slings

Recumbent animal turned

6-8 X/day to prevent decubital ulcers ± Light cast or bandage to prevent contracture of flexors & to protect dorsal fellock

Humeral fxs m/b, remove bone chips & free up

nerve done 6-10 weeks after fracture End-to-end anastomoses of severed nerve



Guarded in mild cases

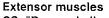
Unfavorable if severed nerve

Poor if dysfunction lasts > 2 wks







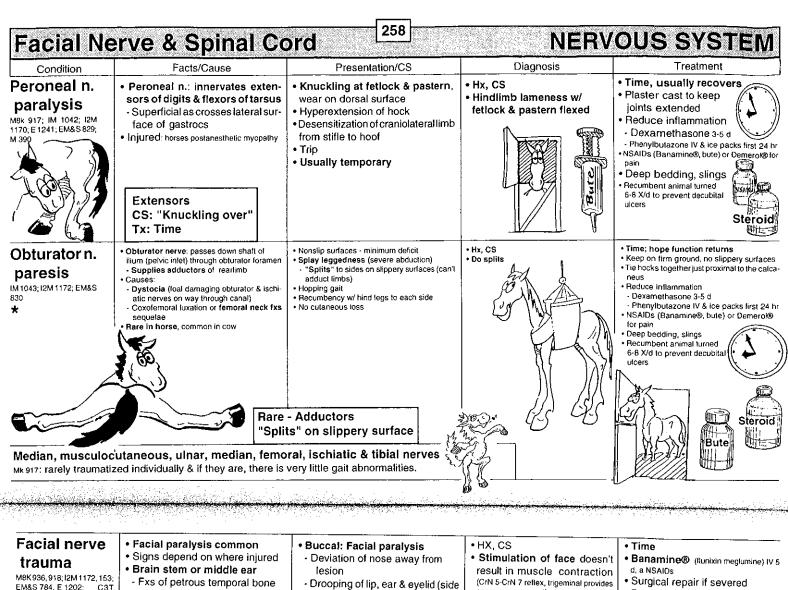


CS: "Dropped elbow", "knuckling"

Tx: Time







EM&S 784, E 1202; 578; M 376; J 1010, LAS



- Fx of stylohyoid bone
- Fx of ramus of mandible
- Midbrain lesions
- Polyneuritis equi Idiopathic facial n. paralysis
- · Buccal branch: Recumbency un-
- der general anesthesia
- "Buccal on buckle" (halter)
- of lesion)
- Flaring of nostril on inspiration (side of lesion)
- Proximal to ramus of mandible
- Facial paralysis
- Exposure keratitis (eye)
- . Inability to close eyelids
- . Lacrimal secretion
- ± Vestibular n. signs if near inner ear (head tilt, nystagmus & circling)
- sensory component)
- Schirmer tear test (for lacrimal gland)
- Atrophy not apparent because muscles thin as apposed to muscles of mastication
- Atrophy does cause distortion of face
- Protect eye if affected



Prognosis:

- Depends on site of lesion
- Peripheral nerve damage good

Prevention:

 Pad side of face when under general

anesthesia

CS: Facial paralysis, dry eye Dx: Hx, Schirmer tear test Tx: Time

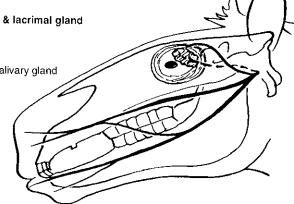
Prevent: Pad head when anesthetize

- Facial nerve (Cranial nerve 7)
- · Innervates muscles or facial expression & lacrimal gland
- · Arises from midbrain
- · Passes in relation to the middle ear
- Branches to lacrimal gland
- Crosses the guttural pouch under parotid salivary gland
- Branches to ear muscles & eyelid
- · Crosses over cheek (masseter muscle)
- Branches to cheek, nose & lips



5th cranial nerve

- Caused by trauma, protozoal migration or cauda equina neuritis
- CS: Dropped jaw, food falling out of mouth; marked atrophy of thick muscles of mastication
- Tx: No specific Tx; Banamine® (anti-inflammatory)



Brain stem lesions

IM 159; I2M 160; E 1176, 1205; C3T 522

Brain stem (midbrain, pons & medulla)

- · RAS, reticular activating system: concerned w/ conscious level
- · Proprioceptive fibers pass through
- Cranial nerves associated w/ brainstem
- UMN, sensory & proprioceptor fibers pass through brain stem
- Walking motion reflexes
- Generated in centers caudal to the midbrain
- Initiated rostral to midbrain (higher centers)

- Consciousness (depression, stupor, coma)
- Profound depression (RAS)
- Deficits in CrN 3-12
- Proprioceptive (Proprcpt) deficit & gait
- Proprept. defc w/ normal gait midbrain or rostral
- Proprept. defe w/ abnormal gait caudal to midbrain
- UMN & proprioceptive deficits all limbs

Causes - Brain stem lesion:

- Verminous migration (pg 251)
- Protozoal myeloencephalopathy (pg 250)
- Brain abscess or brain tumor (pg 269, 262)
- Cauda equina neuritis (pg 248)
- Horner's syndrome (pg 255)
- Eq Rhinopneumonitis (pg 254)



Proprioceptive (proprcpt.) deficit & gait

- Proprioceptive defc w/ normal gait midbrain or rostral
- Proprioceptive defc w/ abnormal gait caudal to midbrain
- Rostral lesions (cerebro-diencephalic) contralateral (opposite) proprioceptive defc, normal gait
- Caudal to the midbrain (brain stem & spinal cord) ipsilateral (same side) proprioceptive deficit, abnormal gait
- Midbrain lesions normal gait & proprioceptive deficit on both sides

Vestibular system lesion

IM 159; I2M 154; EM&S 792; E 1203; C3T 575; S 538

- · Control posture in relationship to gravity; & eye movements in relationship to head movements
- Peripheral vestibular centers: inner ear (labyrinth, receptors, & vestibular n., not proprioceptive fibers)
- · Central vestibular (vestibular nuclei in brain stem & centers in cerebellum) motor, sensory & proprioceptive centers [brain stem] located in area



Head tilt

- Nystagmus
- Ataxia
- Possibly circling & falling towards the lesion
- Strabismus

Central vestibular diz (brain stem) also shows:

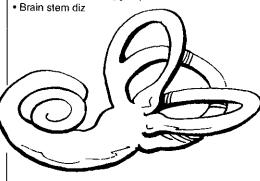
- Postural deficits (proprioception) & paresis (UMN)
- Depression (RAS in area of vestibular nuclei)
- Recumbency, lesion side down
- · Lean against wall
- Loss of perception of sensation

Peripheral vestibular diz also shows:

- No UMN signs (paresis)
- · Ataxia because of loss of balance, not due to proprioception
- · Not decr. sensorium or depression

Causes:

Otitis media/interna (pg 263)



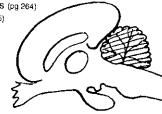
Cerebellar lesions

IM 159; I2M 164; EM&S 793; E 1176, 1208; C3T 524; S 538

- Coordinating movements, but does not initiate them
- Vestibular system connections to help coordinate balance & posture
- Proprioceptive fibers don't pass through cerebellum
- · Menace response, cerebellum plays an unknown role in the menace response
- Incoordination (ataxia) (excessive range, rate & force of movement) (base-wide stance from balance deficits, not proprioception)
- Tremors
- Abnormal movements of the head
- Vestibular diz signs, including:
- Head tilt
- Nystagmus
- Loss of menace response, but not blind
- · BAR (bright, alert & responsive) bec. RAS not affected
- No proprioceptive deficits

Causes:

- Cerebellar abiotrophy (pg 265)
- Grass staggers (pg 264)
- Locoism (pg 265)



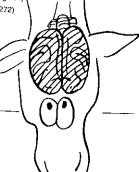
Cerebral lesions

IM 159; I2M 156; E 1175; C3T 523; S 537

- · Cerebral hemispheres & basal nuclei
- Voluntary motor control, behavior, & mental status
- Interprets vision & audition, proprioception, & general sensations
- Thalamus: functionally, it is closely related to the cerebrum to which it relays information
- Mild to marked depression (less than brain stem lesion)
- Alterations in behavior (aggression, rage, hypersexuality)
- Seizures
- Propulsive circling (wide)
- Head pressing, continual chewing (odontoprisis)
- Normal gait in straight line w/ abnormal postural reactions (proprioception loss, stumbling, knuckling over at fetlock)
- Blindness (occipital lobe) w/ normal pupillary responses

Causes - Cerebral lesions

- Protozoal myeloencephalopathy (pg 250)
- Hepatoencephalopathy (pg 268)
- Viral encephalomyelitis (pg 266)
- Rabies (pg 271)
- Leukoencephalomalacia (pg 267)
- Trauma/hematoma (pg 262)
- Hydrocephalus (pg 263)
- Verminous migration (pg 251)
- Idiopathic epilepsy (pg 272)
- Narcolepsy (pg 272)
- Brain abscess/ meningitis (pg 269)



Hypothalamus

 Controls the autonomic nervous system (ANS) & the endocrine system

Autonomic & endocrine abnormalities

- Polyuria/Polydipsia
- Altered sleep patterns
- Rage to affectionate behavior
- Abnormal appetite

Brain Stem Lesions

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NERVOUS SYSTEM

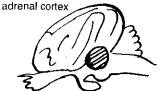
Brain tumors

M8k 951; IM 966; I2M 1083; EM&S 778

Condition

 Cause compression of brain stem Cranial nerves 5, 7 & 8

 Pituitary tumors of old horses rarely produce CNS CS, but stimulate



Hypermetric gait, ataxia

Depression

· Facial paralysis · Facial anesthesia or analgesia

Head tilt, strabismus & nystagmus

Presentation/CS

 Exophthalmos (iffacial tumor [SCC]) migrates through foraminae into cranium

· Horner's syndrome

• CS

CNS tumors

-Pituitary adenomas, microgliomas, medulloepithelioma, choroid plexus papillomas, ependymoma, neurofibromas, meningiomas, meningeal carcinoma

• None

2° tumors

- Melanoma, lymphosarcoma, adenocarcinoma, squamous cell carcinoma, hemangioma & osteoma

 Metastasize via vascular channels or extension along peripheral nerves

Compression No Tx

Brain trauma

IM 932; I2M 1037; C4T 301; EM&S 782; E 1199;



. Poll (top of head) trauma common

Fxs of basisphenoid, petrosal bones, occipital bones

Disrupt foramen lacerum & jugular foramen

. Dysfunction of CrN 9, 10 & 12

Midbrain: swelling - compression (herniation through foramen

· Medullary/inner ear syndrome

CS depend on part of brain

Extreme swelling - midbrain

- Cran. nn. dysfunction (see optic below)

Disturbances of consciousness

Abnormal respiration

Decerebrate rigidity

- Vestibular disturbances (nyslagmus, ventrolateral strabismus, head tilt, blindness,

 Ataxia, recumbency, depression & coma (struggle violently)

Vestibular CS

 Head tilt, nystagmus, circling, facial paralysis

Hx (History)

• CS

DDx:

Herpes myeloencephalopathy (pg 254)

Diagnosis

Degen. myeloencephalopathy (pg 249)
 Protozoal myeloencephalitis (pg 250)

Verminous myelopathy (pg 251)
Rabies (pg 271)

 Botulism (pg 252) Tetanus (pg 253)

Congenital abnormalities

Meningitis (pg 268)



Early recognition

Control ★ CSF pressure

Steroids

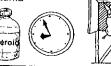
- Mannitol \$ (not if active hemorrhage)

Treatment

- IV DMSO

 Control seizures: Valium®. phenobarbital or pentobarbital

Protect from decubitus & self trauma



Traumatic optic nerve blindness IM 935, EM&S 782

Severe, blunt, head trauma - pushes brain back & stretches optic nerve in young, as in poll trauma (see above)

· CS: Blindness, loss of pupillary reflexes, pupillary dilation

Tx: None

Poll

• Px: Permanent condition



Vestibular diz. Otitis media/ interna

IM 976; I2M 1096; E 1202; EM&S 791, 794; C4T 323;



 β-hemolytic Strep., Staph., Aspergillus versicolor

2 types:

- Peripheral otitis media/interna: localized in temporal bone (less

-Central: extends to tympanohyoid joint & stylohyoid bone (severe)

. Fuses the joint between temporal bone & stylohyoid bone

. Fusion fractures w/ extension into brain case

. Results in hematoma w/in CNS .. Septic meningitis

Causes:

Extension of pyogenic bacterial infection from guttural pouch

Polyneuritis equi

Viral fabyrinthitis

Traumatic fractures

Peripheral - tocalized in temporal bone

- Dysfunction of CrN 7 (facial) & 8 (vestibulocochlear)

. Mild proprioceptive deficit (side of lesion), Ataxia

. Head tilt

. Lacrimation (CrN 7)

. Facial paralysis (drooped ear & lip. drooling, ptosis) (CrN 7)

- Exposure keratitis (can't close eyes)

- Nystagmus (not changed by head movement, rapid phase toward lesion)

. ± Ventrolateral strabismus (side of lesion)

Lean against wall (or circle) . BAR (bright, alert & responsive)

Central w/ fx

- Above signs

- Fall & become recumbent

- Depressed, febrile & anorexic

- If 2° septic meningitis

. Rapid deterioration of mental status

. Stiff neck

. Ataxia (proprioceptive)

. Hyperesthesia, fever, dysphagia

. Otorrhea (discharge from ear)

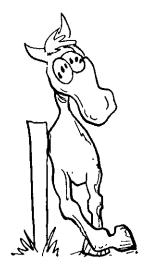
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Hx, CS

Skull radiographs for fxs

Schirmer tear test (lacrimation VII)

- Defc flow < 17 mm/min



Peripheral

- ABs: hi dose penicillin (IV qid) 3rd generation cephalosporins

. Trimethoprim-sulfonamide

- NSAIDs in early stages - Corticosteroids m/b early (problem w/

immunosuppression) - Quiet, heavily-bedded stall on good footing

Exposure keratitis

- Tarsorrhaphy, or

- Ophthalmic ointments, pilocarpine eye drops qid



Prognosis:

 Seemingly recovered horses used carefully because subtle neurological deficits m/ result in catastrophic accidents

Peripheral - CrN 7 & 8

· Depressed, Ataxia

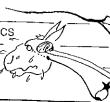
• Dry eye, Head tilt - BAR Central - 7, 8 & Brain stem

Hydrocephalus C3T 530: Dome shaped to cranium, Neurologic deficits

Verminous or protozoal migrations (see pg. 251, 252): aberrant migration through brain stem results in midbrain CS

Brain abscesses or brain tumors (see pg. 278): in midbrain cause midbrain CS

Horner's syndrome (see pg 255): fibers pass through midbrain, lesions to midbrain m/ result in Horner's syndrome



Cerebellar Lesions NERVOUS SYSTEM Condition Facts/Cause Presentation/CS Diagnosis Treatment • CS + Horses occasionally (also sheep, Grass Staggers Remove from grass, spontanecommonly in cattle) Hx of exposure to contaminated Muscle tremors to marked ataxia ously recover staggers, · Dallis grass, other Paspalum grass "Goose stepping" (overstepping) Supplemental nutrition grasses, Rye grass, Bermuda Ryegrass staggers, & falling grasses Bermuda staggers, - Exaggerated hypermetric gait, Ergot, Aspergillus, Penicillium & Dallis grass Ataxia, exacerbated w/ exercise staggers, Claviceps paspali, Tremogenic or excitement Paspalum staggers, alkaloids Paralysis Tremogenic Contaminate grasses Belligerent Prognosis: CS regress in 2-4 d toxins, . ± Nystagmus & salivation Occasional persistent Nervous ergotism locomotor disturbances Mk 1689, 1724; IM 986; I2M 1115, 1892; EM&S 795; E 1212; C3T 377; Tox 428, 435; PP/O 19 CS: "Goose stepping" Dx: CS + Hx Tx: Remove grass = recovery Dallis grass Perennial grass, 2-5 **Bracken** · See TOX pg 322; Bracken fern & horsetail toxicosis Thiaminase causes Vit. B1 (thiamine) deficiency staggers CS: Anorexia, incoordination (staggering), crouching stance, Mk 1641; IM 1648; I2M 1062, death preceded by convulsions & opisthotonus thiamine to 2.5 DDx: Plants, Horsetail, Turnip (Beta vulgaris), Crotalaria spp. Ragwort (Senecio jacobea), Other neurological disorders, Rabie Tx: Vit. Bi highly effective if early, Remove from

Locoweed poisoning, Locoism

Mk 332; IM 995; I2M 1877; EM&S 1784; E 1194, 211; C4T 651; C3T 376; C2T 674



• See TOX pg 328

bracken fern area

· Prognosis: Vitamin therapy usually effective if early

Astragalus & Oxytropis not all are toxic, Specklepod loco (A. lentiginosus), Pursh loco (A. purshii)

Toxic principle: Unknown (m/b barium, selenium, locaine alkaloid or larhyrogen)

- 1st vegetation available in spring, overgrazing & drought, remains green in late fall & winter
- · Horses > cattle > sheep
- Continuous ingestion, Horses that consume 30% of weight over 6-7 weeks
- Addictive (horses)
- Diffuse CNS disorder
- Western USA

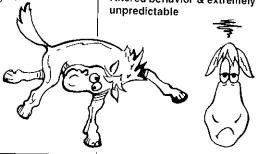
- Incoordination (proprioceplive deficits, ataxia)
- "Stringhalt-like" gait
- Frenzied or maniacal activity alternating w/ depression
- Wander in circles, or straight, Jumping imaginary objects, shying at familiar objects (poor judgement), Violent action, Depressed, Convulsions & falling, Bump into objects, Head press
- Head tremors (intention),
- visual impairments, dysphagia
- · Emaciation death
- Abortion & limb deformities in foals

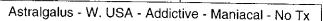
- · History of ingestion
- Tranquilization ineffective in controlling behavior
- Lab: vacuolation of cytoplasm of lymphocytes

• No Tx

Prognosis:

- Poor: horse remains affected long after removal from plants
- Altered behavior & extremely



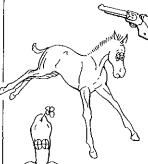


Cerebellar abiotrophy

IM 984; I2M 1105; EM&S 793; E 1209

- Degenerative diz in postnatal horse (abiotrophy)
- Arabians & Arabian crosses
- M/b born w/ it
- Develops CS after 6 mo
- Cause: Unknown, possible familial relationship
- Cerebellar neurologic diz Slow progression, some no progression
 - Conscious proprioceptive deficits
- CS suddenly after 6 mo of age Slight ataxia to complete diffuse
- cerebellar dysfunction
- BAR, No weakness, Hypertonia
- Stiff, hypermetric gait Rear & fall over backwards
- I- Accentuation of CS by stimuli
- Intention head tremors
- Truncal ataxia
- Hyperreflexia of all limbs
- No menace response, not blind

- Arabian cerebellar diz
- Turn animal sharply or blindfold. & obstacle course (exaggerates
- Circling, hypermetric, especially in hind-limbs, outside limb circumducts widely
- Menace response absent (but can see)
- CSF **★** protein
- PM (postmortern) I number of Purkinje
- NO Tx outcome euthanasia
- Council owners on heredity & suggest don't breed





- OAAM (pg 247), rads R/O
- · Neonatal maladjustment syndrome (NMS) (pg 270)
- Septicemia w/ 2° meningitis



Arabians - BAR - Ataxia - Euthanasia

Recovery gives "Dummies"

267

CS: Thrash, Dummies

Tx: Euthanasia

nasia

about damage to liver

Long term if not showing CS, worry

Inorganic Arsenic
 Moldy feeds (pg 267)

Vitamin A deficiency

1° cattle

Tx: Ca** EDTA

CS: Emaciation, "Roaring"

Condition Facts/Cause Presentation/CS Listeriosis. Very rare in horses, mainly a DDx for encephalopathies, Neonatal toals - meningoencephalitis, Listeria monocytogenes,

Circling Diz M8k 479; Mk 382; IM 969;

Dx: Isolation & ID organism (difficult, brain, liver, spleen & lymph nodes), seldom done before death EM&S 754; E 1246

· DDx: Rabies, Encephalitis, Lead poisoning

Common in ruminants & humans

• Tx: ABs considered ineffective, probably administered too fate in diz; Vaccines have given poor results

Vitamin A defc

· Not in horses, mainly feedlot ruminants w/o access to succulent plants

"Dummies", Neonatal maladjustment

syndrome, NMS, "Barkers",

"Wanderers" "Convulsants"

M8k 966, 925; Mk 610; IM 341; EM&S 764; E 1197; C4T 589; C3T 432; C2T 219; M 290; Pop 35-1/98



 Cause: unknown m/he multiple: possible asphyxia during difficult birth, but some have normal births

· Abnormal behavior at birth or w/in 24 hr

 Deranged cerebral, cerebellar function or spinal cord CS

· Foals if force fed colostrum, will not be depressed & sick as w/ septicemia, just

- Sequelae
- Gastric uicers
- Entropion & corneal ulcers
- FPT (failure of passive transfer)



CS: Day old Dummies Tx = OK

• "Dummies"

Cerebral CS (diffuse, most common)

- Loss of suckle reflex
- Aimless wandering
- Apparent central blindness

· CS: Encephalitis, depressed, † temperature, anorexia, weakness, difficulty feeding, † RR & HR, death in 2-10 d, Abortion reported

- Hyperexcitability or depression
- Extensor spasms or convulsions
- Seizure in severely affected
- Excessive chewing or salivation
- "Barking" abnormal vocalization
- Abnorm respiration Spinal cord CS
- Weakness in front, hind, or all limbs
- Ataxia
- Depressed spinal reflexes
- M/b normal behavior w/ periods of worsening neuro functions
- BAR (opposite of septicemia)

Lack affinity for mare



. CS (Rule out other causes of CS)

- History of hypoxia m/b
- Lab: Normal WBC, fibrinogen & electrolytes (opposite of septicemia)
- Postmortem: pathologic lesions associated w/ spinal cord
- Scattered foci thru/o brain, ischemic necrosis & cerebral edem
- No way to definitely Dx
- · CSF tap, m/ be normal or evidence of xanthrochromia

DDx:

- · Senticemia
- Meningitis (pg 268)
- Metabolic disturbance:



- Good: for those managed intensively & not septicemic
- Complete recovery w/ no neuro deficits in wk Best Px associated w/ normal IgG levels at 18 hrs
- · Bad Px if 2° infections due to FTP or if never stood
- Recovering foals, plateau of CS at 24 or 48 hrs. improve after 3 d
- · Guarded if no improvement in 4 d

Can be Tx successfully -Labor intensive

/(Treatment

- Control seizuring & aimless thrashing, diazepam, then maintenance w/ barbiturates, pentobarb TID then wean down to lowest level to control seizures
- Corticosteroids not helpful
- Manual restraint on padded surface when down
- Once coherent
- Relearn to walk, nurse & follow its mother
- Colostrum 1st 6 hrs of life
- 1 L of colostrum (Sp gr. > 1.06)
- · If FPT give plasma & ABs
- Monitor for infections
- Anti-ulcer Rx empirically, monitor for gastric ulcers
- DMSO m/ b benefit for cerebral edema





Rabies

M8k 966, 925; Mk 575, 604, 1608; IM 922; I2M 1024; C4T 336; C3T 545; EM&S 749; M 382;



 Rhabdovirus (Lyssavirus) Worldwide, except some free islands

Progressive, fatal neurologi-

cal diz of warm blooded animals

- Reservoirs: skunks & raccoons, bats, foxes; less dogs & cats because of vaccinations - Impossible to eliminate in endemic areas
- Asymptomatic carriers Pathophysiology
- Transmission: bites (in saliva)
- Migrates to CNS over peripheral nerves
- Then antigrade over nerves to salivary glands
- Shed in saliva (dies in dried saliva)

Human immunization

for veterinarians

strongly recommended

If unvaccinated & then

exposed - Tx is no fun

• IP 3 wk - 3 mo (m/b > 6 mo)

CS highly variable

- Dysfunction of nerves & brain
- Behavioral changes (1st)
- ± Excess "salivate"
- Progressive lameness, ataxia & posterior paresis
- Prodromal, excitative & paralytic phases
- FURIOUS FORM
- Muscle tremors, tenesmus
- Aggression, attack, pursue & bite (as progresses, less beligerent)
- Crash frantically through fences, etc.
- Hypersexuality, mounting inanimate objects
- Pruritic, regional
- Ataxia: proprioceptive deficits
- Recumbent, convulse & die w/in 2-4 d

DUMB FORM:

- Severely depressed
- Frothy sativation, inability to swallow (pharyngeal paralysis), "hydrophobia" (inability to drink) inappelent, lemp > 103° F, drooped head Flaccid paralysis, wide base stance, difficulty rising
- Hypersexuality, paraphimosis
- Flaccid facial mm., tail, tongue, bladder (dribbling) Grinding teeth (odontoprisis), head pressing, circling,
- Die: laryngeal paralysis
- PARALYTIC FORM (common in cattle):
- Flaccid tetraparesis or paraparesis, hyporeflexia, recumbency
- Comatose or convulse, thrash wildly before death in 10 d
- Forms can overlap

Daynloaded From Vet-a Books com

All forms rapidly fatal

- Notify authorities
- · PM done by state officials
- FA staining tech. (Ag/Ab reaction)
- Negri bodies (microscopic brain sections [hippocampus], not seen in all cases)
- Intercerebral inoculation mice examine brain in 5 d

DDx - anything

- Abnormal behavior Digestive disorders
- Injury
- FB in mouth
- Poisoning (Lead) (pg 269)
- · Early infectious disease

Inability to swallow

- Choke (pg 23)
- FB lodged between teeth
- Ingestion of irritating substances
- Obstruction

CNS

- Hepatoencephalopathy (pg 258, 84)
- Leukoencephalomalacia (pg 267)
- Togavirus encephalitis
- EHV-1 myeloencephalitis (pg 254)
- Space occupying masses
- Meningitis (pg 268)
- Protozoal myeloencephalitis (pg 249)
- Toxicity
- Lameness Musculoskeletal problems

Euthanize & notify authorities

Valuable animal, isolate for



- Control: Unlikely to eradicate from
- endemic areas Mass vaccination of dogs & cats
 - Approved vaccines: safe & effective in horses (see pg 12)
- 3 & 4 mo-old Revaccinate annually recommended in endemic areas were horses
- have contact w/ wild animals Islands remain free because of 6 mo quarantine of entering dogs & cats



Horses & humans resistant to pseudorables

DDx for abnormal behavior, anything Progressively fatal neurologic diz CS: Furious, dumb & paralytic forms

Dx: Authorities

Tx: Euthanize & report

Equine Motor Neuron Diz

NERVOUS SYSTEM

Equine motor neuron diz, **EMND**

Condition

C4T 321, 314

- New diz, first reported in 1990, rare Progressive muscle atrophy, weakness & debilitation
- NE & NC USA (most cases)
- Adults: 2-25 years Individual horses housed at riding stables
- Absence of grazing for 1 year, poor quality hay Quarter horses over represented (boarded more?)

