10th NVF INDIAN VETopia 2025

26th to 28th February, 2025

Venue:

Hotel Intercontinental,

Jaipur, Rajasthan

Prime Sponsor

drools
Feed Real. Feed Clean.

VET EXPERT · SAVAVET Shaping the Future · TACO · ROYAL CANIN INCREDIBLE IN EVERY DETAIL · ENAVANT PETCARE · SKY EC

ORGANISER

NVF VETERINARY FOUNDATION

Regd. Office: 5/39, Old Rajinder Nagar, New Delhi-110060
E-mail: nvfworld@gmail.com • Website: www.nvf.world

10th NVF INDIAN VETopia 2025

26th to 28th February, 2025

at Hotel Intercontinental, Jaipur, Rajasthan

SCHEDULE

TIME	DAY 1 Wedneday 26-02-2025	
11.30 am - 1.00 pm	Registration Intercontinental Hotel, Jaipur/ check in	
1.00 pm - 2.30 pm	Lunch	
2.30 pm - 2.25 pm	Inauguration Ceremony	
2.45 pm - 3.30 pm	Abdominal anatomy - emphasis on surgical approaches	Prof. Dr. Mohan Kumar
3.30 pm - 4.15 pm	Gastrointestinal surgery - a review of common surgical gastrointestinal diseases and their surgical treatment	Prof. Dr. Chad Schmiedt
4.15 pm - 5.00 pm	Acute Abdomen in Cat	Dr Philip Judge
5.00 pm - 5.15 pm	Tea & Stall visit	
5.30 pm - 6.15 pm	Fluid Therapy in Cat	
6.15 pm - 6.45 pm	Is Laser Therapy Useful In Veterinary Practice	Dr. S.Yathiraj
7.00 pm onwards	Award Ceremony followed by Gala Dinner and cocktails	

TIME	DAY 2 Thursday 27-02-2025	
7.15 am - 8.00 am	CKD Current Diagnostic Approach (ENAVANT)	Dr. S.Yathiraj
8.00 am - 8.45 am	CKD What's New in the Management (ENAVANT)	Dr. K. G. Umesh
9.00 am - 9.45 am	Abdominal anatomy – emphasis on surgical approaches	Prof. Dr. Mohan Kumar
9.45 am - 10.30 am	Gastric dilation volvulus - a discussion of the condition in dogs and a review of pre-operative stabilization, operative management, and post-operative care. This will including gastropexy techniques and a discussion on prophylactic gastropexy.	Prof. Dr. Chad Schmiedt
10.45 am - 11.15 am	Stall Visits (Tea/ Coffee)	
11.15 pm - 12.00 noon	Hemoabdomen and splenectomy - a discussion of perioperative management of dogs with nontraumatic hemoabdomen most commonly from splenic neoplasia. This will include techniques for splenectomy.	Prof. Dr. Chad Schmiedt
12.00 noon - 12:45 pm	Surgery of the urinary tract – Including basic upper and lower urinary tract surgeries including nephrectomy, renal biopsy, cystotomy, basic ureteral surgery	**Prof. Dr. Chad Schmiedt**
12.45 pm - 1.30 pm	Laser Therapy in Dog - Case Presentations	Dr. S.Yathiraj
1.45 pm - 2.30 pm	Lunch Break	
3.00 pm - 3.45 pm	Anatomy and Physiology of the Kidney (Urinary System)	Prof. Dr. Mohan Kumar
3.45 pm - 4.00 pm	Surgical approach to treatment of chylothorax	Prof. Dr. Chad Schmiedt
4.30 pm - 5.00 pm	Stall Visits Tea/Coffee	
5.00 pm - 5.45 pm	Approach to a Dyspnoic Cat	Dr Philip Judge
5.45 pm - 6.30 pm	Update on Management of Respiratory Infections in Cats	Dr Philip Judge
7.30 pm Onwards	Gala Dinner cocktails	

TIME	DAY 3 Friday 28-02-2025	
9.00 am - 9.45 am	Anatomy of the thoracic cavity - Heart and circulatory system	Prof. Dr. Mohan Kumar
9.45 am - 10.30 pm	Approaches to the thorax - a discussion of median sternotomy and intercostal thoracotomy for thoracic exploration, pericardiectomy, and lung lobectomy.	Prof. Dr. Chad Schmiedt
10.30 am - 11.00 am	Stall Visits Tea / coffee	
11.15 am - 12.00 noon	Cardiac Arrhythmia in Cats	Dr Philip Judge
12.00 noon - 12.45 pm	Anethesia and analgesia in an emergency Cat patients	Dr Philip Judge
12.45 pm - 1.30 pm	Sepsis and Septic shock in Cat	Dr. Philip Judge
1.30 pm - 2.15 pm	LUNCH	
2.30 pm - 3.15 pm	Anatomy and physiology of the lungs and respiratory system	Prof. Dr. Mohan Kumar
3.15 pm - 4.00 pm	Management and surgical approach to diaphragmatic herni arepair including congenital and traumatic diaphragmatic hernias.	Prof. Dr. Chad Schmiedt
4.00 pm Onwards	TEA & curtains & closing ceremony	

NVF BOARD MEMBERS

Dr. K.G. Umesh
President
Board Member

Dr. Lakshmi Srinivasan
Board Member

Dr. S. Ayyappan
Board Member

Dr. Yathiraj
Board Member

Dr. Jasjeet Josan
Board Member

Dr. Rishi Sood
Board Member

Dr. Praydut Sarmah
Board Member

NVF Scientific Advisory Committee (NVF-SAC)

Dr. Umesh Karkare
Vet. Surgeon, Mumbai
Chairman, Scientific Advisory

Dr. G N Purohit
Professor, Dept. of Veterinary Gynaecology And Obstretics, RUVAS, Rajasthan

Dr. Nagarajan L
Professor, TANUVAS, Presently at University of The West Indies, St. Augustine

Dr. Nambi A P
Professor and Head, Dept. of Veterinary Preventive Medicine, TANUVAS, Chennai

Dr. T K Gahlot
Professor and Head, Dept of Veterinary Surgery & Radiology, RUVAS, Rajasthan

Dr. Naresk K Rakha
Professor and Dean-PGS, LUVAS, Hisar, Haryana

Dr. Ramani
Professor,Dept. of Veterinary Surgery And Radiology, TANUVAS, Chennai

Dr. Jeyaraja K.
Professor,Dept. of Veterinary Clinical Medicine, Madras Veterinary College, Chennai

Dr. P Sridevi
Professor,Dept. of Clinics, TANUVAS, Chennai

Dr. S P Tyagi
Professor and Head, Dept of Veterinary Surgery & Radiology, CSKHPKV, Palampur

NVF CE Moderators and Advisory Committee (NVF-CMAC)

Dr. Shiwani Tandal
Vet. Surgeon, Mumbai

Dr. Sunita Nauriyal
Vet. Surgeon, New Delhi

NVF Animal Welfare and Ethics Advisory Committee (NVF-AWEAC)

Maj.Gen. R.M. Kharb AVSM
Chairman, Animal Welfare Board of India

Dr. S. Chinny Krishna
Board Member, World Society For the Protection of Animals (WSPA)

NVF Advisory Committee (NVF-AC)

Dr. Amardeep Singh
Vet. Surgeon, Uttarakhand

Dr. Amarnath M.
Vet. Surgeon, Salem

Dr. Amber Mishra
Vet. Surgeon, UP

Dr. Sanjib Kakoti
Vet. Surgeon, New Delhi

Dr. Annie Varghese
Vet. Surgeon, Trivandrum

Dr. Praveen Kumar
Vet. Surgeon, Hyderabad

Dr. S.P. Singh
Vet. Surgeon, Shimla

Dr. Vinay Gorhe
Vet. Surgeon, Pune

Dr. Parampal Singh
Vet. Surgeon,Bathinda

Dr. Bhanu Khajuria
Vet. Surgeon, Jammu

Dr. Aparajita
Vet. Surgeon, Kolkata

Dr. Manmohan Sharma
Sr. Vet. Surgeon, New Delhi

Dr. Neelam Singh Josan
Vet. Surgeon, Gurgaon

Dr. Kunal Sharma
Vet. Surgeon, New Delhi

Dr. Manjeet Singh
Vet. Surgeon, Punjab

Dr. Sanmant Jain
Vet. Surgeon, Ghaziabad

drools VETERINARY HEALTH

drools
Feed Real. Feed Clean.

Reduce occurrence of CKD in Cats with TelPet™

℞ Telmisartan Oral Suspension

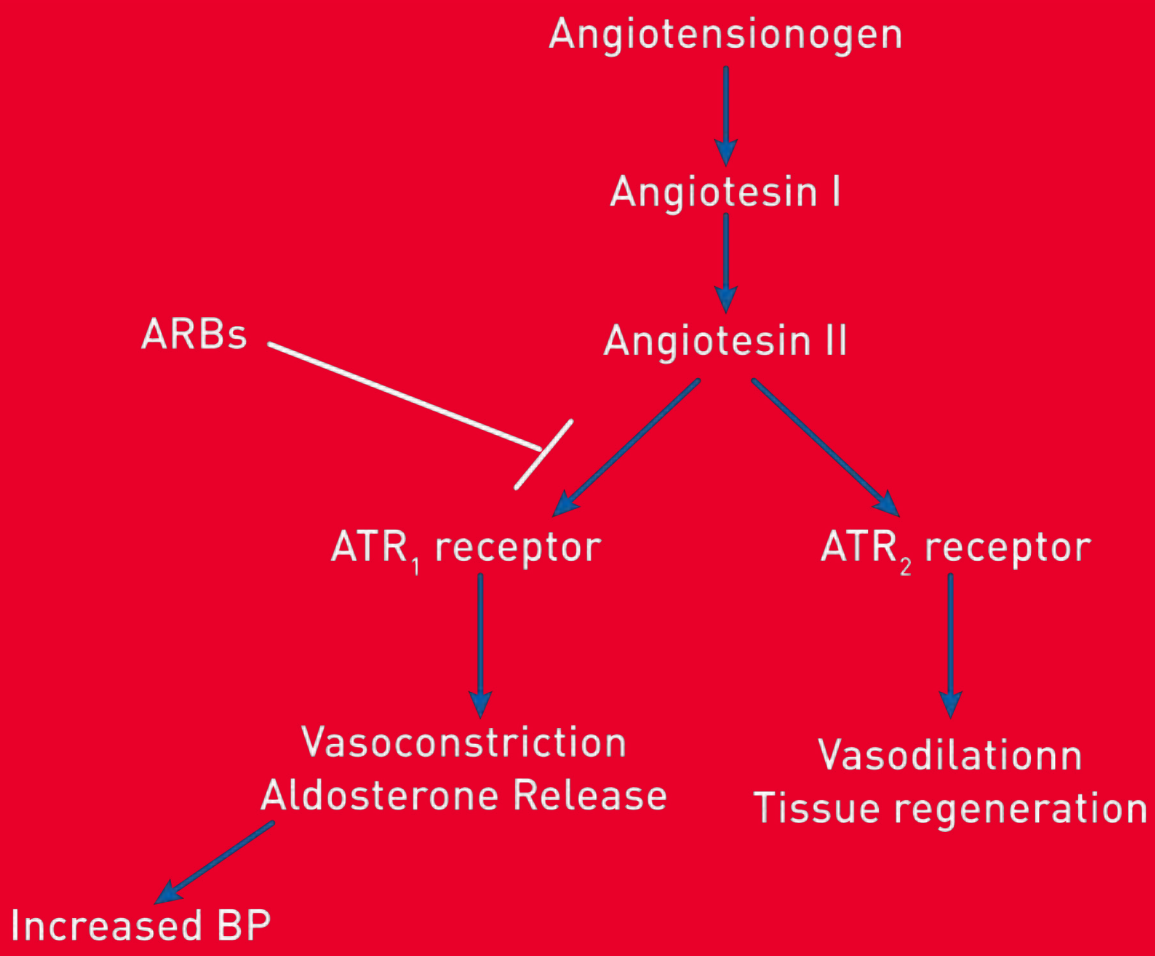
"Caring for Pets, One Dose at a Time."

1st time in India

*1] Telmisartan significantly decreased SABP [Systemic Arterial Blood Pressure] to a clinically relevant extent and was well tolerated in hypertensive cats . ACVIM 2019.

*2] Telmisartan solution (PO) was effective in reducing SABP in hypertensive cats with SABP ^ 160mmHg and ^ 200mm Hg and was persistent over time . ACVIM 2018

PROVEN TREATMENT FOR PROTEINURIA AND HYPERTENSION IN BOTH CATS AND DOGS



MOA:-

Kidneys produce Renin which convert Angiotensinogen to Angiotensin I (Ang I) , With the help of Angiotensin Converting Enzyme - it converts from Ang I Angiotensin II (Ang II) - Angiotensin II then binds to the AT1 Receptor which which causes vasoconstriction, sodium reabsorption, and increased cardiac contractility. This leads to increased blood pressure.

ARBs block the AT1 receptors, preventing Ang II from binding and constricting blood vessels. This allows blood vessels to relax and stay open, which lowers blood pressure.

Dosage:-

For Cats

- For Systemic Hypertension

  To be given at 1.5mg/kg PO BID for 14 days, Followed by 2 mg/kg PO OID

- For Proteinuria associated with Chronic Kidney Disease

  To be given at 1 mg/kg PO OID

For Dogs

- For Management of Proteinuria for dogs with Glomerular Disease

  Start at 1 mg/kg PO OID , and can be increased by 0.5mg/kg. Do not exceed a dose of 2 mg/kg PO OID

Drools Pet Food Private Limited, 436/2, IB Corporate House, Village Indamara, Post Pendri, Rajnandgaon, Chhattisgarh - 491441, India. W: www.drools.com, E: veterinaryhealth@drools.com

SCAN TO KNOW MORE

SIGNATURE

Healthier Pets Happier you !

GRAINZERO Complete Nutrition | FREEDOM 100% NATURAL FOOD FOR ALL PETS

For a lifelong healthy pet

100% ORGANIC PREMIUM DOG FOOD

FORMULATED IN USA · HYPOALLERGENIC

DEEPLY SATISFYING, PROUDLY TANGIBLE!

Natural Antioxidants | Healthy Digestion | Skin & Shiny Coat | Joint Health

For orders and enquiries call:

Delhi - Haider Mehdi +91 98712 66227 | Punjab - Paras Saini +91 96800 07783 | Mumbai - Mohammad Akram Shaikh +91 93276 88078
Bangalore - Krishnaa Kurugod +91 98863 21579 | Chennai - V. Vigneshwaran +91 73731 23420

wecare@signaturepetfoods.com | signaturepetfoods | signaturepetfoods

www.signaturepetfoods.com | Toll-Free: 1800-102-5962

VET EXPERT
BASED ON EVIDENCE

vet planet
ANIMALS' WELFARE IS OUR PASSION

UNLEASH THE POWER OF EVIDENCE-BASED SUPPLEMENTS

DERMATOLOGY

VetoSkin Ultra
- to multidirectionally support the condition of hypersensitive and atopic skin

VetoSkin
- to manifest with a dry and mat coat, hair loss, and flaking skin

NEURO SUPPORT

Neuro Support
- to support the optimal functions of nervous system

KalmVet
- to reduce stress and anxiety. The product has a relaxing effect

DIGESTION

BioProtect
- to optimize the proper functioning of the gastrointestinal microflora

LIVER

Hepatiale Forte Advanced

- to support liver function in cases of failure or dysfunctions

JOINTS

ArthroVet Collagen

- to maintain proper function of articular cartilage and joints dysfunctions

ArthroVet
- to maintain proper function of joint cartilage and joints

LOWER URINARY TRACT

UrinoVet Dog
- to support the optimal functions of the urinary system

LOWER URINARY TRACT

UrinoVet Cat
- to support the optimal functions of the urinary system

VITAMINS & MINERALS

VetAminex
- to balance the daily food intake and meet increased food demands

HEART

CardioVet
- to supprt proper function of the heart and the cardiovascular systen

IMMUNITY

VetoMune
- to support non-specific immunity mechanism

KIDNEYS

RenalVet
- to Support proper renal function

Imported and Marketed By:

Vet Planet India Private Limited

Ph: 9711140505, 7827836303

ROYAL CANIN®

INCREDIBLE IN EVERY DETAIL

MOST RECOMMENDED BY VETERINARIANS

VETERINARY DIET

TACO
The Animal Care Organization

ANIL AGARWAL FOUNDATION

Launched in April'22, TACO aims to establish best in class facilities for animals in need of care, support & shelter, while upskilling professionals through academic & training programmes.

VISION

A compassionate India where every animal lives with dignity & respect.

MISSION

To establish a sustainable & scalable ecosystem for the wellbeing of animals based on the principles of One Health.

FOCUS AREAS

SHELTER

HOSPITAL

ACADEMY

WILDLIFE CONSERVATION

DISASTER RELIEF EFFORTS

SECTORAL ENGAGEMENT

ANIL AGARWAL FOUNDATION

Core - 6, 3rd Floor, Scope Complex, 7, Lodhi Road, New Delhi - 110003

Email: taco@vedanta.co.in

SAVAVET
Shaping the Future

Healthy skin strengthens bonds.

CHEVIK

4% Chlorhexidine gluconate, TrisEDTA

Shampoo **Spray**

Skin infections requires effective and gentle solutions

- Non-irritating to ulcerated or abraded skin
- Supports healthy skin for animals with conditions responsive to chlorhexidine
- 4% chlorhexidine is active against skin pathogens
- Has residual activity
- **Triz-EDTA** has antimicrobial and antibiotic potentiating activity

Spray has residual activity upto 10 days

SAVAVET
Shaping the Future

SAVA HEALTHCARE LIMITED
SAVA House, Off New Airport Road, Viman Nagar, Pune - 411 014, INDIA.
Tel: +91 20 3051 6100 | Email: companionanimal@savaglobal.com | Web.: www.savavet.com
For detailed information, reach us on Customer Care: +91 976 444 3740

ENAVANT PETCARE

Patented in India, Europe, Canada and Eurasia

K9PEA

FIRST TIME IN INDIA

Palmitoylethanolamide (PEA) 300 mg, Daidzein 50 mg, Genistein 4 mg Tablets

For Chronic Pain, Neuropathic Pain & Dermatitis

FIRST TIME IN INDIA

LAFK9

Lactoferrin 100 mg and Disodium Guanosine 5-Monophosphate 10 mg Tablets

For Bone Remodelling

BOULK9

Saccharomyces Boulardii CNCM I-1079 Capsules 250 mg (5 Billion CFU)

For Gut Health

ENAVANT PETCARE

TUMK9

Streptococcus thermophilus 30 billion, Bifidobacterium longum 30 billion, Lactobacillus acidophilus 30 billion Capsules

90BN CFU PROBIOTIC FOR CKD

FIRST TIME IN INDIA

GUT DIALYSIS REDEFINED

- Simplified Dosing
- Superior Efficacy
- Convenience to owner
- Removes the Uremic Toxins through Gut Dialysis

The Only Brand That Maintains Cold Chain Throughout Supply Chain

Manufacturer > Distribution Branch > Veterinary Hospital/Clinic > Pet Owner

Dr. P.S. Mohan Kumar
Department of Biomedical Sciences
Josiah Meigs Distinguished
Teaching Professor

Biography

Research Interests

Neuroendocrinology Reproductive Aging, Stress Axis and Metabolic Function Neuroendocrine-Immune Interactions Prenatal Programming and its Neuroendocrine Consequences Neuroendocrine Effects of Exposure to Environmentally Relevant Chemicals

Educational Background

BVSC (1988)- Madras Veterinary College, India

PhD (1993) Neuroendocrinology, Kansas State University, Manhattan, KS

Postdoctoral (1994-1997) Neuroendocrinology, Department of Molecular & Integrative Physiology, University of Kansas Medical Center, Kansas City, KS

Clinical Anatomy Lectures 1-6

Puliyur S. MohanKumar
Josiah Meigs Distinguished Professor
College of Veterinary Medicine
University of Georgia, Athens, GA 30602, USA

Importance of anatomy in clinical practice – Lecture 1

Puliyur S. MohanKumar, BVSc, PhD
Josiah Meigs Distinguished Professor
College of Veterinary Medicine
University of Georgia, Athens, GA 30602, USA

Canine Abdominal Anatomy: Implications for surgical procedures – Abdominal wall, peritoneum and omenta (Lecture 2)

Puliyur S. MohanKumar, BVSc, PhD
Josiah Meigs Distinguished Professor
College of Veterinary Medicine
University of Georgia, Athens, GA 30602, USA

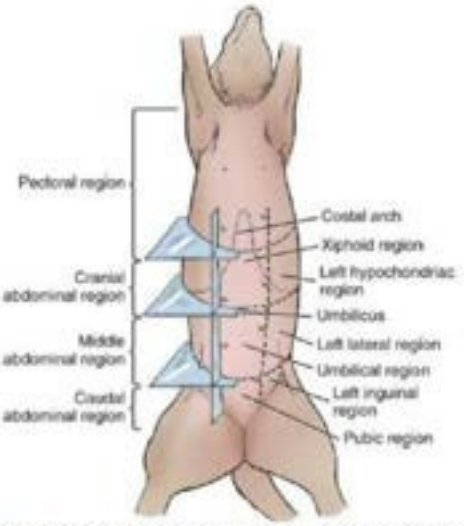
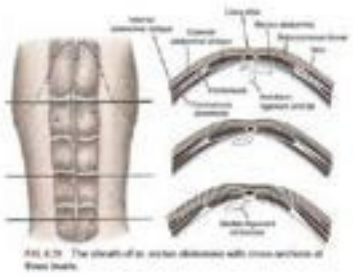


FIG. 7.33 Regions of the abdomen as determined by sagittal and transverse planes.

Abdominal wall

- Muscles
  - Ventrolateral group – 4 muscles
  - 3 broadsheets superimposed on each other with different fiber orientation
  - An aponeurotic tendon inserting upon the midline – ***LINEA ALBA***
    - ***External abdominal oblique (EAO)*** – *arises from lateral ribs and lumbar fascia – caudoventral direction to its insertion on* ***linea alba*** *and pelvic brim.*
    - ***Internal abdominal oblique (IAO)*** – *arises from tuber coxae (and surrounding fascia and lumbar transverse processes) – passes cranioventral to last rib and* ***linea alba.***
    - ***Transverse abdominal*** – *arises from inner surfaces of last ribs and transverse processes of lumbar vertebrae - inserts upon* ***linea alba*** *- does not extend beyond the caudal part of tuber coxae – the caudal part remains uncovered dorsally*
    - ***Rectus abdominus*** – *arises from ventral ribs and sternum and inserts on the* ***pelvic brim*** *and* ***prepubic tendon***

Dorsolateral group (Sublumbar): Psoas Minor, Iliopsoas (psoas major and iliacus), Quadratus lumborum



From Miller and Evans' Anatomy of the Dog

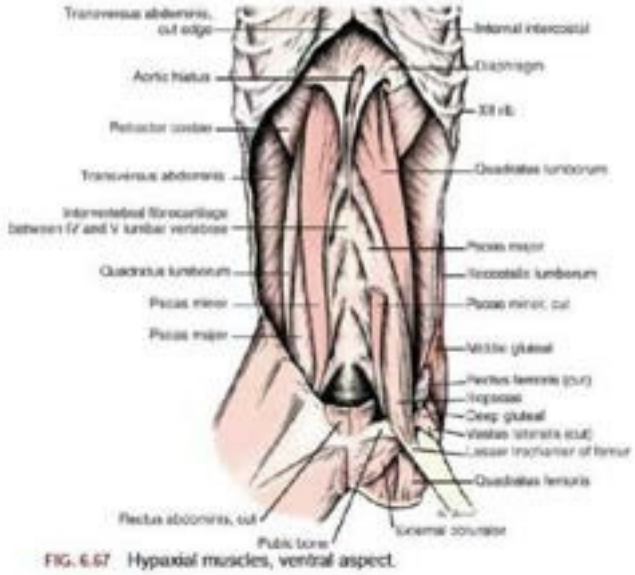


FIG. 6.67 Hypaxial muscles, ventral aspect.

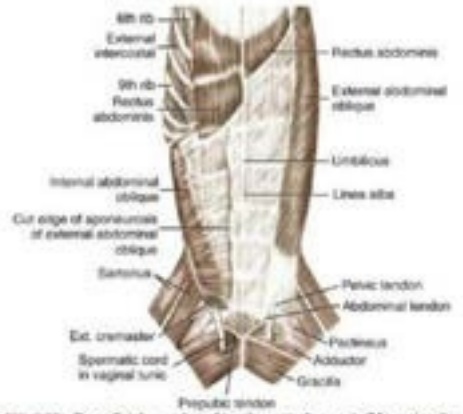
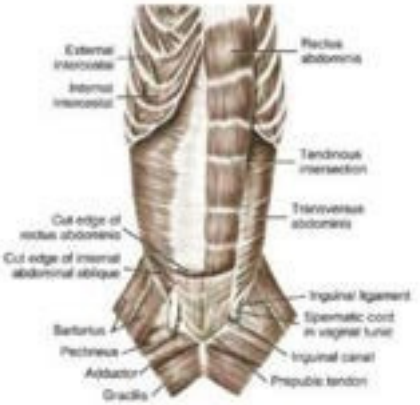


FIG. 6.37 Superficial muscles of trunk, ventral aspect. (M. pectoralis profundus removed.)

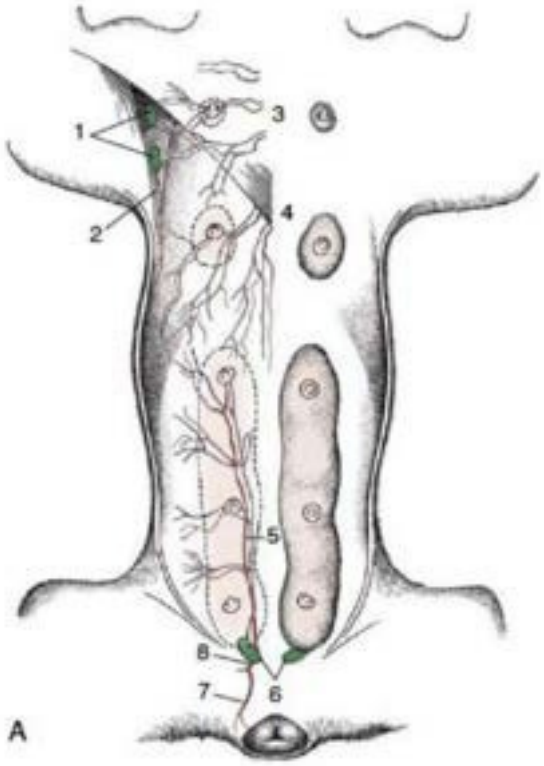


Interactive exercises

Blood supply to abdominal wall

- Segmental lumbar arteries from aorta
- Cranial abdominal a. from Phrenicoabdominal
- Deep circumflex a. – paralumbar fossa area
- Cranial superficial epigastric a.
- Caudal superficial epigastric a.

The superficial epigastric arteries are well developed in lactating animals



From Textbook of Veterinary Anatomy, Dyce, Sack and Wensing

Interactive exercises

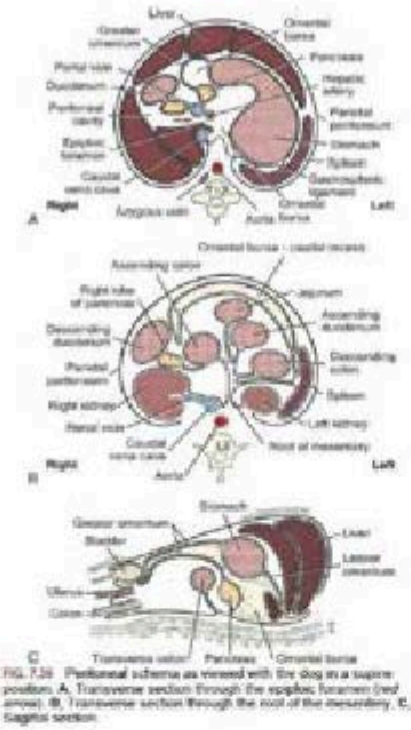
- Virtual skills/dissection

Innervation to abdominal wall

- More relevant in large animals
- Epaxial muscles – dorsal branches of spinal nerves
- Skin on the dorsal ½ of the abdomen – lateral thoracic nerve

Peritoneum

- **Serous membranes lining the abdominal and pelvic cavities and the abdominal organs**
  - Parietal – lining of the abdominal and pelvic cavities
  - Visceral – covering of the abdominal organs
- Organs covered on only one surface by peritoneum are called **'retroperitoneal'** and organs that receive nearly complete covering are called **'intraperitoneal'**.
- Parietal and visceral layers are continuous with one another at a number of sites – consists of two layers – Ex. Ligaments, mesenteries, omenta
- Between the parietal and visceral layers are various amounts of connective tissue, fat and lymph nodes. Vessels and nerves supplying organs also run through these layers.
  - None of these structures lie in the peritoneal cavity



From Miller and Evans' Anatomy of the Dog

Peritoneum

Ligaments:

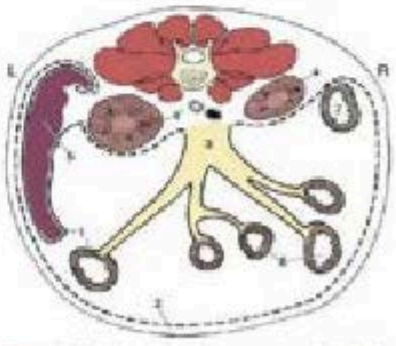
Peritoneal ligaments connect organs, or to the lateral and ventral body wall.

Mesentery:

Suspends organs from dorsal body wall – Prefix 'meso'

Root of mesentery:

Mesentery 'bunching up' like a curtain – intestine in suspended in from a small area – vessels and nerves to the intestines arise at this area – Constant between species – **Ventral to L2** – important surgical radiographic landmark.



From Miller and Evans' Anatomy of the Dog

Omentum

- **Greater omentum**
  - Is the dorsal mesogastrium
  - **Attaches the greater curvature of the stomach to the body wall**
  - Superficial leaf
  - Deep leaft
  - **Omental bursa** - hernia
  - **Epiploic foramen** – right of the median plane – Boundaries: craniodorsal – caudate process of the liver and the caudal veneca; Caudoventral – portal vein
  - **Phrenicosplenic ligament; Gastrosplenic ligament**
- **Lesser omentum**
  - Is the ventral mesogastrium
  - **Attaches the lesser curvature of the stomach to the liver**
  - **Hepatoduodenal ligament** – portion of the lesser omentum that goes from the liver to the duodenum
  - **Hepatogastric ligament** – the lesser omentum that goes from the liver to the stomach

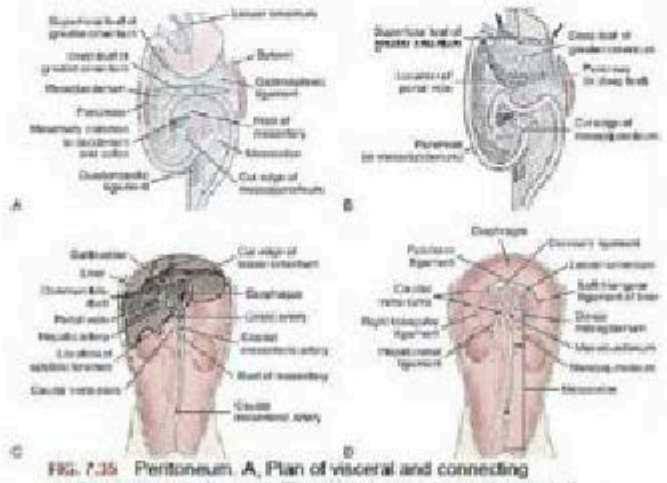


FIG. 7.35 Peritoneum. A, Plan of visceral and connecting peritoneum, ventral aspect. The greater omentum is transected caudal to the stomach. Red arrow in epiploic foramen. B, Plan of peritoneum with greater omentum reflected cranially. The transverse colon is displaced caudally. C, Plan of the dorsal reflections of the connecting and parietal peritoneum. The stomach and intestines removed. D, Plan of the dorsal reflections of the connecting and parietal peritoneum. All abdominal viscera removed.

From Miller and Evans' Anatomy of the Dog

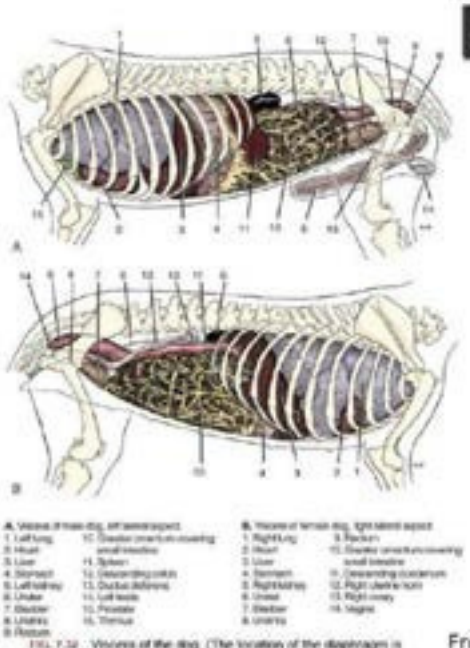


FIG. 7.32 Viscera of the dog. (The location of the diaphragm is indicated by a dotted circle.)

From Miller and Evans' Anatomy of the Dog

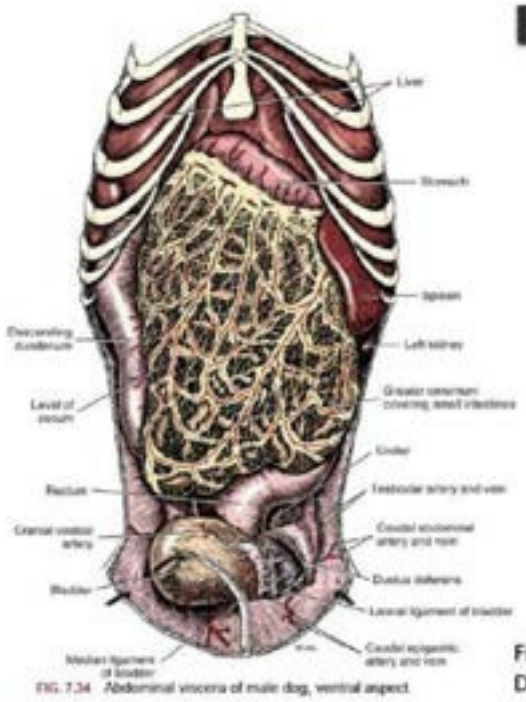


FIG. 7.34 Abdominal viscera of male dog, ventral aspect.

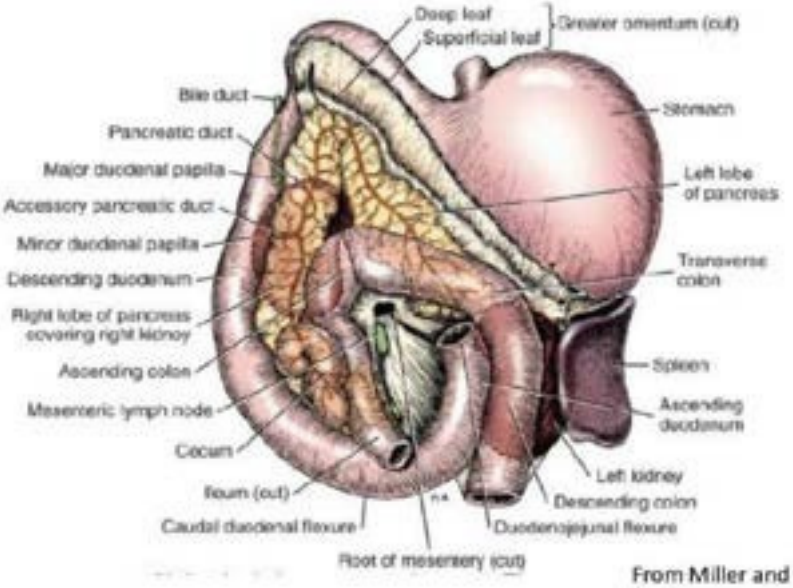
From Miller and Evans' Anatomy of the Dog

Canine Abdominal Topographical Anatomy: Implications for surgical procedures (Topography, Peritoneum, Diaphragm) (Lecture 3)

Puliyur S. MohanKumar, BVSc, PhD
Josiah Meigs Distinguished Professor
College of Veterinary Medicine
University of Georgia, Athens, GA 30602, USA

Topographical Abdominal Anatomy - interactive

- Structures on the left of midline
  - Parts of the stomach, spleen, ascending duodenum (?), left lobe of pancreas, lobes of liver, left kidney, descending colon
- Structures on the right of midline
  - Parts of the stomach, descending duodenum, caecum, ascending colon, right kidney, right lobe of the pancreas,
- Structures along the midline
  - Abdominal aorta
  - Caudal venecava
  - Root of mesentery
  - Vessels providing the abdominal viscera – orgination
  - Autonomic structures



From Miller and Evans' Anatomy of the Dog

Importance of knowing the topography

- Palpation
- Ultrasound
- Surgeries

Interactive exercises

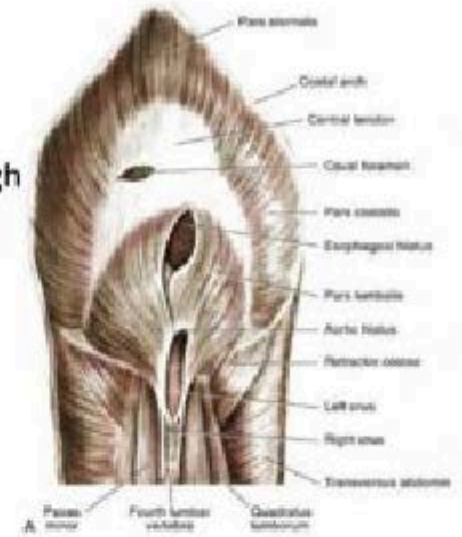
- Virtual skills

Diaphragm

- Musculotendinous separation between thoracic and abdominal cavities
- Projects into the thoracic cavity like a dome
- Attaches to the ventral surfaces of the vertebrae, the ribs and sternum
- The central tendon
- Thoracic side
  - Pleura??
  - Separated from the pleura by the endothoracic fascia
- Abdominal side
  - Separated from the peritoneum by transversalis fascia

Diaphragm (continued)

- Crus of the diaphragm
- Openings for structures to pass through
  - Aorta
  - Venecava
  - Esophagus



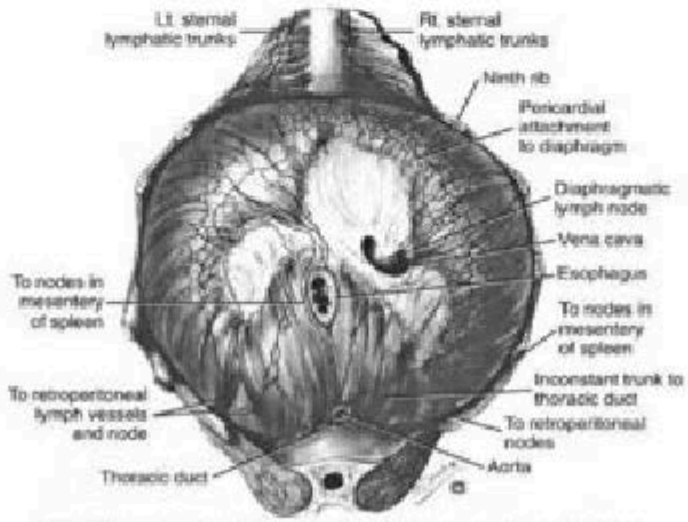


FIG. 13.11 The pleural surface of the diaphragm in a dog after the intraperitoneal injection of a graphite preparation. (From Higgins GM, Graham AS: Lymph drainage from the peritoneal cavity in the dog, *Arch Surg* 19:453–465, 1929. Copyright 1929, American Medical Association.)

From Miller and Evans' Anatomy of the Dog

Blood supply and innervation

- Blood supply: Caudal phrenic and other branches from aorta
- Nerve: Phrenic nerve

Interactive exercises

Canine Abdominal Anatomy: Stomach, Liver, Gall bladder, Spleen and Kidney (Lecture 4)

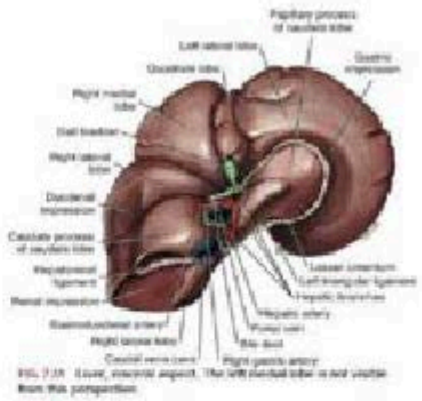
Puliyur S. MohanKumar, BVSc, PhD
Josiah Meigs Distinguished Professor
College of Veterinary Medicine
University of Georgia, Athens, GA 30602, USA

Liver

- Lies almost entirely on the intrathoracic part of the abdomen
- Immediately caudal to the diaphragm
- Diaphragmatic surface (cranial) is convex (adapts to the concavity of the diaphragm)
- Visceral surface (caudal) is concave and is related to stomach.
- Dorsal border is related to esophagus, right crus of the diaphragm and caudal venecava

Blood supply and ligaments

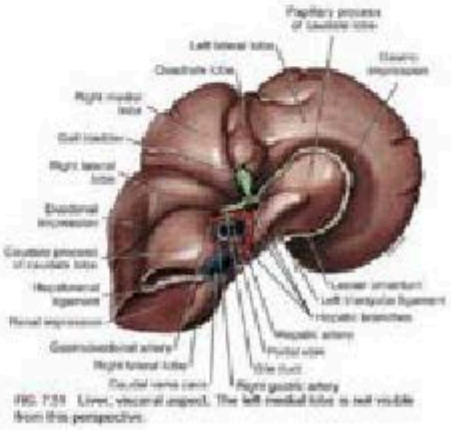
- Receives arterial and venous supply
- Hepatic artery
- Portal vein
- Falciform ligament
- Round ligament of the liver
- Coronary ligament
- Triangular ligaments
- Hepatorenal ligament
- Lesser omentum



From Miller and Evans' Anatomy of the Dog

Lobes of the liver

- Left medial
- Left lateral
- Right medial
- Right lateral
- Quadrate
- Caudate – Caudate and papillary processes
- Renal fossa/impression



From Miller and Evans' Anatomy of the Dog

Gall bladder

- Embedded in the fossa between _____ lobe and _____ lobe
- Cystic duct
- Hepatic duct
- Bile duct (Major duodenal papilla – where is it located?)

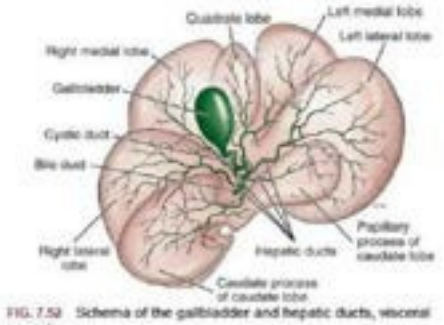


FIG. 7.52 Schema of the gallbladder and hepatic ducts, visceral aspect.

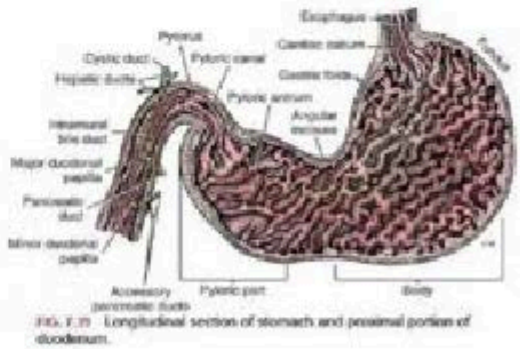
From Miller and Evans' Anatomy of the Dog

Spleen

- Located on the left side of the abdominal wall
- Vessels enter and leave the hilus
- Blood supply: splenic artery

Stomach

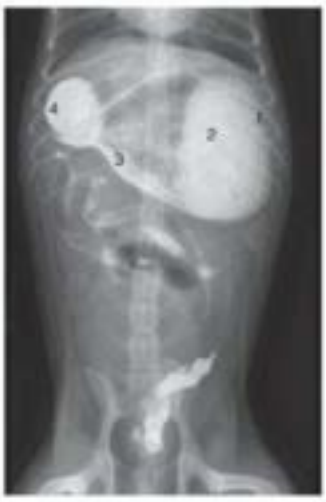
- Greater and lesser curvatures
- Cardiac, Fundus and Body
- Pyloric part (consists of all three below)
  - Pyloric antrum – funnel shaped proximal part
  - Pyloric canal – the narrower, tubular distal part
  - Pylorus – opening between stomach and duodenum – pyloric sphincter

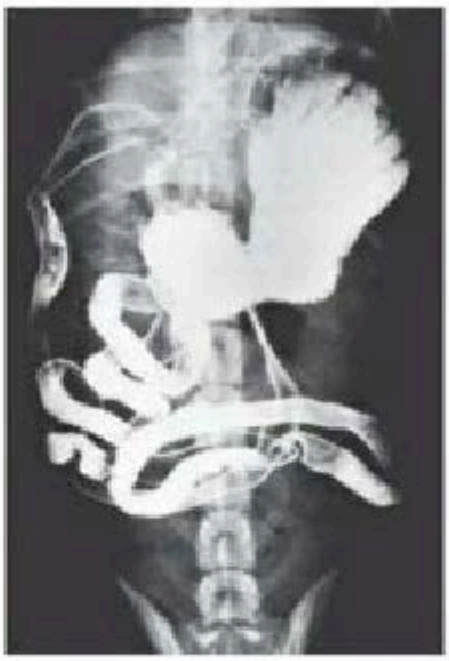


Longitudinal section of stomach and proximal portion of duodenum.

From Miller and Evans' Anatomy of the Dog

Interactive exercises



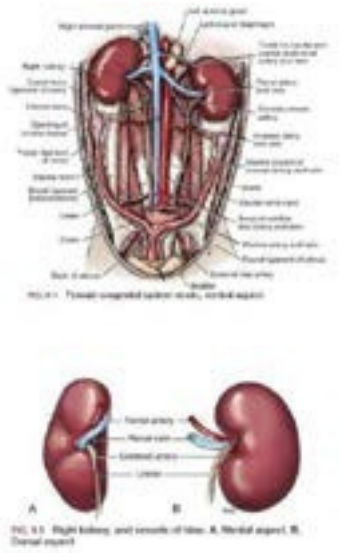


Blood supply and innervation

- Branches of abdominal aorta
- Autonomic nervous system

Kidneys

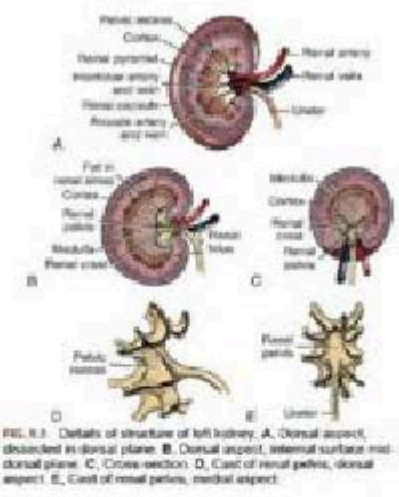
- Paired – which is more cranial?
- Blood supply
  - Renal artery
- Innervation
  - Autonomic



From Miller and Evans' Anatomy of the Dog

Kidneys

- Enclosed by a tough fibrous tissue capsule
- Cortex – contains most of the glomeruli
- Medulla – primary tubules
- Dog: Cortex, Medulla, Renal crest, Pelvis, ureter
- Renal pyramid, Renal papilla
- Hilus – receives renal vessels and has the ureter
- Renal sinus



From Miller and Evans' Anatomy of the Dog

Ureter

- Penetrates the dorsal wall of the bladder at an oblique angle near its neck
- Ectopic ureters – may end blindly – causes hydronephrosis or may terminate in uterus, vagina or urethra causing urinary incontinence

Bladder

- Apex, body, neck, trigone
- Detrusor muscle
- Sphincter – Primarily a physiological (not an anatomical) structure
- Ligaments:
  - Lateral ligaments, Median ligament
- Blood supply:
  - Cranial and caudal vesicular arteries
- Innervation
  - Autonomic and somatic

Urethra

- Intrapelvic and extrapelvic
- Female urethra is short and is completely intrapelvic

Canine Thoracic Anatomy - 1
(Lecture 5)

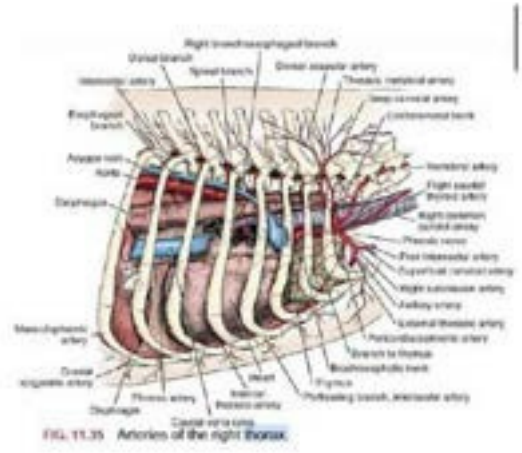
Puliyur S. MohanKumar, BVSc, PhD
Josiah Meigs Distinguished Professor
College of Veterinary Medicine
University of Georgia, Athens, GA 30602, USA

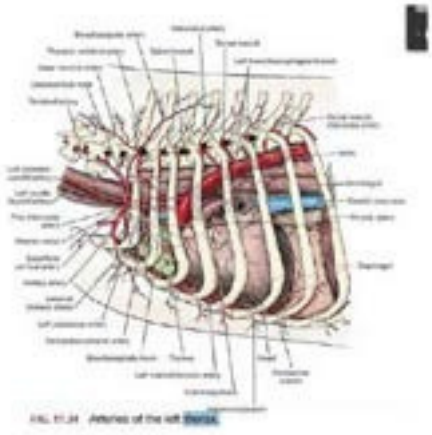
Bony thorax

- Thoracic vertebrae dorsally, ribs laterally and sternum ventrally
- Thoracic inlet
  - T1 vertebra, first pair of ribs, manubrium of sternum
- Caudal thoracic opening (thoracic outlet)
  - Last thoracic vertebrae, last pair of ribs, costal arch and xiphoid process of sternum – sealed by the diaphragm
- The first 4-5 pairs of ribs are straight and relatively fixed and provide support
  - Caudal pairs are curved and swing in a 'bucket handle' fashion; swinging cranially widens and shortens the thoracic cavity and coupled with caudal movement of the contracting diaphragm increases the volume of the thoracic cavity and draws air into the lungs
- Epaxial muscles
- Hypaxial muscles

Blood supply and innervation

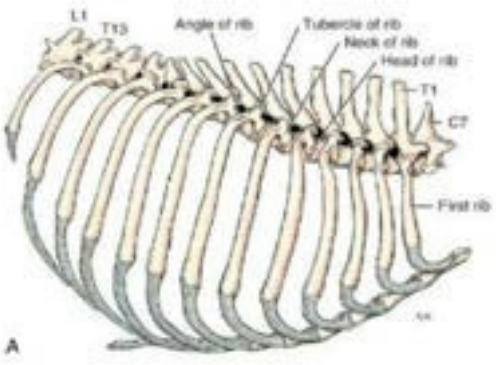
- Arterial
  - Intercostal arteries
  - Dorsal from aorta
  - Ventral from internal thoracic artery
- Venous
  - Intercostal veins
    - Dorsal intercostal veins drain into azygos
    - Ventral intercostal veins drain into the internal thoracic vein (unpaired) and then into the cranial venecava
- Innervation

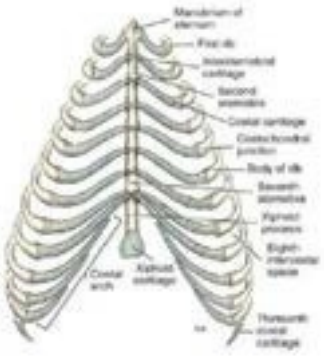




From Miller and Evans' Anatomy of the Dog

Ribs and sternum





From Miller and Evans' Anatomy of the Dog

Interactive exercise

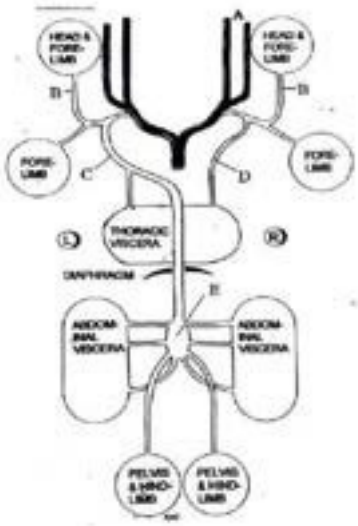
- Bifid sternum

Mediastinum

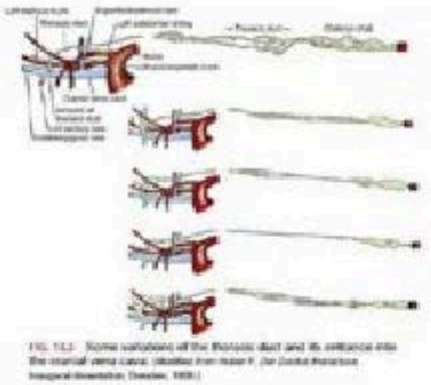
- Space between left and right pleural sacs
- More or less median in the thorax
- Heart and other organs are located
- It is unclear whether the two pleural sacs communicate
  - Unilateral Pneumothorax or pyothorax tends to remain unilateral, but to be safe one should consider any traumatic pneumothorax including surgical thoracotomy to be bilateral and provide mechanical assistance to breathe.

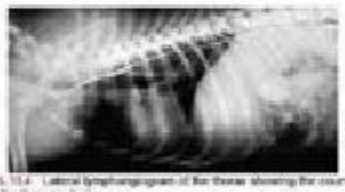
Lymphatic system in the thorax

- Lymph vessels return fluids from the tissue interstitium to the blood stream via the great veins
- Lymph nodes - situated along the length of the lymphatic vessels are like filters – remove and destroy any particulate matter, microorganisms within the lymph.
- The left lymphatic duct – Thoracic duct
  - Major – drains the left side of the thoracic viscera and from the entire body caudal to the diaphragm
- The right lymphatic duct
  - Drains from the right side of the thorax



Lymphatics





From Miller and Evans' Anatomy of the Dog

Chylothorax

Canine Thoracic Anatomy - 2
(Lecture 6)

Puliyur S. MohanKumar, BVSc, PhD
Josiah Meigs Distinguished Professor
College of Veterinary Medicine
University of Georgia, Athens, GA 30602, USA

Swallowing vs normal respiration

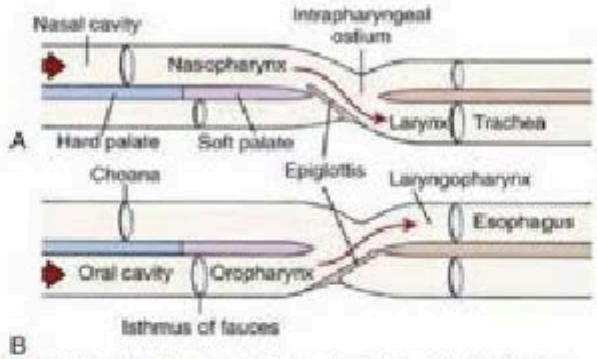


FIG. 8.10 Diagram showing relation of portions of pharynx to esophagus and trachea. A, During normal respiration. B, During swallowing.

From Miller and Evans' Anatomy of the Dog

Trachea and airways

- Trachea bifurcates above base of heart
  - Left and right principal/primary bronchi
  - Lobar/secondary bronchi which enter each lung lobe
- Lung lobes are determined by the presence of lobar bronchi and NOT by external fissures

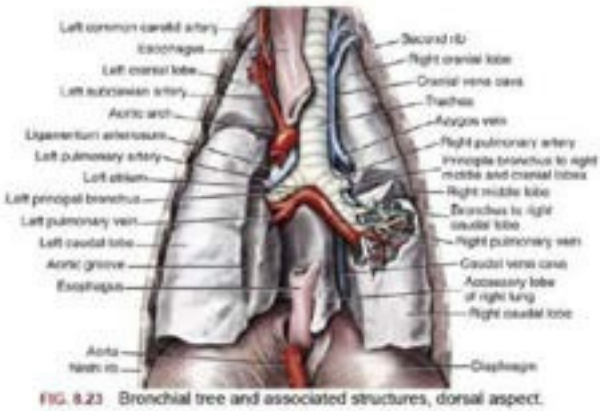
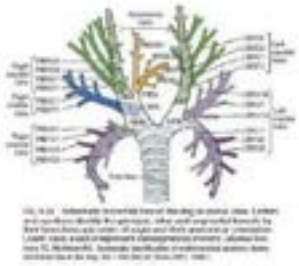


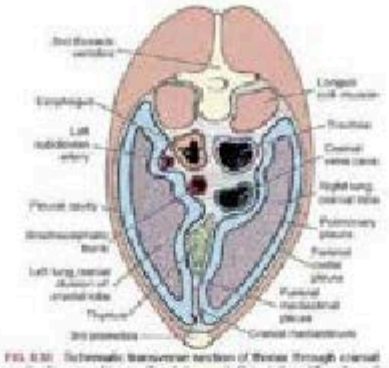
FIG. 8.23 Bronchial tree and associated structures, dorsal aspect.



From Miller and Evans' Anatomy of the Dog

Pleural membranes

- Pleurae
- Visceral and parietal pleurae are held together by negative pressure (vaccum) existing within the pleural sac
  - Surgical or traumatic opening in the chest wall may allow an intake of air into the pleural cavity – recoils the lungs – pneumothorax
  - Fluid (pus, blood, lymph, etc.) may accumulate between the pleural layers – difficulty in breathing – named after the type of fluid
  - Inflammation of the pleural membranes (pleuritis or pleurisy) – can cause them to adhere to one other – difficult and painful respiratory movements



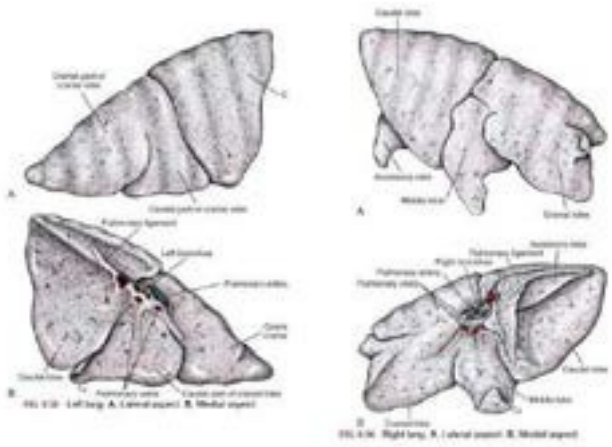
From Miller and Evans' Anatomy of the Dog

Pleurae and recess

- Lungs don't occupy the thoracic cavity fully
  - Costodiaphragmatic recess
  - Pleural cupula
    - May extend past the first rib
    - Vulnerable for puncture wounds of the neck

Lungs

- Left
  - Cranial lobe
    - Cranial part
    - Caudal part
  - Caudal lobe
- Right
  - Cranial lobe
  - Middle lobe
  - Caudal lobe
  - Accessory lobe



From Miller and Evans' Anatomy of the Dog

Cardiac notch

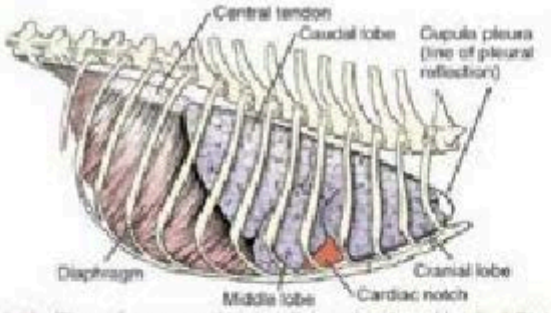
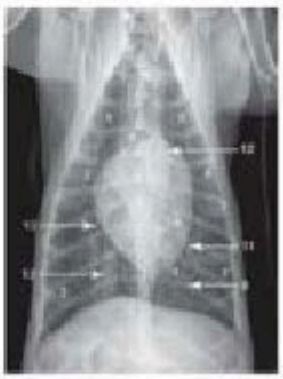
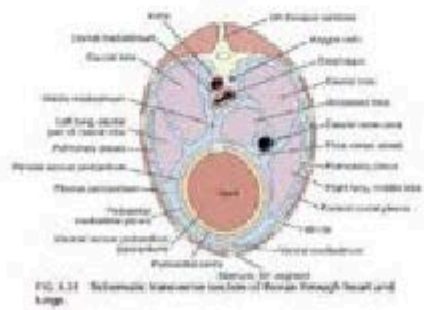


FIG. 8.33 Thoracic cage and lungs. (Lungs hardened in situ.) Right side.

