Small Animal Medicine Module 9

Gastroenterology 1 – The Liver and Pancreas

Richard A. Squires
Massey University
New Zealand

Certificate in SA Medicine

THE ROYAL COLLEGE OF VETERINARY SURGEONS

CERTIFICATE IN SMALL ANIMAL MEDICINE - COMMENTARY AND SYLLABUS

Commentary

1. The syllabus for small animal medicine relates only to the dog and cat and is presented as a guide to candidates. It should not be considered comprehensive. It details the major areas upon which the examination will be based. Examiners will assume that candidates have a good general knowledge of the pathophysiological basis of disease and of clinical pharmacology. Examiners will also expect candidates to be familiar with current literature in small animal medicine.

2. Candidates should possess both theoretical and practical knowledge of small animal medicine as it applies to general practice. Emphasis will be placed on a systematic problem solving approach to procedures required to establish a specific diagnosis and on therapeutic modalities. Candidates must have gained experience in the management of common medical diseases.

3. A detailed knowledge of rare conditions and a practical knowledge of highly sophisticated methods of diagnosis and therapy will not be required.

4. Candidates should have a sound knowledge of suitable patient selection and prognosis.

5. Welfare and ethical considerations in treatment of small animal cases - candidates are reminded of their commitment as registered members of the Royal College of Veterinary Surgeons to pay attention to the welfare of animals under their care.

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6. **RESPIRATORY DISORDERS**
Clinical evaluation of the respiratory system
Principles and applications of diagnostic aids (radiology, endoscopy)
Diagnosis and management of the common disorders

7. **GASTROINTESTINAL DISORDERS**
Clinical evaluation of the gastrointestinal system, including the liver and pancreas
Principles and applications of diagnostic aids (laboratory tests, radiology, endoscopy, ultrasonography)
Diagnosis and management of the common disorders

8. **ENDOCRINE AND METABOLIC DISORDERS**
Clinical evaluation of the endocrine system
Principles and applications of diagnostic aids (laboratory testing, radiology, ultrasonography)
Diagnosis and management of the common disorders

9. **UROGENITAL DISORDERS**
Clinical evaluation of the urinary and genital system
Principles and applications of diagnostic aids (laboratory testing, radiology, ultrasonography)
Diagnosis and management of the common disorders

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1. A good general knowledge of the pathophysiological basis of disease and of clinical pharmacology.
2. Familiarity with current literature in small animal medicine.
3. Theoretical and practical knowledge of small animal medicine as it applies to general practice.

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4. Systematic problem-solving approach.
5. Experience in the management of common medical diseases.
6. Detailed knowledge of rare conditions and practical knowledge of highly sophisticated diagnostic methods and therapy **not** required.

Case challenge

A 7 year-old FS Doberman pinscher is presented to an emergency room in a coma. The owner reports some depression and 'vacancy' over the last several weeks. Faeces are black and tarry on rectal examination. PCV and TPP are both moderately low. There is moderate hypoglycaemia. A buccal mucosal bleeding time is markedly prolonged but the activated clotting time is within the normal range.

Case challenge

Ultimately this dog turns out to be comatose as a consequence of advanced chronic hepatitis and gastrointestinal bleeding. Reflect upon this information and generate some plausible pathophysiological explanations for the clinical observations. Why wasn't the chronic active hepatitis detected earlier?

What is a 'clinical problem'?

"Any deleterious deviation from normality, described at the level you currently understand it"

Why focus on clinical problems, rather than specific diseases, patterns or syndromes?

- Because problems are what you are presented with in clinical practice, and you have to try to solve them
- Thinking in terms of syndromes encourages 'pattern recognition' diagnosis, which can be less inclusive

A problem can be:

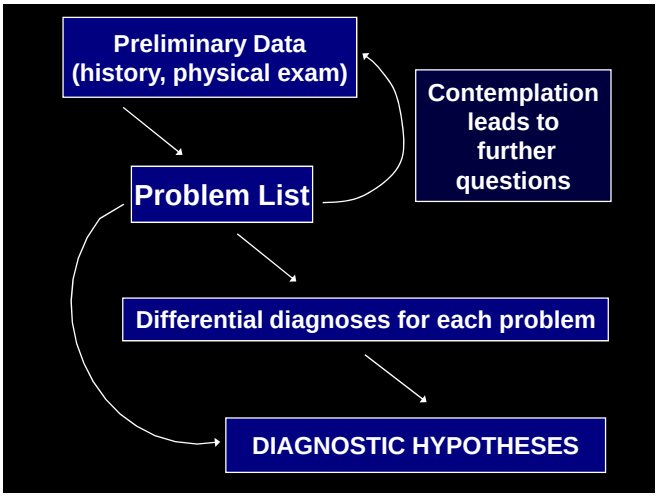
- The owner's presenting complaint
- A historical finding you elicited
- A physical examination finding
- An abnormal laboratory test result
- An abnormal radiographic finding
- Any other detrimental abnormality
- A problem deduced from combinations of the above

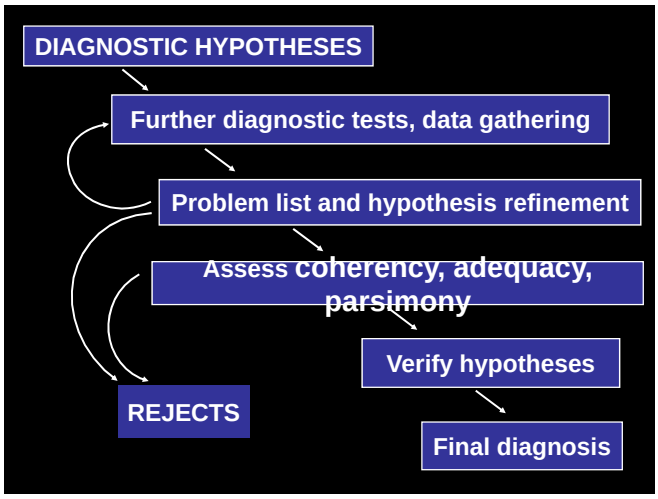
Table 1-1 Some common medical problems of dogs and cats

Abdominal mass	Abdominal pain	Abdominal swelling /enlargement
Abortion	Aggression	Alopecia
Anisocoria	Anorexia / inappetance	Arthritis
Ascites	Ataxia	Back / neck pain
Bleeding (excessive, prolonged)	Blindness	Cachexia
Cardiac arrhythmia	Cardiac murmur	Coma / stupor
Constipation / tenesmus	Cough	Cyanosis
Deafness	Diarrhoea	Drooling
Dysphagia	Dyspnoea	Dysuria / pollakiuria / stranguria
Epistaxis	Faecal incontinence	Fever
Flatulence / borborygmi	Gagging	Growth retardation
Haematochezia	Head shaking	Head tilt
Hepatomegaly / microhepatica	Hypothermia	Icterus / jaundice
Inappropriate urination	Incontinence (faecal, urinary)	Infertility
Joint swelling(s)	Lameness	Lymphadenopathy
Melena	Muffled heart / lung sounds	Muscular pain / swelling
Nystagmus	Obesity	Ocular pain / blepharospasm
Oedema	Otitis	Pallor (mucous membranes)
Paralysis	Paresis	Petechiae / ecchymoses
Pica	Pleural effusion	Pneumothorax
Polydipsia / polyuria	Polyphagia	Preputial discharge
Proprioceptive deficits	Pruritus	Red eye(s)
Regurgitation	Retinal abnormalities	Seizure(s)
Skeletal / bone pain	Skeletal swelling / deformity	Skin lesions (*e.g.*, pustules, scales)
Sneezing / nasal discharge	Splenomegaly	Stridor / stertor
Syncope	Tachypnoea	Urinary incontinence
Urine discolouration	Urinary outflow obstruction	Urine spraying
Vaginal discharge	Vomiting (acute or chronic)	Weakness (episodic or persistent)
Weight loss		

Plus:

- Problems identified as a result of clinicopathological laboratory tests (e.g., nonregenerative anaemia)
- Problems identified during imaging studies (e.g., splenomegaly)
- Problems identified by surgery, endoscopy, biopsy
- Others





Law of Parsimony

"the assertion that no more causes or forces should be assumed than are necessary to account for the facts"

Etymology
ME f. L parsimonia, parcimonia f. parcere pars- spare

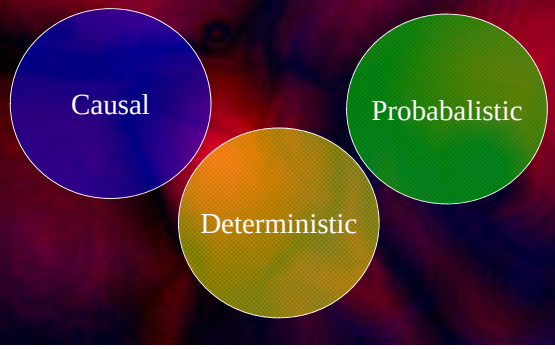
Compared to experts, novices tend to...

- Misidentify or miscategorize problems
- Consider fewer diagnostic possibilities; *i.e.*, narrow down too soon
- Cling more tenaciously to their diagnostic hypotheses, even in the face of strong 'conflicting' data

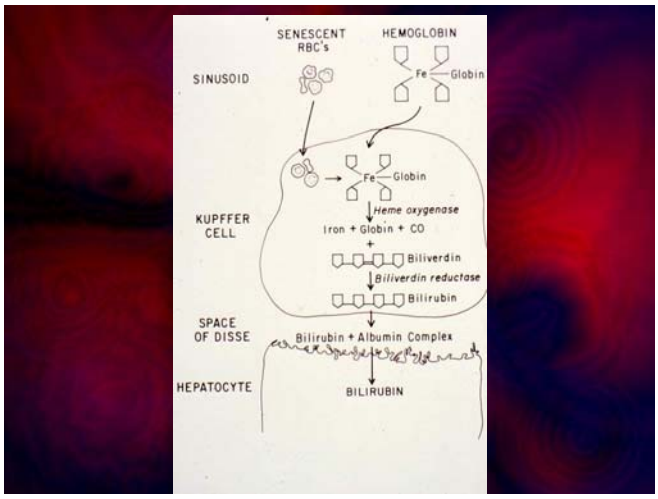
Diagnostic reasoning is...