Facts/Cause

- Cause: unknown
- Resembles Lou Gehrig's diz (amyotrophic lateral sclerosis/ALS)
- Oxidative diz? Preference for Type 1 muscle fibers, deficiency in dietary antioxidants
- Pathophysiology: motor denervation atrophy
- Particularly of type 1 muscle fibers

- Presentation/CS · Muscle fasciculation: more pronounced if forced to stand for long periods (stocks, trailer) May disappear later
- Marked weight loss w/in 2 months
- Muscle atrophy symmetrical: scapular, triceps, quadriceps, lumbar & neck muscles avenous appetites, coprophagia
- · Muscle weakness: sternal recumbency, frequently (head on ground), inability to hold head & neck up Hyperesthesia (increased sensitivity) of muscles
- Stand w/ limbs close together under body
- Constantly shift weight on hindlimbs (unable to lock stifles), buckling of carpus
- Ataxia rare, no proprioceptive loss, normal tail & anal tone
- Walk better than stand
- Short strided gait, especially in tight circle
- Respiratory distress
- . Signs may stabilize & even improve for a few months None return to effective use, euthanized

Diagnosis

- · No definitive antemortem Dx None - Euthanasia
- . Hx (stabled horses), CS
- Laboratory
- Mild + in serum muscle enzymes, CK (creatine kinase, normal 90-350 IU/I) & AST (aspartate transaminase, normal 150-550 IU/I) Low plasma Vit E
- CSF: + protein & creatine kinase
- EMG (electromyography): frequently denervation (positive sharp waves, fibrillation, Pseudomyotonic bursts)
- Necropsy:
- Degeneration of peripheral molor nerves
- Neurons swollen, chromatolytic & ghostlike
- Aggregates of glial cells in ventral horn



Treatment

Vitamin E supplementation

Access to pasture



- Botulism (decreased tail tone)
- Laminitis
- Myositis/rhabdomyolysis
- White muscle diz
- · Spinal cord dizs (alaxia) Cervical stenosis
- Degenerative myelopathy
- Protozoal myelitis (asymmetry)
- Ilíac thrombosis
- Other causes of neuromuscular diz



effective use, euthanized

- Divers TJ et al. Eq motor neuron diz. Proc 13th ACVIM Forum, p918, 1995
- Divers TJ et al, Eq Motor neuron diz. Cont. Ed 14(9)1222, 1992
- Cummings JF et al. EMND; a preliminary report
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- Hahn CN et al. Does EMND exist in UK. Vet Rec 132 p133, 1993

Px: Grave Ascariasis 283 Epizootic cellulitis Epizootic lymphangitis Amyloidosis 289 Angioneurotic edema 281 Ergotism Erythema multiforme Arabian fading syndrome 278 Aural plaque 292 Exuberant granulation Basal cell tumor 290 Eve Black flies/Buffalo gnat 287 Eye laceration Bullous pemphigoid 289 Fibroma

NE & NCentr USA, Motor neuron degeneration, Cause?

CS: Weakness, fasciculations & muscle atrophy

Dx: Hx, CS, Muscle enzymes

Tx: None, euthanasia

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Mange

Lymphosarcoma, cutaneous 291

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Presentation/CS

. Suppurative crusts (proliferative)

"Paintbrush" appearance (pus

Back, rump, head, muzzle, neck,

If untreated, can generalize

Mange (pg 283)

Saddle sores (pg 277)

Ringworm (pg 277)

· Pemphigus foliaceus

Seborrhea (pg 279)

Pus under yellow crusts

sticking to matted hair)

lower hindlimbs

DDx

Painful, not pruritic

Hair breaks & falls off

Self limiting

weekly until resolved

or pen-strep IM

Oxytetracycline

Prevention:

Keep dry

mild soap

Treatment

Remove crusts w/ brush &

Dispose of infective crusts

Betadyne® (povidone-iodine) sham-

Severe or generalized infections

poo or chlorhexidine daily for 7 d, then

- Oxytetracycline or penicillin

Condition

Dermatophilosis, "Rain scald"

Streptothricosis M8k 613; Mk 787, C4T 382, 368; I2M 1411; IM 1266, E 827; EM&S 1694; M



351; LA-D 136



Bacteria Dermatophilus Damaged skin + Zoospores CS: "Paintbrush" crusts

Dx: Hx, CS -"Railroad tracks"

Tx: Bath, Keep dry

Equine staphylococcal cellulitis

IM 1269; I2M 1413; EM&S 1696

Staph. aureus

CS: Dissects, sloughing skin Tx: Aggressive ABs *C&S)

Facts/Cause

 Superficial bacterial infection · Dermatophilus congolensis

(gram pos., branching aerobic organism) Zoospores germinate in moist, damaged skin to form mycelium

- Mycelium proliferate in living layers of

Suspected in soil, but can't be isolated

· Enter through damaged skin

- Macerated or traumatized - Rainy season (constant wetness

damages skin) External parasites

- Non-hygienic conditions

Ruminants, horses

 Transmission: direct contact w/ reservoir host, fomites or insects, crusts

- Crusts contain organisms up 42 months (accounts for repeated outbreaks in contaminated areas)

Severe, deep suppurative process, in-

fection dissects through tissue planes

Cause unknown (handlers & grooming

Staph, aureus, S. intermedius

Thoroughbred racehorses

equip.?)



Initially: Acute swelling &

(pg 280)

Rapidly dissects along fascial planes

Overlying skin devitalized Frequent sloughs

Sequelae:

Laminitis (same or opposite limb)

Bacteremia

Osteomyelitis

· Hx, CS

Direct smear of pus

 Minced preps of crust, on slide (microscope)

Diagnosis

Stain w/ Diff-Quik®

-"Railroad track", long chains of cocci, branching, filamentous

Il negative submit crusts or punch biopsy to microbiologist before ruling out (R/O)



CS (clinical signs)

• Hx, CS

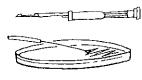
Biopsy

Gram stain pus

Gram positive cocci

· Culture & sensitivity for Tx

Bacterial culture & biopsy



Aggressive & early

 Broad spectrum ABs (pen G & gentamicin sulfate or trimethoprim-sulfamethoxazole) until Culture & Sensitivity

· Remove underlying factors:

(damage superficial layers of skin)

moist conditions, parasites

♣ Edema, promote weight bearing, re duce chance of laminitis - NSAIDs

Hydrotherapy

Support wraps



Betadyne

Px: Guarded for complete recovery

Tack & hygiene: eliminate

Time: mild cases m/ resolve w/o Tx

Betadyne® or peroxide solution

ABs ointments w/ no steroid.

Topical Tx m/not cause resolution

Trimethoprim Sulfa, orally by owner

Rested, no contact w/ saddle or tack

ABs

Furunculosis or unresponsive

folliculitis: Systemic ABs,

Sedate because painful

- Clipping areas & cleaning w/

predisposing factors

Severe cases

Saddle sores Folliculitis/ Furunculosis.

Impetigo, Pyoderma, Collar galls

M8k 718, 617; Mk 828, 829; IM 1268; I2M 1413; EM&S 1696; E 813: C4T 383 C3T 700: M 344; LA-D 134

Inflammation of hair follicles

mon in horse & goat Impetigo common in cattle & goats

· Cause: #1 Staph.

- D. congolensis, Corynebacterium pseudotuberculosis, C. equi

Predisposing factors

- Trauma to skin from tack

. Points: contact points of saddles on trunk & withers

Insect bites

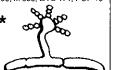
Pastern folliculitis (special form)

- Staphylococcus spp. or B- hemolytic strep. Limited to palmar/plantar pastern or fetlock of 1 or more limbs

Hair follicles, tack CS: Papules, scabs, alopecia Tx: Hygiene, time

Ringworm, Dermatophytosis, Girth itch

M8k 628; Mk 791; IM 1272; I2M 1419; E 823; EM&S 1698; C4T 384, 368; C3T 698; M 353; LA-D 171; POP 18-



Fungus, Trichophyton CS: Alopecia, crusting Tx: Self limiting

Folliculitis & furunculosis com-

Mycotic - fungal

Trichophyton equinum,

Microsporum equinum, T. mentagrophyles, M. canis, M. gypseum Gram +, branching, aerobic organism - Generally invades nonliving layer of skin

Saprophyte in soil Young >> adults (lack of exposure & immunity)

 Horse & cattle >> sheep & goats Predisposing factors

Resistant to infections (diz, poor nutrition, crowding, immunosuppressive drugs)

Environment: hot, humid weather

Winter stabled animals, anytime

Transmission: contaminated grooming equipment, saddle blankets & harnesses Papules

Multiple 3 mm foci of erect hairs - Each hair projects from center of a papule

Papules m/ regress, or

Enlarge, exude serum & scab

Alopecia (denuded areas common)

Painful, not pruritic

Pastern folliculitis (special form)

Papules

 Ulceration & suppuration if left untreated

• Folliculitis = inflammation of hair follicles 2°

to bacterial infection Impetigo = superficial inflam, not involving hair follicles

• Furunculosis = Inflammation rupturing hair follicle & extending into surrounding dermis & subcutis

Carbuncle or boil = coalescing areas of furunculosis forming fistulous tracts

DDx:

Rain scald (pg 276)

Ringworm (pg 277)

Prognosis: Good 🥪

Problem w/ chronic pastern folliculitis deep w/ scar tissue & fissures

 Self limiting (1-4 months) - Betadyne (povidone iodine) & crust re-

Fungal products (topical) to

♣ spread (lime sulfur, Chlorox, tamed) iodine shampoos, thiabendazole (Tresaderm®), miconazole (Conofite®), Na tolnaftate (Tinactin®), chlorhexidine, fulvacin in feed

 Orthocide® (captan) plant lungicide from nurseries, not recommended

Systemic Tx controversial; griseofulvin not recommended



Prevention: disinfect grooming equipment & tack frequently



 Alopecia (hair breaks & falls off) • Crusting (not wartlike as in cattle)

Early looks like urticaria (hairs

stand erect) then crusting (horse) · Head, around eyes, neck, shoul-

ders & sides of thorax Pruritus mild or absent

DDx:

Mange (pg 283)Folliculitis (pg 277)

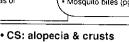
• Rain scald (pg 276)

Ringworm (pg 277)

 Pemphigus foliaceus (pg 280) Flat sarcoid (pg 290) Hives (pg 281)

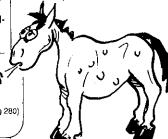
Pemphigus foliaceus





Fungal cultures (of broken hairs at





Purpura hemorrhagica

See CIRC, pg 138; Sequel to other diz, #1 Strangles, Arthrus Type 3 hypersensitivity, vascular damage, Ag/Ab complexes · CS: 2-4 wk after resp. infec., pitting edema of head, belly & limbs, wheals, petechiation, dyspnea, diarrhea, anemia

• Dx: Hx, PE, skin biopsy (vasculitis), mild anemia, normal platelets

• Tx: Aggressive: ABs (2 x), NSAIDs, steroids, tracheotomy; some refractory to Tx

Hives, Urticaria, Nettle rash

M8k 612; Mk 867; IM 1260; I2M 1405; EM&S 1651; M



· Wheals or plaques: localized edema in dermis

• #1 horse, but in all domestic species

Type 1 hypersensitivity

. Precipitating or intensifying factors - Pressure, sunlight, heat, exercise, psychologic stress, genetic abnorm.

Cause: Hives

Drugs: ABs (pen, & sulfonamides) NSAIDs (bute), lidocaine

Ingestion (feeds)

 Inhaled antigens (pollens, molds, & dust) . Insect bites blamed, but rarely cause

Stinging nettle

Chemicals - carbolic acid, turpentine, carbon disulfide or crude oil

Horse #1, Type 1 hypersensitivity

CS: Wheals, edema

Dx: Hx, CS

Tx: Spontaneously recover

Erythema multiforme *

IM 1264; EM&S 1653

Rare in horse, Benign, Self limiting, M/b hypersensitivity reaction, 50% of time cause never found

CS: Urticaria-like lesions which tend to persist

Tx: Remove cause

Px: Spontaneous resolution w/in 3 months

• Wheals or plaques: transient swellings in | • Hx, CS: wheals skin appear few min or hrw/in exposure to - Elevated, rounded, flat-topped, 0.5-5 inches

in diameter, m/b depressed in center Anywhere: back, flanks, neck,

eyelids & limbs

 Severe m/b preceded by fever, anorexia or dullness

Excitation & restlessness

 Advanced: plaques on mucous membranes of mouth, nose, conjunctiva, rectum & vagina

Develop & suddenly disappear generally (w/in few hours)



Pathophysiology - Hives:

Wheals result from vasodilation & leakage of fluids from small vessels

Immunological & non-immunological factors trigger release of mediators from mast cells & basophils that cause wheals

immunological Type I hypersensitivity (IgE) most common immunological cause

• Pit w/ pressure

Intradermal skin testing

DDv.

 Other nodular diz (they don't pit on pressure)

Infec. granulomas

· Chronic urticaria (pg 281)

Neoplasia

Amyloidosis (pg 289)

Erythema multiforme (pg 281)

Cysts (pg 292)

· Rain scald (pg 276)

Panniculitis (pg 289)

Foreign body granulomas

 Purpura hemorrhagica (pg 144) Calcinosis circumscripta (pg 289)

Spontaneously recover w/o Tx

Avoid allergen

- Change feed & see if better

Short acting corticosteroids

Prednisone or prednisolone (gradually #

dose & give every other morning)

Dexamelhasone (long acting) causes

laminitis

Hyposensitization when from inhaled allergens

Antihistamine questionable, m/ cause urticaria if type 4

· Epinephrine if life threatening

Local Tx not generally required (cold packs,

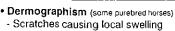
vinegar, or alcohol (70%)



Good - most





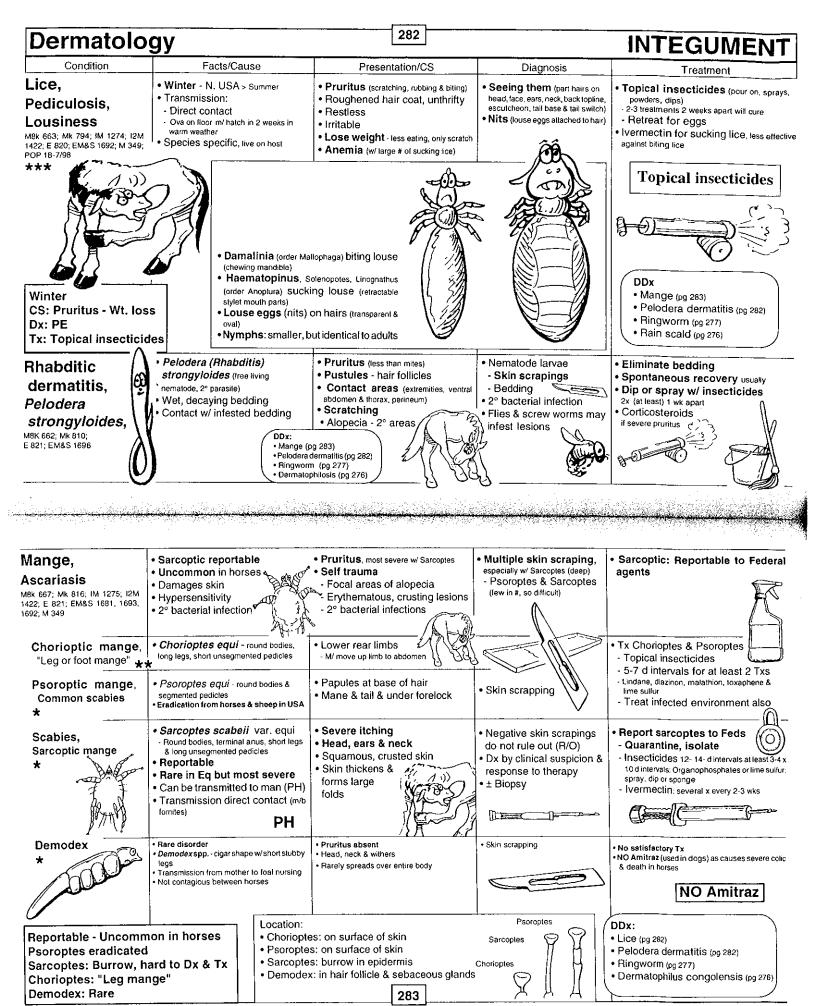


(urticaria-like skin testons) - No clinical significance

Angioneurotic edema; life threatening variation of urticaria

Diffuse SQ edema often localized to head limbs & perineum





Summer sores.

Cutaneous habronemiasis,

Jack sores, Bursatti M8k 658; Mk 806, 201; E 798; IM 1283; I2M 1431; EM&S 1673; M 342; S 274; POP 18-7/98

Sweet itch,

Queensland itch,

hypersensitivity

Mk 804; IM 1277; I2M 1424;

Allergy, seasonal

CS: Dorsal itching

Dx: Hx, CS, Tx

Tx: Steroids

Culicoides

E 814; C3T 693





H. microstoma &

Drashia (Habronema) megastoma

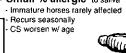
Larvae of stomach worm

- Emerge from flies feeding on wounds or eye or genital excretions
- Migrate & irritate tissue
- Granulomatous reaction
- . Hypersensitivity to dead/dying larvae
- Develops in same horse year after year
- Springtime (fly season)
- Recurs in subsequent years

Stomach worm, Migrates CS: Granulomas: eye, penis, legs Tx: Difficult



- Allergic reaction Type 1 Culicoides gnats (midge, no-see-
- ums, punkies, sandflies) - Small, blood sucking flying insects
- Breed in stagnant water Can only fly 1/4-1/2 mile from water
- Only warm weather (April Nov.) Before or during dusk
- Small % allergic to sativa Immature horses rarely affected Recurs seasonally



- Uicerative, nodular & tumorous masses
- Chronic slow healing lesion
- Locations
- Medial canthus of eye
- Male genitalia
- (sheath, urethral process) Lower extremities

Life cycle:

- Adults in stomach: normally little problem in stomach, except Drashia
- Eggs passed in feces House or stable fly intermediate host
- Normally horses ingest fly

 Larvae m/ emerge from flies feeding on
- wounds or from eye or genital excretions Can't penetrate normal healthy skin

• Pruritic

- Dorsally: poll, mane & tail (rarely belly), Forehead, neck, withers, shoulder, rump
- Self trauma
- Crusting alopecia (excoriation)
- Hair & mane broken & matted
- Severe chronic cases
- Lichenification (thickening of skin due to rubbing) & scarring



Hx of midge exposure

Nonhealing ulcerative

SCC (pg 291)

Sarcoids (pg 290)

Proud flesh (pg 292)

Rain scald (pg 276)Ringworm (pg 277)

· Pruritic, crusting alopecia of dorsum (mane & tail region)

greasy skin granulomas

cified, dead larvae

(spiny knobs on tail)

trale, larvae

DDx:

-Contain rice-sized, yellow, cal-

Scraping: rarely larvae found

Biopsy - granulation tissue, eosinophilic infil-

≪=lvermec

- Warmer months
- Positive response to Tx

DDx:

- · Onchocerciasis (nonseasonal, less pruritic, head, neck & belly) (pg 285)
- Haematobia irritans (horn fly) ventr. midline (pg 286)
- · Stomoxys calcitrans (stable fly) back, chest, neck & legs (pg 287)

Most give poor results, difficult to eliminate due to locations

- Insect repellents Organophosphates topically
- Surgical removal or cauterization or cryosurgery & hope doesn't granulate in
- Ivermectin eliminate adult in stomach. slows down progression of diz
- Steroids, systemically & intralesionly
- Prednisone or prednisolone (10-14 d)
- If conjunctive involved
- Frequent steroid ophthalmic sol. Topical DMSO-corticosteroids

Prevention: Fly control

- Regular removal of manure
- Regular anthelmintic Tx

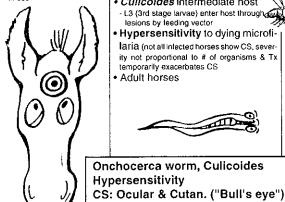
- Move 1/2 mile from midge habital Slable before & during dusk,
- midges stay out of barns
- Fan on horses (fly poorly)
- Insect repetient
- Steroids: prednisolone (PO BID Dexamethasone (PO, BID) for severe cases
- + Dose gradually after 5 ds
- to lowest alternate daily dose Antihistamines
- ineffective in most

culaneous hypersensitivity dize of horses



Cutaneous onchocerciasis: Diffuse midline dermatitis.

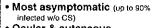
M8k 659; Mk 808; IM 1279; 12M 1427; EM&S 1687; E816;



Mk 781; IM 1281; I2M 1429;

Onchocerca cervicalis

- Adults coiled in funicular part of ligamentum nuchae
- . Produce calcified nodules
- Microfilaria
- . Migrate through connective tissue to dermis
- . Ventral midline, lower eyelid & laterallimbus of eve
- Culicoides intermediate host L3 (3rd stage larvae) enter host through
- lesions by leeding vector Hypersensitivity to dying microfilaria (not all infected horses show CS, sever-
- ity not proportional to # of organisms & Tx temporarily exacerbates CS
- Adult horses



- Ocular & cutaneous
- Ocular
- Uveitis, conjunctivitis, keratitis, depigmentation of lateral limbus
- **Cutaneous lesions**
- Diffuse/patchy alopecia, erythema & scaling
- Ventral midline, face, base of mane, craniomedial forearm, cranial pectoral
- "Bull's eye" lesion in center of forehead
- . Nonseasonal & nonpruritic
- ± Edema on ventral midline & face

• Ringworm (pg 277)

DDx:

- · History, CS Exclusion of DDx
- . #1 Response to therapy
- Microfilaria preparation Not diagnostic because many
- have no CS - Lack of microfilaria doesn't



- Ineffective against adults so re-
- currence possible in 2 mo, but most free for 6-12 months

Minor adverse reactions in 25% of horses

- Retreatment at 4-mo intervals
- Banamine® for ventral edema

• Ivermectin TOC (orally)

- Most improve in 2-3 wks

Corticosteroids for severe reactions (usually unnecessary because most resolve in 1-3 days)



Microfilarial preparation

- Punch biopsy (6 mm)
- Half preserved in buffered formalin histopath.
- . Eosinophilic & lymphocytic perivascular dermatitis (nonspecific & common in other parasitic dermatoses)
- Half on dampened sponge in closed container
 - . Mince w/ razor blade on slide, add few drops of nonbacteriostatic saline
 - . Incubate for 15 min at room temperature
- . Scan at 4x objective for slender, delicate microfilaria
- . If negative, incubate overnight & reexamine

Mange mites (pg 283) Dx & Tx: Ivermectin Nodule w/ breathing hole Larvae (grub) of Hypoderma Warbles, . Usually only 1 or 2 grubs Season bovis & H. lineatum Small nodular lesions on Hypoderma Common & serious problem in cattle

- withers or back Sporadically in horses Breathing pore on pastured near cattle nodule Adults bee-like, spring & early summer
- Posterior paresis, neuro deposit eggs on legs or lower body, rump Migrate through body, see NEURO pg 251 pg 251

285

Ventral midline dermatitis (horn fly) (pg 286)

Sweet itch (no improvementw/ivermectin) (pg284)

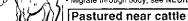
- - DDx:
- Nodular necrobiosis Mastocytoma
- Sterile nodular panniculitis
- Amyloidosis