From Miller and Evans' Anatomy of the Dog





From Miller and Evans' Anatomy of the Dog

Interactive exercises

Heart

- Chambers
- Flow of blood in the fetus
- Flow of blood in the adult
- Vascular ring abnormalities
- Developmental abnormalities
- Membranes of the heart

Interactive exercises

Nerves in the thoracic cavity

- Somatic structures
- Autonomic structures

With Best Compliments From:

VENKY'S PET®
The expression of loyalty

Scientific Solution for
Spinal Health & Cardiac Strength

Venworld®
THE BEST IN ANIMAL HEALTHCARE

Vendisc™
व्हेनडिस्क
Back support formula for pet

Presentation: 50 tablets

Benefits:

- **Bovine tracheal cartilage** is rich in chondroitin sulphates, which makes up the matrix of connective tissue and is found in high concentration in tissues of spine and intervertebral discs
- **Taurine** is a sulfur containing amino acid involved in synthesis of connective tissues
- **Serine** is an amino acid which links the glycosaminoglycans structure to the protein portion of connective tissues
- **Zinc** is necessary for tissue respiration and important in the healing process
- **Vitamin C** support collagen production and glycosaminoglycan metabolism

VenCard™
वेनकार्ड स्ट्रेंथ Strength
Heart Support Formula For Pets

Presentation: 50 tablets

Benefits:

- **L carnitin:** supports the use of fat for energy metabolism, which helps to balance cholesterol and triglycerides level, as well as supporting the heart's energy levels
- **N,N- Dimethylglycine (DMG)** supports proper blood circulation as an antioxidant
- **Coenzyme Q10** supports oxygenation of heart tissues and helps to protest against oxidation. Coenzyme Q10 helps strengthening the heart muscles as lipid soluble antioxidant.

INDIA'S FIRST MONOCLONAL ANTIBODY THERAPY IN VETERINARY MEDICINE

CYTOPOINT®

(Lokivetmab)

– LONG LASTING –

Comfort

An injectable that puts long-lasting control of canine atopic dermatitis in your hands.

TARGETED

Targets and neutralizes canine interleukin-31 (cIL-31), a key itch-inducing cytokine in atopic dermatitis[2]

WORKS FAST & LASTS

Begins working within 1 day and delivers 4 to 8 weeks of relief^ from the clinical signs of atopic dermatitis; in-office administration ensures compliance[1]

SAFE*

Safe* for dogs of all ages, even those with concomitant diseases, and can be used with many common medications[3,4]

^Repeat administration every 4 to 8 weeks as needed in the individual patient1. | *When compared to conventional therapy, steroids and cyclosporine | 1. Data on file, Study Report No. C863R-US-12-018, Zoetis LLC. | 2. Gonzales AJ, Humphrey WR, Messamore JE, et al. Interleukin-31: its role in canine pruritus and naturally occurring canine atopic dermatitis. Vet Dermatol . 2013;24(1):48 - 53. doi:10.1111/j.1365-3164.2012.01098.x. | 3.Data on file, Study Report No. C362N-US-13-042, Zoetis LLC. | 4. Data on file, Study Report No. C961R-US-13-051, Zoetis LLC. | *Monoclonal antibody (mAb): a single, homogeneous antibody preparation cloned from a single lineage of B cells. mAb therapy is designed to mimic a normal immune function by recognizing and binding to one single segment, or epitope, on a particular target antigen. Interleukin 31 (IL-31): a cytokine that triggers the process of sending itch signals to the brain. cIL-31 is canine IL-31. Cytokine: a protein that signals other cells by binding to receptors on those cells. mAbs are catabolized within cells into amino acids and peptides. They are not metabolized in the liver or kidneys, converted into reactive or toxic metabolites, or excreted in urine. Thus, they are highly unlikely to induce liver or kidney toxicity.6,7 | 6. Greenfield EA. Generating monoclonal antibodies. In: Greenfield EA, ed. Antibodies: A Laboratory Manual. 2nd ed. Cold Spring Harbor, NY: Cold Spring Harbor Laboratory Press; 2014:201-221. | 7. Olivry T, Bainbridge G. Clinician's Brief: Advances in veterinary medicine: therapeutic monoclonal antibodies for companion animals. March 2015. http://www.cliniciansbrief.com/sites/default/files/attachments/ZoetisCN_Mar_FNL.pdf. Accessed June 21, 2016. | Adverse reactions: Hypersensitivity reactions, Vomiting and/or diarrhoea, Neurological signs, Application site disorders, Clinical signs of immune-mediated diseases, have been reported very rarely in spontaneous reports, in such cases appropriate treatment should be administered immediately. For more information, please refer to the pack insert. | For more information, please contact: indiaanimalhealth@zoetis.com | | Zoetis India Limited, 31, 3rd Floor, Kalpataru Synergy Opp. Grand Hyatt, Santacruz (E), Mumbai-55 | To be sold by retail on the prescription of a veterinary doctor only.

zoetis

STIMUDERM ULTRA

DERMOCOSMETICS

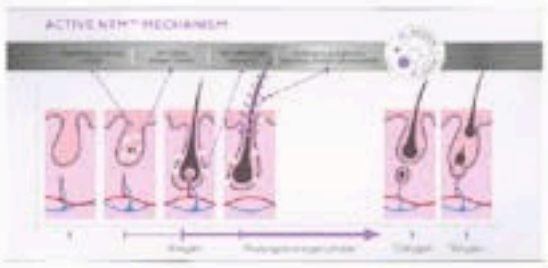
A COMPREHENSIVE SUPPORT OF
VET EXPERT IN SKIN PROBLEMS

VET EXPERT

Visible improvement in hair regrowth among:

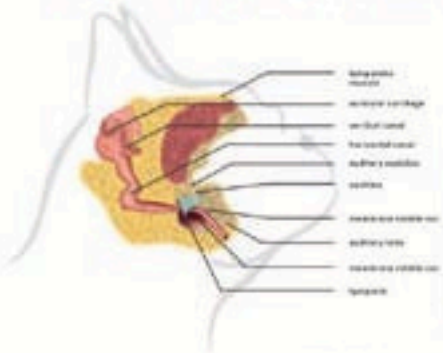
- 71% of dogs with alopecia caused by atopic dermatitis
- 79% of dogs with excoriations caused by dermatomycosis
- 67% of dogs with alopecia caused by hypothyroidism
- 71% of dogs with thinning hair caused by adverse food reactions.

the first line of innovative veterinary dermocosmetics for dog skin and coat care with excessive hair loss of various origins.



Oticurant®

For Daily Ear Hygiene and OTITIS



- It comes in powder form & absorbs moisture
- It supports the normal physiological condition of the ear
- Restores the physiological balance by binding the moisture and wax
- have strong absorptive properties

MOISTURIZING SPRAY RECOMMENDED FOR

- All atopic patients
- Excessively dry skin
- After flea allergy dermatitis
- As adjuvant therapy in endocrine conditions (hypothyroidism, Cushing disease)
- As adjuvant therapy in the benzoyl based shampoo treatment of mange and deep purulent dermatitis
- As adjuvant therapy during/after the antibiotic treatment of skin conditions (that involve itching)

HOT SPOT SPRAY RECOMMENDED FOR

- Allergic Reactions to flea bites
- Mechanical Irritation
- Food Allergies
- Atopic disease and the Airborne allergens that cause it

Dr. Philip Judge

BVSc MVS PG Cert Vet Stud MACVSc (Veterinary Emergency and Critical Care, Medicine of Dogs) Senior Lecturer, Veterinary Emergency & critical care, James Cook University, Australia

Dr. Philip Judge is an adjunct senior lecturer in veterinary emergency and critical care at James Cook University. Philip is also director of Vet Education Pty. Ltd, an independent online education company that conducts a comprehensive continuing professional development program for the veterinary profession. Philip's key interests are in the field of canine and feline sepsis, toxicology, and emergency therapy.

Emergency Anaesthesia of the Cat

Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (Vet. Emergency and Critical Care; Medicine of Dogs)

Introduction

Anaesthesia of patients should always begin with an assessment of the patient. Without this assessment, we may miss subtle changes or abnormalities that can potentially lead to complications, or even death under anaesthesia. Make the most of the opportunity afforded by pre-anaesthetic assessment – particularly in those patients that are having anaesthesia for non-routine procedures. It will enable identification of patient abnormalities that require correction or consideration in the development of an anaesthetic plan. Application of this approach will reduce the chances a potential anaesthetic risk factor –and hence the risks of anaesthetic death - may be overlooked. The risks associated with not looking for anaesthetic risk factors make this approach well worthwhile.

Patient Evaluation

A systematic body system review and should be done. Organ systems of greatest concern are the cardiovascular system, respiratory system, neurological system, kidneys and liver.

Obviously, the more unwell a patient is, the greater the anaesthetic risk is. With this in mind, it is important to look at the whole patient when conducting your physical examination.

When performing a full body systems examination, the mnemonic A CRASH PLAN is often used as a framework, as it helps cover most body systems:

A = Airway
C = Cardiovascular and circulatory
R = Respiratory
A = Abdomen
S = Spine
H = Head
P = Pelvis (including a rectal examination)
L = Limbs (including the tail)
A = Arteries
N = Neurological Evaluation (including cranial nerves, reflexes, and pain assessment)

When performing physical examinations, it is critical to look at as many parameters as possible, so that a complete list of patient risk factors, and desired diagnostic tests can be assimilated prior to the anaesthetic.

Identification of Anaesthetic Risk Factors

Following collection and analysis of patient history, physical examination and data collection, it should be possible to determine a clear list of patient risk factors. In classifying the patient PRIOR to anaesthesia, we allow a prognosis for anaesthesia to be estimated. This can then be conveyed to owners, in order to keep them informed of the likelihood of potential misadventure from the procedure. This also allows the veterinary team to make necessary anaesthetic protocol adjustments in order to improve patient outcome potential as well. For example, when we anaesthetise patients with emergency or critical illness, we are most concerned with abnormalities of the cardiovascular system, the respiratory system, the neurological system, and renal and gut perfusion.

To this end, it is vital that we select drugs and protocols that minimize the likelihood of damage to these organ systems. Another example may be that we avoid an anaesthetic completely in a patient with severe liver disease if possible, or dramatically reduce drug doses, because the liver is where most of our anaesthetic drugs are metabolized.

The American Society of Anaesthesiologists anaesthetic risk classification system classifies anaesthetic risk in patients based on result of physical examination and diagnostic work-up procedure findings, according to the table below…

American Society of Anaesthesiologists
Classification of Physical Status and Anaesthetic Risk

Category	Physical Status	Possible Examples
I	Normal Healthy Patients	No discernible disease; animals entered for ovariohysterectomy, castration
II	Patients with mild local, or generalized (systemic) disease	Skin tumour, localized infection (e.g. cat bite abscess), compensated heart disease (e.g. murmur with no symptoms)
III	Patients with systemic disease	Fever, mild dehydration, anaemia, anorexia, weight loss etc.
IV	Patient with systemic disease that is a constant threat to life	Uraemia, toxaemia, severe dehydration, any patient in shock, anaemia, cardiac disease with symptoms despite medication, emaciation, high fever, severe vomiting or diarrhoea
V	Moribund patients that are not expected to survive more than 24 hours	End-stage shock, terminal malignancy or infection, severe trauma, GDV, ruptured pyometra, respiratory distress, massive haemorrhage

Obviously, the lists given under possible examples are not exhaustive, but they do give an idea of the sorts of conditions represented by each anaesthetic risk category. So, how to these categories translate into anaesthetic risk?

- **Patients in category 1 have been found to have a mortality rate due to general anaesthesia of around 1-2 deaths per 1000 anaesthetics (or 0.2%).**
- **Patients in category 5 have an overall mortality rate due to general anaesthesia of between 5 and 15%, depending on the underlying disease.**
- **Grades 2-4 have between a 1 and 4% mortality rate due to general anaesthetic.**

These statistics indicate that a patient that has fever, or even mild dehydration or pre-anaesthetic anorexia (reduced or absent intake of food due to disease for longer than 2-3 days) can have up to 5-6 times the anaesthetic mortality rate of a normal healthy individual. It stands to reason then, that the more we can do to normalize body function BEFORE the anaesthetic, the lower the risk of anaesthetic death will likely be. Add into this scenario, that the incidence of anaesthetic complications (such as short term apnoea, low blood pressure, etc.) is higher in ill patients than normal patients, and it becomes even more important to identify and classify anaesthetic risk.

Correction of Anaesthetic Risk Factors

Following identification of patient-related anaesthetic risk factors, it is essential that as many risk factors as possible be corrected prior to anaesthesia, in order to reduce overall risk to the patient.

In most cases, it is safer for anaesthesia to be delayed until all potential risk factors are corrected or managed. However, there may be instances, such as in patients with airway obstruction, or GDV for example, where anaesthesia is indicated shortly following patient presentation (almost immediately for airway obstruction!). In these cases, it is still important to begin correction of abnormalities either just prior to anaesthesia, or vigorously correct them during the early period of anaesthesia, to avoid complications developing later during the anaesthetic period, or during recovery from anaesthesia. Let's now examine some of the management tools we have to assist in reducing anaesthetic risk.

Correction of Respiratory Tract Risk Factors

In EVERY anaesthetic, we need to ensure the patient has adequate respiratory support and are especially important in any patient having an anaesthetic that presents in a state of being unwell – largely because these patients are much less tolerant of anaesthesia, and are at a higher risk of complication.

1. Provide supplemental oxygen for **AT LEAST 5 minutes** prior to induction of anaesthesia. This increases the reserve of oxygen available to your patient during anaesthetic induction. Even young kittens and puppies require the ready availability of oxygen prior to anaesthetic induction – particularly if they become stressed, or appear anxious prior to – or during anaesthetic induction as a partial mitigation of the stress response. Failure to do so can result in activation of the stress response that can have a negative effect on blood pressure, circulation and organ function, well after the anaesthetic period

2. Ensure thoracocentesis is performed prior to anaesthesia in patients with pneumothorax or pleural fluid accumulation, in order to improve respiratory reserve during anaesthetic induction and maintenance.

3. Intubate the patient at the earliest opportunity following induction to gain control of the airway, and administer 100% oxygen via the anaesthetic machine. Most anaesthetic drugs depress ventilation or respiratory function to some degree. In addition, during anaesthetic induction, tidal volume decreases and atelectasis – or collapse of alveoli – occurs, decreasing the potential efficiency of respiration. Early intubation and provision of augmentation of the patient breathing efforts and/or supplemental brea thing shortly following induction can reduce or reverse atelectasis and the transient hypoxia that frequently occurs following induction.

4. Provide ventilation support using an ambu-bag or anaesthetic re-breathing bag. The aim of ventilation support is to provide augmentation of the patient's own respiratory efforts shortly following anaesthetic induction and intubation or intermittent positive pressure ventilation, in order to reverse lung atelectasis that will have occurred during anaesthetic induction. The patient should receive intermittent positive pressure ventilation if the patient becomes apnoeic or hypo-ventilates (respiratory rate lower than 10-12 breaths per minute) following induction of anaesthesia (i.e. most patients), OR if the patient has one of the following conditions

    a. Pneumonia
    b. Pulmonary oedema
    c. Pulmonary contusions
    d. Collapsed lungs – pleural effusion etc.
    e. Airway obstruction
    f. Diaphragmatic hernia, pyothorax, chylothorax etc.
    g. Chest wall disease (neoplasia, rib fractures etc.)

How do weassist ventilation? The basic technique involves an 'augmentation' of the patients own breathing efforts, if the patient is breathing, or providing respiratory efforts at 12-15 breaths per minute, and 8-15 ml/kg if the patient is not breathing at all. Inspiratory time should be kept to approximately half the length of the time allowed for exhalation (i.e. inspiration 1/3 of the time for the respiratory cycle: exhalation and pause prior to next breath 2/3 of respiratory cycle time) to minimize interference with blood flow through the thoracic vessels, which predominantly occurs during exhalation. Peak anaesthetic circuit pressure should be less than 20-22 cm water pressure during positive pressure ventilation efforts.

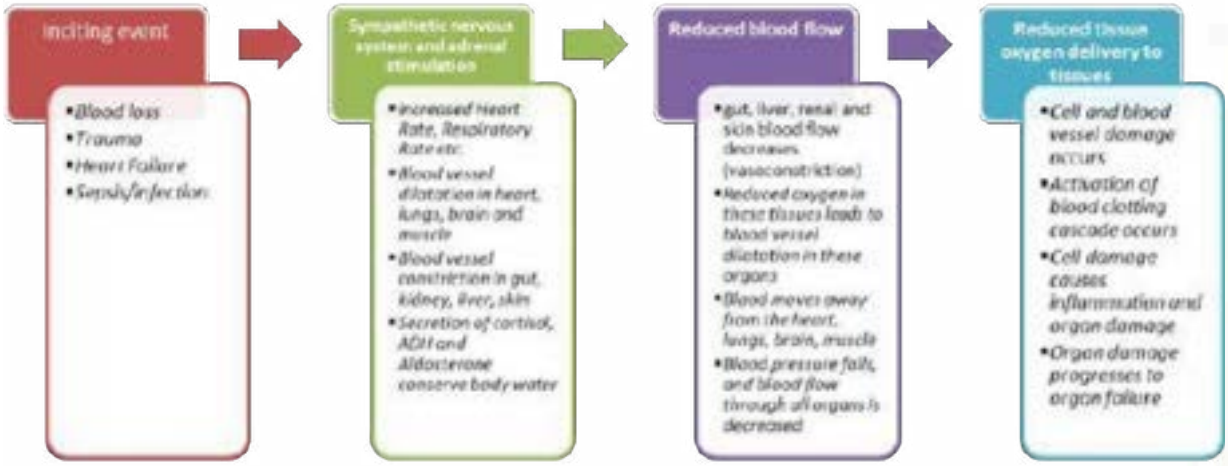
5. Monitor the patient using blood gas, capnography, pulse oximetry, apnoea alert systems and most importantly physical observation

In addition to the aforementioned strategies, specific therapy should be directed at the underlying cause of respiratory compromise. For example, nebulised adrenaline +/- inhaled corticosteroids should be applied to patients with brachycephalic airway obstruction or laryngeal disease; antibiotic therapy should be given to patients with pneumonia; diuretic therapy should be given to patients with heart failure etc.

Correction of Cardiovascular Risk Factors

The cardiovascular system should receive assessment and appropriate support in every anaesthetic patient. The following outlines key components of this support.

1. **Assess and correct shock** – Shock occurs commonly in veterinary patients. Shock is a complex syndrome that has many different causes, stages and physical and physiological characteristics. Clinical signs of shock usually include elevations in heart rate, altered mucous membrane characteristics (pale or bright pink), strong or weak pulses, and central nervous system depression. The following flow-chart outlines the neuro-hormonal cascade initiated in shock, and physiological effects that result from shock.



The Underlying Process is Abnormal Oxygen Delivery to Tissues

Patients with clinical signs of shock should have treatment is administered rapidly, in order to halt the cascade of events outlined above, to prevent organ failure and death.

a. **Correction of intravascular volume deficits** – for patients displaying symptoms of shock or haemodynamic compromise, such as those with weak pulses, tachycardia, bradycardia, collapse, tacky mucous membranes; initial fluid resuscitation should begin with rapid intravenous administration of a balanced isotonic crystalloid solution such as lactated Ringer's solution, or Normosol-R. Initial fluid rates should be 5-7 ml/kg IV over 10 minutes, followed by patient reassessment, and for this procedure to be repeated until signs of haemodynamic compromise are beginning to resolve – as evidenced by improved pulse quality, systolic blood pressure, improvement in capillary refill time, improved mentation, a trend towards normal heart rate etc.

b. **Assess and Manage Packed Cell Volume** – most complications resulting from anaesthesia in critical patients result from inadequate tissue oxygen delivery to organs and tissues. Red blood cell mass is critical for oxygen delivery. Patients undergoing anaesthesia should have a PCV above 27-30% at all times, if possible, as red cell mass below this level is associated with reduced tissue oxygen delivery.

c. **Assess and Correct Plasma Protein and Albumin Concentrations** – the total protein level in the blood - particularly albumin – is critical in ensuring the maintenance of blood volume. Low total protein or albumin concentrations are associated with decreased blood volume, and poor tissue and organ perfusion. Correction , or maintenance of protein or albumin deficits using fresh frozen plasma can assist in improving blood flow through tissues, assists in protein-bound drug and hormone delivery, and may assist in reducing organ dysfunction or failure following anaesthesia. Suggested doses of fresh frozen plasma are 10-30 ml/kg IV as required

d. **Assess blood clotting** – Coagulation assessment is extremely important, particularly for patients undergoing surgery. Perform an activated clotting time (ACT), or APTT/PT along with an estimation of platelet numbers before induction of general anaesthesia. Most anaesthetics lower body temperature, which tends to prolong clotting times. If anaesthesia is super-imposed on top of a pre-existing clotting abnormality, patients may bleed excessively into vital organs such as the brain, or lungs, leading to serious, if not fatal complications. Clotting times should be normalized prior to induction of general anaesthesia using blood or plasma transfusions to effect.

e. **Assess and normalize cardiac rhythm** – Evidence of pulse deficits, tachycardia or bradycardia should prompt evaluation of cardiac rhythm with an ECG. Specific treatment of an abnormal heart rhythm depends on the rhythm, and the underlying cause. In most cases, before specific drug treatment is initiated, the patient should have the following checklist ticked off

   i. Make sure the animal is not in shock. If the patient is in shock, begin intravenous fluid resuscitation as outlined above.

   ii. Check and normalize blood electrolyte concentrations. Sodium, potassium chloride, and calcium in particular, if present in abnormal concentrations in the blood, can cause or predispose a patient to developing abnormal heart rhythm. Normalize electrolyte concentrations prior to anaesthetic induction.

   iii. Manage pain. Pain can trigger the stress response, causing release of adrenaline into the circulation, as well as activating the sympathetic nervous system. Adrenaline, cortisol and activation of the sympathetic nervous system increase myocardial susceptibility to abnormal rhythms. An essential part of the management of abnormal heart rhythm should therefore be to identify pain, and to provide adequate and effective analgesia to the patient

   iv. Ensure normal blood lactate and blood gas values. Measure blood lactate and/or blood gas if theseanalysers are available to assess respiratory function and tissue perfusion. These parameters should be tested in patients with abnormal heart rhythm, and any abnormalities corrected. Initial correction of abnormalities in blood gas and lactate concentrations is usually achieved by providing oxygen and respiratory support for the patients, and the use of intravenous fluid therapy to improve blood flow through tissues to improve tissue oxygen delivery.

f. **Assess blood pressure and urine output** – Hypotension or low blood pressure is associated with the development of acute renal failure, a risk which is increased when superimposed with a general anaesthetic. Hypertension is also a risk factor for acute renal failure, as it often can signify the presence of shock which also can induce renal failure, particularly when superimposed with a general anaesthetic. **Insertion of a urinary catheter in compromised patients, as well as paediatric or geriatric patients requiring anaesthesia allows assessment of urinary production, which in many cases will correlate with kidney**

perfusion. Normal urine output is 1.4 ml/kg/hr. Patients receiving intravenous fluids may have urine output of greater than 2 ml/kg/hr. A urine output below these levels, or a falling urine output of below 1 ml/kg/hr should prompt immediate action by the anaesthetic team to improve blood flow to the kidneys through the use of intravenous fluid therapy, decreasing the depth of anaesthesia, provision of analgesia, cautious use of vasopressors and positive inotropes, and in some cases where acute kidney injury is suspected, diuretics such as mannitol and furosemide.

g. **Assess and Correct Blood Glucose** – Sick patients frequently have abnormal blood glucose concentrations. Any sick or unwell patient receiving an anaesthetic should have blood glucose level evaluated. If blood glucose concentration is low, immediate treatment should begin with 5-10 ml/kg IV 7% glucose solution given over 10 minutes, followed by a 2.5-5% solution given IV at a rate of 2-5 ml/kg/hr, (in addition to surgical rates of intravenous lactated Ringer's solution). Likewise, hyperglycaemia should be managed – particularly in the diabetic or septic patient, using intramuscular or intravenous regular insulin protocols.

h. **Assess and Correct Hydration Deficits -** Hydration deficits are assessed using physical parameters. The presence of dry or tacky mucous membranes, prolapsed third eyelids, skin tenting, and elevations of PCV or TP may indicate dehydration. Hydration deficits should be corrected prior to anaesthesia if possible. Dehydration should be corrected (usually over a period of 12-24 hours), using an isotonic crystalloid fluid such as lactated Ringer's solution. Fluid volumes for rehydration should be based on the following equation

Fluid to be administered (ml) = Bodyweight (g) x % dehydration/100

For example, a 5 kg dog that is 7% dehydrate will need

Fluid to be administered (ml) = 5,000 g x 7% dehydration

= 5,000 x 0.07

= 350 ml

i. **Provide Fluid Therapy for Maintenance and Ongoing Losses –** In addition to correction of hydration deficits, the patient requires fluid therapy for maintenance of normal body functions, PLUS replacement of ongoing fluid losses through the gastrointestinal tract, wounds or injured or inflamed tissues as they occur. Maintenance rates of fluid in the cat are approximately 2.5 ml/kg/hr.

Overall, the goal of circulatory system therapy prior to anaesthesia should be to achieve the following…

- Normalize heart rate and rhythm
- Normalize capillary refill time and mucous membrane characteristics
- Normalize pulse quality
- Normalize PCV
- Normalize serum albumin and total serum protein concentrations
- Normalize urine output
- Normalize respiratory function, blood gas and acid-base physiology
- Normalize electrolyte concentrations
- Normalize hydration
- Provide fluid therapy for maintenance and ongoing losses as they occur

Correction of Neurological Risk Factors

Patients with abnormal mentation are at particular risk under anaesthesia, principally because most anaesthetic drugs affect central nervous system function, the effects of which may be profoundly magnified or altered in patients with neurological disease.

Particular at-risk patients include patients with head trauma, seizures, and abnormal mentation (excitement or depression). Key steps in correcting neurological dysfunction include paying careful attention to oxygenation, ventilation and circulatory function. Pre-oxygenation, rapid intubation and mild hyperventilation are critical steps in preventing these problems from worsening under general anaesthesia. In addition, selection of medications that do not depress blood flow to the brain excessively, or do not predispose to harmful neurological behaviours is also crucial to avoiding neurological damage.

Conclusion

Correction of anaesthetic risk factors is a key component to reducing anaesthetic risk to compromised patients. Obviously, in many patients, complete correction of risk factors is not possible without correction of the underlying disease – for which anaesthesia may be required e.g. intestinal foreign body removal in a vomiting patient with a perforated bowel. In these patients, normalizing as many abnormalities as possible reduces anaesthetic risk often sufficiently to allow a safer anaesthetic period.

Anaesthetic Protocols for Sick or Injured Patients

There is no "ideal" anaesthetic drug for use in animals. However, the pharmacokinetic properties of some anaesthetic drugs do make them safer than others for use in compromised patients. Let's take some time to review anaesthetic drug options in compromised patients, and classify them into drugs we can potentially use, and drugs we should probably avoid using due to their potential for undesirable side effects in the patient.

Drugs to Use in Sick or Injured Patients

The following drugs, when used carefully in combinations, can assist in providing safe and effective anaesthesia for critically ill or injured patients. It should be noted that critically ill and emergency patients can be extremely sensitive to ANY anaesthetic drug. Label drug doses are generally given as a guide for healthy individuals. Dose recommendations for anaesthetic drugs in emergency patients have not been established, but are generally much lower than label doses (up to 10 times lower) When administering anaesthetic medications to emergency or critically ill patients, a guide is to administer approximately 5-10% of the calculated critical patient dose and assess the response to the medication over 30-60 seconds. Following this, an incremental administration of drug may take place every 20-60 seconds until the desired level of anaesthesia has been achieved.

a. Benzodiazepines – these drugs include diazepam, and midazolam. They are classified as minor tranquilizers or sedatives in dogs and cats, and they usually have to be combined with another drug such as ketamine or an opioid such as morphine or fentanyl in order to provide reasonable sedation. They have no analgesic effect, but they do induce amnesia following administration. The advantage of these drugs is that they do not depress cardiovascular or respiratory function significantly, they are anti-epileptic medications, and are therefore reasonably safe to use in patients with neurological dysfunction. They are, however, metabolised in the liver, and so should be used with caution in patients with liver disease and evidence of hepatic encephalopathy

b. Opioids – opioids include drugs such as morphine, methadone, butorphanol, buprenorphine, hydromorphone and fentanyl. Opioids are useful anaesthetic drugs to use in critical patients because they provide good analgesia – particularly fentanyl, morphine, methadone and hydromorphone, and they have minimal deleterious effects on the cardiovascular system. Methadone, hydromorphone and morphine can however; produce profound central nervous system and respiratory depression, and this can necessitate provision of ventilation support following administration. When combined with benzodiazepines, opioids can produce good, dose-dependent sedation that can facilitate endo-tracheal intubation in some patients. Butorphanol and buprenorphine are much less effective analgesics than fentanyl, methadone, hydromorphone and morphine, but are generally safe agents to use as sedatives in compromised patients (particularly butorphanol), because they produce minimal respiratory and central nervous system depression following administration.

c. Ketamine – Ketamine is a dissociative anaesthetic with good analgesic properties. Ketamine increases heart rate and myocardial contractility following administration, and has minimal effects on blood pressure. Ketamine can cause post-induction apnoea following rapid IV administration, which are not seen significantly with slow IV administration over 1 -2 minutes. A combination of an opioid, a benzodiazepine, and ketamine produces minimal cardiovascular and respiratory depression, and facilitates endotracheal intubation in most patients. This combination is preferred in most critically ill patients. Relative contraindications to ketamine administration include animals with head trauma and evidence of increased intracranial pressure, and those with high ocular pressures or ocular trauma (due to increases in blood pressure observed with ketamine)

d. Propofol - Despite having a relatively short duration of action, propofol can cause a profound decrease in ventilation rate and respiratory function, and dramatic decreases in blood pressure which can severely compromise circulation in critical patients. Barbiturates are longer acting than propofol, and cause slightly less depressions in respiratory and cardiovascular function, but are more arrhythmogenic. Their use in critically ill or injured patients is generally reserved for patients with head trauma, seizures, or evidence of increased intra-cranial pressure or ocular pressure i.e. conditions in which ketamine is relatively contra-indicated

e. Alfaxalone – has similar effects on cardiovascular and respiratory systems to barbiturates; however, the arrhythmogenic effects do not seem to be as marked or severe. This makes Alfaxalone a good substitute for barbiturates and propofol in patients in which ketamine may be contraindicated

f. Inhalation anaesthesia – isoflurane is commonly used in veterinary anaesthesia. It is an inhalation anaesthetic that produces rapid induction and rapid recovery from anaesthesia, properties making isoflurane ideal for many patients undergoing anaesthesia. However, isoflurane has many effects that are undesirable, particularly in emergency, critically unwell or compromised patients. Isoflurane is one of the most hypotensive anesthetic agents used in dogs and cats. In addition, isoflurane depresses cardiac contractility. This makes isoflurane a drug that, while useful, should be used with extreme caution in compromised patients. Isoflurane should be used in emergency and critical patients at the LOWEST concentration possible in order to minimise deleterious cardiovascular side effects.

Drugs to Avoid in Sick or Injured Patients

There are several anaesthetic drugs that are considered contraindicated in the ill or injured patient and that should not be used except under exceptional circumstances when specifically indicated. These drugs belong to the following classes of sedatives the alpha-2 agonist drugs (xylazine and medetomidine), phenothiazines (acepromazine) and Tiletamine/zolazepam (Zoletil). Let's examine why.

a. Alpha-2 Agonists – Xylazine and Medetomidine offer good sedation, muscle relaxation and analgesia. However, both drugs cause profound depression in heart rate, myocardial contractility, and blood pressure that are unacceptable in ill or injured patients. Reversal agents are available for use with these drugs; however, reversal of sedation does not equate reliably with a return of cardiac output, or a return of normal blood pressure. Whilst dose ranges exist for these drugs for provision of analgesia in critically unwell patients, analgesic doses can produce profound decreases in blood pressure, heart rate and mentation in some patients. These drugs should be avoided in ill or injured patients

b. Phenothiazines – Acepromazine offers predictable sedation in healthy patients. However, acepromazine administration produces decreases in blood pressure, which can be severe in the critical patient. In addition, acepromazine has a long duration of action, and its effects are non-reversible, as there is no specific antidote. They should be avoided in ill or injured patients

c. Zoletil – is a long-acting combination of two drugs– zolazepam – a drug similar in action to diazepam– and tiletamine– a drug similar to ketamine. This drug combination, while not causing significant depression of cardiovascular function, can cause respiratory depression. In addition, the drug is long-acting, and is non-reversible. This drug is relatively contra-indicated in ill or injured patients primarily due to its long duration of action.

Anaesthetic Drug Protocols for Compromised Patients

Now that we have briefly reviewed potential anaesthetic drugs, what drugs might we choose for our emergency and critical patient? Because each of the drugs reviewed has at least some undesirable side effects, how do we select an anaesthetic protocol that minimizes these undesirable effects? In most patients, we do this by using a combination of drugs – to make the most of the positive effects, while minimizing the undesirable effects. Potential combinations of drugs for use in critically ill patients are many and varied, and might include combinations such as these…

1. Potential anaesthetic combination 1

    - Premedication:
        - Fentanyl 2-4 micrograms/kg IV OR
        - Butorphanol 0.1 mg/kg IV
    - Induction:
        - Diazepam 0.25-0.5mg/kg IV PLUS
        - Ketamine 2.5-5 mg/kg IV
    - Maintenance
        - Fentanyl CRI @ 8-10 micrograms/kg/hr PLUS
        - Isoflurane to effect

2. Potential anaesthetic combination 2

    - Premedication:
        - Fentanyl 2-4 micrograms/kg given IV OR
        - Butorphanol 0.1 mg/kg IV
    - Induction:
        - Diazepam 0.25-0.5mg/kg IV PLUS
        - Ketamine 2.5-5 mg/kg IV
    - Maintenance:
        - Midazolam CRI @ 0.2 mg/kg/hr PLUS
        - Fentanyl @ 5-10 micrograms/kg/hr CRI PLUS
        - Isoflurane +/-
        - Ketamine CRI @ 2-5 mg/kg/hr

3. Potential anaesthetic combination 3

    - Premedication:
        - Fentanyl 2-4 micrograms/kg given IV, PLUS
        - Midazolam 0.25 mg/kg IV OR diazepam 0.25 mg/kg IV
    - Induction:
        - Alfaxalone IV
    - Maintenance:
        - Fentanyl CRI @ 8-10 micrograms/kg/hr +/-
        - Midazolam CRI @ 0.25 mg/kg/hr PLUS
        - Isoflurane to effect
    - This combination may be used for animals with head trauma, increased intra-ocular pressure

4. Potential anaesthetic combination 4
    - Premedication
        - Fentanyl 2-4 micrograms/kg IV PLUS
        - Ketamine 3 mg/kg IV followed immediately by…
        - Constant rate infusion of ketamine 5-6 mg/kg/hr combined with fentanyl 8-10 microgram/kg/hr
    - Induction:
        - Diazepam 0.2-0.5 mg/kg IV PLUS
        - Ketamine 3-5 mg/kg IV
    - Maintenance
        - Fentanyl/ketamine CRI as above PLUS
        - Isoflurane given to effect (0.4-0.7%)
5. Potential anaesthetic combination 5
    - Premedication
        - Fentanyl 2-4 micrograms/kg IV PLUS
        - Ketamine 3 mg/kg IV
        - Constant rate infusion of fentanyl 10 micrograms/kg/hr combined with ketamine 3-5 mg/kg/hr
    - Induction
        - Diazepam 0.2-0.5 mg/kg IV PLUS Ketamine 3-5 mg/kg IV OR
        - Alfaxalone IV to effect
    - Maintenance
        - Fentanyl-ketamine constant rate infusion PLUS
        - Isoflurane to effect

The aim with all of these protocols is to minimize cardiovascular and respiratory compromise, maintaining isoflurane – the most hypotensive drug we may use in these patients, at the lowest possible dose. In addition, the use of local anesthesia, nerve blocks and administration of intravenous lignocaine can provide additional analgesia to patients having surgery, and will reduce the amount of sedative and maintenance anesthesia required.

Opioids such as morphine or methadone may be substituted at low doses in many patients, and continued as constant rate infusion throughout surgery. Care must be taken with these longer-acting opioids as profound respiratory and/or central nervous system depression may occur, occasionally necessitating endotracheal intubation and provision of ventilation support +/- opioid reversal with methadone

Top Anaesthetic Differences between Cats and Dogs

Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (Vet Emergency and Critical Care; Medicine of Dogs)

Introduction:

Cats receiving anaesthesia are documented to have higher mortality rates and dogs. There may be many reasons for this disparity, including patient physiology, small patient size, difficulties in patient monitoring, differences in drug metabolism, the presence of underlying undiagnosed covert disease (such as hypertrophic cardiomyopathy), and the magnitude of peri-anaesthetic anxiety, among other factors.

These differences require that veterinarians and nurses approach anaesthesia in the cat with a different mindset to anaesthesia in the dog, so that appropriate monitoring and support measures are applied, to minimise anaesthetic risk.

Cats vs. Dogs: Key Differences

1. **Fasting before anaesthesia**
    a. Traditional fasting times of 6-12 hours are frequently recommended but are not evidence-based. Shorter fasting times have been associated with less gastro-oesophageal reflux in dogs, but results are conflicting among different studies.
    b. Gastric emptying time:
        i. The gastric emptying in the dog and cat usually begins within 15 minutes of ingestion of food and should be complete within 1-4 hours in the dog, and as little as 30-120 minutes in the cat.
        ii. The shorter gastric emptying time in cats may imply the requirement for a shorter fasting period prior to anaesthesia. However, other factors may influence the likelihood of gastroesophageal reflux in the cat, including:
            1. Anaesthetic and pre-anaesthetic drugs may influence the incidence of gastroesophageal reflux e.g. morphine, methadone and dexmedetomidine/medetomidine/xylazine all can increase the risk of gastroesophageal reflux in the cat
            2. Stress, meal size and low dietary moisture can slow gastric emptying rate in cats, increasing the risk of gastroesophageal reflux under anaesthesia
    c. Recommendations:
        i. There are currently no evidence-based recommendations for fasting in the cat prior to anaesthesia
        ii. Because of the risk of delayed gastric emptying due to a range of factors, the anaesthetic team should be prepared for vomiting or gastroesophageal reflux in any patient
        iii. At the clinician discretion, shorter fasting times of 3-4 hours may be advocated, as long as the final pre-anaesthetic meal is a small wet food feeding
        iv. Reduction of stress appears to offer potential benefits in normalization of gastric emptying. To this end, pre-clinic visit administration of gabapentin (50-100 mg PO) may be recommended, provided there are no contraindications
        v. Water should be made available until the time of premedication

2. Pre-Anaesthetic Assessment

a. Pre-anaesthetic testing should include thorough patient history review, physical examination, and laboratory and imaging assessments as appropriate
b. Many cats can be stressed at the time of physical examination and assessment, and this may be reflected in abnormal physical and laboratory findings, making interpretation of abnormal findings challenging. For example, clinical and laboratory findings that may result from either stress or underlying pathology may include
    i. Elevated blood and urine glucose levels
    ii. The presence of cardiac gallop or murmurs
    iii. The presence of tachycardia or bradycardia
    iv. The presence of hypertension
c. Testing for hyperthyroidism in middle-aged to older cats can be valuable. Hyperthyroidism affects up to 10% of cats aged 10 years and older, and can result in hypertension, hypertrophic cardiomyopathy, and cardiac arrhythmias
d. Abnormal cardiac findings on physical examination should be evaluated. Cats with cardiomyopathy are frequently asymptomatic. Cats with clinical examination findings of gallop or murmur are recommended to have echocardiography performed, as well as NT-ProBNP screening to facilitate diagnosis of cardiac disease.

3. Anaesthetic Equipment

a. Because of their small size and small lung capacity, cats are uniquely susceptible to deleterious side effects of inappropriate or malfunctioning anaesthetic equipment
b. Optimal anaesthetic equipment for cats includes
    i. Anaesthetic machine with precision out of circuit vaporiser
    ii. A non-rebreathing anaesthetic circuit with pressure manometer and safety pop-off relief valve. Alternatively, in cats larger than 3 kg, a paediatric rebreathing circuit with low-resistance tubing, and lightweight plastic rebreathing valves and minimal dead space may also be considered
    iii. Standard anaesthetic equipment including supplies for intravenous access, endotracheal intubation, and monitoring equipment (ECG, pulse oximeter, blood pressure (doppler), thermometer, oesophageal stethoscope, capnography etc.
c. The risk of high airway pressures, and associated barotrauma and lung damage is high in cats, because of their small lung volume (400 ml in a 3-5 kg cat), coupled with the high flow rates of oxygen required in non-rebreathing anaesthetic circuits (>200 ml/kg/minute; minimum flow rate 500 ml/minute). These two factors can lead to damage to a cats' airways in as little as 30 seconds, if the circuit is accidentally closed. As a result, essential safety features in feline anaesthesia should include:
    i. In-circuit manometer
    ii. Safety pop-off valve and/or a high-pressure alarm, placed in the expiratory limb of the non-rebreathing circuit, that emits a loud noise if circuit pressure become excessive.
d. The oxygen flush valve should never be used when a cat is connected to an anaesthetic circuit, as the flow rate can be as high as 40 litres/minute and can result in significant lung and airway damage.
e. Minimising anaesthetic circuit dead space is crucial in the cat, owing to their small size, which can lead to carbon dioxide rebreathing, reduced tidal volume as well as respiratory fatigue. Dead space should ideally be less than 2-3 ml/kg (less than 20% of tidal volume). Where possible, use of low-dead space endotracheal tube adaptors fir attachment to side-stream sampling lines should be used. Important sources of dead-space include:
    i. The patient end of breathing circuits
    ii. Side-stream capnograph adaptors
    iii. In-line capnographs – note that in-line capnographs increase circuit resistance also.

iv. Elbow adaptors – Note that a single elbow adaptor can add up to 8 ml of dead space

f. Minimise circuit resistance – to reduce the effort required for the cat to breathe.
   i. In-line capnographs increase anaesthetic circuit resistance.
   ii. The diameter of any capnograph adaptor should always exceed the internal diameter of the patient endotracheal tube to reduce circuit resistance

4. Anaesthetic Circuits and Oxygen Flow Rates

a. The small patient size, and the use of non-rebreathing anaesthetic circuits means oxygen flow rates and rebreathing bag sizes are quite different to those used in rebreathing systems for dogs over 7 kg.
b. Oxygen flow rates:
   i. Non-rebreathing circuits – Recommended oxygen flow rate > 200 ml/kg/minute with a minimum flow rate of 500 ml/minute
   ii. Rebreathing circuits –
      1. First 15 minutes: 2-3 litres/minute
      2. Maintenance: 0.5 litre/minute
c. Rebreathing bag size
   i. 0-5kg: 500 ml
   ii. >5kg: 1 litre

5. Drug metabolism

a. Kittens up to 5 months of age have variable (reduced) ability to metabolise drugs via the hepatic P450 enzyme systems, leading to profound or prolonged effect of sedative and anaesthetic medications metabolised in the liver.
b. Senior/geriatric cats older than 10 years of age:
   i. Have a higher risk of anaesthetic-related death (independent of ASA status)
   ii. Are more sensitive to the respiratory and cardiovascular effects of sedative and anaesthetic drugs
   iii. Have limited compensatory responses to homeostatic changes e.g. cardiac output etc.

6. Blood volume

a. The blood volume of the cat is smaller than that of the dog, as a percentage of bodyweight.
b. Fluid therapy boluses should be dosed at 5-7 ml/kg, given over 10 minutes
c. Anaesthetic fluid rates for maintenance fluid therapy are 2.5-3 ml/kg/hr
d. Pulmonary oedema may develop with prolonged fluid therapy at rates higher than maintenance rates, including traditional "surgical fluid rates" of 5 ml/kg/hr and above.

7. Emergency and critical illness anaesthesia

a. Cats have unique physiology – particularly notable when they suffer shock, hypovolaemia, inanition, and cardiac, respiratory and abdominal disease
b. Clinical signs of a cat in shock are
   i. Bradycardia
   ii. Hypothermia
   iii. Hypotension
c. Cats have limited ability to contract their spleen during haemorrhage. Splenic volume is also smaller than in the dog (1.5 ml/kg vs. 3.5 ml/kg)

d. Lack of nutritional support in acute illness can result in rapid development of a catabolic state, leading to development of hepatic lipidosis, as well as thiamine deficiency
e. Cats presenting with seizures require thorough evaluation for secondary (intra-cranial and extracranial diseases) illness, as idiopathic epilepsy is uncommon in the cat
f. Acute heart failure is characterised most frequently by respiratory distress, +/- dysfunction of a limb (due to thromboembolism), but can also be characterised by abdominal organ dysfunction and abdominal pain
g. Acute abdominal disease may result in abdominal pain in less than 35% of cases, with some cats showing respiratory distress as the primary presenting sign – necessitating a thorough clinical evaluation of the patient using diagnostic imaging, blood tests/urinalysis etc.

8. Monitoring

a. Monitoring the feline patient under anaesthesia can be more challenging than in the dog, owing to the small patient size, and the impacts on sensitivity of monitoring equipment.
b. Optimum monitoring equipment for feline anaesthesia includes
    i. Clinical observation of physical parameters and anaesthetic depth assessment
    ii. Heart rate – particularly valuable with oesophageal stethoscope
    iii. Respiratory rate and effort – does not indicate effective ventilation or oxygenation. Tidal volume is difficult to assess in the feline patient due to small patient size – necessitating instrumentation such as pulse oximetry or blood gas analysis to verify oxygen saturation
    iv. Temperature
    v. Pulse palpation
    vi. Pulse oximetry – reliable when signal quality if good and pulse rate accurate
    vii. ECG – small patient size often results in poor ECG signal
    viii. Capnography – helpful in determining if alterations in fresh gas flow are required. Inspired carbon dioxide in a non-rebreathing circuit should not exceed 5 mm Hg.
    ix. Blood pressure
        1. Ideal cuff width affects accuracy of reading. Optimum cuff width is 40% of the limb circumference
        2. Doppler blood pressure is more reliable than oscillometric blood pressure devices
        3. Normal systolic blood pressure in the cat is 100-120 mm Hg; hypotension is classified as systolic arterial pressure <90 mm Hg

9. Laryngeal spasm

a. Cats have a very sensitive laryngospasm reflex in response to laryngeal stimulation or irritation, which can result in difficult endotracheal intubation.
b. Placement of 1-2 drops of 2% lidocaine (20 mg/ml) on the arytenoid cartilages, 60-90 seconds prior to intubation attempts will reduce the risk of severe laryngospasm during intubation attempts

10. Airway pressures

a. Endotracheal cuff inflation should be at the lowest volume and pressure required.
b. Inflate the endotracheal tube cuff using 0.5 ml increments of air until no leak can be heart when the rebreathing bag is squeezed to a pressure of no greater than 16-18 cm water
c. Endotracheal cuff pressure measurement devices are also available e.g. Tru-Cuff syringes. Optimum cuff pressure is 20-24 cm water pressure in the cat.

Conclusion:

Feline anaesthesia presents several challenges that are distinct from canine anaesthesia. Whilst this list is not exhaustive, it provides the basis for consideration of several key areas that require conscious thought and evaluation when performing anaesthesia in the cat. It is important to tailor each anaesthetic to the individual patient, and to optimise patient monitoring from the pre-anaesthetic period, through to full patient recovery – with knowledge that the highest period of anaesthetic mortality in cats is in the anaesthetic recovery period (up to 60%) – irrespective of ASA patient risk score.

References and Suggested Reading

1. Grubb, Tamara, Jennifer Sager, James S. Gaynor, Elizabeth Montgomery, Judith A. Parker, Heidi Shafford, and Caitlin Tearney. "2020 AAHA anesthesia and monitoring guidelines for dogs and cats." Journal of the American Animal Hospital Association 56, no. 2 (2020): 59-82.
2. Davis, Harold, Tracey Jensen, Anthony Johnson, Pamela Knowles, Robert Meyer, Renee Rucinsky, and Heidi Shafford. "2013 AAHA/AAFP fluid therapy guidelines for dogs and cats." Journal of the American Animal Hospital Association 49, no. 3 (2013): 149-159.
3. Robertson, Sheilah A., Susan M. Gogolski, Peter Pascoe, Heidi L. Shafford, Jennifer Sager, and Gregg M. Griffenhagen. "AAFP feline anesthesia guidelines." Journal of feline medicine and surgery 20, no. 7 (2018): 602-634.
4. Steagall, Paulo V., Sheilah Robertson, Bradley Simon, Leon N. Warne, Yael Shilo-Benjamini, and Samantha Taylor. "2022 ISFM consensus guidelines on the management of acute pain in cats." Journal of Feline Medicine and Surgery 24, no. 1 (2022): 4-30.
5. Rodan, Ilona, Nathalie Dowgray, Hazel C. Carney, Ellen Carozza, Sarah LH Ellis, Sarah Heath, Lee Niel, Kelly St Denis, and Samantha Taylor. "2022 AAFP/ISFM cat friendly veterinary interaction guidelines: approach and handling techniques." Journal of Feline Medicine and Surgery 24, no. 11 (2022): 1093-1132.
6. Matthews, Nora S., Thomas J. Mohn, Mingyin Yang, Nathaniel Spofford, Alison Marsh, Karen Faunt, Elizabeth M. Lund, and Sandra L. Lefebvre. "Factors associated with anesthetic-related death in dogs and cats in primary care veterinary hospitals." Journal of the American Veterinary Medical Association 250, no. 6 (2017): 655-665.
7. Garcia, R. S., P. C. Belafsky, A. Della Maggiore, J. M. Osborn, B. H. Pypendop, T. Pierce, V. J. Walker, A. Fulton, and S. L. Marks. "Prevalence of gastroesophageal reflux in cats during anesthesia and effect of omeprazole on gastric pH." Journal of veterinary internal medicine 31, no. 3 (2017): 734-742.

Circulatory Shock in the Cat

Dr. Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (VECC; Medicine of Dogs)

Why Talk About Shock in Cats?

Despite the large numbers of studies investigating both physiology and treatment of shock syndromes in both people and dogs, there is a relative paucity of studies on this topic in cats[1]. Let's begin with describing shock in cats. The clinical syndrome of shock in cats[2] has been characterised by 3 things…

1. Hypotension
2. Bradycardia
3. Hypothermia

This contrasts with the situation in dogs, where we tend to see – at least in hypovolaemic shock – a transition from stage 1 shock – in which hypertension, tachycardia and bounding pulses are seen– to stage 2 shock – in which tachycardia, falling blood pressure, and weaker pulses are seen –to end-stage shock, characterised by tachycardia or bradycardia, hypothermia and low blood pressure[1].

Why the difference? What is going on in cats, that is so different to dogs?

The answer is multifactorial but is thought to principally lie in the acute physiological responses to hypovolaemia or haemorrhage in the cat, that differ from those in the dog.

To help explain this, let's briefly review the response to hypovolaemia in the dog[1-3]–

- Stage 1 shock (compensatory shock)
  - Acute hypovolaemia results in activation of the sympathetic nervous system, leading to
    - Tachycardia
    - Positive inotropy
    - Vasoconstriction in gastrointestinal, liver, renal and skin vasculature
    - Vasodilatation in heart, lungs, brain and muscle

  This physiological response is manifested clinically with tachycardia, bounding pulses, heightened mentation and pink mucous membranes with rapid capillary refill time.

- Stage 2 shock
  - Compensatory shock is very short-lived in most patients, and leads to ATP-depletion in vasoconstricted tissues, with progressive vasodilatation in the gastrointestinal tract, skin, liver and kidneys – massively increasing vascular volume. Clinically, this is manifested as a patient with tachycardia, weak pulses, pale mucous membranes, and prolonged capillary refill time.
- Stage 3 shock
  - As stage 2 shock progresses, vascular endothelial damage results from tissue hypoxia caused by poor tissue perfusion. Coagulation activation ensues, followed by widespread thrombosis of micro-vasculature. Organ failure results. Clinically, this is manifested as bradycardia, hypothermia, organ dysfunction (vomiting, diarrhoea, acute kidney failure, arrhythmias, etc.) and death.

Whilst stage 1 shock may occur in cats, it is rarely seen clinically[2]. Likewise, the "classic" decompensatory characteristics of stage 2 shock are rarely noted in the cat with bradycardia and hypotension being the predominant clinical signs[2].

Why Are Symptoms of Shock in Cats Different?

1. The Feline Ventricle
    a. Within the wall of the ventricle, the cat has mechanical and chemical volume receptors that respond to distension
        i. Activation of these mechanoreceptors in hypovolaemia reduces heart rate via a cardiac inhibitory reflex, allowing ventricular contraction only when normal ventricular filling pressures are reached in diastole[4].
2. The Feline Atria
    a. The feline atria have feline-specific type-B atrial volume receptors that respond to atrial distension[4]
        i. Feline-specific type-B receptors discharge during atrial filling and higher pressures, and lead to increases in heart rate; hypovolaemia reduces these receptor stimulation, with bradycardia being the predominant effect.
        ii. In both cats and dogswith hypovolaemia, sympathetic nervous system activity increases, which leads to shunting of blood into cardiac and respiratorycirculation , increasing atrial distension. In cats, however,there is an inhibition of the cardiac sympathetic efferent nerve activity following stimulation of type B atrial receptors, as atrial pressures rise.
        iii. In hypovolaemia,decreased atrial stretch activity related toa ction of the atrial volume receptors occurs before carotid sinus and aortic baroreceptors and leads to hypothalamic-mediated increases in vasopressin concentrations, increasing blood volume.
3. Noradrenaline and Hypothermia
    a. Noradrenaline release from sympathetic nerves leads to a centrally-mediated hypothermia[5,6]
        i. Hypothermia reduces noradrenaline release in the CNS and in the heart, blunting sympathetic nervous system responses.
    b. Parasympathetic Nervous Outflow
        i. Increased parasympathetic outflow and inhibition of sympathetic outflow, occurs by the Benzold-Jarisch reflex (BJ reflex) in hypothermic cats, producing bradycardia and vasodilatation[7].

As shock progresses, prolonged tissue hypoxia leads to extensive cellular death, organ failure and eventually, patient death (stage 3 – or end-stage shock)

The Causes of Shock in the Cat

The causes of shock in the cat are broadly[2,3]–

1. Hypovolaemic shock
    a. Caused by fluid loss from conditions such as
        i. gastrointestinal losses
        ii. Third-space losses
        iii. Inadequate fluid intake
        iv. Haemorrhage
        v. Trauma
        vi. Systemic inflammation
2. Cardiogenic shock
    a. Caused by cardiac conditions, including
        i. Cardiomyopathy
        ii. Myocardial ischaemia
        iii. Mitral valve insufficiency
        iv. Cardiac arrhythmias
        v. Sepsis
3. Distributive shock
    a. Caused typically by severe illness or infection, among other causes, including
        i. SIRS
        ii. Sepsis
        iii. Severe liver disease
        iv. Anaphylaxis
        v. Spinal cord injury
4. Obstructive shock
    a. Results from obstruction to venous return to the heart, or increased afterload. Causes include
        i. Pneumothorax
        ii. Venous thrombosis
        iii. Cardiac tamponade
        iv. Caval syndrome (neoplastic occlusion, heartworm)
        v. Positive-pressure ventilation

Treatment of Feline Circulatory Shock

Aside from basic supportive care, including provision of oxygen therapy, analgesia etc., the treatment of shock in the cat may be divided into four (4) phases[1-3, 8] :

- Phase 1: Determine the type of shock present
    - Assess the following todetermine the type of shock present, as this will assist in directing treatment
        - History – the presence of trauma, bite wounds, gastrointestinal symptoms, potential toxin exposure, medical history may provide vital information that can inform the veterinary team on possible causes of shock.
        - Clinical examination – physical examination of the patient may reveal the characteristic signs of shock (bradycardia, hypotension and hypothermia), but may also reveal other signs that may help determine aetiology, including
            - Hydration status
            - Abdominal pain
            - Jaundice
            - Wounds
            - Haemorrhage
            - Cavity fluid accumulation
            - Pneumothorax or other pleural space disease
            - Cardiac murmurs, gallop rhythms, jugular pulse etc.
        - Diagnostic evaluation – diagnostic test selection will vary, depending on the patient signalment and clinical examination, but may include the following
            - PCV/TP assessment
            - Blood glucose
            - Blood pressure
            - Serum biochemistry and electrolyte evaluation
            - Complete blood count
            - Urine analysis
            - FAST scan (abdominal, thoracic, lung)
            - Echocardiography
            - Abdominal ultrasonography
            - Blood gas analysis
            - Radiography (chest, abdomen, fracture assessment, etc.)
- Phase 2: Fluid selection
    - Fluid selection in the treatment of shock in the cat is made based on clinical status, combined with laboratory assessment of hematocrit and plasma protein, along with electrolyte and acid/base analysis.
    - Crystalloid fluids will distribute within the entire extracellular space following administration – meaning that up to 75% of the administered crystalloid fluid will distribute to the interstitial spaces, leaving as little as 25% within the intravascular space. These fluids are useful in managing acute hypovolaemia but it is important to note in some diseases, that redistribution of administered fluid into interstitial spaces may lead to patient compromise, e.g., pulmonary contusions – meaning judicious use is necessary.

- Colloid fluids are suspensions of large molecular weight compounds (glucose polymer, starches proteins or gels). These large molecules do not readily pass through intact capillary membranes – meaning more of the administered solution will remain within the intravascular space. These fluids can be useful in managing acute hypovolaemia in patients with intact capillary membranes, e.g., acute hypovolaemia. However, starch-based colloids should be avoided in patients with sepsis, systemic inflammation and potentially, patients with significant increases in capillary permeability, as, at least in humans with sepsis, they are associated with increased risk of kidney damage and mortality.
- Fluid therapy in most cats initially presenting with shock will involve judicious use of crystalloids, with occasional use of small bolus colloids in non-septic patients to support crystalloid use if required.

- Phase 3: Determine resuscitation end-points
  - Resuscitation end-points provide a set of values (heart rate, blood pressure, temperature etc.) that are targets for resuscitation (so-called "goal-directed resuscitation"), following which, the rate of fluid therapy is reduced to maintenance rates, plus amounts sufficient to correct hydration deficits and ongoing patient fluid loss.
  - Whilst traditional goal-directed therapy has reduced mortality rates in humans with sepsis and trauma, it is important to individualise patient treatment, so that optimum outcome is achieved[9].
  - Bradycardia, hypothermia and hypotension are associated with worse outcome in cats[10].
  - Appropriate resuscitation end-points in the cat with shock include
    - Systolic arterial pressure = 100-120 mm Hg
    - Heart rate: 160-180/min
    - Normalisation of body temperature. Note that this parameter should be corrected following commencement of fluid resuscitation and should be raised to an initial level of 36.7 degrees over the first30 -40 minutes of resuscitation. Hypotension will begin to resolve as body temperature rises.
  - In addition, patients with blunt force abdominal trauma, abdominal haemorrhage, pulmonary contusions or traumatic brain injury may benefit from administration of the anti-thrombolytic medication, tranexamic acid.
- Phase 4: Administer fluid therapy
  - Small volume resuscitation is the technique of choice and involves administering 7 ml/kg of isotonic crystalloid fluid over 10 minutes, followed by patient re-evaluation, to determine if subsequent fluid boluses are required. This process is continued for 20-30 minutes initially. A single bolus of isotonic crystalloid may be substituted for a 3 ml/kg bolus of hydroxy-ethyl starch, if desired, in non-septic patients, and in patients without kidney compromise, to prolong the intravascular effectiveness of the crystalloid fluid[11].

The Patient that Does Not Respond to Therapy

Patients with circulatory compromise may sometimes fail to respond to the aforementioned treatment[2]. Failure to respond to therapy to treat shock should prompt investigation to determine the cause. Common causes of persistent circulatory failure in the cat are outlined below:

- Inadequate intravascular volume
- Blood loss
- Third space losses (gastrointestinal tract, body cavity loss, etc.)
- Pain
- Heart disease
    - Cardiac tamponade
    - Heart failure
    - Arrhythmias
- Blood gas or electrolyte abnormalities
- Hypoglycaemia
- Hypocortisolaemia
- Anaemia
- Sepsis
- Pleural space disease
- CNS disease

Knowledge of the causes of poor response to treatment for shock can assist in selection of diagnostic tests to determine the aetiology in a given patient, so that correction of the underlying cause can commence.

Conclusion

Shock in the feline patient presents with a distinct, and separate clinical syndrome to the dog. Understanding the reasons for this, the potential causes, and having a clear outline of how to treat shock in the cat allows timely and appropriate intervention – both of which will reduce mortality and morbidity.