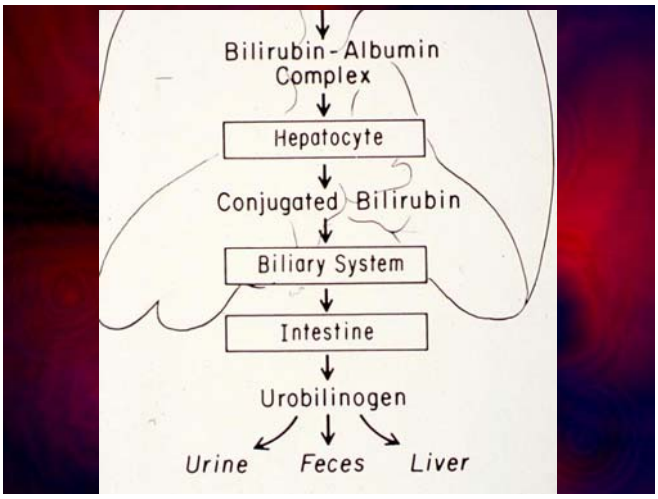
- Intricate
- Integrative
- Multi-layered
  - Causal
  - Probabilistic
  - Deterministic
- Error prone

Diagnostic Reasoning



The problems





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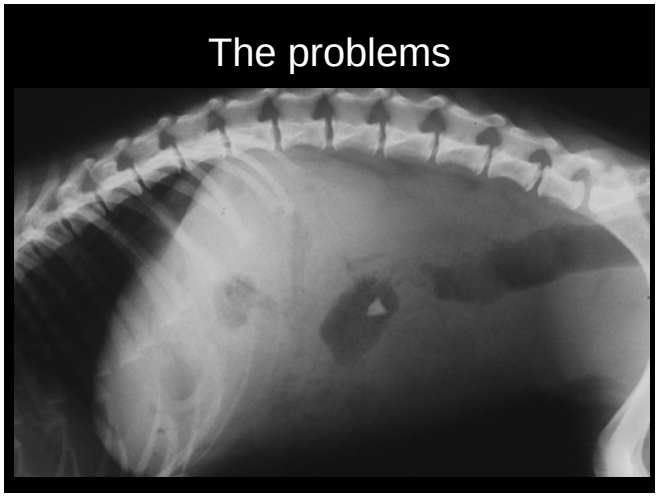

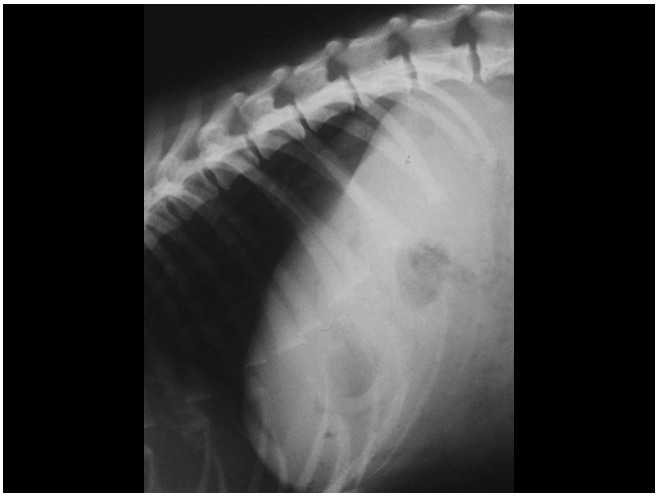

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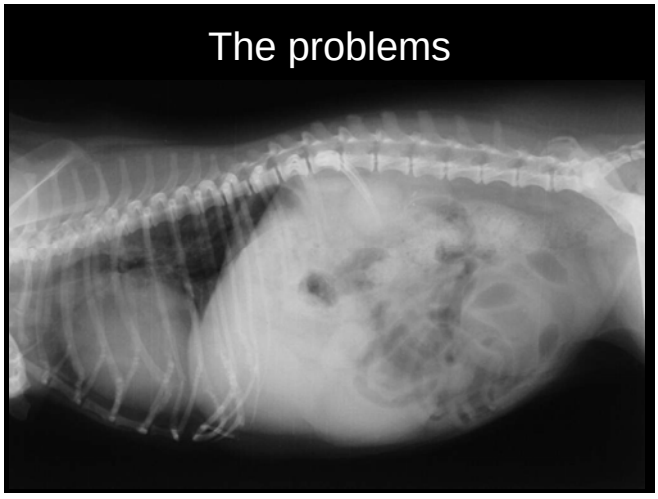
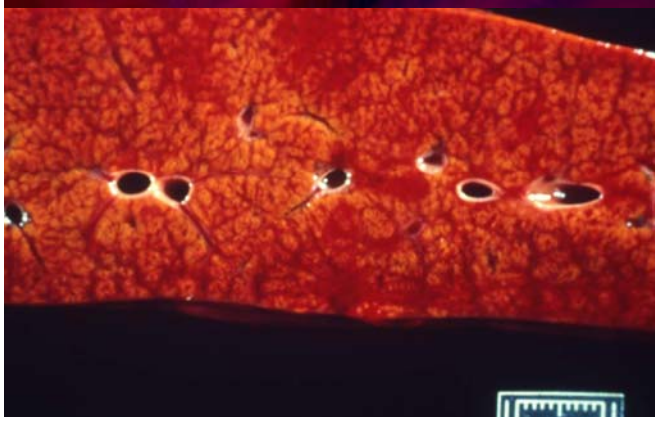
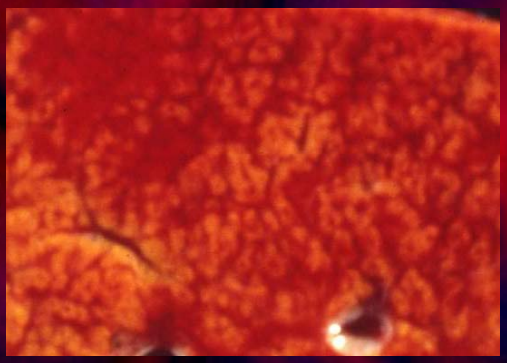


Chronic passive liver congestion



Chronic passive liver congestion



A real nutmeg, cut open

The problems

The problems

The problems

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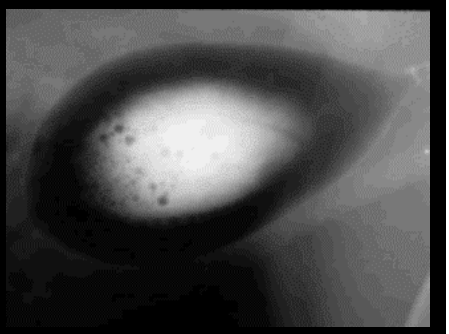

The problems

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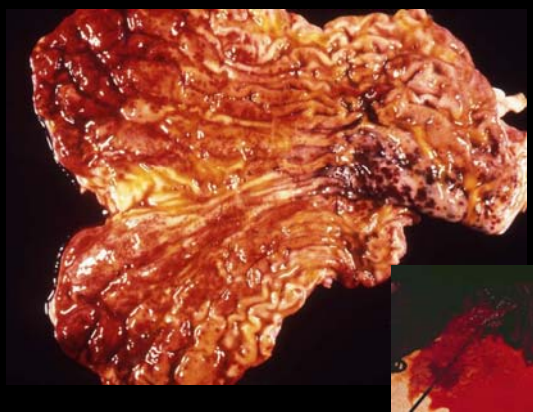


Pattern recognition: Signalment

- Bedlington terrier
- West Highland white terrier, Skye terrier, Dalmatian, Siamese cat (plus, perhaps, many others)
- Doberman pinscher (especially middle-aged females), English and American cocker
- Persian and Himalayan cats (often less than two years of age)

Pattern recognition: Signalment

- Yorkshire terrier, Maltese, Dandie Dinmont terrier, Pug, Miniature schnauzer (often less than two years of age)
- Australian cattle dog, old English sheepdog, Irish wolfhound, Golden and Labrador retrievers (often less than two years of age)
- Cairn terriers

Pattern recognition: Signalment

- Chinese Shar Pei; Abyssinian, Siamese and Oriental cats

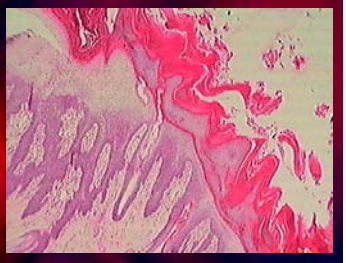
History

History

- Stunted growth;
- Previous cystotomy for ammonium biurate urolithiasis (suggests PSS);
- Recent treatment with a potentially hepatotoxic drug;
- Anaesthetic intolerance;
- Drug intolerance;
- Recent, marked weight loss and anorexia in a previously obese cat (often after stress; suggests hepatic lipidosis).

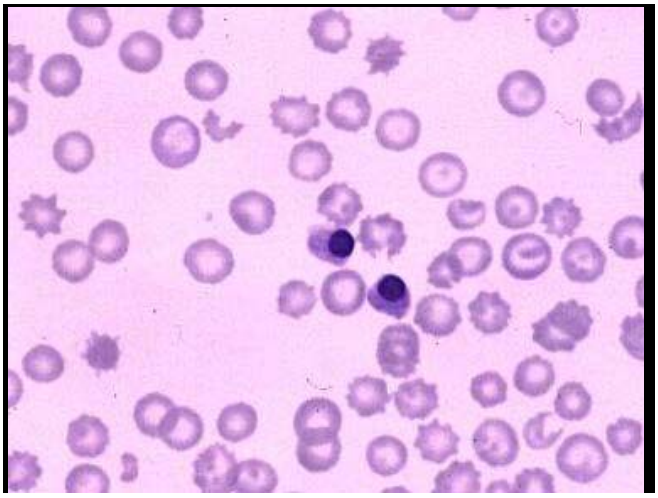
Physical examination

Physical examination



from Vetcutis

Laboratory tests



human

Liver enzymes

- ALT
- AST
- ALP
- KGT
- Arginase

Biochemistry

	Biddy	Units	Ref. Range
Calcium	2.66	mmol/L	2.03 - 2.91
Phosphorous	2.04	mmol/l	1.01 - 3.53
CK	315	IU/l	53 - 821
AST	53	IU/l	2 - 79
Total Protein	72 ↑ slight	g/L	47.4 - 69.6
Albumin	40 ↑ slight	g/L	28.7 - 38.7
Globulin	33	g/L	
A/G ratio	1.22		0.83 - 2.01
ALT	298 ↑ 5x	IU/l	3 - 58
ALP	4007 ↑ 25x	IU/l	8 - 167
Urea	3.8	mmol/l	2.5 - 8.4
Creatinine	44 ↓	umol/l	57 - 126
Amylase	442	IU/l	350 - 920
Lipase	221	IU/l	14 - 252
Glucose	(4.0)	mmol/l	3.8 - 5.8 — ? recheck
Bilirubin	1.6	umol/l	0 - 6
Cholesterol	10.5 ↑	mmol/l	3.2 - 9.3

Haematology

Analysis of an EDTA sample using the Abbott CELL DYN 3500 analyser and manual differential

Tests			Units	Ref. Range
RBC	8.1		$x10^{12}/l$	5.5 - 8.5
Hb	181		g/l	120 - 180
HCT	0.53		l/l	0.37 - 0.55
MCV	66		fl	60 - 77
MCH	22.4		pg	
MCHC	339		g/l	310 - 360
WBC	18.20 ↑		$x10^9/l$	6.0 - 15.0
Bands	0.00		$x10^9/l$	<0.54
Seg. Neutrophils	17.11	94% ↑	$x10^9/l$	3.6 - 11.5
Lymphocytes	0.36	2%	$x10^9/l$	1.0 - 4.8
Monocytes	0.73	4%	$x10^9/l$	0.2 - 1.5
Eosinophils	0.00		$x10^9/l$	0.1-1.5

Erythrocyte morphology:
Red cells show mild anisocytosis with occasional Howell Jolly bodies present.

Platelet morphology:
Normal

Alkaline phosphatase

- $T_{1/2}$ cats 6 hours, dogs 70 hours
- glucocorticoid-induced isoenzyme in dogs
- Anticonvulsants also induce this enzyme
- Numerically modest increases in cats (say 250-300 U/L) are very impressive *vs.* dogs

Patient factors we need to consider

- Species
- Age
- Breed
- Gender & pregnancy
- Drugs
- Hydration & nutritional status
- Stress & intercurrent illnesses

Species

Laboratory Value	Species differences
Alanine aminotransferase	Normal feline ALT, ALP and bilirubin concentrations are lower than those of dogs. ALP is particularly important: modest elevations of ALP are much more significant in cats than in dogs
Alkaline phosphatase	
Total bilirubin	
Cholesterol	Cholesterol is usually lower in cats than in dogs
Creatine kinase	CK values can be variable in both species, but particularly in cats
Creatinine	The normal range for creatinine extends higher in cats than in dogs
Phosphorus	The normal range for phosphorus and urea extends lower in dogs than in cats

Age

Laboratory Value	Age differences
Calcium	Associated with active bone growth, serum calcium and phosphorus are higher in puppies than in adults. Phosphorus is usually more substantially elevated than calcium.
Phosphorus	
Alkaline phosphatase	Puppies have two to three fold higher serum alkaline phosphatase than adults throughout the period of skeletal growth. This is a consequence of the bone isoenzyme. Levels are even more impressive (20 to 25 fold elevation over adult levels) during the first few days of postnatal life. This may be because of intestinal absorption of intact alkaline phosphatase from colostrum.
Bilirubin	Slightly higher in very young puppies than in adults. It declines to the adult level by about two weeks of age.
Creatinine	Somewhat lower in young animals than adults, because of relatively low muscle mass.
Urea	Serum urea nitrogen concentration depends heavily on the length of the pre-sample fast and the protein content of food previously ingested. Making the rather artificial assumption of equal duration of fast, and identical food; urea would be somewhat lower in puppies than adult dogs.
Total protein	Concentration is lower in young animals than in adults because of low albumin and globulin levels. Albumin concentration reaches an adult level by about two months of age. Globulin takes longer.