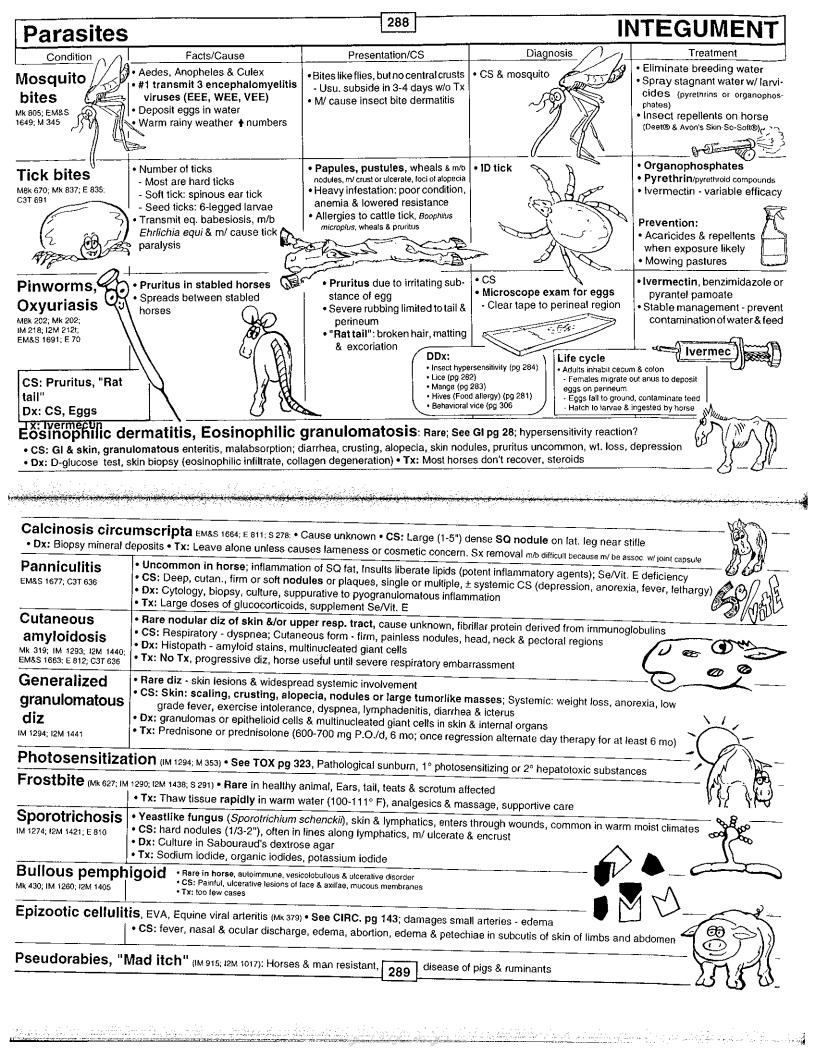
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· Enlarge breathing hole & extract grub







and the second s

onchocerciasis

M8k 364; IM 1244; I2M 1388; E 292; EM&S 1107, 1102

• CS: Conjunctivitis & keratoconjunctivitis, lacrimation, blepharospasm, sm. nodules & corneal opacities, depigmentation (vitiligo) bulbar conjunctivitis, Uveitis, Chorioretinitis, "butterfly-shaped" pattern around optic disc, cutaneous ventral thorax de

• Dx: CS; Corneal or conjunctival biopsy: microfilaria rarely found • DDx: Squamous cell carcinoma, habronemiasis, mycotic infection • Tx: Steroids (Pred-Forte® [prednisolone acetate) & dexamethasone): to control inflammation - 4-6 x/d, once inflam. controlled, eliminate microfilaria

Ocular habronemiasis

M8k 364; IM 1245; I2M 1389; E 1276; EM&S 1101; M 321; C2T 440; S 608

 Larvae of Habronema muscae, H. microstoma or Draschia, deposited on ocular tissue by flies CS: Raised proliferating wounds on med. canthus, contain sulfur granules

• Dx: Biopsy - larvae • DDx: Neoplasia (SCC), sarcoids, phycomycosis, Onchocerciasis, foreign body reaction, exuberant granulation tissue Tx: Oral ivermectin, fly control

Thelaziasis

M8k 363; I2M 1390; EM&S

- Thelazias worm commensal in conjunctival sac, 15-38% of horses affected
- CS: usually asymptomatic, m/b conjunctivitis, superficial keratitis, neovascularization, edema & ulceration

• Dx: Direct visualization of adult worm in conjunctival sac (8-18 mm long)

• Tx: Manual removal (saline flush or forceps after topical anesthetic); topical organophosphates, Ivermectin not constantly effective

Intraocular parasites

IM 1247; I2M 1391

Setaria most common; aberrant migration; into aqueous humor; also Dirofilaria immitis & Onchocerca cervicalis

CS: Serious intraocular inflammation
 Dx: Visualize worm in aqueous humor

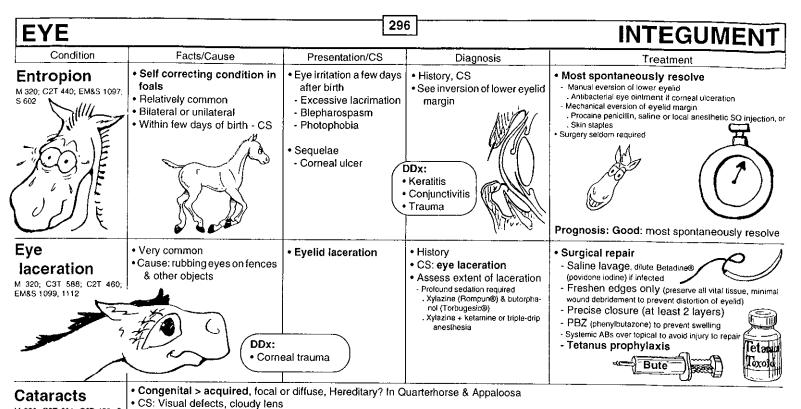
Tx: Symptomatic anti-inflam, Tx & surgical removal

295



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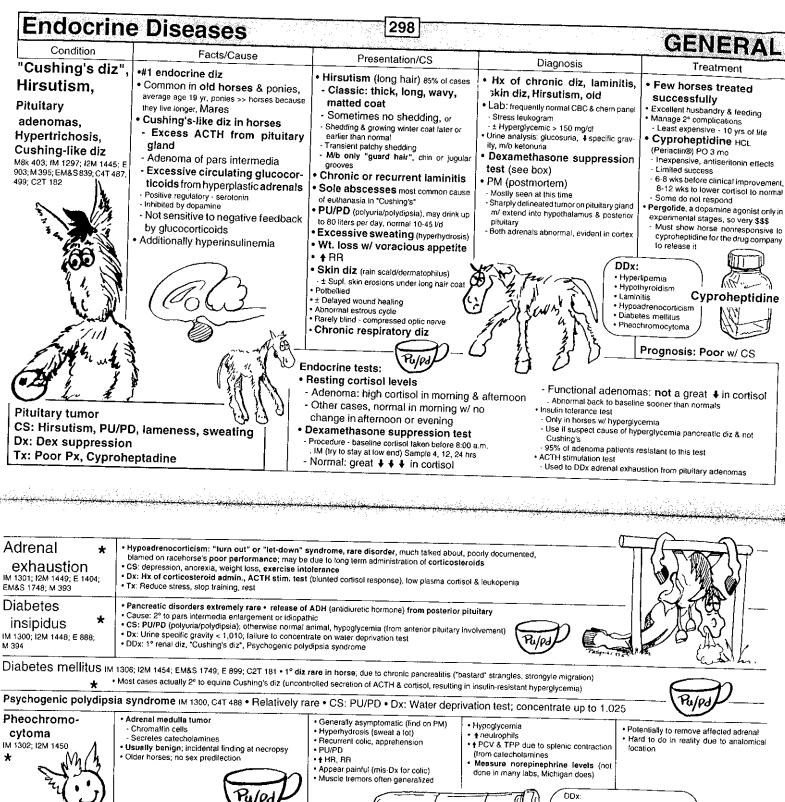
M 326; C3T 601; C2T 456; S 649

• Dx: Visualize, absence of menace response if severe, Ophthalmoscope • DDx: Synechiae, Lens luxation, Uveitis

• Tx: Cataract surgery in foals at a few months of age has good results; Acquired: surgery only if bilateral; difficult & often unsatisfactory

Retinal diseases & Optic nerve M 327; C2T 458: Rare, normal appearing eye; CS: blindness, night vision problems; Dx: Ophthalmoscope; No treatment

A =		A.C		CENIEDA	
Aerophagia	307	Hirsutism	298	GENERA	
Adrenal exhaustion	299	HO, HPO	305		
Adrenal medulla tumor 299		Hyperhydrosis	298		
Adult hypothyroidism	301	Hyperkalemic periodic paralysis	305	Pheochromocytoma	299
Aerophagia 307		Hyperparathyroidism	300	Pica	307
Anhydrosis	299	Hyperthyroidism	301	Pituitary adenomas	298
Anorexia 304		Hypothyroidism	301	Poll evil	303
Anthrax	302	Hypertrichosis	298	Psychogenic polydipsia syndrome	299
Big head diz	300	Hypotrophic osteopathy	305	Self mutilation	306
Biting	306	HYPP	305	Shaking	306
Bran diz	300	Kicking	307	Soil eating	307
Borreliosis	302	Let-down syndrome	299	Splenic fever	302
Brucellosis	303	Licking	307	Shying	307
Charbon	302	Lyme diz	302	Stall walking	306
CID	303	Malignant edema	303	Stereotypic pacing	306
Clostridial myonecrosis	303	Malignant hyperthermia	303	Striking	307
Combined immunodeficiency (CID)	303	Malnutrition	304	Sweating	299
Crib biting	307	Marie's diz	305	Thiocyanates (SCN)	301
Crib-whetting	307	Milzbrand	302	Threatening behavior	306
Cushing's diz	298	Mobile alarm	306	Tongue dragging	307
Diabetes insipidus	299	Myotonia	305	Tularemia	302
Diabetes mellitus	299	Neonatal hypothyroidism	301	Turn-out syndrome	299
Drycoat	299	Nonsweater	299	Weaving	306
Fistulous withers	303	Nutritional 2° hyperparathyroidism	300	Weight loss	
Geophagia	307	Pacing	306	Wind sucking	304
Head nodding	306	Pawing	306	Wood chewing	307
9			500	ood onewring	307



Anhydrosis, Nonsweater

IM 36, 1302; I2M 1451; E 837; C3T 703; C2T 187; Pop 44-7/98

· Inability to sweat in hot climates - "Drycoat", "nonsweater"

- Gradual onset

Some can't sweat in summer, but can

in winter 1° in SE & SW USA & tropical areas

Cause unknown

Neurotransmitter control of sweat glands implicated

Theory: lack of adrenergic receptors in skin Any age, related to heat stress

Can in winter not in summer



- Total body or limited to focal areas

299

RR to dissipate heat

Febrile, especially when

Exercise intolerant

exercised

Alopecia

on face

· Piluitary adenomas (sweating also)

 Anhydrosis (inability to sweat) Hx, CS: Inability to sweat in hot weather at first Lab: Stress leukogram Progresses - don't sweat any time

 Intradermal epinephrine test in neck region

- Use control of sterile saline

0.5 ml of 1/1000 & 1/10,000 + control

Normals will sweat w/n 1 hr

Abnormal - lotally or partially anhydrotic

. No response or delayed response of

Some in early diz will sweat on this in winter, not in summer

 Minimize concentrates in diet Electrolyte solution in drinking

Misting fine, rest until winter

Move to cool environment



Endocrine Diseases Facts/Cause

300

Presentation/CS

Early stiff gait, shifting leg lameness

- Enlarged painful joints (bilateral)

- Lamina dura: resorbed, causing

"Big head": fibro-osteodystrophy of

facial bones, 1° maxilla & mandible (sym-

Teeth fall out & difficult mastica-

Diagnosis

GENERAL Treatment

Correct diet (take off bran)

Alfalfa roughage w/ high

Supplement w/ limestone

Nutritional secondary hyperparathyroidism. Big head diz, Bran diz

IM 1305; I2M 1454, 1469; E 895; C3T 119, 207; M 128; Pic 24



& low in roughage (high in Ca) - Vit. D toxicosis

Absolute Ca deficiency

(also pigs and ruminants)

to understanding nutrition

. Overzealous supplementation

. Plants high in Vit. D activity (Cestrum diurnum [wild jasmine, day cestrum])

Too high in grains (especially bran, low in Ca)

Young, growing horses & ponies

Problem of past, less prevalent now due

Cause: diet - excessive P over Ca

"Pampered" horse diets

Pasture high in oxalate plants (unavailable Ca) (Selaria sphacelata, Cenchuris ciliaris, Pani

Weight loss

metrically)

teeth to fall out

Lameness

tion



CS: "Big head", lameness, teeth

 Analysis of feed for Ca, P & Vit D content

• Lab: **∱ Serum Ca**

- Normal serum PO4

. A Renal secretion of phosphorus & + Ca excretion

◆ ALP w/ overt bone diz (young naturally have higher ALP)

Rads support Dx, done in field

Loss of lamina dura

Delineation of mandible &

maxilla

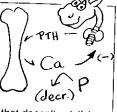


Prognosis:

Ca content

Good if lame, not "big head"

Resolution in 1-2 mos w/ proper diet Poor if "big head", will never resolve due to bony changes & teeth



Growing foals,

♣ Ca

CS: Big Head, lameness, teeth loss, wt. loss

Dx: Hx, SC, Lab Tx: Change diet



Pathophysiology:

 Low Ca stimulates parathyroid gland to secrete excessive PTH

PTH pulls Ca from bone

High phosphorus indirectly affects by lowering blood Ca

Parathyroid hyperplasia & metabolic bone diz

Osteoclastic activity (eats bone) leads to lameness

 Osteoblastic activity (builds bone) leads to fibrohyperplasia which leads to osteoid formation (bone substance that doesn't calcify)

Results in osteodystrophy due to more osteoblasts than osteoclasts

- Widespread pathological calcification of soft tissue (arteries, tendons & ligg.)

Obscure lameness

- Microfracture of subchondral bone & resulting degeneration of articular cartilage

- Ligament tearing from periosteal attachments

- Folding fxs of long bones

Neonatai hypothyroidism

IM 1303, 1579t; I2M 1451; E 892; M 394; EM&S 1747; C4T 502; C2T 185; Tox 456, M8k 2128; Mk 283



Neonatal goiters

Dx: TSH stim, test

Tx: T4, No reversal

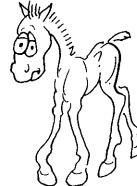
CS: Weak, angular limbs

· Hyperplastic goiter in neonates

- #1 thyroid disorder in horse & small ruminants
- Young foals
- Adult not documented
- Dam usually asymptomatic
- Association between myopathies & hypothyroidism Cause:
- Dam excessive intake of iodine (in kelp feed) or plant goitrogens; See TOX pg
- Thiocyanates (SCN) contained in plant
- . Infect foat in utero
- ^Dathophysiology:
- Low thyroid hormones cause TSH (thyroid stimulating hormone) stimulation of pituitary gland
- Leads to thyroid enlargement

Hypothyroidism

- Goiter, m/ or m/not be seen
- Poor performance
- ·Weak, lethargic
- Incoordination
- Poor suckle reflex
- Poor righting reflex
- Hypothermia (low metabolic rate)
- Angular limb deformities, tarsal & carpal bones - 2° to delayed ossification
- Tendon contraction &/or rupture
- Retarded bone development
- M/b born asymptomatic, but at 2 weeks skeletal lesions (particularly



· History, Clinical signs

PE: Grossly enlarged thyroid

♣ Thyroid hormones

- Bute will give false positive (+ protein carrier so total thyroid hormone less)
- T3/T4 levels & check against age matched controls
- Free T3 is physiologic component of this
- T3 has diurnal variation
- Drugs such as Lasix® interfere w/ binding of T3/T4
- Horses w/ systemic illness will have lower
- than normal values 1° TSH stimulation response test (1 day old foal)
- TRH response test (rare)

mentation (iodine) if hormone level is low, if CS & normal levels then deficiency occurred during critical developmental period - Therapy only effective during

Thyroid hormone supple-

low hormone levels

Developmental lesions can't be reversed



Plants w/ goitrogenic compounds Mustard family (Cruciferae)

Brassica spp

Glycine max

Linum usitatissimum

Rape seed, Mustard, Kale, Broccoli, Cabbage & Turnip Soybean

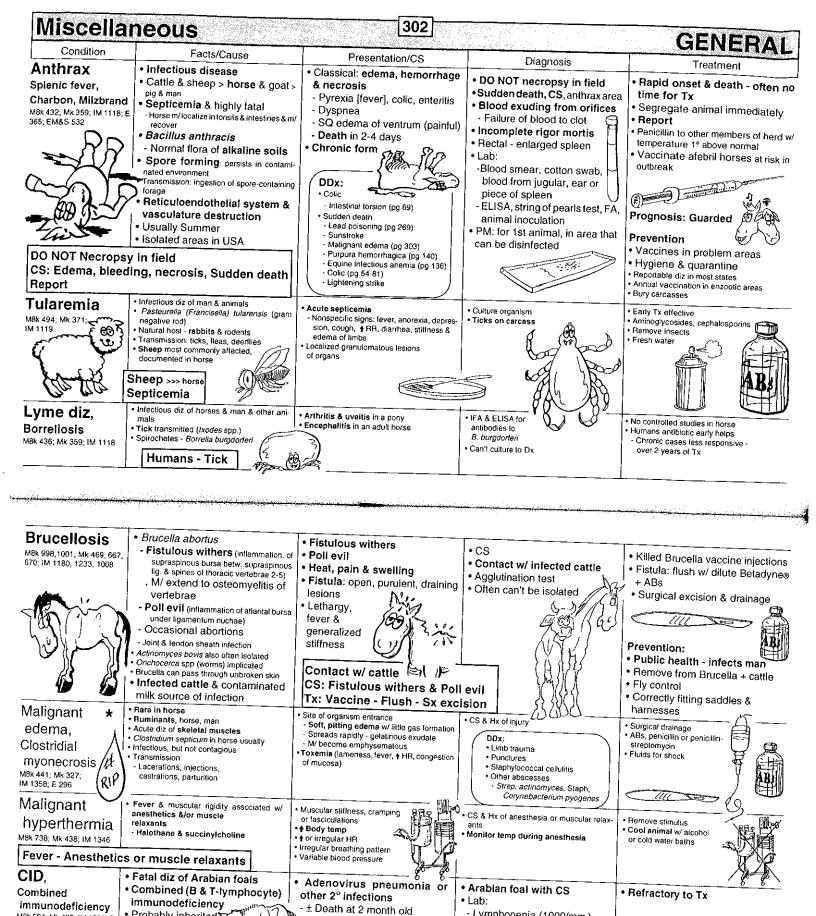
Flax

Adult hypothyroidism IM 1304; C4T 502: not documented in horses, but probably occurs (Standardbreds & Thoroughbreds)

Tx: Adult horse generally 20 mg thyroxin/o in 1000 lb horse - 2 wks to see change (and lots of little pills to grind & put inmolasses) Alternatively iodinated casein which contains 5 grams thyroxin, sprinkled on feed, also takes time to respond

Hyperthyroidism IM 1304; E 894: not documented in horses

301



Refractory to treatment

303

Lymphopenia (1000/mm)

IgM

- Intradermal PHA test (Tresponse) - Immunodiffusion for IgM, no

Postmortem: thymus difficult to ID & has abnormal architecture

M8k 584; Mk 435; IM 1592; E 420; C4T 585; C2T 217; Pop

Probably inherited

Facts/Cause

GENERA

Treatment

Treat 1° cause

Good quality feed

Associated with:

1. Anorexia - usually 2° to a 1° disease condition

Loss of appetite (desire to eat) . Complete or partial . . Usually 2° to 1° disease

Differentiate from dysphagia (difficult swallowing) by observation

♠ Nutrient demands

Physiologic (colds, weather, growth, exercise, pregnancy & lactation)

 Pathologic (sepsis, parasitism, burns, peritonitis, surgery or trauma)

Parasitism

. Common cause of weight loss

. Competition for nutrients

. Inflammation (+ requirements) .. Malassimilation, malabsorption

Migration damage to organs or vasculature Anorexia in advanced stages

3. Malnutrition

Poor quality feed

Deficient micronutrients (copper, cobalt [Vit B12] or Vit A)

Stress of disease increases sympathetic activity

♦ Epinephrine release, impaired insulin release & enhanced glucagon secretion

Hyperglycemia (↓insulin release, enhanced glycogenolysis & gluconeogen-

Granulomatous enteritis

Associated w/ malabsorption or malassimilation syndromes

Anorexia absent, good appetite w/ wt. loss

Presentation/CS

 Weight loss - short or intermediate duration

Dehydration

 Electrolyte imbalances &/or acid-base imbalances

Weight loss (pg 348) Common causes

Parasitism (pg 24, 35)

Malnutrition

Teeth & jaw problems (pg 18-22)
 COPD (chronic obstr. pulm. diz) (pg 121)

Strangles

Pneumonia (pg 111-116)
Lung abscesses (pg 113)

· Rhodococcus equi (pg 113)

Pleuritis (pg 118)
 Sand colic/impaction (pg 78)

Cribbing, Wind sucking (pg 307)

 Pyrrolizidine alkaloid toxicity (pg 89)
 S vulgaris thromboembolism (pg 75) SCC of stomach (pg 26)

· Gastroduodenal ulcers (pg 26) Neoplasia

Obstruction of small colon (pg 77)

Peritonitis (pg 53) Abdominal abscesses (pg 65)

Wound mylasis

Infectious arthritis

From Large Animal Internal Medicine, Smith (see for less common & uncommon causes)



· Note other CS: diarrhea, dysphagia, coughing & polyuria

History (Hx)

Amount of weight loss

Dietary Hx, quality of feed, feeding practices, esp. if fed in groups

Deworming Hx

- Environmental toxic substances

 Physical exam (PE): CS of concurrent disease (diarrhea) fever, dysphagia [difficult swallowing], dentition, melena, icterus,

Can animal eat? Weigh horse

Analyze diet

Adequate intake

Inadequate intake

. Adequate feed available

.. Anorexia due to 1° disease . Inadequate feed available

.. Malnutrition

Fecal exam

Microscopic - parasite ova

Fecal occult blood for melena

- Diarrhea

• Lab - CBC, PP (plasma proteins) & fibrinogen

- Inflammatory process († WBCs, † PMNs, † fibrinogen, ‡ PP: fibrinogen ratio [<10])

If anemia, calculate RBC indexes

- Chemistry: Hypoalbuminemia assoc. w/internal abscessation, malnutrition, liver, renal, granulomatous bowel diz

Function tests:

Title Oral glucose & D-xylose absorption tests to access carbohydrate absorption

Direct (conjugated) bilirubin to access fat absorption

Hyperkalemic periodic paralysis. HYPP

M8k 767, 904; IM 1340, 397; C3T 117; M387; Pop

• "Impressive" syndrome inheritable: autosomai dominant trait

in offspring of Impressive. They do better in halter classes (heavily muscled)

Pure & part quarterhorses. thus in Appys & Paint

Evident by 2-3 yr-old

Cause: Mutation of Nat channels

★ K* (potassium) during episode Not hi between episodes

Temporary loss of muscle function due to persistent depolarization of muscle cell membrane

 Variable: from no episodes to severe • 15-90 min. episodes (variable) ± Prolapse of 3rd eyelid, Sardonic grin

 Sweating (hyperhydrosis) Muscular fasciculations (myotonia)

(starts in front then generalizes, "rippling")

 Muscle weakness, stagger & sway in rear end, bulking, inability to raise head

Foal: stridor, laryngospasms

"Collapse"/recumbency, dog sitting, inability to rise (severe)

BAR, fully conscious, show no pain, anxious ± Loose feces

 CS resolve spontaneously w/o therapy, or may die (cardiac standstill)

Normal between episodes



DNA test

Colic (pg 54-81)

 Hypocalcemia Exercise myopathy

Tetanus (pg 253)

Chronic renal failure (pg 150)



Hx: 1/4 from Impressive

Muscle weakness episodes

 During episodes collect blood - ↑ K+> 5 mEq/L (normal 3-5) usually but

not always, life threatening Bradycardia - cardiac standstill

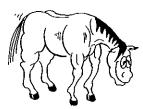
↑ Temp. usually, Hemoconcentration - Normal CPK (not altered unless

muscle damage from recumbency) After episode: K+ returns to normal (all lab parameters normal)

DNA test: accurate, whole blood EDTA

to U of Cal, Davis, takes 3 wks KCI provocation test: risky & replaced w DNA test

EMGs (electromyography): quick screening test for DNA: † activity, doublets



Emergency during episode

- 5% Na bicarbonate (1-2 mEq/kg) drives K• into cells by pulling H• out of cells

Ca gluconate IV beneficial 23% sol)

Dextrose (6 ml/kg · 5% sol.) diuresis

- Potassium-free fluids

Possibly insulin (hypoglycemia) Foals: ± tracheotomy

Bicarb

Prevention:

♦ Renal excretion of K⁺

- Acetylzolamide PO for life (a carbonic anhydrase) (induce kaluresis by proximal tubules)

Phenytoin, hydrochlorthiazide

Diet: low potassium (K) - Timothy or Bermuda grass hay

♣ Alfalfa & brome (Hi in K) Regular exercise or access to pasture



Timothy hay

Acetyizolamide

 Tx & cure 1° problem & HO will disappear

Prognosis: Depends on cause & its treatment

DDx:

Pulmonary lesions

Intra-abdominal mass

HPO, Hypotrophic (pulmonary) osteopathy, HO,

Dx: DNA probe

Tx: Bicarb.