References:

1. Davis H, Jensen T, Johnson A, Knowles P, Meyer R, Rucinsky R, Shafford H. 2013 AAHA/AAFP fluid therapy guidelines for dogs and cats. Journal of the American Animal Hospital Association. 2013 May;49(3):149-59.
2. Rudloff E, Kirby R. Feline Circulatory Shock. In August's Consultations in Feline Internal Medicine, Volume 7 2016 Jan 1 (pp. 843-858). WB Saunders.
3. Pachtinger GE. Hypovolemic shock. Clinician's Brief. 2014:13-6.
4. Fahim M. Cardiovascular sensory receptors and their regulatory mechanisms. Indian J Physiol Pharmacol 203 47 (2): 124-146
5. Myers RD, Beleslin DB, Rezvani AH. Hypothermia: role of 1-and α2-noradrenergic receptors in the hypothalamus of the cat. Pharmacology Biochemistry and Behavior. 1987 Feb 1;26(2):373-9.
6. Kawada T, Kitagawa H, Yamazaki T, Akiyama T, Kamiya A, Uemura K, Mori H, Sugimachi M. Hypothermia reduces ischemia-and stimulation-induced myocardial interstitial norepinephrine and acetylcholine releases. Journal of Applied Physiology. 2007 Feb;102(2):622-7.
7. Mark AL. The Bezold-Jarisch reflex revisited: clinical implications of inhibitory reflexes originating in the heart. Journal of the American College of Cardiology. 1983 Jan 1;1(1):90-102.
8. Perner A, Haase N, Guttormsen AB, Tenhunen J, Klemenzson G, Åneman A, Madsen KR, Møller MH, Elkjær JM, Poulsen LM, Bendtsen A. Hydroxyethyl starch 130/0.42 versus Ringer's acetate in severe sepsis. New England Journal of Medicine. 2012 Jul 12;367(2):124-34.
9. Roberts JK, Disselkamp M, Yataco AC. Oxygen delivery in septic shock. Annals of the American Thoracic Society. 2015 Jun;12(6):952-5.
10. Silverstein DC, Wininger FA, Shofer FS, King LG. Relationship between Doppler blood pressure and survival or response to treatment in critically ill cats: 83 cases (2003–2004). Journal of the American Veterinary Medical Association. 2008 Mar 15;232(6):893-7.
11. Muir WW. A new way to monitor and individualize your fluid therapy plan. Vet Med (2):76-82. 2013.

Fluid Therapy in Traumatic Brain Injury

Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (Veterinary Emergency and Critical Care; Medicine of Dogs)

Introduction

Traumatic brain injury is significantly associated with both morbidity and mortality in dogs, with mortality rates reported in excess of 24% in veterinary studies (Pigott, Rudloff 2021).

Patents with traumatic brain injury frequently present with comorbidities alongside their brain injury (Kuo et al. 2018), including (but not limited to)

- Circulatory shock
- Haemorrhage
- Coagulopathy
- Pulmonary injury
    - Pulmonary contusions
    - Pneumothorax
    - Pleural fluid accumulation
    - Thoracic wall disease (fractured ribs, intercostal muscle tears etc.)
    - Diaphragmatic hernia
- Abdominal injury
    - Liver contusions
    - Haemoabdomen
    - Abdominal hernia
    - Ruptured urinary tract, kidney avulsion etc.
- Orthopaedic injury
- Skin wounds

The ultimate goal of managing the patient with traumatic brain injury is to provide optimal conditions for brain perfusion, which includes supporting normal neuronal function, cardiovascular function and respiratory tract function (Sundstrøm et al 2020; Vella et al. 2017). The rationale for this broad approach is that normal neuronal function optimised by controlling cerebral blood flow – which is determined by (Neus et al. 2019; Sande et al. 2010; Dos Santos et al. 2018):

1. PCO_2 - A change in cerebral CO_2 is the main and most sensitive regulator of cerebral blood flow. Carbon dioxide is a potent cerebral vasodilator, regulated by changes in cerebral pH, and leads to an increase in cerebral blood volume

2. PO_2 - normal oxygen tension in cerebral circulation maintains vascular tone in cerebral blood vessels

3. Blood pressure autoregulation – The cerebral circulation is regulated in such a way that a constant total cerebral blood flow is maintained under varying conditions. This is called autoregulation. Autoregulation maintains cerebral blood flow at a constant level between a mean arterial blood pressure of between 50-150mmHg, outside of which, cerebral blood flow becomes linearly related to blood pressure. Autoregulation is less efficient in states of ischaemia, hypoxia, hypercabia, and increasing blood viscosity, as may be encountered in patients suffering severe dehydration and haemoconcentration, or polycythaemia Vera.

4. The level of neuronal stimulation – Increasing neuronal activity and hence metabolic rate will increase cerebral blood flow, for example, with seizures, hyperthermia etc.

5. Systolic blood pressure. Systolic blood pressure outside the normal range – particularly in the hypotensive range, is associated with significantly worse neurological outcome in humans, which has lead to the recommendation to maintain systolic arterial pressure in the range 100-120 mm Hg

The Problems with Traumatic Brain Injury and Brain Homeostasis

A traumatic insult to the brain causes injury to key components responsible for normal brain homeostasis.

Increased permeability of the blood brain barrier results in increased movement of inflammatory cells and protein into the brain parenchyma, which establishes an inflammatory response within the brain which further increases vascular permeability and fluid loss, causing both vasogenic and cytotoxic oedema. The presence of oedema in brain tissue can result in increased intra-cranial pressure, and regional vascular compression in the brain, which can further compromise brain blood flow, cerebral perfusion and oxygen delivery (Pigott, Rudloff 2021; Vella et al. 2017)

Fluid therapy is an essential component of the management of TBI patients. The goal of fluid therapy is to maintain adequate cerebral perfusion and oxygenation while avoiding complications such as cerebral edema and increased intracranial pressure (ICP). However, the optimal approach to fluid therapy in TBI patients requires a balanced and integrated approach, that must also include adequate support of the respiratory tract, so that carbon dioxide and oxygen concentrations in blood are normalised as much as possible; along with control of cerebral metabolic rate.

Fluid Therapy in Patients with Traumatic Brain Injury

Intravenous fluid therapy is a key component in the management of traumatic brain injury – principally in the treatment of hypovolaemia. The general recommendation is that circulation should be supported to prevent hypotension and to maintain cerebral blood flow, as both of these factors are positively correlated with improved survival and neurological outcome. However, there is considerable controversy regarding fluid type, fluid volume, appropriate end-points and strategy in fluid therapy in patients with traumatic brain injury (Pigott, Rudloff 2021).

Results of human and experimental animal studies have established the following goals of fluid therapy in traumatic brain injury (Vella et al. 2017; Epstein et al. 2014)

1. Maintenance of systolic arterial blood pressures in the range 100-120 mm Hg
2. Avoidance of systolic arterial blood pressures lower than 90 mm Hg, as this is associated with worse neurological outcome
3. Avoidance of coagulopathy (trauma-associated coagulopathy)
4. Optimization of oxygen delivery to cerebral tissues

Further to establishment of these treatment goals is the requirement to mitigate the development or exacerbation of acute traumatic coagulopathy in the setting of traumatic brain injury, which is associated with increases in patient mortality and worse neurological outcome (Epstein et al. 2014)

Isotonic Crystalloid Fluid Resuscitation

Isotonic fluid resuscitation in patients with traumatic brain injury is not associated with survival advantage in most human and experimental animal studies (Santry et al. 2010). Additionally, when compared to synthetic colloids, animals resuscitated with 0.9% NaCl or lactated Ringer's solution required larger volumes of fluid to maintain haemodynamic end-points and developed progressive acidosis (Gantner et al. 2014).

In experimental studies on animals with haemorrhagic shock, isotonic crystalloid-resuscitation fluids were associated with the following outcomes, when compared to resuscitation with fresh frozen plasma or hydroxy-ethyl starch (Chowdhury et al. 2013):

- Lower cerebral perfusion pressure
- Higher intra-cranial pressure
- Lower mean arterial blood pressure
- Lower brain oxygenation
- More brain oedema

- Larger brain lesion size
- Higher brain inflammation
- Greater degree of neurological impairment
- Slower rates of neurological recovery

Finally, in theory, hyponatraemic fluids, such as lactated Ringer's solution should be avoided in the resuscitation of traumatic brain injury, since they may produce an increase an increased osmolar gap that may favour brain water accumulation (Pigott, Rudloff 2021), although this is not reflected in clinical studies, and balanced solutions lead to lower incidence of hyperchloraemic metabolic acidosis (Van Aken et al. 2012; Selmer et al. 2018)

Hyperosmolar Fluid Resuscitation

Hyperosmolar fluids may confer advantages over iso-osmolar fluids in traumatic brain injury resuscitation, by producing an osmotic fluid shift into the intravascular space and reducing brain oedema (Marchesini et al. 2023).

Clinical trials in humans evaluating infusion of 7.5% sodium chloride in patients with traumatic brain injury resuscitation failed to show improvement in survival.

In experimental canine studies of traumatic brain injury and haemorrhage, animals resuscitated with 3% sodium chloride had higher cerebral perfusion pressure, lower intracranial pressure, less cerebral oedema and earlier return of ocular reflexes when compared to animals resuscitated with lactated Ringer's solution.

Another study in a rat model of traumatic brain injury and haemorrhage, animals resuscitated with 7.5% sodium chloride had improved neuronal survival and behavioural recovery compared to animals resuscitated with 0.9% sodium chloride.

At this point in time, a strong preference for hypertonic saline is based on a small number of studies without strong evidence of long-term improvement (Farrokh et al. 2019)

Synthetic Colloid Fluid Resuscitation

Fluid resuscitation with synthetic colloids in patients with traumatic brain injury has been evaluated principally in experimental studies, with the following findings (Sekhon et al 2011; Chowdbury et al. 2013)):

- Smaller resuscitation volume requirement with HES than saline to reach resuscitation end-points
- Less cerebral oedema following resuscitation with HES or blood when compared to 0.9% or 0.45% sodium chloride
- Less cerebral oedema and brain lesion size following resuscitation with HES when compared to 0.9% sodium chloride. However, fresh frozen plasma reduced oedema and brain lesion size to a greater degree than HES.

Clinical trials in traumatic brain injury in humans have failed to demonstrate a change in mortality with the use of HES over crystalloid therapy.

Natural Colloid (Plasma or Albumin) Resuscitation

A large randomized trial comparing infusion of hyperosmolar (4%) albumin to 0.9% sodium chloride in the treatment of haemorrhagic shock (SAFE trial) in the subset of patients with traumatic brain injury found that albumin-treated patients had higher mortality than saline-treated patients. Other studies have shown that treatment with hyperosmolar albumin is associated with increased brain oedema over patients treated with other fluid resuscitation strategies (Van Aken et al. 2012; Gantner et al. 2014)

Infusion of fresh frozen plasma appears to confer improved outcomes over crystalloids and synthetic colloids in experimental studies, with the following findings (Halaweish et al. 2015; Chang et al. 2020):

- Lower infusion volumes required to achieve resuscitation end-points

- Improved vascular endothelial glycocalyx and blood-brain barrier health
- Improved cerebral perfusion pressure
- Higher mean arterial blood pressure
- Improved brain tissue oxygenation
- Reduced brain oedema
- Reduced brain lesion size
- Reduced mitochondrial dysfunction
- Reduced glutamate-mediated excitotoxic secondary brain injury
- Less neurological impairment
- Faster rates of neurological recovery

Clinical data is conflicting (Pigott, Rudloff 2021), with one small study evaluating plasma use in patients with severe trauma showing worse neurological and mortality outcomes with fresh frozen plasma administration, and another small study showing increased coagulopathy and worse neurological outcome, but no mortality difference. Both studies were likely underpowered to accurately predict outcome based on intervention, and both studies enrolled patients having had a variety of fluid types as pre-treatments prior to being assigned to plasma or crystalloid treatment groups.

A larger randomized study of human patients with traumatic brain injury evaluated plasma resuscitation in addition to standard care resuscitation versus no plasma with standard resuscitation (Gruen et al. 2020). Patients with plasma treatment had lower mortality at 30 days, less crystalloid fluid requirement, lower red cell transfusion requirement, lower 24-hour mortality and lower vasopressor requirement within the first 24-hours of treatment. The plasma-treated patients did, however, have a higher incidence of multiple organ failure, longer ICU stays. The study also found:

- Plasma transfusion was of greatest benefit in in the most severely injured patients
- Plasma transfusion was of greatest benefit if given early in treatment versus later in treatment

The benefit of early plasma transfusion has also been documented in a retrospective study on traumatic brain injury in humans (Chang et al. 2017)

Fluid Resuscitation Technique in Traumatic Brain Injury

Results of experimental animal studies, and clinical studies in humans, the following observations on fluid resuscitation techniques in traumatic brain injury are noted:

- There is evidence than even transient periods of hypotension (SAP <90 mm Hg) is associated with worse neurological outcome, in both a time-dependent and severity-dependent manner.
- Experimental studies of traumatic brain injury in the setting of haemorrhage that are resuscitated with large-volume crystalloid resuscitation have higher mortality and worse neurological lesions than those patients in which resuscitation was delayed until haemorrhage control was achieved (Vrettos et al. 2016).
- A step-wise infusion of fluids is associated with less brain oedema than large bolus infusions of fluids.
- 3% sodium chloride may provide superior outcomes than isotonic crystalloids, including lower brain water content, less oedema and improved neurological outcome
- Hydroxy-ethyl starch and fresh frozen plasma infusion result in lower fluid volumes required to achieve resuscitation end-points, as well as less cerebral oedema and brain lesion size
- Fresh frozen plasma is also associated with improved blood flow, neurological outcome and smaller lesion size following traumatic brain injury, especially if administered early in resuscitation protocols

Clearly, there is no fluid strategy, based on currently available evidence, that will likely prove ideal in every patient. However, the following broad recommendations may be made:

1. Patients should have co-morbidities thoroughly assessed and managed as appropriate

2. Respiratory tract support should include therapy such as oxygen supplementation, +/- ventilation therapy to optimise oxygen and carbon dioxide concentrations in arterial circulation
3. Anticonvulsant therapy +/- ketamine infusion should be used to control excess neuronal stimulation and to mitigate glutamine-mediated neuronal dysfunction
4. Control of co-existing haemorrhage should be attempted. The use of hypotensive resuscitation techniques in the presence of traumatic brain injury should generally be avoided except in the context of acute, uncontrollable haemorrhage, in which setting, treatment should include concerted efforts to control haemorrhage, coupled with early transfusion therapy and cautious use of hypotensive resuscitation. Once haemorrhage has been controlled, fluid resuscitation should be attempted to raise systolic arterial blood pressure to - or slightly above 90 mm Hg (Palmer 2017; Vrettos et al. 2016)
5. Fluid therapy should be provided to attain a systolic arterial pressure of greater than 90 mm Hg using the following approach
    a. Hypertonic saline 3% @ 3 ml/kg bolus over 10 minutes, repeated 2-3 times over 30-60 minutes followed by infusion of 0.9% NaCl at maintenance fluid rates
    b. Synthetic colloids such as hydroxy-ethyl starch may be combined with hypertonic saline at a dose of 3 ml/kg over 10 minutes, given once or twice over 10 minutes in the first 30 minutes of treatment only.
    c. Fresh frozen plasma may be administered at a dose of 3 ml/kg over 10 minutes, repeated 2-4 times over the first 30-40 minutes of treatment, followed by 0.9% NaCl treatment at maintenance fluid rates

Deteriorating Clinical Signs

Deteriorating clinical status can be the result of progression or development of co-morbidities, including respiratory and cardiovascular abnormalities (including haemorrhage); and increasing intra-cranial pressure. Detection of deteriorating clinical status therefore requires the clinician evaluate the whole patient, so that key abnormalities are detected, and appropriate treatment administered e.g. thoracocentesis in the patient with pleural space disease; ventilation therapy in the hypoventilating patient etc.).

Detection of central nervous system deterioration associated with increased intra-cranial pressure can ben managed using hyperosmolar therapy – although there is no strong evidence regarding the most appropriate agent, administration rate (bolus vs. continuous infusion) or timing (Vella et al. 2016; Pigott, Rudloff 2021; Jagnnatha et al 2016). A single study in dogs and cats evaluating mannitol and 3% NaCl showed both agents reduced intracranial pressure and improved cerebral perfusion pressure for a short period of time following administration (Ballocco et al. 2019).

A summary of findings in clinical trials involving hypertonic saline and mannitol include (Pigott, Rudloff 2021):

- There is some evidence that higher doses of mannitol may b associated with greater reductions in intra-cranial pressure than lower doses, with less rebound effect
- Continuous infusion of hypertonic saline may be associated with improved outcome, however sustained elevations in sodium and chloride content can be associated with worse clinical outcome in critically ill patients with SIRS and sepsis
- Mannitol may reduce concentrations of reactive oxygen species in previously hypoxic tissues
- No clear advantage exists between hypertonic saline and mannitol for adjunctive management of increased intra-cranial pressure
- Neither agent is associated with improved survival, despite producing reductions in intracranial pressure.

Conclusion

Fluid therapy recommendations in traumatic brain injury remain controversial – largely due to a lack of clear evidence in the clinical setting, coupled with conflicting results of experimental studies. Additionally, response to treatment can be complicated by the presence of comorbidities, including acute haemorrhage.

Clear recommendations are difficult to provide. Saline-based fluids are possibly more beneficial than solutions such as lactated Ringer's solution, with 3% hypertonic saline possibly conferring benefits over isotonic saline for initial resuscitation. Early plasma transfusion may confer improved outcome, reducing coagulopathy, and providing a colloid solution that minimises fluid loss into 3rd spaces, whilst potentially modulating glycocalyx loss, among other benefits (Pigott, Rudloff 2021; Maegele 2013; Epstein et al. 2014).

Further research will assist the clinician in making better informed treatment decisions regarding fluid therapy in traumatic brain injury.

References:

Pigott A, Rudloff E. Traumatic brain injury—a review of intravenous fluid therapy. Frontiers in Veterinary Science. 2021 Jul 9; 8:643800.

Kuo, Kendon W., Lenore M. Bacek, and Amanda R. Taylor. "Head trauma." Veterinary Clinics: Small Animal Practice 48, no. 1 (2018): 111-128.

Sundstrøm T, Grände PO, Luoto T, Rosenlund C, Undén J, Wester KG, editors. Management of severe traumatic brain injury: evidence, tricks, and pitfalls. Springer Nature; 2020 Jun 8.

Elias, Neus, Ana-Maria Rotariu, and Tobias Grave. "Traumatic brain injury in dogs and cats." Companion Animal 24, no. 9 (2019): 480-487.

Sande A, West C. Traumatic brain injury: a review of pathophysiology and management. Journal of veterinary emergency and critical care. 2010 Apr;20(2):177-90.

Dos Santos, L. O., G. G. Caldas, C. R. O. Santos, and D. B. Junior. "Traumatic brain injury in dogs and cats: a systematic review." Veterinarni medicina 63, no. 8 (2018): 345-357.

Vella MA, Crandall ML, Patel MB. Acute management of traumatic brain injury. Surgical Clinics. 2017 Oct 1;97(5):1015-30.

Epstein, Daniel S., Biswadev Mitra, Gerard O'Reilly, Jeffrey V. Rosenfeld, and Peter A. Cameron. "Acute traumatic coagulopathy in the setting of isolated traumatic brain injury: a systematic review and meta-analysis." *Injury* 45, no. 5 (2014): 819-824.

Santry HP, Alam HB. Fluid resuscitation: past, present, and the future. Shock (Augusta, Ga.). 2010 Mar;33(3):229.

Gantner D, Moore EM, Cooper DJ. Intravenous fluids in traumatic brain injury: what's the solution? Current opinion in critical care. 2014 Aug 1;20(4):385-9.

Chowdhury, Tumul; Cappellani, Ronald B; Schaller, Bernhard1; Daya, Jayesh. Role of colloids in traumatic brain injury: Use or not to be used?. Journal of Anaesthesiology Clinical Pharmacology 29(3):p 299-302, Jul-Sep 2013. | DOI: 10.4103/0970-9185.117043

Van Aken, Hugo K.a; Kampmeier, Tim G.a; Ertmer, Christiana; Westphal, Martina,b. Fluid resuscitation in patients with traumatic brain injury: what is a SAFE approach?. Current Opinion in Anaesthesiology 25(5):p 563-565, October 2012. | DOI: 10.1097/ACO.0b013e3283572274

Semler MW, Self WH, Wanderer JP, et al. Balanced crystalloids versus saline in critically ill adults. N Engl J Med. 2018;378(9):829-839. doi:10.1056/NEJMoa1711586

Zampieri FG, Damiani LP, Biondi RS, Freitas FG, Veiga VC, Figueiredo RC, Serpa-Neto A, Manoel AL, Miranda TA, Corrêa TD, Azevedo LC. Effects of balanced solution on short-term outcomes in traumatic brain injury patients: a secondary analysis of the BaSICS randomized trial. Revista Brasileira de Terapia Intensiva. 2023 Mar 3;34:410-7.

Marchesini N, Fernández Londoño LL, Boaro A, Kuhn I, Griswold D, Sala F, Rubiano AM. Hyperosmolar therapies for neurological deterioration in mild and moderate traumatic brain injury: A scoping review. Brain Injury. 2023 Mar 19:1-9.

Farrokh, Saliaa,b,•; Cho, Sung-Minb,•; Suarez, Jose I.b. Fluids and hyperosmolar agents in neurocritical care: an update. Current Opinion in Critical Care 25(2):p 105-109, April 2019. | DOI: 10.1097/MCC.0000000000000585

Sekhon MS, Dhingra V, Sekhon IS, Henderson WR, McLean N, Griesdale DE. The safety of synthetic colloid in critically ill patients with severe traumatic brain injuries. Journal of Critical Care. 2011 Aug 1;26(4):357-62.

Halaweish I, Bambakidis T, He W, Linzel D, Chang Z, Srinivasan A, Dekker SE, Liu B, Li Y, Alam HB. Early resuscitation with fresh frozen plasma for traumatic brain injury combined with hemorrhagic shock improves neurologic recovery. Journal of the American College of Surgeons. 2015 May 1;220(5):809-19.

Chang R, Folkerson LE, Sloan D, Tomasek JS, Kitagawa RS, Choi HA, Wade CE, Holcomb JB. Plasma Transfusion Is Associated with Improved Survival after Isolated Traumatic Brain Injury in Patients with Multifocal Intracranial Hemorrhage. Journal of the American College of Surgeons. 2016 Oct 1;223(4):e203.

Gruen DS, Guyette FX, Brown JB, Okonkwo DO, Puccio AM, Campwala IK, Tessmer MT, Daley BJ, Miller RS, Harbrecht BG, Claridge JA. Association of prehospital plasma with survival in patients with traumatic brain injury: a secondary analysis of the PAMPer cluster randomized clinical trial. JAMA Network Open. 2020 Oct 1;3(10):e2016869-.

Palmer, Lee. "Fluid Management in Patients with trauma: restrictive versus liberal approach." Veterinary Clinics: Small Animal Practice 47, no. 2 (2017): 397-410.

Vrettos T, Poimenidi E, Athanasopoulos P, Balasis S, Karagiorgos N, Siklis T, Gatzounis G, Fligkou F. The effect of permissive hypotension in combined traumatic brain injury and blunt abdominal trauma: an experimental study in swines. Eur Rev Med Pharmacol Sci. 2016 Jan 1;20(4):620-30.

Jagannatha, Aniruddha Tekkatte, Kamath Sriganesh, Bhagavatula Indira Devi, and Ganne Sesha Umamaheswara Rao. "An equiosmolar study on early intracranial physiology and long term outcome in severe traumatic brain injury comparing mannitol and hypertonic saline." Journal of Clinical Neuroscience 27 (2016): 68-73.

Ballocco I, Evangelisti MA, Deiana R, Cubeddu F, Pinna Parpaglia ML, Serra G, Carta G, Manunta ML. A pilot study evaluating the effect of mannitol and hypertonic saline solution in the treatment of increased intracranial pressure in 2 cats and 1 dog naturally affected by traumatic brain injury. Journal of Veterinary Emergency and Critical Care. 2019 Sep;29(5):578-84.

Maegele M. Coagulopathy after traumatic brain injury: incidence, pathogenesis, and treatment options. Transfusion. 2013 Jan;53:28S-37S.

Fluid Therapy in Lung Disease

Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (Veterinary Emergency and Critical Care; Medicine of Dogs)

Introduction:

Fluid therapy is an essential component of the management of many types of lung disease. However, the optimal fluid management strategy in patients with lung disease is often unclear, given normal physiology of fluid balance in the lung is frequently disrupted by disease. The type and amount of fluid required by patients with lung disease depend on various factors such as the underlying disease, disease severity, and the presence of other comorbidities. This review aims to summarize the current evidence on fluid therapy in the treatment of patients with lung disease.

In the normal lung, there are several mechanisms which limit accumulation of extravascular lung water:

- Capillary hydrostatic pressure can increase without significant leakage of fluid into the alveolar tissue
- The pulmonary interstitium is non-distensible, which limits accumulation of extravascular lung water.
- Pulmonary lymphatics are able to significantly increase flow in response to extravascular lung water accumulation

In disease, alterations in microvascular permeability resulting from glycocalyx damage, infection, tissue injury and inflammation rapidly overwhelm these homeostatic mechanisms, leading to the development of significant increases in lung water content. This leads to development of pulmonary interstitial oedema, alveolar oedema, and, depending on the underlying disease, the presence of cellular infiltrates and high protein pulmonary effusion e.g. pneumonia, ARDS etc.

The Effects of Fluid Therapy on Pulmonary Function

In the absence of pulmonary pathology, experimental studies have documented an increase in lung weight, histological oedema and water content – without an effect on PaO_2 - following intravenous fluid boluses in a rat model of haemorrhagic shock and crystalloid fluid resuscitation. Other experimental studies in healthy, dehydrated dogs have shown pulmonary oedema secondary to fluid overload developed with fluid doses of between 270 and 360 ml/kg/hr for 1 hour – doses many times those delivered in acute volume resuscitation. The results of these studies suggest that in the absence of lung disease, administration of intravenous fluids to manage hypovolaemia and to correct hydration deficits are likely well tolerated.

However, in the presence of lung disease, or sustained hypervolaemia, fluid therapy will contribute to a positive lung fluid balance, and the development of pulmonary oedema.

The presence of lung disease, or sustained increased in capillary hydrostatic pressure, however, is present in many disease, including:

- Pneumonia
- Acute respiratory distress syndrome
- Congestive heart failure
- Pulmonary hypertension
- Pulmonary trauma/contusions

Additionally, conditions that produce endothelial glycocalyx damage may result in increased rates of fluid extravasation in pulmonary tissue. Conditions associated with glycocalyx damage with potential to result in increased pulmonary fluid accumulation include:

- Systemic inflammatory response syndrome
- Sepsis
- Hypervolaemia/volume overload

- Hyperglycaemia
- Ischaemia-reperfusion injury
- Trauma and haemorrhage
- Hypoalbuminaemia

Fluid overload has been identified in critically ill dogs and is associated with increased mortality. Pulmonary fluid overload results in the following:

- Dilution of lung surfactant
- Reduced lung compliance
- Ventilation-perfusion mismatching and venous admixture
- Reduced efficiency of gas exchange
- Increased work of breathing

The Diagnosis of Excess Lung Water

The most effective tools for evaluating excess lung water include:

- Physical examination – physical examination of the patient may reveal increased respiratory rate, increased respiratory effort, evidence of rales and crackles in thoracic auscultation, and the presence of altered mucous membrane colour, among other parameters
- Pulse oximetry – pulse oximetry may show falling oxygen saturation with development of increased lung water content
- Point of care ultrasound – lung ultrasound techniques allow detection of pulmonary infiltrates with fluid, lung consolidation and other pathology. Good correlation exists between the number of B-lines observed in lung ultrasound, and extravascular water in lung tissue, with the technique being more sensitive than thoracic radiography at detecting early development of lung oedema.
- Thoracic radiography – thoracic radiography may reveal the presence of interstitial and alveolar fluid accumulation, although interpretation may be complicated by patient co-morbidities such as contusions, pneumonia, and ARDS, among others.
- Blood gas analysis – blood gas analysis may reveal a low PaO_2, increased A-a gradient, or a reduced P/F ratio (PaO_2/FiO_2)

Fluid Therapy in Pulmonary Contusions[1-6]

Pulmonary contusions result from a compression-decompression injury to the thoracic structures during some form of trauma. As a result of the contusion, alveoli and capillaries are torn, and blood and interstitial fluid leak into the alveoli and tissues. Additionally, tissue damage results in leukocyte chemotaxis and cytokine-mediate inflammatory response, which further increases pulmonary capillary permeability, with extravasation of protein and cells into extravascular lung tissue, which peaks 12-24 hours following trauma.

Administration of appropriate fluid therapy to patients with pulmonary contusion is of critical importance to the patient – as excesses in intravenous fluid administration may exacerbate trauma-associated coagulopathy and further intra-pulmonary haemorrhage; whilst insufficient fluid therapy may adversely affect outcome in co-morbidities, such as traumatic brain injury.

A summary of conclusions from relevant published literature in experimental animal studies, and literature regarding fluid therapy strategy in human patients with pulmonary contusions follows:

- In experimental studies in pigs with blunt-force lung injury, use of crystalloid fluid therapy was associated with higher lung water volumes than synthetic colloid solutions
- The rate of fluid replacement is directly related to lung weight and clinical and pathological changes of respiratory insufficiency.
- Plasma fluid resuscitation results in lower water volume than isotonic crystalloid resuscitation

- Increasing the total volume of isotonic crystalloid solution administered in patient resuscitation is positively correlated with development of hypoxaemia, as well as development of acute respiratory distress syndrome following chest trauma
- Administration of blood products during resuscitation is associated with lower mortality
- Positive fluid balance in the first 3 days of hospitalisation is associated with higher mortality, worse oxygenation and prolonged requirement for ventilation therapy

There is a paucity of evidence regarding optimal fluid therapy strategies for management of pulmonary contusions in both humans, as well as dogs and cats. The aforementioned conclusions from the current studies does however, provide some insight into some broad recommendations:

Management of Hypovolaemia:

Hypovolaemia, if present, should be treated using the smallest volume of fluid required to achieve haemodynamic stability. The optimal choice of fluid utilized to achieve stabilisation is currently unclear, with options being isotonic crystalloid, synthetic colloid or plasma.

Synthetic colloids may result in lower lung water volumes following resuscitation – however, their use is associated with an increased potential for reduced blood clot strength, which in the setting of pulmonary contusions, may exacerbate intra-pulmonary haemorrhage.

Use of fresh frozen plasma may be of benefit early in resuscitation – but the cost of transfusion and lack of firm recommendation at present precludes a universal recommendation. Potential benefits include mitigation of trauma-associated coagulopathy in the acutely traumatised patient, and lower lung water volume.

Cautious use of isotonic crystalloid fluids to achieve haemodynamic stability, followed by a conservative fluid strategy, to minimise positive fluid balance over the first 3 days of hospitalisation is a rational choice for the majority of patients.

Maintenance Fluid Therapy:

In all cases, following acute volume resuscitation, fluid therapy rates should be reduced to provide for maintenance therapy only, in order to reduce risk of volume overload and positive fluid balance. At the earliest opportunity following patient stabilisation, de-escalation of fluid therapy (weaning) should be applied to allow for establishment of neutral fluid balance.

Fluid Therapy in Patients with Acute Respiratory Distress Syndrome (ARDS)[7-15]

ARDS is a severe form of acute lung injury characterized by inflammation and increased permeability of the alveolar-capillary membrane, leading to non-cardiogenic pulmonary oedema and respiratory failure.

The most common precipitating causes of ARDS are pulmonary infections, non-pulmonary sepsis, shock, aspiration pneumonia, thoracic trauma, near drowning, and major trauma (including traumatic brain injury).

Following systemic dissemination of inflammatory mediators associated with precipitating causes, an overwhelming pulmonary inflammatory process is initiated leading to alveolar epithelial and vascular endothelial injury, and extravasation of inflammatory cells, protein and extracellular fluid into the pulmonary interstitial and alveolar spaces. Histopathology reveals of affected lungs reveals diffuse alveolar damage with neutrophil infiltration, alveolar hemorrhage and hyaline membrane formation.

Regarding fluid requirements, as the airspaces are becoming flooded with fluid, the systemic inflammation and ensuing endothelial permeability present in many ARDS patients lead to third spacing and relative intravascular volume depletion, manifesting as hypotension in a large proportion of ARDS patients. Further complicating the problem, increases in pulmonary hydrostatic pressure can directly exacerbate local inflammation and increased pulmonary vascular permeability.

One of the key challenges in the care of the ARDS patient is how to reduce fluid accumulation in the alveolar space whilst also providing adequate intravascular volume to support cardiac output and vital tissue perfusion.

Goals for fluid therapy in ARDS:

As stated above, optimal fluid management for patients with ARDS should provide adequate oxygen delivery to the body, while avoiding inadvertent increase in lung oedema which further impairs gas exchange.

What the literature says:

Comprehensive reviews and meta-analysis of published literature shows the following regarding fluid therapy in patients with ARDS:

- Positive fluid balance has been associated with
    - Prolonged mechanical ventilation
    - Longer ICU and hospital stay
    - Higher mortality
- Patients who received appropriate resuscitation fluid volumes given early in patient management, coupled with later restrictive fluid volumes are associated with better outcomes than patients who received restricted early fluid resuscitation and/or liberal later fluid volumes.
- Patients having a lower fluid balance at 7 days had fewer ventilator days, and fewer ICU days than patients with higher fluid balance.
- Patients resuscitated with albumin have better oxygenation, serum albumin concentration and lower cumulative fluid balance than non-albumin-treated patients. Addition of furosemide to albumin therapy resulted in similar benefits, with the addition of lower fluid excess balance

Specific ARDS Trial Results:

Several randomized controlled trials and meta-analyses have investigated the optimal fluid management strategy in patients with ARDS.

Cumulative positive fluid balance is associated with worse clinical outcomes in patients with ARDS. A phase III study conducted by the ARDS network (the FACTT study) compared liberal versus conservative fluid strategy in patients with acute lung injury. They observed an improvement in oxygenation, lung injury score

(LIS), and shortened duration of mechanical ventilation without any increase in other organ failure in the conservative group, but no difference in hospital mortality

A meta-analysis of 17 randomized controlled trials found that the use of albumin-containing solutions for the resuscitation of patients with sepsis (including ARDS) was associated with lower mortality compared with other fluid resuscitation regimens.

Conclusion:

Fluid therapy is a critical component of the management of patients with lung disease. The optimal fluid management strategy depends on the underlying disease, disease severity, and comorbidities. For patients with both pulmonary contusions and ARDS, fluid restriction may be beneficial – although in both conditions, early resuscitative efforts should be pursued to achieve improvements in tissue oxygen delivery and resolution of hypovolaemia, prior to fluid restriction to maintenance rates, and eventually de-escalation to achieve fluid restriction.

It is essential to carefully monitor fluid intake in patients with lung disease to prevent respiratory failure and worsening of lung function. Further research is needed to determine the optimal fluid management strategy for patients with lung disease.

References:

1. Adamantos S. Fluid Therapy in Pulmonary Disease: How Careful Do We Need to Be?. Frontiers in Veterinary Science. 2021 Aug 9;8:624833.
2. Rendeki S, Molnár TF. Pulmonary contusion. Journal of thoracic disease. 2019 Feb;11(Suppl 2):S141.
3. Klein Y, Cohn SM, Proctor KG. Lung contusion: pathophysiology and management. Current Opinion in Anesthesiology. 2002 Feb 1;15(1):65-8.
4. Magret, M. (2010). Lung Trauma. Clinical Pulmonary Medicine, 17(2), 75–81.
5. Johnson, J. L. (2013). Chest Trauma and Lung Contusions. In Management of Musculoskeletal Injuries in the Trauma Patient (pp. 305–319). Springer New York.
6. Murphy CV, Schramm GE, Doherty JA, Reichley RM, Gajic O, Afessa B, Micek ST, Kollef MH. The importance of fluid management in acute lung injury secondary to septic shock. Chest. 2009 Jul 1;136(1):102-9.
7. Valentine SL, Sapru A, Higgerson RA, Spinella PC, Flori HR, Graham DA, Brett M, Convery M, Christie LM, Karamessinis L, Randolph AG; Pediatric Acute Lung Injury and Sepsis Investigator's (PALISI) Network; Acute Respiratory Distress Syndrome Clinical Research Network (ARDSNet). Fluid balance in critically ill children with acute lung injury. Crit Care Med. 2012 Oct;40(10):2883-9. doi: 10.1097/CCM.0b013e31825bc54d. PMID: 22824936; PMCID: PMC3455114.
8. Carmen Sílvia Valente Barbas, Gustavo Faissol Janot Matos, Marcelo Britto Passos Amato, Carlos Roberto Ribeiro Carvalho, "Goal-Oriented Respiratory Management for Critically Ill Patients with Acute Respiratory Distress Syndrome", Critical Care Research and Practice, vol. 2012, Article ID 952168, 13 pages, 2012.
9. Kuan WS, Tam WWH, Chong SY, et al. Restrictive fluid management in acute respiratory distress syndrome: A systematic review and meta-analysis of randomized controlled trials. Journal of Critical Care. 2018;44:362-368.
10. van Mourik N, Metske HA, Hofstra JJ, Binnekade JM, Geerts BF, et al. (2019) Cumulative fluid balance predicts mortality and increases time on mechanical ventilation in ARDS patients: An observational cohort study. PLOS ONE 14(10): e0224563.
11. Delaney, Anthony P. MD, FCICM; Dan, Arina MD, FCICM; McCaffrey, John MD, FCICM; Finfer, Simon MD, FCICM. The role of albumin as a resuscitation fluid for patients with sepsis: A systematic review and meta-analysis*. Critical Care Medicine 39(2):p 386-391, February 2011. | DOI: 10.1097/CCM.0b013e3181ffe217
12. Silversides JA, Major E, Ferguson AJ, et al. Conservative fluid management or deresuscitation for patients with sepsis or acute respiratory distress syndrome following the resuscitation phase of critical illness: a systematic review and meta-analysis. Intensive Care Medicine. 2017;43(2):155-170.
13. Schuller D, Schuster DP. Fluid-management strategies in acute lung injury. N Engl j Med. 2006 Sep 14;355(11):1175.
14. Lee J, Corl K, Levy MM. Fluid therapy and acute respiratory distress syndrome. Critical Care Clinics. 2021 Oct 1;37(4):867-75.
15. Dong WH, Yan WQ, Song X, Zhou WQ, Chen Z. Fluid resuscitation with balanced crystalloids versus normal saline in critically ill patients: a systematic review and meta-analysis. Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine. 2022 Apr 18;30(1):28.

Treatment of Traumatic Haemorrhagic Shock

Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (Veterinary Emergency and Critical Care; Medicine of Dogs)

Definition

Haemorrhagic shock is a potentially life-threatening consequence of whole blood loss. Haemorrhagic shock results in loss of fluid, and oxygen-carrying capacity and coagulation factors from the vasculature, and as such will produce clinical signs of hypovolaemia, as well as anaemia, and in some cases, coagulopathy particularly following resuscitation effort, if these are made using crystalloid or synthetic colloid fluids, in the absence of blood products.

Haemorrhagic shock is diagnosed when there is evidence of poor perfusion, combined with evidence of bleeding.

Aetiology

Trauma may result in either external haemorrhage, or internal haemorrhage. External haemorrhage is typically readily diagnosed, and managed with application of compression bandages, and/or isolation and ligation of the source of bleeding. Internal haemorrhage can originate from any internal organ, is more challenging to detect, and requires careful physical examination, coupled with the use of imaging modalities, including point or care ultrasound.

Internal haemorrhage of sufficient severity to cause shock usually occurs into the pleural cavity or abdominal cavity – and most commonly results from hepatic contusions, hepatic laceration, splenic contusion or rupture, avulsion of the kidneys, or contusions of other abdominal organs.

The Approach to the Patient

A thorough history should be obtained in all cases of suspected haemoabdomen, including questioning regarding possibility of trauma, duration and progression of any clinical signs.

Symptoms of the patient with haemorrhage can vary, depending on the location of haemorrhage and further complicating factors, including the presence of hypovolaemia, anaemia, infection, and any co-morbidities, such as respiratory tract trauma, diaphragmatic hernia, urinary tract rupture, organ avulsion, herniation, and traumatic brain injury, among others.

Clinical signs referable to haemorrhage may include:

- Cardiovascular compromise
  - Tachycardia
  - Bradycardia (esp. Cats)
  - Pale mucous membranes
  - Prolonged capillary refill time
  - Depressed mentation
  - Cardiac arrhythmias
  - Weak pulses
  - Patients with mild haemorrhage, or slow bleeding may present in compensated shock.
- Respiratory symptoms
  - Tachypnoea
  - Rapid, shallow respiration (due to abdominal pain)
- Abdominal symptoms
  - Abdominal pain
  - Abdominal distension
  - Palpable abdominal mass
  - Abdominal organ displacement
  - Palpable abdominal fluid wave (requires approx. 40 ml/kg abdominal fluid to produce in many animals)
  - Skin discolouration about the inguinal region (Cullen sign), umbilicus testicles.

- Other symptoms
    - Bruising of the skin (in cases of trauma)
    - Wounds

Note that some patients – especially those with either mild, or early haemorrhage, may present with no overt clinical signs of haemorrhage, and may display only symptoms of weakness and lethargy.

Initial Patient Assessment

Following physical examination, the patient should be assessed as follows:

1. Blood tests:
    a. PCV/TP analysis
    b. CBC
    c. Blood smear
    d. Serum biochemistry – will assist in identifying any concurrent organ system injury and electrolyte concentrations
    e. Coagulation assessment – will allow determination of clotting defects, which should be managed prior to any surgical intervention to reduce risk of catastrophic haemorrhage at the time of any surgical interventions.
        i. APTT
        ii. PT
        iii. Platelet count; BMBT
        iv. Fibrinogen
        v. FDP/D-dimer
        vi. TEG (if available)
2. Diagnostic imaging
    a. Radiography – perform once stable, or if FAST ultrasound is inconclusive
        i. Abdominal radiography:
            1. Left lateral, right lateral, and ventro -dorsal radiographs should be taken, to evaluate the presence of free abdominal gas (which may indicate rupture of intestines, or septic peritonitis), or the presence of organ displacement or torsion
        ii. Thoracic radiography
            1. Left and right lateral, and ventro-dorsal +/- dorso-ventral radiography should be performed to evaluate lungs and pleural space
    b. Ultrasound
        i. AFAST scan of the abdominal cavity allows detection of fluid in 4 key areas of the abdomen
        ii. Use of the AFAST fluid scoring system allows documentation of an advancing/worsening abdominal bleed
        iii. It is essential for a sample of any fluid detected to be evaluated and confirmed as haemorrhagic effusion

Emergency stabilisation

The patient with traumatic haemorrhage should be managed medically until the following is achieved:

1. Acute patient resuscitation has been achieved
    a. Provide oxygen supplementation. Avoid nasal oxygen therapy in patients with documented coagulopathy, as catheter-related trauma to the nasal mucosa can result in uncontrolled haemorrhage from the nose in these patients
    b. Provide analgesia. Most patients with abdominal haemorrhage are in pain. Opioid analgesia should be provided using pure mu agonists, such as fentanyl or morphine. Continuous infusions are preferred, as they avoid peak/trough serum levels, and the potential for patients to experience pain in-between dosing periods. Addition of ketamine and/or lidocaine continuous infusions improves analgesic effectiveness, and is recommended in most patients progressing to surgical intervention.

2. Diagnosis and management of shock
    a. Clinical signs of decompensated shock include tachycardia (bradycardia in cats); pale mucous membranes, prolonged capillary refill time; weak pulses and dull mentation.
    b. Fluid therapy should be provided with an isotonic crystalloid, such as lactated Ringer's solution, given at 10 ml/kg (dog); 7 ml/kg (cat) over 10 minutes, followed by patient reassessment. Further boluses of fluid (+/- blood transfusion – see below) are administered to patients in which sustained symptoms of shock are present.
    c. Endpoints of fluid resuscitation vary, depending on patient comorbidities, but a target systolic arterial blood pressure of 90 mm Hg should ideally be achieved; and a mean arterial pressure of 60-70 mm Hg. In addition, the patient should show improved mentation, reduction in heart rate and improved pulse quality as other measures of improvement circulation.
    d. Note that sustained administration of high rates of intravenous fluids beyond those required to achieve target blood pressures (see below) is generally not advised, as these rates of fluid administration can lead to increased blood pressure in diseased or damaged abdominal organs, and can further exacerbate abdominal bleeding. Additionally, they may result in dilutional coagulopathy, which can exacerbate bleeding tendency.
    e. Once target blood pressure and other endpoints are achieved, the patient should have fluid rates reduced to maintenance rates, plus allowance for rehydration (if the patient is dehydrated).
    f. Note that in patients that are continuing to bleed into their abdominal cavity, resolution (or partial resolution) of symptoms of shock is likely to be short-term, necessitating further patient intervention. Application of abdominal compression bandages, anti-plasminogen medications, blood transfusion, and definitive surgical repair may be required in order to resolve clinical signs of haemorrhagic shock in some patients.
3. Alleviation of life-threatening anaemia using blood transfusion
    a. Patients with acute anaemia resulting from abdominal haemorrhage will generally display symptoms of shock (tachycardia, etc.) when the PCV falls below 20-24%.
    b. Whole blood transfusion is recommended to raise the patient PCV to approximately 27-30% in the pre-operative period
    c. Excessive blood transfusion volume may exacerbate ongoing abdominal haemorrhage by raising abdominal organ blood pressure in the presence of active pathology e.g. liver contusions, bleeding vascular neoplasia etc.
    d. Autologous Transfusion Therapy
        i. Autologous transfusion therapy (also called "Autotransfusion") is an effective method for rapidly providing red blood cells and intravascular volume support when imminent death precludes the preparation of allogenic transfusion or when other blood products are not available.
        ii. Intra-abdominal, or intra-pleural blood may be collected aseptically by aspirating into a sterile syringe by paracentesis or by suctioning into a sterile container at the time of surgery.
        iii. Acute haemorrhage: When haemorrhage is acute and rapid, insufficient time may have elapsed between the time of haemorrhage, and the time of blood collection, for the blood to defibrinate. As a result, it is necessary to use anti-coagulant during collection. Typically, 7 mL of citrate–phosphate dextrose adenine should be added to each 50 mL of abdominal blood collected.
        iv. Chronic haemorrhage: Abdominal blood associated with chronic haemorrhage can usually be collected and infused without anticoagulant because the blood is defibrinated when it comes in contact with the peritoneal surface.
        v. The collected blood should be administered through a blood administration set or in-line blood filter.

4. Administration of adjunct medications to slow the rate of haemorrhage (if appropriate)
    a. Patients with abdominal trauma may benefit from administration of the anti-plasminogen medication, tranexamic acid.
    b. Tranexamic acid is administered at an initial dose of 10-20 mg/kg (dogs) given slow intravenously over 20-30 minutes, followed by a continuous infusion of 10 mg/kg IV delivered over the subsequent 4-6 hours
5. Application of external compression bandages to slow the rate of abdominal haemorrhage (if appropriate)
    a. Patients with traumatic haemoabdomen, or haemoabdomen caused by anticoagulant toxicity, may benefit from abdominal compression bandages
    b. A soft, layered, cotton-wool bandage is applied circumferentially around the abdomen, and is compressed using compression gauze, or self-adhesive elastic bandage to a pressure sufficient to just allow 2 fingers to be placed between the bandage and the skin without difficulty.
    c. Compression bandages increase intra-abdominal pressure, thereby compressing bleeding blood vessels, and increasing resistance to blood flow into the abdominal cavity.
    d. Compression bandages are most helpful in post-trauma patients with blunt abdominal trauma, with liver or splenic contusions, as these patients are generally not amenable to surgery – but are at risk of continued bleeding without compression support.
    e. Bandages may be left in place for several hours, as long as intra-abdominal pressure is maintained less than 10-12 mm Hg, after which time, they may be removed slowly, by gradually incising the bandage 1-4 cm (depending on patient size) to reduce intra-abdominal pressure every 30-60 minutes for a further 2-4 hours.
6. Determination of the requirement for surgical intervention
    a. Surgical intervention may be considered in the following instances:
        i. When medical therapy is either ineffective, or transiently effective at stabilising the patient
        ii. Progressively decreasing PCV, despite interventions including fluid therapy, transfusion therapy, abdominal compression bandages and anti-thrombolytic therapy
        iii. Increasing abdominal fluid score despite active interventions to reduce the rate of bleeding (compression, anti-plasminogen therapy
        iv. Identification of a bleeding abdominal mass
        v. A penetrating abdominal wound
        vi. Evidence of rupture of a hollow abdominal viscous (urinary tract, gastrointestinal tract, biliary tract, reproductive tract)
        vii. Organ torsion or volvulus

Surgical Management

In traumatic haemorhage, the primary indications for surgery are the presence of a penetrating, or the presence of a distinct fracture of the liver or spleen in the patient that is not stabilising with medical treatment alone. Most cases of traumatic haemoabdomen are caused by liver or splenic contusions, and are not normally amenable to surgical repair, and are best managed with fluid resuscitation, transfusion therapy, tranexamic acid, and abdominal compression bandages.

The main goals in surgical management of haemoabdomen are:

1. To identify source(s) of haemorrhage
2. To effect rapid and timely haemostasis
3. To remove or repair damaged abdominal organs resulting in haemorrhage
    a. Partial liver lobectomy
    b. Splenectomy
    c. Nephrectomy for avulsed kidneys or kidney neoplasia
    d. Adrenalectomy
    e. Partial enterectomy for intestinal neoplasia
4. To remove neoplastic tissue with wide surgical margins using techniques listed above

Prognosis

The prognosis for traumatic haemorrhage is variable. Short-term prognosis is dependent on several factors, including:

1. The severity of haemorrhage, and the severity of clinical signs at presentation
2. The patient response to treatment and resuscitation efforts
3. The ability to readily detect and manage ongoing haemorrhage

Longer-term survival is largely dependent on the presence and severity of co-morbidities, such as traumatic brain injury.

References

1. Pesillo-Crosby SA. Hemoperitoneum. Textbook of Small Animal Emergency Medicine. 2018 Sep 24:528-34.
2. Pratschke K. Approach to haemoabdomen in small animal patients. In Practice. 2020 Jan 1;42(1):5-19.
3. Lux CN, Culp WT, Mellema MS. Hemoperitoneum. Small Animal Surgical Emergencies. 2015 Nov 9:105-15.
4. Caldwell DJ, Petras KE, Mattison BL, Wells RJ, Heffelman VL. Spontaneous hemoperitoneum and anaphylactic shock associated with Hymenoptera envenomation in a dog. Journal of Veterinary Emergency and Critical Care. 2018 Sep;28(5):476-82.
5. Knight R, McClaran JK. Hemoperitoneum Secondary to Liver Lobe Torsion in a Cat. Journal of the American Animal Hospital Association. 2020 Jan 1;56(1).
6. Marik PE, Weinmann M. Optimizing fluid therapy in shock. Current Opinion in Critical Care. 2019 Jun 1;25(3):246-51.
7. Ramesh GH, Uma JC, Farhath S. Fluid resuscitation in trauma: what are the best strategies and fluids?. International journal of emergency medicine. 2019 Dec;12:1-6.
8. Milford EM, Reade MC. Resuscitation fluid choices to preserve the endothelial glycocalyx. Critical Care. 2019 Dec;23(1):1-1.

Management of Acute Heart Failure in the Cat

Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (VECC; Medicine of Dogs)

Acute Congestive Heart Failure (CHF)

Acute congestive heart failure in cats is relatively common particularly in cats with advancing age. Congestive heart failure may result from any of the following

- Cardiomyopathy: Dilated cardiomyopathy, hypertrophic cardiomyopathy, restrictive/intermediate cardiomyopathy
- Congenital heart disease
- Bacterial endocarditis
- Heartworm disease
- Pericardial disease (rare)

The sequela to congestive heart failure depends on whether left or right sided heart failure are present. Typically, left-sided CHF results in pulmonaryo edema; isolated right-sided CHF causes ascites +/- pleural effusion; and biventricular CHF often causes pleural effusion and pulmonary oedema.

Chylothorax can result from congestive heart failure whenever the pressure in the cranial cava exceeds the pressure in the thoracic duct.

Echocardiography is one of the most useful diagnostic tools in assessment of the cat with a cardiac emergency, and can beused to identify pleural or pericardial effusion, to assess the degree of left atrial enlargement in the emergency situation. More advanced exams can identify the degree of contrle function and other specifics once the patient has been stabilised.

Causes of Acute Heart Failure in the Cat

Hypertrophic cardiomyopathy (HCM):

Definition:

Hypertrophic cardiomyopathy is the term given to a thickening of the heart muscle. The most recent ACVIM guidelines on the classification and management of cardiomyopathies in cats uses the definition that hypertrophic cardiomyopathy in the cats diffuse or regional increasedleft ventricular (LV) wall thickness with a nondilated LV chamber (Luis Fuentes et al 2020). In cats, this is the most common form of heart disease. Hypertrophic cardiomyopathy may be caused by what is termed idiopathic (meaning of undetermined cause), hyperthyroidism, acromegaly, neoplastic infiltration of the myocardium, and primary or secondary hypertension (high blood pressure). Hypertrophic cardiomyopathy results in an increase in the size of myocardial cells, particularly in the left ventricle. This has the effect of decreasing the internal (luminal) diameter of the left ventricle- effectively reducing the internal volume of the left ventricle, so that each ventricular contraction results in a smaller volume of blood being delivered to the general circulation. This results in triggering the compensatory mechanisms associated with reduced cardiac output:

- Activation of the sympathetic nervous system and adrenaline release
- Activation of the renin-angiotensin-aldosterone (RAAS) axis
- Vasopressin release
- Cortisol release etc.

These mechanisms result in sustained elevations in both preload and afterload, with the development of volume overload, left atrial enlargement, pulmonary oedema and heart failure following.

Clinical Signs and Physical Examination:

The most common presenting clinical signs in cats with HMC is laboured breathing (Lui Fuentes et al 2020; Ware et al 2021). Other clinical signsof heart failure caused by hypertrophic cardiomyopathy include lethargy, anorexia and mild gastrointestinal disturbance (vomiting).

The presence of thromboembolic disease may give rise to clinical signs of hindlimb paresis, organ dysfunction or respiratory distress. On physical examination, many cats with hypertrophic cardiomyopathy (up to 80%) have a detectable cardiac murmur or gallop rhythm detected on physical examination - compared to 30-45% of cats without cardiomyopathy. Gallop rhythms may be present in up to 16% of cats with HCM - whereas they are rare in normal cats. It should be noted that cats with HCM may show no clinical signs or abnormalities on physical examination. Evidence of pleural effusion +/- hepatomegaly may be detected in some cats. Additionally, hypothermia may be observed as cardiac failure advances.

In cats that present in acute heart failure, a gallops rhythm or arrhythmia are reportedly more common than the presence of a heart murmur.

Diagnosis:

Imaging is crucial in making a diagnosis of hypertrophic cardiomyopathy, and includes echocardiography, lung ultrasound and thoracic radiography.

Cardiomegaly may be identified on thoracic radiographs (usually left atrial enlargement) but the concentric nature of the hypertrophy can result in unimpressive cardiomegaly in some cases (Ware et al 2021). Most cats with significant left atrial enlargement will also have enlargement of both pulmonary artery and vein on the DV radiographic projection. On the lateral radiographs cats with chronic left atrial enlargement often have a tortuous pulmonary vein returning to the left atrium from the caudal lung lobes. Pulmonary edema can be initially manifested as an increased interstitial pattern in the lungs that coalesces into an alveolar pattern as CHF worsens. In many cats this radiographic pattern develops ventrally or is distributed into multifocal, patchy areas of edema rather than in the classic perihilar regions seen in dogs. Note that pleural effusion can also be seen - often in association with hepatomegaly and enlargement of the caudal vena cava. A 2012 study found that poorer left atrial function (which was not related to left atrial size or diameter) and increased right ventricular dimensions were associated with pleural effusion in cats with left-sided heart disease (Johns et al 2012). Radiographic assessment of left atrial size using a modified vertebral heart score has been described (Guglielmini et al 2015). Whilst this measurement has good sensitivity and specificity in dogs, a study evaluating the accuracy of this measurement in cats found low value in predicting left atrial size in cats with heart disease diagnosed at cardiac ultrasound (Schober et al 2014). Conversely, a lateral vertebral heart score above 7.9 has a high diagnostic accuracy in distinguishing cats with left sided cardiac disease (Guglielmini et al 2014)

Echocardiography is the gold standard for ante-mortem diagnosis of HCM. typically reveals hypertrophy of the interventricular septum and left ventricular wall, good contractile function, and marked to severe left atrial enlargement - often with an Aorta: Left atrial ratio of 1:2 or greater (Haggstrom et al 2015) . In cats with long standing left atrial enlargement and secondary pulmonary hypertension, the pulmonary artery is often enlarged and bigger than the aorta and these cats often have accompanying right sided heart failure. Pericardial effusion resulting from congestive heart failure may be identified in some cats; however, the volume of pericardial fluid is usually small, and infrequently results in cardiac tamponade.

Other diagnostics that are desirable include a complete blood count, biochemistry profile including electrolytes), urine analysis, arterial blood pressure, and thyroid concentration - especially in cats >6 years of age (Haggstrom et al 2015).

It is suspected that HCM in cats has a genetic basis, and it has been reported to be familial in some cat breeds, including:

- American short-hair
- Maine Coon
- Norwegian Forest
- Sphynx
- Persian

HCM is inherited as an autosomal dominant trait in Maine Coon cats, and may well be in other breeds also. Mutations in the gene for myosin binding protein C (MWBPCF3) have been reported in Maine Coon and Ragdoll cats. As such, genetic testing has been recommended for some breeds (Ware et al, 2021, Haggstrom et al 2015). More than 40% of Maine Coon cats are estimated to have mutations in the MYBPC3-A31P gene, and between 17-30% of ragdoll cats are estimated to have mutation of the MTBPC3-R820W gene. Genetic testing for the MyBPC3-A31P mutation and the MyBPC3 R820W mutation istherefore recommended in Maine Coon and Ragdoll cats (respectively) intended for breeding, with the aim of decreasing the incidence of these mutations and HCM in these breeds. The same genetic tests can be considered in nonbreeding Maine Coon or Ragdoll cats to determine the relative risk.
Feline hypertrophic cardiomyopathy associated with a variant in the MYH7 gene has also been reported, and should be tested in animals intended for breeding.

The NT-Pro-BNP assay carries a high sensitivity for the presence of cardiac disease in the cat with respiratory disease – but lacks some specificity– meaning false positives are possible. It is considered a useful test for discriminating non-cardiac from cardiac diseases in situations where diagnostic ultrasound is not available, or where findings areequivocal, with a negative result making cardiac disease unlikely as a cause for the patient's clinical signs (Ware et al 2021, Haggstrom et al 2015).

Measuring circulating cardiac troponin I (cTnI) concentrations can help distinguish between cardiac and noncardiac causes of respiratory distress – as it is a marker of myocardial injury. In addition, cTnI also might be considered for its prognostic value, because an increased circulating cTnI concentration is associated with increased risk of cardiovascular death independent of LA size.

Electrocardiography should be utilized in cats as a general screening tool for arrhythmias. A variety of arrhythmias can occur in cats with cardiomyopathy, and can contribute to clinical signs such as weakness, syncope, and hypoxic-anoxic seizures.

Measurement of serum thyroxine should occur in middle-aged to older cats with suspected HCM, along with systolic blood pressure to evaluate for hypertension as a complicating or causative factor (Haggstrom et al 2015).

Dilated Cardiomyopathy (DCM):

Definition:

Dilated cardiomyopathy is a disease characterized by excessive loss of heart muscle cells, which results in a thinning of the heart muscle, and subsequent heart chamber dilatation. Dilative cardiomyopathy used to be the most common heart disease in cats in the 1970's and 1980's, because many cat foods at the time contained inadequate amounts of taurine. Subsequent to this becoming known in 1987, pet food manufacturers increased the amount of taurine in their manufactured diets, and the incidence of dilated cardiomyopathy in cats has now fallen to less than 10% of pre-1987 levels. The condition, however, may still be seen in cats fed a vegetarian or vegan diet. Other potential causes for DCM in cats include prolonged doxorubicin therapy, or end-stage cardiomyopathy, infection or toxin-induced (Luis Fuentes et al 2020). Cats with DCM typically have systolic dysfunction, with associated loud S3 gallop rhythm on cardiac auscultation, and may also have a soft murmur or arrhythmia present. Pleural effusion is frequently present in cats with DCM (Luis Fuentes et al 2020).