Some extrahepatic causes of serum liver enzyme elevation

Cause	ALT	AST	ALP	γ-GT
Glucocorticoid overexposure	+	+	+++	+/++
Anticonvulsant therapy	+	+	+/++	+
Feline hyperthyroidism	+		+	
Canine hypothyroidism			+	
Diabetes mellitus			+	
Muscle damage	+	++		
Young growing animal			++	
Late pregnancy (queen)			+/++	
Severe anaemia / hypoxaemia	++	+	-/+	-/+

Liver enzymes decline in advanced chronic disease (there's less remaining tissue to leak or produce enzymes)

Case scenario

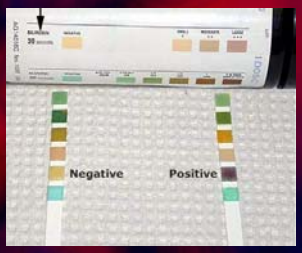
An apparently healthy 14 year-old FS Beagle is presented for an annual health check. Physical examination reveals no significant abnormalities. Routine blood work and UA reveals an ALT of 312 U/L (ref. range 8-60) but no other abnormalities.

What would be your approach?

Serum chemistry

- Albumin / globulin
- Cholesterol
- Glucose
- Urea
- Bilirubin

Urine analysis



Serum bile acids

1: Aust Vet J. 1995 Apr;72(4):121-6. Related Articles, Links

Erratum in:
- Aust Vet J 1995 Jun;72(6):238.

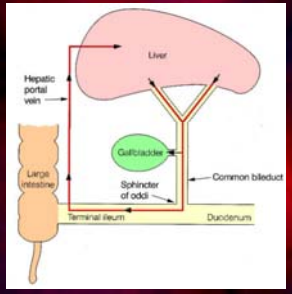
Post-prandial serum bile acid concentrations and ammonia tolerance in Maltese dogs with and without hepatic vascular anomalies.

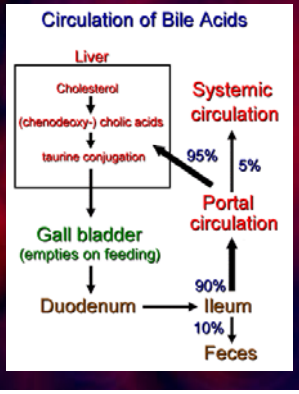
Tisdall PL, Hunt GB, Tsoukalas G, Malik R.

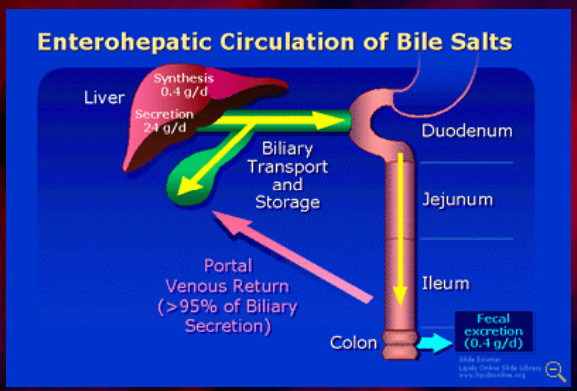
Department of Veterinary Anatomy, University of Sydney, New South Wales.

Post-prandial serum bile acid concentrations were measured in 200 Maltese dogs in an attempt to identify those with subclinical portosystemic shunts. Five of these were later shown to have hepatic pathology or abnormal liver function. In the other 195 Maltese, bile acid concentrations ranged from 1 to 362 mumol.L-1 (mean +/- SD, 70 +/- 50 mumol.L-1; median, 65.0 mumol.L-1). Of these, 79% were above the reference range (0 to 31 mumol.L-1) established from 23 mixed-breed control dogs. It was therefore not possible to determine the prevalence of subclinical portosystemic shunts on the basis of bile acid determinations. Further investigation of liver function was performed to investigate why bile acid concentrations were increased in these dogs. Rectal ammonia tolerance tests were normal in 102 of 106 Maltese tested and liver samples (11 dogs) and plasma biochemistry profiles (9 dogs) demonstrated no significant hepatic disease or dysfunction. Of 2 Maltese with hyperammonaemia after administration of ammonium chloride, one had a large congenital portosystemic shunt that was confirmed at surgery. In the other there were no macroscopic portosystemic communications, but a liver biopsy showed histological changes consistent with microscopic portovascular dysplasia. Total serum bile acid concentrations were consistently lower when assessed by high-performance liquid chromatography than by an enzymatic spectrophotometric method. This discrepancy was substantially larger in Maltese than in control dogs, suggesting the presence of an additional reacting substance in the serum of Maltese dogs.

Serum bile acids







Blood ammonia

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Invited critical review

Diagnostic methods in hepatic encephalopathy

Juan C. Quero Guillén*, Juan M. Herrerías Gutiérrez

Department of Digestive Diseases, University Hospital Virgen Macarena, Avda. Dr. Fedriani 3, 41007 Seville, Spain

Received 15 April 2005; received in revised form 3 August 2005; accepted 3 August 2005

Abstract

Hepatic encephalopathy (HE) is a neuropsychiatric syndrome in patients with liver disease and/or portosystemic shunting that affects quality of life and prognosis. The diagnosis is primarily based on clinical criteria that classify HE into 5 grades of severity ranging from normal mental status (grade 0) to coma (grade 4). As this clinical classification is rather subjective, additional diagnostic methods are required. Biochemical diagnostic tests can be used to confirm or exclude the diagnosis and to monitor the effect of treatment. An elevated ammonia level plays a central role in the pathogenesis of HE and can be determined in arterial, venous and capillary blood.

Blood ammonia

J S Afr Vet Assoc. 1997 Jun;68(2):66-8.

Related Articles, Links

Transient hyperammonaemia in an adult German shepherd dog.

Lobetti RG, Miller DB, Dippenaar T.

Department of Medicine, Faculty of Veterinary Science, University of Pretoria, Onderstepoort, South Africa.

A 3-year-old male German shepherd dog was presented with severe generalised seizures. The dog was protein-intolerant and showed severe hyperammonaemia on ammonia stimulation. The hyperammonaemic state was present for at least 6 weeks and then spontaneously resolved. No obvious cause (liver disease, portocaval shunts, urea cycle enzyme deficiencies, drug therapy or urinary tract obstruction) could be identified. It is possible that this dog had a variation of transient hyperammonaemic syndrome, described in man and recently in a juvenile Irish wolfhound, that extended into adulthood.

Hyperammonaemic encephalopathy secondary to selective cobalamin deficiency in a juvenile Border collie

An eight-month-old Border collie was presented with anorexia, cachexia, failure to thrive and stupor. Laboratory tests demonstrated a mild anaemia, neutropenia, proteinuria and hyperammonaemia. Serum bile acid concentrations were normal, but an ammonia tolerance test (ATT) was abnormal. The dog responded to symptomatic therapy for hepatoencephalopathy. When a low serum cobalamin (vitamin B_{12}) concentration and methylmalonic aciduria were noted, the dog was given a supplement of parenteral cobalamin. Two weeks later, a repeat ATT was normal. Cobalamin supplementation was continued every two weeks, and all clinical signs, except for proteinuria, resolved despite withdrawing all therapy for hepatoencephalopathy. A presumptive diagnosis of hereditary selective cobalamin malabsorption was made, based on the young age, Border collie breed, low serum cobalamin concentration and methylmalonic aciduria. Although hereditary selective cobalamin malabsorption in Border collies, giant schnauzers, Australian shepherd dogs and beagles has previously been reported in North America, to the authors' knowledge this is the first report of the condition in the UK and the first to document an abnormal ATT in a cobalamin-deficient dog.

I. A. Battersby, U. Giger* and E. J. Hall

Journal of Small Animal Practice (2005) 46, 339–344

This report describes the case of a juvenile Border collie with severe encephalopathy associated with hyperammonaemia and methylmalonic aciduria occurring secondarily to selective Cbl deficiency.

An eight-month-old, neutered male Border collie was referred to the University of Bristol, with a three-month history of progressive anorexia, lethargy and poor weight gain. Pretreatment blood samples taken by the referring veterinarian three weeks earlier had revealed a mild anaemia (creatinine 120 μmol/l, reference range 0 to 105 μmol/l; urea 10·5 mmol/l, reference range 1·7 to 7·4 mmol/l) and neutropenia ($2{\cdot}22 \times 10^9$/l, reference range 3·30 to $12{\cdot}0 \times 10^9$/l). The dog recovered from neutering uneventfully, but a protein-restricted diet (Hill's k/d) was prescribed on the assumption that renal insufficiency was present. The diet change was associated with a transient improvement in the dog's demeanour and appetite. However, the dog had been anorexic for five days before referral.

At presentation (day 1), the puppy was very underweight (8·9 kg, condition score 1/5; expected weight for age and breed 15 to 20 kg) and stuporous. Neuro-ophthalmic examination revealed normal retinas but a sluggish menace response

Hereditary selective cobalamin (Cbl) malabsorption in dogs has previously been

Blood ammonia

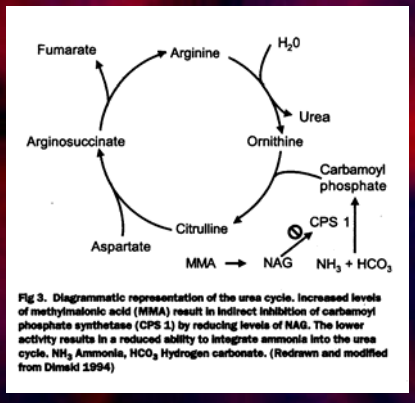
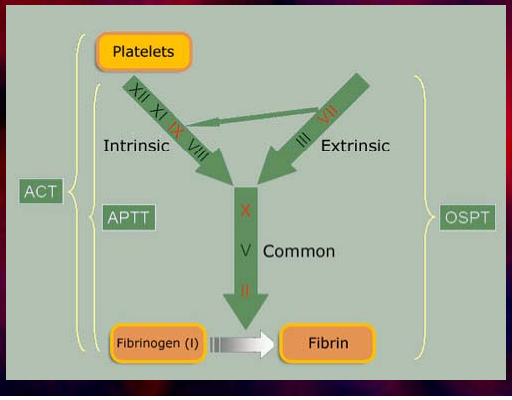