Quarterhorse - Inheritable

CS: Episodes of weakness

Marie's diz

Prevent: Acetylzolamide, Timothy hay Associated w/ pulmonary diz

(most commonly, but not always) Pulmonary abscesses or neo-

plasia, rib fx w/ pleural adhesions - 1° Abd. disorders (ovarian tumors)

Formation of subperiosteal new bone growth of distal diaphyses of long bones, axial Nonedematous swelling of extremities (over distal diaphyses)

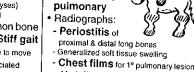
Warm & throbbing, Asymmetrical Taut skin over dorsal cannon bone

Swelling & pain of joints, Stiff gait

 Pain on manipulation, reluctance to move ± Pulmonary signs if associated

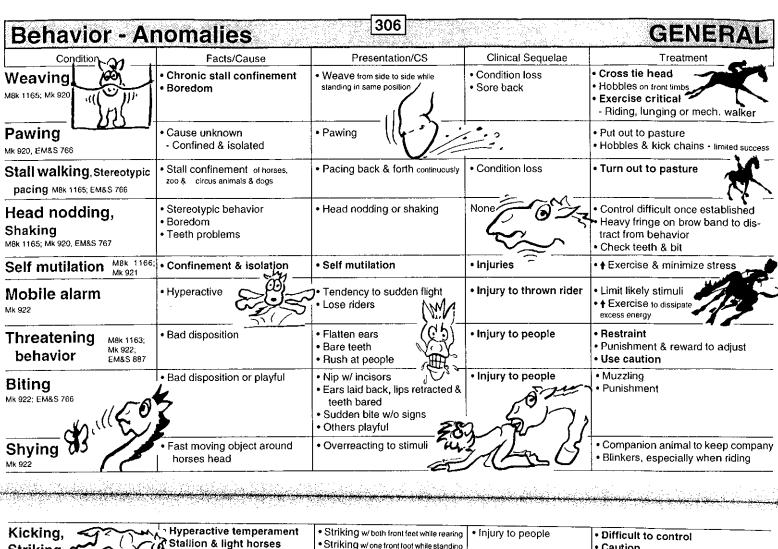
• CS: swelling + pulmonary

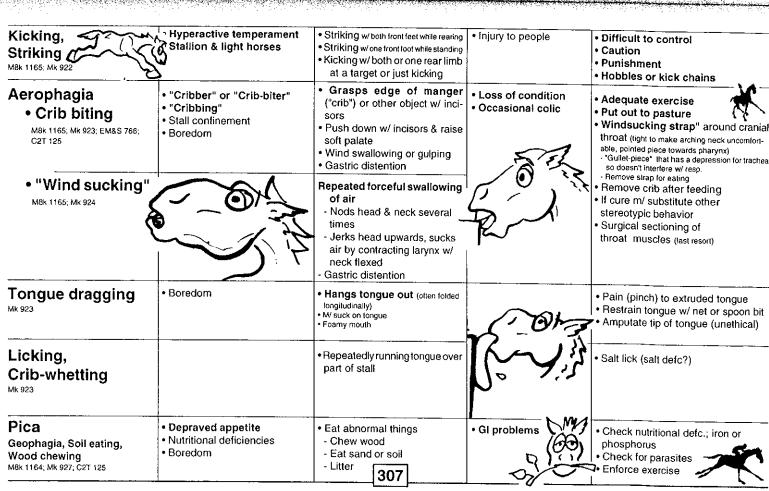
Abd. films for 1° abd. lesion





- Cough skeleton & facial bones - Nasal discharge Ultrasound of abdomen & thorax for 1° lesion 305





Anthelmintic	040				
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Anticoagulants	141	Ethylene glycol	317	Phenothiazine	
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ANTU	315	Hydrocarbons	312	Poisonings	
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		Metaldehyde	314	Toxic gases	
		Metals	313	Treatment of poisonings	
		* * * * * * * * * * * * * * * * * * * *		,	

Molluskicide

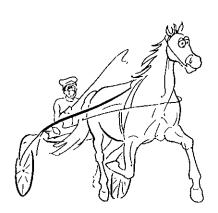
OPs (Organophosphates)

PCP (Pentachlorophenol)

Organochlorine insecticides

Monensin

NSAIDs



TOXICOLOGY

Wood preservatives

Zinc phosphide

Urea toxicity

Vacor

Zn₃P₂

Warfarin

Poisonous Plants

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Red maple leaf	139, 32		j gadonus	329

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2-PAM (protapam chloride, pralidoxime)

5% Dioctyl sodium sulfosuccinate (1/2 L/450 kg)

5% sodium bicarbonate

20% Magnesium sulfate sol.

Oral medications
• Activated charcoal

Mineral oil
 Miscellaneous
 Oxygen

MOLLUSKICIDE

Metaldehyde M8K 2074, 2145; Mk 1676; IM 1637; EM&S 777; Tox 325

Molluskicide: slug & snail bait, Snarol®: looks & tastes like dog food, palatable, reported in dog, cat, sheep & children

· CS: Acute win 3 hrs, vomiting, colic, diarrhea, dyspnea, tachycardia, frothing, CNS (tremors, hyperesthesia, spasms, convulsions), death: resp. failure

• Dx: Lab: stomach contents for acetaldehyde (break down product of metaldehyde), PM: formaldehyde odor

• Tx: No specific antidote, symptomatic, sedate: diazepam, emetic or gastric lavage, fluids (Na lactate)

CNS/GI - No antidote

WOOD PRESERVATIVE, FUNGICIDE

PCP, penta.

Pentachlorophenol M8K 2097; Mk 1696; IM 1631; EM&S 1708; C4T 663; C3T 362;

• Pressure wood treatment (soil fungi & insects), Horses like to lick "leaky" wood, Not street drug called PCP (angel dust). ↑ O2 demand by blocking oxidative phosphorylation & ATP formation, Irritating to skin & respiratory tract

 CS: Acute: muscular weakness, anorexia, lethargy, fever, gasping, dehydration, sweating, salivation, lethal: cardiac & muscular collapse & death . Chronic: anemia, weight loss

Dx: Treated wood in environment, 🛊 BUN, proteinuria, Blood: 40-80 ppm PCP, Rapid rigor mortis

Lick "leaky" wood" • Tx: No specific therapy, Fluids to flush kidney • Control: prevent use of oily, freshly treated wood where contact can occur TOXIC GASES

Hydrogen sulfide, H₂S

IM 1643; C3T 315; Tox 372

Colorless, heavier than air, toxic gas, "Rotten egg" smell, Liquid manure holding pits underneath animal housing, H2S retained in liquid, when liquid agitated prior to pumping out released (m/b 1000 ppm), > 500 ppm imminent threat to life, fritiant to eyes & respiratory system CS: Pulmonary edema, hyperpnea († respiration initially followed by apnea); asphyxia follows if not immediate artificial respiration

Dx: Smell in air at low levels, Hi levels can't smell (olfactory paralysis)

• Tx: Fresh air, artificial respiration • Prevention: Remove animals & humans before agitating manure pit, Ventilate area • Public Health: Asphyxiation to humans, Don't enter manure pit

'Rotten egg" - Manure pit - Respiratory • Tx: Fresh air

Ammonium C3T 315 • Ammonia most common noxious gas found in stables

Smoq, **SO**₂,

Smog, Sulfur oxides + H2SO4 smog major factor in air pollutants; deaths of man & animals • CS: Eye irritation & salivation, emphysema, respiratory distress; cattle & horses in urban environments

 Tx: None, supportive Sulfur oxide Tox 376

Eyes - Respiratory

Smoke

inhalants

IM 602, 1643; C4T 460; EM&S

Barn fires, Alveolar damage, interstitial edema, hypoxia & 2º bronchopneumonia

CS: Oral burns, Conjunctivitis, Laryngospasms, Respiratory problems (cough, stridor, tachypnea)

· Dx: Hx, CS, Bronchoscopy, Transtracheal wash

• Tx: Patent airway (intubation, tracheostomy), O2 therapy (up to 100%) (Careful bec. O2 m/ cause lung damage) IV fluids (monitor for pulmonary edema), ABs for 2° pneumonia, Bronchodilators (aminophylline) (relieve soot-induced bronchospasms)

Respiratory - O2

RODENTICIDES

M8k 2143; Mk 1721; IM 1635, E

 Rare: dogs, cats & swine. Alpha-naphthyl thiourea, Use declining because of more effective rodenticides. † permeability of pulmonary capillaries, Strong emetic (protects some animals that vomit), rodents unable to vomit

CS: Pulmonary edema "drowns in own fluid", Death w/o convulsions
 Tx: No specific Tx, emetics early before edema

Rare - Respiratory



M8k 2156; Mk 1732; IM 1635;

· See Neuro pg 272; Used for burrowing rodents & coyote, No rationale for its use! Stimulates CNS, loss of "damping" of spinal reflexes CS: Uncontrolled reflex activity, extensor rigidity, easily induced convulsions, tonic seizures

. Dx: CS, Strychnine tissue & urine levels

Tx: Control seizures, muscular relaxation, Oxygenation, Quiet environment, charcoat, Diuresis

No rational fore use - CNS/Seizures

Sodium fluoroacetate. 1080

M8k 2146; Mk 1723; IM 1635; C2T 661; Tox 340

CS: Cardiac CS, pain, anxious, hyperexcitability, profuse sweating, trembling, ataxia, terminal convulsions, sudden death m/b only CS

Dx: Hyperglycemia, Rapid course, Rapid rigor mortis (extensor rigidity), Detection of 1080 in tissue difficult, Elev. kidney citrate levels suggestive

• Tx: No specific antidote, calcium gluconate or calcium chloride il hypocalcemia, glycerol monacetate (Monacetin®) IM before CS

Px: Grave once CS

To kill coyotes - Rapid death - No antidote • Px: Grave

Zinc phosphide, * ZN3P2

Tx: NO specific Tx, Gastric lavage w/ 5% Na bicarbonate (raise pH to delay formation of gas), Symptomatic

• Kilrat®, Goph-rid®, Release of phosphine gas on contact w/ water (more rapid at low pH), Odor of rotten fish, Emetic properties: rodents can't "bart" • CS: Rapid, Die w/in 2 d, Anorexia, lethargy, Rapid deep breathing, Colic, Alaxia, weakness, prostration, convulsions, Garlic-breath
• Dx: Exposure, Rapid death, Dyspnea, PM - Garlic smell to stomach, Histo: kidney & liver damage

Mk 1724; IM 1636; C2T 662; Tox

Phosphine gas in stomach · Garlic odor, Corrosive & hepatotoxic, Protoplasmic poison, rare, infrequently used as a rodenticide today, dogs may get phosphorus in fireworks Phosphorus

CS: Biphasic, GI irritation (Colic, Hemorrhagic diarrhea) M/b coma & death before 2nd phase, Latent phase - apparent recover, 2nd phase, 2-4 of after start of CS, Depression, Hepatic & renal failure - death, Colic m/ recur, oliguria, icterus & bleeding tendencies.

Dx: Biphasic CS, elev. liver enzymes, † BUN, PM: enlarged liver, icterus, G1 irritation, hemorrhage & necrosis, Histo: liver & renal failure.

Tx: Emergency, Symptomatic Tx, Mineral oil, activated charcoal, No fat in diet for 3-4 d (absorbs phosphorus)

Px: Grave

Garlic - Biphasic

Pyrimilin, "Vacor" IM 1637; E 196; C2T 662; Tox 357

M8k 2145; Mk 1722; IM 1636, C2T 663; Tox 358, 49, 54

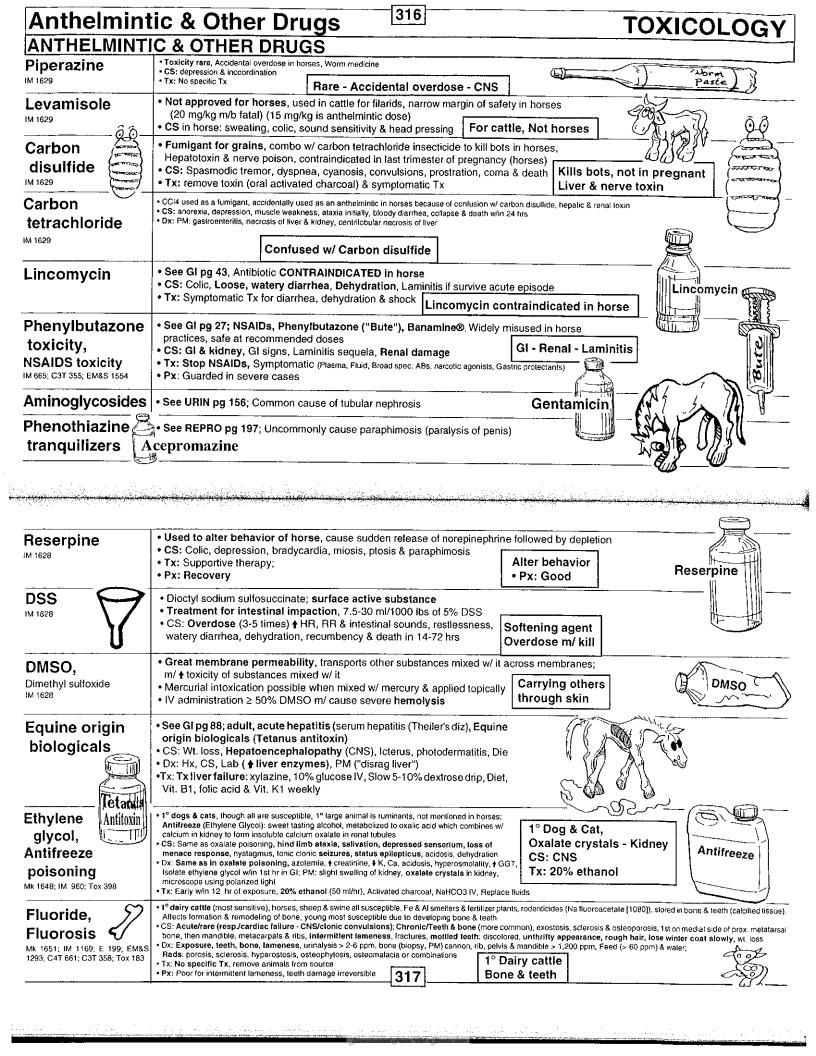
> Not currently available, but m/ be on premises, Mech.: Vit. B antagonism, destroys pancreatic cells & depresses glucose uptake by cells CS: Apprehension, Mental confusion, Visual disturbances, Colic. † peristalsis, Dilated pupils, Weak pupillary response, † HR, Dehydration, Muscle fasciculations & profuse sweating, Hindlimb weakness, Ataxia
>
> Tx: Many recover spontaneously if protect from self trauma & give supportive care, Nicotinamide reported as specific antidote (disappointing

In human poisonings)









Poisonous plants E 205; PP/Mt 3

Facts:

- · Rule of thumb: poisonous plants usually not palatable
- Usually eat only when nothing else (drought, early Spring)
- Or when mixed w/ hay or grain
- . Fortunately horses need to eat 1-3% of body weight to be poisoned

Economics:

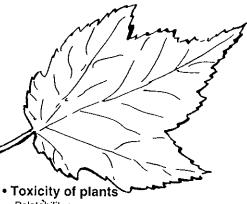
- Western USA 3-5% annual deaths for cattle, horses & sheep
- Poor weight gain & poor reproduction

Variables:

- Plants vary in toxicity
- Animals affected (some OK for cattle & horses, but kill sheep)
- Conditions of poisonings:
- . Some nutritious, except during certain seasons when they are poisonous
- . Some nutritious unless only thing eaten (choke cherry for cattle)

Poisonous substances:

- Poisonous substances themselves to livestock (Alkaloids & oxalates)
- Harmless substances transformed by decomposition or ingestion (choke cherry nontoxic amygdalin changed to toxic prussic acid)
- Substances absorbed from soil into plant (ex. milkvetch accumulates selenium)
- Substances making animal hypersensitive (e.g., St. John's wort causes photosensitization)
- Miscellaneous substances (toxic metals: fluorine, arsenic)



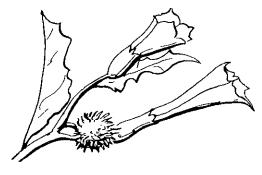
- Palatability (influences how much eaten, herbicide or fertilizers m/ make more palatable)
- Available nonpoisonous forage; will eat instead of nonpalatable toxic plants usually
- Stage of development: usually more toxic when immature, some when mature
- Drought or freezing (stress) commonly † toxicity
- Soil (e.g., some toxic on selenium soil, but good on other soils)
- Moisture contents: drying m/ ↑, ↓ or leave toxicity the same
- Parts of plants: some more toxic than others (seeds, leaves, stems or root)
- Toxic substances: some lethal in small amounts. others require large amounts
- . Some accumulate in animal while others don't

Animal factors

- Some toxic to certain species, others toxic to all
- Young usually more susceptible than adults
- White animals: photosensitization
- Variability of susceptibility of individual animals
- Stressed animals more susceptible (temperature, exertion)
- Fasted animals more susceptible
- Horses are more selective grazers than cattle

Management factors

- Overgrazing most common cause of poisonings, have to eat poisonous plants
- Turning hungry animals into new pasture, eat first thing they see
- Early turn out onto range, toxic plants often 1st to "green up" (low larkspur, lupine, death camas)
- Crowding animals, let spread out
- Inadequate nutrients (salt defc eat greasewood or arrowgrass)
- Feeding contaminated hay (Blister beetle)
- Grazing during dangerous seasons
- toxic plants
- Grazing snow covered fields, taller toxic plants m/b only available feed (tall larkspur or lupine)
- Grazing after heavy rainstorm, some more toxic after rain, some more palatable, easier to pull up toxic
- Grazing during drought, some toxic plants remain green, while good forage is gone



Clinical signs m/b confusing

- Similar signs for different plants
- · Incomplete or nontypical CS of poisonings by usually nontoxic plants

Diagnosis:

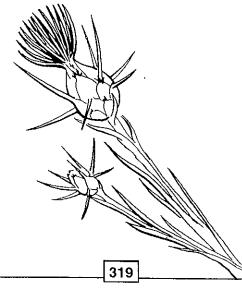
- Hx: of injestion (requires more than just identification of plant, need proof of ingestion also)
 • Clinical signs: confusing
- · Suspected plant poisonings:
- Sudden onset of illness w/ no apparent cause (after turned out into new pasture)

• ID plant

- Check pasture, especially fence rows, ditches & springs for poisonous plants
- Check GI for plant material in those that die
- Collect plants & send to local county agent for ID . Press & dry between 2 pieces of paper
- Send between 2 pieces of stiff cardboard
- Detailed record of events
- If alive: observe & record CS

Treatment

- Tx less satisfactory then for chemical poisonings
- Therefore prevention is key to control
- · Usually symptomatic because few antidotes
- Absorbents: activated charcoal
- Astringents (bismuth subnitrate) constrict intestinal capillaries (prevent further absorption)
- Stimulate elimination:
- Stomach pump Emetics Purgatives Diuretics
- Treat symptoms
- Cramps & convulsions: sedative & depressants
- Depression & paralysis: stimulants





Prevention key to controlling

• DON'T OVERGRAZE

- Remove plants
- Remove animal from pasture during poisonous season for certain plants (e.g., late summer & fall for yellow star thistle, because horses like to eat it)
- Put on range when sufficient growth of desirable
- Spread out herd & move slowly, so they can be selective
- · Supplement forage or extra hay during poisonous seasons (prefer over nonpalatable poisonous plants)
- · Mineral supplements, mineral deficiencies m/ stimulate to eat poisonous plants (e.g., hi nitrate have salty
- Check hay for too many weeds (e.g., oleander)
- No hungry animals into new pastures, will eat anything
- · Don't throw lawn clippings where animals can eat (e.g., oleander)
- Salt & phosphorus blocks (supplement & spread livestock out)
- Adequate water supply
- Keep off sprayed or fertilized land for 2 weeks
- · Feed supplements when snow covers forage except tall toxic plants
- Supplement during drought or reduce number of animals
- Fence uncontrollable areas

Cyanide, HCN.

Hydrocyanic acid, Prussic acid M8k 2154; Mk 1647; IM 1649; E 214; C2T 678; Tox 455

· Cyanogenic glycosides - hydrolyzed to release hydrocyanic acid (prussic acid, HCN), Cattle > sheep/goats > equine > swine Sources: Rodenticides, Plants 1° (see box), damage (willed, trampled, drought) cause glycoside to change to HCN, Not cumulative (must eat a lot in short time, 1 hr), cellular hypoxia, hemoglobin (Hb) can't release oxygen (bright red blood)

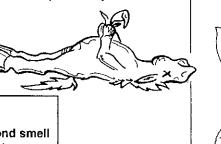
 CS: Found dead (rapidly acting); excitement & muscle tremors; dyspnea; salivation; lacrimation; voiding of feces; goes down, gasps for breath, m/b clonic convulsions due to anoxia; dilated pupils; mucous membranes bright red; If survives longer than 2 hours usually recover

 Dx: Exposure - resp. distress, cherry red blood, red mucous membranes, odor of bitter almond to GI; GI & lung hemorrhage

Tx: Na nitrate IV (Breaks HCN bonds & is excreted), Na thiosulfate IV (HCN to thiocyanate

Px: Poor, so rapid, usually too late to Tx

& excretion), Rare toxicity because it can be treated specifically & effectively



Cyanide plants

- Apple
- Arrow grass
- Birdsfoot trefoil
- Cherries, choke cherries, apricot, peach
- Corn, maize
- Elderberry
- Flay
- Hydrangea
- Lima bean
- · Poison suckleya
- Sudan grass, Johnson grass Sorghum grass hybrids
- Velvet grass
- Vetch seed
- White clover

Suckleya suckleyana Sorghum spp.

Hydrangea spp. Phaseolus lunatus

Pyrus malus

Lolus corniculatus

Prunus spp.

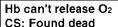
Linum spp.

Zea mays Sambucus canadensis

Triglochin maritima

Hoecus lunatus Vicia sativa Trifolium repens

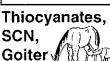
Cherry Red Blood



Dx: Cherry red blood, Almond smell

Tx: Na nitrate - Na thiosulfate

Px: Poor



 SEE ENDO pg 301; Goitrogenic plants or excess iodine (kelp) eaten by pregnant mare; most common thyroid disorder in neonates, inhibit thyroid hormone production

CS: Goiter, hypothyroidism (incoordination, poor suckle response, poor righting reflex, hypothermia, tendon contracture, retarded bone development, born normal - skeletal lesion in wks

Tx: Thyroid hormone supplementation (iodine): No way to reverse lesions

Dx: Thyroid stimulation hormone response test



- Prunus virginiana, Cattle & sheep, all susceptible
- Description:
- Large shrub or tree
- Leaves: dark green, 2-4" long oval w/ pointed tip, saw-toothed margins
- Flowers: small, yellow-white, dense clusters
- Fruit: dark red to black (3/8*)

Plants w/ goitrogenic compounds

Mustard family (Cruciferae)

Brassica spp

· Lineum usitatissimum

Rape seed, Mustard, Kale, Broccoli, Cabbage & Turni Soybean



Selenium toxicity, Alkali diz. Bob-tailed diz, Blind staggers

M8k 2150; Mk 1727; IM 1653; E 192; EM&S 1681; C4T 667; C3T 125; C2T 670; Tox 132; PP/US/C

Dx: Exposure, Levels Tx: Arsenic for cattle

- Cattle, sheep & horses, arid & semiarid areas, "accumulator" plants require Se & m/ contain > 1000 ppm, Fortunately only eaten if starving, "converter" plants don't require Se, but absorb it (commonly grazed & cause poisoning), western 1/3 of USA. Se supplementation may result in toxicosis
- CS; Acute (rare, because avoid plants) dyspnea, frothy nasal discharge, recumbency, diarrhea, polyuria, death
 - Chronic "alkali diz", weight loss, depression, lameness, abnormal hoof conformation
 - Coronary band inflam. & break, grows down & sloughs
- "Bob-tailed" diz, hair loss from mane & tail, reproductive compromise (soft testicles)
- Chronic "blind staggers", aimless wandering or circling, incoordination, forelimb weakness, dyspnea, blindness
- Dx: Exposure & CS, Se blood levels 1-2 ppm "alkali" diz; 1.5-4 ppm in "blind staggers"
- Tx: Chronic: Ruminants: arsenic application to feed reduces sorption; arsenic orally dangerous in equine because they live so long; Acute: no treatment

Se Plants

Obligate Se-indicator plants (often >1000 ppm) **Oonopsis**

- Goldenweed
- · Milk vetch
 - Astragalus bisulcatus
- Poison vetch
- Astragalus pectinatus Prince's plume

- Woody aster

Stanleya pinnata

Xylorrhiza

Facultative Se-indicator plants

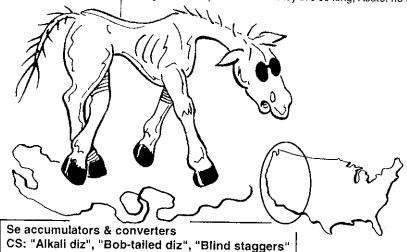
- Broom snakeweed Gutierrezia sarothrae
- Groundsel varieties

Senecio spp.

Gumweed

Aster

Grindelia



Astragalus spp., Milkvetch, Poison vetch

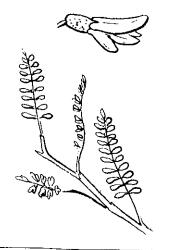
M8k 2128; Mk 1727; IM 1653; Tox 132; PP/Mt 36

- Astragalus spp. (over 300 species), largest
- genus of legume family
- Catile, sheep, horse, all species Difficult to ID species of astragalus
- Oxytropis, point locoweed, pointvetch so closely resemble astragalus that some botanist treat as
- Unpalatable, garlic odor



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- Low growing perennial
- Leaves pinnately compound
- Flowers: white to purple, rounded keel petal (lowermost petal)



Thiamine defc, Bracken fern toxicosis, Bracken staggers

M8k 2140, 2136; Mk 1641; IM 1648; E 208; EM&S 805; C3T 676; PP/US/C

• See NEURO pg 264, Bracken fern & Horsetail Western USA; Thiaminase leading to thiamine defc (B1) in monogastric animals, Not in ruminants (bone marrow depletion - aplastic anemia) Accumulates over 1-3 months, Palatable

 CS: Bracken staggers, anorexia, weight loss, incoordination, crouched stance, arched neck & feet wide apart, convulsion, clonic spasms & opisthotonus, death

• Tx: B1, thiamine (IV qid, then IM for several days)

· Prevention: Remove bracken fern

Thiaminase producing plants Bracken fern, w. bracken Pteridium apuilinum

 Crotalaria Horsetail Equisetum arvense

Perennial ryegrass

Pteris aquilina

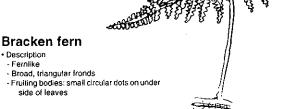
 Ragwort Senecio jacobea



Thiaminase CS: Staggers Dx: Exposure, Levels

Tx: B1







Horsetail, Scouring rush, Jointfir

Horses most common, all susceptible

Long rush-like, green stems, jointed, hollow

Cone-like fruiting bodies on top of

Photosensitizing plants, **Pyrrolizidine** alkaloid

toxicity. Trefoil dermatitis, Rape scald

M8k 2132, 2138, 2114; Mk 820, 1698; IM 850; 1652; E 205; EM&S 1555, 1652; C2T 672; C4T 76, 650; C3T 680, PP/US/C 271, 171; PP/MI 44 . See GI pg 89; Not common sunburn, exposure to sunlight plus chemical deposited in dermal tissue

Western USA

1° photosensitizing plants - photodynamic substances

- CS: Sun burn of light colored skin (ears, nose, lips, vulva, udder, coronary band), erythema, edema, blister & serum exudate

- Tx: No specific Tx, Avoid sunlight & plants

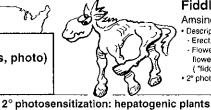
2° Photosensitizing plants: Pyrrolizidine alkaloid (more common)

Cause hepatic damage, cumulative & progressive, preventing clearance of normal chlorophyll breakdown products which are photodynamic & deposited

- CS: Weight loss, liver failure: hepatoencephalopathy (ataxia, wandering), Icterus, photosensitization (white areas)

- Tx: Euthanasia, remove plant source, Tx for liver failure

- Px: Poor to grave, due to fibrosis of liver



Fiddleneck,

Amsinckia

Description

Erect, 1-3', Hairy Flower, coiled racemes.

flowers on one side "fiddleneck")

Narthecium ossifragum

Heliotrophium europeaum

Holocalys glaziovii

Panicum coloratum

Lippia rehmanni

Panicum spp.