Clinical Signs:

Clinical signs of cats with DCM are similar to those in cats with HCM and include

- Lethargy
- Reduced appetite
- Open-mouth breathing with mild exertion
- Collapse
- Hypothermia
- Symptoms of shock – including bradycardia
- Tachypnoea
- Hypokinetic pulses may be palpated
- Heart murmurs are often present, and tend to be quite soft
- Gallop rhythms and jugular pulses may also be present
- Pleural effusion is not uncommon
- Aortic thromboembolism (symptoms dependent on location of thromboembolism)
- Retinal degeneration (30% of cats with taurine deficiency)

Diagnosis:

Diagnostic tests include clinical pathology, thoracic radiography, lung ultrasound and echocardiography. Radiographic findings often reveal

- Generalised cardiomegaly, with rounding of the cardiac apex
- Evidence of pleural effusion
- Pulmonary venous congestion
- Hepatomegaly +/- ascites may be evident

Characteristic echocardiography findings include

- Dilation of all 4 cardiac chambers
- Reduced left ventricular systolic function
- Thinning of the walls of the ventricles – although focal areas of ventricular wall hypertrophy can occur
- Intra-cardiac thrombi may be evident

Plasma and whole blood measurement of taurine is indicated in order to help anticipate the response to taurine supplementation.

Treatment:

Treatment goals for cats with DCM are similar to those for cats with HCM (see later). Oxygen therapy, thoracocentesis, and judicious use of diuretics is recommended in early patient stabilisation. Excessive diuretic doses should be avoided, as, due to poor systolic function, falls in circulating blood volume can precipitate cardiogenic shock. Pimobendan should be administered early in treatment to provide inotropic support. In patients unable to tolerate oral medication, intravenous pimobendan is preferred over oral medication. If intravenous Pimobendan is unavailable, dobutamine continuous infusion should commence (Luis Fuentes et al 2020).

Blood pressure support, management of hypothermia, and assessment and management of cardiac arrhythmias, as well as management of electrolyte disorders are crucial in acute patient management alongside management outlined above.

The long-term outcome is often related to the presence or absence of taurine deficiency as cats with taurine deficiency often respond well, with 50% survival at 1 year after diagnosis, if they can survive for the first 2 weeks. Cats with normal taurine concentrations often respond poorly to medical therapy and may only live for a short time following presentation (days to months).

Restrictive (intermediate) cardiomyopathy:

Definition:

Restrictive cardiomyopathy refers to a subset of heart muscle diseases characterized by normal-looking ventricular muscle on ultrasonography, but with a dilated left atrium – suggesting that the ventricles are unable to adequately distend (restricted) to cope with normal blood volume in diastole, which subsequently results in an increase in preload, and accumulation of blood in the left atrium, which leads to atrial distension, and the development of signs and symptoms of heart failure. Cats with restrictive cardiomyopathy have extensive endocardial or myocardial fibrosis with prominent left atrial or bi-atrial enlargement, with some atrial hypertrophy. In some cases, thick bands of fibrous tissue may bridge between the left ventricular wall and the septum, and can cause intraventricular obstruction (Luis Fuentes et al 2020).

Clinical Signs, Treatment and Prognosis:

Physical examination findings are not specific for RCM/ICM and can include respiratory distress, cardiac murmur, cardiac gallop, or cardiac arrhythmia. Affected cats have a mean age of presentation between 7-10 years old. Cats with isolated left heart failure and pulmonary edema usually develop loud pulmonary crackles, often with severe respiratory distress. In cats with biventricular heart failure (usually the result of longstanding left heart failure) respiratory distress is often accompanied by jugular vein distention, hepatomegaly, and muffled lung sounds ventrally due to pleural and/or pericardial effusion. A small volume ascites may be present in cats with biventricular heart failure however, it should be noted that marked cardiogenic ascites in the cat is usually due to isolated right heart disease.

Radiography usually shows left atrial enlargement with left ventricular or generalised cardiomegaly, evidence of interstitial infiltration/pulmonary oedema, pleural effusion, and distension and tortuous proximal pulmonary veins (Luis Fuentes et al 2020).

The ECG is frequently abnormal, with abnormal ventricular conduction evident, +/- atrial tachyarrhythmias.

Echocardiography reveals moderate to severe left atrial enlargement (+/- right atrial enlargement) and patchy or generalised areas of hyperechoic, thickened endocardium in the ventricles. Generally, the ventricular wall is normal thickness (<6 mm), but may have focal regions of increased thickness. Bridging septa may be observed in the ventricles of some cats. Thrombi may be observed within the left atrial lumen in some patients, as can varying degrees of mitral and tricuspid valve insufficiency (Luis Fuentes et al 2020).

This is a cardiac disease with a poor prognosis following development of acute heart failure, with reports of median survival time ranging from less than 2 months, with survival times possible in excess of 21 months for cats without respiratory distress and heart failure at diagnosis (Luis Fuentes et al 2020).

Transient Myocardial Thickening (TMT) of Cats with Heart Failure:

TMT is a condition that was reported in a study of 21 cats that presented with acute congestive heart failure. In the study, TMT was defined as initial maximal left ventricular wall thickness (LVWT)≥ 6 mm with left-sided CHF, with subsequent resolution of CHF, reduction in left atrium/aorta (LA/Ao), and LVWT<5.5 mm. 100% of cats with TMT survived in the study, compared with 60% of cats with HCM. Cardiac troponin (cTnI) was measured on presentation and repeated at the time of LVWT normalization. Median cardiac troponin (cTnI) was elevated at presentation and normalized once myocardial thickening resolved.
TMT appears to preferentially affect young cats and often follows an antecedent event but is difficult to differentiate from HCM at presentation. The recognition of TMT could have an important impact in daily clinical cardiology, leading to clinicians and owners attempting treatment in cases classically thought to have a poor prognosis, and potentially avoiding premature euthanasia (Novo Matos et al 2018).

Myocarditis:

Inflammation of the heart muscle in cats is called myocarditis, and is most commonly caused by infection caused by Feline Infectious Peritonitis (FIP) virus, but can also be caused by feline immunodeficiency virus (FIV), with infiltration of T lymphocytes and macrophages into the myocardium occurring. Other causes might include trauma to the myocardium following thoracic trauma or surgery, sepsis, and bacterial infection with Bartonella, toxoplasma or Trypanosoma.
Typically, myocarditis results in cardiac arrhythmias, diastolic failure, acute-onset congestive heart failure, and sudden death
Management protocols are aimed at diagnosing underlying causes (including immunologic testing for Bartonella toxoplasma and viral causes), and providing supportive care for acute heart failure and management of arrhythmias (Luis Fuentes et al 2020).

Pericardial disease:

Pericardial disease is less common in cats than in dogs. The pericardium is essentially a "sac" of fibrous and connective tissue that surrounds the heart – and whose function is to lubricate the outer surface of the heart whilst it is contracting, to maintain the heart in a relatively stable position within the thoracic cavity, and to offer some protection from physical trauma. Disease of the pericardium may occur due to infection– where the pericardium becomes infected, inflamed and subsequently thickened and scarred; due to neoplasia– where the pericardial sac fills with fluid produced by neoplasia or unknown causes; foreign bodies, or trauma, among other conditions. When these things occur, the pericardium may restrict normal cardiac chamber filling, and can produce signs of reduced cardiac output and congestive heart failure typically right sided heart failure

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC):

ARVC is an uncommon idiopathic cardiomyopathy, similar to that described in dogs. The disease is characterised by right atrial and ventricular distension, right ventricular wall thinning, and the development of atrial/supraventricular or ventricular tachyarrhythmias. Right heart failure is common, with sudden death being a possible presenting sign. Thoracic radiography reveals right heart (and sometimes left heart) enlargement. Pericardial effusion may augment appearance of cardiomegaly on radiographs. Echocardiography reveals right atrial and ventricular dilatation, right ventricular wall thinning, and tricuspid regurgitation. Treatment involves managing acute heart failure, arrhythmias, and hypercoagulation (clopidogrel). Prognosis is generally guarded to poor once clinical signs of heart failure are present (Luis Fuentes et al 2020).

Acute Heart Failure: Clinical Signs

As has been alluded to above, the clinical signs in cats with congestive heart failure can be vague and non-specific, and include lethargy, depression, tachypnoea, cough, vomiting and open-mouth breathing, among other symptoms of respiratory distress associated with pleural fluid accumulation and pulmonary oedema (Luis Fuentes et al 2020).

Physical examination findings may include

- Respiratory distress, characterised by tachypnoea, increased bronchial sounds
    - Present in up to 33% of cases on presentation
    - Usually associated with moderate to severe left ventricular diastolic dysfunction
- Gallop or murmur on cardiac auscultation
    - Murmurs are detected in approximately 80% of cats with occult hypertrophic cardiomyopathy.
    - Causes of heart murmur include physiological (functional murmurs); congenital heart defects, and cardiomyopathy.
    - A gallop rhythm provides stronger suspicion of cardiomyopathy, especially in younger cats. Gallop rhythms are associated with increased ventricular stiffness, or increased left atrial pressure.
    - Other causes of gallop rhythm include hyperthyroidism, hypertension, anaemia and anxious or stressed states.
- Hypothermia
- Hepatomegaly
- Cyanosis
- Prolonged capillary refill time
- Dehydration
- Weak arterial pulses are common once congestive signs develop

As mentioned previously, thoracic radiographs and TFAST/cardiac/VetBLUE ultrasound are indicated to establish the diagnosis of CHF and to differentiate heart disease from respiratory or heartworm disease. Additionally, NT-pro BNP can assist in differentiating cardiac from non-cardiac disease also – in particular, a negative MT-pro BNP test result rules out cardiac disease. However the test does not reliably distinguish normal cats from those with mild disease (false positives)

Acute Heart Failure: Treatment

Emergency management of acute feline congestive heart failure generally follows the outline below (Luis Fuentes et al 2020):

1. Oxygen therapy: by oxygen cage, fly by or other low-stress method. Oxygen concentrations of 50-100% may be required initially, however oxygen concentrations should be reduced to 40% within 12 hours to avoid the potential for oxygen toxicity.
2. Stress reduction: butorphanol 0.05-0.2 mg/kg IV, IM or SC can be useful in providing mild sedation in cats that are overtly distressed on presentation
3. Thoracocentesis: If pleural effusion is detected, thoracocentesis should be performed to remove the effusion.
4. Diuretic therapy: Furosemide is the drug of choice in the emergency setting. Dose ranges quoted in literature vary widely, but a dose of 1 mg/kg IV or IM q 2-4 hrs., or 0.33-0.66 mg/kg/hr continuous infusion (following an initial IM or IV bolus of up to 1 mg/kg) are appropriate starting doses. Note that there is often considerable lag between the onset of diuresis and the resolution of respiratory distress – particularly in patients in which there is capillary basement membrane damage secondary to vascular hypertension in capillary beds of the lungs. Whether it is wise to continue high doses of furosemide beyond the establishment of diuresis (as evidenced by weight loss following administration) is a subject of ongoing discussion in literature. Current thought is that as clinical signs of respiratory distress and tachypnoea begin to resolve, the infusion or dose should be reduced or stopped for the remainder of the stabilisation period to avoid potentially harmful dehydration.
5. Preload reduction: transdermal glyceryl trinitrate may be used to reduce preload, although its effectiveness is questionable. Doses of intravenous glyceryl trinitrate 0.5-1.0 micrograms/kg/minute CRI (solution should be diluted to 100-300 micrograms/ml in 5% glucose) may be used as a potentially more efficacious treatment. Alternatively, a continuous infusion of nitroprusside may be used at a starting dose of 0.5- 2 micrograms/kg/minute (titrated up to 2 micrograms/kg/minute if required). Blood pressure monitoring is recommended.
6. Ventilation therapy: positive pressure ventilation therapy should be attempted in cats not responding to medical therapy within 1 hour of presentation, and continued for up to 12 hours following presentation (depending on response to therapy). Positive end-expiratory pressure of at least 5 cm water may be applied to facilitate aeration of alveoli.
7. Inotropic support: off-label use of pimobendan in cats with acute congestive heart failure lacks high grade evidence. However, clinical experience suggests cats with congestive heart failure may benefit from pimobendan in the following situations
    a. Cats without a heart murmur, or with a low-intensity murmur
    b. Cats affected by moderate to large pleural effusions
    c. Cats in cardiogenic shock (low body temperature, low blood pressure, low heart rate)

    Dobutamine can be used in place of pimobendan to provide inotropic support when indicated.
8. ACE inhibitors: are currently not recommended in acute heart failure, but may be commenced once the cat begins to eat, as part of a three-pronged pharmacology approach (furosemide, clopidogrel and ACE inhibitor) to chronic heart failure management in the cat. The aim of long-term ACE-inhibitor therapy is to reduce adrenaline and angiotensin-mediated peripheral vasoconstriction.
9. Heart Rate Control: following emergency stabilisation, diltiazem or beta-blockers such as atenolol may be used to attempt to reduce heart rate to increase the time of ventricular diastole, and thereby improve cardiac filling and cardiac output.
    a. Diltiazem: demonstrated clinical benefit in early studies, producing mild reductions in heart rate and contractility, thereby reducing myocardial oxygen demand. However, 3 x per day dosing reduces clientcompliance , and long-acting diltiazem has an unpredictable absorption and serum concentrations.

b. Beta-blockers. Beta blockers produce more profound heart rate reduction than calcium channel blockers and are preferred for cats with atrial fibrillation or marked tachycardia. By reducing catecholamine influence of heart rate and myocyte damage, beta blockers may reduce myocardial fibrosis. However, their use is not correlated with improved survival. Esmolol, sotalol or atenolol may be used.

10. Anticoagulation: aggressive anticoagulation is recommended in cats with clinical evidence of thromboembolic disease, and in those with echocardiographic evidence of left atrial spontaneous contrast
    a. Clopidogrel reduces platelet activation, reduces platelet degranulation, and inhibits modification of glycoprotein IIb/IIIa receptor which leads to reduced aggregation. Dose: 18.75 mg PO q 24 hrs
    b. Daltaparin (low-molecular weight heparin): Dose 150-175 IU/kg SC q 8-12 hrs
    c. Enoxaparin (low molecular weight heparin): Dose 1 mg/kg SC q 12 hrs
11. Management of cardiac arrhythmias as indicated

12. Other treatments
    a. Cilobradine – Cilobradine is a bradycardic agent derived from the calcium channel blocker, verapamil. It has been shown to slow heart rate in cats, whilst having minimal effects on vasomotor tone and myocardial contractility. Theoretically, a reduction in heart rate may reduce myocardial ischaemia, cardiac remodelling and arrhythmias in cats with cardiomyopathy. However, a recent study found that the use of Cilobradine in cats with cardiomyopathy following diagnosis of heart failure did not alter clinical outcome(mortality or morbidity) (Riesen et al. 2022)

Monitoring the Patient with Cardiac Failure

In-Clinic Monitoring in Patients with Acute Congestive Heart Failure

Monitoring patients with cardiac failure is an intensive exercise. For all cardiac patients, however, monitoring of the patient should include a full TPR and body systems evaluation every 15-30 minutes, until the patient is stable. The overall goals of our monitoring include the following -

Aims of monitoring and therapy are to:

- Decrease respiratory rate and respiratory effort; establish normal lung sounds and reduce cough
- Decrease heart rate, Improve arterial pulses, capillary refill time, and mucous membrane colour
- Improve level of consciousness
- Stabilize body temperature
- Normal hydration status (PCV/TP/skin turgor)
- Resolution of pre/renal azotemia – measure BUN/Creatinine/Phosphorus, and re-measure every 2 days
- Electrolyte changes - measure sodium, potassium magnesium, and chloride every q 12-24 hours and maintain within normal limits
- Normalise ECG abnormalities (see later)
- Urine output – urine output is a really useful monitoring tool. Urine output often reduces in acute heart failure – due to a reduction in blood flow to the kidneys due to decreasing cardiac output, and through release of hormones like "anti-diuretic hormone". In sick cardiac patients, insertion of a urinary catheter can assist monitoring urine output- particularly in patients with azotemia, or in patients that have not urinated, despite receiving diuretics like furosemide in their treatment protocols.
- Fluid therapy – many cardiac patients, despite having congestive heart failure, may be dehydrated. A clue to whether your patient is dehydrated or not may be gained by measuring the PCV and TP of your patients. An elevated TP +/- PCV may support a diagnosis of dehydration. Dehydrated patients require re-hydration. Rehydration should commence using low sodium containing fluids, e.g. 0.45% NaCl/2.5% glucose +/- potassium (given at 20 mEq/L in most instances) and magnesium supplementation as indicated by blood test measurements. Intravenous fluids should be administered at a rate of 0.75% to 100% of the patient recommended daily allowance (1.7- 2 ml/kg/hr). When starting a patient with heart failure on intravenous fluids, monitor heart rate, respiratory rate, lung sounds etc. very carefully every 10-15 minutes or so. ANY development of signs of stress (increased heart rate, respiratory rate etc.) or congestive heart failure (stress, tachypnoea, coughing, restlessness) should prompt the following actions…
    - Stop the intravenous fluids immediately
    - Administer furosemide 2 mg/kg IV - wait for 30-60 minutes, then re-start intravenous fluids at 5–75% of the previous rate
    - In severely ill patients, the following should be monitored
    - Parameters as outlined above PLUS
    - Insert a jugular catheter to measure central venous pressure
    - Insert ECG wires to facilitate 24 hr ECG monitoring
    - Insert urinary catheter to monitor urine output

Prognosis

Negative prognostic indicators for cats presenting with heart disease include the following:

- The presence of acute congestive heart failure
- Left atrial enlargement
- Reduced left atrial fractional shortening
- Reduced left ventricular fractional shortening
- Extreme left ventricular hypertrophy
- The presence of aortic or left atrial thromboembolism
- Atrial fibrillation

Negative prognostic indicators for cats that present with acute heart failure include:

- Hypothermia on presentation
- Thromboembolism
- Concurrent organ failure secondary to reduced cardiac output e.g. acute kidney injury etc.

The 5-year incidence of death from heart disease in cats diagnosed with HCM in the REVEAL study was 23% (Fox 2018). In cats that developsigns of acute congestive heart failure, or thromboembolic disease, or in patients with left ventricular systolic dysfunction had a mean survival time of less than 2 years in 2 studies Payne et al 2015; Payne et al 2015; Fox et al 2018).

Cats that presented with thromboembolism and acute congestive heart failure generally live for less than 6 months. However, some patients who respond to therapy for congestive heart failure, and are managed with clopidogrel may live for extended periods of time, with one study showing median survival time of 346 days, compared to aspirin-treated cats who had a mean survival time of 4 months (Hogan et al 2015)

References:

Guillaumin J, Gibson RM, Goy-Thollot I, Bonagura JD. Thrombolysis with tissue plasminogen activator (TPA) in feline acute aortic thromboembolism: a retrospective study of 16 cases. J Feline Med Surg 2019;21:340-346

Borgeat K, Wright J, Garrod O, Payne JR, Fuentes VL. Arterial thromboembolism in 250 cats in general practice: 2004-2012. J Vet Intern Med 2014;28:102-128

Riesen, S. C., et al. "Effect of cilobradine in cats with a first episode of congestive heart failure due to primary cardiomyopathy." Journal of Veterinary Cardiology 41 (2022): 179-193.

Ware, Wendy A., John D. Bonagura, and Brian A. Scansen. "Myocardial diseases of the cat." Cardiovascular Disease in Companion Animals. CRC Press, 2021. 649-694.

Novo Matos, J., et al. "Transient myocardial thickening in cats associated with heart failure." Journal of veterinary internal medicine 32.1 (2018): 48-56.

Häggström, Jens, Virginia Luis Fuentes, and Gerhard Wess. "Screening for hypertrophic cardiomyopathy in cats." Journal of Veterinary Cardiology 17 (2015): S134-S149.

Guglielmini, Carlo, and Alessia Diana. "Thoracic radiography in the cat: identification of cardiomegaly and congestive heart failure." Journal of Veterinary Cardiology 17 (2015): S87-S101.

Guglielmini, Carlo, et al. "Diagnostic accuracy of the vertebral heart score and other radiographic indices in the detection of cardiac enlargement in cats with different cardiac disorders." Journal of feline medicine and surgery 16.10 (2014): 812-825.

Fox, Philip R., et al. "International collaborative study to assess cardiovascular risk and evaluate long-term health in cats with preclinical hypertrophic cardiomyopathy and apparently healthy cats: the REVEAL study." Journal of veterinary internal medicine 32.3 (2018): 930-943.

Payne, Jessie Rose, David Charles Brodbelt, and Virginia Luis Fuentes. "Cardiomyopathy prevalence in 780 apparently healthy cats in rehoming centres (the CatScan study)." Journal of Veterinary Cardiology 17 (2015): S244-S257.

Payne, J. R., et al. "Risk factors associated with sudden death vs. congestive heart failure or arterial thromboembolism in cats with hypertrophic cardiomyopathy." Journal of Veterinary Cardiology 17 (2015): S318-S328.

Hogan, Daniel F., et al. "Secondary prevention of cardiogenic arterial thromboembolism in the cat: the double-blind, randomized, positive-controlled feline arterial thromboembolism; clopidogrel vs. aspirin trial (FAT CAT)." Journal of Veterinary Cardiology 17 (2015): S306-S317.

Feline Cardiogenic Arterial Thromboembolism

Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (Vet. Emergency and Critical Care; Medicine of Dogs)

Thrombosis is defined as a pathological clot formation leading to occlusion of blood supply and Ischemia. Feline cardiogenic arterial thromboembolism (CATE) is a well-recognized and devastating clinical morbidity with feline cardiomyopathy.

Aetiology

- Arterial thromboembolism is most commonly found in association with cardiomyopathy. The altered ventricular morphology, and subsequent alterations in rheology (blood flow dynamics) in cats with hypertrophic cardiomyopathy are thought to play a significant role in the pathophysiology of arterial thromboembolism in the cat
- Non-cardiac risk factors for thromboembolism in cats may also include
  - Post-ischemic injury
  - Conditions associated with antithrombin III loss (e.g. glomerulonephropathy), reduced synthesis (e.g. liver disease) or increased consumption (e.g. DIC)
  - Systemic inflammatory disease
  - Immune-mediated disease
  - Endocrinopathies e.g. diabetes mellitus, hyperthyroidism
  - Shock
  - Hepatopathy
  - Neoplasia e.g. pulmonary carcinoma

Pathophysiology of Feline Cardiogenic Thromboembolism

- **Endocardial Injury**
  - Cardiac endothelial injury- Turbulent blood flow within the left ventricle, and so-called "jet" lesions in the left atria due to mitral valve insufficiency, damage cardiac endothelium, and expose ventricular sub-endothelium, activating the intrinsic clotting cascade. Endocardial fibrosis may also affect the atrial and ventricular endocardial surfaces

- **Increased Blood Coagulability**
  - Altered blood flow is present in both dilated and constricted cardiac chambers, and promotes platelet aggregation and adhesion, and subsequent activation of intrinsic clotting pathway
  - Altered coagulation Disseminated intravascular coagulopathy due to consumptive coagulopathies (e.g. in sepsis, viral infection, poor peripheral blood flow, or vascular endothelial damage resulting from cardiac disease or other illness), hepatic coagulopathy or from the effects of thromboembolism, contributes to a state of systemic hypercoagulation
  - Vasoactive mediators - Serotonin and prostaglandins released from activated platelets, result in vasoconstriction of collateral vasculature about the site of a thromboembolism

- **Congestive Heart Failure – Diastolic Failure**
  - In hypertrophic cardiomyopathy, the stiff, non-compliant ventricular wall results in failure of diastole, and reduced end-diastolic volume, resulting in low stroke volume, and reduced cardiac output. Fractional shortening is generally greater with hypertrophic cardiomyopathy (70% vs. 50% in the normal cat) Reduced cardiac output results in increasing preload, left atrial dilatation, mitral valve insufficiency, and eventually systolic anterior motion of the mitral valve, left ventricular outflow tract obstruction.
  - Pulmonary oedema results from a combination of
    - Elevated left ventricular end-diastolic pressure
    - Renin-angiotensin-aldosterone system activation, leading to sodium and water retention
    - Mitral regurgitation
    - Left atrial distension and enlargement
  - Mitral valve insufficiency may also result in systolic anterior motion (SAM). SAM of the mitral valve is abnormal process of the anterior (septal) mitral valve leaflet being pushed or pulled into the left ventricular outflow tract during ventricular contraction. This motion obstructs flow out of the left ventricle (dynamic sub-aortic stenosis), contributing to further mitral valve dysfunction, and increasing preload
  - Pleural effusion is a common presenting sign. With high left ventricular end diastolic pressure (LVEDP), congestion of pulmonary veins results in pulmonary oedema and pleural effusion -due to hypertension in pulmonary visceral lymphatics

Clinical Signs of Cardiogenic Aortic Thromboembolism

- Symptoms of cardiac failure and pulmonary oedema may be superimposed on clinical signs of thromboembolism, and include
  - Tachypnoea
  - Dyspnoea
  - Exaggerated breathing efforts
  - Panting
  - Cough
  - Haemoptysis
  - Sudden death
- Symptoms of thromboembolism are dependent on the location of the thromboembolus
  - Common sites of thromboembolism are the caudal aortic bifurcation, forelimb, pulmonary, renal, and cerebral vasculature
  - Occlusion of the distal aorta causes symptoms of pain, paresis, absence of femoral pulses, cyanotic nail beds**ischaemic neuropathy, ischaemic myopathy** , flaccid paralysis of the hindlimbs, with hypo-reflexia, or areflexia of the hindlimbs. With partial occlusion due to micro-embolization, mild muscle weakness, "dropped hocks" and ataxia of one or both hindlimbs may be observed.
  - Occlusion of renal vasculature may result in oliguric or anuric renal failure
  - Occlusion of intestinal vasculature can cause bowel necrosis, diarrhoea, and abdominal pain
  - Heart murmurs, gallop rhythms, arrhythmias, tachypnoea, pleural effusion, or muffled heart sounds may be present on thoracic auscultation
  - Other non-specific symptoms unrelated to the location of a thrombus include
    - Tachypnoea
    - Vomiting
    - Vocalization
    - Open-mouth breathing – even in the absence of overt congestive heart failure

Diagnosis

- Clinical pathology may reveal
  - stress leukogram
  - elevated ALT/AST/CPK
  - azotemia
  - metabolic acidosis +/- respiratory alkalosis
  - hyperglycaemia (stress response)
  - thrombocytopaenia and DIC
- Thoracic radiography – may be consistent with that of cardiac disease, including left atrial enlargement; prominent pulmonary veins; and the presence of alveolar and interstitial lung patterns with congestive heart failure. The presence of lung neoplasia or pleural fluid accumulation may also be observed
- Ultrasonography may reveal
  - Ventricular wall thickening of the free wall, septal wall, or both
  - Left atrial enlargement with an increased LA:Ao ratio (greater than 2:1)
  - Reduced left atrial contractility
  - Spontaneous echogenic contrast (smoke) swirling in the left atrium
  - Visible thrombus
  - Right-sided atrial enlargement may also be evident in patients with pulmonary hypertension and thromboembolism
- Electrocardiography is indicated for arrhythmia detection
- Detection of thrombus location can be achieved with
  - Non-selective angiography- however, the procedure causes morbidity in many patients, ventricular tachyarrhythmias, and can cause mortality.
  - Contrast CT angiography may also be performed– but requires heavy sedation or anaesthesia, which may be contraindicated, depending on the clinical status of the patient
  - Regional blood flow determination using blood pressure doppler, or ultrasonography doppler may be useful
- **Note:** Gallop rhythms result from the atria contracting against elevated left end-diastolic pressure, which results from increased preload, left ventricular outflow tract obstruction and systolic anterior motion. They are relatively uncommon in cats without cardiac disease, and should prompt cardiac diagnostic work-up

Treatment

The goals of therapy for aortic thromboembolism include:

- Provide supportive patient care to stabilise the patient
- Prevent extension of the thrombus, and additional thromboembolic events
- To reduce the size of existing thrombi and restore perfusion when possible

Cats with thromboembolic disease are critical patients, that can readily succumb to fatal cardiac arrhythmias if stressed. They should be handled with extreme care at all times during stabilisation.
Additionally, complications of thromboembolism include permanent nerve damage, ischaemic reperfusion injury, acute kidney injury, limb necrosis with associated toxemia, and septicemia. These problems should be anticipated, and hospital staff and owners should be aware and informed.

Supportive Care
General supportive careinvolves treating cardiac failure, pulmonary oedema, providing fluid therapy for treatment of shock and dehydration, and provision of analgesia as follows:

1. Oxygen therapy
2. If the patient is determined to be in acute heart failure
    a. Diuretic therapy - furosemide IV/IM 1 mg/kg, repeat in 4-6 hours if necessary or continuous infusion as previously outlined
    b. Glyceryl Trinitrate, beta adrenergic blockers/calcium channel blockers may be used to manage heart failure as indicated
    c. Manage pleural effusion with thoracocentesis if present
    d. Beta-adrenergic blocker (atenolol, sotalol etc.) or calcium channel blocker (diltiazem) administration
3. Fluid Therapy: in the non-congestive heart failure patient, fluid therapy should be used to improve tissue perfusion, support blood pressure, correct electrolyte and acid/base abnormalities, and provide rehydration as indicated
4. Patient warming
5. Analgesia
    a. Analgesia should be provided using opioid medication with one of the following
        i. Butorphanol: 0.1 mg/kg IM/IV initially, followed by 0.05-0.1 mg/kg/hr CRI
        ii. Morphine 0.05-0.1 mg/kg IM q 4 hrs
        iii. Fentanyl 2 micrograms/kg IV bolus followed by CRI @ 2-4 micrograms/kg/hr
        iv. Methadone 0.1-0.2 mg/kg IV q 4 hrs.
        v. Buprenorphine 0.01-0.02 mg/kg IM, IV or transmucosal

Management of thromboembolism

Management of thromboembolism is achieved using the following:

1. Treat/aid prevention of thrombi in collateral circulation
    a. acepromazine 0.05-0.1mg/kg IV q 8 hrs - alpha adrenergic antagonist
    b. OR - hydralazine 0.5-0.8mg/kg q 8 hrs - direct acting smooth muscle relaxant
    c. OR - diltiazem - inhibits calcium dependent contraction of vascular smooth muscle and aids in inhibiting platelet aggregation by inhibiting calcium uptake by activated platelets, 7.5 mg/kg PO q 8 hrs
2. Anticoagulants
    a. Are *ineffective* in causing clot lysis, but may reduce the rate of new thrombi formation.
    b. Heparin is the drug most commonly used to prevent further enlargement of the thrombus. Heparin complexes with antithrombin, which in turn prevents fibrin formation; and also reduces thrombin-induced activation of platelets and factors V, VIII and XI. Heparin also stimulates the release of tissue factor inhibitor, which reduces thrombus initiation from sites of vascular injury.
    c. Unfractionated heparin
        i. Binds thrombin as well as antithrombin.
        ii. Optimal dosing schedules for animals are undefined
        iii. Heparin doses range from 75–500 IU/kg q 6–8 hours with unknown efficacy
        iv. Doses are given either intravenously or subcutaneously. IM doses are avoided due to the potential for haemorrhage after injection
        v. A continuous infusion of unfractionated heparin at 15 to 25 Units/kg/hour can be used and titrated to increase the PTT to 1.5–2 times the baseline value– although this form of monitoring does not consistently predict serum heparin concentrations
        vi. Because of its complex haemodynamics, the risk of profound haemorrhage and the difficulty in monitoring safety and efficacy, the use of unfractionated heparin is not recommended as a routine.
        vii. The long-term use of unfractionated heparin is likewise not usually advisable.
    d. Low molecular weight heparins
        i. Binds to antithrombin, but their small size prevents simultaneous binding to thrombin, thereby reducing the risk of bleeding.
        ii. They do not significantly affect clotting times
        iii. They have less protein binding and have a longer half-life than unfractionated heparin
        iv. Suggested doses for cats are
            1. Daltaparin: 150 IU/kg SC q 4 hrs.
            2. Enoxaparin: 1.5 mg/kg SC q 6 hrs.
    e. Direct factor Xa inhibitors
        i. There are several new factor Xa inhibitors that show predictable pharmacokinetics, and may show promise in cats
        ii. Optimal dosing has yet to be established in cats, but are being investigated
    f. Antiplatelet Drugs
        i. Antiplatelet drugs are used to prevent thrombosis in cases with high risk, and as secondary prevention then a thrombotic event has already occurred
        ii. Clopidogrel
            1. Is the preferred antiplatelet drug for use in cats.
            2. In the FATCAT study, clopidogrel at 18.75 mg/cat PO q 24 hrs. significantly reduced the occurrence of thromboembolism in cats that have a prior thromboembolic event.

3. In addition, clopidogrel more than doubled the time interval to thromboembolism recurrence to over 440 days, when compared to cats treated with aspirin
4. Clopidogrel is a platelet membrane receptor antagonist that inhibits ADP binding, which reduces platelet binding to fibrinogen and von Willebrand's factor and leads to reduced ADP-mediated platelet aggregation. It also reduces platelet release of serotonin, ADP and other factors that contribute to vasoconstriction and platelet aggregation
5. Clopidogrel is less likely to cause gastric ulceration than aspirin
6. May be used for longer-term patient management.

iii. Aspirin alters platelet function and reduces platelet aggregation by irreversibly inhibiting TxA_2 synthesis. TxA_2 promotes vasoconstriction and platelet aggregation through release of ADP. Aspirin may aid in promoting collateral circulation and decreasing size of the clot. Aspirin also inhibits vascular endothelial prostacycline synthesis. Note that prostacycline inhibits platelet aggregation and promotes vasodilatation. Aspirin is less effective than clopidogrel at preventing recurrence of thromboembolism.

3. Thrombolytic agents – attempts to dissolve the thrombus have been made for many years. Normal thrombolysis involves plasminogen activators catalyzing plasminogen conversion to plasmin. Plasmin hydrolyses fibrinogen and fibrin, aiding dissolution of thrombi. Several drugs have been trialed to emulate this process, thereby resulting in dissolution of the thrombus in FATE. For optimum efficacy, thrombolytic therapy should ideally be attempted within the first 6 hours of illness.
    a. Plasminogen activators used in therapy are urokinase and streptokinase. These agents require adequate circulating plasminogen to be effective. These drugs have minimal affinity for fibrin; therefore, they activate circulating, and fibrin-bound plasminogen indiscriminately, potentially inducing a systemic fibrinolytic state, and predisposing the patient to haemorrhage. They are no longer available in many countries.
    b. Tissue plasminogen activator (t-PA) - is an intrinsic protein present in all mammals - it is produced using recombinant DNA technology. It has lower affinity for circulating Plasminogen, has high affinity for fibrin bound in a clot (as with plasminogen) thereby by proximity potentiating conversion of plasminogen to plasmin by p-TA. The dose is 0.25-1 mg/kg, repeated once if required. Clinical trials are variable in terms of survival, with early studies reporting a high rate of complications, including bleeding, and severe reperfusion injuries. More recent literature, using lower doses, and a shorter time interval between FATE diagnosis and treatment report lower complication rates – similar to the current standard of care, and an overall mortality rate of 60%. However, case numbers are small, and studies both on efficacy and optimal dosing are currently ongoing.
    c. Reperfusion injury is a potential complication of thrombolytic agent therapy, and results from tissue depletion of oxygen in ischaemic tissues, leading to anaerobic metabolism due to depletion of ATP (necessary for oxidative phosphorylation). Depletion of oxygen, and inability to reform ATP leads to accumulation of products such as hypoxanthine within the cells - on reperfusion, and subsequent delivery of oxygen to the tissues, hypoxanthine is converted to xanthine in the presence of oxygen, leading to the formation of oxygen free radicals, and subsequent cellular damage. Release of activated clotting factors, toxic cellular metabolites, and vasoactive substances into systemic circulation contribute to the syndrome of reperfusion injury. Hyperkalaemia may result during reperfusion. During ischaemic myopathy, potassium will be released from damaged myocytes. On reperfusion, this potassium can enter systemic circulation, and can result in death within minutes of reperfusion

Prognosis

The prognosis for cats with arterial thromboembolic disease. In general, prognosis

a. Positive prognostic indicators include
    a. Prognosis is better when only one limb is affected
    b. Survival is better when some motor function is present in affected limbs
b. Negative prognostic indicators include
    a. Hypothermia (rectal temperature below 37.2 degrees Celsius is associated with 50% mortality
    b. Hyperphosphataemia
    c. Progressive azotemia
    d. Progressive hyperkalaemia
    e. Progressive limb injury
    f. Ischaemia sufficient to cause necrosis of limbs
    g. Significant embolization of kidneys, intestines or other organs carries a grave prognosis

With successful management -motor function may take 2-4 weeks to return, proprioception may take several months. 50% of affected cats will regain all or part use of the leg(s) within 1-6 weeks with rest, and treatment of their cardiomyopathy

Recurrence rate despite therapy for cardiomyopathy is 75% following the first episode. Recurrence occurs generally within a few months, but can be delayed with use of clopidogrel as previously outlined.

A recent retrospective study in cats showed that the presence of aortic thromboembolism, and the presence of reduced left atrial fractional shortening of less than 30% were independently associated with dying with aortic thromboembolism within 2 years. Additionally, cats that presented with symptoms of congestive heart failure on presentation, or with left atrial or ventricular systolic dysfunction were associated with dying from congestive heart failure within 2 years of diagnosis.

References:

Guillaumin J, Gibson RM, Goy-Thollot I, Bonagura JD. Thrombolysis with tissue plasminogen activator (TPA) in feline acute aortic thromboembolism: a retrospective study of 16 cases. J Feline Med Surg 2019;21:340-346

Borgeat K, Wright J, Garrod O, Payne JR, Fuentes VL. Arterial thromboembolism in 250 cats in general practice: 2004-2012. J Vet Intern Med 2014;28:102-128

Hogan DF, Fox PR, Jacob K, et. al. Secondary prevention of cardiogenic arterial thromboembolism in the cat: the double-blind, randomized, positive-controlled feline arterial thromboembolism; clopidogrel vs. aspirin trial (FAT CAT). J Vet Cardiol 2015;17:S306-S317.

Payne, J. R., et al. "Risk factors associated with sudden death vs. congestive heart failure or arterial thromboembolism in cats with hypertrophic cardiomyopathy." Journal of Veterinary Cardiology 17 (2015): S318-S328.

Luis Fuentes, Virginia, et al. "ACVIM consensus statement guidelines for the classification, diagnosis, and management of cardiomyopathies in cats." Journal of Veterinary Internal Medicine 34.3 (2020): 1062-1077.

Management of Life-Threatening Arrhythmias in Cats

Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (Veterinary Emergency and Critical Care; Medicine of Dogs)

Introduction

Cardiac arrhythmias are the result of abnormalities in cardiac conduction. Cardiac arrhythmias are classified according to the rate of the rhythm (tachycardia vs. bradycardia) and their origin (ventricular vs. supraventricular). They are also sub-classified as premature or escape, and refined according to fine location (junctional vs, non-junctional), and behavioural (re-entrant etc.).

Whilst many arrhythmias may not result in clinical signs of illness, clinically significant arrhythmias can produce a range of clinical signs referrable not only to the abnormal cardiac rhythm itself, but also related to underlying disease. It is the aim of this short article to focus on clinically significant, life-threatening cardiac arrhythmia diagnosis and management.

Causes of Arrhythmias in the Cat

Cardiac arrhythmias can develop as a result of cardiac disease, or non-cardiac disease

Cardiac causes of arrhythmias include:

- Cardiomyopathy (dilative or hypertrophic)
- Congenital heart defects
- Endocarditis
- Myocarditis – including traumatic/trauma-related myocarditis
- Pericardial disease
- Congestive heart failure from any cause
- Cardiac neoplasia

Non-cardiac causes of arrhythmias include:

- Hyperthyroidism
- Electrolyte abnormalities
    - Calcium disorders
    - Potassium disorders
    - Magnesium disorders
- Anaemia of any cause
- Trauma
- Neoplasia – primary cardiac, secondary metastatic neoplasia, paraneoplastic syndromes
- Drug/medication-related – anaesthetic medications, toxins, cardiac medications

Clinical Signs of Life-Threatening Arrhythmias

Cats may not show any symptoms of abnormal cardiac rhythm if the rhythm does not disrupt cardiac output to a significant degree. However, as cardiac output becomes impaired as a result of the arrhythmia, clinical signs may manifest in one or more of the following:

- Depressed mentation
- Weakness
- Collapse
- Dyspnoea
- Tachypnoea
- Sudden death

Physical examination findings may include:

- Tachypnoea
- Dyspnoea
- Abnormal lung sounds
  - Rales
  - Crackles
  - Reduced lung sounds
- Cyanosis
- Hypothermia
- Hypotension
- Weak and/or irregular pulses
- Evidence of underlying cause (urethral obstruction, thyroid enlargement etc.)

Diagnosis of Arrhythmias in the Cat

The diagnosis of an arrhythmia relies on electrocardiography. Further diagnostic tests are usually required to ascertain the presence of primary or secondary heart disease, and to also evaluate the patient for the presence of underlying disease, and also co-morbidities associated with poor cardiac output resulting from the arrhythmia.

The diagnostic tests that may be applied to the cat with abnormal cardiac rhythm includes:

- ECG analysis
- Cardiac ultrasound
- TFAST ultrasound
- Complete blood count
- Serum biochemistry analysis
- Serum electrolyte analysis
- Urine analysis
- Blood gas and blood lactate
- Blood pressure
- Abdominal ultrasound

Regarding ECG analysis, it is important to note that the cat has some peculiarities when compared to the dog:

- Sinus tachycardia is common in cats, due to anxiety and heightened arousal in the veterinary setting
- A heart rate of less than 160/min on physical examination may indicate relative bradycardia, and warrants investigation
- QRS complexes in normal cats may appear inverted/negative, because cats have a wide range of mean electrical axis within the ventricular muscle
- QRS complexes in cats can be very small. No minimum height of the R wave is recognised in cats in lead II

The Arrhythmias:

As in dogs, feline arrhythmias are classified as either ventricular in origin, or supraventricular in origin. Ventricular arrhythmias are characterised by wide, bizarre QRS complexes, whereas supraventricular arrhythmias are characterised by narrow (normal) QRS complexes.

In addition to the ventricular vs. supraventricular distinction, feline (as well as canine) arrhythmias are also classified according to the heart rate produced – bradyarrhythmias or tachyarrhythmias.

Bradyarrhythmias:

Sinus Bradycardia

Sinus bradycardia is a regular rhythm of normal appearance that originates from the sino-atrial node, but occurs at a rate that is inappropriately slow. Sinus bradycardia is abnormal in cats unless they are well acclimated to the practice, and the presence of a serious underlying disorder should be investigated.

Common causes of sinus bradycardia in cats are listed in the table below:

• Hypothermia
• Excess vagal tone (thoracic or abdominal disease, neoplasia etc.)
• Cardiomyopathy
• Trauma
• Shock (hypovolaemia, haemorrhagic)
• Anaphylaxis
• Neurological disease (including traumatic brain injury)
• Feline dysautonomia
	• Medications
	  ○ Beta blockers
	  ○ Calcium channel blockers
	  ○ Digitalis
	  ○ Morphine
	  ○ Methadone
	  ○ Alpha-2 agonists
	• Sepsis
	• Hyperkalaemia, hypokalaemia
	• Hypercalcaemia, hypocalcaemia

Clinical signs of sinus bradycardia usually include symptoms of lethargy and depressed appetite, along with symptoms associated with underlying disease e.g. urethral obstruction and associated hyperkalaemia.

Treatment is usually unnecessary unless clinical signs are evident. The requirement for treatment also depends on the underlying cause

- Acute treatment
  - Draw blood for analysis of electrolyte levels (calcium, potassium, acid/base status) and serum drug concentrations (digoxin)
  - Atropine at 0.01-0.04 mg/kg slow IV, isoproterenol may be administered in clinically unwell patients that do not respond to atropine in the acute setting. Glycopyrrolate 0.005-0.01 mg/kg IV may be used instead of atropine. Alternatively, terbutaline 5-10 micrograms/kg SC may be used.
  - Provide a fluid bolus – this is an essential component of acute management of severe sinus bradycardia caused by drugs, electrolyte disturbances, or head trauma. The fluid of choice is lactated Ringer's solution, or 0.9% sodium chloride. Administer a bolus of 5-7 ml/kg given rapidly intravenously over 10 minutes. The fluid bolus achieves dilution of serum electrolytes, dilution of drugs, and an immediate increase in preload, afterload, and cardiac output. Titrate fluid therapy beyond the initial fluid bolus according to the response to therapy and the fluid requirements of the patient
- Pacemaker therapy is rarely required, but may be considered in cases refractory to chronic medical therapy.

Atrial Standstill

Atrial standstill occurs when the atrial myocardium is unable to depolarize. QRS complexes may be normal (narrow and tall) or abnormal (wide and bizarre) in appearance. There are no P waves present.

Hyperkalaemia is the most common cause of atrial standstill in the cat – from endocrinopathy (DKA); urinary tract rupture, acute kidney injury, sepsis, urethral obstruction.

Clinical signs are similar to those of other bradyarrhythmias, and include weakness, ataxia, stupor, coma, sudden death. Symptoms of acute congestive heart failure may be present in patients with underlying heart disease.

An ECG diagnosis is made based on the presence of a slow heart rate, regular rhythm, and ventricular escape rhythms – wither junctional (normal QRS complexes) or non-junctional (wide QRS complexes). It is important that these complexes are not suppressed with lignocaine or other anti-arrhythmic drugs, or cardiac arrest will occur. In patients with hyperkalaemia, junctional escape complexes may have tall, spiked T waves – often similar in size to QRS complexes – coupled with abnormal (wide and bizarre) QRS complexes

Treatment is directed at identifying and correcting hyperkalaemia (if present), and correcting or managing the underlying cause.

- Acute Treatment
    - Obtain blood for serum biochemistry and electrolyte analysis
    - Treat hyperkalemia with an intravenous bolus of lactated Ringer's solution (or 0.9% NaCl) at 5-7 ml/kg IV over 10 minutes, followed by a glucose bolus (7-10% solution at 5 ml/kg IV over 10 minutes) together with regular insulin 0.1 unit/kg IV. Calcium gluconate 10% given at 0.2-1.0 ml/kg given SLOW IV may be used in refractory cases, where atrial standstill persists despite treatment for hyperkalaemia.
    - In patients without hyperkalaemia, administer atropine 0.01 mg/kg slow IV or SC as a trial
    - Cautious use of intravenous fluids in patients with underlying cardiac disease is advised due to the presence of atrial disease, and the risk of development of congestive heart failure.

Sinus Arrest and Sinus Block

Sinus arrest is defined as a complete loss of SA node automaticity. Sinus block results from failure of impulses formed in the sino-atrial node to depolarize the atria, or results in a delay in atrial depolarization. Either condition may produce loss of atrial depolarization, and ventricular asystole if secondary pacemakers do not initial ventricular depolarization.

Sinus arrest in the cat is caused most commonly by atrial disease, leading to failure of the SA node to depolarize for extended periods of time. As such, it is most commonly seen in cats with marked atrial enlargement secondary to heart disease (cardiomyopathy), in patients with neoplastic infiltration of the atrial myocardium, secondary to some cardiac medications (beta-adrenergic blockers, class 1a antiarrhythmic drugs etc.), and in patients with hyperkalaemia or increased vagal tone due to concurrent disease.

Clinical signs are referrable to intermittent decreases in cardiac output associated with the arrythmia, and include syncope, weakness and occasional loss of consciousness. Symptoms of congestive heart failure, and sudden death are also noted.

An ECG diagnosis is made based on the presence of sinus rhythm, containing intermittent periods of electrical inactivity lasting longer than 2 P-P intervals. Occasional junctional or non-junctional ventricular escape complexes may also be present. A syndrome of intermittent supraventricular tachycardia is observed in some patients (bradycardia-tachycardia syndrome).

Treatment involves identification of the underlying cause (electrolyte disorder, neoplasia etc.) and in patients with primary cardiac disease, anticholinergic drug therapy, +/- terbutaline and in patients in which the arrhythmia persists despite treatment of the underlying disease, pacemaker implantation is recommended.

Atrio-Ventricular Block (AV Block)

Atrio-ventricular block exists when there is a delay or complete block of atrial impulse conduction through the atrio-ventricular node

First degree AV Block

First degree AV block is characterised by a prolonged P-R interval. Causes include increased vagal tone, cardiomyopathy, electrolyte disturbances (hyperkalaemia), endocarditis (including traumatic endocarditis), beta-adrenergic blockers, calcium channel blockers, and sedative medications.

Treatment involves identification and management of the underlying cause.

Second Degree AV Block

There are three (3) types of second-degree AV block:

- Mobitz Type I (Wenckebach) - gradual increase in P-R interval until a P wave occurs without an R wave. The QRS complex is normal duration. Mobitz Type 1 AV block occurs within the AV node tissue
- Mobitz Type II - P-R interval constant but intermittent non-conducting P wave (P wave occurs without an R wave). Mobitz Type II AV block occurs within or below the Bundle of His (referred to as an infra-nodal block). Because the conduction block occurs below the His Bundle bifurcation, the QRS complexes may demonstrate abnormal morphology.
- Advanced 2nd Degree Heart Block - only one beat in a group is conducted to the ventricles - in ratios of 2:1, 3:1, 4:1 etc.

The cause of 2nd-degree AV block are similar to those causing 1st-degree AV block. Mobitz Type I AV block generally occurs secondary to increased vagal tone, or medications that depress AV node conduction, such as beta-adrenergic blockers, morphine, and calcium channel blockers. Mobitz Type II AV block is associated with organic heart disease, electrolyte abnormalities, and medications as for Mobitz Type I AV block.

Patients with 2nd-degree AV block usually present with symptoms referrable to reduced cardiac output +/- their underlying disease (if present) – with weakness, syncope and symptoms of congestive heart failure being most common.

Diagnostic evaluation should include full blood evaluation, urine analysis, echocardiogram and thoracic or abdominal imaging, depending on the suspected aetiology.

Treatment and clinical management of 2nd degree AV block involves increasing the heart rate, and management of the underlying cause

- Treatment of Mobitz Type I AV block is generally not required; monitor electrolyte and acid-base status and treat as required
- Mobitz Type II AV block is managed in the acute setting with administration of an atropine trial, glycopyrrolate, terbutaline or dobutamine administration.
- Management of symptoms of acute congestive heart failure may be managed by administration of furosemide, and benazepril as indicated by the patients' condition.
- Definitive management for long-term management is cardiac pacemaker implantation

Third Degree AV Block

Complete AV block occurs when all atrial impulses are blocked at the AV node or Bundle of His. An independent idioventricular pacemaker coordinates ventricular depolarization. As a result, the atria and ventricles are controlled by independent pacemakers.

- P waves, when present, have no fixed relationship to QRS complexes
- Ventricular rate is slow
- Ventricular escape beats are present. When ventricular escape complexes originate from the AV junction, they have normal morphology; when they originate from below the His Bundle bifurcation, they will be wide and bizarre in appearance

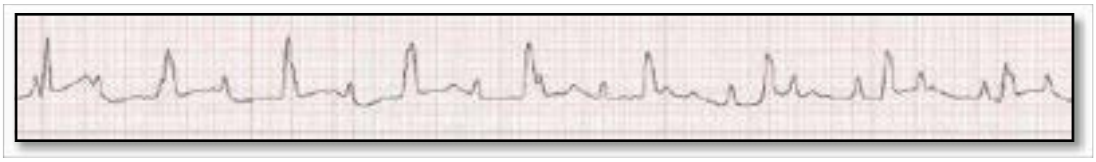


Image showing non-junctional escape rhythm at a rate of 120 beats per minute in a 15-year-old cat with HCM and 3rd degree AV block. Note the independent rates of atrial and ventricular depolarisations. Image: Anderson EL. Arrhythmias in Feline Cardiomyopathies. Guide to Canine and Feline Electrocardiography. 2018 Aug 15:301-14.

The causes of 3rd degree AV block are similar to those for 1st and 2nd degree AV block, including cardiomyopathy, atrioventricular infarction, cardiac inflammation, sepsis and neoplasia, along with advanced heart disease secondary to inherited cardiac defects.

As with other forms of AV block, clinical signs are generally referrable to the decreased in cardiac output, the severity of the underlying cause, and the rate of the ventricular escape rhythm. Typical clinical signs include weakness, syncope, collapse, seizures, congestive heart failure (tachypnoea etc.) or sudden death.

Diagnostic evaluation should include full blood evaluation, urine analysis, echocardiogram and thoracic or abdominal imaging, depending on the suspected aetiology.

Medical treatment has limited benefits for long-term management. However, emergency treatment and stabilization can be life-saving. The use of atropine, glycopyrrolate, or terbutaline in the emergency setting is preferred. Oral medication with propantheline bromide or terbutaline may be useful prior to cardiac pacing implantation.

Escape rhythms

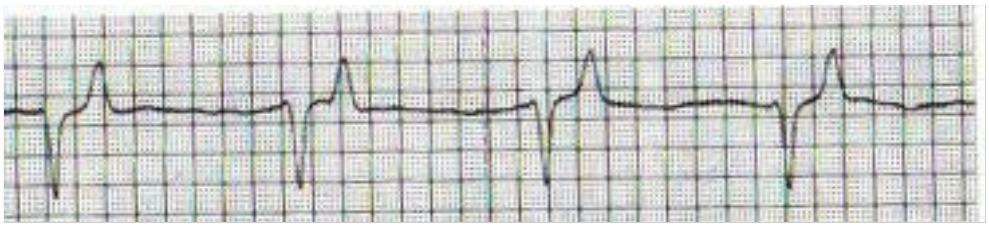
Escape rhythms arise from a physiologic (as opposed to pathologic) ectopic focus, when a superior focus (pacemaker) fails to fire. Escape rhythms originate from pacemaker cells that have a slower rate of discharge than that of the sino-atrial node, and therefore only appear when there is a sinus arrest, or sinus (sino-atrial, or atrioventricular) block. The ectopic pacemaker foci exist in the heart at one of two levels – supraventricular, or ventricular.

For junctional supraventricular escape rhythms, the QRS is generally narrow, with or without a P wave; the escape rhythm rate is 40-70 beats per minute (bpm)

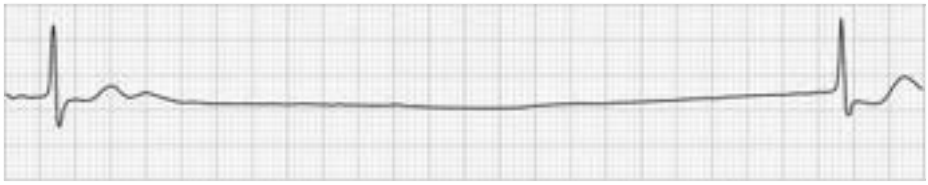
For ventricular escape rhythms, the QRS is generally wide, and resembles a VPC in appearance; the escape rhythm rate is 20-40 bpm.

Escape rhythms are generally regular. However, the ECG tracing will depend on the presence and type of rhythm disturbance that lead to the appearance of the escape rhythm in the first place

ECG of ventricular escape complexes



Non-junctional escape complexes in a patient with sinus arrest. Image: Tilley, L.P., "Essentials of Canine and Feline Electrocardiography" Tilley (Ed) LWW (1992)



Junctional escape complexes in a patient with sinus arrest. The narrow QRS complexes with T-waves suggests ventricular depolarization originates in junctional (AV node) tissue

Tachyarrhythmias

Pathological tachycardia should be suspected in the cat if the heart rate is above 240 beats per minute, in the absence of an identifiable systemic disease. Tachyarrhythmias can originate in the ventricular muscle (ventricular tachycardias) or in the supraventricular tissue (supraventricular tachycardia).

Supraventricular Tachyarrhythmias:

The most common clinically significant supraventricular tachyarrhythmias are:

1. Atrial tachycardia
2. Atrial flutter
3. Atrial fibrillation

The main difference between supraventricular tachycardia, atrial flutter, and atrial fibrillation is the rate at which the ectopic focus depolarizes. Standard definitions for atrial depolarisations in supraventricular tachycardias in cats are not described, but in dogs, the rate of pacemaker firing for supraventricular tachycardia is between 150–350 beats per minute; for atrial flutter, the rate is greater than 350 beats per minute, and for atrial fibrillation, the rate is generally greater than 500 per minute. The ventricular response-rate produced by AV nodal depolarisation in response to atrial depolarisation is variable, but typically leads to ventricular rates of 180-280 depolarisations per minute. In atrial tachycardia and atrial flutter, the ventricular rate is fairly regular; whereas in atrial fibrillation, the ventricular rate and pattern is highly variable (irregularly irregular).

The causes of supraventricular tachycardia are listed in the table below:

• Chronic obstructive pulmonary disease • Myocardial hypoxia of any cause • Shock • Sepsis • Cardiomyopathy with atrial distension • Congestive heart failure • Myocarditis (including traumatic myocarditis)	• Congenital heart defects • Electrolyte abnormalities (hypokalaemia, hyperkalaemia) • Pericardial effusion • Cardiac or metastatic neoplasia • Medications e.g. barbiturates • Systemic inflammation

Clinical signs associated with supraventricular tachycardias are related to the underlying disease, as well as reduced cardiac output, which occurs due to reduced diastolic filling time of the ventricles (in response to rapid stimulation of the AV node by rapid supraventricular depolarisations). As such, symptoms of acute congestive heart failure, weakness, lethargy and end-organ dysfunction (acute kidney injury etc.) may be present.

Diagnostic evaluation of the patient with supraventricular tachycardia involves complete blood evaluation (CBC, serum biochemistry, electrolytes), urine analysis, and diagnostic imaging. Further diagnostic tests are selected based on physical examination and diagnostic evaluation.

ECG characteristics of supraventricular tachyarrhythmias include:

- Variable, rapid atrial depolarisation rate
- The presence of P' or F waves, or complete absence of P waves (atrial fibrillation) coupled with rapid ventricular ate with normal QRS morphology
- Normal QRS complexes occurring at a rapid rate
- P in T complexes, where the P wave is embedded in the preceding T wave (seen in atrial tachycardia)
- Occasional ventricular ectopic depolarisations

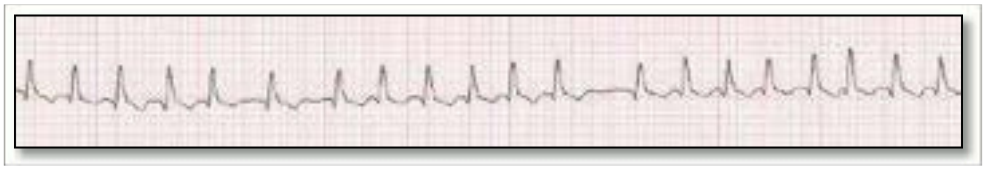


Image showing atrial fibrillation, with ventricular response rate of 240 beats per minute in a 5-year-old cat with HCM and congestive heart failure. Image: Anderson EL. Arrhythmias in Feline Cardiomyopathies. Guide to Canine and Feline Electrocardiography. 2018 Aug 15:301-14.

Treatment of supraventricular tachycardia has the following goals

- To diagnose and manage the underlying cause
- To slow ventricular depolarisation rate, in order to improve cardiac output
- To slow the atrial depolarisation rate

Treatment recommendations are outlined below:

1. Oxygen supplementation
2. Diltiazem
    a. If heart rate >180/min with evidence of poor perfusion (hypothermia, poor pulse quality, evidence of azotemia, poor mention etc.)
    b. Diltiazem 0.1-0.4 mg/kg IV slowly over 10 minutes, followed by continuous infusion at 1-5 micrograms/kg/minute
3. Beta-adrenergic blockers
    a. May be used instead of diltiazem
    b. In cats with known systolic dysfunction, esmolol is preferred, due to its short duration of action
    c. Esmolol is used at a dose of 50-500 micrograms/kg slow IV over 1 minute, followed by a continuous infusion @25-200 micrograms/kg/minute
    d. Propranolol is used if systolic function is normal at 0.02-0.1 mg/kg IV over 5-10 minutes; or 0.1-0.2 mg/kg PO q 8-12 hrs
4. Procainamide
    a. May be used instead of diltiazem
    b. 1-2 mg/kg slow IV over 10 minutes followed by continuous infusion of 10-20 micrograms/kg/minute
    c. Procainamide can cause vasodilatation and hypotension if given too rapidly
5. Furosemide
    a. Used if congestive heart failure is present
    b. 2 mg/kg IV bolus OR
    c. Continuous infusion of 0.66-1.0 g/kg/hr
6. Glyceryl trinitrate
    a. Used if congestive heart failure is present
7. Dobutamine
    a. Used if patient is in cardiogenic shock
    b. 2.5 micrograms/kg/minute continuous infusion; titrate the dose upwards to maintain systolic arterial pressure >90 mm Hg

Ventricular Tachycardia

Ventricular tachycardia is defined as 3 or more ventricular premature complexes (VPCs) in a row. Ventricular tachycardia may be intermittent (paroxysmal) or sustained. Typically, QRS complexes are wide and bizarre in appearance; with the heart rate being rapid.