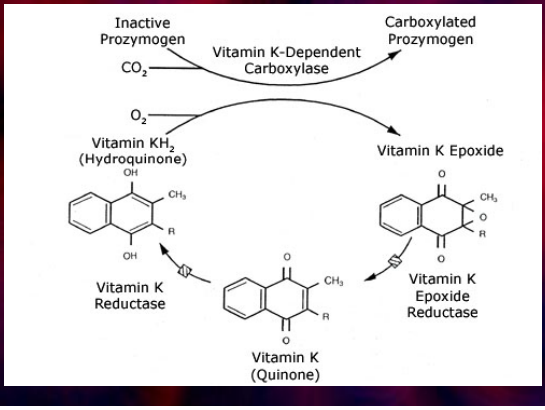
Fig 3. Diagrammatic representation of the urea cycle. Increased levels of methylmalonic acid (MMA) result in indirect inhibition of carbamoyl phosphate synthetase (CPS 1) by reducing levels of NAG. The lower activity results in a reduced ability to integrate ammonia into the urea cycle. NH_3 Ammonia, HCO_3 Hydrogen carbonate. (Redrawn and modified from Dimski 1994)

Ammonia tolerance test

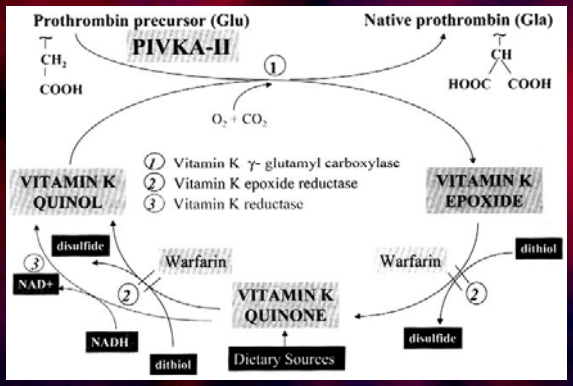
Coagulation profile



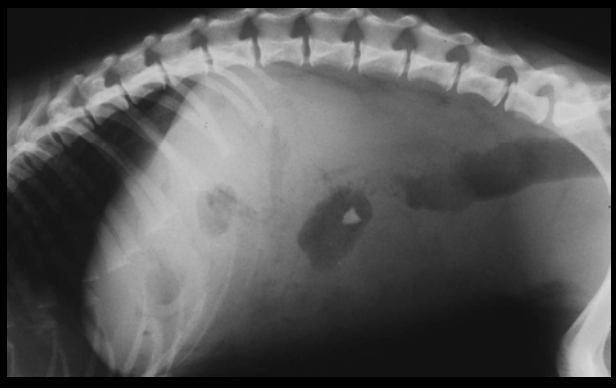
Coagulation profile



Coagulation profile



Imaging



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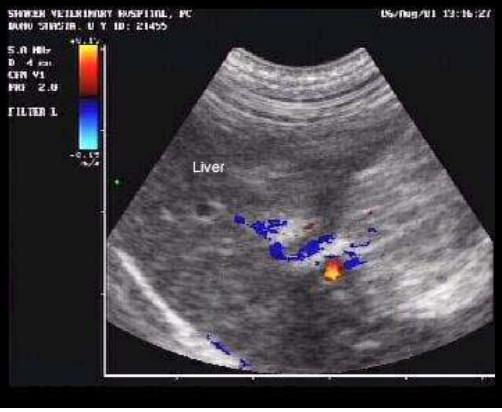

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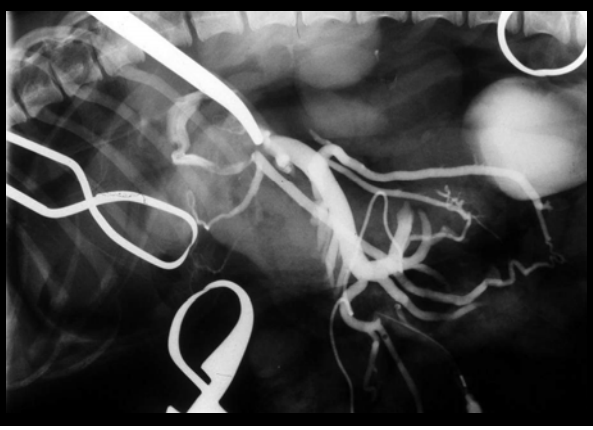

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1: J Vet Intern Med. 1991 Jan-Feb;5(1):23-7. Related Articles, Links

Per rectal portal scintigraphy using 99mtechnetium pertechnetate to diagnose portosystemic shunts in dogs and cats.

Daniel GB, Bright R, Ollis P, Shull R

Department of Urban Practice, College of Veterinary Medicine, University of Tennessee, Knoxville 37901-1071.

Per rectal portal scintigraphy using 99mTechnetium pertechnetate (99mTcO4-) was used to diagnose portosystemic shunts (PSS) before surgical confirmation in seven dogs and two cats. Shunt fractions, representing the percent of portal blood that bypasses the liver, were determined by computer analysis of the scintigraphic images. Animals with portosystemic shunts had a mean preoperative shunt fraction of 84.02% (n = 9). The mean postoperative shunt fraction in four animals was 58.22%. The mean shunt fraction in ten control dogs was 5.00%. Per rectal portal scintigraphy is an innovative, easily performed, inexpensive method to diagnose congenital portosystemic shunts in dogs and cats.

PMID: 1850482 [PubMed - indexed for MEDLINE]

Display Abstract Show 20 Sort by Send to

Imaging

1: Vet Radiol Ultrasound. 2005 Mar-Apr;46(2):153-61. Related Articles, Links

Use of 99mTCO4(-) trans-splenic portal scintigraphy for diagnosis of portosystemic shunts in 28 dogs.

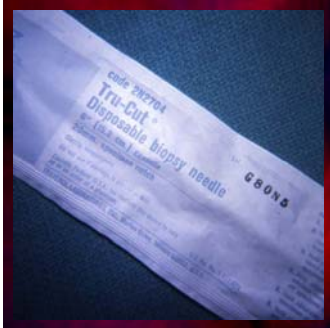
Morandi F, Cole RC, Tobias KM, Berry CR, Avenell J, Daniel GB

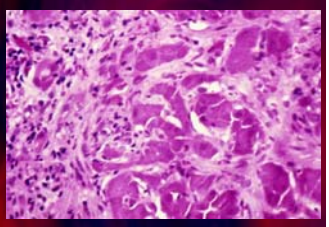
Department of Small Animal Clinical Sciences , Veterinary Teaching Hospital, University of Tennessee, Knoxville, TN 37996-4544, USA. fmorandi@utk.edu

Ultrasound-guided percutaneous trans-splenic portal scintigraphy (TSPS) using 99mTcO4(-) has been used to image the portal venous system in normal dogs. Compared with per-rectal portal scintigraphy, it provides higher count density, consistent nuclear venograms of the splenic and portal vein, and significantly decreased radiation exposures. This paper describes the use of TSPS for the diagnosis of portosystemic shunts in 28 dogs. TSPS was performed injecting 70 +/- 28 MBq of 99mTcO4(-) (mean +/- SD) into the splenic parenchyma with ultrasound guidance. A dynamic acquisition at a frame rate of four frames/s for 5 min was initiated after placement of the needle and approximately 2s prior to injection. All dogs had diagnoses confirmed via exploratory laparotomy or ultrasonographic identification of the shunting vessel(s). Three studies (10.7%) were nondiagnostic because of intraperitoneal rather than intrasplenic injection of the radionuclide. Three pathways were recognized on the scintigraphic images: (1) portoazygos shunts--the 99mTcO4(-) bolus traveled dorsally, running parallel to the spine and entering the heart craniodorsally, (2) single portocaval or splenocaval shunts--the 99mTcO4(-) bolus ran from the area of the portal vein/splenic vein junction in a linear fashion toward the caudal vena cava entering the heart caudally, (3) internal thoracic shunt-the 99mTcO4 bolus traveled ventrally along the thorax and abdomen entering the cranial aspect of the heart. Single and multiple shunts were easily distinguished. There were no distinguishing features between single intra and extrahepatic portocaval shunts.

Publication Types:
- Evaluation Studies

Liver biopsy





- Category of disease
- Degree of severity
- Degree of chronicity

Liver biopsy

J Am Vet Med Assoc. 2004 Jan 1;224(1):75-8. Related Articles, Links

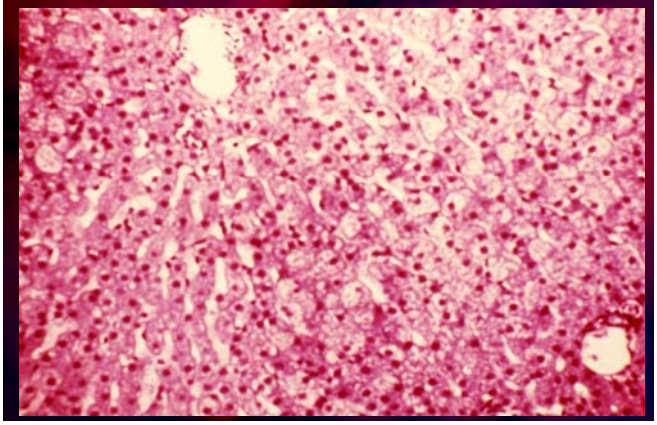
Accuracy of ultrasound-guided fine-needle aspiration of the liver and cytologic findings in dogs and cats: 97 cases (1990-2000).

Wang KY, Panciera DL, Al-Rukibat RK, Radi ZA

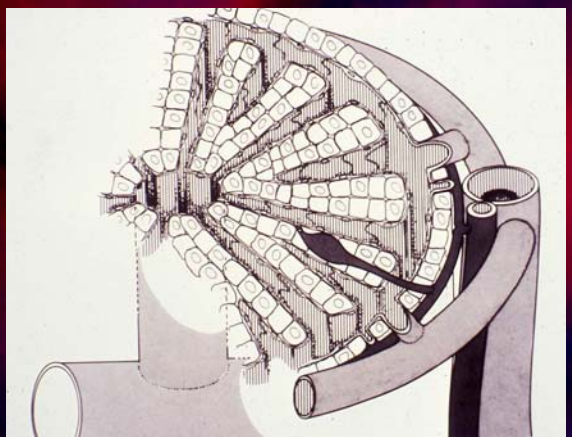
Department of Small Animal Clinical Sciences, Virginia-Maryland Regional College of Veterinary Medicine, Virginia Tech, Blacksburg, VA 24061, USA.

OBJECTIVE: To evaluate the accuracy of ultrasound-guided fine-needle aspiration of the liver and cytologic findings in dogs and cats. DESIGN: Retrospective study. ANIMALS: 56 dogs and 41 cats. PROCEDURE: Medical records of dogs and cats evaluated from 1990 to 2000 by use of cytologic and histopathologic examination of the liver were reviewed. Histologic and cytologic diagnoses were categorized as vacuolar hepatopathy, inflammation, neoplasia, cirrhosis, primary cholestasis, shunt, normal, and other. RESULTS: Overall agreement between the histopathologic diagnosis and cytologic diagnosis was found in 17 of the 56 (30.3%) canine cases and 21 of the 41 (51.2%) feline cases. Vacuolar hepatopathy was the category with the highest percentage of agreement. Vacuolar hepatopathy was identified via cytologic examination in 7 of 11 and 15 of 18 dogs and cats, respectively, in which histopathologic examination revealed that it was the predominant disease process. However, it was also the category that was most commonly misdiagnosed via cytologic examination. Inflammatory disease was accurately identified cytologically in 5 of 20 and 3 of 11 dogs and cats, respectively. CONCLUSIONS AND CLINICAL RELEVANCE: Acknowledging the limitations of cytology and the extent of discrepancies between cytologic and histopathologic findings in dogs and cats will help clinicians make better decisions in diagnosing liver disease.