Amsinckia intermedia

Senecio redellii, S. longilobus

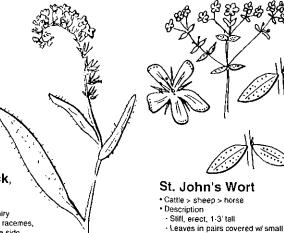
Tetradymia canescens

B. napus, B. hyssopifolia, B. brizantha

2º nhotosensitization

Lantana camara

Nolina texana



clear to black dots

Flowers: numerous, bright yellow

• 1° photosensitization

 Puncture vine, caltrop Tribulus terrestris

Senecio

Crotalaria Tansy ragwort Senecia jacobea

 Viper's bugloss Echium plantagineum

Medicago

Trifolium son

Unclassified photosensitizing plants

· Alfalfa

Clover species

· Fireweed, summer cyprus

 Milk purslane • Oats

Ragwort

Rattlebox

Smartweed

Avena

Kochia scoparia Euphorbia maculata

Polygonium

Tx: Euthanasia, Px: poor 1° Photosensitizing plants

2° > 1°, Pyrrolizidine alkaloid

Bishop's weed

Dx: Exposure

Buckwheat Castor bean

Clover

Oat orass

Perennial rye grass

• Bane

Rutaceae

Spring parsley

Klamathweed Trefoil

Ammi majus

Fagopyrum esculemtum Ricinus communis Trifolium Cooperia pedunculata

CS: Wt. loss, Liver Failure (CNS, icterus, photo)

Avena fatua

Brassica

St. John's Wort,

Cymopterus watsoni

Umbelliferae

Hypericum perforatum Erodium

• Horsebrush (gray) Kleingrass

Fiddleneck

Groundsels

· Blue green algae

Bunch grass, texas sacahuiste
Common heliotrope

· Bog asaphodel

Broomcorn millet

Brassica

Mycotic agents · Panic grass, witch grass

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Pithomyces chartarum & P. minutissima

Velches

Sudan grass

Sorghum var. sudanense

Vicia spp.

TOXICOLOGY

poisoning,

M8k2142, 2110; Mk1720; IM 814; C2T 673; Tox 468

Quercus spp., SW USA associated w/ browsing of buds, new leaves in spring, midwest & NE; associated w/ eating acorns in fall, Windstorms drop acorns, Hi rainfall softens acorns (more palatable), Rarely in horses, #1 Cattle (less selective eating

· Common in fresh water, N. plain states, summer when bloom (green soum) of algae, algae dies, toxin in water - foul fish smell, Wind

- CS: Peracute or acute, sudden death, colic, tenesmus, hemorrhagic diarrhea, not urinary problem seen in cattle
- Dx: Consuming oak buds or acorns, acorn husks or shells in feces, Urinary phenolic content to DDx from other colics Tx: as all colics, fluids IV, analgesics, oral laxatives, diuresis, acid base balance & serum calcium levels

blows to shore, Animals drink water - die, ruminants more sensitive than monogastrics

Description: Trees to shrubs, Acorns, 60 spp. in USA & Canada

Rare in horse, GI; not urinary

Toxic bluegreen algae,

Algae poisoning; Algal poisoning,

- Tx: No specific antidote, often animal dead or dying before Tx, supportive care, activated charcoal & oil
- necrosis) & kidney
- Prevention: Organic herbicides or copper sulfate (bluestone) Tx of water, follow label direction to avoid toxicity, fence off water when bloom of algae on water

CS: Acute death w/in minutes, rapid onset (15-45 min), rapidly progresses to prostration & death; nausea, colic, bloody diarrhea, prostration,

muscle tremors, dyspnea, cyanosis, ± CNS (general paralysis, seizures, prostration & death), chronic (depression, anorexia, hemorrhagic

Dx: Very difficult, exposure, no practical way to isolate & identify toxins, PM: gastroenteritis, degenerative changes of liver (centrolobular



Ruminants> Horse, CS: Acute death, Dx: difficult; Tx: prevention; Px: poor die in 24 hrs

Trauma plants,

Mechanical traumaproducing plants IM 1659; PP/US/C 487

- Bristle grass, Yellow bristle grass, Foxtail, Cheatgrass, Needle grass, Povert grass, Crimson clover, Cocklebur (Xanthium strumarium)
- Common in hay, mechanical injury

gastroenteritis, photosensitization, hepatic diz

- CS: Mouth uicers, trauma to skin around mouth & eyes (horse), lodge alongside mouth (salivation)
- Tx: Removal of source, remove from animal, Antibiotics heal rapidly
- · Description: Short, sharp, straight awns

American black walnut shavings

M8k 2124; IM 1656; C3T 374

- · Toxic principle: Jugulone (anaphthoquinone), Horses bedded on black walnut shavings
- CS: Signs in 12-24 hr, laminitis, weight on rear limbs, limb edema, blackish/brown staining to mouth & teeth if eat walnut hulls
- Tx: Remove bedding, treat laminitis
- · Prevention: Don't use black walnut bedding

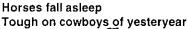
Bad horse bedding



Sleepy grass

IM 1658; C2T 681; PP/US/C 494

- · Description: Grass: erect clump 2-4' tail
- Stipa spp.; Sierra Blanca Mountains of N. Mex. · CS: Profound stuporous sleep
- (comatose), lasts only a few days
- Tx: Avoid further ingestion complete recovery





Grass sickness, Equine dysautomia

C4T 203;

- Unknown cause
- High mortality rate
- No histologically confirmed cases in N. Amer.

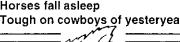


Milkvetch, Vetch toxicosis, Timber milkvetch

- Astragalus miser var. (wasatch, Columbia & vellowtone milkvetch)
- 1° Cattle, all species

Hemolytic anemia Heinz bodies

- Toxic principle: Glucosides
- · CS: CNS & respiratory
- Tx: Symptomatic & supportive



Red maple leaf toxicity,

Heinz body hemolytic anemia

- See CARDIO pg. 139, Acer rubrum, Oxidizing agents causing acute hemolytic anemia & Methemoglobin, also Phenothiazine, onions & Brassica plants
- CS: Weakness, exercise intolerance, brownish mucosa, No fever
- Dx: CS, anemia, Heinz bodies (crystal violet)
- Tx: Remove source, supportive
- · Px: Good if modest anemia, unfavorable if renal damage

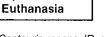


Nigropallidal encephalomalacia Yellow Star Thistle poisoning, "Chewing diz"

See NEURO pg 267, Centauria solstitialis, Centauria repens (Russian knapwéed)

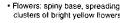
- Western US, Summertime, Long term ingestion addiction
- CS: "Chewing diz", Lips don't work, Die of starvation/dehydration
- Dx: PM: necrosis of substantia nigra & globus pallidus pathognomonic
- Tx: Euthanasia recommended
- Px: No recovery

Lips don't work Euthanasia





- Leaves: covered w/ cotton hair

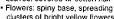














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TOXICOLOGY

Jimsonweed. Datura. Stramonium, **Thornapple**



- Description
- Leaves: long petioled (stalk attaching leaves), alternate Flowers: solitary, showy, large, tubular, born at fork of
- stem & branches Fruit: spiny capsule
- · Toxin: Datura stramonium; Atropine-like alkaloid (scopolamine), distasteful
- · Horse > cattle, sheep & goats
- CS: Depression & parasympatholytic nervous system, restlessness, irritability, weakness, mydriasis, photophobia, constipation, thirst, incoordination & paresis, respiratory failure, delirium, convulsions
- Dx: Drop of urine in lab animal's eye = mydriasis
- Tx: Symptomatic, parasympathomimetic (physostigmine), activated charcoal, laxatives; Convulsions: diazepam (not phenothiazine), anticholinergics

Atropine-like CS: P-ANS Vallum



Description

- Shrub or tree 40' tall
- Rough deeply furrowed bark
 Paired spines at base of each leaf
- Leaves odd pinnate 12" long
- Flowers: white, drooping racemes

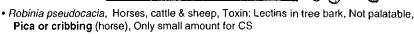
Black locust

IM 1657; C3T 678; PP/A 43

CS: Colic, diarrhea

Pica - Bark

Tx: Symptomatic, fatalities rare



- · CS: Colic, diarrhea, weakness, posterior paralysis, mental depression, fatal cases rare
- Dx: Exposure, Irregular HR
- Tx: Symptomatic & supportive, digitalis in severely affected, Avoid exercise (heart)

Blister beetle &

• See GI pg 45; Horses > cattle, swarm in alfalfa during harvest, Cantharidin: potent irritant & vesicant, SW • CS: Endotoxic shock & renal failure, Found dead peracute, GI irritation • Dx: CS, beetle in alfalfa, cantharidin in urine or stomach, BUN & creatinine

• Tx: No antidote, Supportive: IV fluids: protectants, mineral oil, charcoal, diuretics, analgesics

· Prevention: Hay not from Southwest, ID feed source

Px: Guarded if obvious CS & a lot of blood in feces & urine

Alfalfa





Dogbane, Indian hemp

M8k 2116; IM 1656; PP/US/C 263

 Apocynum cannabinium (Indian hemp. dogbane), A. androsaemifolium (Spreading dogbane)

- Cattle, horse & sheep
- CS: Urinary system
- . Dx: Urinalysis, crystals in urine
- Tx: Dilute toxins (diuretic)
- Description
- Erect herb
- Woody stem milky sap
- · Leaves: opposite pairs
- Pods hanging in pairs

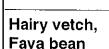
CS: Urinary system Dx: Crystals in urine

Tx: Diuretic

Bitter rubberweed, Bitterweed, Hymenoxys

M8k 2108; IM 1655

- Hymenoxys odorata
- · Sheep in SW USA, also cattle & horses



IM 1659, EM&S 1680; PP/US/C 362

- Vicia billosa, Horse & cattle
- Toxic principle: unknown
 Horse infrequently reported
 CS: Pyrexia, Progressive SQ edema of lip,
- spreads to rest of body, Marked wt. loss, Corneal ulcers; SQ papules & plaques, pruritus & alopecia
- Tx: No Tx, Frequently refractory to Tx

Castor bean (ricin) toxicity

M8k 2138; IM 1653; E 207; C2T 673; PP/US/C 194

Seed in feed

CS: GI irritation, convulsions, death

Dx: Exposure, Levels

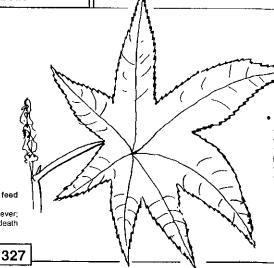
Tx: Sedate & Tx shock



· Ricinus communis, Toxin: Ricin · seed-borne protein phytotoxin, plant not usually ingested, but seeds in feed as an oil by-product (2 soldiers chewed the seeds as a laxative and died)

CS: Gastric irritation - colic; profuse, watery diarrhea; anaphylactic shock if enough eaten; Hi fever; depression; incoordination; profuse sweating; muscle twilching; pounding heart beat; convulsion before death
 Dx: Hx of ingestion, seed in feed material, RBC agglutination or precipitation test for ricin

 Tx: Sedation & Tx shock initially, keep warm, antihistamines, IV fluid, prevent further absorption (Oral laxatives, intestinal protectants & activated charcoal)





- Shrub 12' tall
- Stem: reddish ourolish
- Leaves: large, alternate, deeply
- paimated, 6-11 lobes
- Fruit: spiny capsule
- Seed: large brown female tick-like



Poisonous Plants

CHEST Locoweed poisoning

"Locoweed diz", Texas loco

- See NEURO pg 265; West, Astragalus spp. & Oxytropis; Toxin: unknown, unpalatable, starvation, Addictive m/b, Horses > cattle > sheep
- CS: Stagger, loco, Wander in circles, convulsions & falling, death

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- Tx: None
- Px: Bad remains effected long, extremely unpredictable

Addictive - Maniacal



• See URIN pg 156; Usually valuable forage, Myelomalacia of lower spinal cord

 CS: CNS: Posterior incoordination, "dribbling", cystitis . Dx: Hx & CS, no specific tests, urinalysis for cystitis

• Tx: Withdraw Sorghum, No specific Tx, ABs for urinary tract infections

• Px: Recovery rare • Control: diversify diet (Sorghum not complete diet)

Description - Greasewood

Erect, woody, spiny shrub, 3-5' eaves: fleshy, 1/4-1 1/2" long

Flowers: sexes separate, small flowers

1° in cattle - rare in horses - West Crystals - Renal • Px: Grave

Oxalate

M8k 2110, 2122; Mk 1709, IM 1651; Tox 471; PP/US/C 235, 240

- · Halogeton, Greasewood, 1º in cattle, Rare in horse, West USA
- Insoluble pcpt. w/ Ca, Vascular necrosis & renal tubular blockage,
 CS: Colic, Weakness, Frequent urination, Crystals in urine
 Tx: Hopeless once CS; Fluids, Ca gluconate



Halogeton - description

- · Leaves: fleshy
- · Flowers: "microscopic"

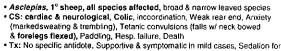


M8k 2126; IM 1656; C3T 374; PP/Mt 36; PP/US/C 267



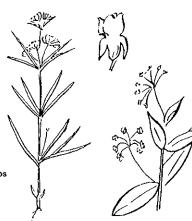
Description

- Perennial w/ milk juice, erect
- Flowers: umbrellas
- Narrow & broad leaved groups



convulsions, Antiarrhythmic Rx: alropine &/or diphenylhydantoin

Prevention: Keep out of hay



TOXICOLOGY

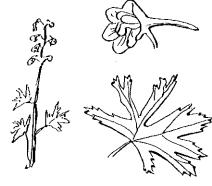
Larkspur toxicosis, Delphinium

M8k 2112; IM 1647, PP/Mt. 14, 15; PP/US/C 25

- . Toxic principle Delphinine alkaloids (similar to substance isolated from Aconitum)
- · Horses: don't eat enough to show CS, sheep resistant
- · #1 poisonous plant of cattle in Western USA

Description

- · Tall & low larkspurs
- Tall at high allitude 3-6';
- · Low at low altitude < 3'
- Perennial
- · Leaves deeply divided to palmately lobed
- Flower: distinctive spur



Solanum toxicosis, Nightshade, Deadly nightshade

M8k 2120; IM 1646, CT 376; C2T 677; PP/O 13; PP/US/C 290

- Minor importance of intoxication of horses
- CS: CNS & GI
- Description:



Description:

Shrub or bush up to 20" Leaves: leathery, lanceolate Flowers: showy clusters





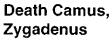


Oleander toxicosis

M8k 2136; IM 1649; E 208; C2T 674; PP/US/C 264

- Nerium oleander Ornamental hedge W & S USA
- Toxin Cardiac glycoside similar to digoxin
 Bilter taste (infrequent ingested, clippings mixed in grass)
- CS: Cardiac, "thumps" (+ HR), diarrhea, sweating,

muscle twitching, death



M8k 2110; IM 1646; PP/Mt 28; C2T 677; PP/O 14

Description:

- Perennial, single stem, bulb
- · Flower: terminal racemes
- Sheep, all classes susceptible, W USA, very early spring; Distasteful, onion-like bulb
- CS: Cardiac & respiratory: salivation, dyspnea, weakness & staggering, coma, death
- DDx: Oniori (has onion odor).
- Tx: No specific Tx, Symptomatic & supportive



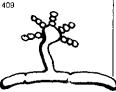


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TOXICOLOGY

Mycotoxicosis

M8k 2076, 232, 2078; Mk 1678; C4T 83, 668; EM&S 1708; Tox



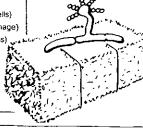
• Mycotoxin: 2º toxic metabolite produced by mold, Not from growth of mold in animal

- Characteristics:
- Cause of problems not readily identifiable
- Tx by drugs or ABs not effective
- Not contagious or infectious
- Often seasonal outbreaks (climate dependent)
- Conditions needed
- Substrate carbohydrates of feedstuff Moisture of grain (10-18%) or 70% humidity
- SE USA warm humid conditions
 - · SW USA irrigated cotton production
 - Grains shipped long distances
 - · Rarely responsible for acute diz or large death loss

- Vague chronic conditions (poor performance, ill thrift or
- ◆ susceptibility to infect. dizs)
- Reduced growth
- Hepatic fatty infiltration: aflatoxins
- Vague chronic conditions
- Confirmation ID toxin
- Feed analysis 10 lbs (4.5 kg), many individual samples mixed together
- . Dry samples preferred, shipped in paper or cloth, not plastic
- No known Tx
- Remove source of toxin (moldy feedstuff)



- Hepatotoxins (degeneration, fatty changes, hemorrhage, necrosis of hepatic cells)
- Nephrotoxins (exalic acids by Aspergillus & Penicillium spp. renal tubular damage)
- Hematopoietic & coagulation changes (hemorrhage, anemia, weakness)
- Direct irritation (oral ulcers, gastroenteritis)
- Reproductive &/or endocrine disturbances
- Feed refusal m/b only CS
- CNS (tremogens from ergot & Penicillium spp.)
- Immune system (* susceptibility to infectious dizs)



Aflatoxicosis

Dx: difficult

M8k 2077; C4T 669; C3T 369;

CS: Vague, chronic

Tx: ABs not effective

- Poultry > dogs > swine > horse > calves > cattle > sheep, Aspergilius flavus, Allatoxin; Hepatic necrosis & fatty changes, Immunosuppressive
- CS: Anorexia, fever, tachycardia, ataxia, colic, icterus, bloody feces, abdominal straining, reduced weight gain
- Dx: Hepatoloxic diz depression, icterus, hemorrhages; anemia, \$ BSP secretion, icterus, \$ serum transferase, ALP, allatoxin in feed, UV light: greenish-yellow "firefly"-like glow
- Tx: Methionine or methionine + cysteine & Na thiosulfate, 🛊 protein in diet, Vit. supplementation (A, D, E, K, 8 complex), aggressively Tx infections (since immune system is compromised)

 Ergotism, **Ergot**

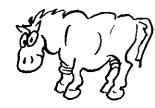
M8k 2083, 2078; Mk 1684; C4T 83; Tox 428

- Parasitic fungi, Claviceps purpura (rye, oats, wheat & Kentucky blue grass), Claviceps paspali (Paspalum or Dallis grass); swine, sheep, horses & poultry; warm moist conditions; Toxicity: alkaloids: ergotamine, ergotoxine & ergometrine; † muscle motor activity of uterus, arterial & venous constriction, vascular stasis, thrombosis, gangrene
- · CS: Gangrenous: gangrene of extremities (limbs, nose, ears, tail), depression, anorexia, rapid RR, general unthriftiness
 - GI: vomiting, colic & constipation or diarrhea;
 - Lameness, swelling & tenderness of fetlock, then darkening & discoloration below fetlock, Sloughing of hoof
 - Convulsive: hyperexcitability, belligerence, ataxia, limb flexure, recumbency, convulsion & opisthotonus (dorsal recumbency & arched back)
- Dx: Ergot ID on grain (rye, wheat or barley), dark purple to black banana-shaped body > grain itself, chromatographic analysis of feed
- DDx: Foot rot, Chronic selenium toxicosis, Laminitis, Frostbite, Fescue toxicosis
- Tx: Remove contaminated grain, supportive Tx, supplemental feeding, antibiotics, Pain control



Description

- Leaves: 3 oval
- Flowers: racemes



Moldy sweat clover

See CIRC pg 141; Melilotus spp., mold on plant synthesizes dicoumerol

- #1 cattle, all susceptible
- CS: Swelling & hemorrhages
- Dx: Exposure, hemorrhages, prolonged PT time, no other clotting abnormalities, anemia
- Tx: Remove source, Vit K1, Fresh plasma
- Px: Good if early Tx



 Black patch diz, Moldy red clover, Slaframine

M8k 192, 2089, 2080; C4T 668; Tox 436; PP/US/C 360; PP/O 18

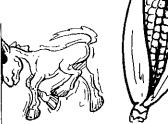


Description: Leaves: in 3 leaflets

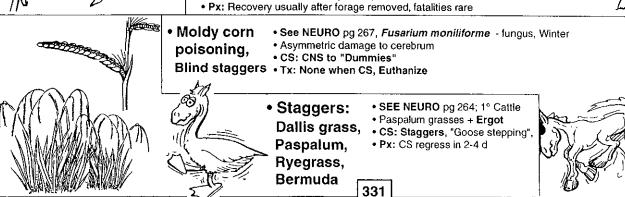
- Flowers: round clusters

- Molded red clover (Trifoleum pratense) or other legumes, Fungus: Rhizoctonia leguminicola, Piperidine alkaloid (slaframine), Wet weather or high humidity
- · Horse, Cattle, sheep & goats; Eastern & central USA
- . CS: "Slobbers", salivation (copious), anorexia, frequent urination, m/b watery diarrhea
- Dx: ID fungus w/ dissecting scope, Chem. ID of Slaframine in hay
- . Tx: Remove from toxic forage, atropine









TOXICOLOGY PP - Snake Bite • 1º Cattle, all susceptible, Cicuta douolasii Water Early Spring if low forage, watery environment hemlock, · CS: Death - resp. paralysis, muscle twitches, tremors, violent convulsions (not w/ poisonous hemlock), salivation, frothing at mouth Cicuta, Tx: Seldom eaten if appropriate feed available, No specific Tx, Poison parsnip, Sedate to control convulsions Description Wild parsley, Both similar: erect 4-8' Snakeroot Flowers: "umbrellas" Mk2 2108; IM 1646, C2T 675; PP/Mt 17; PP/US/C 373 Root Water H. - bundles or root w/ "air chambers" Poison 4 Poison H.: fleshy root Cattle & pig; sheep & horse less susceptible Conium maculatum, alkaloid, extremely toxic - CNS hemlock Confused w/ Water hemlock found in aquatic areas Spring w/ no other forage - nauseating taste Mk2 2130; IM 1646; CS: Similar to water hemlock, except absence of convulsions Tx: Seldom eaten if appropriate feed available, Stimulants, Charcoal PP/ Mt 17; PP/US/C 379 Poison hemlock Pokeweed Nicotine poisoning toxicosis. IM 1647: E 194 Nicotiona tabaccum, wild tobacco Inkberry, Pigeon berry, Nicotine alkaloids Garget CS: Stirnulation of ANS, Paralysis, Death Mk2 2108; IM 1657, PP/O 15; Dx: Exposure CS PP/US/C 225 Description Phytolacca americana Annual, 1-3', sticky Cattle & sheep, occasionally horse & pig. · Leaves alternate, large on bottom CS: GI irritation, chemical oral burns, Lupine toxicity, § hemolytic crisis Lupinosis Mk2 2078, 2088, 2134; IM 1647, PP/Mt 33 Description Description · Perennial, 9', fleshy taproot Perennial shrub 1° Sheep, Cattle common · Leaves: alternate, ovate · Leaves: alternate. Horses infrequent (discriminating eaters) Flowers: racemes CS: agitation, depression palmately compound, 2- Fruit: purple berry Flowers: racemes Venomous Pit vipers Pit vipers · CS, bite wounds • Rarely fatal, not emergency Copperhead (Agkistrodon) - Distinct teeth marks on muzzle snake bites Keep calm Soap Cottonmouth (Agkistrodon pescivorus) - Marked warm edema of lips Thorough cleaning, M8k 2152; Mk 1729; IM 1662; E - Rattlesnake (Crotalarus) - Skin discoloration common w/ rattle 215; EM&S 764; C2T 663; LA-D 86; disinfection & irrigation Massasauga or pygmy rattlesnake Pop 72-7/98 snakes, infreq. w/ copperheads • 1/2" incise & suck venom for 30 min (Sistrurus) Swollen nasal passages (do under 30 min. of bite) Coral snakes - Elapines - Dyspnea Antivenin (Micrurus) w/in 2 hr of bite Eastern or Texas coral snakes Local tissue necrosis - Probably unnecessary in adult horse because (Micrurus) of large body wt. & low fatality 2° clostridial infections Arizona or Sonoran coral snake - Foal may be helpful Cardiac, neurologic & resp. CS rare in - Have epinephrine for anaphylactic shock ready (Micruroides) Toxic principles adult equid, except swollen nose Tracheostomy if dyspnea from swollen nose Coral snakes: neurotoxic - respira- No NSAIDs early (aggravate Coral snakes tory paralysis possible thrombocytopenia) OK in - Pain & swelling minimat Pit vipers: hemotoxic, necrotizing & later stages for pain & edema - Systemic neurologic signs anticoagulant (some neurotoxin) • Antibiotics predominate Identification