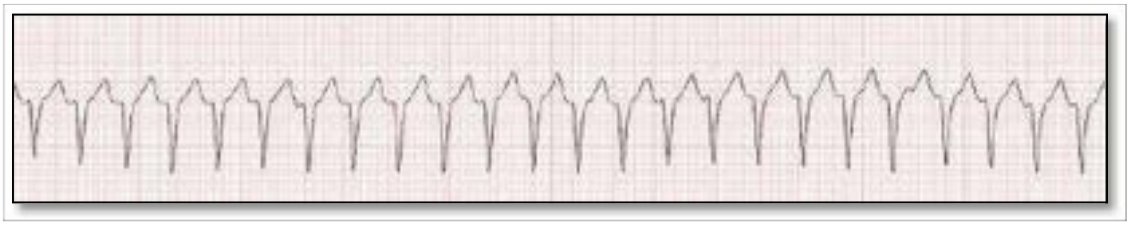


Image showing ventricular tachycardia at a rate of 240 depolarisations per minute in a 14-year-old cat with HCM and congestive heart failure. Image: Anderson EL. Arrhythmias in Feline Cardiomyopathies. Guide to Canine and Feline Electrocardiography. 2018 Aug 15:301-14.

The causes of ventricular tachycardia are listed in the table below:

• Cardiomyopathy
• Traumatic myocarditis
• Shock
• Sepsis
• Systemic inflammatory disease
• High sympathetic tone
• Anaemia
	• Electrolyte disorders
	  ○ Hypokalaemia
	  ○ Hypomagnesaemia
	• Metabolic acidosis
	• Pain
	• Medications – barbiturates, sympathomimetic agents etc.

Clinical signs associated with ventricular tachycardia include those associated with underlying disease, as well as with reduced cardiac output, owing to reduced diastolic filling time due to tachycardia, and include lethargy, collapse, signs of acute heart failure, or sudden death.

The diagnostic approach to the patient with ventricular tachycardia is aimed at diagnosing potential predisposing causes, as well as cardiac assessment, and include complete blood count, serum biochemistry, electrolyte analysis, urine analysis and diagnostic imaging as appropriate.

Treatment of ventricular tachycardia is recommended if the ventricular rate is in excess of 220-240 beats per minute, and associated with symptoms of perfusion abnormalities, and cat involves:

1. Oxygen supplementation
2. Diagnosis and management of underlying disease or predisposing condition e.g. correct electrolyte disorders, provide analgesia, fluid resuscitation, transfusion therapy etc.
3. Antiarrhythmic therapy is attempted using either of the following
    a. Procainamide 3-8 mg/kg slow IV over 10 minutes
    b. Propranolol 0.25-0.5 mg/cat slow IV over 10 minutes
    c. Esmolol 50-500 micrograms/kg IV over 1 minute; followed by continuous infusion @ 25-200 micrograms/kg/minute
    d. Lidocaine 0.25 mg/kg initial dose – increasing to 1.0 mg/kg if required; administered slowly IV followed by continuous infusion @ 10-40 micrograms/kg/min

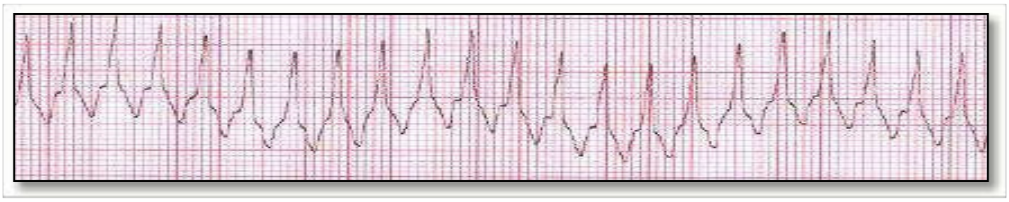


Image of rapid ventricular tachycardia in a cat with arrhythmogenic right ventricular cardiomyopathy – an uncommon primary myocardial disease of cats, in which right ventricular myocyte death occurs, with replacement by fatty deposits and fibrous tissue. Image: Anderson EL. Arrhythmias in Feline Cardiomyopathies. Guide to Canine and Feline Electrocardiography. 2018 Aug 15:301-14.

References and Suggested Reading:

1. Côté E. Feline arrhythmias: an update. Veterinary Clinics: Small Animal Practice. 2010 Jul 1;40(4):643-50.
2. Anderson EL. Arrhythmias in Feline Cardiomyopathies. Guide to Canine and Feline Electrocardiography. 2018 Aug 15:301-14.
3. Kittleson MD, Côté E. The Feline Cardiomyopathies: 3. Cardiomyopathies other than HCM. Journal of Feline Medicine and Surgery. 2021 Nov;23(11):1053-67.
4. Sleeper MM. Management of Life-Threatening Arrhythmias. Feline Emergency and Critical Care Medicine. 2022 Oct 7:169-76.
5. McMichael M, Fries R. Life-threatening Cardiac Emergencies for the Small Animal Practitioner. John Wiley & Sons; 2016 Aug 1.
6. Pariaut R, Saelinger C. Cardiovascular disorders. In Clinical Medicine of the Dog and Cat; Schaer/Gaschen/Walton (Ed) 2023 (pp. 237-288). CRC Press.

Feline Respiratory Emergencies

Dr. Philip R Judge BVSc MVS PG cert Vet Stud MACVSc (VECC; Medicine of Dogs)

Upper Airway Disease

Key Clinical Points

- Loud respiratory noises that are able to be heard without a stethoscope usually reflect upper airway obstructive diseases, located in the…
  - nasal cavity
  - nasopharynx
  - oropharynx
  - larynx.
- Respiratory difficulty associated with upper airway obstruction generally occurs on inspiration.
- Other clinical signs associated with upper airway disease may include sneezing and nasal discharge, (in animals with nasal disease), or altered vocal character (voice change), decreased vocalization, abnormal purr, loss of meow, or dysphagia (in animals with laryngeal diseases).

Nasal Cavity Diseases

Animals with nasal disease causing obstruction and respiratory distress generally have decreased nasal airflow on physical examination. Cats with nasopharyngeal disease can generally breathe well through the mouth but exhibit distress when one nostril is occluded or when the mouth is held closed. This pattern of clinical examination (alleviation of clinical signs with open-mouth breathing, but worsening with a closed mouth) localises the disease to the nasopharynx. Whenever possible, the caudal aspect of the soft palate should be palpated for abnormalities. Generally, the soft palate is easily depressed into the roof of the nasopharynx with digital palpation. A nasopharyngeal mass or polyp can be felt as a space-occupying lesion dorsal to the soft palate.

Conditions involving the nasal cavity that can result in respiratory distress in cats include

- Nasopharyngeal stenosis – which may be congenital or acquired as a result of chronic upper respiratory disease or regurgitation/vomiting into the nasopharynx. Mucopurulent nasal discharge may be present, but it is generally uncommon. The condition is diagnosed on nasopharyngeal endoscopy or CT scan. Treatment is balloon dilatation or surgical removal of stenotic scar tissue or cartilage.
- Nasal Neoplasia – Nasal neoplasia presents with clinical signs of nasal disease, including sneezing, nasal discharge and occasionally epistaxis, along with facial asymmetry (70% of cases). Behaviour changes and seizures may also be seen, and are highly suggestive of neoplastic invasion into the central nervous system. Tumour types encountered include
  - Lymphosarcoma
  - Adenocarcinoma
  - Squamous cell carcinoma
  - Fibrosarcoma

  Diagnosis is confirmed using CT and tumour biopsy. Most nasal tumours will exhibit local tissue invasion, but metastasis to lymph nodes and lungs may also occur. The most common recommended treatment for nasal tumours is radiation therapy. Median survival time is 9-23 months. Nasal lymphoma may also be managed with chemotherapy.
- Mycotic rhinosinusitis – aspergillosis is rare in cats, but is an emerging disease, most common among Persian or Himalayan breeds. The disease affects young to middle-aged cats, with immune compromise and previous viral upper respiratory tract infection or recurrent antimicrobial therapy

thought to be additional risk factors. Diagnosis is typically based on biopsy, cytology, culture and CT imaging.

- Cryptococcus infection is likewise rare in cats, and causes chronic nasal disease, as with aspergillosis, often accompanied nasal discharge, skin lesions, chorioretinitis or central nervous systemic signs, including seizures. Diagnosis involves cytological identification and/or serum agglutination tests. Treatment with itraconazole is efficacious in most cases.
- Nasopharyngeal polyps - typically affect young cats. They are pedunculated fibrous inflammatory tissue that originates in the Eustachian tube, and invades the nasopharyngeal area. They can lead to unilateral or bilateral nasal obstruction - and as a result can cause stertorous inspiration and inspiratory dyspnoea +/- serious nasal discharge. In severe cases, persistent open-mouth breathing is necessary for ventilation, leading to inappetence and poor body condition.
- Acute infectious upper respiratory tract disease is a significant cause of morbidity in cats. The primary organisms responsible for feline respiratory infections are:
    - Feline herpes virus - also called feline rhinotracheitis virus, or FHV-1
        - FHV-1 generally induces the most severe respiratory disease in cats
        - FHV-1 does not survive for longer than 18-hours in a warm damp environment, and is readily inactivated by common disinfectants
        - Once a cat becomes infected with FHV-1, and recovers, it will invariably become a carrier of the virus. Carriers of viral infections remain clinically healthy much of the time, but will intermittently shed virus into the environment, particularly during times of stress, treatment with corticosteroids or concurrent illness. Approximately 1 week after the onset of stress, infection or corticosteroid therapy, these cats will begin shedding virus, and are a source of contagious infection to other cats.
    - Feline Calicivirus (FCV)
        - FCV is the most common respiratory infectious agent in cats
        - FCV can survive for up to 7 days in the environment
        - FCV is more resistant to many disinfectants that FHV-1, but is readily inactivated by 1:32 dilution of hypochloric acid (bleach)
        - Many strains of FCV exist - vaccination does not offer full protection against all strains of the virus.
        - Carrier states exist following FCV infection. The carrier animals usually shed virus continuously - rather than intermittently as with FHV-1. The carrier state usually last approximately 100-300 days, with up to 20-30% of cats remaining lifelong carriers.
    - Bordetella bronchiseptica
        - Is a gram negative aerobic bacteria usually associated with canine kennel cough, but is also a primary pathogen in feline respiratory infections
        - Up to 9% of cats may be carriers of the bacteria and may shed into the environment, acting as a reservoir of infection for other cats
    - Chlamydophila felis (formerly Chlamydia psittaci var. felis)
        - Chlamydophila felis is usually associated with conjunctivitis in cats, but can cause respiratory symptoms

Clinical signs observed differ depending on the causative organism, and include:

- Feline Herpes virus
  - Incubation period = 2-6 days
  - Early signs include depression, sneezing, anorexia, serous ocular and nasal discharge
  - Later signs include excessive salivation and drooling, conjunctivitis, copious ocular and nasal discharge, which becomes mucopurulent, occasional cough and oral ulceration
  - Chronic sequelae include chronic nasal discharge if damage to nasal turbinates has been severe. This is often accompanied by chronic bacterial nasal infections
- Feline calicivirus
  - Pyrexia
  - Depression
  - Oral ulceration – much more common that with FHV-1
  - Conjunctivitis
  - Anorexia and salivation may occur due to oral discomfort resulting from oral mucosal ulceration
  - Sneezing and conjunctivitis may occur but are less severe than with FHV-1 infection
  - Facial swelling and oedema may occur, along with pyrexia and signs of respiratory infection in severe infections. Severe infections are also associated with pancreatitis, pneumonia and inflammation of the pericardium (pericarditis)
- Bordetella infection can result in sneezing, nasal and ocular discharge, through to cough, severe dyspnoea and clinical signs of pneumonia. Symptoms are most likely to be severe in young cats or kittens

Treatment of acute infectious upper respiratory tract disease includes

- Anti-viral chemotherapy has received some attention in the management of feline viral respiratory disease, with varying success. Currently, no specific anti-viral treatment is in widespread use, although systemically administered famciclovir has been trialled in Feline Herpesvirus infections and resulted in reduced duration and severity of respiratory illness in virus-affected cats. Likewise, topical antiviral agents such as cidovir are very effective in reducing severity of clinical signs associated with feline herpesvirus.

Other treatments under investigation include

- Lysine – mixed results; one clinical review suggested the treatment was ineffective
- Interferon – both feline and human interferon have shown good efficacy in reducing disease severity, especially in feline herpesvirus infections

General treatments to support cats with infectious respiratory disease are as follows

- Antibiotic therapy
  - Indicated to help control secondary bacterial infection.
  - Tetracycline antibiotics– especially doxycycline- are the antibiotics of choice for Bordetella infection in dogs but may not effectively treat the organism in cats.
  - Enrofloxacin is the preferred agent at present in cats

- Nursing care
  - Intravenous fluid therapy is indicated in cats unable to eat or drink
  - Analgesia – opiate analgesia may be indicated, especially for painful oral ulcers
  - Nebulization or steam therapy may be indicated to break down thick mucus and facilitate clearance from the airways
  - Cleaning the oral cavity with 0.05% chlorhexidine may reduce bacterial populations in severely moribund cats
  - Cleaning or the external nares with saline-soaked swabs

Laryngeal Disease

Cats with laryngeal disease present with variable degrees of respiratory distress – usually inspiratory dyspnoea +/- noise whilst breathing, exercise intolerance (although this can be difficult to detect in cats), tachypnoea, and cough. Gagging and dysphagia may also be seen. A change in vocal quality/sound of meow, or an alteration in purring may also be detected. In cats with laryngeal neoplasia, a soft tissue mass may be palpable in the region of the larynx. Diagnosis is usually confirmed by laryngoscopy and biopsy.

The most common causes of laryngeal disease causing respiratory distress in cats include

- Laryngeal mass
  - Due to inflammatory laryngitis – often caused by laryngeal neoplasia
  - Laryngeal neoplasia – the most common neoplasia of the larynx in cats are lymphosarcoma and squamous cell carcinoma. Chemotherapy may be helpful in reducing tumour size in lymphoma. COX-2 inhibition with Piroxicam 0.3 mg/kg/day may be of some benefit in squamous cell carcinoma, along with debulking surgical treatment. Frequently, tracheostomy is required – although cats are susceptible to stoma occlusion more so than dogs.
- Laryngeal paralysis – is relatively rare in the cat, and is most commonly an acquired condition secondary to trauma or neoplasia along the pathway of the recurrent laryngeal nerve. Radiographs may reveal caudal retraction of the larynx during inhalation, and/or gas distension of the oesophagus. The diagnosis is made in a similar manner to dogs- by direct visualization of failure of abduction of arytenoid cartilages during inhalation, when the cat is at a light plane of anaesthesia. Laryngeal ultrasound or radiography may be used to detect soft tissue neoplasia that be a complicating causative factor. Conservative management with weight loss and anti-inflammatory therapy, combined with surgery is associated with good outcome.

Tracheal Disease

Cats are uncommonly affected with intraluminal tracheal disease causing respiratory distress, being more commonly affected by extra-luminal thyroid mass compression of the trachea, and traumatic injury to the rachea. Causes of intraluminal obstruction include neoplasia (most commonly lymphoma, adenocarcinoma and squamous cell carcinoma, and less commonly fibrosarcoma, chondrosarcoma and plasmacytoma), and granulomas

Clinical signs include stridor, loud breathing, cyanosis, and coughing. Neck or chest radiographs may show an intraluminal mass outlined by air however mural masses may become relatively large before they become visible.

The diagnosis of tracheal disease requires visualization and biopsy. Tracheal resection and anastomosis can be attempted to relieve obstruction caused by tracheal neoplasia. Lymphoma and mastocytoma may respond to chemotherapy.

Lower Airway Disease

Feline bronchial syndrome (feline asthma or bronchitis) is a common cause of respiratory distress and/or coughing in cats. In fact, cats with cough most frequently have lower airway disease over other types of airway and lung disease. Cardiac disease is an uncommon cause of cough in cats, although it is not uncommon for airway disease and cardiomyopathy to co-exist. Other differentials for cough include lungworm or heartworm infection, bacterial infection (e.g., *Bordetella*), lung or tracheal tumors, and rarely pleural effusion. However, underlying cause surrounding the development of lower airway disease remains undetermined in most cats.

Lower airway disease will increase airway resistance, by narrowing the conducting airways, secondary to bronchoconstriction (reversible), increased airway mucus, and/or airway smooth muscle hypertrophy.

Aetiology

Feline asthma is thought to be an allergic disorder based on evaluation of cytology and histopathology, and the apparent response to glucocorticoid therapy. Clinical signs are associated with airway constriction and obstruction linked to smooth muscle hypertrophy, excessive airway mucous production secondary to mucous gland hypertrophy, and bronchial wall oedema. Some patients progress to having severe lower airway obstruction, lung hyper-inflation, emphysema and bronchiectasis.

Clinical Syndrome

Feline bronchial syndrome typically has the following clinical presentation:

- Age: young to middle-aged cats
- Clinical signs:
    - Cough
    - Increased respiratory effort
    - Open-mouth breathing
    - Dyspnoea – typically both inspiratory and expiratory components of increased effort; wheezing and/or crackles may be auscultated
    - Cyanosis

Diagnostic Evaluation

Thoracic radiographs are a mainstay of diagnosis. Radiographic features of bronchial disease include

- A bronchial or broncho-interstitial pattern
- Lung hyperinflation due to air-trapping
- Occasionally, collapse of the right middle lung lobe due to mucus plugging.

Laboratory testing is commonly unremarkable in cats with lower airway disease, but should be performed to exclude co-morbidities. Occult heartworm antibody and antigen testing is indicated in endemic areas, even in indoor cats. Biermann faecal sedimentation may also be performed to evaluate for lung worm. Older cats may be evaluated for hyperthyroidism.

Tracheal cytology (and perhaps culture) may be performed to evaluate cats for airway eosinophilia, and lack of other causes of respiratory distress (infection, neoplasia etc.). Note that airway cytology should not be collected from cats that showing respiratory distress, as the procedure (broncho-alveolar lavage or tracheal wash) may temporarily worsen lung function.

Treatment

Acute therapy includes supplemental oxygen therapy, glucocorticoids and bronchodilators as outlined below.

1. Provide oxygen therapy
2. Methylprednisolone sodium succinate 5 mg/kg slow IV q 8 hrs, then transition onto oral medication within 12 hrs
3. Bronchodilatation: terbutaline 0.01 mg/kg subcutaneously q 6-8 hrs as required; or inhaled salbutamol or albuterol
4. Sedation: butorphanol 0.05 mg/kg IV can produce mild sedation which reduces dynamic airway collapse.

Key Therapeutic Points

- Limit exposure to airway irritants.
- Oral steroids (prednisolone 5 mg q 12 hours X 10 days, then tapered to 2.5 to 5 mg once a day. It is likely that cats will need life-long therapy, and the goal is NOT to get them off steroids, but rather control airway inflammation.
- Inhaled steroids (fluticasone 110–220 µq q 12 hours, using a feline spacer (e.g. Aerokat) may be substituted. In this case, fluticasone and oral prednisolone are administered for 7-14 days, followed by tapering of the oral prednisolone.
- Oral cyclosporine has been documented as a safe and effective treatment for feline asthma in a cat with diabetes mellitus and congestive heart failure, where the use of corticosteroids was considered relatively contraindicated
- Doxycycline or azithromycin if infection is suspected (uncommon).

Drugs, Dosages and Indications

Drug	Drug class	Dose range	Frequency	Route	Indications
Prednisolone	Glucocorticoid	0.25–1.5 mg/kg	Q 24-48 hrs	Oral	Long-term
Fluticasone	Inhaled glucocorticoid	110 mcg (rarely 44 mcg or 220 mcg)	Q 12 hrs	Inhaled	Long term
Terbutaline	Beta-2 adrenergic agonist	0.01 mg/kg	PRN	SC/IM	Acute respiratory distress
Albuterol	Beta-2 adrenergic agonist	1 "puff"	PRN	Inhaled	Acute respiratory distress
Theophylline	Bronchodilator	10-20 mg/kg	Daily at night	Oral	Bronchodilatation

Conclusion

Feline lower airway disease represents a heterogeneous mixture of lower airway disease, with some cats have a more compelling "asthma" phenotype with acute bronchoconstriction and some cats having a more "chronic bronchitis" picture with cough and excessive airway mucus. Diagnostic efforts should be directed at excluding other potential causes of cough or respiratory distress.

Pulmonary Parenchymal Disease

Cats can suffer from a range of pulmonary parenchymal diseases, including pneumonia, pulmonary infiltrates with metastatic neoplasia, neurogenic pulmonary oedema and heart failure, among many others.

Aspiration Pneumonitis and Pneumonia

Aspiration pneumonitis and pneumonia are consequences of aspiration of chemical irritants, such as acidic stomach contents, water (salt or fresh water), or hydrocarbons. The pathophysiology of both of these conditions involves

- Initiation of localised inflammation, with resultant impairment of respiratory function
- The possible development of acute respiratory distress syndrome and/or sepsis

Risk factors for the development of aspiration pneumonitis/pneumonia include the following…

- Gastric pathology – including delayed gastric emptying due to conditions such as stress, trauma, intestinal foreign body, gastric outflow obstruction, pregnancy, obesity etc.
- Oesophageal pathology – including oesophageal foreign body, megaoesophagus, gastroesophageal reflux, drug administration-induced oesophageal stricture, myasthenia gravis, nasogastric tube placement etc.
- Impaired consciousness – including general anaesthesia or sedation, head trauma, seizures, etc.
- Impaired laryngeal function and airway protection – including paralysis in conditions such as botulism, snake bite, tick paralysis, anaesthesia, tetanus, laryngeal paralysis etc.

Predisposing Causes for Aspiration Pneumonia

Gastrointestinal	Nervous system	Altered airway defenses
• Reflux esophagitis • Oesophageal motility disorder • Vascular ring anomalies • Esophageal foreign body • Megaoesophagus • Cricopharyngeal achalasia • Chronic vomiting • Cleft palate • Force feeding • Nasoesophageal feeding • Laryngeal paralysis	• Altered consciousness • Seizures • Coma • Immobility • Myasthenia gravis • Polyradiculoneuritis • Botulism • Elapid envenomation • Ixodes tick paralysis • Tetanus	• Anaesthesia • Pharyngostomy tube • Endotracheal tube • Tracheostomy tube • Misplaced feeding tubes

Pathophysiology

Aspiration of material from the oral cavity, pharynx, oesophagus, and stomach and proximal duodenum results in a cascade of reactions that rapidly result in interference with the gas exchange units in the lungs, resulting in systemic hypoxia, tissue damage, and severe illness. The cascade of reactions is outlined below

Phase 1 (0-4 hrs following aspiration)

- Collapse of alveoli - acid gastric contents denatures and dilutes lung surfactant leading to atelectasis, V/Q mismatching, decreased lung compliance, and increased respiratory effort
- Pulmonary oedema - occurs as a result of epithelial cell necrosis caused by gastric acid, and increased capillary permeability resulting from stimulation of sensory nerves in the trachea and bronchi. Fluid, protein and cells move into the interstitium and alveoli, where they subsequently provide oncotic pull for further fluid movement into the alveoli and interstitium

- Broncho-constriction – results from stimulation of sensory nerves in the bronchi and trachea in response to foreign material, and stimulates cough, bronchial oedema, increased bronchial mucous production, and reflex bronchoconstriction, all of which lead to narrowing of the conducting airways.

Phase 2: (4-72 hrs following aspiration)

- Pulmonary oedema – the release of inflammatory mediators secondary to cell injury and death leads to activation of the inflammatory cascade; recruitment of neutrophils and macrophages, and the subsequent release of inflammatory cytokines, that dramatically increase vascular permeability, and contribute to the accumulation of protein and cell-rich fluid extravasation into the pulmonary tissues
- Inflammation – damage to the pulmonary tissues leads to activation of the inflammatory response by tissue macrophages and neutrophils, which results in not only pulmonary oedema as described above, but also lung consolidation, leading to ventilation-perfusion mis-matching, which can lead to generalized tissue hypoxia if inflammation is widespread throughout the lungs
- Systemic hypotension - occurs through extravasation of fluid into the interstitium and alveoli, and the inflammatory response to pulmonary injury, hypoxia, and the development of shock
- Airway obstruction occurs due to fluid, particulate material; bronchospasm due to foreign material, granulomatous inflammatory reaction, hemorrhage and edema
- Infection – results from inhalation of contaminated material, damage to respiratory defense systems (mucociliary clearance, mucosal barrier), and poor oxygen delivery to affected tissues, decreasing effectiveness of mucosal defence systems

Diagnosis

- Patient status – These patients have a multitude of problems, all of which can result in death of the patient if they are not addressed promptly. Dyspnoea, poor tissue perfusion/oxygenation, VQ mis-matching, toxic shock, and infection are all presumed to be present in the patient.
- Physical examination:
  - Provide oxygen supplementation at all times during patient examination and stabilization
  - Clinical signs suggestive of aspiration pneumonia include
    - Acute-onset respiratory distress
    - +/- cyanosis
    - +/- cough
    - +/- mucopurulent nasal discharge
    - +/- pyrexia
    - +/- collapse
  - Auscultation – is frequently abnormal. Findings may include
    - Asymmetry of lung sounds
      - May be quieter on one side of the chest than the other if lung consolidation has occurred, or if bronchial obstruction is present
      - May be louder/noisier if air is passing into a non-consolidated lung
    - Crackles or rales may be auscultated, particularly during inspiration
    - Wheezes may be auscultated if bronchiolar constriction is present
  - Complete patient evaluation – may reveal a predisposing cause e.g. abnormal mentation, intestinal foreign body etc.

- Radiography – is best reserved for stable patients, as radiography of the unstable or severely dyspnoeic patient may lead to patient decompensation and death
  - Alveolar lung patterns predominate, but interstitial lung patterns often co-exist
  - Lesion distribution is dependent on the orientation of the patient when aspiration occurred
  - Differential diagnoses for radiographic lesions often observed are…
    - Bronchopneumonia
    - Pulmonary haemorrhage
    - Pulmonary contusions
    - Pulmonary neoplasia
    - Lung lobe torsion or collapse
  - Megaoesophagus may be observed, as may foreign bodies, etc.
- VetBLUE Ultrasound – frequently reveals either generalized or lobar lung consolidation, and may also reveal pleural effusion.

Acute management

- **Oxygen therapy**
  - Provide oxygen via nasal oxygen catheter @ 50-100 ml/kg/minute
  - Do not exceed 40-60% oxygen concentration for longer than 12-24 hrs to avoid the potential for oxygen toxicity.
- **Airway patency**
  - Examine oral cavity, palpate trachea, and larynx
  - Remove vomitus from mouth, pharynx
  - Observe respiratory pattern - marked inspiratory efforts and stridor are supportive of airway obstruction. In case of upper airway obstruction, sedation or light anaesthesia may be required to secure the airway and permit removal of foreign objects, and to allow airway suctioning. Perform tracheotomy if required.
  - Airway suctioning is only indicated in the early stages following aspiration, as aspirated contents are quickly dispersed from the larger airways following aspiration
- **Breathing**
  - Significant ventilation/perfusion mismatching occurs. Provide oxygen supplementation via endo-tracheal tube, nasal oxygen, or trans-tracheal catheter
  - Due to infiltration of blood, oedematous fluid, and inflammatory exudate into the airways, many alveoli will not receive oxygen, therefore mechanical ventilation with application of positive end-expiratory pressure (PEEP) to relieve atelectasis may be beneficial; humidify oxygen. Specific indications for provision of ventilation support include hypoxaemia, and hypercabia despite conventional non-invasive methods of improving ventilation efficacy (such as oxygen supplementation, broncho-dilatation etc.)
  - Due to V/Q abnormalities, patients should be rotated 20-30 degrees along the longitudinal axis every 2 hours, or maintained in sternal recumbency
- **Circulation**
  - Monitor PCV/TP/electrolytes, capillary refill time and colour
  - Intravenous fluid therapy should be administered judiciously to avoid excessive fluid administration and worsening of pulmonary oedema. There is much controversy over ideal fluid rates in animals with pneumonia. Below are some recommendations…
    - Diagnose and treat shock using small volume resuscitation in the following manner
      - Lactated Ringer's solution @ 5-7 ml/kg IV over 10 minutes, then…
      - Lactated Ringer's solution @ 5-7 ml/kg IV over 10 minutes , OR Hydroxy-ethyl starch @2-3 ml/kg IV over 10 minutes, (in the non-septic patient) then…

- Lactated Ringer's solution @ 5-7 ml/kg IV over 10 minutes
- If clinical signs of shock are not resolved within 30 minutes, fluid rates should be reduced to maintenance rates, to avoid fluid overload, and a cause of poo response to fluid (presence of sepsis etc.) should commence.

The use of hydroxy-ethyl starch in human and animal models of pneumonia is associated with reduced lung water volume. However, use beyond acute volume resuscitation is no longer recommended, particularly if the patient is showing signs of sepsis or septic shock, as there may be an increased risk of adverse outcome.

- Provide fluid therapy for rehydration slowly over 24 hours if the patient is dehydrated
- Provide fluid therapy for maintenance using a low-sodium fluid such as 0.45% NaCl + 2.5% glucose + 20 mEq/L KCl at a rate of 2.5 ml/kg/hr
- Provide fluid for ongoing losses. The patient with aspiration pneumonia suffers ongoing fluid losses into third spaces such as the pulmonary parenchyma and pleural space, and, in patients who subsequently develop sepsis, from third space losses into body interstitial spaces. Administration of isotonic crystalloids at 1 ml/kg/hr is often sufficient, when combined with maintenance fluid therapy rates, to provide for both maintenance and ongoing losses. However, frequent patient assessment is required to ensure the patient does not become dehydrated or over-hydrated, with subsequent fluid rate adjustments made based on the results of physical examination findings.

- **Bronchodilators**
  - Bronchodilator use in the patient with pneumonia is controversial. Providing dilatation of the bronchi reduces conducting airway resistance, and the work of breathing, both of which are beneficial in patients with dyspnoea. Bronchodilators may also aid in mucociliary clearance of respiratory secretions. However, bronchodilators may also suppress the cough reflex, and may worsen ventilation-perfusion mis-matching within the lung. Recommendations are that patients that are experiencing profound dyspnoea may benefit from bronchodilator use in order to reduce the work of breathing, tissue oxygen demand and patient fatigue
    - Terbutaline 0.01 mg/kg SC or IM
- **Antibiotics**
  - Damage to natural airway defense mechanisms of the respiratory tract, and inhalation of contaminated material means infection is a likely sequel to aspiration.
  - Antibiotic therapy choice should be ideally based on results of gram stain, and antimicrobial culture and sensitivity following a trans-tracheal wash
  - Aerobic bacteria will be the predominant source of infection for most patients
  - Empirical antibiotic choices may include…
    - First generation cephalosporin (cephazolin @ 22 mg/kg IV q 8 hrs) OR
    - Enrofloxacin 5-10mg/kg slow IV q 12 hrs, or 10 mg/kg slow IV q 24 hrs.
    - Adjust antibiotic therapy as indicated
- **Search for underlying aetiology**
  - Altered consciousness - sedation, seizures, CNS depression, head trauma, unconsciousness, cleft palate, force feeding, vascular ring abnormality, esophageal stricture, esophageal motility disorders; megaoesophagus; gastro esophageal reflux, gastric motility disorders, vomiting etc.

Pulmonary Contusions

Introduction

Pulmonary contusion is a serious anatomical and physiological lesion of the lung following non-penetrative, compression-decompression injury to the chest wall. The disruption of alveolar-endothelial integrity results in the pathology of haemorrhage and oedema (Roch et al., 2011). A study of humans with pulmonary contusions determined that 80% of human patients with pulmonary contusions also suffered non-thoracic injuries (Fulton 1970) necessitating the clinician conduct a thorough patient evaluation and consideration of pulmonary contusions in patients presenting with injuries following trauma.

Aetiology

Pulmonary contusions result from compressive-decompressive injury to the chest wall. They may be encountered in many different types of trauma, including road traffic trauma impact, dog attack injuries, incidents where the owners stand on a young or small animal, or falling injuries, and other types of injuries.

Pathophysiology

- Pulmonary contusions result in intra, and extra pulmonary haemorrhage. Haemorrhage into the alveoli causes interference with the gas exchange unit, and results in hypoxia, and reflex-mediated increased ventilatory rate and effort mediated via central nervous system chemoreceptors and the respiratory centre in the brain.
- Bronchospasm occurs due to pulmonary trauma. In addition, the presence of fluid in airways reduces airflow within the larger airways and bronchioles. Lung surfactant is diluted, reducing lung compliance, increasing the work of breathing, and leading to atelectasis - further interfering with alveolar ventilation and gas exchange.
- Concurrent traumatic injury to the myocardium, the presence of circulatory shock, and intra-pleural diseases (haemorrhage, effusion, pneumothorax, fractured ribs, diaphragmatic hernia, and flail chest) may also interfere with gas exchange and respiration
- A secondary inflammatory reaction occurs in response to extravasation of blood into the interstitium and alveoli, from concussive trauma (and subsequent damage) to the cells of the lungs, and hypoxia-induced lung damage. It is important to note that extravasation of fluid and cells into pulmonary interstitial and alveolar spaces may result in progressive impairment in gas exchange and related increase in respiratory distress for up to 12-18 hours following trauma.

Treatment

- **Oxygen therapy**
  - Oxygen should be supplemented initially by fly-by or oxygen hood or cage, but should be transitioned to intra-nasal oxygen within the first few hours of admission to hospital (except in cases of head trauma or increased intra-cranial pressure) to avoid the potential for excessive inhaled oxygen concentration and oxygen toxicity.
  - Failure to improve oxygen saturation in a patient receiving oxygen supplementation may be an indication for provision of positive pressure ventilation therapy.
- **Management of Pleural Space Disorders**
  - Drainage of pleural fluid and/or air will reduce lung atelectasis, reduce the work of breathing, and may improve gas exchange in the lung.
  - Stabilisation of fractured ribs, and/or flail chest using external stabilization techniques will reduce the effort required for the patient to breathe. Provision of local anaesthesia in the form

of local nerve blocks to intercostal nerves in patients with rib fractures/displacement can ease discomfort of breathing in affected patients.

- Ventilation assessment and therapy – Patients with pulmonary contusions may require ventilation assistance if supplementation with oxygen therapy, and other supportive measures do not result in a return to normal SpO_2 or PaO_2. Given that respiratory function may deteriorate over the first 24 hours or so following trauma (owing to the development of an inflammatory reaction in response to tissue trauma within the pulmonary parenchyma), close monitoring of patients during this period is essential in order to detect deteriorating respiratory effectiveness and function, so that timely intervention is possible.
- **Intravenous fluid therapy**
    - Damage to the pulmonary capillary walls results in haemorrhage and extravasation of fluid into the interstitial fluid compartment in the lung, and into the alveoli. In addition, pulmonary tissue damage results in leukocyte chemotaxis and cytokine-mediated inflammatory response, which further increases pulmonary capillary permeability.
    - Administration of appropriate intravenous fluid therapy to patients with pulmonary contusions has long been an area of controversy, owing to concerns about excessive fluid administration raising pulmonary capillary pressures and contributing to worsening of pulmonary oedema. At present, scientific studies offer conflicting evidence regarding appropriate fluid administration. As a result, a case-by-case assessment must be made, following some general guiding principles (outlined below).
    - Clinicians must therefore achieve a balance between limiting pulmonary pressures and providing adequate fluid resuscitation to avoid hypoperfusion complications of other organ systems
        - Administration of large volumes of isotonic crystalloids e.g. lactated Ringer's solution should be avoided, as they are associated with excessive lung water accumulation and a deterioration of respiratory function and gas exchange.
        - Several studies have showing the benefit to mild fluid restriction in patients with pulmonary contusions, although the studies are flawed in many respects, through exclusion of key patient subsets, and treatment criteria.
        - A study in 2009 explored the concept of biphasic (early and late) fluid management of patients suffering septic shock complicated by acute lung injury – which has many facets similar to those observed in pulmonary contusions – including the presence of high pulmonary capillary permeability and inflammation. Their study evaluated the relationship between adequate initial fluid resuscitation (AIFR), where patients received an initial fluid bolus corresponding to a positive fluid balance, and conservative late fluid management (CLFM), defined as an even-to-negative fluid balance measurement during the first 7 days after lung injury. The results revealed both AIFR and CLFM to have lower mortality rates if used separately when compared to not being used at all; however, mortality rates were lowest if used in combination suggesting an additive effect of both fluid strategies (Murphy el al., 2009).
        - Given many patients with pulmonary contusions have traumatic injury to other organ systems (such as head trauma, fractures, open wounds etc.) – all of which require positive fluid balance to ensure adequate tissue oxygen delivery for optimal healing to take place, a strategy of fluid resuscitation to restore cardiac output and tissue oxygen delivery in acute resuscitation, followed by a more conservative fluid administration protocol seems appropriate for most patients with pulmonary contusions.
    - Fluid type for administration to patients with pulmonary contusions…
        - Most studies demonstrate little difference in patient survival, requirement for ventilation therapy, or lung function when isotonic crystalloid fluids, colloid fluids or

hypertonic crystalloid fluids are used for patient resuscitation. However, one study in humans, and one in pigs, demonstrated lower lung water volumes in patients with acute lung injury in sepsis that received synthetic colloids (hydroxy-ethyl starch) than those that did not, suggesting there may be benefit in providing colloids in some patients with severe inflammatory lung disease

- Current recommendations – it is difficult to provide recommendations regarding fluid resuscitation in pulmonary contusions for all patients, as all patients are different, and require individual assessment. However, the following may be used as a guide…
  - For acute patient resuscitation
    - Lactated Ringers' solution 7 ml/kg IV over 10 minutes, and repeated until clinical signs of shock have resolved
    - One bolus of lactated Ringer's solution may be substituted for a single bolus of hydroxy-ethyl starch (HES; Voluven) @ 2-3 ml/kg given over 10 minutes if desired to prolong the effectiveness of lactated Ringer's solution – however, this is not necessary, and may result in increased risk of bleeding in some patients
  - Following acute volume resuscitation
    - Lactated Ringer's solution or other buffered poly-ionic isotonic solution should be administered at rates not exceeding 2-3.5 ml/kg/hr to provide for maintenance fluid requirements and ongoing losses

- **Monitor the patient** – regular assessment of cardiovascular status (heart rate, pulse quality, mucus membrane color) and respiratory function (respiratory rate, effort, pulse oximetry) is essential to aid in determining the progress of the patient, and the requirement for further intervention e.g. mechanical ventilation. Blood gas analysis and venous blood lactate can also be used as a monitoring tool to evaluate global tissue perfusion adequacy, and the effectiveness of pulmonary gas exchange.
- **Analgesia** – pulmonary injury is painful, especially if it occurs with concurrent pleural or thoracic cage injury. Analgesia should be provided with an opioid analgesic, +/- ketamine. Anti-anxiety medications such as midazolam may also be considered to reduce stress associated with respiratory difficulty.
  - **Opioid medications**: fentanyl constant rate infusion is considered the agent of choice in most patients with acute pulmonary contusions and/or thoracic wall injury, owing to its short-acting pharmacodynamics, and minimal cardiovascular and respiratory depressant effects. Morphine and methadone, particularly if administered at dose rates exceeding 0.1 mg/kg, can produce profound depression in the compromised patient, along with respiratory depression, both of which are undesirable in the acute trauma patient with breathing difficulty. Pain caused by mild injury may be successfully managed with butorphanol, which, like fentanyl, is associated with minimal respiratory depression.
  - **Adjunct medications**: Ketamine constant rate infusions may be employed alongside fentanyl or butorphanol constant rate infusions if patients remain painful despite opioid medication. Likewise, stressed patients may benefit from mild sedation with ketamine or midazolam constant rate infusions. Lidocaine intercostal local nerve blocks administered to patients with fractured ribs can provide useful analgesia in combination with the constant rate infusions outlined above.

Pulmonary Thromboembolism

Definition

The formation of a clot (coagulation factors, platelets, fibrin, cellular material) or and embolus (fat, air, tumor fragment, hair etc.) at one site, followed by migration to another site in the pulmonary vasculature, with associated vascular occlusion - usually occurs in the smaller pulmonary arteries.

Predisposing factors

Virchow's Triad

- Hyper-coagulable state (most important factor)
- Vascular stasis
- Disruption of the vascular endothelium

Diseases associated with pulmonary thromboembolism (PTE)

- Polycythemia, dysproteinaemia, obesity, pregnancy, immobilization (human data)
- Hypothyroidism
- Pancreatitis
- Nephrotic syndrome, renal amyloidosis
- DIC
- Antithrombin III deficiency
- Hyperadrenocorticism
- Immune-mediated hemolytic anemia (associated with IV catheters, blood transfusions, hyperbilirubinemia)
- Hyperthyroidism

Clinical signs

- Acute onset of dyspnoea
- Persistent tachypnoea is an early and common sign
- Acute, nonspecific cardiopulmonary signs
- Evidence of pulmonary hemorrhage
- Harsh or loud bronchovesicular lung sounds may be present; crackles and wheezes are rare
- Other symptoms of underlying disease

Diagnosis

- PaO_2 is frequently low, but may be normal
- Radiography
  - Frequently normal
  - Occasional evidence of pleural effusion is present.
  - Blunting of pulmonary vessels
- Coagulation profile may reveal coagulopathy, elevated fibrin degradation products or D-dimers
- CT angiography, ventilation-perfusion scanning
- Echocardiography may detect right heart disease or pulmonary hypertension
- ECG may indicate P-pulmonale, however, this is not sensitive nor specific
- Pulmonary angiography - intra-luminal filling defect, sharp cut-off (i.e. - occluded pulmonary artery) are diagnostic

Treatment

- Often based on clinical appearance, as diagnosis is difficult
- Oxygen therapy

- Cage rest
- Mechanical ventilation
- Fluid therapy if hypotension is present
- Heparin therapy
    - See section on aortic thromboembolism in cardiac diseases
    - Low molecular weight heparin enoxaparin 1 mg/kg SC q 12 hrs
- Plasma therapy is indicated if ATIII deficiency is confirmed or suspected (hepatic failure, glomerulonephropathy etc.)
- Anti-platelet therapy: using clopidogrel 50 mg/kg PO q 24 hrs.
- Thrombolysis – no improvement in survival has been documented
- Manage underlying disease
- Warfarin - dose 0.1 mg/kg PO q 24 hrs; warfarin is the treatment of choice in patients with ATIII deficiency

Prognosis

- Initial presentation - some patients die, overall prognosis poor
- If patients survive the initial episode, the prognosis is guarded to poor

Pleural Space Disease

Pleural space disease is relatively common in the cat, and can produce significant respiratory compromise and distress. The emergency management of pleural space disease has already been discussed in the approach to the respiratory patient. What follows is a more detailed description of the approach to the patient with pleural fluid

Emergency Patient Stabilization

Respiratory distress from pleural effusion is caused by the inability of the lungs to expand – therefore immediate oxygen supplementation and thoracocentesis is indicated to stabilize these patients. Thoracocentesis is also useful for diagnostic purposes as well, as fluid collected during initial patient stabilization can be submitted for microbial culture and sensitivity, cytology, and biochemical analysis.

Thoracocentesis should be continued until no more fluid is able to be removed from the pleural cavity, in order to provide for maximum lung expansion during respiration to relieve respiratory compromise. Pleural fluid should be collected in EDTA, Lithium heparin and plain tubes for analysis, and a smear made for cytology analysis. Full fluid analysis includes…

1. Cytology, including gram stain evaluation
2. Biochemistry analysis
3. PCV/TP
4. Microbial culture and sensitivity

Following thoracocentesis, the patient will frequently require cardiovascular stabilisation to manage shock, dehydration, haemorrhage and ongoing fluid loss (from drainage of pleural fluid).

Provision of General Supportive Care

Once the patient is stabilized, a complete general physical examination should be performed, and blood and urine samples taken for routine laboratory analysis (including FeLV/FIV analysis), blood coagulation profile, urinalysis and culture etc.

The patients' systemic needs should be met, including continued provision of oxygen supplementation, intravenous fluid therapy, monitoring of urine output, and provision of nutritional and gastrointestinal support. In addition, showing signs of systemic infection or inflammation should receive broad spectrum antibiotics until a definitive therapy can be determined through microbial culture and sensitivity of pleural effusion.

When to Consider Placement of a Chest Drain

Chest drains are placed when there is an ongoing requirement for aspiration of fluid, air, or both from the pleural space. In almost all cases in which an exudate is present in the pleural space, chest drain placement is warranted, whereas the presence of transudate or modified transudate pleural fluid does not always require placement of a chest drain once definitive treatment is begun.

Placement of the Chest Drain

1. Induce a light plane of general anesthesia using a short-acting intravenous anesthetic agent such as diazepam/ketamine or alfaxalone. Avoidance of drugs which induce significant hypotension and/or hypoventilation, such as medetomidine should be avoided in the compromised patient.
2. Clip and aseptically prepare a 5 cm square are of skin on the lateral thoracic wall, starting from rib 4-5 cranially, and ending at rib 11-12 caudally, and starting ventral to the costo-chondral junction ventrally, and ending at the vertebral-costal junction dorsally.
3. Chest drains are inserted at the junction between the middle and upper 1/3 of the thoracic cage. Infiltrate local anesthetic (2% lignocaine) at the intended skin incision site at this level over the 10th intercostal space, and infiltrate cranially towards the 7th or 8th intercostal space.
4. Make a stab skin incision at the 10th intercostal space.
5. Tunnel the subcutaneous tissue forward to the 7th or 8th intercostal space using one of the following techniques
    a. Have an assistant grasp the thoracic skin of the patient and pull it cranially until the skin incision rests over the 7th or 8th intercostal space, OR
    b. Blunt dissect the subcutaneous tissues to form a narrow tunnel between the skin incision and the 7th or 8th intercostal space
    c. If the chest drain has a trocar, the trocar is used to "tunnel" under the skin from the skin incision to the 7th or 8th intercostal space
6. Push the chest tube into the pleural space using one of the following techniques
    a. Chest drain with trocar – following tunneling to the 7th or 8th intercostal space, the chest tube and trocar is elevated perpendicular to the chest wall, and is grasped with one hand just above the skin to ensure the trocar does not penetrate too deeply into the chest cavity on entry into the pleural space, and is gently tapped into the pleural space with the other hand
    b. Blunt dissection – the intercostal muscles may be bluntly dissected into the pleural space for passage of the chest tube. This technique offers greater control on entering the pleural space, and is associated with a reduced incidence of lung laceration

Following entry into the pleural space, the chest tube is advanced in a cranial and ventral direction so that the distal end of the tube should be imagines to be lying along the ventral floor of the pleural space, ending at the 2nd to 3rd rib. All tube fenestrations must be contained within the pleural space. The end of the chest tube must be clamped off to prevent entry of air into the thoracic cavity and iatrogenic pneumothorax.

7. Secure the chest tube – the skin incision is closed around the chest tube with a purse-string suture, and the tube is then secured in place with a Chinese Finger-Trap suture, anchored to a suture placed though the skin and through the periosteum of the 10th rib.
8. Preventing chest tube leakage and pneumothorax
    a. Clamping the tube – all chest tubes should have a minimum of two (2) points of closure/clamping at the open end. Recommended clamping include
        1. One clamp should be placed on the chest tube as close to the patient as possible. This clamp is placed to assist in preventing pneumothorax should the patient dislodge connections to suction or 3-way stopcock apparatus when the tube is not being suctioned
        2. A second clamp, or tube closure device, such as a 3-way stopcock or valve should be placed at the end of the chest tube, and is used to facilitate chest drain opening and closure during periods of chest tube suctioning.
    b. Connecting the chest tube – the chest tube may be connected via a tube adaptor, syringe adaptor and 3-way stopcock to facilitate removal of fluid. It is important to secure all components and attachments (adaptors, 3-way stopcock and chest tube) together using

a18-guage wire in a double figure-8 pattern to ensure the attached connectors do not loosen and result in pneumothorax.

c. Bandaging the chest tube – the skin incision is covered with iodine or chlorhexidine ointment and a soft, sterile, non-adherent pad, and a circumferential self-adhesive wrap is placed around the chest, covering the incision. This self-adhesive wrap should be secured to the patients' skin at the cranial edge with adhesive tape/bandage, to prevent the bandage from migrating caudally along the patient body, and exposing the skin incision
d. Elizabethan collar – chest drains can be irritating to patients – and Elizabethan collar or other such device should be employed to assist preventing the patient from biting or pulling their chest drain out.

Establishing a Diagnostic Pathway

Pleural effusion results from either

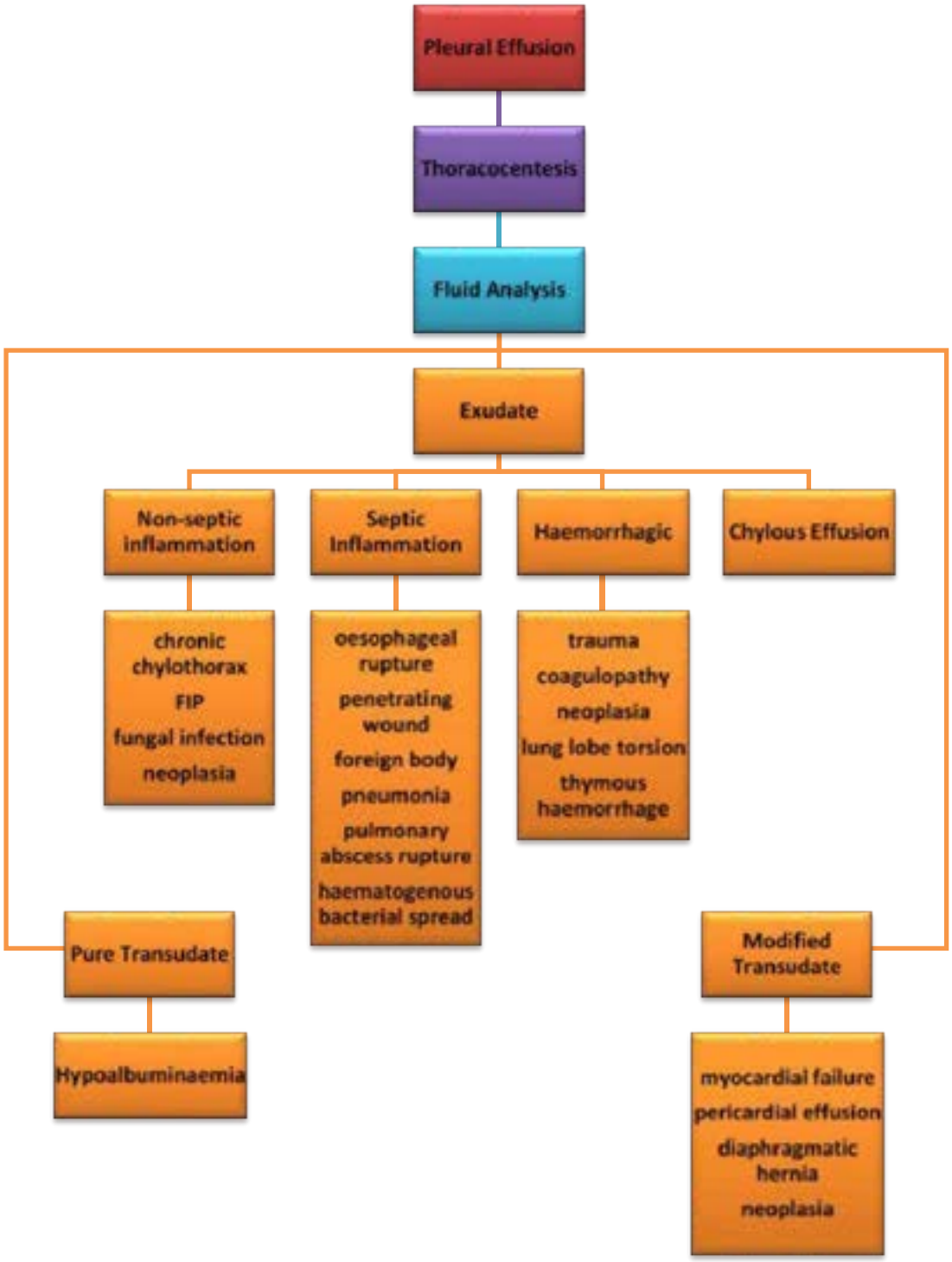
- increased vascular permeability
- increased capillary or lymphatic hydrostatic pressure
- decreased intravascular colloid oncotic pressure
- trauma
- coagulopathy
- blood vessel erosion (neoplasia)
- inflammation (pleuritis)

Usually, analysis of pleural fluid, and the presence of clinical signs, radiographic findings, ultrasonography and laboratory findings representative of underlying disease facilitate a diagnosis in the majority of cases of pleural effusion. Analysis of pleural effusion is one of the most important steps in identifying the underlying cause of fluid accumulation. Characteristics of pleural fluid and likely causes are listed in the table below

Characteristics and Causes of Pleural Effusion

Fluid Type	Characteristics	Possible Causes
Transudate	1. Color - colorless to pale yellow color 2. Turbidity - low turbidity/clear 3. total protein <25 g/L 4. total cells < 1500/µL 5. Cell types a. Mesothelial b. Occasional red blood cells	• Hypoproteinaemia
Modified Transudate	1. Color - yellow or pink 2. Turbidity - clear to slightly cloudy 3. total protein 30 g/L 4. total cells 1500-5000/µL 5. Cell types a. Red blood cells b. Macrophages c. Mesothelial cells	• Myocardial Failure • Pericardial Effusion • Diaphragmatic Hernia • Neoplasia • Long-standing transudate
Exudate	1. Color - yellow to brown 2. Turbidity - cloudy 3. total protein >30 g/L 4. total cells >5000/µL	***Non-septic Inflammation*** • Chronic chylothorax • FIP • Fungal Infection • Neoplasia ***Septic Inflammation*** • Ruptured oesophagus • Penetrating wound • Foreign body • Ruptured tumor • Para-pneumonic spread • Pulmonary abscess • Haematogenous bacterial spread
Haemorrhage	1. Color - red 2. Turbidity - cloudy 3. total protein > 30 g/L 4. total cells - similar to peripheral blood if recent or active	1. Trauma 2. Coagulopathy 3. Neoplasia 4. Lung lobe torsion 5. Thymic bleed
Chylous	1. Milky white 2. turbid 3. total protein >25 g/L 4. total cells 500-20,000/µL 5. Cell types a. Mature lymphocytes b. Neutrophils c. Macrophages	• Mediastinal mass • Lymphatic trauma • Heartworm • Cardiomyopathy • Lung lobe torsion • Idiopathic

Flow-Chart of the Diagnostic Approach to Pleural Effusion



Pleuritis and Pyothorax

Inflammatory conditions of the pleura may be dry, sero-fibrinous, pyogranulomatous, or purulent.

- *Dry pleuritis* often precedes inflammatory pleural effusions. Dry pleuritis may be caused by bacteria, viruses, or trauma. A diagnosis of dry pleuritis is suggested by clinical findings of a rapid and shallow respiratory pattern, obscure thoracic pain, non-productive cough, and auscultation of a pleural friction rub.
- *Sero-fibrinous pleuritis* is reported with canine hepatitis, canine leptospirosis, canine distemper, canine and feline upper respiratory viruses, and parasitic diseases such as *Aelurostrongylus* in cats. Occasionally, bile present in the pleural cavity will cause a sero-fibrinous pleuritis
- *Pyogranulomatous pleuritis* is associated with feline infectious peritonitis. The effusion is secondary to virus-induced vasculitis affecting all serous membranes.
- *Purulent pleuritis*, also referred to as *pyothorax* or *empyema*, is invariably the result of bacterial or fungal sepsis of the pleural space. Sources of bacterial contamination include
    - Penetrating thoracic wounds e.g. bog bites, impalement, or malicious attack with knives or other weapons; gunshot etc.
    - Extension of bacterial pneumonia
    - Migrating foreign bodies
    - Oesophageal perforations
    - Extensions of cervical, lumbar or mediastinal infections
    - Haematogenous spread from distant sites e.g. Discospondylitis, pneumonia etc.

Thoracic bite wounds are frequently implicated in feline pyothorax. *Pasteurella multocida* and anaerobes are the most prevalent isolates in cats.

Clinical Signs

Pleuritis and pyothorax frequently have an insidious course and presentation may be delayed. Tachypnoea, inspiratory dyspnoea, orthopnoea, or more subtly exercise intolerance may be described. Lethargy/depression and anorexia, vomiting and diarrhoea may be described. Occasionally, patients may present in the advanced stages of disease with signs of septic shock. Dogs presenting with a traumatic body wall defect most often have history that relates to trauma. In most cases, moderate to severe respiratory distress usually is present. The patient may also show signs of systemic infection characterized by anorexia, weight loss, malaise, and fever.

Diagnosis

- Diagnostic thoracocentesis and radiography - Physical and radiographic findings are those of pleural effusion. Diagnosis is made typically on the presence of an inflammatory exudate on thoracocentesis. Typically, thoracocentesis is recommended prior to thoracic radiography, as thoracocentesis is of therapeutic and diagnostic value and may improve the diagnostic yield of subsequent radiographs; as a primary or accompanying disease process (mass/abscess, pneumothorax, or pneumo-mediastinum) may be more readily identified after evacuation of the effusion.
- Laboratory evaluation
    - Cytologic evaluation of the collected fluid is generally diagnostic for pyothorax (see below).
    - An inflammatory leukogram, hypoalbuminaemia, blood glucose and electrolyte derangements are the most common findings on a blood profile.
- Other Imaging Techniques
    - Thoracic ultrasound may be indicated and useful in identifying consolidated lung masses, mediastinal masses, and abscessed or neoplastic lung nodules.
    - Advanced imaging is not commonly used although CT or MRI are proving to be more useful in veterinary medicine and are utilized routinely in human medicine.

If one is obtaining fluid for analysis and culture, it makes sense to remove as much of the fluid as possible, as in most cases the effusion contributes significantly to the morbidity of the patient. Bilateral evacuation is most beneficial since bilateral effusion is typical. Hypodermic needles, butterfly catheters, and over-the-needle catheters can be used. Larger (18 or 16 gauge) catheters may be fenestrated to prevent plugging but the fenestrations should be small and staggered to avoid kinking or breakage. Needles/catheters are placed in the ventral third of the thorax caudal to the fifth rib to maximize yield and minimize iatrogenic cardiac trauma. Fluid can be removed with a syringe or low-power suction system attached to extension tubing and a three way stop cock.

- ***Inflammatory exudates*** typically have a total protein greater than 30 g/L, a specific gravity greater than 1.018, and a total cell count greater than 30 X 10^3 cells/ul. Inflammatory exudates may be non-septic or septic.
    - *Non-septic exudates* usually have a sero-fibrinous or sero-sanguineous appearance. A typical example of a non-septic exudate is that produced by feline infectious peritonitis. Feline infectious peritonitis produces a non-septic exudative pleural effusion that appears yellow, translucent, and viscous on gross examination. Total protein values will approach serum levels ranging from 40 to 80 g/L. Electrophoresis will reveal an elevated gamma globulin fraction. The predominant cell types present in non-septic exudates are non-degenerative neutrophils and macrophages. Total cell counts are generally not high, ranging from 5 to 15 X 10^3 cells/ul.
    - *Septic exudates* are characteristic of pyothorax. The fluid is frequently viscous, opaque, and varies in color from white or yellow to green or red. The fluid may clot or exhibit fibrinous debris, and often produces a foul odour. Cell counts range from 30 to 200 X10^3 cells/ul, although accurate cell counts are difficult due to extensive cellular degeneration. Degenerate neutrophils predominate. Bacteria are often visualized in bacterial infection. Gram stains may give an early indication of the types of bacteria present. Fluid should be cultured for aerobic and anaerobic bacteria. Macrophages and plasma cells increase as an exudative process becomes longstanding.

Treatment

Treatment of pyothorax must be prompt and aggressive.

1. The initial goal of therapy is to relieve respiratory embarrassment by thoracocentesis, preferably under minimal restraint with the patient sternal recumbency, or whilst standing.
2. Supportive care with intravenous fluids is necessary to correct dehydration, acid-base and electrolyte imbalance, and to provide fluid for ongoing losses, which predominantly are provided to replace the high-protein fluid obtained via thoracic drainage. As such, a combination of isotonic crystalloids and synthetic colloids such as hydroxy-ethyl starch are required.
3. Systemic antibiotic therapy should be initiated immediately, and then adjusted based on culture and sensitivity results. Due to the high incidence of anaerobic infections, antibiotics with an anaerobic spectrum should be started upon diagnosis of pyothorax and continued throughout the course of disease. Penicillin/penicillin derivatives, cefoxitin, and enrofloxacin are considered effective against the most commonly isolated aerobic bacteria. Subsequent therapy should be based on culture and sensitivity. Anaerobic bacteria and *Actinomyces* can be difficult to culture, and treatment for these organisms should continue even if cultures are negative. *Actinomyces* species are more likely to be found on cytology of pleural fluid, and are effectively treated with extended-term (at least 6 weeks) beta-lactam antibiotics.
4. Many animals with pyothorax will have bacteraemia or septicaemia so intravenous antibiotics are indicated in the initial treatment period. Antibiotic therapy should be continued for at least four to six weeks after diagnosis. Broad-spectrum systemic antimicrobial therapy is the first line of treatment for pyothorax. Empiric therapy is initiated pending culture, and selection of medications is based on the most common causative organisms.