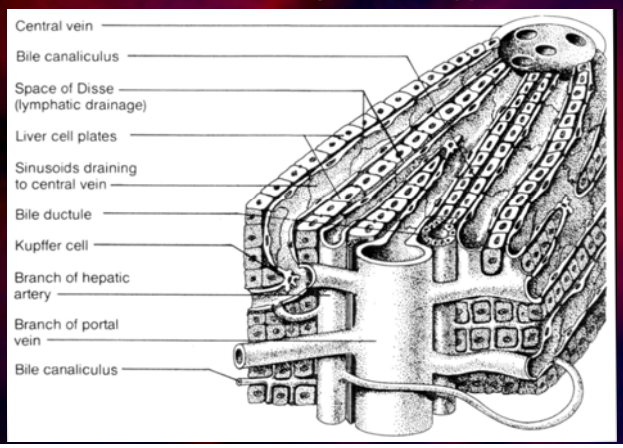
Liver histopathology



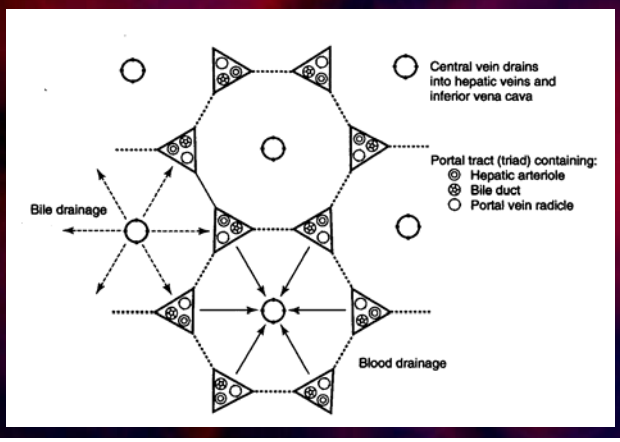
Liver histopathology



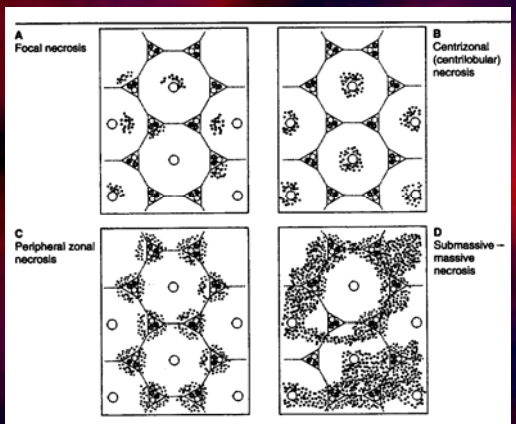
Liver histopathology



Liver histopathology



Liver histopathology



Rupes

A skinny, spaced-out, Yorkshire Terrier puppy
8 month-old, male

Rupes -- history

- Since the age of 4 months, Rupes has failed to thrive
- He is thinner than he should be
- His appetite is mediocre, but he drinks quite well
- 2-3 hours after eating, he often becomes ‘vacant’ and stares into space. Sometimes it is difficult to rouse him.

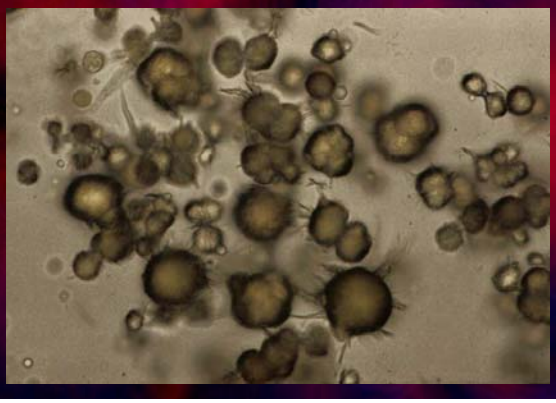
Rupes -- P.E.

- Underweight, body condition score 3 or 4 /9
- Distinctly dull for a puppy of this breed and age
- TPR normal
- No abnormal auscultation or palpation findings. Abdomen feels rather empty

Routine blood work

- Mild, non-regenerative anaemia with slight microcytosis (small RBCs)
- Serum albumin, urea and glucose all slightly low

Routine urine analysis

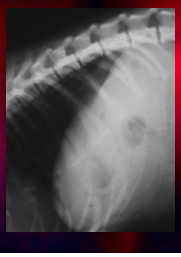


Routine urine analysis

- S.G. 1.013
- Ammonium biurate crystals present in abundance on sediment examination, otherwise normal

Plain abdominal radiographs

- A tiny, 'sliver' of a liver



Abdominal ultrasonography

- Numerous small bladder stones seen. *Why not seen on x-ray?*
- Very small liver
- A single, large, extrahepatic portosystemic shunt with turbulent blood flow was observed

Serum bile acids

- Pre-prandial: slightly raised above normal
- 2 hours post prandial: markedly elevated

Blood ammonia

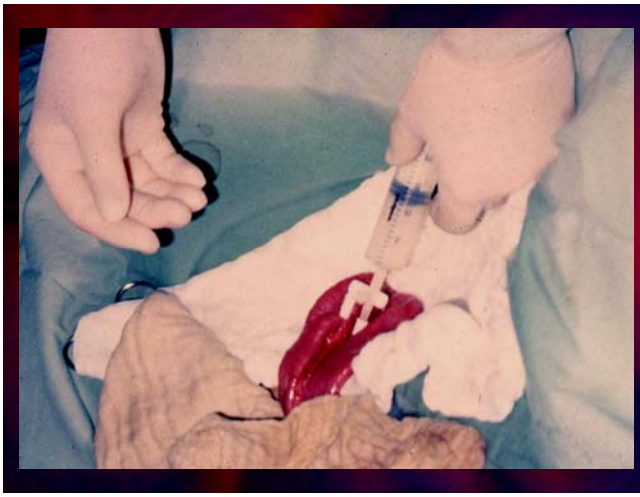
- Abnormally high 4 hours post prandially

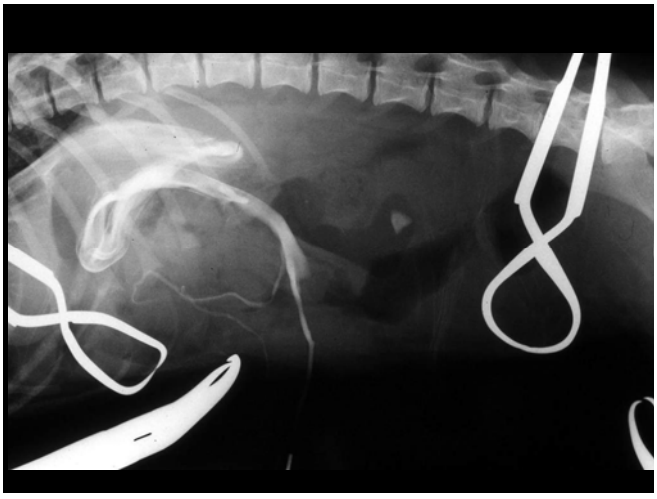
Temporary medical management

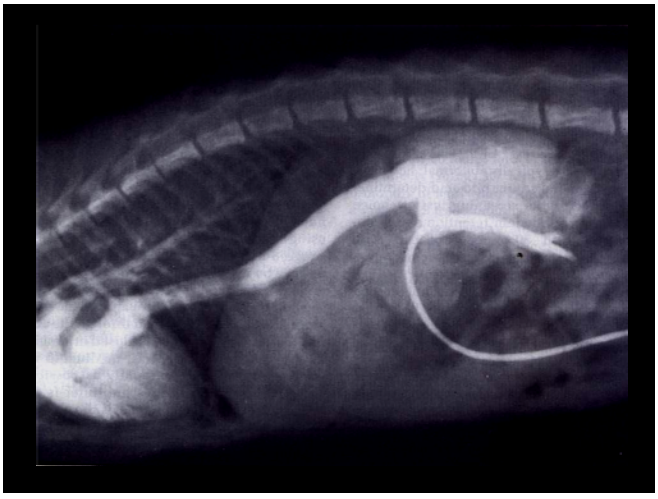
- Rupes was sent home for several days on a low protein diet, an oral antibiotic and lactulose (a laxative)
- The owner reported that Rupes was noticeably brighter and did not become ‘vacant’ 2-3 hours after meals

Case progress

- Rupes was anaesthetized and taken to surgery
- The plan was to carry out a mesenteric portogram (dye study) if a ligatable shunt was not rapidly identified







Case progress

- The abnormal shunting vessel was identified and partially ligated
- Bladders stones were removed at cystotomy
- Rupes was castrated

Vet Surg. 2004 Jan-Feb;33(1):25-31. Related Articles, Links

Full text

Outcomes of cellophane banding for congenital portosystemic shunts in 106 dogs and 5 cats.

Hunt GB, Kummeling A, Tisdall PL, Marchevsky AM, Liptak JM, Youmans KR, Goldsmid SE, Beck JA.

Veterinary Cardiovascular Unit, Faculty of Veterinary Science, University of Sydney, Sydney, Australia. gbhunt@mail.usyd.edu.au

OBJECTIVE : To report outcomes after cellophane banding of single congenital portosystemic shunts in dogs and cats. STUDY DESIGN : Retrospective study of sequential cases. ANIMALS : One hundred and six dogs and five cats. METHODS : Medical records were reviewed for breed, sex, age at surgery, shunt anatomy, results of pre- and postoperative biochemical analysis, development of postligation neurologic dysfunction, portal hypertension or other serious complications, and the owners' perception of their animal's response to surgery. RESULTS : Ninety-five dogs and all 5 cats had extrahepatic shunts. Eleven dogs had intrahepatic shunts. Six dogs (5.5%) died as a result of surgery from portal hypertension (2 dogs), postligation neurologic dysfunction (2), splenic hemorrhage (1) and suspected narcotic overdose (1). Serious complications were more common in dogs with intrahepatic shunts than those with extrahepatic shunts (P=.002). Postligation neurologic dysfunction necessitated treatment in 10 dogs and 1 cat; 8 dogs and the cat survived. Clinical signs attributed to portosystemic shunting resolved or were substantially attenuated in all survivors. Postoperative serum bile acid concentrations or results of ammonia tolerance testing were available for 88 animals; 74 (84%) were normal and 14 (16%) were abnormal. Multiple acquired shunts were documented in two animals. CONCLUSIONS : Cellophane banding is a safe and effective alternative to other methods of attenuation. CLINICAL RELEVANCE : Slow occlusion of portosystemic shunts using a variety of methods is being evaluated world wide. Cellophane banding is a relatively simple procedure with comparable safety and efficacy to previously reported techniques.

Welcome to **Research Instruments NW**. We are a small business that has been operating in the Mid-Willamette Valley in Oregon since the spring of 1999. The primary product that we manufacture and sell is the **Ameroid Constrictor**.

Ameroid Constrictors are used in surgical treatment when a Portosystemic Shunt has been diagnosed. The diagnosis of PSS is becoming more common as veterinarians, owners, and breeders are becoming more aware of the clinical signs.

Ameroid Constrictors are being used for gradual shunt ligation across the United States in many of the veterinary teaching hospitals at universities and in private practices. Internationally, we now provide Ameroid Constrictors to veterinarians in twenty-two countries.

You may order the constrictors directly from us via phone, fax, or email. Please see our Products page for further information about the different sizes available, prices, and options. Use our convenient Order Page to place your order — you may type in your choices to be printed, or print out the form and write in your choices by hand. Please print clearly. The form may then be faxed or mailed.

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Case progress

- Rupes made an uneventful recovery and was much brighter and more active after surgery.
- He gained weight and his condition score improved

Web-based information on PS shunts

- http://www.upei.ca/~cidd/Diseases/cardiovascular diseases/portosystemic shunt.htm
- http://www.malteseonly.com/shunt.html

Google: DOGS portosystemic

Bridget

An 8½-year-old female spayed English Springer Spaniel

Bridget -- Current complaint

Over the past 6 weeks, Bridget's owners have noticed that she has been drinking and urinating much more than usual. She has started to leak urine while lying down asleep. In the last week, she seems to have been having some difficulty seeing in the dark.