- Pit vipers: pit organ on sides of nose
- Rattlesnakes easy: rattle; broad, flat head
- Coral snakes: bands of yellow, red & black (yellow bands in contact w/ red bands in USA), short fangs
- "Pseudo" coral snakes black bands bordering yellow bands on both sides
- Horse: bitten on nose or face (curiosity)
- Rarely fatal in adult equid or bovid
- CS delay of hours for coral snakes



- . Tetanus toxoid (+ antitoxin in unvaccinated horses)
- Foals w/ hypotension, cardiac dysrhythmias, CNS signs
- IV fluids (careful for pulmonary edema)
- Neostigmine for CNS signs (rare)

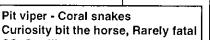


- Abscesses
- Spider bites
- Allergic reaction to insect bites or stings









CS: Swelling, dyspnea Dx: Hx, Cs, bite

Pit viper - Coral snakes

Tx: Cleaning, T. toxoid, Abs

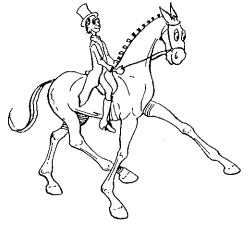
"Red on yellow

- Kill a fellow. Red on black
- Friend of Jack" Good for USA Not for Mexico









DIFFERENTIAL DIAGNOSIS

Abdominal pain	336	Ataxia	349	Chest pain	341	Diarrhea 3	2, 46, 337
Abortion 2	214, 342	Behavior	306	Chloride	357	chronic diarrhea	46
Acute renal failure 1	49, 343	Bilirubin	355	Choke	23	Dysphagia	337
Alkaline phosphatase	354	Blood loss	135	Chronic diarrhea	46	Dyspnea	339
Alopecía	347	Blood Urea Nitrogen	355	Chronic renal failure 15	0, 343	Dystocia	225
Altered sexual function		Brain & brain stem lesions	260	Colic 5	5, 336	Dysuria	343
in stallion	342	Bulla	347	Congestive heart failure	125	Edema	344
Anemia 1	35, 350	BUN	355	Coughing	341	Electrolytes	356
Anestrus 1	62, 342	Calcium	357	CPK (creatine phosphokinase)	354	Elevated FDPs	353
Antithrombin III	353	Cardiac murmur	350	Creatinine	355	Enlarged lymph node	es 350
AP (alkaline phosphatase)	354	Cardiomyopathy	126	Creatine phosphokinase	354	Eosinophilia	351
APTT (activated PTT)	353	Causes of impotency	194	Crusting	347	Epistaxis	340
Ascites	344	Cerebellar lesions	261	Cyanosis	341	Erosions	346
AST (aspartate aminotransferas	e) 354	Cerebral lesions	261	Cystitis	152	Exercise intolerance	348

Colic, Abdominal pain

IM 128, 127; I2M 30, 125; M 25

COMMON CAUSES

- GI common causes
- Gas distention of intest., cecum or colon
- Hypermotility & intestinal spasms
- Feed impaction, constipation (pg 77, 78)
- Meconium impaction (pg 83)
- Gastroduodenal ulcers (pg 26)
- Anterior enteritis (pg 67)
- Extra GI
- Mesenteric abscess (pg 65)
- Ovarian tumor, abscess, or hematoma (pg 188)
- Parturition (pg 224)
- Acute hepatitis (pg 88)
- Hepatic lipidosis (pg 91)
- Diaphragmatic hernia (pg 71)
- Ruptured bladder (foal) (pg 155)
- Urinary tract or renal diz (pg 152)
- Urolithiasis (pg 155)
- Uterine torsions (pg 232)

LESS COMMON CAUSES

- GI less common
- Thromboembolism (cr. mesenteric a.) (pg 75)
- Intest. FB (sand, enterolith, phytobezoar) (pg 79)
- Volvulus of small intestine (pg 69)
- Volvulus or displacement of bowel (pg 29)
- Sand colic (pg 78)

- Pedunculated lipoma w/ bowel strangulation (pg 67)
- Hernia (inguinal, epiploic, umbilical, diaphragmatic) (pg 72, 73)
- Nephrosplenic lig. entrapment (pg 81)
- Ascarid impaction (pg 65)
- Massive strongyle infection (pg 47, 75)
- Gastric dilation (pg 29)
- Peritonitis (pg 53)
- Parasympathomimetic drugs
- Irritant cathartics
- Necrotizing enterocolitis (pg 34)
- Psychogenic colic
- Rectal tears (pg 82)
- Rupture of stomach or intestine (pg 29)
- Ileus (pg 66)
- Intussusception (pg 72) - Cecal dilation (pg 76)
- Enteroliths (pg 79)
- Strangulating lipoma (pg 69)

Extra - Gl less common

- Rabies (pg 271)
- Pleuritis or pericarditis (referred pain) (pg 118)
- Retained placenta (pg 238)
- Uterine rupture or retroflexion
- Spermatic cord thrombosis or torsion (pg 204)
- Rupture of prepubic tendon (pg 233)
- Splenitis
- Splenic abscess
- Splenomegaly
- Cauda equina neuritis (w/ retention of feces or urine) (pg 284)

• TOXIC CAUSES

- Cantharidin toxicity (pg 45)
- Warfarin (pg 143)
- Herbicides
- Lead (pg 269)
- Vacor (pg 315)
- Phenylbutazone or other NSAIDs (pg 27)
- Poisonous plants (pg 313-332)

UNCOMMON CAUSES

- GI Uncommon causes
- Abdominal adhesions (pg 66)
- Intramural hematomas of stomach or intest
- Stenosis or stricture of bowel lumen
- Botulism (og 252)
- Tetanus (pg 253)
- Potomac fever (pg 43)
- Exhaustion
- Anaphylaxis (pg 117)
- Rhodococcus equi gut abscesses (pg 35) Cribbing or wind sucking (pg 307)
- Abdominal fibroma
- Segmental ischemic necrosis following mesocolic tearing
- Eq. viral arteritis (EVA) (pg 143)
- Anthrax (pg 302)
- Displaced colon (pg 80)
- Malignant mesothelioma
- Gastric or intestinal tumor (pg 28, 51)
- Moldy sweet clover (pg 141)

Extra-G1 - uncommon causes

- Perirectal abscess
- Pheochromocytoma (pg 299)
 Purpura hemorrhagica (pg 140)
- Biliary atresia
- Vaginal or vulvar tear
 Cholelithiasis (pg 91)
 White muscle diz (pg 128)

Colic - Neonate

Diarrhea

IM xx; ; I2M xxi, xxii, 121, 122; M 32 Common cause:

- · Idiopathic (pg 40, 46)
- · Salmonellosis (pg 42)
- · Colitis (pg 44)
- Potomac fever (pg 43)
- Endotoxemia (pg 52)
- · Sm. strongyles (pg 36, 47
- Stress induced (pg 40) • Nutritional (pg 40)

Less common causes:

- Gastroduodenal ulcers (pg 26)
- Clostridiosis (pg 44) Chronic liver diz (pg 84)
- Peritonitis (pg 53)
- Eosinophilic gastroenteritis (pg 28)
- Chronic granulomatous bowel diz (pg 50)
- Enterotoxemia (pg 52)
- Intestinal lymphosarcoma (pg 30, 51)
- Proximal enteritis (pg 67)
- Intussusception (pg 72)
- Sand (pg 79)
- Enteroliths (pg 79) • Gut stenosis (pg 66)
- · Antibiotic use (lincomycin,
- tetracycline) • Rhodococcus equi (pg 35, 48)
- Cryptosporidiosis (pg 37)
- Giardiasis
- Parasympathomimetics
- Cathartics, laxatives

- Heart failure (pg 125)
- Large strongyles (pg 47)

- Monensin toxicity (pg 129)
- Arsenic toxicity (pg 313) Mercury toxicity (pg 313)

- Slaframine toxicity (pg 332)

- Jimson weed (pg 326)
- Necrotizing enterocolitis (clostridium) (pg 34)
 Oleander poisoning (pg 329)

 - Mycotoxicosis (pg 330)
 - Avocado poisoning

- · Campylobacter jejuni

- Phenylbutazone toxicity (pg 27)
- Blister beetle toxicity (pg 45)
- Selenium toxicity (pg 321)
- Organophosphate toxicity (pg 312)
- Sulfur toxicity
- Phosphorus toxicity (pg 315)
- Nicotine toxicity (pg 329)

- Poisonous plants:
- Algae poisoning (pg 324)
- Acorn or oak poisoning (pg 324)
- St John's wort (pg 323)
- Castor bean poisoning (pg 327)
- Potato poisoning Japanese Yew poisoning

Diarrhea in foals I2M 391; IM 347

- · Idiopathic
- Foal heat (pg 35) Failure of passive transfer (pg 39)
- Drug induced (ABs.
- phenobulazone) (pg 43, 27) • Foal heat (pg 35)
- · Bacterial septicemia (pg 38) • Salmonellosis (pg 38)
- Rhodococcus equi (pg 35)
- Clostridium perfringens (pg 34)
- Rota virus (pg 38) Corona virus
- Nutritional (pg 38) Parascaris equorum (pg 37)



- S. westerii (pg 36)

- Campylobacter



Dysphagia

- I2M 135; C4T 141; M 33
- Teeth problems (pg 18-20) Choke (pg 23)
- Pharyngeal paralysis Strangles (pg 96)
- Hepatoencephalopathy (pg 84)
- Guttural pouch diz (pg 98)
- Yellow star thistle (pg 267) · Equine protozoal
- EEE, WEE (pg 266)
- Botulism (pg 252)

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- S. vulgaris (pg 36) Cryptosporidia (pg 37)
 C. difficile
- Rabies (pg 271) Trauma (pg 262)
- Laryngeal surgery (pg 105) Lead poisoning (pg 269)
- Esophageal stricture (pg 22)
- myeloencephalitis (pg 249)





I2M 129

Peritonitis (pg 53) 12M 861; IM 674

Infectious:

- Perforation of abdomen or intestine w/ migration of bacteria
- Septicemia
- Enteritis
- Abdominal abscesses (pg 65)
- Surgical complications
- Anastomoses
- Asepsis
- Dead tissue
- Gut trauma
- Wound dehiscence
- Metritis (pg 183)
- Uterine rupture (pg 240)
- Ascending urinary tract infections (pg 152)
- Castration (pg 208)
- Equine influenza (pg 109)
- Equine infectious anemia (pg 136)
- Bastard strangles (og 96)
- Chemical irritants + 2° infection

Noninfectious

- Gastric rupture (pg 29)
- Hepatitis (pg 88)
- Cholelithiasis (pg 91)
- Pancreatitis (pg 91)
- Splenitis
- · Ruptured bladder (pg 154)
- Urinary tract obstruction (pg 155)

• Urolithiasis (pg 155)

- Ovarian tumor (pg 188)
- Hemoperitoneum
- Drugs
- Foreign bodies (drain, sponge)
- Chem (bile, gastric or pancreatic juice, barium, talc, chyle)

Traumatic:

- Perforation of abdominal wall
- Blunt abdominal trauma
- Ruptured diaphragm
- Bleeding accidents
- Foaling accident
- Rupture of esophagus

latrogenic:

- Rectal tear (pg 82)
- Intraperitoneal injection
- Uterine perforation
- Liver biopsy
- · Cecal trocharization (pg 76)
- Enterocentesis
- Peritoneal dialysis

Parasitic:

- · Larval migration
- Verminous arteritis (pg 127)
- · Perforation (tapeworm, ascarids)

Icterus, Jaundice

12M 139; IM 117, 142; Pic 79; Pop 90-2/98

Hemolytic anemia

- Common causes
- , Immune-mediated hemolytic anemia (pg 141)
- . Ehrlichiosis (pg 142)
- , Neonatal isoerythrolysis (pg 137) . Hepatic hyperlipemia (pg 91)
- Less common causes
- . Babesiosis (pg 144)
- . Equine infectious anemia (pg 136)
- . Transfusion reaction (pg 145)
- . Snake bite (pg 332)
- . Cold isoagglutinins
- . Erythrocytosis
- Toxins
- , Phenothiazine poisonings (pg 139)
- , Red maple toxicity (pg 139)
- . Onion toxicity (pg 139)

Hepatic diseases

- Common causes
- , Serum hepatitis (Theiler's) (pg 88)
- . Equine infectious anemia (pg 136)
- . Acute hepatitis (pg 88)
- . Chronic active hepatitis (pg 90)
- . Cholangitis (pg 91)
- . Bile stones (pg 91)
- . Fasting (hyperbilirubinemia)
- . Poisonous plants
- .. Pyrrolizidine alkaloid (pg 89)



- Less common causes

. Lupinosis (pg 329)

. Drug toxicity

. Phenol toxicity

. Mycotoxicosis (pg 330)

. Tyzzer's disease (pg 87)

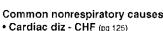
. Hepatic abscess (pg 87)

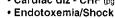


Dyspnea (respiratory distress) 12M 70, 73; IM 76; M 34

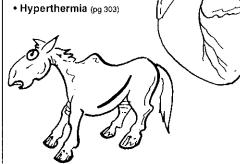
Common respiratory causes

- Pneumonia (bacterial, viral) (pg 111-116)
- Pleuritis (pg 118)
- Pulmonary abscessation (pg 113)
- COPD (Chronic obstructive pulmonary diz) (pg 121)
- Strangles (pg 96)
- Prematurity (RDS foals) (pg 121)
- Neonatal septicemia (foals) (pg 35)
- · Pharyngeal/retropharyngeal abscesses or trauma (pg 97)
- · Partial upper airway obstruction





• Anemia (pg 134) • Pain



Tachypnea

I2M 70, 73; IM 76; M 34 78

Less common respiratory causes

- Fungal minitis (pg 94)
- Nasal polyps
- Ethmoid hematoma (pg 94)
- · Nasal trauma/neoplasia (pg 94)
- Cleft palate (pg 22)
- Arytenoid chondritis (pg 104)
- Guttural pouch empyema, tympany (pg 99)
- Foreign bodies anywhere in resp. tract
- Exercise induced pulmonary hemorrhage (pg 101)
- Lung worms (pg 116)
- Neonatal maladjustment syndrome (pg 270)
- Pneumothorax (pg 121)
- Mediastinal abscess
- Thoracic trauma (rib fxs, etc.)
- Coccidioidomycosis, cryptococcosis
- Bronchospasm

Less common nonrespiratory causes

- Gastric distention (pg 29)
- Pericarditis (pg 124) Cardiovascular defects (pg 130)
- Endocarditis (pg 132)
- Atrial fibrillation (pg 133)

- Left AV valve problems
- Purpura hemorrhagica (pg 270)
- · Blood or plasma transfusions Acidosis
- Intracarotid injection (pg 272)
- Tetanus (pg 253)
- Malignant edema (pg 303)
- · Injection abscess (Clostridium)
- Procaine penicillin G reaction IV
- Liver failure (pg 84)
- Anaphylaxis (pg 117) Tooth root tumors

- Toxic cause • Red maple toxicity (pg 129)
- Blister beetle (pg 45)
- Organophosphates (pg 312)
- · Lead (pg 269) Selenium (pg 321)
- Strychnine (pg 273) • Ryegrass (pg 264)
- Pyrrolizidine alkaloids (pg 89) • Locoweed (pg 265)
- Monensin (pg 129)
- Iron (pg 86) Sodium fluoroacetate (1080) (pg 31)
- · Larkspur (pg 329)
- Oleander (pg 329)
- Water hemlock (pg 329) • ANTU (pg 315)
- Vit. D

From Large Animal Internal Medicine, Smith; I2M 70, 73; IM 76; M 34 for less common causes & more toxic causes









Epiglottic entrapment + infection (pg 107)

- Paranasal sinusitis (pg 102)
- Guttural pouch mycosis (pg 99, 100)
- Inhalation pneumonia (pg 116)
- Diaphragmatic hernia (pg 71)

- Small intestinal obstruction (pg 64) • Pulmonary edema (pg 117)
- · Fluid therapy
- 339

Nasal discharge 12M 56, 60; IM 60- 64; M 42

Serous discharge

- Influenza (pg 109)
- EHV-1 (pg 143) • Rhinovirus (pg 110)
- Other viruses
- Pharyngitis/hyperplasia (pg 95)
- · Nasal/sinus infections, cysts, polyps, tumors
- Early bacterial pneumonia/pleuritis (pg 111-116)
- Early strangles (pg 96)
- Guttural pouch infections/mycosis (pg 100)
- · Overflow of nasolacrimal ducts
- COPD (pg 121)

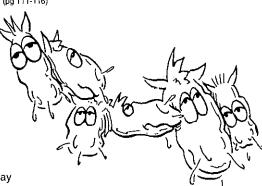
Mucopurulent

- 2° bacterial infections (postviral) (pg 111-116)
- Strangles (pg 96)
- · Bacterial rhinitis
- Pharyngitis (pg 95)
- Pneumonia (pg 111-116)
- Guttural pouch empyema (pg 99)
- Lung abscess (pg 113)
- Pharyngeal abscess (pg 97)
- · Paranasal infections (pg 103)
- Fungal rhinitis (po 94)
- · Nasal foreign body
- · Conchal necrosis
- Ethmoid hematoma (pg 95)
- · Nasal tumors, polyp, cyst
- · Trauma: nasal, skull, upper airway



Ingesta in nasal discharge

- Choke (pg 23)
- Cleft palate (pg 22)
- · Pharyngitis (pg 95)
- Strangles (pg 96)
- Dorsal displacement of soft palate (pg 106)
- · Guttural pouch infection, mycosis, neoplasia (pg 100)
- · Glossopharyngeal nerve damage
- Botulism (pg 252)
- Retropharyngeal abscess (pg 97)



Epistaxis 12M 68; IM 67)

Common Cause

- Ethmoid hematoma (pg 95)
- Nasal polyps
- Fungal granulomas (pg 94)
- · Tumors of nasal cavity
- Trauma from nasogastric tube or endoscope
- Guttural pouch mycosis (pg 100)
- Thrombocytopenia (pg 142)
- Purpura hemorrhagica (pg 140)
- EIPH (exercise induced pulmonary hemorrhage) (rarely see epistaxis) (pg 101)

Less common causes

- Toxic hepatic failure (pg 86)
- Pharyngeal lymphoid hyperplasia (pg 95)
- Cryptococcal rhinitis (pg 94)
- Coccidiomycosis
- Fungal granuloma (pg 94)
- · Guttural pouch empyema, neoplasia, or FB

- Arsenic (pg 313)
- Warfarin/dicoumerol (pg 143)
- Moldy sweet clover (pg 141)
- Pyrrolizidine alkaloids (pg 89)

Uncommon causes see I2M 68, IM 67

Stridor

I2M 85, 90; IM 90; M 44)

Common dauses:

- Laryngeal hemiplegia (pg 104)
- · Dors. displacement of soft palate
- Epiglottal entrapment (pg 73)
- · Retropharyngeal abscess (pg 97
- Strangles (pg 96)
- · Guttural pouch empyema/ mycosis (pg 99, 100)
- Pharyngitis (pg 95)
- Arytenoid chondritis (pg 104)
- · Laxity of alar cartilage

Less common causes: 5

- Botulism (pg 252)
- Guttural pouch tympany (pg 98)
- · Nasal polyps/neoplasm/trauma (pg 94)
- Ethmoid hematoma (pg 95)
- · Sinusitis/tumors (pg 103)
- Tracheal problems/stenosis (pg 95)
- Purpura hemorrhagica (pg 140)
- Anaphylaxis (pg 117)
- Equine influenza (pg 109)
- EIPH (Ex. ind. pulm. hem.) (pg 101)
- Subepiglottic cyst (pg 107)
- · Rostral displacement of palatopharyngeal arch

Toxic causes:

- Lead (pg 269) OPs (pg 312)
- From Large Animal Internal Medicine, Smith for uncom-
- Reserpine (pg 317)

Cyanosis 12M 80, 425; IM 83)

Common causes:

- Bacterial/viral pneumonia (pg 111-116)
- Pleuritis (pg 118)
- Pulmonary abscesses (pg 113, 114)
- Rhodococcus equi, Strep.
- COPD (Chronic obstructive pulmonary diz) (pg 121)
- Aspiration pneumonia (pg 116)
- Prematurity (RDS foals) (pg 121)
- VSD/ tetralogy of Fallot, ASD
- Toxic methemoglobinemia
- Anaphylaxis (pg 117)
- Hypovolemic shock

341

Less common causes:

- Stenotic nares
- Neoplasia
- Nasal granulomas
- Pneumothorax (pg 120) • Pułmonary edema (pg 117)
- Smoke inhalation (pg 314)

Toxic causes:

- Sulfur/hydrogen sulfide (pg 314)
- ANTU (pg 315)
- Chlorinated hydrocarbons (pg 312)
- OPs (Organophosp./carbamate) (pg 312)
- Red maple (pg 139)

From Large Animal Internal Medicine. Smith for uncommon causes

Coughing 341_{12M 46, 54; IM 54; M 30}

Common causes:

- Equine influenza (pg 109)
- Equine herpesvirus-1 (pg 111)
- Rhinovirus, Reovirus (pg 110)
- Bact./aspiration pneumonia (pg 116) • Bacterial pleuritis (pg 118)
- COPD (C. obstr. pulm. diz)(pg 120)
- · Mechanical (dust)

Pharyngitis (pg 95)

- Less common causes: Strangles (pg 96)
- Retropharyngeal abscess (pg 97)
- Pharyngeal/laryngeal trauma or surgery
- Pharyngeal paresis
- Inhalation pneumonia (pg 116)
- Lung worms (pg 116)
- Tracheal collapse/stenosis (pg 107)
- Chondritis of arytenoid cartilages (pg 104) Choke (pg 23)
- Guttural pouch empyema (pg 99) Guttural pouch mycosis (pg 100)
- Parascaris migration (pg 117)
- Pulmonary abscessation (pg 113)
- Epiglottic entrapment (pg 107)
- Subepiglottic cyst or abscess (pg 107)
- EIPH (Exercise induced pulm. hem.) (pg 101) • Neonatal septicemia (pg 35)
- · Left heart failure (pg 125)
- Congestive heart failure (pg 125)
- Equine viral arteritis (pg 111)
- Laryngeal surgery (pg 105)

Toxic causes:

- ANTU (pg 315)
- Pentachlorophenol (pg 314)
- Organophosphate (pg 312) Carbamate (pg 312)

From Large Animal Internal Medicine,



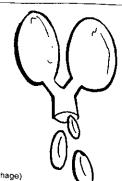
Chest pain

12M 32; IM 30

- Common cause
- Lung abscess (pg 133) Pneumonia (pg 111-116)
- Pleuritis (pg 118)
- Pleuropneumonia (pg 118)

Less common

- Choke (pg 23)
- Ruptured esophagus (pç '2)
- Mediastinal masses (pg):
- Fractured ribs
- Osteomyelitís (pg 246)
- Tying up syndrome





The state of the property of the state of th

Polyuria

(pg 297)

12M 206; IM 202; M 46; C4T 486

Steroid administration

· Diuretic administration

Cushing's syndrome (pg 296)

• Diabetes mellitus (pg 297)

Diabetes insipidus (pg 297)

· Cl, K or urea deficiencies

• Blister beetle (pg 45)

. Salt toxicity or deficiency (pg 313)

• Fluid administration

Hyperglycemia

Chronic renal failure (pg 150)

Psychogenic water intoxication

Acute renal failure (pg 148)

Anestrus 12M 247; IM 243, 1367

- Pregnancy
- Seasonal anestrus (pg 164)
- Transitional (pg 164)
- Prolonged estrus ("split estrus") (pg 165)
- Persistent corpus luteum (pg 166)
- Unobserved estrus (pg 169)
- · Silent heat (pg 166)
- Nymphomania (pg 168)
- Nutritional infertility (pg 168)
- Twinning (pg 176)
- EED/Early embryonic death (pg 176)
- · Genital infections (pg 169)
- Endometritis (pg 183)
- Metritis (pg 183)
- Pyometra (pg 183)
- Cervicitis (pg 184)
- Vaginitis (pg 184)
- "Windsucker" (pg 177)
- Urine pooling (pg 186)
- · Granulosa theca cell tumor (pg 188)
- Ovarian hypoplasia/dysgenesis (pg 190)
- . Lymphangectasis of uterus (pg 191)
- Chronic diz
- Pituitary tumors
- Weight loss



Repeat Breeding (RB)

12M 249; IM 246; M 37

- Common causes
- Transitional season (pg 164)
- Windsucker (pg 177)
- Endometritis/fibrosis (pg 183)
- Twins (pg 176)
- Poor Al timing
- Poor heat detection (pg 169)
- EED (early embryonic death) (pg 176)
- Uterine lymphagectasis (pg 191)
- · Less common causes
- Uterine pooling (pg 186)
- Rectovaginal fistula (pg 236)
- Malnutrition (pg 168)
- Metritis (pg 183)
- Pyometra (pg 183)
- Heat stress
- Poor semen quality
- Uncommon causes:
- CEM (contagious equine metritis) (pg 179)
- Gonadal dysgenesis (pg 190)
- Neoplasia, uterine or cervical (pg 190)
 Ovarian neoplasia (pg 188)
- Parovarian cyst (pg 190)
- Oophoritis
- Salpingitis (pg 190)
- Iodine deliciency
 Phosphorus deficiency
 Zearalenone toxicity
- Intersexuality

Abortion 12M 252; 1M 1397; C4T 532

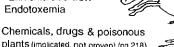
Common causes:

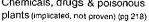
- Twinning (pg 220)
- Early embryonic death (pg 220) Congenital malformations (pg 219)
- Herpesvirus-1 (pg 215)
- Streptococcal (pg 217)