5. Chest Drain Placement - Once the patient is stable, a thoracotomy tube should be placed utilizing local anaesthesia and sedation, or light general anaesthesia. Frequently, bilateral thoracotomy tubes are required, as, although the mediastinum in dogs and cats is considered an incomplete division between left and right hemi-thorax, fibrin and debris from the pyothorax often leads to compartmentalisation of the pleural space, and inadequate drainage when only one chest drain is used.
    a. Thoracic drainage should be attempted every 2-4 hours following tube placement. After complete evacuation of the pleural space, pleural lavage is initiated. The pleural space should be lavaged 2-4 times daily with approximately 10-20 ml/kg of warmed 0.9% saline or Ringer's solution. The lavage fluid should be instilled slowly and discontinued if respiratory distress occurs. The lavage fluid should remain in the pleural space for 10-20 minutes (as long as the patient is not exhibiting respiratory distress). Approximately 75% of the instilled fluid should be removed. Lavage is performed in the first 48 hours of medical treatment. Lavage is not recommended in feline patients. Aspiration should occur with the patient positioned in right lateral, left lateral, sternal, and dorsal recumbency. This allow for more adequate drainage. The fluid volume and character should be recorded and monitored
    b. Complications associated with thoracostomy tubes include iatrogenic pneumothorax, ascending infection, kinking or clogging of the tube, and traumatic removal of the tube. Complications associated with lavage include increased risk of ascending infections and inability to retrieve the instilled fluid. Approximately 25% of the initial lavage volume will be absorbed by the patient, and the remainder should be aspirated.
    c. Efficacy of treatment is monitored by clinical findings, thoracic radiographs, and cytology of the pleural effusion. Most animals with successful treatment will have a decrease in fever and improvement in general attitude within the first 48 hours. Cytology of pleural fluid can be used to assess the response to therapy. Neutrophils, both total number and percentage of degenerate cells, and bacteria should gradually subside over three to five days. The combination of repeated thoracic drainage, pleural cavity lavage, and antibiotic therapy has been reported to resolve 50 to 60% of cases of pyothorax in small animals.
6. Thoracic Surgery – One study reported a 5.4 times greater success with surgical intervention compared to non-surgically treated patients in the successful management of pyothorax. The increased success with surgical intervention may be due to the debridement of fibrotic and infected tissues, removal of foreign material, more adequate lavage with removal of organisms, and allowing better antimicrobial penetration into the tissues. Thoracic surgery may be indicated in the following situations…
    a. Lack of significant clinical improvement within 48 to 72 hours
    b. Radiographic demonstration of un-drained or encapsulated fluid
    c. Radiographic evidence of lung lobe consolidation and pneumothorax – which may suggest the possibility of a ruptured pulmonary abscess.

Exploratory thoracotomy should be undertaken by medium sternotomy in order to access to both left and right thoracic compartments. Adhesions and areas of fluid pockets should be gently broken down during surgery. Removal of the mediastinal membrane is often necessary as the ventral mediastinum frequently becomes thickened and filled with small abscesses. The pericardium also may require excision if it is thickened and abscessed. Consolidated lung lobes which cannot be inflated should be excised by partial or complete lobectomy. Large lung lacerations created by adhesion breakdown must be repaired or the damaged tissue excised. Before closure, the thoracic cavity is vigorously lavaged with copious amounts of warm isotonic crystalloid solution. Closed pleural lavage should be continued postoperatively for at least two to three days. The probability of success with surgical management of refractory pyothorax is better for dogs than for cats.

Chylothorax

Chylothorax results when chyle from the cisterna chyli-thoracic duct system gains access to the pleural space.

- **Anatomy -** In the cat, the caudal thoracic duct typically courses dorsal and to left of the aorta.
- **Aetiology -** The aetiology of chylothorax is poorly understood in the dog and cat.
    - **Trauma -** Trauma is an often cited cause of chylothorax in dogs and cats, but may in fact be quite uncommon. Thoracic duct rupture might result from
        - Blunt or penetrating injuries
        - Traumatic diaphragmatic herniation
        - Thoracic surgery
        - Severe coughing or vomiting episodes.
    - **Obstruction** - Experimental obstruction of the thoracic duct alone rarely results in chylothorax. However, ligation of the cranial vena cava produces lymphangiectasia (distension) of the thoracic duct and a high incidence (> 50%) of chylothorax in both dogs and cats. It is speculated that lymphangiectasia may allow extravasation of chyle through the lymphatic vessel wall.
        - Malignancies or thrombosis that occludes the cranial vena cava might induce chylothorax by obstructive mechanisms.
        - Cardiac disease - Chylothorax occurs with cardiomyopathy, tricuspid dysplasia, and heartworm disease.
    - **Idiopathic** - Lymphangiectasia of unknown origin has been demonstrated in dogs and cats with spontaneous chylothorax.
- **Diagnosis** – The diagnosis of chylothorax is based on recognition of clinical and radiographic findings of pleural effusion and by demonstration of chyle on fluid analysis.
    - *Chylous effusions* are typically opaque and milky white to yellow in colour. Chyle retains its milky appearance upon standing and may form a creamy top layer. Physical and chemical properties of chylous effusions are similar to obstructive transudates since lymph composes a large portion of thoracic duct chyle. Total protein ranges from 30 to 50 g/L. Cytological examination reveals a predominance of small and large lymphocytes. Chronic chylous effusions will show increased neutrophils, macrophages, and mesothelial cells. Total cell counts generally do not exceed 20 X 10^3 cells/ul. Chylomicrons within an effusion confirm its chylous nature. Chylomicrons can be visualized on direct smears or with supra-vital stains such as Sudan III or IV. The presence of chylomicrons is most reliably confirmed by determination of triglyceride levels in the serum and fluid. Chylous fluids typically show triglyceride levels that are 12 to 100 times greater than levels measured in the serum, although anorexic animals may have low chylomicron concentrations, and may require feeding of a fatty meal in order to demonstrate a definitive diagnosis of chylous effusion. Cholesterol levels in chylous effusions are not elevated when values are compared to values from serum.
    - *Pseudo-chylous effusions* - Pleural effusions high in cholesterol or lecithin-globulin complexes appear grossly similar to chylous effusions, but are due to degenerating cells associated with chronic inflammatory or malignant processes. These effusions are termed *pseudo-chylous effusions*. Pseudo-chylous effusions will be low in triglycerides and may have a high cholesterol level.

- **Determining Aetiology** - Once chylothorax is diagnosed, an attempt to determine its cause should be undertaken.
    - Owners should be questioned regarding the possibility of recent trauma.
    - Thoracic radiographs taken after complete pleural drainage should be evaluated for the presence of masses particularly in the cranial mediastinum.
    - Echocardiography and ultrasound examination of the mediastinum also can be undertaken.
    - Animals with chylothorax should be evaluated for dirofilariasis using microfilaria concentration techniques or serum adult antigen detection tests, or both.
    - Cytologic examination of the chylous effusion for the presence of neoplastic cells helps rule out neoplasia.
    - If a cause for chylothorax is not found, the patient is presumed to have a diagnosis of idiopathic chylothorax.
- **Treatment** - Treatment of chylothorax may be medical or surgical.
    - Medical management is directed at draining the pleural space and reducing the formation of chyle.
        - Pleural drainage is indicated to relieve respiratory distress and may either be intermittent or continuous.
        - Low-fat diets have been used to decrease the triglyceride content of chyle, but there is no evidence that the volume of chyle is similarly decreased.
        - Because repeated drainage of chyle can lead to significant loss of lipid and protein, as well as fluid, protein-calorie malnutrition is a concern with long-term intermittent fluid drainage. Animals with chylothorax should therefore receive aggressive nutritional support and be supplemented with fat soluble vitamins.
    - Surgical management of chylothorax involves ligation of the caudal thoracic duct. The following indications for surgery are suggested…
        - failure to significantly diminish the flow of chyle after 5 to 10 days of medical management
        - Losses of chyle exceeding 20 ml/kg/day over a five day period
        - Protein-calorie malnutrition and hypoproteinaemia.

Transthoracic ligation of the thoracic duct is accomplished through a right ninth or tenth intercostal thoracotomy in the dog. Failure to ligate all collateral branches of the caudal thoracic duct is thought to be a most common cause of operative failure. For this reason, *en bloc* ligation of all structures in the caudal mediastinum dorsal to the aorta is recommended. The success rate for surgical management of chylothorax alone is generally less than 60%. Combined mechanical pleural pleurodesis and thoracic duct ligation, both performed via median sternotomy, is currently undergoing clinical evaluation and shows early promise for improving the success of surgery.

Despite vigorous attempts at medical and surgical management, a significant number of animals with chylothorax will fail to respond to therapy.

Haemothorax

The most common cause of haemothorax is trauma - either accidental or surgical. . Haemorrhage can originate from internal structures such as the heart, great vessels, or lungs, or from laceration of intercostal or internal thoracic arteries. Coagulopathies including thrombocytopenia or anticoagulant rodenticide toxicity can also manifest with haemothorax as a primary finding. In addition, neoplasia or parasites, e.g. *Dirofilaria immitis*, can cause rupture of thoracic vessels and spontaneous haemothorax.

- **Diagnosis** – The diagnosis of haemothorax is based on demonstration of blood in the pleural space.
    - Specific gravity, total protein, total cell counts, and cytologic findings of *haemorrhagic effusions* are similar to values in the peripheral blood. The myeloid-erythroid ratio is approximately 1 to 100, similar to peripheral blood. Haemorrhagic effusions generally will have a haemoglobin level of at least 25% of the blood level, whereas sero-sanguineous effusions rarely have haemoglobin values exceeding 1 gm/dl.
    - Haemorrhage within the pleural space generally does not clot due to mechanical defibrination and activation of fibrinolytic mechanisms. Clotting also is impaired by the disappearance of platelets within approximately eight hours following acute haemorrhage.
    - Coagulation assessment - Diagnostic evaluation of coagulation parameters is indicated in animals with haemothorax, especially in the absence of obvious trauma. A platelet count and activated clotting time should be performed. If a platelet count and activated clotting time are normal but coagulopathy is still suspected, a mucosal bleeding time should be performed to assess platelet function particularly in breeds that commonly have von Willebrand's disease. A citrated blood sample should be collected before blood transfusion, centrifuged immediately, and frozen for future assessment of specific factor deficiencies if needed.
- **Treatment –** The specific treatment of haemothorax depends on the volume and flow of haemorrhage in the pleural space.
    - Mild haemothorax that does not induce significant respiratory distress may be managed conservatively to allow resorption of pleural blood.
    - More significant haemothorax sufficient to cause respiratory symptoms requires thoracocentesis to relieve respiratory compromise.
    - Patients with blood loss sufficient to cause symptoms related to blood loss and anaemia, such as elevated heart rate, increased respiratory rate and symptoms of cardiovascular shock and sympathetic nervous system activation may require blood transfusion to maintain an adequate packed cell volume. Non-autologous red cell or whole blood transfusions can be administered if available. Auto-transfusion of blood removed by pleural drainage provides a readily available source of compatible blood in patients with severe haemothorax in which a ready source of blood is unavailable. Blood can be collected directly into a standard blood collection bag and returned to the patient by standard gravitational infusion or collected into 60 ml syringes anti-coagulated with citrate phosphate dextrose (CPD) or anticoagulant citrate dextrose (ACD) at a dose rate of 8 to 10 ml/100 ml blood, and administered via micro-pore filter to remove micro-thrombi. Potential problems associated with auto-transfusion include micro-embolization, haemolysis, cardiac arrhythmias and coagulopathy.
    - Rarely pleural haemorrhage will be such that pleural drainage and transfusion are insufficient to keep ahead of accumulation. In this case, exploratory thoracotomy is indicated in an attempt to effect repair the site of haemorrhage. Exploratory thoracotomy should be undertaken by a median sternotomy approach. If exploratory surgery for severe haemothorax is undertaken, the surgical team should be prepared for intraoperative collection of blood for auto-transfusion.

Neoplastic Effusion

Neoplastic disease can result in pleural effusion. Common tumours that result in thoracic effusion include…

- Lymphosarcoma
- Pulmonary or bronchogenic carcinoma
- Metastatic carcinomas
- Haemangiosarcoma
- Mesothelioma (rare)

Neoplastic effusions are suspected when physical, radiographic, and laboratory findings suggest the presence of a thoracic neoplasm. *Neoplastic effusions* may resemble and exudate or transudate fluid pattern and are therefore identified by the presence of neoplastic cells on cytological examination.

- Thymic lymphosarcoma is the most common cause of neoplastic effusions in cats. Neoplastic lymphocytes are usually pro-lymphocytes or lymphoblasts. These cells are large, variable in size, and have intensely basophilic cytoplasm and multiple nucleoli.
- Metastatic carcinomas and occasionally sarcomas will produce neoplastic effusions.
- It is important to note that differentiation of neoplastic cells from reactive mesothelial cells is difficult even for experienced cytologists. Therefore, care must be used in diagnosing neoplasia on cytological examination alone. Cytological findings should be correlated with other clinical findings. A punch biopsy of the pleura is indicated when pleural neoplasia such a mesothelioma is suspected. Thoracoscopy can be used to explore the thoracic cavity and perform biopsy of the pleura.

Treatment of a neoplastic pleural effusion is directed at the causative neoplasm. The prognosis for animals with neoplastic effusions is poor with the exception of effusions associated with lymphosarcoma. Intermittent pleural drainage gives temporary relief from respiratory distress.

Pneumothorax

Aetiology

Pneumothorax is one of the most common emergency respiratory presentations following traumatic injury to cats. It results primarily from traumatic chest wall compression, with rupture of alveoli secondary to increase in intra-thoracic pressure against a closed glottis, direct penetration of thoracic wall (sharp objects, rib fractures), or rupture of major airway. Note that rupture of the trachea or mainstem bronchi will typically cause pneumo-mediastinum +/- pneumothorax.

Pathophysiology

The pleural space is normally at sub-atmospheric pressure, with small amount of fluid forming a cohesive bond between the lungs and parietal pleura. If air enters the pleural space, the cohesion is lost and the lungs collapse. The initial response of the patient is tachypnoea, leading to decrease in pCO_2, and increasing pH, tachypnoea due to Herring Breuer reflex (triggered by pulmonary deflation) or hypoxia (triggered by aortic and carotid chemoreceptors which relay to the respiratory centre. Hyperventilation decreases physiologic dead space, and increases efficiency of gas exchange BUT does increase energy needs, and compounds cellular hypoxia. Note- dogs can increase the degree of chest wall expansion by 2.5-3.5 times normal during compromised pulmonary function – but this does come at a cost of increased energy expenditure. It is estimated that the energy of breathing can increase by up to 600% in times of severe dyspnoea, and this energy expenditure, in the face of decreasing tissue oxygen delivery usually cannot be sustained. As the pneumothorax becomes worse, compensatory mechanisms fail, and the patient will develop hypercapnoea, severe metabolic and respiratory acidosis and death.

Definitions

There are some broad definitions of different types of pneumothorax

- Open pneumothorax – an open chest wound is present
- Closed pneumothorax - tears in visceral pleura, with an intact thoracic cage
- Valvular pneumothorax - air enters chest during inspiration e.g. with a tension pneumothorax due to traumatic causes, the rupture of an emphysematous bulla, lung granulomas, lung cyst
- Tension pneumothorax - increase intra-pleural pressure, therefore chest expansion is not possible; therefore, the patient succumbs more rapidly. Intra-thoracic pressure eliminates the thoracic pump of venous return - therefore venous return decreases. Tension pneumothorax collapses the vena cava. The net result is decreased cardiac output, and systemic arterial blood pressure

The collapsed lung lobes in pneumothorax result in ventilation/perfusion deficits, and right to left shunting of blood without gas exchange, as blood circulates through damaged and collapsed pulmonary parenchyma, resulting in decreased pO_2, which contributes to shock

Clinical signs

Clinical signs of pneumothorax are typical of other pleural space diseases, and include

- Tachypnoea
- Anxiety
- Restlessness
- Cyanosis
- Pale mucous membranes
- Mouth breathing
- Barrel shaped thorax
- Increased inspiratory +/- expiratory effort

Approach to the patient with pneumothorax

Following initial patient evaluation (history, physical examination), thoracocentesis is recommended – prior to radiography – to provide both diagnostic confirmation, and therapeutic relief from lung collapse. Note that in many trauma patients, a respiratory rate of 45-60 breaths per minute, together with findings on auscultation supportive of diminished lung sounds, indicates thoracocentesis is required.

- Clip and prep skin between ribs 5-9. Insert a 22g needle attached to 3 way stopcock and 10ml syringe at junction of the dorsal and middle 1/3 rib at rib 7, and withdraw as much air as possible. Tap BOTH SIDES due to incomplete mediastinum in dogs and cats

Thoracocentesis should be repeated every 30-60 minutes following patient presentation, with the volume of air removed recorded. The frequency of thoracocentesis can be reduced to 60-120 minutes if the volume of air being aspirated from the chest cavity is falling within 1-2 hrs of patient presentation. If the volume of air is increasing, or not diminishing within the first 6-8 hours of presentation, a chest drain should be placed, and attached to a continuous suction apparatus to prevent excessive pneumothorax development, patient decompensation and death. Other indications for chest drain placement with continuous suction include…

- Patients status is not significantly improving
- Vacuum cannot be established using simple aspiration

Patient progress and pneumothorax resolution can be checked by intermittently (every 2-4 hours) turning off continuous chest suction drainage, and monitoring the patient for 30-60 minutes (unless the patient deteriorates, in which case continuous suction is resumed), and performing a thoracocentesis or suction from the chest drain via syringe to determine the volume of air accumulating in the thoracic cavity within a 30-60 minute time period. Most small pulmonary tears show some sign of resolving – evidenced by decreasing volumes of air suctioned off the chest cavity every 2-4 hrs – over the first 24-48 hours of hospitalization.

If no negative pressure can be established despite thoracocentesis or continuous suction, an exploratory thoracotomy is recommended to isolate damaged airways and effect surgical repair
Note that patient with pneumothorax ma also present with severe pulmonary contusions and other injuries. Failure to establish normal respiratory pattern may be an indication for anaesthesia, immediate intubation, intermittent positive pressure ventilation (IPPV), followed by thoracocentesis or immediate percutaneous thoracotomy to relieve intra-thoracic pressure, to allow for an improvement in venous return, diastolic function and cardiac output. Follow-up with thoracotomy and/or chest drain placement.

References:

Dickson D, Little CJ, Harris J, Rishniw M. Rapid assessment with physical examination in dyspnoeic cats: the RAPID CAT study. journal of small animal practice. 2018 Feb;59(2):75-84.

Janson CO, Hezzell MJ, Oyama MA, Harries B, Drobatz KJ, Reineke EL. Focused cardiac ultrasound and point-of-care NT-proBNP assay in the emergency room for differentiation of cardiac and noncardiac causes of respiratory distress in cats. Journal of Veterinary Emergency and Critical Care. 2020 Jul;30(4):376-83.

O'Byrne L, Cole L. Cats are not small dogs: assessment and stabilisation of emergency presentation. Companion Animal. 2024 Nov 2;29(11):2-6.

Johnson LR. Canine and feline respiratory medicine. John Wiley & Sons; 2024 Nov 4.

Levy N, Ballegeer E, Koenigshof A. Clinical and radiographic findings in cats with aspiration pneumonia: retrospective evaluation of 28 cases. Journal of Small Animal Practice. 2019 Jun;60(6):356-60.

Ward JL, Lisciandro GR, Ware WA, Viall AK, Aona BD, Kurtz KA, Reina-Doreste Y, DeFrancesco TC. Evaluation of point-of-care thoracic ultrasound and NT-proBNP for the diagnosis of congestive heart failure in cats with respiratory distress. Journal of veterinary internal medicine. 2018 Sep;32(5):1530-40.

Tong CW, Gonzalez AL. Respiratory Emergencies. Veterinary Clinics: Small Animal Practice. 2020 Nov 1;50(6):1237-59.

Chalifoux NV, Drobatz KJ, Reineke EL. Predictors of inflammatory lower airway disease in cats presented to the emergency room in respiratory distress: a case-control study. Journal of Feline Medicine and Surgery. 2021 Dec;23(12):1098-108.

Drobatz KJ. Approach to the critically ill cat. Feline emergency and critical care medicine. 2022 Oct 7:1-8.

Clarke DL. Upper airway disease. Feline Emergency and Critical Care Medicine. 2022 Oct 7:109-18.

Feline Sepsis and Systemic Inflammation

Dr. Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (Vet. Emergency and Critical Care, Medicine of Dogs)

The diagnosis of sepsis in cats can be challenging, owing significantly to the absence of many of the clinical signs in cats, that are used to diagnose sepsis in both humans in dogs[1].

In order to diagnose sepsis in cats, the clinician must maintain a high index of suspicion – particularly in critically ill cats – as well as recognise the unique features of the feline response to sepsis[1].

Definitions of SIRS and Sepsis

Traditional criteria for the recognition of patients with systemic inflammation, sepsis and septic shock are based on evidence in humans, stemming from the American college of chest physicians/society of critical care medicine consensus conference: definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis, published in 1992[2].

In these definitions, systemic inflammatory response syndrome, or SIRS was defined as having two of the following clinical criteria: tachycardia, tachypnoea, fever or hypothermia, and leukocytosis or leukopaenia[2].

Sepsis was defined as evidence of infection, plus meeting SIRS criteria. Severe sepsis was defined as the presence of sepsis with concurrent organ dysfunction, hypotension and/or hypoperfusion; with septic shock being defined as severe sepsis with hypotension refractory to fluid therapy and vasopressor therapy[2].

These criteria were adapted for dogs and cats based on their normal physiological parameters according to the table below:

Criteria	Cats	Dogs	Humans
Temperature	>39.7 deg C <37.8 deg C	>39.2dec C <38.1 deg C	>38.0 deg C <36.0 deg C
Heart Rate	>225/min <140/min	>120	>90
Respiratory Rate	>40	>20	>20
White Blood Cell Count	>19,500 <5,000	>16,000 <6,000	>12,000 <4,000

The Sensitivity and Specificity Problem

The difficulty with these traditional SIRS criteria is the low specificity and sensitivity for detection of sepsis in animals. It is widely thought that most cats will fit at least 2 criteria for SIRS without any evidence of clinical illness when presented for evaluation at a veterinary clinic. In a study evaluating the clinical use of SIRS and sepsis criteria in dogs, sensitivity was found to be between 77-97%, but specificity varied between 64 and 77%[3].

In 2001, SIRS criteria were further refined to include more physical parameters and biomarkers, designated PIRO to assist improvement in specificity and sensitivity[4].

PIRO incorporates understanding of Predisposition, Infection, Response of the host, and Organ dysfunction assessment[4].

When applied to cats, PIRO factors have potential to increase specificity for the diagnosis of SIRS and sepsis[1].

Application of PIRO to the Diagnosis of Sepsis in Cats

There are no large-scale studies on sepsis in cats, with most literature involving relatively small numbers of cats with clinical sepsis[5-7], and one study of experimentally induced sepsis in cats[8]. Based on available literature in cats with severe systemic illness and sepsis, the following information is available on the application of PIRO in cats, and how it may inform the diagnosis of sepsis in this species[5-10].

1. Predisposition
    a. There is no known breed or sex predilection for sepsis in cats
    b. Pyothorax is more common in multi-cat households
    c. Septic peritonitis is more common in male cats
    d. Immune suppression, feline leukaemia virus, feline immunodeficiency virus etc. likely increases the risk of sepsis in cats
2. Infection
    a. The most common reported causes of severe sepsis in cats are
        i. Pyothorax (24%)
        ii. Septic peritonitis (14%)
        iii. Endocarditis (14%)
        iv. Pyelonephritis (7%)
        v. Osteomyelitis (3%)
        vi. Pyometra (3%)
        vii. Bite wounds (3%)
    b. The most common bacteria associated with sepsis in cats are
        i. Enteric origin
            1. Escherichia coli
            2. Other enteric bacteria
        ii. Pyothorax
            1. Pasteurella
            2. Clostridium
        iii. Endocarditis
            1. Bartonella species
            2. Gram positive bacteria
    c. Potential sources of infection in sepsis in cats include
        i. Pneumonia
        ii. Pyothorax
        iii. Septic peritonitis
        iv. Pancreatitis
        v. Pyelonephritis
        vi. Bacteraemia secondary to severe gastrointestinal disease or gastrointestinal blunt trauma
        vii. Pyometra
        viii. Hepatic abscess
        ix. Endocarditis
        x. Meningitis

3. Response
    a. The feline response to systemic inflammation and sepsis is less predictable than in dogs or humans.
    b. The feline clinical response to SIRS and sepsis has the following characteristics
        i. Bradycardia
        ii. Variable temperature: Afebrile, pyrexia, or hypothermia
        iii. Abdominal discomfort (with or without abdominal involvement)
        iv. Tachypnoea
    c. The feline biomarker response is largely unknown, as very few studies have been carried out in cats. However, in an experimental study of 39 cats with SIRS or sepsis, the following biomarker response was documented8:
        i. TNF, IL-6, IL 10 and chemokine CXCL-8 increase
        ii. WBC counts decrease, then increase
        iii. Hyperglycaemia, followed by hypoglycaemia
        iv. Leukocytosis or leukopaenia may be seen
        v. Lactate, glucose PT, bilirubin increases
        vi. Hypoalbuminaemia is common, and is likely multifactorial in origin, with reduced hepatic production, and increased protein loss occurring
        vii. Anaemia may develop due to frequent blood sampling, gastrointestinal blood loss, antibody or mechanical-mediated haemolysis, and renal or hepatic disease. Additionally, TNF and IL-1 inhibit erythroid precursor cells, reduce erythropoietin production and response
        viii. Ionised Ca decreases are common
    d. Clinically, cats with SIRS and sepsis show the following in addition to the aforementioned[7]
        i. Hyperbilirubinaemia
        ii. Coagulopathy
        iii. Hepatic dysfunction
        iv. Encephalopathy
4. Organ Dysfunction[8,11]
    a. Organ dysfunction in the cat usually relates to underlying organ disease resulting from either the aetiology or effect of systemic inflammation and sepsis
    b. Common organ dysfunction in cats with SIRS or sepsis includes:
        i. Pulmonary dysfunction
        ii. Pleural effusion
        iii. Acute kidney injury
        iv. Gastrointestinal failure
        v. Liver dysfunction

The Diagnosis of Sepsis in Cats[1,7-10,11]

The diagnosis of sepsis in cats is based on the principles of sound clinical examination, selection of appropriate diagnostic tests, and repeated patient evaluation. Given the physiological response to severe disease and sepsis in cats is more subtle than in dogs – with lethargy and anorexia being the most common clinical signs – the clinician must remain vigilant in seeking patient re-evaluation.

Diagnostic tests applied to the diagnosis of sepsis in cats may include some or all of the following:

1. History
2. Physical examination
    a. Initial patient evaluation to determine
        i. Identification of sepsis criteria
        ii. Identification of emergent issues with vital organs
    b. Full body systems evaluation to determine
        i. Aetiology and/or infection source
        ii. Abnormalities in vital organ function
        iii. Detection of gross abnormalities
3. Laboratory analysis
    a. CBC
    b. Biochemistry
    c. Electrolytes
    d. Blood Gas + Blood Lactate
    e. Urine analysis
    f. Blood culture, wound culture as indicated
    g. CSF analysis as indicated
    h. Peritoneal lavage as indicated
4. Diagnostic Imaging
    a. Thoracic and abdominal radiography
    b. Thoracic and abdominal ultrasonography
5. Blood pressure

Note that repeated patient evaluation is often necessary to detect underlying or causative disease. Abdominal pain may be present in the absence of abdominal disease – and conversely, absence of abdominal pain does not preclude the presence of serious abdominal disease, necessitating thorough and complete patient evaluation using both clinical examination, laboratory and diagnostic imaging in evaluating patients meeting criteria for serious illness.

Treatment of Sepsis in Cats

SIRS and sepsis are potentially devastating disease syndromes, associated with the possibility of high morbidity and mortality in both human and small animal veterinary medicine. There is growing evidence-based medicine regarding best practice for management of people with sepsis with somewhat less evidence-based data in dogs and cats[12]. The outline presented here is based on recommendations from available published literature in human and veterinary fields.
Key tenets of managing the patient with sepsis are included in the table below…

Action	Rationale
High Index of Suspicion	The earlier sepsis is detected, the earlier treatment can begin. There is a high degree of correlation between a delay in the onset of treatment of sepsis, and increasing mortality rate
Obtain Samples for Culture	Samples should be obtained from any injured, damaged or infected tissue for culture and antibiotic sensitivity at the earliest possible time.
Use Appropriate Antibiotics - and treat Early	Antibiotic therapy with appropriate antibiotics should commence early in the treatment of sepsis. In humans, a 1-hour delay in commencement of antibiotic therapy in septic patients leads to increased mortality.
Identify and Manage Shock Aggressively	Shock should be identified and corrected early, with an attempt to achieve normal cardiovascular parameters, normalizing blood lactate concentrations, normal urine output and normal systolic blood pressure
Instrument Early	Intravenous catheters, arterial catheters, urinary catheters, oxygen and feeding tubes should be placed early, to facilitate ready vascular access, early monitoring of urine output and blood pressure, prior to the development of peripheral oedema or respiratory compromise that can make placement of these instruments more difficult.
Ensure Early Source Control	Once the patient is stable, surgical and/or medical control of the source of sepsis must be achieved. Without source control, the patient is likely to show ongoing clinical deterioration of sepsis.
Beware Intestinal Surgery	Poor blood flow in hypotensive and hypovolaemic states, and the high bacterial load within the gastrointestinal lumen place the gut at high risk of surgical or non-surgical dehiscence. Patients with gastrointestinal dysfunction or surgery should be monitored with vigilance for the presence of wound dehiscence and septic peritonitis
Hospital-Acquired Infections	All personnel handling septic patients should observe barrier nursing techniques, and strict asepsis when placing or monitoring intravenous lines, urinary catheters and feeding tubes. Patient cleanliness should be of paramount concern
Inflammation Lasts	The inflammatory cascade will be present as long as tissue injury is present - and will persist through the natural phases of inflammation and debridement, which typically lasts about 3-5 days. Patients are at risk for deterioration at any time during this period, particularly if faced with additional trauma, surgery or procedures that cause tissue injury or vascular compromise (shock etc.)
Monitor for MODS	Be prepared for MODS. Monitoring blood test results for liver, clotting, and renal dysfunction, and monitoring urine output for the presence of oliguria are key elements. Be aware of the requirements for intervention for oliguria, ARDS, fulminant DIC, and other manifestations of MODS, and intervene early if organ dysfunction occurs.

What follows is a description of a suggested checklist of medical therapy for the patient with sepsis[9].

1. Fluid Therapy[13,14]

   Fluid deficits and derangements are common in sepsis and are typically managed using intravenous fluid therapy. The aim of fluid therapy is to restore a normal circulatory volume to restore normal cardiac output, and to optimize tissue oxygen delivery. To achieve these goals, the clinician must treat hypovolaemia, whilst also attending to other key determinants of tissue oxygen delivery, such as ventilation, haemoglobin concentration, cardiac performance, electrolyte status, and hydration deficits, followed by replacement of ongoing fluid losses.

   a. Treat Shock

      Traditionally, it has been suggested that one blood volume of isotonic crystalloid be administered by rapid intravenous infusion to the patient showing clinical signs of shock, within the first hour of patient presentation to provide intravascular support. This equates to providing 25-50 ml/kg/hr. for the cat. Because of the large volumes of crystalloid fluid administered, this approach is associated with the extravasation of a large volume of crystalloid into the interstitial spaces. In the patient with systemic inflammation and sepsis, the increase in vascular permeability and pre-existing excess interstitial fluid may produce symptoms of apparent "volume overload", particularly in pulmonary vessels, producing symptoms of pulmonary oedema. Dilution of plasma proteins and coagulation factors are other potential problems with the traditional large volume fluid therapy approach

      The practice of 'small volume resuscitation' with fluids has sound rationale in the patient with sepsis because it enables titrating the volume of fluid a patient receives, to ideally achieve a set of ideal endpoints listed below…

      - Normal mucous membrane colour
      - Trend to normal heart rate, normal respiratory rate
      - Return of normal blood pressure
      - Normal blood gas analysis
      - Establishment of normal or urine output

      In patients with sepsis, lactated Ringer's solution is the fluid of choice for small volume resuscitation and is administered at a rate of 5-7 ml/kg intravenously over 10 minutes, and repeated until cardiovascular parameters begin to resolve. Synthetic colloids are generally avoided in sepsis, following the discovery of an increased risk of kidney damage demonstrated in humans. Additionally, 0.9% NaCl is also avoided due to higher mortality risk in humans as well. Following successful stabilization of the patient with bolus or 'pulse' fluid therapy, the patient should be placed on fluid therapy at maintenance rates, to avoid excessive fluid loss into the extravascular space. Replacement of hydration deficits should be added to the maintenance fluids. Ongoing losses should be calculated and administered as bolus replacements every 4-6 hrs as required. This is preferable to maintaining the patient on several times maintenance fluid rates.

   b. Maintain optimum haemoglobin concentration.

      In addition to providing intravenous fluid therapy, it is also necessary to ensure patients have sufficient haemoglobin concentrations. The optimum packed red cell mass in a critically ill patient is approximately 27-30%. This level of red cell mass provides adequate blood haemoglobin concentrations, while producing a reduction in blood viscosity which is thought to be optimal in the critical patient in improving oxygen delivery. In humans, the incidence of thromboembolism (DVT) in critical patients is lower when patients have mild anaemia (PCV 27-30%). In critically ill animals, packed red blood cells or whole blood should be administered to maintain a haematocrit of at least 0.27 (PCV = 27%). Transfusion to a haematocrit higher than 30% is not associated with improved survival. However, a PCV lower than 20-24% may result in inadequate oxygen delivery to tissues, patient decompensation, and the development of organ damage. Inadequate oxygen delivery to tissues may acutely be evidenced by clinical signs of

physiological stress, including symptoms of shock, pale mucous membranes, tachypnoea and altered mentation. These symptoms should prompt investigation and transfusion therapy.

c. Optimise Coagulation
Assessment of coagulation is essential in patients with sepsis early in-patient management, as well as at timed intervals (every 12 hours) following initial patient stabilisation, using platelet count, aPTT/PT, activated clotting time and (if available) TEG.

Detection of clinical coagulopathy has, for many years, prompted administration of fresh frozen plasma at an initial dose of 10-30 ml/kg – repeated until coagulopathy has resolved. However, this approach has not led to reduced mortality in humans. It is now recommended to transfuse with fresh frozen plasma if there is occult bleeding, if the patient is at risk of catastrophic haemorrhage, or if invasive surgical or diagnostic procedures are to be carried out. Note that it is not possible to adequately transfuse significant numbers of platelets with whole blood, and thrombocytopenia is best managed by treating the underlying disease, improving tissue oxygen delivery, and providing increased numbers of secondary clotting factors in fresh frozen plasma to manage the patient in fulminant DIC

d. Coagulation and colloid oncotic pressure
Hypoalbuminaemia is common in patients with sepsis. Reduced plasma protein concentrations – particularly albumin – is associated with significant decreases in colloid oncotic pressure, loss of fluid from the intravascular space, and the development of excess tissue oedema and hypovolaemia. This reduces oxygen delivery to tissues, contributes to shock and organ damage. Support of colloid oncotic pressure is challenging. Recent reviews of human literature show limited effectiveness of plasma transfusion in critically ill patients, except those with fulminant coagulopathy, or acute haemorrhage following trauma. If used in the presence of active coagulopathy, or before invasive procedures or surgery, plasma should be administered@ 10 - 30 ml/kg IV. Use of plasma to maintain albumin concentrations is challenging in the critical patient, and is not associated with improved survival. Transfusions of large quantities of plasma or albumin to maintain serum albumin is not currently recommended, as it is not associated with improved survival.

e. Maintain cardiac output and tissue blood flow[14].
Systolic failure is documented in both humans and dogs suffering from sepsis and septic shock, and is associated with negative outcome. Failure of the patient to show signs of improving tissue perfusion (urine output etc.) despite seemingly adequate amounts of intravenous fluid support indicate that the cardiovascular system requires further support. Dobutamine is recommended to improve cardiac output – beginning at a dose of 1-3 micrograms/kg/minute and titrating upwards to clinical effect. Dobutamine has primarily positive inotropic effect (mediated by beta-1 receptor agonist effect), with relatively little effect on heart rate – except at high doses. In cats, seizures have been documented if used continuously beyond 24hrs.

f. Maintain adequate mean arterial blood pressure[15].
Hypotension is defined as a mean arterial pressure below 70 mm Hg, and systolic pressure less than 90 mm Hg. Hypotension is managed by administration of intravenous fluid therapy until the patient is volume replete, with the goal of achieving a systolic blood pressure of between 100 and 120 mm Hg. If hypotension persists, despite fluid resuscitation, vasopressors may be administered. Noradrenaline 0.1-3 micrograms/kg/min is the current vasopressor of choice in cats, and should be titrated to effect, with the dose adjusted according to the physiological response. In patients that fail to respond to fluid therapy and vasopressor therapy, physiological doses of corticosteroids should be considered[17,18].

g. Maintain Normal Cardiac Rhythm[13].
Cardiac arrhythmias are common in patients with severe systemic illness and sepsis. Common predisposing factors include hypovolaemia, pain, electrolyte and acid -base balance abnormalities, thromboembolism and cytokine-mediated cardiac dysfunction. In all cases, a search for, and management of the underlying disease process that has led to the abnormal cardiac rhythm is the most effective means of managing the arrhythmia, followed by judicious use of anti-arrhythmic drugs if required. It is important to note that many anti-arrhythmic drugs have toxic or undesirable side effects if they are administered inappropriately to patients, and indiscriminate use should be avoided. Prior to starting anti-arrhythmic therapy, it is therefore recommended that all patients have the following

i. Assessment of, and correct of intravascular volume and hydration deficits
ii. Correction of electrolyte and acid-base status
iii. Assessment and provision of effective analgesia
iv. Adequate management of the underlying disease process (e.g. sepsis, infection, heart failure etc.).
v. An assessment of whether the abnormal cardiac rhythm is causing hemodynamic compromise to the patient prior to starting anti-arrhythmic therapy must be made, so that an assessment of the effectiveness of therapy can be made, using clinical parameters as well as ECG parameters.

h. Maintain adequate urine volume
Adequate urine output is achieved through management of hypovolaemia and maldistribution of blood flow as has been outlined above. The goal in most patients is urine output. Oliguria may be firstly managed by administration (if appropriate) of fluid boluses using lactated Ringer's solution at 5-10 ml/kg IV over 5-10 minutes, +/- inotropic cardiac support if required. Failure to initiate diuresis may prompt addition of diuretics such as mannitol and/or furosemide. Failure to increase urine output in a normo-volaemic patient following diuretic use is an indication for peritoneal or haemodialysis.

i. Maintain normal blood glucose, electrolyte, and acid-base balance
Electrolyte balance is essential to ensure normal tissue metabolism, cell function, normal cardiac rhythm and vascular tone. Supplementation of intravenous fluids with potassium, magnesium, and glucose is usually based on measurement of serum levels. However, because most body potassium and magnesium is located within the intracellular space, serum measurements poorly reflect total body levels. Supplementation of potassium and magnesium may be based on expected urinary losses, or based on urinary electrolyte measurement to provide a more accurate replacement guide.

2. Maintenance of pulmonary function and adequate gas exchange[13]
Maintenance of pulmonary function and gas exchange involves providing oxygen supplementation by nasal catheter, tracheal catheter, or oxygen-enriched air. Ensuring the patient has an optimal haemoglobin level is also critical in ensuring adequacy of oxygenation of arterial blood. Frequent evaluation for the presence of pleural space disease is critical, particularly in post-trauma, pyothorax, or thoracic surgery patients. Ventilation therapy is indicated in those patients where oxygen supplementation fails to increase SpO_2 above 95%, or in patients where excessive work of breathing is present. Strenuous use of the muscles of respiration can increase oxygen consumption by up to 800%, and decrease cerebral blood flow by as much as 50%. Providing ventilation assistance to these patients is critical in reducing oxygen demand and improving cardiac output, and can be a life-saving measure.

3. Body temperature control[13]
   Low body temperature causes poor blood vessel tone and abnormal blood flow through organs. Normal body temperature is achieved through obtaining normal tissue perfusion and the provision of warm active warming, including humidified air, and warming of intravenous fluids. The goal is a normal rectal temperature of 38.0-39.2^{0}C.

4. Manage infection[13,19-21]
   Infection and sepsis is managed through ensuring adequate tissue perfusion and tissue oxygen delivery as outlined above. Selection of antibiotics should ideally be based on culture and sensitivity from isolated organisms. Prior to obtaining culture and antimicrobial sensitivity results, empirical therapy is used –and can be guided by the most likely source of sepsis, and commonly-associated bacteria, along with a gram stain and identification of rods or cocci on a sample smear. For example, in septic peritonitis, the gastrointestinal tract is the most likely source of infection- appropriate antimicrobial choices would include beta-lactam antibiotics +/- metronidazole +/- fluoroquinolones may be appropriate.
   Empirical choices for initial antibiotic therapy should be restricted to a few antibiotics to minimize development of multiple resistance.
   It is important to begin antimicrobial therapy early. In humans, there is an 8% increase in mortality risk for every hour delay in starting empirical antibiotic therapy.
   Persistent pyrexia, high or low WBC counts in sick animals on apparently appropriate antibiotic therapy suggests the possibility of anaerobic bacterial, viral, or fungal infections. Always consider anaerobes in gastrointestinal tract, liver and biliary disease.

5. Provide nutritional support[13]
   Nutritional support should be provided to the patient with systemic inflammation at the earliest opportunity. Ideally, all calories should be provided by the enteral route of nutrition, as this is associated with superior outcomes when compared to parenteral nutrition provision.
   Patient caloric requirements should be calculated using resting energy requirements (RER) only. Addition of "illness" factors to the RER is generally not favoured in patients with critical illnesss as patients may develop increased incidence of feeding complications, such as hyperglycaemia, vomiting and diarrhoea.

6. Provide support for the gastrointestinal tract[13]
   In addition to nutritional support, the use of antiemetic medications, with or without the use of gastric acid-suppression can improve patient comfort, and reduce the risk of aspiration pneumonia and the development of oesophagitis
   Provide a mechanism for gastric decompression- Cats with sepsis and SIRS have decreased gastric motility. Accumulation of gastric secretions can result in vomiting or regurgitation, both of which are risk factors for aspiration pneumonia and even vagal arrest. Placement of a nasogastric or gastrostomy tube (placed during surgery) facilitates removal of gastric secretions, and additionally serves as an important portal for provision of nutritional support.

7. Provide good nursing care.
   Frequent turning (every 1-2 hours) should be provided if the patient is immobile along with passive range of motion exercises. Catheter insertion sites should be assessed daily and rewrapped at that time. Gloves should be worn at all times when handling critically ill patients to reduce the possibility of introducing hospital-acquired infection to the patient.

8. Analgesia[1,13]
   The patient with septic peritonitis is likely to have significant pain. Due to haemodynamic instability associated with peritonitis, analgesic drugs with minimal depression of cardiovascular function that are non-toxic to poorly perfused tissues are recommended. Fentanyl, delivered by CRI at 3-5µg/Kg/hr is short acting, reversible, and provides a steady state of analgesic support, and is considered the initial analgesic of choice. Addition of ketamine may be considered in patients with poor analgesic control despite therapy with fentanyl.

9. Mentation[13]
   Patients with SIRS or sepsis frequently suffer from alterations in mentation. Repeated patient evaluation is essential to monitor for changes in mentation, as patients with depressed mentation are at a high risk of aspiration pneumonia and death. In general, the following rules apply to managing the patient with sepsis/SIRS and altered mentation
   - Provide postural support that reduces risk of gastro-oesophageal reflux
   - Evaluate intracranial causes of depressed mentation
   - Measure serum osmolality, electrolyte levels, cardiac and respiratory function
   - Maintain euglycaemia - avoid hyper/hypoglycemia

Conclusion

Sepsis, SIRS and MOF/MODS are challenging to manage. In humans, the best success in managing these patients is early, pre-emptive aggressive management of the patient underlying disease, fluid therapy, pulmonary support and intensive monitoring. All of these things are in the ultimate pursuit of improving tissue oxygen delivery, in concert with effective management of the underlying disease. Once organ failure is detected, prompt and aggressive management strategies are required to prevent further patient deterioration and should be pursued.
The elimination of any septic focus is essential [1] and should be carried out at the earliest time possible. Because nutritional support is so crucial to management of patients with SIRS, if an anaesthetic is required, placement of a feeding tube is essential in anticipation of post-operative anorexia.

Management of Hyperglycaemia in Sepsis

Hyperglycaemia is not uncommon in patients with sepsis, and may be associated with stress, and insulin resistance associated with severe disease. Hyperglycaemia has been associated with negative outcome in several human studies of sepsis, and recommendation is therefore made to normalize blood glucose concentrations in these patients. The following protocol outlines the use of insulin continuous infusion, or intermittent intramuscular insulin injection for the control of blood glucose in septic patients.

Insulin Therapy

1. Begin insulin therapy after acute blood volume resuscitation is complete, and rehydration of the patient is well underway, to minimise fluid shift and electrolyte changes during insulin therapy. Additionally, the blood pH should ideally be at or above 7.2 prior to commencement of treatment, as insulin is less effective at pH below this level.
2. Insulin should **never** be administered subcutaneously in the emergency setting, as its absorption rate will vary considerably, leading to erroneous conclusions regarding subsequent doses.
3. Intravenous regular insulin using CRI is the preferred method for administration. However intramuscular regular insulin protocols may also be effective.

Insulin Continuous Infusion:

a. Use regular insulin only for the emergency treatment of hyperglycaemia
b. Dose
    a. Dogs - dose = initially 2.2 U/kg/day. This equates to 0.09 U/kg/hr CRI
    b. Cats - dose = initially 1.1 U/kg/day. This equates to 0.05 U/kg/hr CRI
c. Solution Preparation
    a. Dogs: Add 2.2 units/kg regular insulin to 100 ml 0.9% NaCl
    b. Cats: Add 1.1 units/kg regular insulin to 100 ml 0.9% NaCl
    c. Allow 20 ml of the prepared solution to run through administration set, and discard, as insulin will bind to plastic tubing.
d. Administer the continuous infusion in the following manner:

Blood Glucose	Insulin dose	Insulin Solution Infusion Rate
>15 mmol/L	0.15 U/kg/hr	4.4-7.6 ml/hr
13-15 mmol/L	0.08 U/kg/hr	4 ml/hr
11-13 mmol/L	0.05 U/kg/hr	2.8 ml/hr
8.5-11 mmol/L	0.04 U/kg/hr	2 ml/hr
5.5-8.5 mmol/L	0.04 U/kg/hr	2 ml/hr
<5.5 mmol/L	STOP	STOP

Once the blood glucose drops to below 13 mmol/l, the insulin administration rate is decreased (as above) and 2.5 or 5% dextrose is added to the fluids in the following manner
NB - once glucose is stable at or below 11 mmol/l, begin SC insulin at 0.5 U/kg q 6-12 hr

Blood Glucose (mmol/L)	Glucose Supplementation
13-15 mmol/L	None
11-13 mmol/L	5% dextrose @ 0.5-2.5 ml/hr
8.5-11 mmol/L	5% dextrose @ 0.5-2.5 ml/hr
5.5-8.5 mmol/L	5% dextrose @ 1-5 ml/hr
<5.5 mmol/L	5% dextrose @ 1-5 ml/hr

Insulin Intramuscular Injection Protocol:

a. Patients >10 kg 0.25 U/kg initially, then 0.1 U/kg/hr
b. Patients 3-10 kg 2 U initially, then 1 U/hr
c. Patients <3kg 1 U initially, then 1 U/hr
d. Decrease frequency of administration to every 4 hours once glucose concentrations stabilize or fall below 13 mmol/L, at a dose of 0.1-0.4 U/kg
e. Supplement with intravenous glucose infusion as outlined above once glucose concentrations fall below 11 mmol/L

References:

1. Otto, Cynthia M., and Merilee Costello. "Fresh look at identifying sepsis in cats." *Veterinary medicine* (2010).
2. American college of chest physicians/society of critical care medicine consensus conference: definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. Crit Care Med 1992;20(6):864-874.
3. Hauptman JG, Walshaw R, Olivier NB. Evaluation of the sensitivity and specificity of diagnostic criteria for sepsis in dogs. Vet Surg 1997;26(5):393-397
4. Levy MM, Fink MP, Marshall JC, et al. 2001 SCCM/ESICM/ ACCP/ATS/SIS international sepsis definitions conference. Intensive Care Med 2003;29(4):530-538.
5. Babyak, Jonathan M., and Claire R. Sharp. "Epidemiology of systemic inflammatory response syndrome and sepsis in cats hospitalized in a veterinary teaching hospital." Journal of the American Veterinary Medical Association 249, no. 1 (2016): 65-71.
6. Costello, Merilee F., Kenneth J. Drobatz, Lillian R. Aronson, and Lesley G. King. "Underlying cause, pathophysiologic abnormalities, and response to treatment in cats with septic peritonitis: 51 cases (1990-2001)." Journal of the American Veterinary Medical Association 225, no. 6 (2004): 897-902.
7. Klainbart, Sigal, Limor Agi, Tali Bdolah-Abram, Efrat Kelmer, and Itamar Aroch. "Clinical, laboratory, and hemostatic findings in cats with naturally occurring sepsis." Journal of the American Veterinary Medical Association 251, no. 9 (2017): 1025-1034.
8. Troìa, Roberta, Giulia Mascalzoni, Stefano Calipa, Ilaria Magagnoli, Francesco Dondi, and Massimo Giunti. "Multiorgan dysfunction syndrome in feline sepsis: prevalence and prognostic implication." Journal of feline medicine and surgery 21, no. 6 (2019): 559-565.
9. DeClue, Amy E., Cherlene Delgado, Chee-hoon Chang, and Claire R. Sharp. "Clinical and immunologic assessment of sepsis and the systemic inflammatory response syndrome in cats." Journal of the American Veterinary Medical Association 238, no. 7 (2011): 890-897.
10. Brady CA, et al. Severe sepsis in cats: 29 cases (1986-1998). J Am Vet Med Assoc. 2000; 217(4):531.
11. Troìa, Roberta, Giulia Mascalzoni, Stefano Calipa, Ilaria Magagnoli, Francesco Dondi, and Massimo Giunti. "Multiorgan dysfunction syndrome in feline sepsis: prevalence and prognostic implication." Journal of feline medicine and surgery 21, no. 6 (2019): 559-565.
12. Waddell, Lori S., Colleen A. Brady, and Kenneth J. Drobatz. "Risk factors, prognostic indicators, and outcome of pyothorax in cats: 80 cases (1986–1999)." Journal of the American Veterinary Medical Association 221, no. 6 (2002): 819-824.
13. Kirby, Rebecca, and Andrew Linklater, eds. Monitoring and intervention for the critically ill small animal: the rule of 20. John Wiley & Sons, 2016.
14. Davis, Harold, Tracey Jensen, Anthony Johnson, Pamela Knowles, Robert Meyer, Renee Rucinsky, and Heidi Shafford. "2013 AAHA/AAFP fluid therapy guidelines for dogs and cats." Journal of the American animal hospital association 49, no. 3 (2013): 149-159.
15. Ince, Mehmet Ege, Kursad Turgut, and Amir Naseri. "Echocardiographic Assessment of Left Ventricular Systolic and Diastolic Functions in Dogs with Severe Sepsis and Septic Shock; Longitudinal Study." Animals 11, no. 7 (2021): 2011.
16. Pascoe, Peter J., Jan E. Ilkiw, and Bruno H. Pypendop. "Effects of increasing infusion rates of dopamine, dobutamine, epinephrine, and phenylephrine in healthy anesthetized cats." American journal of veterinary research 67, no. 9 (2006): 1491-1499.
17. Pisano, Simone RR, Judith Howard, Horst Posthaus, Alan Kovacevic, and Ivayla D. Yozova. "Hydrocortisone therapy in a cat with vasopressor-refractory septic shock and suspected critical illness-related corticosteroid insufficiency." Clinical case reports 5, no. 7 (2017): 1123.
18. Creedon, Jamie M. Burkitt. "Controversies surrounding critical illness-related corticosteroid insufficiency in animals." Journal of Veterinary Emergency and Critical Care 25, no. 1 (2015): 107-112.
19. Zubert S, *et al.* Antibiotics in sepsis and septic shock: Like everything else in life, timing is everything. Crit Care Med. 2010; 38(4):1211.
20. Kumar A, *et al.* Initiation of inappropriate antimicrobial therapy results in a fivefold reduction of survival in human septic shock. Chest. 2009; 136(5):1237.
21. Kumar A, *et al.* Early combination antibiotic therapy yields improved survival compared with monotherapy in septic shock: A propensity-matched analysis. Crit Care Med. 2010; 38(9):1773.

The Acute Abdomen in the Cat

Philip R Judge BVSc MVS PG Cert Vet Stud MACVSc (Veterinary Emergency and Critical Care; Medicine of Dogs)

Introduction

Acute abdomen refers to the acute onset of abdominal pain. Acute abdominal discomfort is frequently a sign of significant, and potentially life-threatening abdominal disease – but may represent either minor abdominal disease, or even extra-abdominal disease.

Most references on acute abdomen in dogs and cats focus on the condition in canine patient – and whilst many of the causes, and the diagnostic work-up of acute abdomen in the cat are similar to those in the dog, there are a few crucial differences. The focus of this short reference is to review published literature in the acute abdomen of the cat, as well as known causes of abdominal pain in the cat, to better inform the condition.

Common Causes of Acute Abdomen in the Cat[1-4]

The following table outlines some of the most common causes of acute abdominal pain in the cat:

Gastrointestinal system • Foreign body • Obstruction • Intestinal perforation • Ischaemia • Neoplasia • Gastroenteritis • Colitis • Intussusception • Ileus	Urogenital system • Urinary tract obstruction • Rupture of the urinary tract • Pyelonephritis • Urolithiasis • Acute kidney injury • Neoplasia
Peritoneal disease • Septic peritonitis • Haemo-peritoneum • FIP • Disseminated neoplasia	Hepatobiliary • Cholangiohepatitis • Biliary obstruction • Cholecystitis • Neoplasia
Body wall • Penetrating injury • Hernia	Pancreatic • Pancreatitis • Neoplasia
Referred pain • Intervertebral disc disease • Spinal neoplasia • Pelvic trauma	Pyometra

Diagnostic Approach[1-4]

The diagnostic approach to the acute abdomen in the cat involves attention to the following:

1. History: Obtain a complete history, including the following:
    a. History of in-contact animals
    b. Vaccination history
    c. Indoor/outdoor activity
    d. Trauma history
    e. Diet and dietary indiscretion
    f. Exposure to toxins
    g. Medications
    h. Symptoms
        i. Gastrointestinal
        ii. Urinary
        iii. Reproductive
        iv. Neurological
        v. Respiratory
        vi. Musculoskeletal/mobility
        vii. Weight change
        viii. Abdominal conformation
2. Physical examination:
    a. Vital organ systems evaluation
        i. An assessment should be made of respiratory, cardiovascular and neurological systems to determine if emergency life-saving intervention is required (oxygen therapy, respiratory support, fluid or transfusion therapy etc.)
        ii. Repeat this assessment as indicated, and intervene if abnormalities arise
    b. Abdominal assessment
        i. Inspection: Inspect the abdominal wall for the following
            1. Bruising
            2. Puncture
            3. Distension
            4. Defect resulting in herniation, or abnormal shape
        ii. Palpation
            1. Abdominal palpation has relatively low sensitivity for detection of many abdominal diseases, but can nevertheless be valuable in many patients and should not be overlooked[5]
                a. The sensitivity of abdominal palpation for detection of ascites was found to be 32% in one study[5]
                b. The sensitivity of abdominal palpation for detection of abdominal masses was found to be 43.7% in the same study[5]
            2. Determine presence of abdominal pain
            3. Determine region of abdominal pain
            4. Determine the location and size of abdominal organs

c. Complete physical examination
    i. Oral examination – look for evidence of linear foreign bodies that may be anchored in the sub-lingual region
    ii. Spinal palpation
    iii. Rectal examination
    iv. Musculoskeletal examination

3. Diagnostic imaging
    a. Radiography
        i. Abdominal radiography may allow assessment of intestines, and other organs for location, size and shape, foreign bodies, and radiopaque structures, such as cholelithsor uroliths.
        ii. Evaluation of the pelvis and thoraco-lumbar spine should also be conducted in cases or trauma
        iii. Thoracic radiography may allow determination of diaphragmatic integrity, evidence of pulmonary or pleural fluid accumulations, evidence of cardiac disease, or neoplastic pulmonary infiltrations
    b. AFAST and TFAST ultrasonography
        i. AFAST sonography allows detection of abdominal fluid accumulations, which should be sampled for cytology and biochemistry analysis +/- microbial culture
        ii. AFAST sonography allows cursory evaluation of abdominal organ structure and size, prior to complete abdominal evaluation
        iii. TFAST sonography allows detection of peripheral pulmonary fluid accumulations (pulmonary oedema, pneumonia etc.), pulmonary nodules (fungal nodules, or metastatic neoplasia) or peripheral pulmonary masses, which may direct further diagnostics
        iv. TFAST may allow detection of pleural fluid accumulation, which should be aspirated for evaluation (cytology, biochemistry +/- culture
        v. TFAST allows early assessment of cardiac structure. In its simplest form, a left atrium: aorta diameter ratio should be determined, as this can herald the presence of underlying cardiac disease
    c. Complete abdominal ultrasonography
        i. Ultrasound examination has a high degree of sensitivity and specificity for detection of abdominal disease, and a correlation of approximately 86% when compared to surgical findings in patients with surgical abdominal disease[5]
        ii. Abdominal ultrasonography compliments the AFAST ultrasound and abdominal radiography and provides more detailed organ system evaluation.

4. Blood and urine analysis
    a. Routine blood analysis should include complete blood count, serum biochemistry, blood gas, serum electrolyte analysis and blood lactate, and blood smear evaluation
    b. Routine urine analysis should include chemistry, urine specific gravity, and cytology, +/- urine culture if appropriate.