Bridget is up to date on her routine vaccinations. There is no history of previous illness or surgery apart from the ovariohysterectomy. She has no known allergies. Her appetite is excellent. There has been no vomiting, diarrhoea, coughing or sneezing.

Bridget -- Physical examination

T 38.3°C, P 90 (strong), R Panting

Bright, alert, well hydrated

Dilated pupils, hyper-reflective tapetal fundi, direct and consensual pupillary light responses intact.

Retinal exam: diffuse retinal atrophy, abnormally small retinal vessels.

Abdomen slightly tense on palpation. No abnormalities on rectal exam

WHY IS A RECTAL EXAM PARTICULARLY IMPORTANT IN A CASE LIKE THIS?

Bridget

Complete blood count

Test	Patient	Reference Range
WBC	6.9	6.1 -17.4 x 10^9/L
Seg	4.899	3.0 -11.5 x 10^9/L
Band	0	0.0 - 1.0 x 10^9/L
Lymph	1.173	1.0 - 4.8 x 10^9/L
Mono	0.483	0.15 -1.35 x 10^9/L
Eos	0.345	0.1 - 1.25 x 10^9/L
Baso	0	Rare
RBC	7.51	5.5 - 8.5 x 10^{12}/L
HGB	185	120 - 180 gm/L
HCT	0.52	0.37 – 0.55 L/L
MCV	67	66 - 77 fl
MCHC	360	310 - 340 gm/L
Plasma Protein	72	50 - 75 gm/L
Platelets	Adequate	145 - 440 x 10^9/L

Bridget

Serum Chemistry Profile

Test	Patient	Reference Range
SODIUM	144	145 - 158 mmol/L
POTASSIUM	3.7	3.6 - 5.5 mmol/L
CHLORIDE	112	105 - 122 mmol/L
TOTAL CO2	19	18 - 30 mmol/L
TOTAL CALCIUM	2.52	2.20 - 2.58 mmol/L
PHOSPHORUS	0.83	0.80 - 1.6 mmol/L
GLUCOSE	3.0	3.9 - 6.1 mmol/L
UREA NITROGEN	4.64	3.6 - 7.1 mmol/L
CREATININE	88.4	50 - 110 µmol/L
TOTAL SERUM PROTEIN	69	50 - 75 g/L
ALBUMIN	38	22 - 35 g/L
ALKP	670	0 - 200 U/L
CREATINE KINASE	80	0 - 460 U/L
AST	78	10 - 50 U/L
ALT	498	0 - 130 U/L
TOTAL BILIRUBIN	6.84	0 - 6.9 µmol/L
CHOLESTEROL	6.32	2.58 - 5.85 mmol/L

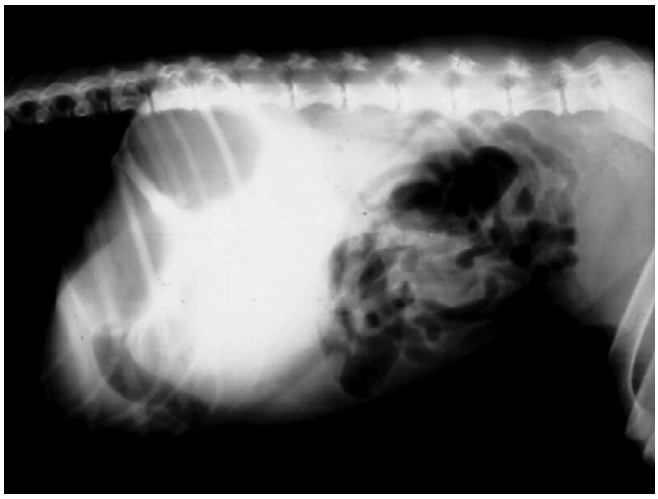
Bridget

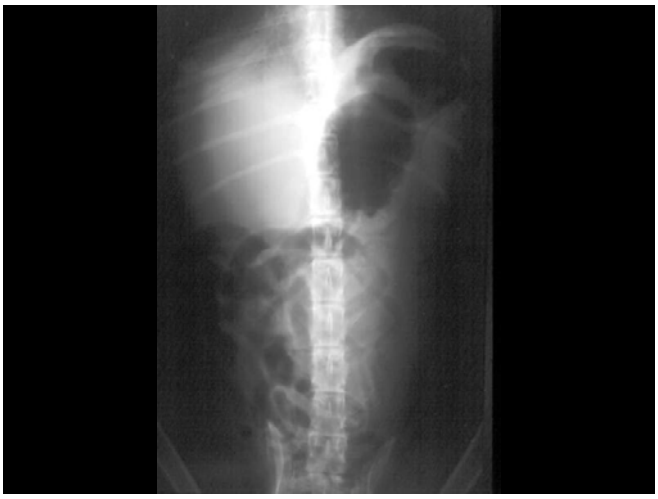
Urine Analysis

Source	cystocentesis
Volume	7 ml
Colour	light yellow
Turbidity	clear
S.G.	1.008
pH	8.0
Protein	negative
Glucose	negative
Ketone	negative
Bilirubin	positive
Hb	2+ (mod)
Urobilinogen	0.2

Sediment Exam

Epithelial cells	few
Crystals	few amorphous urates
RBCs	too numerous to count
WBCs	0 - 3 / hpf
Debris	large amount





You are considering the possibility that Bridget's urinary incontinence is perhaps being exacerbated, or made manifest, by her polydipsia/polyuria. Given the blood test results, renal insufficiency (but <u>not</u> failure), liver disease or Cushing's disease are reasonable differential diagnoses. Diabetes mellitus is ruled out. In fact, the low blood glucose is a little troubling. You plan to repeat a blood glucose measurement on a fresh, appropriately-handled sample; and to carry out further investigations of liver and adrenal function.

Bridget

Repeat blood glucose:

3.1 mmol/L (normal 3.9 - 6.1)

Low Dose Dexamethasone Suppression Test

Resting level	221	(normal 83 - 221 nmol/L)
4 hours post	69	(normal < 27.6 nmol/L)
8 hours post	83	(normal < 27.6 nmol/L)

Bridget

Serum bile acids (pre & post prandial)*

Pre	14.7	(normal <12.25)
Post	53.9	(normal < 36.75)

* *Done by a colleague one day after an episode of bloating. Bridget was brought to the practice out-of-hours because of a distended abdomen. A stomach tube was passed and the distension was relieved easily. The next day, serum bile acids were measured. Several days later, Bridget bloated a second time. Again, a stomach tube was passed easily and the gastric distension was resolved.*

Bridget

Repeat LDDST:

Resting	70 nmol/L	(normal 83 - 221 nmol/L)
4 hour	63 nmol/L	(normal < 27.6 nmol/L)
8 hour	154 nmol/L	(normal < 27.6 nmol/L)

Repeat Abdominal Radiographs

An 8 cm diameter soft tissue mass is present in the cranial, dorsal, right abdomen. It displaces the stomach into an abnormal position, so that the stomach appears to be partially twisted.

Abdominal Ultrasound findings

A mass is present within the caudate lobe of the liver. It is about 10 cm in diameter. It is very close to the caudal vena cava, but does not appear to be invading that vessel. The remainder of the liver is of normal echogenicity, except for one or two hypoechoic nodules, consistent with nodular hyperplasia, or some other infiltrative process.

Diagnosis: Consider neoplastic liver mass, primary or secondary.

Bridget

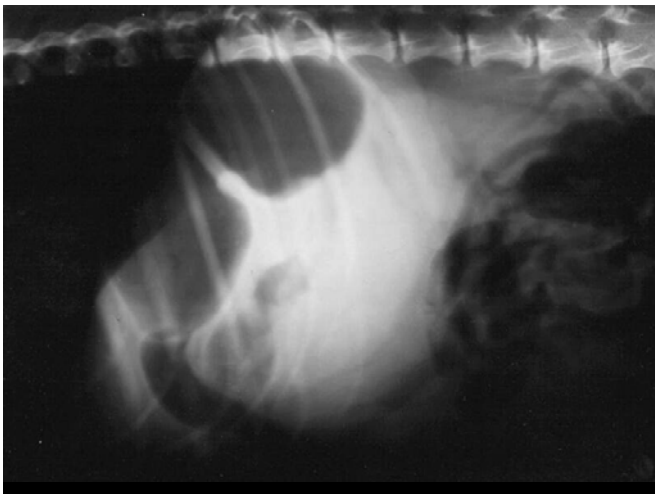
Surgical Pathological Findings

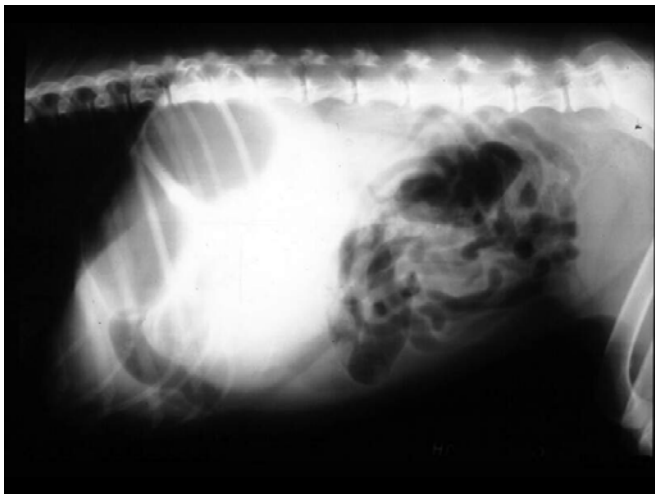
When the abdominal cavity was opened, the stomach was in an abnormal position, displaced by a liver mass. A moderately firm, 10 cm diameter, round purple mass occupied the caudate lobe of the liver. This lobe was resected with some difficulty, since it extended to the pedicle of that lobe, very close to the caudal vena cava. Resection was considered to have been incomplete. The mass was submitted for histopathological examination. The pancreas was palpated: it felt normal. The adrenal glands were inspected. A small mass was found on the right adrenal gland. This was biopsied. The left adrenal gland was normal. A gastropexy was performed. Histologically, the liver mass was reported to be a hepatoma and the adrenal mass was reported as adrenal cortical hyperplasia.

Follow up (2 weeks post op)

Urinary incontinence and polydipsia / polyuria resolved completely within days of surgery.

Glucose	6.1 mmol/L
ALKP	267 U/L
LDDST	normal suppression at 4 and 8 hours.