Less common causes:

- Malnutrition (pg 168)
- Equine viral arteritis (EVA) (pg 216)
- EIA (equine infectious anemia) (pg 216)
- Salmonella (pg 217)
- Leptospira (pg 218)
- Mycotic (pg 217)
- Placental insufficiency (pg 218)

- Premature placental separation (pg 218)
- Body pregnancy (pg 218)
- Chromosomal abnormalities (pg 219)
- Fetal diarrhea (pg 219)
- Oxytocin (pg 219)
- Prostaglandins (pg 219)
- Glucocorticoids/severe stress (pg 219)
- Uterine torsion (pg 232)
- Uterine body pregnancy
- Ehrlichia abortion
- Endotoxemia





- Ryegrass (pg 264) Locoweed (pg 265)
- Sorghum (pg 156)Death camus (pg 329)
- · Slaframine (pg 332)





Altered sexual function in stallions

12M 242

Dysuria/stranguria

I2M 34, 196, 426; IM 195; C4T 432

- 1° causes:
- Urethral calculi (pg 154)
- Cystic calculi (pg 154)
- Cystitis (pg 152)
- Sorghum cystitis (pg 259)
- Habronemiasis (pg 200)
- Smegma accumulation (pg 200)
- Penile or preputial trauma (pg 196)
- Urethritis (pg 200)
- Ruptured bladder (pg 155)
- Rabies (pg 271)
- Herpes myeloencephalopathy (pg 254)
- Estrogen-responsive dysuria

2° causes:

- Severe spinal cord diz (pg 245)
- Vertebral body infection or fxs (pg 246)
- Laminitis
- Recumbency
- Mvositis

• Amyloidosis (rare) (pg 151)

Idiopathic glomerulosclerosis (pg 151)

- Peritonitis (pg 53) - Colic (pg 54-81)

Hematuria

I2M 199; IM 198; C4T 489

- Urethra:
- Urethritis (pg 200)
- Calculi (pg 155)
- Habronemiasis (pg 200)
- Idiopathic in males
- Urinary bladder:
- Cystitis (pg 152)
- Calculi (pg 155) - Tumors
- Debris
- Warfarin/dicoumerol (pg 143)

Kidney:

- Trauma
- Calculi (pg 155)
- Nephritis (pg 153)
- Glomerulopathy (pg 149)
- Vascular anomaly
- Parasitic migration (pg 151) Papillary necrosis (rarely hematuria)

Pain on urination

Common causes:

- Bladder calculus (pg 155)
- Rupture of bladder (pg 154)

- Cystitis (pg 152)
- Urethral calculi (pg 155)
- Vaginitis (pg 184)

Acute renal failure

I2M 204; IM 200

- Vasomotor nephropathy:
- Hemorrhagic shock (pg 145)
- Septic shock
- Sepsis
- Congestive heart failure (pg 125) Dehydration
- Thromboembolism

- Aminoglycosides (pg 157)
- NSAIDs (pg 27)
- Oxalate containing plants (pg 157)
- Pigment nephropathy (pg 149)

Heavy metals (Hg, arsenic, lead)

Immunologic:

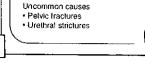
- Glomerulopathy (rare) (pg 149)
- Drug-induced nephritis
- Acute interstitial nephritis (sporadic) (pg 149)

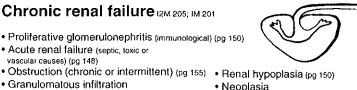
Less common causes:

- Urethritis (pg 152)
- Neoplasia

Oliguria 12M 426; M 46

- Chronic renal failure (pg 150)
- Acute renal failure (pg 148)
- Dehydration (severe)
- Ruptured urinary bladder (pg 155)
- Urolithiasis (pg 154)
- Eq. herpes virus myelitis (pg 156)





 Neoplasia Chronic pyelonephritis (rare) (pg 151) • Parasite migration (pg 151)











Sudden death 12M 288; IM 283-290; M 45; C4T 651

- Noninfectious
- Lightning
- Electrocution
- Gun shot
- Ruptured aorta
- Ruptured heart
- Anaphylaxis (drug reactions) (pg 117)
- Trauma
- GI torsion & rupture (pg 69)
- Heat stroke (rare)
- CNS embolism
- Myocarditis (pg 125)
- Uterine arterial rupture (pg 221)
- Splenic rupture
- Thrombi (verminous) (pg 127)
- Uterine torsion (pg 232)
- Skull fractures
- Diaphragmatic hernia (pg 71)
- Birth trauma
- Infectious
- Malignant edema (pg 303)
- CI. perfringens endotoxemia (pg 34)
- Endotoxins (pg 52)
- Botulism (usually slower) (pg 252)
- Babesiosis (pg 144)
- Potomac fever (pg 43)
- Salmonellosis (pg 34, 42)
- Tyzzer's disease (pg 87)
- Hemorrhagic enterotoxemia (toals)

- Colitis-X (pg 44)

- Anthrax (rare) (pg 302)
- Metabolic/Nutritional
- White muscle disease (pg 128)
- Transit tetany (hypocalcemia, eclampsia)
- Monensin (pg 129)
- Mycotoxins (pg 330)
- Blister beetle (cantharidin) (pg 45)
- Organophosphates/carbamate (pg 312)
- Serum sickness (pg 88)
- Black flies (pg 287)
- Snake bite (pg 333)
- Poisonous plants
 - Sweet clover (pg 141)
 - . Cyanogenic plants (pg 320)
 - Blue-green algae (pg 324) Castor bean (ricinus) (pg 327)

 - Poison hemlock (pg 329) Water hemlock (pg 329)

 - Oleander (pg 329)
 - Tobacco (pg 329)



Peripheral edema/Pleural effusion/Ascites

I2M 101; IM 101; M 35; C4T 269

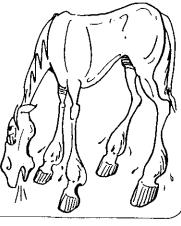
Common causes:

- Pericarditis (pg 124)
- · Heart problems:
 - Congestive heart failure (pg 125)
- Vegetative endocarditis (pg 132)
- Cardiomyopathy (pg 126)
- Lt. or rt. AV valve regurgitation
- Aortic valve insufficiency
- Chronic atrial fibrillation (pg 133)
- Congenital heart defects (pg 130)
- Infectious myocarditis (pg 154)
- Heart base tumor
- Monensin toxicity (pg 129)
- Pleuritis (pg 118)
- Liver disease (pg 85)
- Kidney disease (pg 150)
- Equine viral arteritis (pg 143)
- Vasculitis (pg 127)
- Purpura hemorrhagica (pg 138)
- Equine ehrlichiosis (pg 142)
- Equine infectious anemia (pg 136)
- Thrombophlebitis (pg 127)
- Starvation
- · Hypoproteinemia:
- GI malabsorption (pg 50)
- Peritoneal or pleural effusions
- Ulcerative lymphangitis (pg 293)
- Lymphatic obstruction/
- lymphadenitis

- Trauma
- Tumor
- Cellulitis
- Parasitism
- Drug reactions - Phenobutazone toxicity (pg 27)
- Confinement
- Rhodococcus infection (foal) (pg 35)

Uncommon causes:

- Pregnancy
- Ruptured bladder (pg 154)
- · Angioneurotic edema



Fever 12M 37-45; IM 37-42

Infectious causes:

- Common causes
- Strangles (pg 96) - Pneumonia - bacteria/viral
- (pg 111-116) Upper respiratory viral infections
- (pg 94-107) - GI parasite infection (pg 36)
- Enteritis
- Salmonellosis (pg 34, 42)
- Potomac fever (pg 43)
- Septicemia (pg 35) - Endotoxemia (pg 52)
- DPJ (duodenitis prox. jejunitis) (pg 67)
- Rotavirus diarrhea (foals) (pg 38)
- Peritonitis (pg 53)
- Localized abscesses
- Tumors
- Tetanus (pg 253)
- Tenosynovitis, Cellulitis (pg 143)
- Metritis (pg 183)
- Less common causes:
- EEE/WEE (pg 267)
- EIA (pg 136)
- EVA (pg 143)
- Tyzzer's diz (foals) (pg 87) - Osteomyelitis (adults)
- Bacterial endocarditis (pg 132)
- Mastitis (pg 241)
- Pyelonephritis (pg 151, 153)
- Otitis media/interna (pg 263)
- Vesicular stomatitis
- Malignant edema (pg 303)

Fever - Neonate



- Neoplastic: Common causes:
- Squamous cell carcinoma (pg 294)
- Metastatic melanoma (pg 291)
- Lymphosarcoma (pg 30)
- Fibrosarcoma (pg 291)
- · Less common cause: - Granulosa cell tumors (mares) (pg 188)
- Reticuloendothelial cell sarcoma
- Adenocarcinomas - Myeloproliferative diz (pg 144)

Immunologic causes of fever:

- Common cause:
- Urticaria (pg 281)
- Purpura hemorrhagica (pg 140)
- Drug induced fever
- Less common causes:
- CID (Combined immunodefc, diz) (pg 303)
- IgM deficiency
- Pemphigus foliaceus (pg 280)
- Neonatal isoerythrolysis (pg 137)
- Immune mediated hemolytic anemia (pg 141)
- Thrombocytopenia (pg 142)
- Chronic necrotizing vasculitis

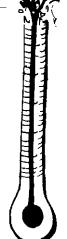


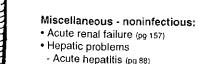
Drugs

- · Common: - Penicillins
- Sulfonamides
- Procainamide
- Quinidine
- Amphotericin B - Antihistamines
- Less common:
- Levamisole
- Cephalosporins
- lodides
- Rifampin
- Furazolidone - Cimetidine

- Toxins: Blister beetle (pg 45)
- Mercury (pg 313) Propylene glycol
- Dinitrophenol
- Arsenic (pg 313)
- Selenium (pg 321) Chlorinated hydrocarbons (pg 312)
- Trichloroethylene Plant toxins:

 - Algae (pg 324) - Castor bean (pg 327)
- Pyrrolizidine alkaloids (pg 89) - Jimson weed (pg 326)
- Water hemlock (pg 329) - Mycotoxicosis (pg 330)





- Chronic active hepatitis (pg 90) - Hyperlipidemia (pg 91) - Chlolelithiasis (pg 91)
- · Foreign bodies (nasal, oral, pharyngeal, tracheal, bronchial) Thrombophlebitis (pg 127)
- Ocular trauma/recurrent uveitis (pg 295)
- Snake bite (pg 332)
- Burns Smoke inhalation (pg 314)







Ulceration & Erosions

- Nodules (see nodular diseases)

- Contact dermatitis (pg 278)

· Pruritus (see pruritic diseases)

Pruritus 12M 226; 1M 221; M 43

- Boredom
- · Pemphigus foliaceous (immune mediated)
- · Infectious diseases (bacteria, viral, parasites, chemical & photoactive dermatitis)
- · Contact dermititis (Allergens: seasonal, pollen, parasite) (pg 278)
- Culicoides hypersensitivity (pg 284)
- . Hypersensitivity reaction (food, drugs or inhalants)
- Rain scald (pg 276)
- Ringworm (99 277)
- Ectoparasites
- Lice (winter) (pg 282)
- Mange (pg 283)
- Mosquito bites (pg 288)
- Pinworms (pg 288)
- Biting files (pg 286)
- Early anaphylaxis (pg 117)
- Sweet itch (pg 284)
- Eosinophilic dermatitis (pg 280)
- Photosensitization (pg 89)
- Neurological dizs (Rabies, Polyneuritis equi, self mutilation syndrome)

Nodules/Tumors/Swellings

I2M 228: IM 223

- Cutaneous & SQ neoplasms (pg 290
- · Hives, urticaria (pg 281)
- Erythema multiforme (rare) (pg 281)
- Cutaneous cysts
- Abscess
- Hypersensitivity reaction
- Insect, spider bite (pg 288)
- Food or inhalant allergies
- Infectious disease
- Cutaneous lymphosarcoma (pg 291)
- Melanoma (pg 291)
- · Sarcoids (pg 290)
- Nodular necrobiosis (pg 280)
- Amyloidosis (pg 289)
- Anaphylaxis (pg 117)
- Cutaneous habronemiasis (pg 284)
- Panniculitis (pg 289)
- Calcinosis circumscripta (pg 289)



Pruritus at head of tail

- Food allergies
- Culicoides hypersensitivity
- Pin worms



- See nodular diseases DDx this page
- Hypersensitivity
- Culicoides (pg 284)
- Food & drug
- Parasites

I2M 228: IM 224

- Tumors

- Hypersensitivity

• External trauma

Hypersensitivity

Cvsts

2° to:

- Biting files (pg 286)
- · Infectious diz (bacterial, viral, fungal)
- Neoplasia
- Papillomas (pg 292)
- Sarcoids (pg 290)
- Pemphigus foliaceus (pg 280)
- Generalized granulomatous diz

Pustules (accumulation of pus)

I2M 226230; IM 225, 226

- Bacterial infections
- Demodex (pg 283)
- Drug eruptions
- Rain scald (pg 276)
- Pemphigus foliaceous (pg 280)



Vesicles/Bulla (accumulation of fluid)

12M 230; IM 225

- Contact dermatitis (pg 278)
- Burns
- Autoimmune diseases
- Pemphigus foliaceus (pg 280)



Scaling/Crusting 12M 231; IM 227

- Self trauma from pruritus (see pruritic diz)
- Rain scald (pg 276)
- Ringworm (pg 277)
- Pemphigus foliaceous (pg 280)
- Cushing's diz (og 298)
- Cutaneous filariasis (pg 286)
- Photosensitization (pg 289, 328)
- . Contact irritation (pg 278)
- · Generalized granulomatous diz (pg 289)
- Anhydrosis (pg 298)
- · Aural plaques (pg 292)
- Reticulated leukotrichia (pg 278)
- Seborrhea (pg 279)
- Lice (pg 282)
- Folliculitis (pg 277)



Hives, Uticaria

pg 281; I2M 1407; IM 1261

Allergic urticaria:

- Ingestants
- Food or beverages
- . Strawberries

- Drugs

- Dust
- Volatile chemicals
- Feather down

fungal or parasitic

- Animal products
- Plastic
- Plant material

- Penicillin
- Quinine

- . Citrus fruits
- . Some fish . Food additives

Inhalants:

- Pollens
- Dander
- Smoke

- Mold spores

Infections:

- Foci of bacteria, viral,

Contactants:

- Cosmetics
- Many chemicals
- Drugs:



- Insulin
- Cocaine
- Morphine
- Codeine - Atropine
- Polymyxin B

Injectants:

- Drugs
- Insect bites/stings
- Serums
- Blood - Diagnostic agents

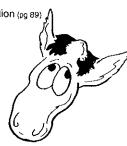
- Toxins: Cobra venoms
- Plant toxins
- Jellyfish toxin

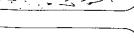
- Insects

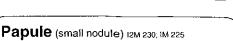
- Physical: - Heat
- Cold
- Light - Dermographic
- Secondary:
- Infections Neoplasia
- Psychogenic Collagen vascular disease

Alopecia 12M 2331; IM 228; M 26

- Self trauma (pruritus or ulceration)
- Trauma to skin • Stress from high fever or severe illness &
- parturition
- · Congenital alopecia
- · Contact dermatitis (pg 278)
- Rain scald (pg 276)
- Ringworm (pg 277) · Dietary deficiencies
- · Drug hypersensitivity
- Black hair follicle dystrophy of Appaloosas
- Ectoparasites - Lice (pg 282)
- Mange (pg 283)
- Biting flies (pg 286) - Mosquitoes (pg 288)
- · Grease heel (pg 279)
- Anhydrosis (pg 299) · Sarcoids (pg 290)
- Mercury blister • Photosensitization (pg 89)









Exercise intolerance 12M 92, 98, 111; IM 97; M 36

Respiratory:

- · Roaring (pg 104)
- Displacement of soft palate (pg 106) Degenerative joint disease Monensin (pg 129)
- Epiglottic entrapment (pg 73)
- Arytenoid chondritis/chondroma
- Pharyngeal lymphoid hyperplasia Fractures
- Guttural pouch infections (pg 99)
- Stenotic nares/enlarged alar fold/ nasal polyps
- Tracheal problems (pg 95)
- Ethmoid hematoma (pg 95)
- Paranasal sinus empyema (pg 103) Fluid & electrolyte imbalances
- EIPH (Exercise induced pulmonary hemorrhage)(pg 101)
- Pneumonia viral/bact (pg 111-116)
 Liver diseases (pg 84)
- Pleuritis (pg 118)
- Pulmonary abscessation (pg 113)
- Diaphragmatic hernia (pg 71)

Cardiovascular causes:

- · Atrial fibrillation (pg 133)
- · Heart block
- CHF (pg 125)
- Valvular insufficiency or stenosis
- VSD/ASD/ Tetralogy of Fallot
- Endocarditis/myocarditis (pg 132)
- Pericarditis (pg 124)
- Thrombosis (pg 127)

Musculoskeletal causes:

- Tying up syndrome

- Back problems
- Tendonitis/desmitis
- Chronic sarcocystosis
- Sacroiliac problems Osteochondrosis
- Hoof imbalance

Metabolic/Systemic causes:

- Heat exhaustion
- Diarrhea diseases (pg 32-49)
- · Skin diseases generalized • Anemia (pg 134)
- Anhydrosis (pg 298)
- · Febrile diseases
- · Myeloproliferative disease
- Thyroid tumors

General causes:

- Unconditioned
- Poorly trained
- · Slow horse





Toxic cause:

- Locoweed (pg 265)

- · Ryegrass (pg 264) Pyrrolizidine
- alkaloids (pg 89) Organophosphates
- (pg 312)
- Lead (pg 269)
- Vit. D

Weight loss - Foals M 47

Common causes:

- Septicemia (pg 38)
- Malnutrition
- Failure of passive transfer (pg 39)

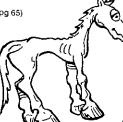
Less common causes:

- Parascaris equorum (pg 37)
- Strongyloides westerii (pg 36)
- Rhodococcus equi (pg 35)
- · Bacterial GI infections (Salmonella,
- E. coli, Clostridium, Rhodococcus) (pg 35)
- Small intes. intussusception (pg 72)
- Chronic diarrhea (pg 46)
- White muscle diz (pg 128)
- Liver diz (pg 84)
- Idiopathic

Weight loss 12M 171, 180, 189; IM 188; M 46) Common causes • Parasitism (pg 24, 35) Malnutrition

- Teeth & jaw problems (pg 18-22)
- COPD (chronic obstructive pulm. diz) (pg 121)
 - Strangles
 - Chronic infections
 - Liver diz (pg 84)
 - Pneumonia (pg 111-116)
 - Lung abscesses (pg 113)
 - Rhodococcus equi (pg 113)
 - Pleuritis (pg 118)
- Sand colic/impaction (pg 78)
- Cribbing, Wind sucking (pg 307)
- Pyrrolizidine alkaloid toxicity (pg 89)
- S. vulgaris thromboembolism (pg 75)
- SCC of stomach (pg 26)
- · Gastroduodenal ulcers (pg 26)
- Neoplasia
- Obstruction of small colon (pg 77)
- Peritonitis (pg 53)
- Abdominal abscesses (pg 65)
- Wound mylasis
- Infectious arthritis
- Heart failure • Renal diz (pg 150)
- Idiopathic

See Large Animal Internal Medicine, Smith, for less common & uncommon causes



Ataxia M 29; C4T 651

Common causes:

- Head/spinal cord trauma (pg 245, 262) /
- Protozoal myeloencephalitis (pg 250)
- Cervical vertebral malformation (pg 247)
- Herpes myeloencephalitis (pg 254)

Less common causes:

- Equine degenerative myeloencephalopathy (pg 249)
- Viral encephalomyelitides (pg 266)
- . Hepatoencephalopathy (pg 268)
- · Lead poisoning (pg 269)
- Vestibular syndrome (pg 263) Botulism (pg 252)
- Locoweed toxicosis (pg 265)
- Organophosphate toxicosis (pg 312, 273)
- Cauda equina neuritis (pg 248) • Neonatal maladjustment syndrome (pg 270)
- · Vertebral osteomyelitis (pg 246)
- Septicemia (pg 38) • Otitis media/interna (pg 263)
- Rabies (pg 271)
- Narcolepsy/epilepsy (pg 272)
- · Idiopathic epilepsy (Arabians)
- Verminous myeloencephalitis (pg 251)





Metabolic acidosis 12M 457; 1M 402

Common causes:

- Acute diarrhea (pg 32-49)
- Colic w/ strangulate bowel (pg 54-81)
- Peritonitis (pg 53)
- Ruptured bladder (pg 155) Excess exercise



Respiratory acidosis 12M 458; IM 404

Common cause:

- Obstruction of upper
- respiratory tract
- · Laryngeal edema
- Pneumonia (pg 111-116)
- Pneumothorax (pg 121)
- Chronic obstructive pulmonary disease (pg 120) • Depression of respiratory center of CNS
- General anesthesia
- Drugs (opiates, anesthetics & tranquilizers)
- CNS disease

Metabolic alkalosis 12M 457: IM 404

- · Gastric reflux w/ iteus (pg 67, 66) · Excessive sweating in endurance horses
- · Salivary loss w/ esophagostomy
- Diuretic · Chloride &/or potassium depletion



Respiratory alkalosis 12M 458; IM 405

Common causes:

- Hypoxemia
- Pulmonary disease (pg 93)
- Severe anemia (pg 134)
- Congestive heart failure (pg 125)
- Stimulation of CNS respiratory center
- Neurologic disorders - Psychogenic hyperventilation
- Septicemia (pg 35)
- Transport, pain, fear, excitement





↑ PCV 12M 478; 1M 424

- Relative erythrocytosis:
- Dehydration
- Endotoxic shock
- . Strangulating intest, obstruction (pg 66)
- . Salmonellosis (pg 34, 42)
- . Colitis -X (pg 44)
- . Septic metritis (pg 183)
- Splenic contraction
- · Absolute erythrocytosis:
- Common causes
- . COPD (Chronic obstr. pulm. diz) (pg 121)
- . Residence in high latitudes
- . Congenital cardiovascular diz
- Less common cause
- . Hepatoma (pg 87)
- . Pheochromocytoma (pg 288)
- . Myeloproliferative diz (og 144)
- . Chronic hepatic diz (pg 90)
- . Hemangioblastoma

From Large Animal Internal Medicine, Smith pg 424 for uncommon causes

Cardiac murmur 12M 108



Anemia 12M 476; IM 423; M 27

Blood loss:

- Common causes:
- Intestinal parasites (strongylosis) (pg 36)
- Ectoparasites (lice [pg 272], ticks)
- Gastric ulcers (pg 26)
- Immune-mediated thrombocytopenia
- Gastric SCC (squamous cell carcinoma) (pg 28)
- Equine purpura hemorrhagica (pg 140)
- Less common causes:
- DIC (disseminated intravascular coagulation) (pg 142)
 Moldy sweet clover toxicosis (pg 143)
- Warfarin poisoning (pg 143)
- Hemophilia A or other congenital factor deficiency
- Guttural pouch mycosis (pg 100)

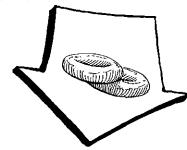
Hemolysis:

- · Common causes:
- Neonatal isoerythrolysis (pg 139)
- EIA (equine infectious anemia) (pg 138)
- Red maple leaf toxicosis (pg 141)
- Equine ehrlichiosis (pg 135)
- Less common causes:
- Onion poisoning (pg 141)
 Autoimmune hemolytic anemia
- Babesiosis (piroplasmosis) (pg 144)
- Clostridial infection
- Incomplete blood transfusion

Inadequate RBC production

- Common causes:
- Chronic inflammatory dizs
- Abdominal abscess or other chronic abscessation
- Chronic pneumonia/pleuritis (pg 118, 143)
- Equine purpura hemorrhagica (pg 140)
- Equine ehrlichiosis (pg 135)
- Lymphosarcoma (pg 30)
- Chronic liver diz (pg 143)
- · Less common causes:

- Myelogenous leukemia Equine viral arteritis/EVA (pg 134) Chronic renal failure (pg 148)
- Radiation toxicosis
- Idiopathic aplastic anemia



Muffled heart sounds 12M 108

Jugular venous distention 12M 112

Enlarged lymph nodes 12M 115

Neutrophilia (↑ PMNs)

I2M 483; IM 429

Common causes:

- Exercise/excitement
- Stress/corticosteroid administration
- Chronic salmonellosis or colitis (pg 46)
- Chronic peritonitis (pg 53)
- · Abdominal/internal abscesses (pg 65)
- Strangles (pg 96)
- Chronic pneumonia (pg 118)
- Chronic pleuritis (pg 118)
- Purpura hemorrhagica (vasculitis) (pg 138)
- Thrombophlebitis (pg 127)

Less common:

- Chronic hepatitis (pg 90)
- Cholelithiasis (pg 91)
- Lymphosarcoma/internal neoplasia (pg 31)
- · Bacterial endocarditis (pg 132)
- · Autoimmune hemolytic anemia (pg 141)
- · Granulocytic leukemia
- Pyelonephritis (pg 151, 153)
- Cellulitis (pg 276)



Neutropenia (+ PMNs)

I2M 484; IM 430

Common causes:

- Acute peritonitis (pg 46)
- Septicemia/endotoxemia (pg 52)
- Neonatal septicemia (pg 35)
- Acute salmonellosis (pg 42)
- Acute toxic colitis
- Proximal enteritis (DPJ) (pg 67)
- Acute pleuritis (pg 118)
- Equine influenza (pg 109)
- Snotts (EHV-1) (pg 111)
- Acute metritis (pg 183)

Less common causes:

- Potomac fever (pg 43)
- Ehrlichiosis
- Equine viral arteritis (pg 143)
- · Idiopathic aplastic anemia
- Radiation therapy