5. Abdominal fluid analysis should include:
    a. Biochemistry to evaluate
        i. Bilirubin
        ii. Urea and creatinine
        iii. Electrolytes
        iv. Lactate
    b. Cytology

A Review of Literature on the Acute Abdomen in Cats

I. Septic Peritonitis: The Studies

In a review of 26 cats with surgically treated septic peritonitis, the following were noted[6]:

1. Aetiology
    a. In this study cohort, the causes of septic peritonitis included:
        i. Trauma resulting in damage to the gastrointestinal tract, biliary tract or abdominal wall (31%)
        ii. Surgery of the intestine, resulting in intestinal wound dehiscence (23%)
        iii. Neoplasia (12%)
        iv. Miscellaneous (23%)
            1. Intussusception
            2. Abscess formation of undetermined cause
            3. Undetermined
2. Clinical Signs
    a. Lethargy, and anorexia were the most common clinical signs
    b. Abdominal pain was noted in only 38% of cats with septic peritonitis
    c. Vomiting was present in 42% of cases
3. Pathology
    a. Blood lactate was significantly higher in non-survivors (median 3.5 mmol/L) than survivors (median 1.4 mmol/L)
    b. Glucose and total protein concentrations were not statistically different between survivors and non-survivors
    c. White cell counts were not statistically different between survivors and non-survivors
    d. The most common bacterial isolates were
        i. E. coli
        ii. Enterococcus faecalis
        iii. Pasteurella
        iv. Corynebacterium
4. Outcome
    a. Survival to discharge was 46%
    b. Median duration of hospitalisation was 7 days

In another review of 83 cats with septic peritonitis diagnosed by cytology or culture, the following were noted[7]:

1. Aetiology
    a. Trauma, with intestinal dehiscence was the most common cause
    b. Idiopathic septic peritonitis, with no identified underlying cause was the second most frequent cause
    c. Other causes included
        i. Pyometra
        ii. Grass awn migration
        iii. Septic bile peritonitis
        iv. Penetrating wounds
        v. Urinary tract origin
2. Pathology
    a. Most common bacterial isolates were E. coli, Enterococcus, Clostridium and Bacteroides species
    b. CBC and routine biochemistry findings were not predictive of survival, except glucose (see below)
3. Prognosis and associated factors
    a. 69.9% survived to discharge
    b. Body temperature was not correlated with survival or non-survival
    c. Cats with higher blood glucose concentration on presentation had higher mortality (7 mmol/L mean for survivors vs. 9.12 mmol/L mean for non-survivors)
    d. Blood lactate concentration showed no significant difference between survivors and non-survivors
    e. Appropriate early empirical antimicrobial therapy on presentation was associated with increased survival

Septic Peritonitis in Cats: Key Points

1. Abdominal pain may only be present in less than half of cats with septic peritonitis
2. Indicators such as blood lactate and blood glucose may inform prognosis, but at present, available evidence is contradictory
3. Appropriate and early antimicrobial therapy can improve outcome
4. In some cats, the cause of septic peritonitis is not able to be identified
5. Overall prognosis can vary between 46 and 70%

II. Gastroduodenal Ulceration and Perforation: The Studies

1. Gastro-duodenal Perforation[8]
    a. A review of 16 dogs and 7 cats with gastroduodenal perforation revealed the following in relation specifically to cats:
        i. Clinical Signs
            1. Lethargy
            2. Weight loss
            3. Vomiting
            4. Reduced appetite
            5. Dehydration
            6. Shock
            7. Clinical evidence of gastric bleeding, whilst common in dogs, was absent in 80% of cats
            8. Clinical signs of abdominal pain were absent in 80% of cats
        ii. Diagnostic findings
            1. Blood tests
                a. Neutrophilia was common, and may be either mature, or with a left shift
                b. Anaemia was present in 29% of cats
            2. Imaging
                a. Radiographic findings included loss of serosal detail, and/or the presence of free abdominal gas
                b. Ultrasonographic findings included free abdominal fluid
                c. Confirmation of gastroduodenal rupture by ultrasonography was not able to be made in any cat in this study
            3. Predisposing causes
                a. Predisposing illness was present in call cats in this cohort, with illnesses including:
                    i. Liver disease
                    ii. Gastroduodenal neoplasia – including lymphoma, and angiosarcoma
            4. Prognosis
                a. Survival rate was 14% in this study population, with the majority of patients euthanized
            5. Other conclusions
                a. Acute decompensation was not always present, suggesting dramatic signs of acute abdomen following gastroduodenal perforation may not be common, until significant peritoneal reaction occurs
2. Gastric Perforation[9]
    a. A case series detailed three cats with successful surgical management of gastric ulcers.
        i. Clinical Presentation
            1. Lethargy
            2. Reduced appetite
            3. Occasional vomiting
            4. Abdominal enlargement
            5. Tachycardia
            6. Dehydration
            7. Non-steroid anti-inflammatory medication had been administered to all cats within the 7 days prior to admission

ii. Imaging
    1. Radiography: pneumoperitoneum; evidence of intestinal ileus
    2. Ultrasound: free abdominal fluid; pneumoperitoneum; intestinal ileus
iii. Haematology
    1. Neutrophilia with a left shift
    2. Anaemia was present in 1 cat
iv. Conclusions
    1. Vague presenting signs highlight the importance of thorough patient evaluation in any cat presenting with non-specific signs, particularly with history of non-steroid anti-inflammatory medication
    2. Abdominal radiography demonstrating pneumoperitoneum was the most sensitive test

3. Gastroduodenal ulceration[10]
    a. A case series of 8 cats with an eventual diagnosis of gastroduodenal ulceration presented the following findings
        i. Clinical presentation
            1. Vomiting is the most common presenting clinical sign
            2. Abdominal pain was present in only 1/3 of patients
        ii. Clinical pathology
            1. Anaemia was observed in all 8 cats in this study
            2. Neutrophilia was also present in all cats, with some mature, and some immature
        iii. Pathology and clinical course
            1. Neoplasia
                a. Cats with primary intestinal neoplasia e.g. gastrinoma and gastric tumours) had a more protracted disease course, with weight loss etc.
                b. Extra-intestinal tumours were more likely to be located in the duodenum; whereas gastric tumours were associated with gastric ulceration
            2. Non-neoplastic causes
                a. Cats with non-neoplastic causes typically had a shorter duration of disease
                b. Ulcers were more likely to be in the stomach
            3. Prognosis: if appropriate therapy (transfusion therapy, surgical removal of the ulcer, or gastric diversion surgery) was administered, prognosis was fair to good, with most cats surviving beyond 12 months

4. Gastric Ulceration[11]
    a. A case report of a necro-ulcerative haemorrhagic gastritis in a cat, following administration of 3% hydrogen peroxide as an emetic agent described the following:
        i. Acute onset of haematemesis within 24 hours of administration of 15 ml 3% hydrogen peroxide
        ii. Blood tests revealed a monocytosis, eosinopaenia and mild hyperglycaemia and ALT increase
        iii. Radiography revealed no significant findings
        iv. Ultrasound of the abdomen did not reveal significant findings
        v. An exploratory laparotomy revealed widespread ulceration of 60% of the gastric mucosa. Histopathology revealed severe necro-ulcerative haemorrhagic gastritis

Gastroduodenal Ulceration and Perforation: Key Points

1. Lethargy, anorexia and vomiting are the most common clinical signs
2. Abdominal pain may be present in as few as 20% of cases
3. Neutrophilia is common; anaemia may be present with significant bleeding
4. Radiography is an insensitive test, except in cases of gastro-duodenal perforation, where the presence of free peritoneal gas was highly diagnostic for bowel rupture
5. Given the vague symptoms, a complete patient evaluation is essential to allow detection of gastroduodenal ulceration, and determination of an underlying cause

III, Gastric Dilatation and Dilatation-Volvulus: The Studies

Both gastric dilatation, as well as gastric dilatation-volvulus syndrome have been reported in cats.

A case report of 2 cats, along with a review of a further 8 cases of gastric dilatation in cats revealed the following[12]:

i. Signalment:
   a. 11 weeks of age to 11 years
   b. No breed or sex predilection
ii. Presenting signs
   a. Abdominal distension
   b. Dyspnoea
iii. Co-morbidities
   a. Diaphragmatic hernia
   b. Respiratory distress
   c. Gastric torsion
iv. Management
   a. Fluid therapy, analgesia
   b. Decompression in cases of simple dilatation
   c. De-rotation and gastropexy in cases of dilatation-volvulus
   d. Management of co-morbidities e.g. diaphragmatic hernia
v. Prognosis
   a. Patients with respiratory distress had a worse prognosis
   b. Mortality rate overall was 30%

A further case report of 2 cats – both with gastric dilatation-volvulus syndrome, provided some additional information on this interesting condition in cats[13]:

1. Presenting signs: both cats in this paper presented with marked respiratory distress as the primary complaint, characterised by marked increased inspiratory effort, and increased lung sounds in one cat, and tachypnoea, vocalization and foaming at the mouth in the other cat
2. Physical examination revealed a distended, painful abdomen on palpation in both cats
3. Radiography confirmed gastric distension in both cats, but volvulus in only 1 cat.
4. Treatment involved supportive care, and surgical correction of GDV in both cats, with gastropexy performed

Gastric Dilatation and Dilatation-Volvulus: Key Points

1. Respiratory distress and abdominal distension are the most common presenting signs
2. Abdominal radiography may not be diagnostic of torsion in some cases
3. Co-morbidities include diaphragmatic hernia and severe respiratory distress, and these require management
4. Exploratory laparotomy and surgical management may be required in patients with apparent simple gastric dilatation not responding to gastrocentesis

IV. Intussusception: The Studies

A retrospective study looked at 9 cases of intussusception in cats, along with 27 in dogs to evaluate predisposing causes, clinical signs and management[14]. Features of feline intussusception included:

1. Presenting clinical signs included (in order of descending frequency) vomiting, depression, anorexia, diarrhoea and abdominal pain. Abdominal pain was present in 30% of cases. Clinical signs were present for between 1-7 days (median 4 days)
2. Physical examination revealed abdominal pain (30%), dehydration (27%) and a palpable abdominal mass (30%)
3. Predisposing causes were not identified in most cats, with endoparasitism occurring in 1 cat (11%)
4. Radiography revealed the presence of intestinal obstruction or mass in 50% of cases.
5. Surgical findings revealed the most common site of intussusception to be the ileocolic junction (55%). Small intestinal enteric intussusception occurred in 33% of cases. 88% of cats had significant serosal adhesions at the point of intussusception. Peritonitis was evident in 11% of cats. As a result of adhesions, most cats were not amenable to simple reduction or their intussusception, and required intestinal resection and anastomosis.

Another study looked at both medical records and histopathology reports of dogs and cats with intussusception[15]. Of the 18 cats evaluated, the following clinical and histological findings were noted:

1. Signalment: mean presenting age was 36 months; no sex or breed predilection was noted
2. Surgical and histopathology findings:
    a. 17% of intussusceptions were at the ileocecal junction; 72% were in the small intestine; and 11% were in the colon.
    b. 22% of cats had inflammatory disease described as the cause for the intussusception. Causes of the inflammation were identified as lymphocytic-plasmocytic enteritis, eosinophilic enteritis, and a single case of ascarid-associated inflammation
    c. 27% of cats had neoplastic disease as the cause for the intussusception. Neoplasia associated with intussusception included lymphosarcoma, adenocarcinoma and mast cell tumour
    d. Concurrent conditions included patients with no intestinal wall underlying cause included pancreatitis, pyogranulomatous peritonitis, and hepatocellular carcinoma

Intussusception: Key Points

1. About 50% of cats with intussusception have underlying intestinal disease related either to inflammatory bowel disease or neoplasia, making histopathology of the site of intussusception and other intestinal sites important.
2. Endoparasites are also a possible cause and should be evaluated.
3. Concurrent diseases such as pancreatitis, peritonitis and liver disease are relatively common. Clinicians should be aware, and patient evaluation should include investigations of these tissues and organs
4. Radiography has sensitivity of 50% in detection of intussusception in the cat
5. Adhesions at the site of intussusception are common, necessitating intestinal resection and anastomosis in the majority of cases

V. Intestinal Volvulus and Colonic Torsion: The Studies

Intestinal and colonic torsion and volvulus is infrequently reported in veterinary literature. However, isolated case reports do exist, and they describe these serious and potentially fatal conditions that veterinarians should be aware of.

A single case study of colonic volvulus in a cat has been reported[16]. The case was a 12-year old cat that had chronic diarrhoea of several months duration, and a 1 day history of severe depression and loss of appetite immediately prior to presentation.

Clinical examination findings revealed the following:

1. Hypothermia
2. Cachexia
3. Marked dullness
4. Dehydration
5. Abdominal palpation revealed a large, gas-filled non-painful structure, filling the majority of the abdominal cavity. A taut band of tissue was palpated extending from the caudal end of the gas-filled structure to the pelvic inlet

Clinical pathology revealed the presence of hypoglycaemia, hypokalaemia and elevated blood lactate, as well as a metabolic acidosis.

Abdominal radiographs revealed a large, U-shaped, gas-filled intestinal (colonic) structure in the mid and cranial abdomen, and another dilated segment to the left of the midline. The gas-distended descending colon terminated in a rounded end, suggesting complete obstruction. A small diameter, red rubber feeding tube was unable to be passed per rectum to relieve obstruction

Surgical findings included:

1. Volvulus at the root of the colonic mesentery
2. Torsion of the distal colon just cranial to the pelvic inlet
3. Excision of the distal ileum, ileo-colic junction, and devitalized segment of the colon was required

This particular patient remained hypotensive during surgery and was euthanized shortly following completion of surgery.

A two-case series of small intestinal mesenteric volvulus in cats revealed the following[17]:

1. Clinical signs – present for 1 day (1 cat) and 3 days (1 cat)
    a. Vomiting
    b. Lethargy
    c. Anorexia
2. Physical examination
    a. Shock
    b. Abdominal distension and discomfort
    c. Several gas-distended loops of bowel may be palpated, and/or the presence of a palpable abdominal mass

3. Diagnosis
    a. Blood tests revealed
        i. Anaemia
        ii. Leukopaenia or leukocytosis
        iii. Thrombocytopaenia
        iv. Pre-renal azotemia
    b. Radiography
        i. Marked gas distension of small intestinal loops
        ii. Variable loos of serosal detail elsewhere in the abdomen
        iii. One cat had free peritoneal gas present
    c. Surgery
        i. Abdominal effusion with serosanguinous fluid
        ii. 270-360-degree clockwise rotation of the small intestine about the mesenteric root
        iii. Intestines had herniated through a tear in the mesentery
4. Prognosis
    a. Following resection and anastomosis of devitalized bowel, both cats recovered.
    b. Chronic diarrhoea was present due to the large amount of small intestine resected in one cat; with chronic soft faeces present in the other

Both cats in this series survived to discharge, indicating a potentially better prognosis than this condition has in dogs.

Intestinal Volvulus and Colonic Torsion: Key Points

- Abdominal distension associated with gas-distended bowel are common clinical findings, along with the presence of cardiovascular shock, and obtundedness
- Radiography is the most useful diagnostic tool available, and allows visualisation of gas-distended loops of bowel
- Inability to pass a small-bore feeding tube into the colon to relive gas pressure can assist diagnosis of colonic torsion/volvulus
- Small intestinal mesenteric torsion carries a better prognosis than the condition in dogs

Constipation

Constipation: Key Points

Constipation is a relatively common complaint in cats, that can occasionally present as an acute abdomen. A review of risk factors and outcome in cats with constipation found the following, with respect to acute abdomen[18]:

1. Patients with abdominal pain on palpation were less likely to defecate following an enema
2. Adjunctive treatments such as fluid therapy and laxatives increased the likelihood f a successful enema, but were not statistically significant in determining outcome
3. Older and overweight cats were more likely to present with constipation
4. Cats with chronic kidney disease were more likely to suffer from constipation
5. Cats with chronic kidney disease were more likely to suffer from constipation
6. Fluid/lubricant enemas resulted in defecation in 52% of cases

Uro-Abdomen

Uro-Abdomen: Key Points

Uro-abdomen is a common complication in dogs and cats, and occurs following damage to the urinary tract, either as a result of trauma, urethral obstruction, bladder expression or catheterisation, neoplasia or following urinary tract surgery[19].

The presence of urine in the abdominal cavity results in abdominal pain, electrolyte and acid-base disturbances – notably metabolic acidosis and hyperkalaemia.

Diagnosis is usually made using abdominal ultrasound, and abdominal fluid analysis, +/- positive contrast urethrocystography and/or intravenous excretory urography, which demonstrate free abdominal urine.

Stabilisation of patient with uroabdomen involves medical therapy to restore circulating blood volume, normalize serum electrolyte concentrations, and to provide temporary urinary diversion using a combination of urethral catheterisation, as well as peritoneal drainage +/- peritoneal dialysis, prior to definitive surgical repair. Analgesia should also be administered, with antibiotic therapy if indicated by the presence of bacteria evident in the urinary tract or free abdominal fluid[19]. Other indications for antimicrobial therapy in uroabdomen include:

1. Recent urinary catheterisation
2. Chronic kidney failure
3. Prostatitis
4. Urolithiasis
5. Urinary tract neoplasia
6. Neurogenic bladder
7. Recent chemotherapy, glucocorticoid, or immunosuppressive therapy
8. Diabetes mellitus or hyperadrenocorticism, or feline leukaemia or immunodeficiency virus infection

Prognosis for uroabdomen is generally good, if early patient stabilisation can be achieved, and if the underlying condition can be managed.

A common concern when managing patients with urethral obstruction, is that emergency decompressive cystocentesis may result in the presence of uroabdomen and abdominal effusion. An evaluation of 45 cats with naturally-occurring urethral obstruction revealed the following[19]:

1. 33% of cats with urethral obstruction had abdominal effusion prior to cystocentesis
2. 49% of cats with urethral obstruction had abdominal effusion following cystocentesis (a 16% increase)
3. Abdominal effusion noted following cystocentesis was scant in volume, and did not result in complications

The authors concluded that a single decompressive cystocentesis to facilitate stabilisation and management of cats with urethral obstruction in their study population.

Diagnostic Ultrasound

The Value

of Diagnostic Ultrasound: Key Points

A study evaluating the diagnostic utility of abdominal ultrasound in the patient with acute abdomen has been evaluated in dogs and cats[20]. Relevant findings from this study included:

1. There was a good agreement between the ultrasound diagnosis and intraoperative findings of 86.9% (80/92), for both primary and secondary lesions
2. Surgical diseases were not identified by ultrasound in 13% of cases taken to surgery – most notably bile duct rupture, intestinal foreign body and gastrointestinal perforation
3. Cytology and histopathology examinations corroborated ultrasound diagnosis in 86.4% of primary and 66.2% of secondary lesions.
4. Care should be taken with interpretation of gastrointestinal perforation, pancreatic neoplasia, omental tumors, gastrointestinal foreign body and common bile duct rupture, as these lesions have a higher incidence of either not being detected or being misinterpreted with ultrasound. Other diagnostic tests such as palpation and radiography may improve diagnostic sensitivity.

References:

1. Walters PC. Approach to the acute abdomen. Clinical techniques in small animal practice. 2000 May 1;15(2):63-9.
2. Snow S, Beal MW. General approach to the acute abdomen. Feline emergency and critical care medicine. 2010 May 18:229-44.
3. Beal MW. Approach to the acute abdomen. The Veterinary clinics of North America. Small animal practice. 2005 Mar 1;35(2):375-96.
4. Mazzaferro EM. Triage and approach to the acute abdomen. Clinical techniques in small animal practice. 2003 Feb 1;18(1):1-6.
5. Abdellatif A, Kramer M, Failing K, Von Pückler K. Correlation between Preoperative Ultrasonographic Findings and Clinical, Intraoperative, Cytopathological, and Histopathological Diagnosis of Acute Abdomen Syndrome in 50 Dogs and Cats. Veterinary Sciences. 2017 Sep;4(3):39.
6. Parsons KJ, Owen LJ, Lee K, Tivers MS, Gregory SP. A retrospective study of surgically treated cases of septic peritonitis in the cat (2000–2007). Journal of Small Animal Practice. 2009 Oct;50(10):518-24.
7. Scotti KM, Koenigshof A, Sri-Jayantha LS, Kato M, Bishop M, Barr JW, Pashmakova MB. Prognostic indicators in cats with septic peritonitis (2002–2015): 83 cases. Journal of Veterinary Emergency and Critical Care. 2019 Nov;29(6):647-52.
8. Hinton LE, McLoughlin MA, Johnson SE, Weisbrode SE. Spontaneous gastroduodenal perforation in 16 dogs and seven cats (1982–1999). Journal of the American Animal Hospital Association. 2002 Mar;38(2):176-87.
9. Cariou MP, Halfacree ZJ, Lee KC, Baines SJ. Successful surgical management of spontaneous gastric perforations in three cats. Journal of Feline Medicine and Surgery 2010 12:1; 36-41
10. Liptak, JM, Hunt, GB, Barrs, VRD Gastroduodenal ulceration in cats: Eight cases and a review of the literature. J Feline Med Surg 2002; 4: 27–42.
11. Obr TD, Fry JK, Lee JA, Hottinger HA. Necroulcerative hemorrhagic gastritis in a cat secondary to the administration of 3% hydrogen peroxide as an emetic agent. Journal of Veterinary Emergency and Critical Care. 2017 Sep;27(5):605-8.
12. Bredal WP, Eggertsdóttir AV, Austefjord O. Acute gastric dilatation in cats: a case series. Acta Veterinaria Scandinavica. 1996 Jan 1;37(4):445-52.
13. Leary ML, Sinnott-Stutzman V. Spontaneous gastric dilatation-volvulus in two cats. Journal of Veterinary Emergency and Critical Care. 2018 Jul;28(4):346-55.
14. Levitt L, Bauer MS. Intussusception in dogs and cats: A review of 36 cases. The Canadian Veterinary Journal. 1992 Oct;33(10):660.
15. Levien AS, Baines SJ. Histological examination of the intestine from dogs and cats with intussusception. Journal of Small Animal Practice. 2011 Nov;52(11):599-606.
16. Drobatz KJ, Hughes D, Hill C, Walker L. Volvulus of the colon in a cat. Journal of Veterinary Emergency and Critical Care. 1996 Jul;6(2):99-102.
17. Knell SC, Andreoni AA, Dennler M, Venzin CM. Successful treatment of small intestinal volvulus in two cats. Journal of feline medicine and surgery. 2010 Nov;12(11):874-7.
18. Benjamin SE, Drobatz KJ. Retrospective evaluation of risk factors and treatment outcome predictors in cats presenting to the emergency room for constipation. Journal of Feline Medicine and Surgery. 2020 Feb;22(2):153-60.
19. Gerken KK, Cooper ES, Butler AL, Chew DJ. Association of abdominal effusion with a single decompressive cystocentesis prior to catheterization in male cats with urethral obstruction. Journal of Veterinary Emergency and Critical Care. 2020 Jan;30(1):11-7.

20. Abdellatif A, Kramer M, Failing K, Von Pückler K. Correlation between Preoperative Ultrasonographic Findings and Clinical, Intraoperative, Cytopathological, and Histopathological Diagnosis of Acute Abdomen Syndrome in 50 Dogs and Cats. Veterinary Sciences. 2017 Sep;4(3):39.

The Approach to the Dyspnoeic Cat

Dr. Philip R Judge BVSc MVS PG cert Vet Stud MACVSc (VECC; Medicine of Dogs)

The Approach to the Cat in Respiratory Distress

Any patient in respiratory distress has a potentially life-threatening emergency that requires immediate attention to prevent tissue hypoxia, alleviate shock and prevent death. However, the cat in respiratory distress is uniquely susceptible to acute deterioration during handling and manipulation, which adds an additional degree of complexity to the establishment of a diagnosis.

Clinical signs of respiratory distress in the cat include the following:

- Rapid, shallow, or noisy breathing
- Open-mouth breathing may be present
- Orthopnoea – sitting in one position – often sternal and hunched – in order to optimise comfort during difficult respiration.
- Dilated pupils
- Excessive salivation
- Lethargy
- Reduced food intake

Respiratory distress is commonly described using the term "Dyspnoea". Dyspnoea by definition refers to difficult or laboured breathing and signs may be associated with abnormalities at any level of the respiratory system. The causes of dyspnoea can be categorized depending on the level of the respiratory system affected – upper airway, the lung or the pleural cavity. Dyspnoea can also be characterized depending on the particular part of the breathing cycle that is affected by difficult or laboured respiration. Let's explain that another way.

Difficulty in breathing can result from any of the following

1. **Airway obstruction** – airway obstruction can be partial or complete, and can occur at any level within the respiratory tract, from the external nares, nasal passages, pharynx, larynx, trachea, and bronchi to the small bronchioles. The causes of airway obstruction are numerous, and include foreign bodies; anatomical deformity; haemorrhage or vomitus in the airways; neoplasia; oedema or tissue swelling; and trauma resulting from skull fractures, laryngeal fractures, haematoma formation in the airways etc.
2. **Lack of pulmonary expansion (collapse of the lung)** – lack of lung expansion occurs when the lungs physically cannot expand to allow air movement into the alveoli, to allow gas exchange to occur. This can occur as a result of the following
    a. ***Damage to central nervous system*** control over the respiratory centres and diaphragm – this is the type of ventilation failure we see in tick paralysis, snake bites, head trauma, and other neuro-muscular disease.
    b. ***Damage to integrity of chest wall*** – trauma or surgery to the chest wall result in physical disruption to the chest wall, pain, and other injury that can interfere with the ability of the patient to optimally expand the lungs during inhalation. Additionally, these types of injury are commonly associated with damage to the pulmonary parenchyma and pleural space, which further may compromise respiratory capacity.
    c. ***Pleural space disease*** – The presence of air or fluid within the pleural space results in loss of the normal cohesion between the lungs and the thoracic wall, and causes the lungs to collapse under their natural elastic recoil, which reduces the effectiveness of respiration. Additionally, compromise to the volume of the pleural space, caused by conditions such as

diaphragmatic hernia, with herniation of abdominal organs into the pleural cavity, may also compromise the ability of the lungs to expand during inspiratory efforts.

d. ***Damage to the gas exchange unit*** – Damage to the alveolus within the lung may occur with many conditions. Aspiration of stomach/gastric acid into the lungs, infection (pneumonia), infiltration of the lungs with cells (inflammatory cells, blood, or neoplastic cells), or with fluid (for example the oedema that occurs in congestive heart failure, orinfiltration of blood into the alveolus following trauma to the chest) all cause damage to the alveolus, or interference with gas exchange. Additionally, the presence of excess fluid within the pulmonary parenchyma dilutes lung surfactant, which causes a rise in alveolar surface tension, and collapse of alveoli within the lung.

3. Altered pulmonary circulation – In some diseases, the lung function is normal, but the blood flow to the lungs may become disrupted– meaning that oxygen cannot get into the circulation. Trauma to blood vessels in the lungs, coagulopathies, and blood clots in the lung circulation (pulmonary thromboembolism) are some of the more common reasons why this may occur.

What are the symptoms of these diseases? How do I tell them apart?

Depending on the part of the respiratory tract affected, dyspnoea, or increased breathing effort may be present primarily during inhalation, primarily during exhalation, or during both inspiration and expiration. Typically, inspiratory dyspnoea is caused by disorders of the upper respiratory tract; expiratory dyspnoea is caused by disorders of the diaphragm, chest wall, and pleural space, and the presence of both inspiratory and expiratory dyspnoea is caused by disorders of gas exchange alveoli, pulmonary parenchyma, blood vessels/interstitium etc., as well as pleural space and diaphragmatic disease; although there is some overlap.

What does the patient with dyspnoea actually look like?

Patients with breathing difficulty are frequently distressed, and usually have the following general signs…

a. Tachypnoea (rapid respiration)
b. Anxiety
c. Restlessness
d. Cyanosis, and/or pale mucous membranes
e. Mouth breathing
f. Coughing

In addition, as mentioned above, patients with breathing difficulty often have an exaggeration of inspiratory effort, expiratory effort, or both

A Note About Cyanosis?

Cyanosis refers to the presence of a bluish tinge to the mucous membranes of the mouth, conjunctival or reproductive tract. **Cyanosis is a late and unreliable sign of airway obstruction or hypoxia**. However, the presence of cyanosis is never a good sign, and demands immediate action to secure the patient airway and restore normal pleural space and ventilation capacity.

The following diagram provides a diagnostic flow of the approach to the cat in respiratory distress.

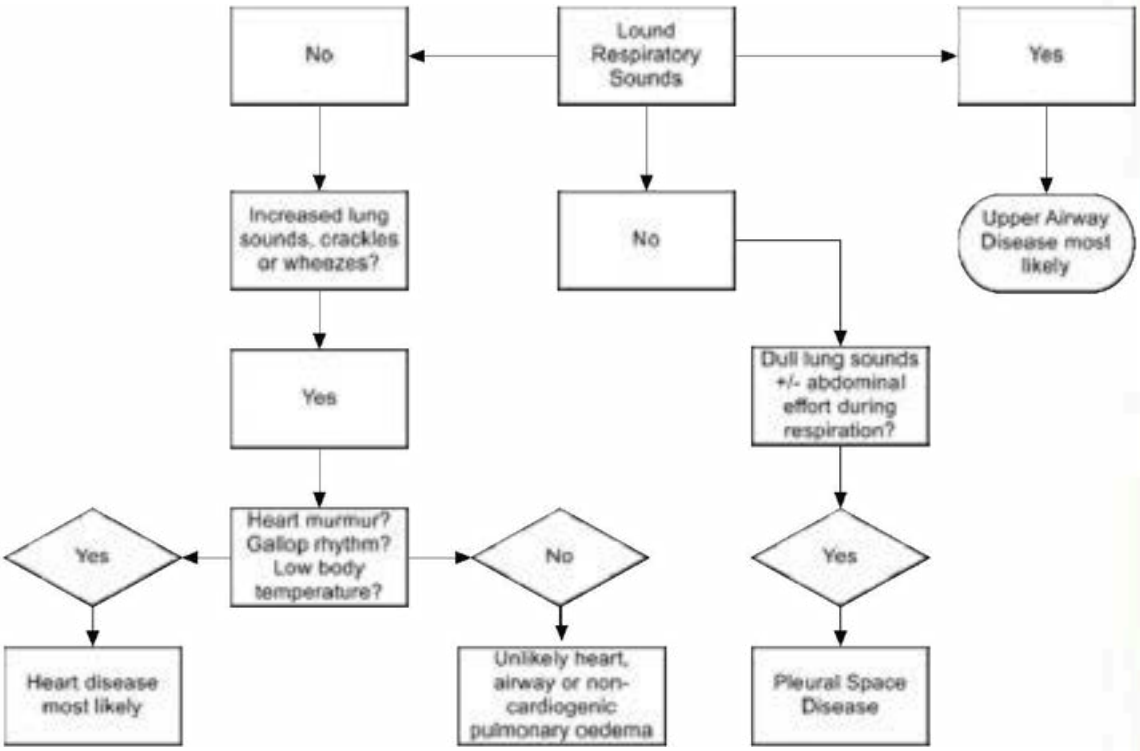


Table 1: Causes of Dyspnoea in the Cat

Inspiratory Dyspnoea	Expiratory Dyspnoea	Inspiratory and Expiratory Dyspnoea
Upper Airway Obstruction ▪ Stenotic (narrow) nares ▪ Elongated soft palate ▪ Laryngeal paralysis ▪ Everted laryngeal saccules ▪ Tracheal collapse ▪ Hypoplastic trachea ▪ Severe bronchitis ▪ Upper Airway Trauma o Pharyngeal trauma o Pharyngeal hematoma o Fractures at the base of the skull (basilar skull fractures) o Hyoid cartilage fractures o Laryngeal trauma causing bronchospasm o Tracheal trauma causing bleeding into the airways ▪ Pharyngeal oedema o secondary to trauma o insect bites or stings o swelling from increased breathing effort ▪ Laryngeal oedema – see causes of pharyngeal oedema ▪ Infection – infectious nasal cavity disease ("snuffles"), abscesses, fungal disease ▪ Neoplasia ▪ Foreign body ▪ Neurological disease – results in decreased clearance of mucus, vomit etc. from the airways. Diseases causing inspiratory and expiratory dyspnoea also cause inspiratory dyspnoea	Pulmonary Parenchymal Disease (diseases of the lower airways and lung tissue) ▪ Pneumonia o Bacterial o Protozoal (rare) o Fungal o Parasitic ▪ Neoplasia o Primary lung neoplasia o Secondary (metastatic) neoplasia ▪ Inflammatory lung disease o Feline allergic lung disease (feline asthma) o Chronic bronchitis o Allergic bronchitis ▪ Trauma to the lung – pulmonary contusions ▪ Aspiration pneumonia ▪ Pulmonary thromboembolism ▪ Pulmonary oedema o Secondary to heart disease (cardiogenic) o Secondary to neurological disease e.g. seizures, electrocution ▪ Acute respiratory distress syndrome – present in severe illness such as pancreatitis Pleural Space Diseases – as for inspiratory and expiratory dyspnoea	Pulmonary Parenchymal Disease – all diseases causing expiratory dyspnoea can cause inspiratory difficulty as well Central Nervous System Disease – can alter respiratory pattern, and respiratory drive ▪ Head trauma ▪ Seizures ▪ Drugs/medications e.g. opiates such as morphine ▪ Toxicities – e.g. tremorgenic toxins ▪ Brain tumours ▪ Meningitis Disorders of the nerve-muscle junction in the chest wall ▪ Snake bite ▪ Tick paralysis ▪ Botulism ▪ Muscle relaxants ▪ Neuro-muscular diseases Pleural Space Diseases ▪ Pneumothorax ▪ Pyothorax ▪ Chylothorax ▪ Haemothorax ▪ Pleural space neoplasia ▪ Cardiac disease ▪ Diaphragmatic hernia Rib Fractures, Flail Chest

Regardless of the diagnosis, stabilisation of the patient in respiratory distress involves the following:

1. **Assess Respiratory Rate, Pattern, and Effort**
2. **Assess and Establish Airway Patency**
3. **Assess the Evacuate the Pleural Space**
4. **Assess Effectiveness of Breathing Efforts**
5. **Provide Effective Ventilation**

Emergency Management of the Airway and Respiration

Many of the following steps in assessing the airway will have been carried out during your initial patient evaluation on arrival at the veterinary clinic (the primary survey). However, it is important to ALWAYS repeat these steps in emergency patients- sometimes as often as every few minutes- because an animals' status can change very rapidly, from being stable, to very unstable. This is because animals with trauma or life-threatening disease are often coping/surviving on their physiological reserves only – and these reserves are limited! This is particularly the case with cats. So, to begin with, we assess, and treat airway patency, starting from the tip of the nose and mouth, working our way caudally towards the chest wall and lungs. We then evaluate the pleural space, and finally, we further evaluate the lungs. The following is a step-by-step guide to the management of life-threatening conditions, and stabilization of airway disease and dyspnoea.

Always minimise stress and handling of the patient in respiratory distress, because decompensation can occur very rapidly in these patients, leading to death.

Essential Equipment to have prepared for the Patient in Respiratory Distress

Airway Equipment	Surgical Equipment	Ancillary Equipment
▪ Endotracheal tubes in all sizes ▪ Good light source – general lighting ▪ Good light source – laryngoscopes ▪ Dog urinary catheters x 4 ▪ Tracheostomy tubes sizes 3, 5, 8, and 10 ▪ Lignocaine spray or drops	Small surgical kit ▪ Mayo scissors ▪ Haemostats – curved and straight ▪ Rat tooth forceps ▪ Needle holders ▪ Scalpel blades – sizes 10 and 11 ▪ Drapes ▪ Lap sponges or swabs Chest Drains – sizes 8, 10, 14, and 16 Fr x 2 of each	▪ Oxygen supply - Flow-past oxygen supply or Anaesthetic machine ▪ Ambu-bags ▪ Umbilical tape ▪ Pulse oximeter ▪ Capnograph ▪ ECG ▪ Butterfly needles 22 G, and 20 G x 5 of each ▪ 3-way stopcocks ▪ Extension tubing ▪ Syringes 10 ml and 20 ml x 5 of each ▪ Clippers ▪ Needles – 18 G x 6

1. Assess Respiratory Rate, Pattern and Effort

It is important not to rush towards an animal in respiratory distress, and begin handling them without first having an idea of what they have wrong with them - even though our instincts tell us to immediately begin resuscitation. Why? If we have an idea of what is causing the patients' breathing difficulty, we can be much more efficient at going about fixing their problem.

Provide supplemental oxygen therapy at all times while evaluating respiratory function, until it is confirmed that the patient does not require supplemental oxygen

Allow the cat time (10 minutes or so) to recover from the stress of transport to the vet clinic, in a low-stress environment with oxygen supplementation. Typically, an oxygen cage is used, although fly-by oxygen, nasal cannula or oxygen hood could be considered if the patient will tolerate it.

Be prepared to intervene quickly (intubate, thoracocentesis, positive pressure ventilation) if respiratory distress worsens.

Many cats will benefit from mild sedation whilst they recover from stress. Butorphanol 0.1 mg/kg may be administered intra-muscularly in these patients to help achieve this. Although intravenous administration is associated with more rapid action, and more reliable sedation.

Assess respiratory rate and pattern

a. Observe the rate and pattern from a distance
b. Determine if the patient is breathing
c. Determine if the breathing rate is increased, decreased or normal
d. Determine the breathing pattern
    i. Normal
    ii. Shallow and rapid
    iii. Slow and deep
    iv. Determine if there is difficulty in breathing during inspiration or difficulty in expiration or both?
e. Assess chest wall movement – Observe closely for any increase in respiratory effort. *Helpful hint – look at your own pets' chest wall movement during breathing. Look at animals presenting for vaccination – how much does the chest wall move? Make a conscious effort to observe each animal that comes into your practice. This will make it easier for you to detect abnormal chest wall movement.*
f. Is there an abdominal component to the breathing? Abdominal movement during inspiration may indicate upper airway obstruction caused by any of the diseases listed in Table 1. Excessive abdominal movement during exhalation usually indicates diseases of the diaphragm, thoracic wall, pleural space, or lung parenchyma.
g. Auscultate the lungs bilaterally – start dorsally, then move ventrally. ALWAYS listen to both side of the chest. Auscultate the trachea. Respiratory sounds that are louder in the trachea than the lung generally come from larger airways. Respiratory soundsthat are softer in the trachea than the lung generally come from the lung/pulmonary parenchyma.
h. Exaggerated chest and/or abdominal expansions that do not result in air movement indicate total airway obstruction.
i. **NO chest movement or expansion supports a diagnosis of respiratory arrest** – these patients require immediate intubation and ventilatory assistance to prevent death.
Have a laryngoscope, lignocaine spray or injection, good light source, ties, and several endotracheal tubes prepared in case intubation is required

2. Assess and Establish Airway Patency

a. Provide supplemental oxygen therapy by flow-past oxygen (via an oxygen hose, or anaesthetic circuit. In most instances, a flow rate of between 1-3 litres per minute is sufficient for oxygen flow past the patients' mouth or nose. If the animal is not tolerant of oxygen flow, reduce the flow rate until the animal no longer resents the flow of oxygen.
b. Gently extend the animal's head and neck. Open the animals mouth, and pull the tongue gently forward (if this can safely be done without trauma or stress), and make sure the tongue, pharynx, and larynx is clear of mucus or debris. If the larynx is not clear, either remove mucus using gauze swabs (particular care needs to be taken in animals that have suffered head trauma, as they may have skull fractures that may lacerate blood vessels if moved) or a soft suction hose or hand-held medical sputum suction pump. Use of a laryngoscope or strong light source is extremely helpful when doing this, and may reduce the risk of damaging pharyngeal and laryngeal structures
c. Check the nose: put a microscope slide near nose to check for condensation to indicate airway movement in and out of the nostrils. Gently clear excessive blood or mucus about the external nares, if this is interfering with respiration. Removal of blood clots from the nose may initiate

more bleeding – if the animal is breathing adequately, it is best not to remove blood clots until the patient has been assessed for coagulopathy, and is otherwise stable.

d. If patient is not trying to breathe, or has an obstructed airway, they will need to be intubated. Once the patient is intubated, provide oxygen supplementation, using an anaesthetic machine or other oxygen source. Depending on the patients breathing effort, positive pressure ventilation may be required (see later under "Provide Effective Respiration").

e. If the patient cannot be intubated due to airway blockage, or airway swelling, the patient may need an emergency tracheotomy to obtain airway patency. Whilst the most dramatic action seen to establish a patent airway is the "slash" tracheotomy, it is rarely required. There are some other things we can do with our patient before we resort to this procedure. They are

i. Insert an 18 G over-the-needle catheter or angiocath between the tracheal rings or through the cricothyroid membrane (at the caudal end of the laryngeal cartilages) and connect to an oxygen supply. The small amount of oxygen delivered will assist in reducing hypoxia, and will make any subsequent attempts to establish a patent airway via tracheotomy under sedation or light anaesthesia much safer for the patient

ii. Use a 4-6Fr dog urinary catheter as a stylette via the oral cavity to assist in guiding an endotracheal tube into the trachea

In most instances, one or other of these procedures will allow short-term delivery of oxygen to the patient in order to stabilise them prior to a planned tracheotomy procedure. The procedure for such a planned tracheotomy is described below…

Tracheotomy Procedure
1. Make a ventral midline incision from the manubrium of the sternum to the laryngeal cartilages **2. Part the sternohyoideus muscles on the midline by blunt dissection** **3. Continue blunt dissection down to the tracheal rings** **4. Incise the trachea at the level of tracheal ring 3-5 distal to the thyroid cartilages in one of 2 ways...** **A. Make an "H" incision or...** **B. Make a transverse incision between 2 tracheal rings** **5. Place stay sutures through the tracheal rings either side of the tracheal incision** **6. Insert the tracheostomy tube (size 3 or 4). The tracheostomy tube should be 2/3-3/4 of the diameter of the trachea, and have a low pressure/high volume cuff. Note: only inflate the cuff if positive pressure ventilation is required, or if it is necessary to prevent aspiration or oropharyngeal contents.** **7. Secure the tube to the patient by typing it around the patient's neck so that it sits snuggly against the stoma and skin.**

3. Assess and Evacuate the Pleural Space

a. Many emergency patients will have an abnormal pleural space. The pleural space is the space between the chest wall, and the outer surface of the lungs. In the normal cat, this space is filled with a small amount of cohesive fluid that allows the lungs to slide back and forth over the chest wall during normal breathing, while keeping the lungs in close proximity to the chest wall, preventing them from collapsing. When animals are traumatized or diseased, the pleural space can become filled with air, blood, or fluid. When this happens, the lungs lose their proximity to the chest wall, and collapse. This results in breathing difficulty, respiratory distress, and exaggerated breathing efforts. This is a serious condition, which can result in rapid patient deterioration and death if left untreated.

b. **Assess the pleural space**
   i. Palpate the chest for rib fractures, intercostal tears, flail chest segments, subcutaneous emphysema
   ii. Auscultation - listen over **entire** thorax. On auscultation, stertor (low frequency snoring sounds) indicates respiration that has an increased effort, Stridor (high frequency squeaking sounds) which may suggest upper respiratory dysfunction. Crackles, Wheezes or broncho-vesicular (lower airway) sounds may indicate lower respiratory dysfunction. Absence of lung sounds or dull sounds could be consistent with air/gas, pus, or blood in the pleural spaces or the lung parenchyma (alveoli and small airways)
   iii. Diagnostic ultrasound – placing an abdominal ultrasound probe to view the lung fields in the intercostal spaces can enable the diagnosis of pneumothorax, pleural fluid, and peripheral pulmonary congestion (due to pneumonia, pulmonary oedema etc.) or the presence of pulmonary masses. The technique for this procedure is termed Vet BLUE (Veterinary bedside lung ultrasound evaluation), and is an easy-to-learn, non-invasive, and low stress diagnostic test, with superior sensitivity to emergency radiographs

c. **Perform thoracocentesis -** In most animals showing clinical signs of restrictive breathing, having reduced lung sounds, with confirmed pleural space accumulations of fluid or air, as determined by Vet BLUE ultrasound, we advise performing what is called a "**diagnostic/therapeutic thoracocentesis**", where a needle is passed into the chest cavity in an attempt to determine if there is any abnormal fluid or air accumulation causing lung collapse. A respiratory rate of 45-60 breaths per minute, or the presence of clinical signs such as inspiratory and/or expiratory dyspnoea, and abdominal efforts during breathing indicates thoracocentesis may be required. In addition, decreased lung sounds on thoracic auscultation may also suggest pleural space disease, and warrants evaluation with diagnostic thoracocentesis. When performing thoracocentesis, be prepared to evacuate the entire pleural space at the first attempt thoracocentesis**. ALWAYS ATTEMPT TO DRAIN BOTH SIDES** of the chest cavity. Take the time to review the procedure below, and take note of the equipment required…

Thoracocentesis – The Procedure

- Prepare a 22-23G butterfly catheter; attach it to a short IV extension tube, a 3 way stopcock, and a 10-20 ml syringe.
- Clip an area of 5 cm X 5 cm **on BOTH sides of the chest**, from the 4 th to the 8th rib, and swab with chlorhexidine surgical scrub and alcohol.
- Insert the butterfly catheter into the chest at the 6-7th intercostal space, and have an assistant draw back on the syringe. The syringe is repeatedly filled and emptied while the butterfly catheter is in the chest cavity until there is no more air or fluid present in the pleural space

4. Establish Effectiveness of Breathing Efforts

a. Provide supplemental oxygen at all times during the initial patient assessment

b. Airway equipment such as laryngoscope, endotracheal tubes of the appropriate size for the animal, cuff inflating syringe and ambu-bag should be ready.

c. Clear mucous, blood and debris from the oral and pharyngeal/laryngeal cavity as previously described.

d. Assess Effectiveness of Breathing Efforts – this is a broad statement, and we all know that animals need to breathe to survive. But how do we know if an animal is breathing *enough*? The answer lies in looking at the patient, and taking a few measurements. We will discuss these now

i. Look at the patient – evaluate respiratory effort. If there is increased effort, and breathing difficulty, the patient probably requires some assistance with their breathing using oxygen supplementation and maybe manual assistance during inspiration using an ambu-bag or anaesthetic re-breathing bag. In fact, animals with exaggerated breathing are frequently unable to supply their intercostal muscles with enough oxygen to allow them to keep working, which leads to lactic acidosis and decreased efficiency of muscle activity. The result? Patients with exaggerated respiratory efforts frequently suffer from respiratory muscle fatigue VERY early in their disease, which can lead to hypoventilation.

ii. Pulse oximetry – Pulse oximetry measures the precent saturation of blood haemoglobin with oxygen. It gives a rough guide or assessment of the ability of the lung to deliver oxygen to the blood so that it can oxygenate haemoglobin. Pulse oximetry, however, does not give an indication of tissue perfusion, tissue oxygenation, or tissue oxygen delivery. Patients not breathing adequately, however, will frequently have low pulse oximetry readings. It is important to note that haemoglobin saturation assessment using pulse oximetry can be misleading. Table 3 may be used as a guide to determine when ventilatory assistance is required. We consider providing ventilation assistance in any patient that is receiving oxygen supplementation that has a reliable pulse oximetry reading of less than 94%. A reliable reading from a pulse oximeter is one where the pulse-wave on the pulse oximeter is strong, the heart rate on the pulse oximeter is the same as the femoral pulse rate, and there is a steady number on the display.

Table 3: Interpretation of Pulse Oximetry Readings

SaO_2	PaO_2	Interpretations
>95%	>80%	Normal
<89%	<60%	Serious Hypoxaemia
<75%	<40%	Lethal Hypoxaemia

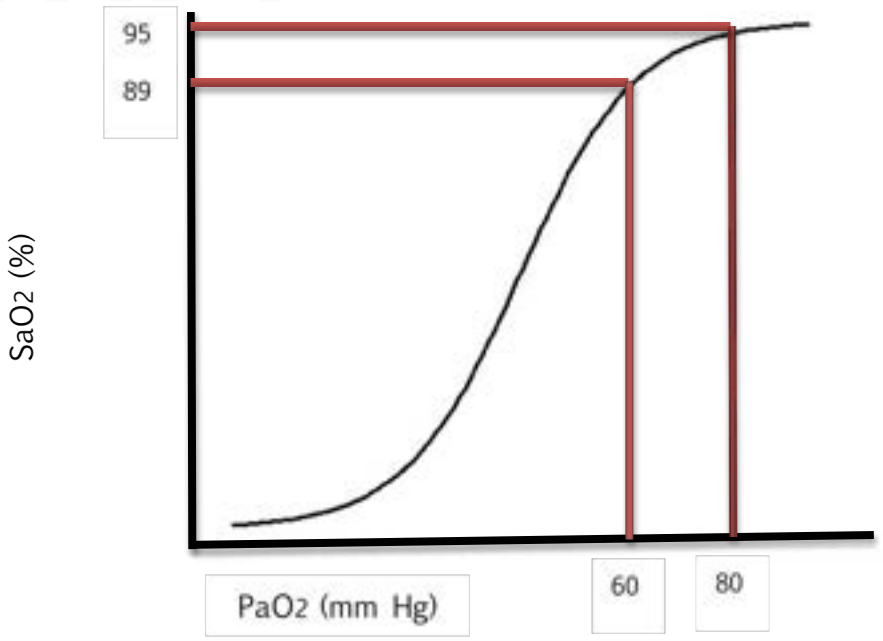


Chart showing the relationship between oxygen saturation (SaO_2) and blood oxygen pressure (PaO_2). Note that at a pulse oximetry reading of 90%, arterial oxygen content indicates serious, life-threatening hypoxaemia.

iii. Capnography – if the patient has been intubated, it is possible to measure end-tidal carbon dioxide levels. End-tidal carbon dioxide is the concentration of carbon dioxide in the patients' breath at the end of expiration. End-tidal carbon dioxide concentration gives an estimate of the effectiveness of breathing efforts. Any decrease in respiratory effort, or any interference with breathing efforts or gas exchange in the lungs may lead to a higher than normal end-tidal carbon dioxide level. The normal end-tidal carbon dioxide level is between 35 and 45 mm Hg. An end-tidal carbon dioxide level of greater than 55 mm Hg provides strong evidence that the patient has inadequate breathing efforts. Patients with elevated carbon dioxide levels require assistance with their breathing, and provision of positive pressure ventilation with either using an ambu-bag, an anaesthetic re-breathing bag or mechanical ventilator. The aim of ventilatory assistance is to 'augment' or 'slightly increase' the depth of the patients' breathing effort, and to maintain a respiratory rate of approximately 12-24 breaths per minute (one breath every 3-4 seconds), with a tidal volume of approximately 8-12 ml/kg

iv. Blood gas analysis – Blood gas analysis is a useful means of assessing pulmonary function, the adequacy of gas exchange and the adequacy of circulatory support, and can assist in providing a guide of when to initiate ventilation support, using PO_2, PCO_2, and other parameters.

Patients with abnormal levels of consciousness i.e. they are depressed, comatose, or semi-comatose, or seizuring – often require assistance with their breathing. These patients often develop airway obstruction due to accumulation of saliva, vomitus, and other debris in their larynx and airways unless you protect the patient from inhaling this material. These patients frequently require intubation with an endotracheal tube. Once the patient is intubated, they will require supplemental oxygen via anaesthetic machine circuit. Depending on their breathing effort, they may also require manual or mechanical ventilation.

Encourage all staff members to practice intubating any dead or euthanized animal that is present in your clinic, both in sternal, lateral and in dorsal recumbency to gain a good skill level and technique.

e. If the patient is not ventilating adequately, sedation and/or light anaesthesia should be administered, and ventilation assistance (manual or with a ventilator) should be provided. The manual rate of ventilation in emergency patients should be 12 -24 breaths/ minute, at an initial tidal volume of 8-12 ml/kg.

A word about sedation in patients with breathing difficulty Patients with airway obstruction are hypoxic and hypoxaemic and therefore are EXTREMELY sensitive to the effects of anaesthetic and sedative agents frequently used in veterinary medicine. In general, the safest anaesthetic to use in the emergency is the anaesthetic with which you are most familiar and comfortable with. However, some anaesthetics are safer than others are. The authors' preference is to sedate any patient in which intubation cannot be achieved without chemical restraint, using an intravenous bolus of diazepam at 0.1- 0.3 mg/kg. If diazepam alone is insufficient to allow endotracheal intubation, addition of ketamine to effect (1-5 mg/kg i/v) or fentanyl (1-4 micrograms/kg i/v), or butorphanol (0.1 mg/kg) are preferred agents.

NEVER administer sedatives or anaesthetic drugs to a patient with respiratory distress without assistance and a full crash-cart on-hand.

The reason for this statement is that these patients are VERY susceptible to lethal hypoxia and death during handling and sedation. The author recommends that a minimum of 2 (preferably 3) people be present when sedating a patient in respiratory distress, in order to assist with CPR if required.

It is important to understand that there are particular disease conditions increase the likelihood for the requirement of ventilation support. The following table (Table 4) lists some of the more common conditions that may result in the need for providing ventilation assistance. If you have a patient in your hospital with one of the following conditions intensive monitoring is advised, frequently assessing the patients breathing efforts, heart rate, mucous membrane colour, pulse oximetry, capnography and blood gases to determine if the patient requires ventilation assistance. Remember to alert staff members monitoring the patient and encourage communication if they have ANY doubt about a patient's ability to breathe normally so that the team can intervene.

Table 4: Conditions that May Require Provision of Positive Pressure Ventilation (PPV)

Disorders of the Neuromuscular Junction	Pulmonary Parenchymal Disease
1. Tick Paralysis 2. Elapid Snake Envenomation 3. Botulism 4. Polyradiculoneuropathy 5. Myasthenia Gravis 6. Muscle relaxant administration 7. Tetanus	1. Pneumonia 2. Neoplasia 3. Pulmonary oedema 4. Pulmonary interstitial disease 5. Pulmonary contusions 6. Congestive heart failure
Central Nervous System Disease	**Hypoventilation**
1. CNS disease causing depression of respiratory drive a. Head trauma b. Neoplasia c. Drugs/medications d. Toxicity e. Seizures f. Infection/inflammation g. Cerebral oedema, increased intracranial pressure	1. Shock a. Hypovolemic shock b. Haemorrhagic shock c. Septic shock d. Cardiogenic shock e. Non-cardiogenic shock 2. Pleural space disease 3. Sepsis 4. Mediastinal disease 5. Pain

Radiographic Imaging in the Cat with Respiratory Distress

When presented with a cat in respiratory distress, it is sometimes tempting to attempt to radiograph the patient early, in an attempt to gain more information about what is going on. So why have we not advocated radiographs in any of the previous pages of notes on respiratory emergency management? I am sure many will have experienced times when we have radiographed a cat having breathing difficulty, struggled with the patient on the x-ray table, seen the patient go cyanotic, been bitten by the patient, or even had the patient die, or come close to it – all for a radiograph – often a single view, that does not give us all the information we need! So why stress the patient, or even kill the patient, for an incomplete diagnostic test? The answer – DON'T!

Radiographs are, however, an important diagnostic tool in evaluating respiratory disease. So, when is the right time to radiograph the patient in respiratory distress? In general, the best time to radiograph a patient with respiratory disease, is when they are not distressed – that is, it is extremely important to stabilize the patient as much as possible by

- Providing supplemental oxygen therapy
- Securing a patent airway
- Evaluating, and evacuating the pleural space in patients with pleural fluid or pneumothorax
- Providing ventilatory assistance for patients with inadequate respiratory effort, or with pulmonary parenchymal disease e.g. pneumonia, pulmonary oedema, pulmonary contusions et.

When taking radiographs, be prepared to take 3 or 4 views –a left lateral, right lateral, and a v/d +/- d/v view. Also be prepared to stop the radiograph session if the patient becomes distressed, and allow the patients to stabilize before proceeding with the remainder of the radiographic examination.

VetBLUE Lung Ultrasound and TFAST Ultrasound

Trans-thoracic ultrasound techniques have been developed that can provide the emergency clinician with essential information on which to be able to base clinical decisions on when to perform diagnostic thoracocentesis, to help enable differentiation of conditions with similar presenting signs, such as heart failure and feline asthma among other things. The presence of B lines has high sensitivity and specificity for alveolar and alveolar interstitial lung disease (cardiac or non-cardiac). In addition, the presence of an enlarged left atrium: aorta ratio, with B lines on lung ultrasound is highly sensitive and specific for cardiac disease, whereas absence of both B-line and a normal LA: Ao ratio is highly sensitive for ruling out cardiac disease as a cause of respiratory distress in the cat.

The Extended Database

Other diagnostic steps that may allow your team to establish a diagnosis include

1. Blood tests
    a. Obtain a PCV/TP, glucose, and lactate level from the blood you collect from the hub of the intravenous catheter you place in the patient following initial stabilization
    b. Obtain blood for biochemistry, CBC, and blood gas (if available) once the patient is stable
    c. Obtain blood for a clotting assessment (activated clotting time – ACT) from a peripheral vein once the patient is stable
    d. Feline leukaemia and immunodeficiency virus tests.
    e. Obtain urinalysis and/or blood for snake venom detection analysis
    f. NT-Pro BNP is highly sensitive for the detection of cardiac disease in the cat (95-100%), and a normal value tends to rule out cardiac disease as a cause of respiratory distress in most cats. Some cats with cardiac disease, however, may have a normal NT Pro-BNP result, and should have further diagnostics, including cardiac ultrasound to confirm the presence or absence of heart disease.
2. Fluid analysis – keep a sterile sample of any fluid obtained from the pleural space for analysis by your laboratory, including culture and sensitivity and cytology. In addition, any non-sterile fluid from the trachea may be kept and examined by cytology
3. Cardiac ultrasonography, pulmonary/lung aspirates
4. Further diagnostic tests are reserved until the patient is breathing well, and is haemodynamically stable, and include tracheal washes, broncho-alveolar washes, antibody tests, etc.

Summary

When faced with an animal with breathing difficulty, remember to do the following things

1. Be prepared – make sure you have airway management equipment ready for the patient
2. Look at the patient –try to work out where their breathing difficulty may lie within the respiratory tract
3. Be methodical
    a. Provide oxygen supplementation at all times until you prove the patient does not need it
    b. Assess and secure the presence of a patent airway
    c. Evaluate the pleural space, and drain the pleural space if necessary, or if clinical signs of pleural space disease are present, prior to radiography
    d. Check adequacy of breathing efforts – provide breathing assistance for the patient if necessary
4. Monitor the patient frequently – patients with respiratory distress are some of the most critical patients you will see and deal with. They need monitoring every few minutes during the stabilization period, and every 15-30 minutes for several hours, once they are stable.

References:

1. Dickson D, Little CJ, Harris J, Rishniw M. Rapid assessment with physical examination in dyspnoeic cats: the RAPID CAT study. journal of small animal practice. 2018 Feb;59(2):75-84.
2. Janson CO, Hezzell MJ, Oyama MA, Harries B, Drobatz KJ, Reineke EL. Focused cardiac ultrasound and point-of-care NT-ProBNP assay in the emergency room for differentiation of cardiac and noncardiac causes of respiratory distress in cats. Journal of Veterinary Emergency and Critical Care. 2020 Jul;30(4):376-83.
3. O'Byrne L, Cole L. Cats are not small dogs: assessment and stabilisation of emergency presentation. Companion Animal. 2024 Nov 2;29(11):2-6.
4. Johnson LR. Canine and feline respiratory medicine. John Wiley & Sons; 2024 Nov 4.
5. Levy N, Ballegeer E, Koenigshof A. Clinical and radiographic findings in cats with aspiration pneumonia: retrospective evaluation of 28 cases. Journal of Small Animal Practice. 2019 Jun;60(6):356-60.
6. Ward JL, Lisciandro GR, Ware WA, Viall AK, Aona BD, Kurtz KA, Reina-Doreste Y, DeFrancesco TC. Evaluation of point-of-care thoracic ultrasound and NT-ProBNP for the diagnosis of congestive heart failure in cats with respiratory distress. Journal of veterinary internal medicine. 2018 Sep;32(5):1530-40.
7. Tong CW, Gonzalez AL. Respiratory Emergencies. Veterinary Clinics: Small Animal Practice. 2020 Nov 1;50(6):1237-59.
8. Chalifoux NV, Drobatz KJ, Reineke EL. Predictors of inflammatory lower airway disease in cats presented to the emergency room in respiratory distress: a case-control study. Journal of Feline Medicine and Surgery. 2021 Dec;23(12):1098-108.
9. Drobatz KJ. Approach to the critically ill cat. Feline emergency and critical care medicine. 2022 Oct 7:1-8.

ADVANCE
VETERINARY DIETS

DISCOVER ADVANCED VETERINARY NUTRITION

CLINICALLY PROVEN

scientific remedies

Marketed by :
Scientific Remedies Pvt. Ltd.
1, Cosmic Enclave, Opp. SBI Bank, Sama, Vadodara (Gujarat), India.
customercare@srplmail.com
www.scientificremediesindia.com

For product related queries, please contact :
Contact No. : 1800 2585 787 | **Email :** customercare@srplmail.com

CLINICALLY PROVEN
Real results supported by research.

HIGH QUALITY RECIPES
High palatability and nutritional excellence for long-term daily feeding.

NO PRESERVATIVES OR ARTIFICIAL COLOURANTS
With natural antioxidants and no artificial additives.

EXPERT NUTRITION
Over 25 years of research and development of veterinary solutions.

Mankind's
PetStar
V Diet

LAUNCHED

MADE IN

Pet Mankind

Finding Pain Relief.. is a Joint Effort

Coxkind®-57/227 Tablets

Nutrition for every **Pet's unique needs**

WELLCON Animal Health

Canine Product Range

APTIFAST PET

- Appetite stimulant
- Mood enhancer
- Hepatoprotective

CANISHINE

- Enriched with Omega 3,6,9 fatty acid
- Maintain skin shine & lusturous coat

SMARTDOG©

Presents

- Packed with flavours highly palatable

PHCAN Dry Bath Shampoo

- Groom & Shine your companion
- Ideal for cold conditions

Contact us : +91 7055546444 customercare@wellcon.co.in

VET EXPERT

Imported and Marketed By:

Vet Planet India Private Limited

Ph: 9711140505, 7827836303

vet planet

Nutritional solutions for specific needs.

Farmina®
Pet Foods

Happy pet. Happy you.

www.Farmina.com/in/

BRAVECTO®

3 THREE-MONTH PROTECTION

TESTED AND TRUSTED AROUND THE WORLD

with 30 Crore doses distributed in 90 countries

PROVEN SAFETY BACKED BY 170 CLINICAL STUDIES & 80+ PUBLISHED PAPERS

3 Month Protection

Against Ticks, Fleas & Mites in One Chew

Intervet India Pvt. Ltd.,
6th Floor, Tower 5, World Trade Center, Survey No 1, Kharadi, Pune – 411014, Maharashtra, India
Ph. No. +91 20 66294700/01 | Email: infoin-india@merck.com | www.msd-animal-health.co.in

MSD Animal Health

FlexiRun™

ADVANCED HIP & JOINT FORMULA

FlexiRun is a scientifically researched and clinically proven joint health supplement.

Formulated with high-quality ingredients, Glucosamine HCl (purity>99%) Chondroitin Sulphate (100% purity).

Proven results can seen within 4-6 weeks of daily use.