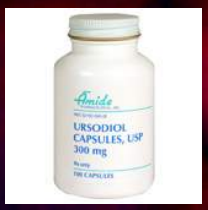


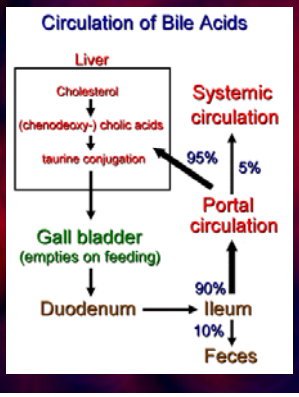


Glucocorticoids

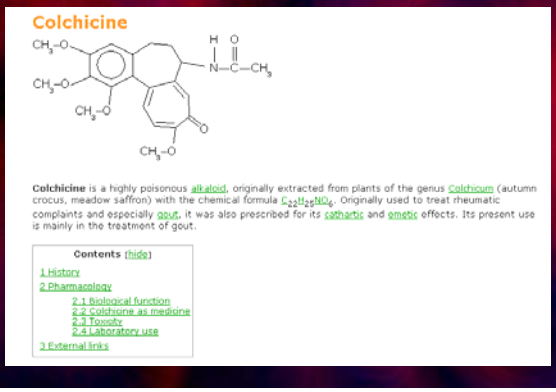
Azathioprine

Ursodiol





Colchicine



Drugs to combat copper accumulation

- D-Penicillamine
- Tetramine (Trientene)
- Zinc gluconate or zinc acetate

Antioxidant drugs

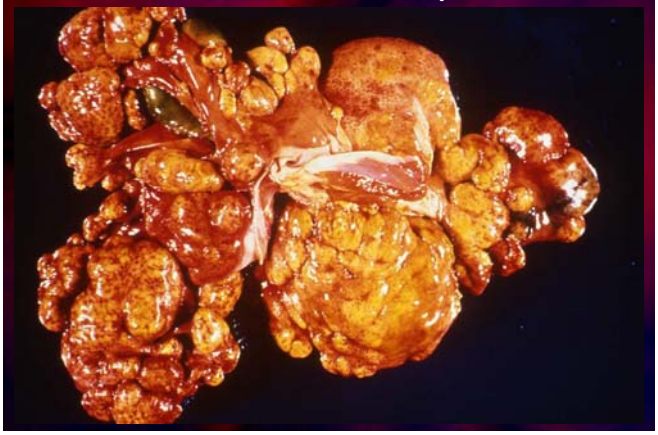
- Silymarin
- s-Adenosyl-L-methionine (SAMe)
- Others

Managing hepatic encephalopathy

- Diet
- Lactulose
- Antibiotic

- Avoid hypokalaemia
- Avoid benzodiazepines

Canine chronic hepatitis

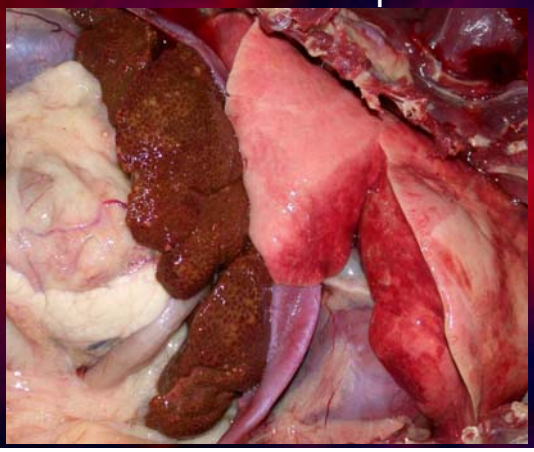


Canine chronic hepatitis

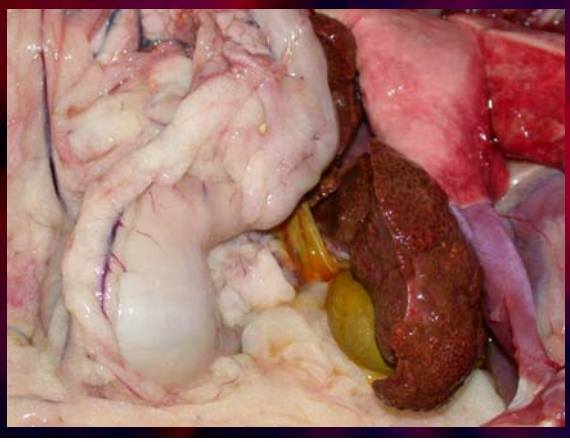
Canine chronic hepatitis

Canine chronic hepatitis

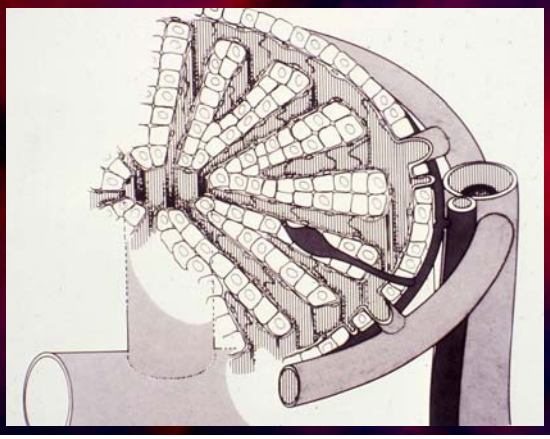
Canine chronic hepatitis



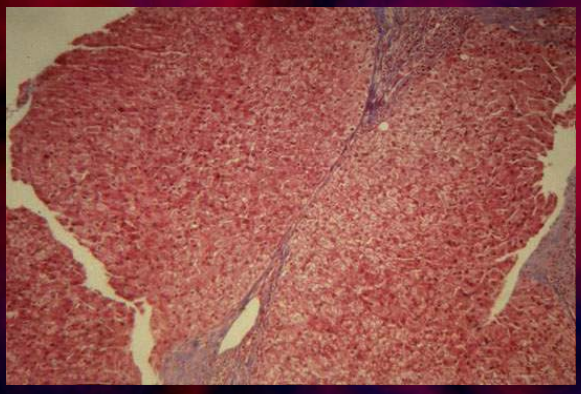
Canine chronic hepatitis



Canine chronic hepatitis



Canine chronic hepatitis



Infectious canine hepatitis

Infectious Canine Hepatitis

- Caused by *Canine adenovirus* type 1 (CAV-1)
- Dogs (esp. puppies), foxes, wolves, coyotes, skunks, bears; but *not* mustelids (e.g., ferrets, stoats)
- Acute hepatitis, respiratory Dz, ocular Dz,
  ± encephalopathy, chronic hepatitis
  ± interstitial nephritis
- *Many* subclinical infections occur

Infectious Canine Hepatitis

- Young, recently-weaned dogs are most susceptible; high mortality ~ 100%
- Most adult dogs recover; ~ 10 to 30% mortality
- However, virus localises in the kidneys of recovered dogs; they continue to shed virus for several months (especially in urine) despite high levels of antibody

CAV-1 can cause :

1. peracute disease with death; *or*
2. acute disease with anorexia, pyrexia then severe abdominal pain (vomiting and diarrhoea) and death; *or*
3. **subclinical or mild infection** – most common

Canine Adenovirus-1 (Infectious canine hepatitis)

Inhalation / Ingestion of the virus

↓

Local replication

(in oropharynx, tonsils, cervical lymph nodes, thoracic duct)

↓

Viraemia

(to eye, liver, kidney and endothelial cells)

↓

Replication

Endothelial cells → **Haemorrhages**

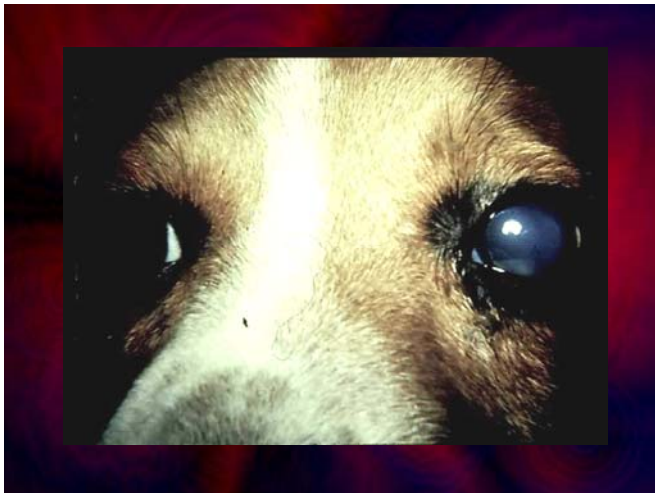
Transmission

Parenchymal cells (**liver, kidney**) → **Necrotic lesions**

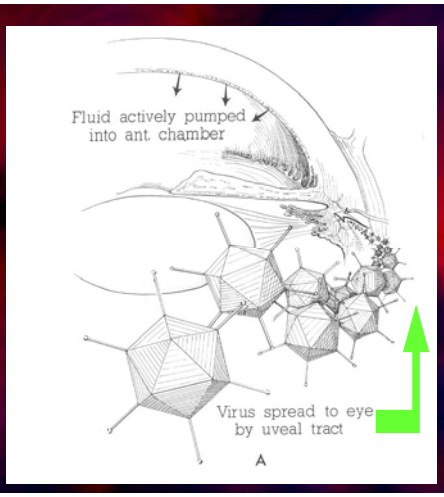
↓

Excreted in faeces, saliva, urine

Tonsils

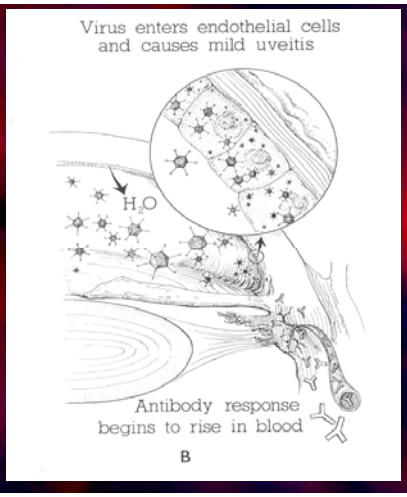


'Blue eye'



Day 4 PI

‘Blue eye’

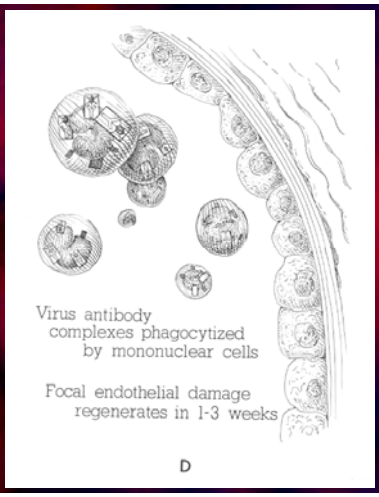


Day 6 PI

‘Blue eye’

Day 7: Ag/Ab complexes (1), complement fixation (2), & leucocyte chemotaxis (3)

‘Blue eye’



Days 8-21: resolving

Confirmation of Diagnosis

- Clinical signs, clinical pathology, gross pathology, histopathology (intranuclear inclusion bodies)
- Rising antibody titres
- Virus isolation, viral antigen in tissues, PCR

Reliability when the disease is so rare?

Control of I.C.H.

- Isolation of infected animals
- Thorough disinfection of infected premises
- Vaccination

Feline cholangitis / cholangiohepatitis

- Suppurative cholangitis / cholangiohepatitis
- Lymphocytic cholangitis

Pathogenesis and outcome of extrahepatic biliary obstruction in cats

J Small Anim Pract 2002 Jun;43(6):247-53

Mayhew PD, Holt DE, McLear RC, Washabau RJ.
Department of Clinical Studies, School of Veterinary Medicine, University of Pennsylvania, Philadelphia 19104-6010, USA.

Extrahepatic biliary obstruction (EHBO) was confirmed at surgery or necropsy in 22 cats. Biliary or pancreatic adenocarcinoma was diagnosed by histopathology in six cats and one cat had an undiagnosed mass in the common bile duct. The remaining 15 cats had at least one of a complex of inflammatory diseases including pancreatitis, cholangiohepatitis, cholelithiasis and cholecystitis. The most common clinical signs were jaundice, anorexia, lethargy, weight loss and vomiting. Hyperbilirubinaemia was present in all cases. Distension of the common bile duct and gall bladder was the most commonly observed finding on abdominal ultrasound. Nineteen cats underwent exploratory laparotomy for biliary decompression and diversion. Mortality in cats with underlying neoplasia was 100 per cent and, in those with non-neoplastic lesions, was 40 per cent. Long-term complications, in those that survived, included recurrence of cholangiohepatitis, chronic weight loss and recurrence of obstruction. Based on these findings, the prognosis for EHBO in cats must be considered guarded.