Lymphocytosis († lymphocytes)

12M 485; IM 431

Common causes:

Exercise/excitement

Uncommon causes:

Lymphocytic leukemia

EIA (equine infectious anemia)



Lymphopenia (+lymphocytes)

12M 485; IM 431

Common causes:

- · Stress/corticosteroid administration
- Endotoxemia/septicemia (pg 52)
- . Acute peritonitis (pg 46)
- Influenza (pg 109)
- Equine herpes virus-1 (pg 111)

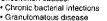
Less common causes:

- Starvation/malnutrition
- CID (combined immunodeficiency) (pg 303)
- EVA (equine viral arteritis) (pg 143)
- Pyometra (pg 183)

Monocytosis (†monocytes)

I2M 486; IM 432

Uncommon causes:





Eosinophilia (+ eosinophils)

12M 486; IM 432

- Uncommon cause: Cutaneous habronemiasis (pg 284)
- Internal parasites
- Lymphosarcoma (pg 31)
- Eosinophilic leukemia · Systemic hypersensitivity reaction



Hyperproteinemia 12M 492; IM 439

Panhyperproteinemia - dehydration

Common cause:

- Salmonellosis (pg 34, 42, 48)
- Acute toxic colitis
- Intestinal clostridiosis (pg 44)
- Potomac fever (pg 43)
- Proximal enteritis (pg 67)
- · Sepsis/endotoxemia (pg 52)
- Intestinal strangulation (pg 66, 79)
- Choke (pg 23)
- Botulism (pg 252)

Less common causes:

- Chronic hepatic disease (pg 90)
- Guttural pouch mycosis w/ dysphagia (pg 100)
- Chronic renal failure (pg 150)
- Protozoal myelitis
- Salt toxicosis (pg 313)
- Toxins/poisonous plants
- Lead toxicity (pg 269)
- Yellow star thistle poisoning (pg 267)
- · Idiopathic dysphagia

Hyperglobulinemia

Common cause:

- Abdominal abscesses (pg 65)
- Bastard strangles (pg 96)



- Pulmonary abscess (pg 96)
- EIA (equine infectious anemia) (pg 136)
- Purpura hemorrhagica (pg 140)

Less common causes:

- Lymphosarcoma (pg 30)
- Strongyloides (pg 36)
- · Chronic hepatic disease (pg 90)

Hypoproteinemia (+ protein) 12M 494; IM 442 • Gastrointestinal ulceration (pg 26)

Hypoalbuminemia

Common causes:

- Parasitism
- Protein losing enteropathy (pg 50)
- Idiopathic granulomatous enteritis (pg 50)
- Intestinal lymphosarcoma (pg 30, 51)
- Saimonellosis (pg 34, 42, 48)
- Potomac fever (pg 43)
- Colitis X (pg 44)
- Clostridiosis (pg 44)
- . Chronic hepatic fibrosis (pg 90)
- Pyrrolizidine alkaloid toxicity (pg 89)
- . NSAIDs (nonsteroidal antiinflammatory drugs)
- Glomerulonephritis (pg 148)
- Pyelonephritis (og 151, 153)

Less common causes:

- Starvation
- Chronic eosinophilic gastroenteritis (pg 28)
- Chronic hepatitis (pg 90)
- · Hepatic neoplasia (pg 87)
- + Amyloidosis (pg 149)
- Tuberculosis
- Histoplasmosis

Panhypoproteinemia \

Common causes:

- · Excessive fluids or water intake
- Strangulation GI obstr./infarction (pg 66, 75, 79)
- Protein-losing enteropathy (granulomatous) (pg 50)
- Acute peritonitis (pg 53)
- · Acute blood loss (pg 145)
- Glomerulonephritis (pg 148)
- NSAIDs (nonsteroidal anti-inflammatory drugs) (pg 27, 43)

Less common causes:

- · Parasites blood sucking internal or external
- Intestinal lymphosarcoma (pg 30, 51)
- Congestive heart failure (pg 125)
- DIC (disseminated intravascular coagulopathy) (pg 140)
- Immune-mediated thrombocytopenia (pg 142)
- Urinary blood loss:

Prolonged APTT

· Acute hepatic necrosis

Less common causes: Moldy sweet clover toxicosis (pg 141)

• Warfarin toxicosis (pg 141)

Pyrrolizidine alkaloids (pg 89)

- Rubratoxins, aflatoxins (pg 330) - Bitterweed (pg 327)

Common causes:

- Congenital vascular disorders, renal trauma, renal calculi (pg 155), pyelonephritis (pg 151, 153), neoplasia, cystic calculi (pg 155)

(Activated partial prothrombin time) 12M 501; IM 448

Congenital deficient factors IX, XI, hi-molecular weight kininogen

DIC (Disseminated intravascular coagulation) (pg 140)

Hyperfibrinogenemia 12M 496; IM 443

Common causes:

- Abscessation
- · GI inflammation
- Salmonellosis (pg 34, 42, 48)
- Chlolelithiasis (pg 91)
- Peritonitis/abscesses (pg 53)
- Pneumonia (pg 111-116)
- Pleuritis (pg 118)
- Purpura hemorrhagica (pg 140)
- Osteomyelitis
- · Septic arthritis
- Cellulitis (pg 143)

Hypofibrinogenemia 12M503; IM450

- Acute severe DIC

Common causes:

coagulation) (pg 140)

(pg 142)

Uncommon causes:

Thrombophlebitis (pg 127)

· Postoperative states

 Massive internal hemorrhage Primary hyperfibrinolysis

- Acute hepatic necrosis



- · Severe hepatic fibrosis

Elevated FDPs (Fibrogen

degradation products) 12M 502; IM 449

• DIC (Disseminated intravascular

· Severe inflammatory disorders

• IMTP (Immune-mediated thrombocytopenia)

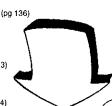
Thrombocytopenia (+ platelets) I2M 499: IM 446

Common causes:

- DIC (disseminated intravascular coagulation) (pg 140)
- Immune-med. thrombocytopenia (INTP) (pg 142)
- Endotoxemia/septicemia (pg 52)
- Acute toxic colitis
- Intest. strangulating obstruction (pg 68, 80)
- Neonatal septicemia (pg 35)
- Lymphosarcoma (pg 30, 51)
- Ehrlichiosis (pg 43)
- EIA (equine infectious anemia) (pg 136)

Less common cause:

- Salmonellosis (pg 34, 42, 48)
- Influenza (pg 109)
- EVA (equine viral arteritis) (pg 143)
 Aplastic anemia
- Stachybotryotoxicosis
- Piasma cell myeloma
- Myeloproliferative disease (pg 144)



Prolonged PT (prothrombin time)

I2M 500; IM 447

Common cause:

- DIC (disseminated intravascular coagulation) (pg 140)
- Warfarin toxicosis (pg 143)
- Acute hepatic necrosis (pg 148)
- . Chronic hepatic fibrosis (pg 150)
- Pyrrolizidine alkaloid toxicosis (pg 89) Aflatoxicosis (pg 330)

Less common causes:

Moldy sweet clover (pg 141)



Reduced antithrombin III

I2M 502; IM 450

Common cause:

Hepatotoxins

Hemophilia A

or prekallikrein

- DIC (Disseminated intravascular coagulation) (pg 140)
- Chronic glomerulonephritis
- Protein losing enteropathy
- Intestinal lymphosarcoma
- Granulomatous enteritis - Phenylbutazone toxicosis
- Starvation

Less common causes: Antithrombin

- Acute hepatic necrosisVenous thrombosis

353

Serum enzyme elevation 12M xxv, 461; IM xxii

SDH

(sorbitol dehydrogenase)

- Common causes:
- Acute liver failure (pg 88)
- Liver abscess (pg 87)
- Damaged bowel
- . Strangulation (pg 66)
- . Acute toxic enteritis
- · Less common cause:
- Chronic liver failure (pg 90)
- Acute & severe anemia (pg 134)
- General anesthesia
- Anorexia

GGT 4

(gamma-glutamyl transferase)

- Common causes:
- Acute liver failure (pg 88)
- Chronic liver failure (pg 90)
- Pyrrolizidine alkaloid toxicity (pg 89)
- Aflatoxicosis (pg 330)
- Cholangiohepatitis (pg 91)
- Cholelithiasis (pg 91)
- Normal range in young

(alkaline phosphatase)

Common cause:

- Acute liver failure (pg 88)
- Chronic liver failure (pg 90)
- Cholangiohepatitis (pg 91)
- Cholelithiasis (pg 91)
- · Hi normal range in young



(creatine phosphokinase)

Common cause:

- Tying up (muscle damage)
- Nutritional myodegeneration (Se/Vit E defc) (pg 128)
- Malignant hyperthermia (pg 303)
- · After endurance rides

Uncommon cause:

- Post exercise increase
- Equine influenza
- Cardiomyopathy
- IM injection
- Purpura hemorrhagica

LDH 4

(lactate dehydrogenase)

Common cause

- Muscle disease
- Tying up (muscle damage)
- Nutritional myodegeneration (Se/vit E defc) (pg 128)
- Malignant hyperthermia (pg 303)
- After endurance rides
- Liver disease
- Acute liver failure (pg 88)
- Chronic liver failure (pg 90)
- Cholangiohepatitis (pg 91)
- Cholelithiasis (pg 91)
- Hemolysis (pg139-143)

- Uncommon cause:
 Acute cardiomyopathy (pg 126)
- Hemolytic anemia (pg 141)
- Equine influenza (pg 109)
- Purpura hemorrhagica (pg 140)
 IM injection



(aspartate aminotransferase)

Same as for LDH

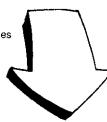


BUN # 4

↓ BUN (blood urea nitrogen) 12M 466; IM 411

Common causes:

- Liver failure (pg 84-91)
- · Normally lower in neonates



♣ BUN (blood urea nitrogen) 12M 466; IM 411

Common causes:

- Prerenal azotemia
- Hypovolemia
- Congestive heart failure (pg 125)
- Reduced renal perfusion
- Dehydration endurance exercise
- Renal azotemia
- Acute renal failure (pg 148)
- Chronic renal failure (pg 150)
- Postrenal azotemia
- Urolithiasis (renal, ureteral, urethral calculi) (pg 155)
- Ruptured bladder (pg 155)



12M 464: IM 408

- Common causes:
- Hemolytic anemia (pg 134)
- Liver failure (pg 84-91)
- Systemic disease
- · Fasting/anorexia
- ↑ Indirect bilirubin

Common causes:

- Hemolytic anemia (pg 134)
- Liver failure (pg 84-91)
- Systemic disease
- · Fasting/anorexia

• ↑ Direct bilirubin

Common cause:

- Liver failure (pg 84-91)
- Cholelithiasis (pg 91)
- Cholangiohepatitis (pg 91)
- Neonatal isoerythrolysis (pg 137)





♦ Hypoglycemia 12M 464; IM 409

Common cause:

- Anorexia in newborn
- Late endotoxic shock

★ Hyperglycemia

I2M 464; IM 409 Common cause:

- Excitement & stress
- Acute colic (pg 54-81)
- Cushing's syndrome (pg 296)
- Xylazine administration
- Glucocorticoid administration

Creatinine ↑ 12M 465; IM 410





- Prerenal azotemia
- Hypovolemia
- Congestive heart failure (pg 125)
- Reduced renal perfusion
- Dehydration endurance exercise
- · Renal azotemia
- Acute renal failure (pg 148)
- Chronic renal failure (pg 150)
- · Postrenal azotemia
- Urolithiasis (renal, ureteral, urethral calculi) (pg 155)
- Ruptured bladder (pg 155)







Hyponatremia (¥ Na) 12M 450; IM 395

Common causes:

- Decreased fluid volume
- Diarrhea (pg 32-49)
- Blood loss (pg 145)
- Excessive sweating
- Fluid drainage
- , Pleural drainage
- , Gastric reflux (pg 67)
- Sequestration of fluid in 3rd space
- . Ascites
- . Peritonitis (pg 53)
- . Ruptured bladder (pg 155)
- . Torsion or volvulus or gut (pg 69, 80)
- Adrenal insufficiency (pg 297)
- Hyperlipidemia (pg 91)
- Hyperglycemia
- . Excitement & stress, Cushing's diz, Acute colic, Steroid administration, xylazine administration
- Excessive 5% dextrose to renal diz patient

Uncommon causes:

- Water retention (normal circulatory volume)
- Renal disease (pg 148) Excess ADH (antidiuretic hormone) (pg 296)
- Psychogenic polydipsia



Hypernatremia (↑ Na) 12M 450; IM 396

Common causes:

- Water loss
- Water deprivation (pg 313)
- · Sodium excess (water restriction/salt poisoning) (pg 313)

Uncommon causes:

- Water loss >> electrolyte loss
 Diarrhea (pg 32-49)
- Vomition
- Burns
- Intrinsic renal diz
- Diabetes insipidus (pg 297)
- Hypertonic saline or NaCO3 administration
- Mineralocorticoid excess (pg 296)



Hypokalemia (↓ potassium) 12M 451; IM 396

Common causes:

- Diarrhea (pg 32-49)
- Gut torsion/volvulus (pg 69, 80)
- Peritonitis (pg 53)
- Metabolic acidosis
- · Dietary deficiencies
- · Prolonged anorexia Vomition

Uncommon causes:

- Mineralocorticoid excess
- Renal tubular acidosis Diuretics
- · Postobstructive diuresis
- Excessive NaCO2 administration
- Excess catecholamines
- · Insulin or glucose administration

Hyperkalemia (↑ potassium) 12M 451; IM 397

Common causes

- Hypovolemia w/ renal shutdown
- · Metabolic acidosis
- Excessive exercise
- · False hyperkalemia
- In vitro hemolysis
- Prolonged storage



- · Renal disease (pg 148)
- Addison's disease
- · Hyperkalemic periodic paralysis in Quarterhorses
- Tissue necrosis
- Diabetes mellitus (pg 297)

Hypochloremia (+ cloride) 12M 452; IM 398

Common causes:

- Diarrhea (pg 32-49)
- Blood loss (pg 145)
- Fluid drainage
- Peritonitis (pg 53)
- Ruptured bladder (pg 155)
- Ascites
- Excessive sweating
- Gastric reflux (pg 66, 67)

False hypochloremia

- Hyperglycemia
- Hyperproteinemia
- Hyperlipidemia (pg 91)

Psychogenic polydipsia (pg 297)

Hypochloremia w/o hyponatremia

Common causes:

- Metabolic alkalosis
- Lasix (furosemide) response in horses

Hyperchloremia (↑ CI) 12M 452; IM 398

Common causes:

- Water deprivation (pg 313) Salt poisoning (pg 313)

Hyperchloremia w/o hypernatremia

Common causes:

- · Hyperchloremic metabolic acidosis
- Renal tubular necrosis (pg 148)

Hypocalcemia (+ calcium) 12M 454; IM 399 Common causes:

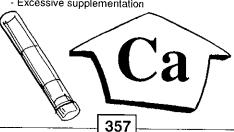
- Lactation tetany
- Hypoalbuminemia
- Transport tetany
- Blister beetle toxicosis (pg 45)
- Diaphragmatic flutter
- Acute renal failure (pg 148)



Hypercalcemia (↑ Ca) 12M 454; IM 400

Common causes:

- Chronic renal failure (pg 150)
- · Excessive, rapid IV calcium
- Hypervitaminosis D
- Excessive supplementation



Hypophophatemia (♦ phosphorus) 12M 45S; IM 400

Common causes:

Chronic renal failure (pg 150)



Hyperphosphatemia (↑ phosphorus) 12M 455; IM 401

Common cause:

- Acute renal failure (pg 148)
- Excessive phosphate intake
- Hi normal range in neonates
- Endurance exercise



Hypomagnesemia (**♦** magnesium) 12M 455, 1M 401

Common cause:

Endurance exercise



Hypermagnesemia

(↑ magnesium) 12M 455, 1M 401

No common cause





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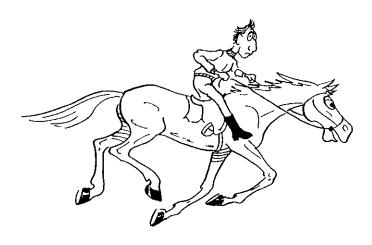
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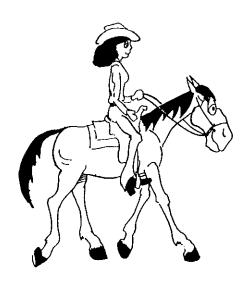
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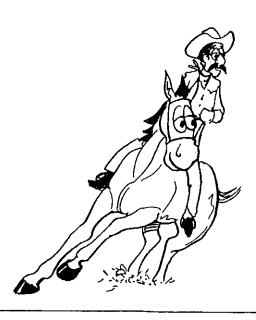
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1 dl (deciliter) $= 100 \text{ ml} (10^2 \text{ml})$

 $= 0.001 L (10^{-3}L)$ = 15 minims (16.23) 1 ml (milliliter)

= 1 cc (cubic centimeter)

 $= 0.000001 L (10^{-6})$ 1 μL (microliter)

1 fl oz (fluid oz) ≈ 30 ml (29.57)

≈ 500 ml (473.2) 1 pt (pint) = 16 floz≈ 1/2 L (473.2 ml) = 2 cups

≈ 1 L (946.4 ml) = 1.136 imperial L 1 qt (quart)

≈ 4000 ml (3785.6) 1 gal (gallon) ≈ 4 L

> = 4 quarts = 4.55 imperial L

= 0.833 imperial gal

≈ 250 ml = 8 fl oz1 cup

= 1/20 ml1 drop = 5 ml1 tsp (teaspoon) $= 15 \, ml$ 1 tbsp (tablespoon)

≈ 0.06 ml (0.062) 1 minim

Length

1 yd (yard) = 91.44 cm≈ 1 m

≈ 30 cm (30.48) 1' (ft/foot)

1" (in/inch) = 2.54 cm

≈ 40 " (39.37) 1 m (meter) ≈ 1 yd

= 100 cm

1 cm (centimeter) ≈ 0.4 " (0.39) $= 10 \, \text{mm}$

1 mm (millimeter) ≈ 0.04 " (0.039)

Weights

1 kg (kilogram) $\approx 2.2 \text{ lb } (2.205)$ = 1000 g

1 mg (milligram) $= 0.1 \text{ g } (10^{-3} \text{ kg})$ = 1/65 (0.015) gr

1 g (gram) ≈ 15 grains (15.43) = 10 mg

= 0.035 oz

1 μg (microgram) $= 0.000001 g (10^{-6})$

1 ng (nanogram) $=(10^{-9})$ 1 pg (picogram) $= (10^{-12})$

1 gr (grain) $\approx 65 \text{ mg } (64.8)$ $\approx 0.065 \text{ g}$

1 oz (ounce) $\approx 30 \text{ g } (28.35)$

1 lb (pound) $\approx 1/2 \text{ kg } (0.454)$ = 16 oz= 1 part per million 1μg/gm = 1 mg/kg

= 2,000 lb1 ton

1 metric ton = 1,000 kg= 2,205 lb

Drug abbreviations

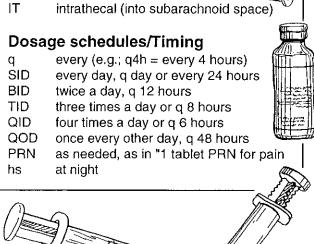
Routes of administration

PO per os, by mouth or orally SQ subcutaneous injection IM intramuscular injection IV intravenous injection

IΡ intraperitoneal (into peritoneal cavity)

IN intranasal IC intracardiac

intrathecal (into subarachnoid space)



Blood Tube "Run l					
Color (ingredients)	Uses	Comments [Resulting testing solution]			
Red (nothing)	Common chemistries Serology Occult heartworm	[Serum]			
Red & black (None, separator)	Common chemistries Serology Occult heartworm	[Serum]			
Green (lithium heparin)	"Stat" chemistries Hormone tests Ammonia	"Stat" = Don't have to wait for clotting [Heparin plasma]			
Black (Batroxobin enzyme + soybean trypsin inhibitor)	Fibrin degradation product: (FDP)	s Used for DIC dogs [serum]			
Gray (NaFI)	Glucose	[Plasma]			
Gray (Diatomaceous earth)	Activated clotting time	[Whole blood clot]			
Gray w/ bright yellow label (nutrients)	Urine culture	[Urine]			
Blue (Na Citrate)	PT & PTT Von Willebrand's diz (hereditary coagulopathy	moor in tabe completely light			
Purple (EDTA)	CBC Fibrinogen Microfilariae heartworm Hormone tests	[EDTA plasma]			

Serology

From Anja's Nerd book

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Abb	reviations
a, aa	artery (ies)

	eviations			Т "			
a, aa	artery (ies)	ext	external	m/b	maybe	R/O	Rule out
abd	abdomen	FB	foreign bodies	med	medial	RR	respiratory rate
abnorm	abnormal	Feds	Federal agents	membr	membrane	rt	right
ABs	antibiotics	fx, fxs	fracture (s)	metab	metabolic	Rx	drug (s)
bact	bacterial, bacteria	gen	general	min	minute	SID	once a day
BID	twice/day	hi	high	MLV	modified live virus	sm	small
bilat	bilateral	hr(s)	hour(s)	mo (s)	month, months	spec	spectrum
C&S	Culture & Sensitivity	HR	heart rate	n, nn	nerve (s)	ŞQ	subcutaneous
caud	caudal	hs	at night	neg	negative	supf	superficial
CrN	cranial nerve	Hx	history	norm	normal	Sx	surgery
conc	concentrate	IM	intramuscular	p-ANS	parasympathetic	thru	through
cran	cranial	IN	intranasal	PCV	packed cell volume	TID	three times/day
CS	clinical signs	incr	increase	perf	perforating	TP	total protein
d(s)	day, days	infec	infection	PM	postmortem	Tx	treatment
decr	decrease	inflam	inflammation	PMNs	neutrophils	UMNs	upper motor neuron:
DDx	differential Dx	int	internal	PO	per os/orally	usu	usually
defc	deficiency	IP	intraperitoneal	pos	positive	unilat	unilateral
degen	degenerative	IV	intravascular	ppt	precipitate	vac	vaccination
dist	distal	jt	joint	PRN	as needed	ventr	ventral
diz	disease	lat	lateral	preg	pregnancy	V, VV	vein (s)
DJD	degenerative joint diz	lb	pound	prblm	problem	w/	with
DOC	Drug of Choice	lg	large	prox	proximal	w/i	within
dors	dorsal	lig (ligg)	ligament (s)	Px	prognosis	w/o	without
Dx	diagnosis	LMNs	lower motor neurons	q	every (as in q 12 hrs)	wk(s)	week, weeks
elev	elevated	In, Inn	lymph node (s)	QID	four times/day	wt.	weight
envir	environment	It	left	repro	reproduction	yr(s)	year, years
esp	especially	m/	may	resp	respiratory	\$ `	expensive

Equine Clinical Chemistry:

Normal ranges

Total bilirubin < 2.5 mg/dl Direct reacting < 0.8 Indirect reacting 0.1-2.0 mg/dl Cholesterol 75-200 mg/dl Creatinine 1-2 mg/dl Glucose 50-115 mg/dl Fibrinogen 100-500 mg/dl Total Protein 5.4-7.9 g/dl 2.3-3.9 g/dl Albumin 0.6-1.5 A/G ratio 10-25 mg/dl BUN (Urea nitrogen)

Enzyme

ALP (Alkaline phosphatase) 149-395 IU/L AST, SGOT (Aspartate aminotransferase) 150-550 IU/L 90-350 IU/L CPK (Creatine phosphokinase) 11-40 IU/L GGT (Gamma-glutamyl transferase) 160-490 IU/L LDH (Lactate dehydrogenase) 3-14 IU/L SDH (Sorbitol dehydrogenase)

Electrolyte

Na (Sodium) 132-147 mEq/L K (Potassium) 2.4-4.7 mEq/L 95-110 mEq/L CI (Chloride) Ca (Calcium) 9.1-13.4 mg/dl P (Phosphorus) 2.9-4.9 mg/dl Mg (Magnesium) 1.3-3.2 mg/dl

Normal values - RBCs & WBCs

PCV (%)(hematocrit) 32-48% RBCs (Erythrocytes) 6-12 x 10⁶/µl Hab (Hemoalobin) 10-18 a/dl MCV (mean corpuscular vol.) 34-58 fL MCH (mean corpuscular Hgb) 13-19 pg MCHC (mean corpuscular Hgb conc.) 31-37 g/dl Reticulocytes (%) 0%

Thrombocytes 100,000-600,000 μΙ

WBC 6-12 x 103/µl PMNs (Neutrophils) 30-75% (3-6 x 103/µl) Bands 0-1% $(0-0.1 \times 10^3/\mu l)$ Lymphocytes 25-60% (1.5-5x 103/µl) Monocytes $(0-0.6 \times 10^3/\mu l)$ 1-8% Eosinophils 1-10% (0-1 x 103/µl) Basophils 0-3% $(0-0.3 \times 10^3/\mu l)$ N/L(Neutrophil:lymphocyte) ratio 0.8-2.8

All Labs vary widely!!!

These are only general guidlines for interpreting laboratory data M8k 2188-2194; C4T 761; I2M 474, 481, 491, 498; IM 103

Acid-base (venous blood)

Rectal temp

Heart rate (HR)

Respiratory rate

& specific gravity

Urine volume

99.7°F (37.6°C)

100°F (37.8°C)

1.025-1.060

40 (26-50) beats/min

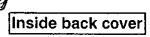
12 (10-14) breaths/min

3-18 ml/kg body wt./d

Stallion

Mare

pΗ 7.32-7.44 PCO₂
HCO₃ (Bicarbonate) 38-46 mm Hg 20.7-28.9 mEq/L Total CO, 24-32 mEq/L



Stix