Feline cholangitis / cholangiohepatitis

- "Triaditis"
  - Cholangitis / cholangiohepatitis
  - Pancreatitis
  - Inflammatory bowel disease

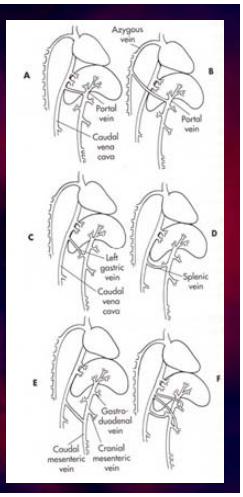
Metabolic and toxic hepatopathies

- Canine vacuolar hepatopathy
- Feline hepatic lipidosis
- Hepatotoxicities
  - Anticonvulsants (e.g., phenobarbital)
  - Antimicrobials (e.g., trimethoprim-sulpha, ketoconazole)
  - Diazepam
  - Methimazole / carbimazole
  - NSAIDs (carprofen, paracetamol)

Feline hepatic lipidosis

Portosystemic vascular anomalies

- Intrahepatic
- Extrahepatic



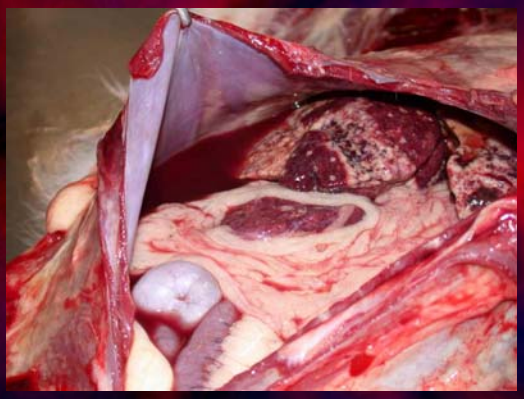
Liver neoplasia

Feline bile duct carcinoma with mesenteric infiltration

Liver neoplasia

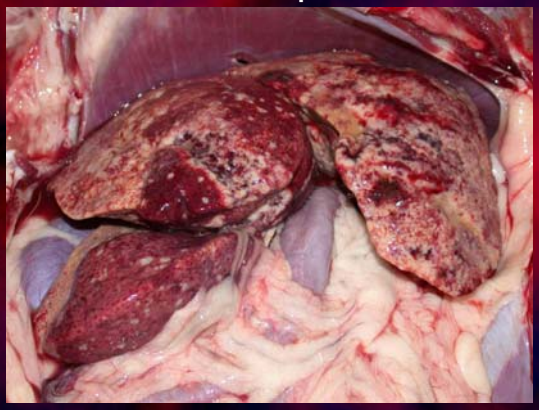
Canine bile duct carcinoma

Liver neoplasia



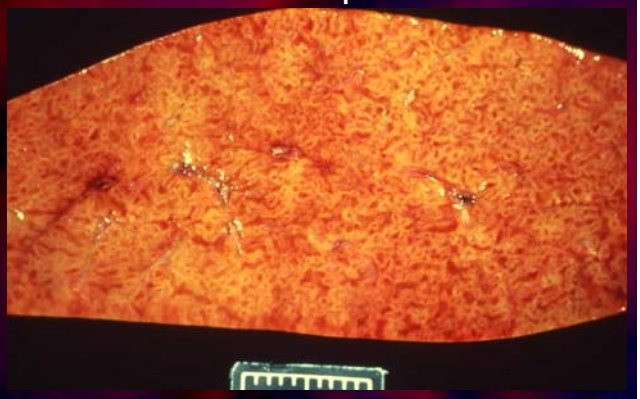
Canine bile duct carcinoma

Liver neoplasia



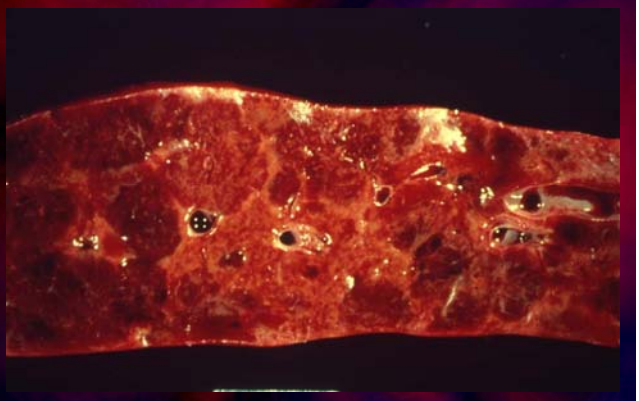
Canine bile duct carcinoma

Liver neoplasia



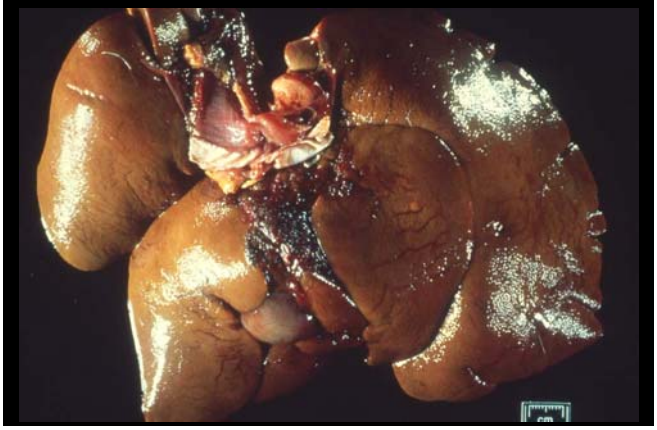
Cat erythremic myelosis

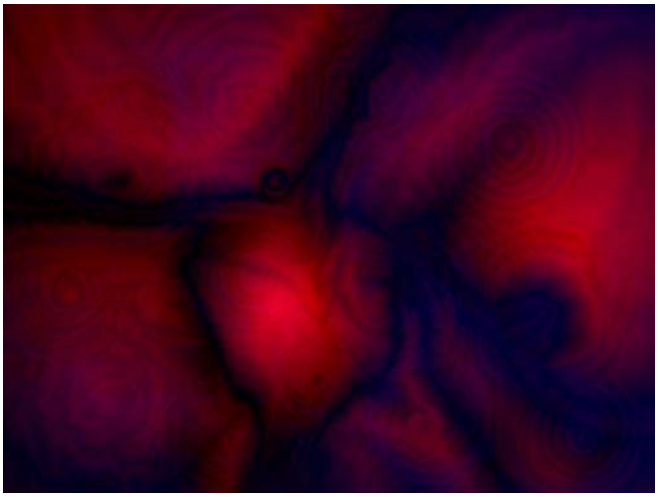
Liver neoplasia



Dog haemangiosarcoma

‘Fractured’ liver (post trauma)

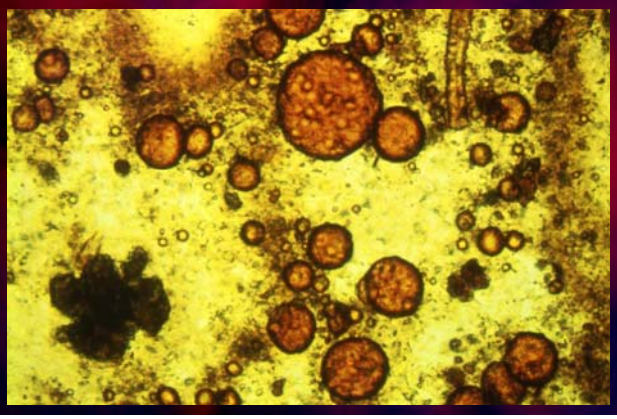




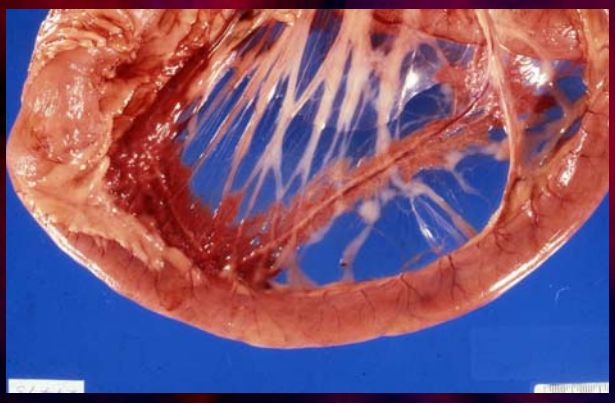
Exocrine pancreatic insufficiency

Exocrine pancreatic insufficiency

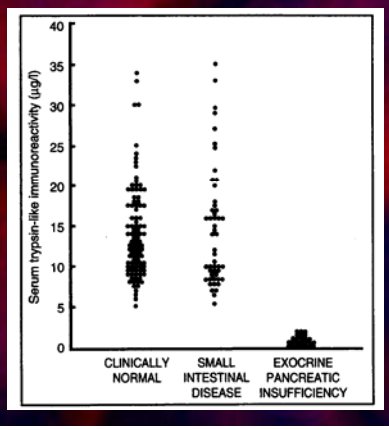
Exocrine pancreatic insufficiency



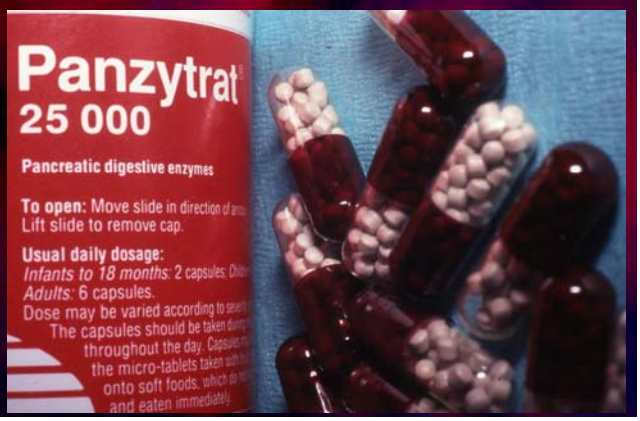
Exocrine pancreatic insufficiency



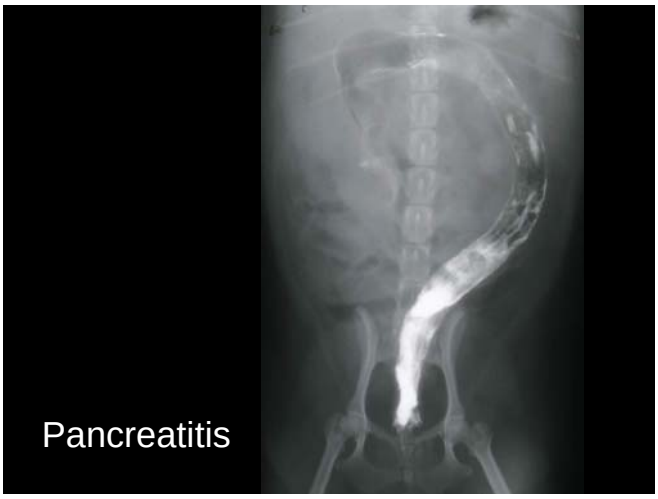
Exocrine pancreatic insufficiency



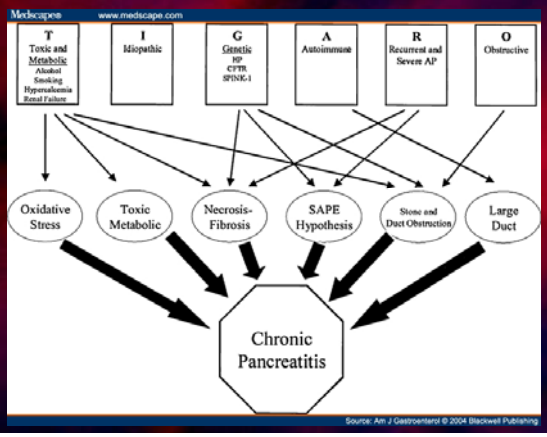
Exocrine pancreatic insufficiency



Pancreatitis



Ideas concerning pathogenesis



Management