Mfg & Marketed by
SKYEC
Committed to Quality

SkyEC Drugs and Pharmaceuticals Pvt. Ltd.
RAM- L1, Industrial Estate, Guindy, Chennai - 600032.
Customer Care No: 044-42700919
Email:contact@skyecpharma.com | www.skyecpharma.com

VET EXPERT

BASED ON EVIDENCE

Imported and Marketed By:

Vet Planet India Private Limited

Ph: 9711140505, 7827836303

PURINA®

PRO PLAN®

NUTRITION THAT PERFORMS®

SUPPORT YOUR PUPPY'S IMMUNITY FOR A **GREAT START IN LIFE**

COLOSTRUM

To help maintain immunity

DHA

Nourish brain and vision development

Gastrointestinal health

Refer pack for details

Easy rehydration of kibble for weaning

Learn more at purina.in/brands/pro-plan | Refer to pack for details

PURINA Your Pet, Our Passion.

Nestlé
Good food, Good life

Keep System HEALTHY with VETRICARE System Care Range

Vetrina®
Animal Nutrition & Health

With best
compliments from:

Boehringer
Ingelheim

Vet care +

Complementary feed

CARDIO FORCE

Heart Support

Creatine | L-arginine | Taurine | L-carnitine

Vetfood PROFESSIONAL

30 capsules

Vet care +

Complementary feed

BRAINACTIV BALANCE

brain health in pet animals

Vetfood PROFESSIONAL

30 capsules

Vet care +

Complementary feed

DERMACTIV

Skin Function Support

Vetfood PROFESSIONAL

30 capsules

Vet care +

Complementary feed

AMYLACTIV MAX

Highest Concentration of Digestive Enzymes

Vetfood PROFESSIONAL

30 capsules

APOCARE

Vet care +

Complementary feed

AMYLACTIV DIGEST

Digestive Enzymes

Vetfood PROFESSIONAL

30 capsules

CMI+

Protection & Defence for your Pet

PARVO

HEPATITIS

DISTEMPER

KENNEL COUGH

Imported & Marketed By:

Navyaa Agencies

Mobile: 9711140505, 7827836303

E-mail: rishi14sood@yahoo.co.in

Dr. Chad Schmiedt

Small Animal Medicine & Surgery,
Soft Tissue Surgery Service,
Veterinary Teaching Hospital
Professor Alison Bradbury Chair in Feline Health

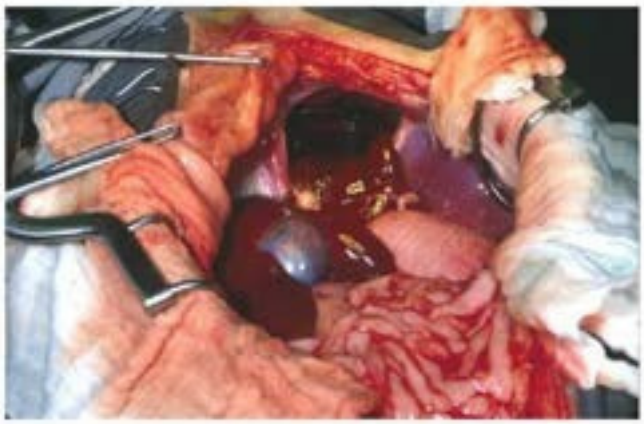
Dr. Chad Schmiedt received his DVM from the University of Georgia, College of Veterinary Medicine in 2000. Following five years of post-graduate surgical training at the University of Tennessee, the Dallas Veterinary Surgical Center, and the University of Wisconsin-Madisonand a two year clinical instructorship at the University of Wisconsin-Madison, in the summer of 2007 Dr. Schmiedt returned to UGA to join the faculty at the College of Veterinary Medicine as a soft tissue surgeon. Dr. Schmiedt is board-certified by the American College of Veterinary Surgeons and holds the Alison Bradbury endowed chair within the Department of Small Animal Medicine and Surgery. He is the section head of the small animal medicine and surgery section and also runs the feline renal transplantation program at UGA. Dr. Schmiedt commonly sees referral and emergency clinical cases on the soft tissue surgery service at the Veterinary Teaching Hospital and runs an active research program focusing on kidney disease in cats.

Diaphragmatic Hernia Repair in Dogs and Cats

Chad Schmiedt DVM, DACVS-SA
Professor, Small Animal Surgery
Alison Bradbury Chair of Feline Health
University of Georgia

Agenda

- Types of hernias
- Diaphragmatic anatomy
- Stabilization
- Anesthetic considerations
- Repair technique
- Post operative care

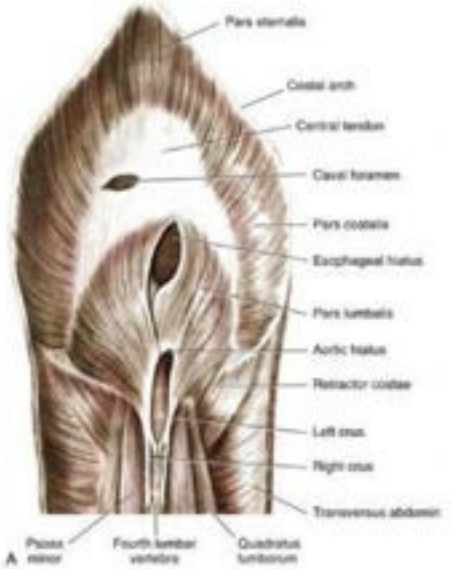


Diaphragmatic Hernia

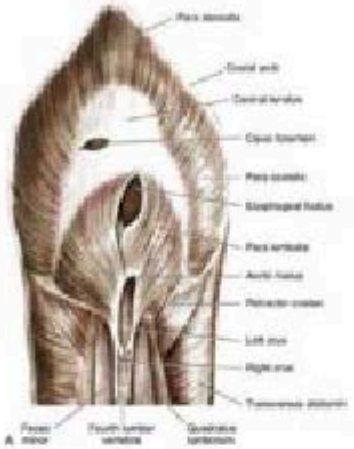
- Traumatic
- Congenital –
  - Pleuroperitoneal -
  - Periteoneal pericardial diaphragmatic hernias (PPDH)
- Hiatal

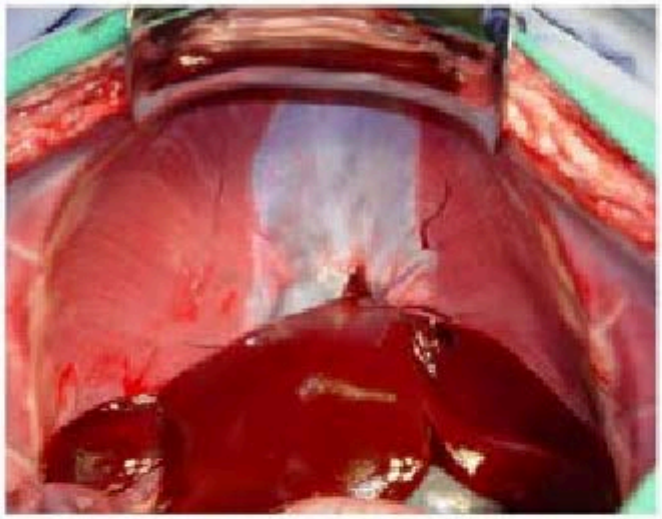
Diaphragm Anatomy

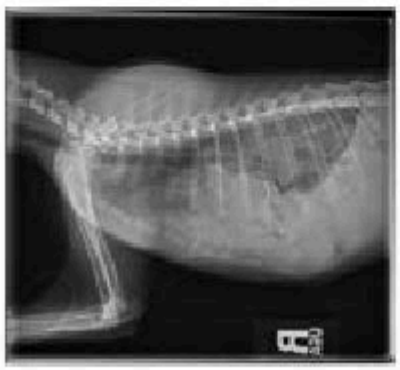
- Central tendon
- Pars costalis, sternalis, lumbalis
- 3 holes:
  - Caval foramen
  - Esophageal hiatus
  - Aortic hiatus
- Local helpful muscles:
  - Transversus abdominus
  - Rectus abdominus

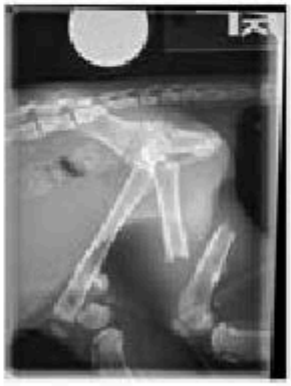


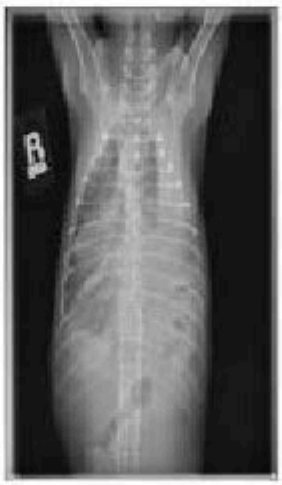
Diaphragm Anatomy











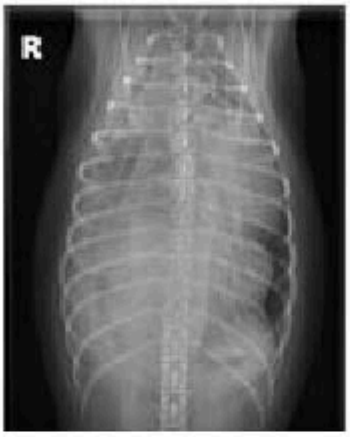
Traumatic Diaphragmatic Hernia

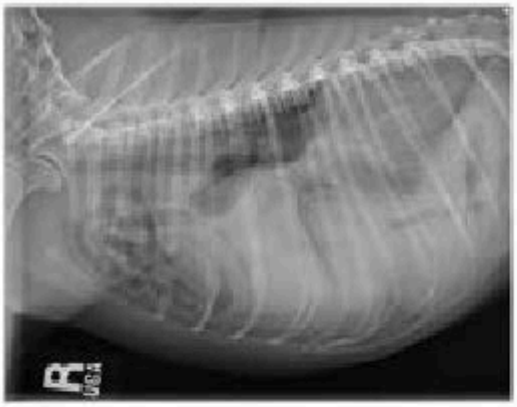
Physical Exam and Diagnosis

- Dyspnea of varying degrees (38% of patients)
- Tucked up abdomen – wasp waist
- Muffled heart or lung sounds
- Unilaterally increased heart sounds (opposite hernia)
- Borborygmi in the thorax
- Orthopnea
- Sitting up
- Gastrointestinal signs (vomiting, dysphagia, diarrhea, constipation)

- Asymptomatic

Radiographic Diagnosis



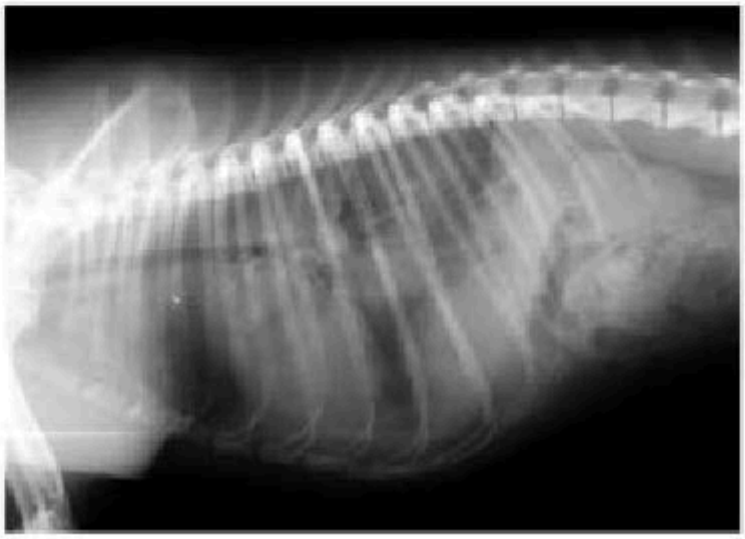


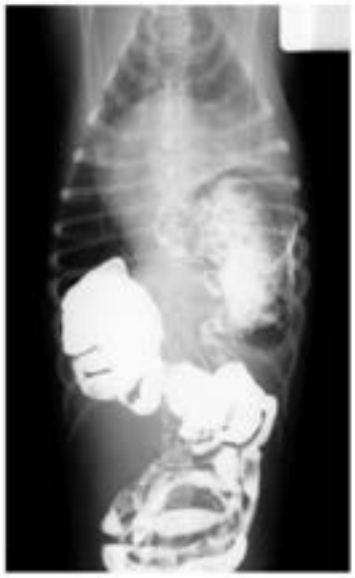
Thoracic radiographs are most useful

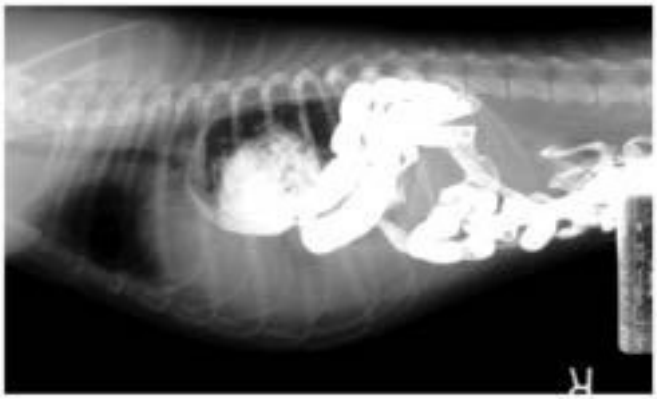
Loss of diaphragmatic line on lateral projection in 66 – 97%

Viscera in thorax, obscured cardiac shadow, pleural effusion

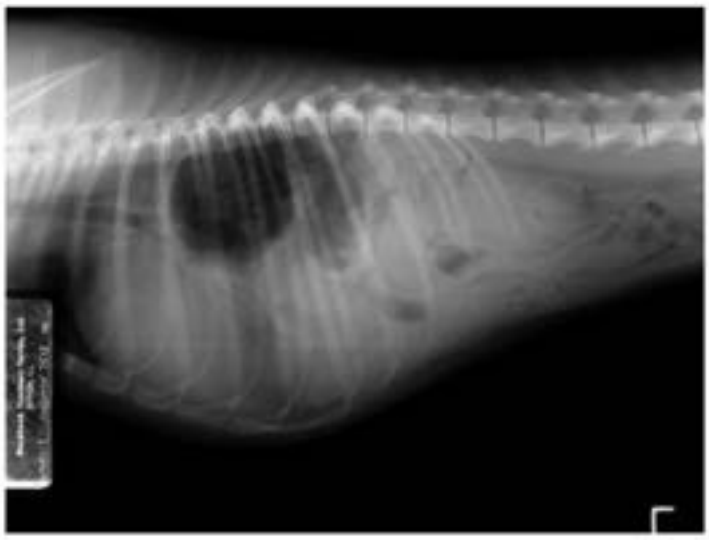
Oral contrast may aid in diagnosis





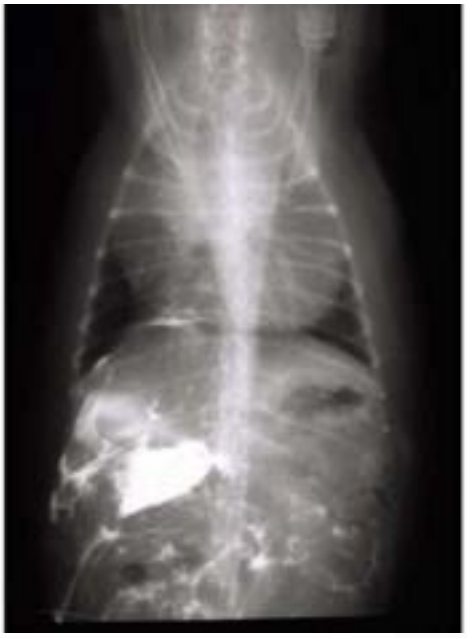


Opposite lateral may also help



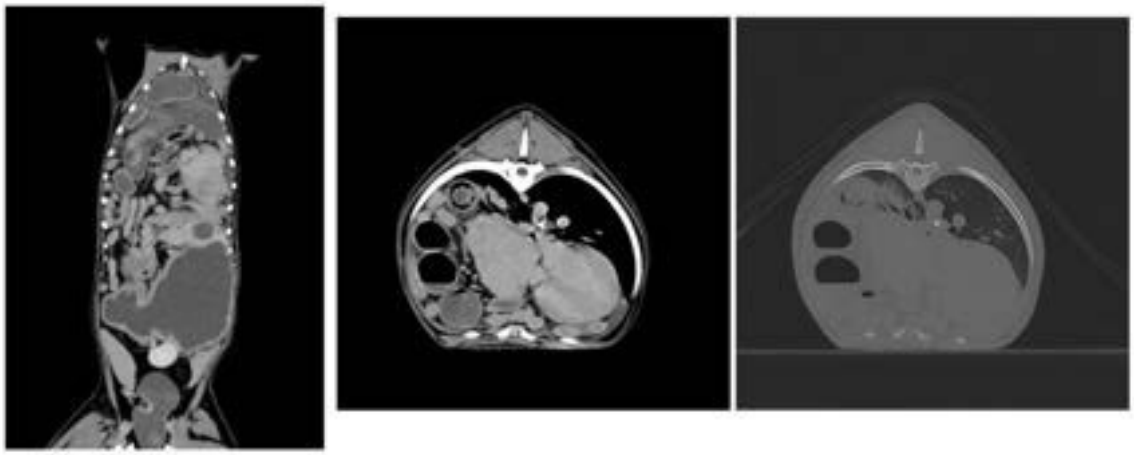
Positive contrast peritoneography

Potential for false negatives (low sensitivity)



CT scan helpful for difficult cases

4 yr, FS, MBD
Chronic DH
History of corn cob ingestion



335941

Timing of Surgery: Acute vs. Chronic Hernias

- Acute hernias –
  - Traumatic injury may have other systemic consequences
  - Bullae, shock, pneumothorax, traumatic myocarditis, hemothorax

- Chronic hernias -
  - Pleural effusion
  - Adhesions
  - Loss of abdominal domain

Survival of Acute vs. Chronic Hernias

Pathophysiology of Traumatic Diaphragmatic Hernia in Dogs

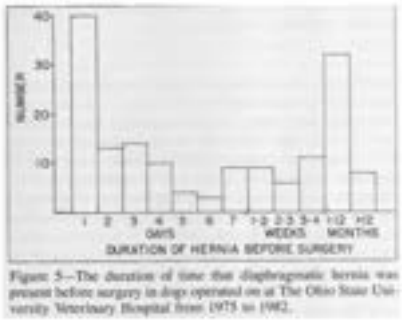


Figure 5—The duration of time that diaphragmatic hernia was present before surgery in dogs operated on at The Ohio State University Veterinary Hospital from 1975 to 1982.

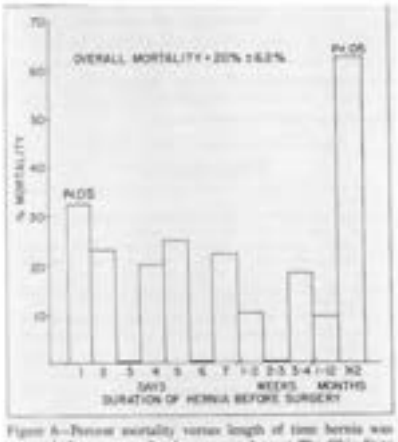


Figure 6—Percent mortality versus length of time hernia was present before surgery for dogs operated on at The Ohio State University Veterinary Hospital from 1975 to 1982.

Comp Small Animal, 1987

Survival of Acute vs. Chronic

Prognostic indicators for perioperative survival after diaphragmatic herniorrhaphy in cats and dogs: 96 cases (2001-2013)

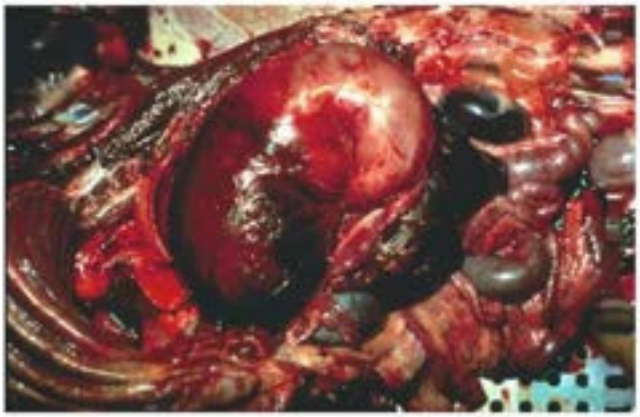
- 79 dogs and 17 cats with DH
- Time between trauma and surgery, trauma and admission, admission and surgery – not associated with survival
- Duration of anesthesia, surgical procedure, concurrent soft tissue or orthopedic injury related to mortality

Perioperative survival rates after surgery for diaphragmatic hernia in dogs and cats: 92 cases (1990–2002)

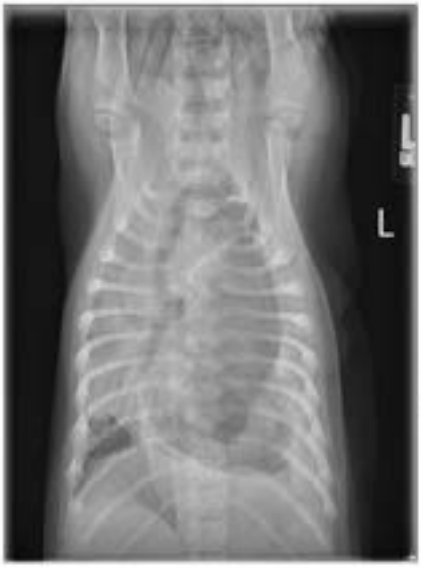
- 63 dogs and 29 cats with DH
- 92% of cases with acute DH received intervention within 24 hours of admission – 94% discharged
- 43% of cases with acute DH received intervention within 24 hours of trauma – 90% survival

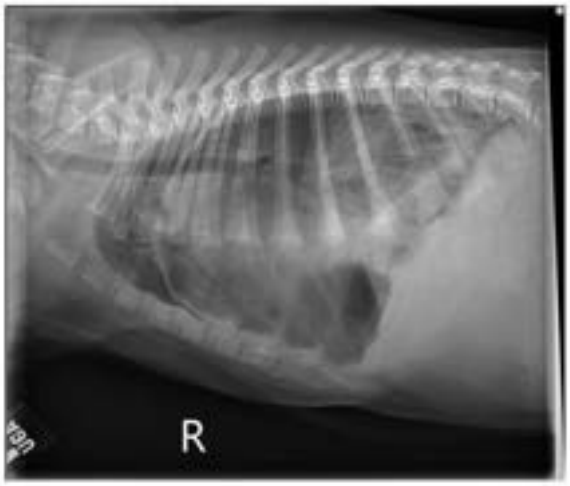
Timing of Surgery: Hernia Contents

- Liver may increase risk of pleural effusion
- Stomach may risk bloat and respiratory compromise
- More likely to take an animal to surgery more rapidly if stomach is herniated (tension gastrothorax)



5 week old Golden Retriever,
Acute respiratory distress

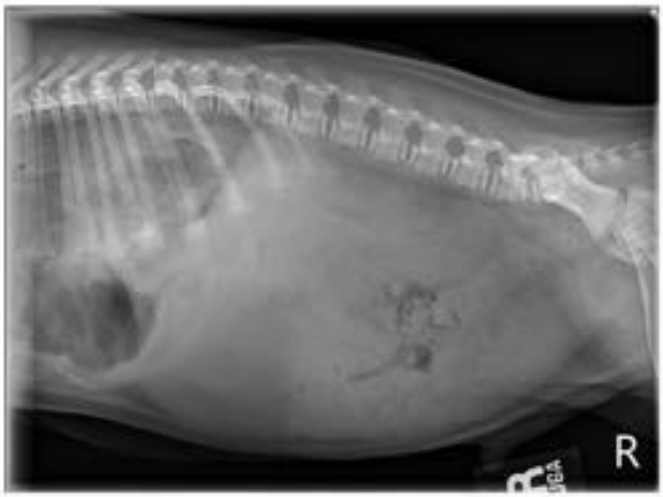




Pleuroperitoneal diaphragmatic hernia with tension gastrothorax

These findings are consistent with congenital pleuroperitoneal diaphragmatic hernia with gastric herniation, entrapment, and distention.

340921



Stomach is not in correct location in the abdomen

Timing: The Bottom Line

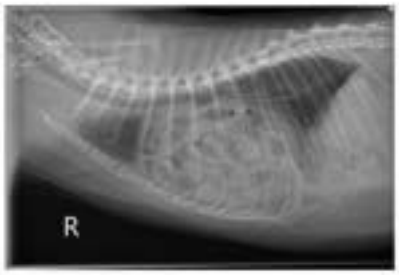
- Surgical intervention should take place when the patient has been adequately stabilized.
- Delay increases the risk of respiratory compromise

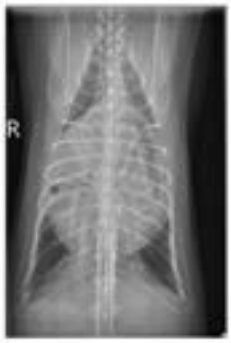
Acute stabilization

- Supplement oxygen
- Analgesia
- Address traumatic injuries
- IV access
- Drain pleural effusion if present
- Emergent surgery maybe indicated

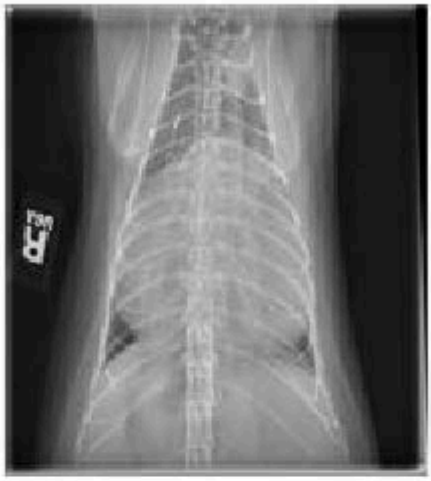
Congenital Diaphragmatic Hernia

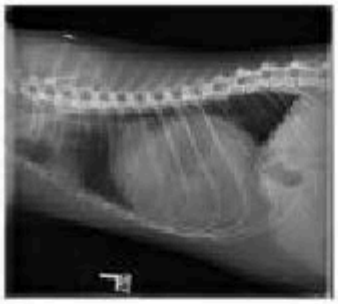
- Heritability is not clear
- Most commonly peritoneopericardial diaphragmatic hernia (PPDH)
- Occurs in cats and dogs
- May be incidental or present with vomiting, inappetance, lethargy, difficulty breathing, pleural or pericardial effusion

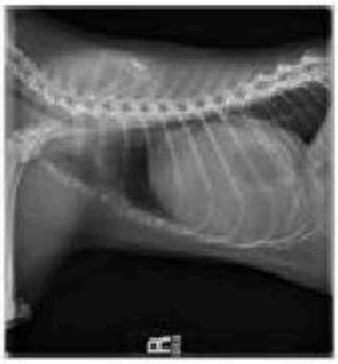




3 yr old, MN, DMH
Vomiting, 3 weeks duration







340972

Surgical vs. Conservative Therapy of PPDH

Surgical and nonsurgical treatment of peritoneopericardial diaphragmatic hernia in dogs and cats: 58 cases (1999–2008)

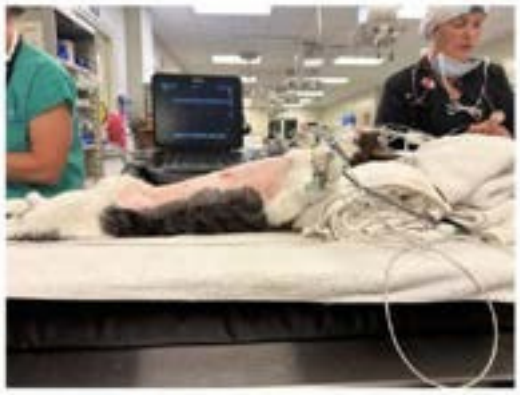
- 34 animals had surgical repair.
- Animals with clinical signs most commonly had surgery
- 9% mortality for surgical treatment
- No difference in long term survival with surgically treated vs. non-surgically treated
- Other congenital abnormalities were common (umbilical hernias, sternal defects, abdominal wall hernias)

Long-term outcome of cats treated conservatively or surgically for peritoneopericardial diaphragmatic hernia: 66 cases (1987–2002)

- 37 treated surgically, 29 treated conservatively
- Post op mortality 14%
- Post op complications in 29/37 cats
- 2 conservatively treated cats had progression of clinical signs which caused death or need for surgical treatment
- 88% owner satisfaction with surgery and 68% owner satisfaction with conservative.

Anesthetic Considerations

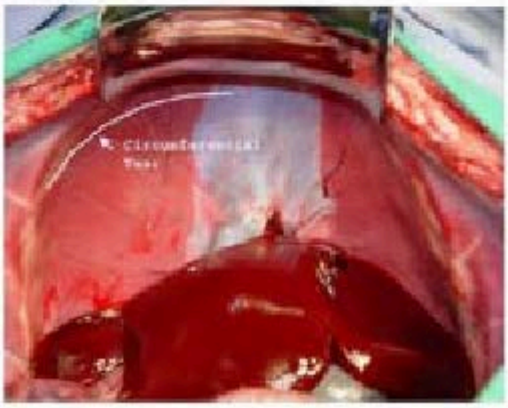
- Preoxygenate
- Elevate the head if possible
- Rapid induction, intubation, and ventilation – induction is a dangerous time
- Reexpansion Pulmonary Edema –Chronic Hernias
  - Avoid high ventilatory pressures,
  - Max around 10 mmHg

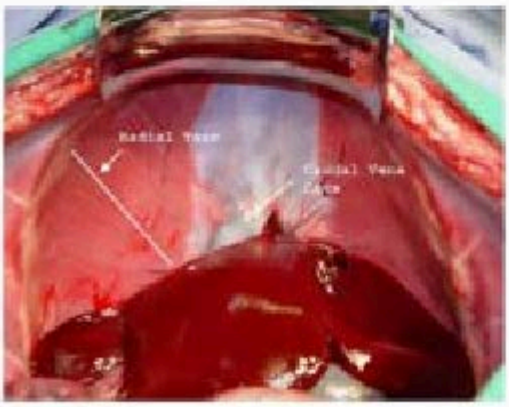


Operative Management: Diaphragmatic Hernia

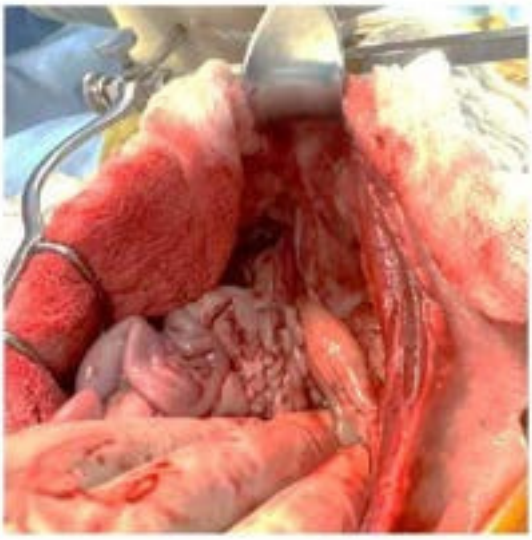
- Approach via the ventral midline
- Drape in extra space for chest tube and extending cranially
- Adhesions and necrosis may be evident
- Debride and close the diaphragm with a simple continuous suture line
- May require muscle flap or mesh is defect is large (rare)
- Drain chest – place a chest tube

Radial or Circumferential Tear



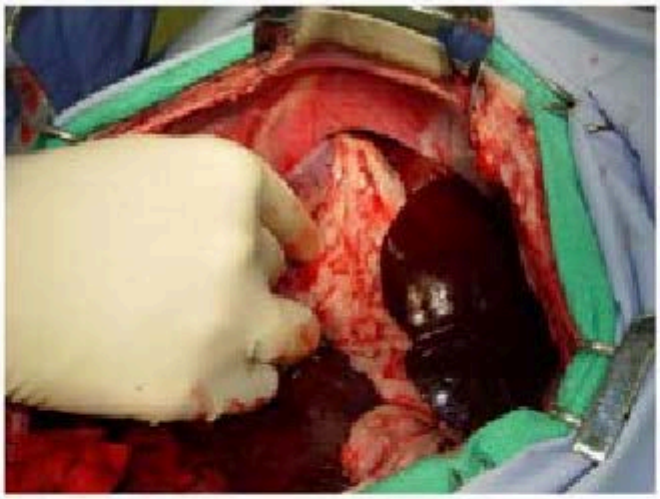


Traumatic hernias can have multiple tears – be sure to inspect the entire diaphragm. Particularly dorsally.



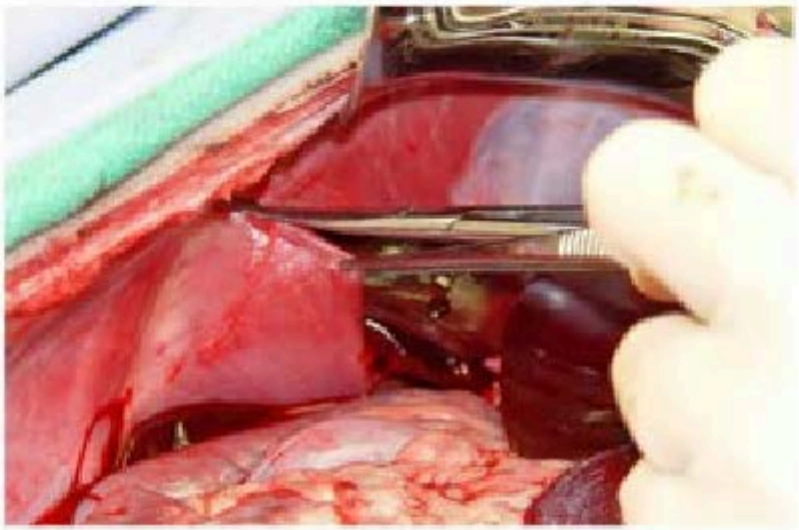
Retractors are important to be able to visualize and repair the defect

Gently Milk Hernia Contents back into Abdomen

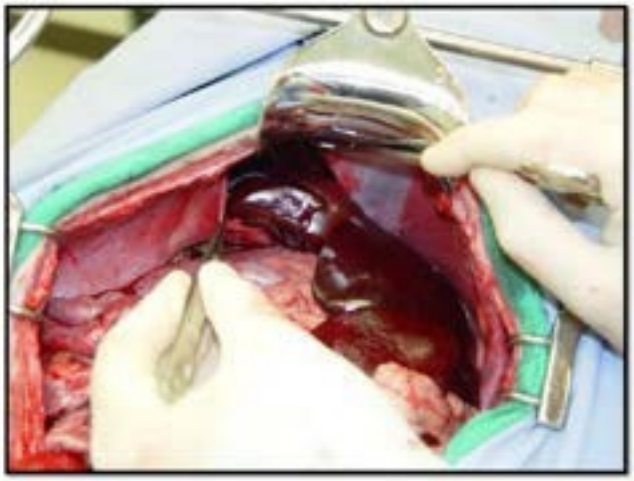


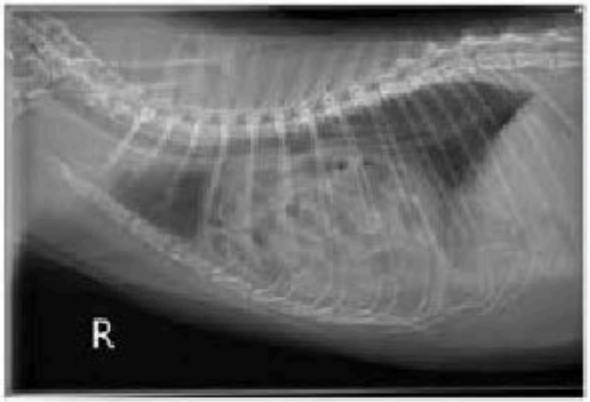
If reduction is difficult,

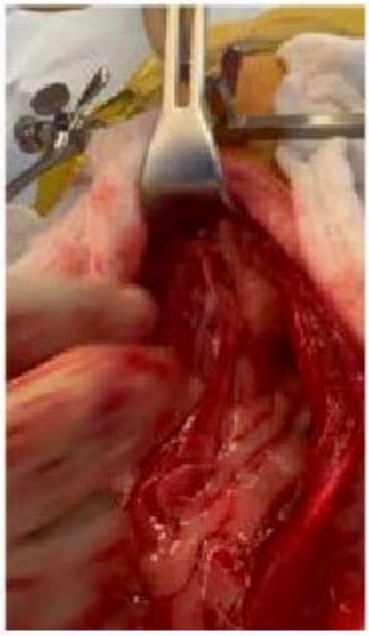
enlarge the hernia



Now organs can be *nontraumatically* reduced

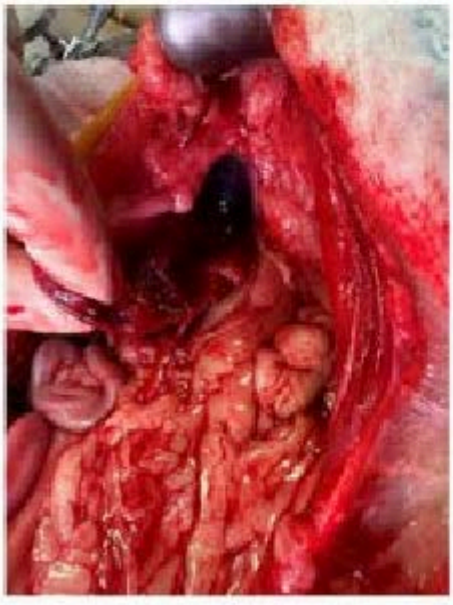




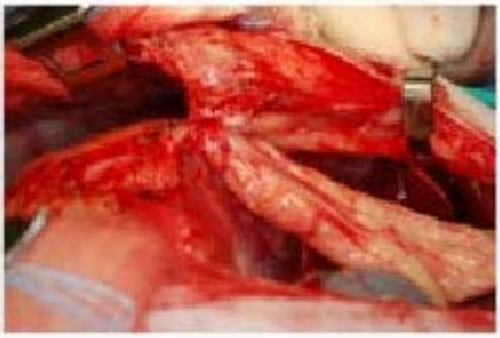


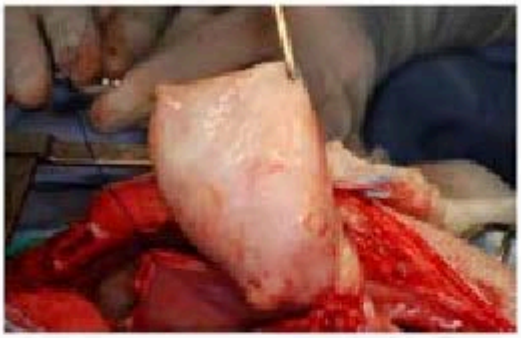
Hernia sac

- Peritoneal mesothelial cells will create a pocket or sac around the hernia
- Can be debrided or left in place.

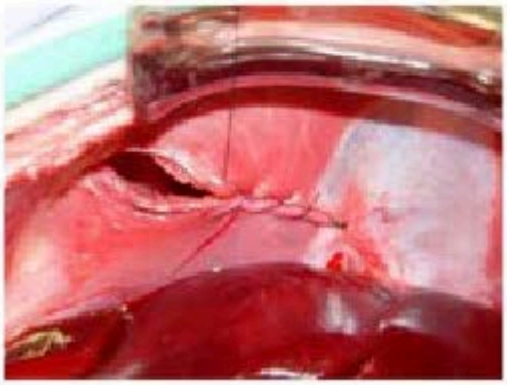


Extending ventral midline abdominal incision into caudal medial sternotomy is helpful if significant adhesions are present

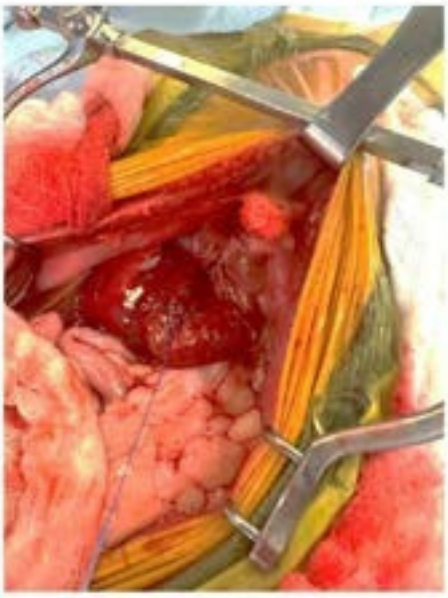


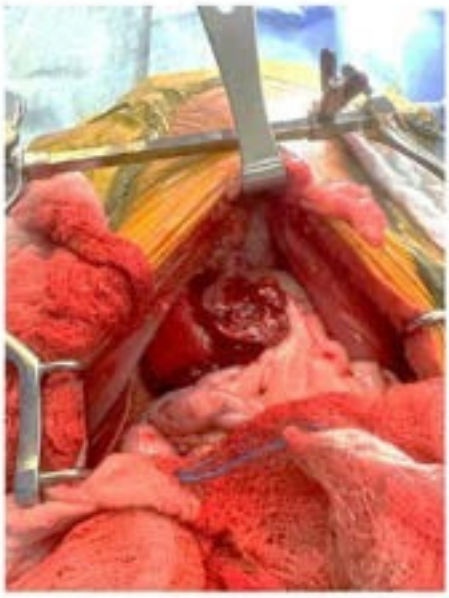


Hernia is closed from dorsal to ventral, medial to lateral (hardest to easiest)

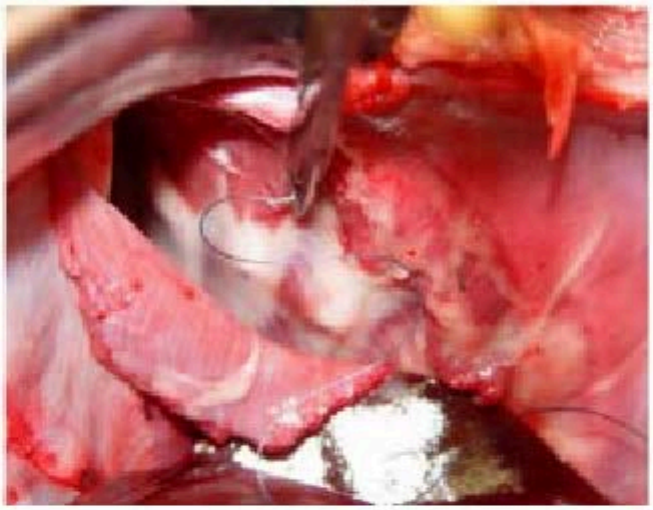


Place and tighten sutures carefully around caudal vena cava hiatus





Circumferential Tears are Bolstered with Circumcostal Sutures, then Oversewn



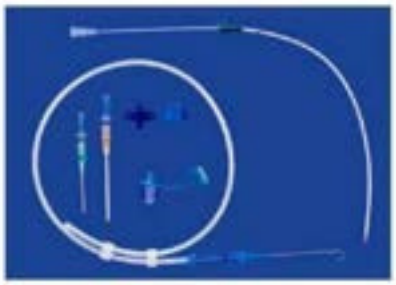
What if you can't close it?

- Transect rib and mobilize thoracic wall
- Rectus Abdominus flap
- Transversus abdominus flap
- Mesh
- Porcine submucosa (SIS)

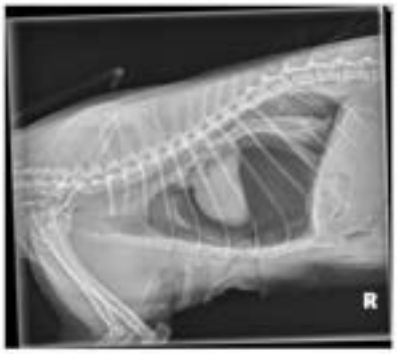
Vet Comp Orthop Traumatol 2/2013

Chest tubes post op

- Can use a red rubber placed through the diaphragm
  - Incorporated into diaphragm closure or,
  - Stab incision and purse string
  - Typically exited through abdominal incision and pulled prior to recovery.
- Or…place a thoracostomy tube prior to closure of the chest.



Complications



- Pneumothorax
- Pneumopericardium
- Reexpansion pulmonary edema
- Sudden cardiac death
- Death from other traumatic injury
- Ascites
- Recurrence of hernia
- Hiatal hernia
- Transient megaesophagus/esophagitis

Prognosis

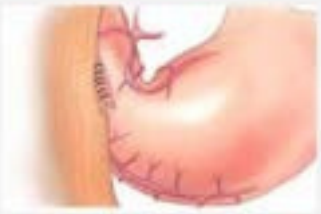
- **~15% mortality prior to presentation**
- **Guarded peri-operatively because of acute risks before, during, and just after surgery. Traumatic hernias survival 82 – 89%.**
- **Post operatively if animal is doing well prognosis becomes much better**
- **Risk of recurrence is low**

GASTRIC DILATATION VOLVULUS

Chad Schmiedt DVM, DACVS
Professor
Small Animal Surgery

OUTLINE

- Signalment, history, exam, diagnostics
- Pathophysiology
- Treatment
  - Pre-operative
  - Intra-operative
  - Post-operative
- Prognosis, prophylactic gastropexy
- Questions and discussion

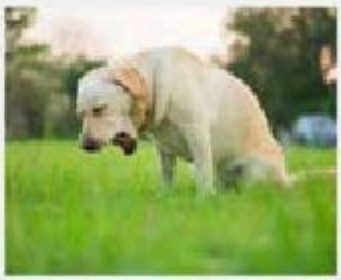


SIGNALMENT

- Deep-chested, large breed dogs
  (Others: Shar Pei, Bassett, Cocker Spaniel)
- Usually middle aged to older

HISTORY

- Restlessness
- Retching
- Non-productive vomiting
- Hypersalivation
- Distended abdomen
- Weakness
- Collapse



PHYSICAL EXAM

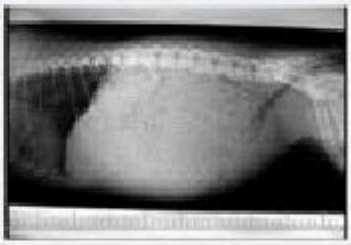
- May be nearly normal
  - Abdomen may not be that distended, femoral pulses may feel OK, may even be wagging tail

May be collapsed, in shock, or obtunded or dead

Or anywhere in between

VS. FOOD BLOAT?

- Typically a history of food thievery is known
- Gastric distention can be as severe as GDV
- Dogs with food engorgement frequently presented with acid-base and electrolyte derangements (including hyperlactatemia)
- Outcome with only supportive care is excellent

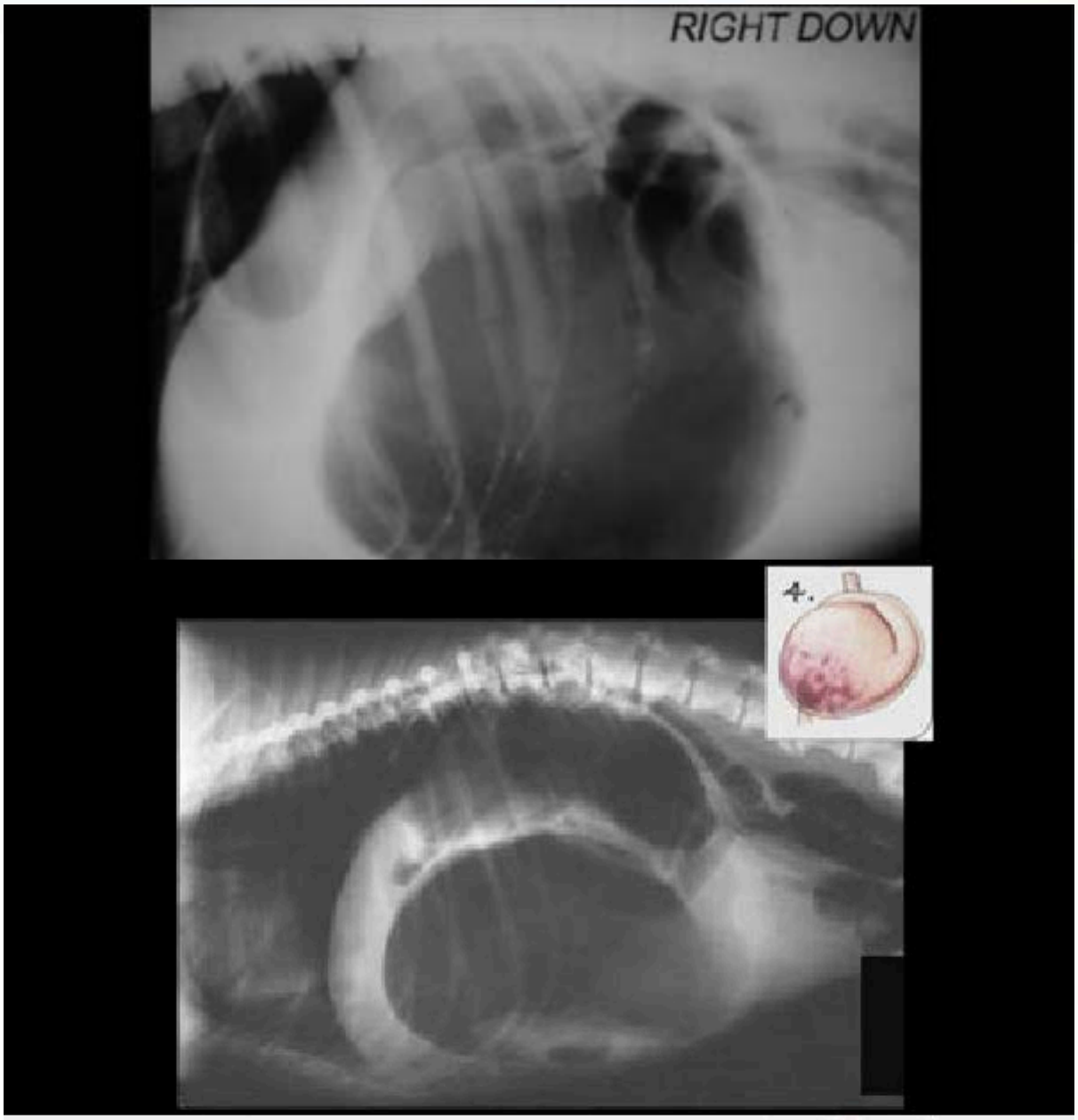


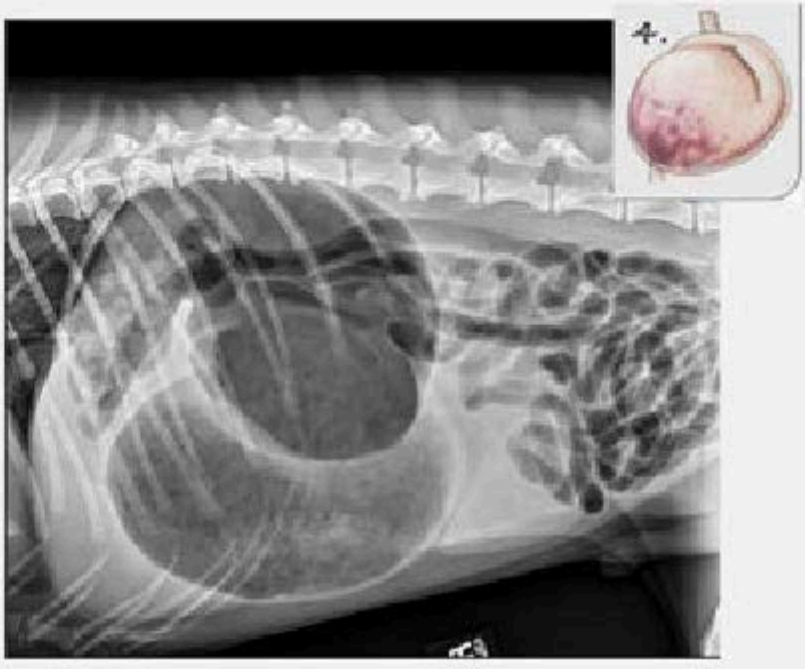
Smart, Reese, and Hosgood, VetRecord, 2017

CLINICOPATHOLOGIC DIAGNOSTICS FOR GDV

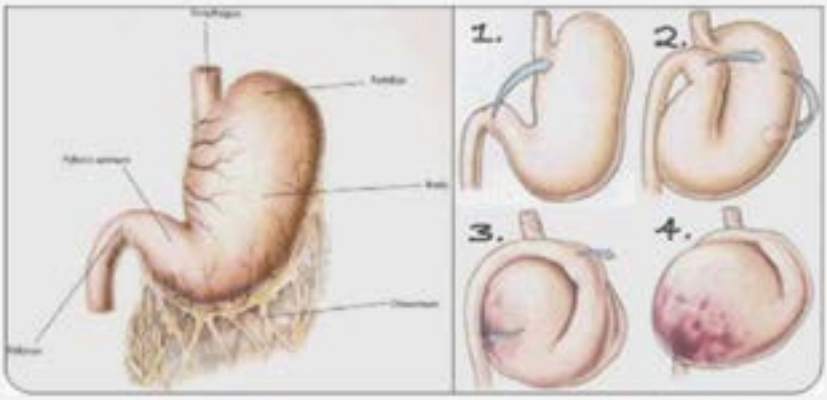
- Right lateral abdominal radiograph
  - Should also obtain an orthogonal view (VD)
- CBC, Chemistry, electrolytes
- ±Coagulation profile
- BP and EKG
- ±3 view thoracic radiographs (older dogs)

For the severely affected animal, stabilization must be performed in parallel with diagnostics


RIGHT DOWN



PATHOPHYSIOLOGY



RISK FACTORS

Increased Risk

- Purebred or giant breed
- History of GD or GDV in a 1st degree relative
- Deep chested
- Fewer meals/day
- Fed dry kibble
- Smaller kibble size
- Anxious/aggressive/fearful
- Spending 5 hours a day with owner
- Intact females
- Splenectomy
- Activity after eating
- Elevated food bowls

Reduced Risk

- Playing with other dogs
- Running the fence after meals
- Fish and egg supplements
- Equal time indoors and out

Brief Communication

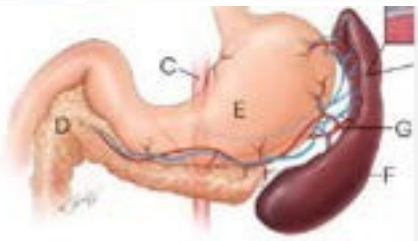
Stomach Gas Analyses in Canine Acute Gastric Dilatation with Volvulus

H.F. Van Kruiningen, C. Gargasoli, J. Hovior, S. Frisch, L. Jin, and S. Salb

- Authors postulate gas is not from aerophagia but a result of bacterial fermentation

Association between previous splenectomy and gastric dilatation-volvulus in dogs: 453 cases (2004–2009)

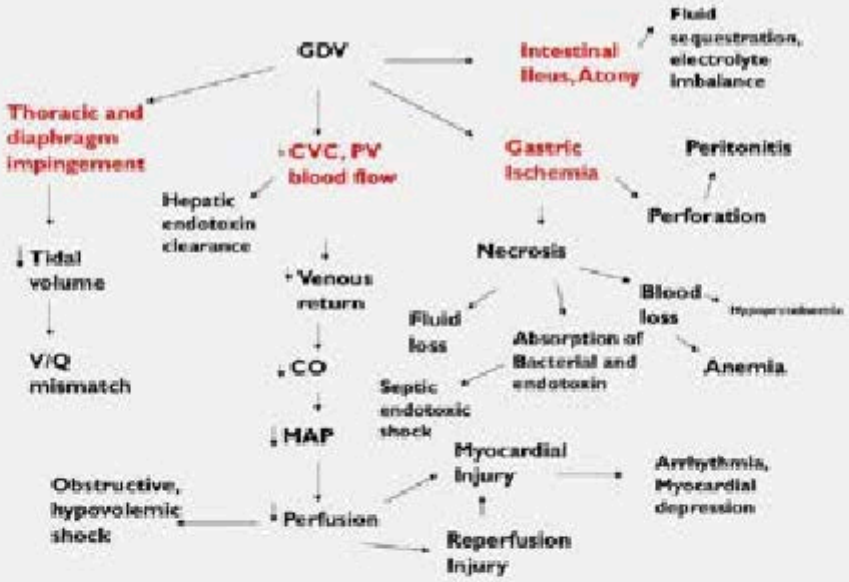
Angela J. Sutter, DVM, Adrienne M. Bentley, DVM, MACVS, Dorothy C. Brown, DVM, MSCI, DACVS



- Previous studies showed no evidence that splenectomy was associated with an increased incidence of subsequent GDV
- Odds of GDV 5.3x with previous splenectomy vs. dogs without splenectomy

Results—6 (4%) dogs in the GDV group and 3 (1%) dogs in the control group had a history of previous splenectomy. The odds of GDV in dogs with a history of previous splenectomy in this population of dogs were 5.3 times those of dogs without a history of previous splenectomy (95% confidence interval, 1.1 to 26.8).

Conclusions and Clinical Relevance—For the patients in the present study, there was an increased odds of GDV in dogs with a history of splenectomy. Prophylactic gastropexy may be considered in dogs undergoing a splenectomy, particularly if other risk factors for GDV are present. *J Am Vet Med Assoc* 2013;242:1381–1384)



ETIOLOGY OF CARDIOVASCULAR COMPROMISE IN DOGS WITH GDV

Ventilation

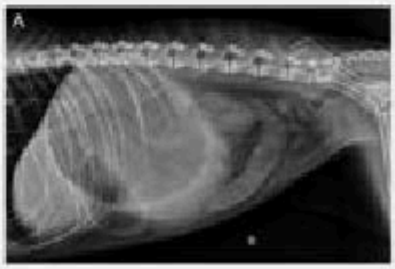
- Diaphragmatic pressure reduces ability to ventilate

Blood Flow

- Reduced cardiac return from abdomen via vena cava
- Reduced portal vein blood flow

Cardiac Dysfunction

- Reduced return means reduced stroke volume and cardiac output
- Electrocardiographic abnormalities in 40 – 70% of cases
  - Myocardial ischemia/necrosis
  - Other?



SECONDARY COMPLICATIONS

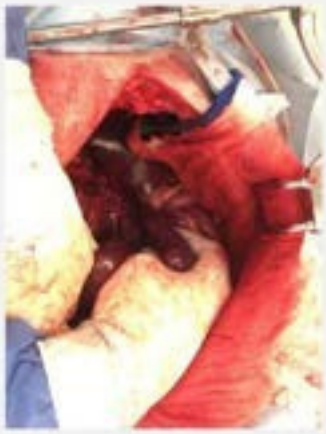
Gastric Wall Necrosis

- Secondary to high intragastric pressure
- Systemic hypotension

Bacterial Translocation

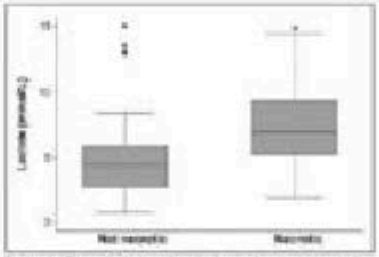
- Unknown significance. Actual occurrence hasn't been documented over control dogs.

Reperfusion Injury



Evaluation of plasma lactate concentration and base excess at the time of hospital admission as predictors of gastric necrosis and outcome and correlation between those variables in dogs with gastric dilatation-volvulus: 78 cases (2004–2009)

- Gastric Necrosis in 12 dogs (20%)
- 65 dogs (83%) survived to discharge
- Dogs with gastric necrosis
  - 8/65 survivors
  - 4/8 non-survivors
- Plasma lactate cutoff 7.4 mmol/liter
  - 82% accurate for predicting gastric necrosis
  - 88% accurate for predicting outcome



Association between outcome and changes in plasma lactate concentration during presurgical treatment in dogs with gastric dilatation-volvulus: 64 cases (2002–2008)

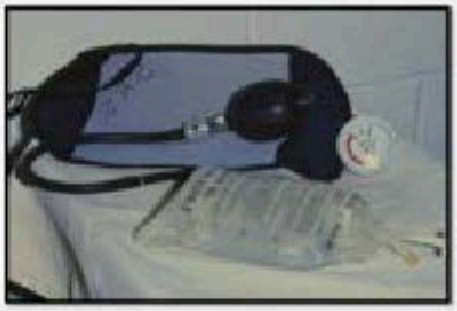
- Dogs with initial lactate concentration below 9 mmol/L = 90% survival (36/40 dogs)
- Dogs with an initial lactate concentration over 9 mmol/L = 54% survival (13/24 dogs)
- Within the dogs with initial lactate over 9 mmol/L
  - Final lactate concentration (after fluid resuscitation) over 6.4 mmol/L = 28% survival
    (vs. 91% for final lactate concentration less than 6.4 mmol/L)
  - Absolute change in lactate concentration less than 4 mmol/L = 10% survival
    (vs. 86% for absolute change greater than 4 mmol/L)
  - Percentage change in lactate concentration less than 42.5% = 15% survival
    (vs. 100% for percentage change greater than 42.5%)
- So initial lactate is important, but better prognosis dogs with a high initial lactate that can be reduced with pre-surgical resuscitation

Zacher JAVMA 2010

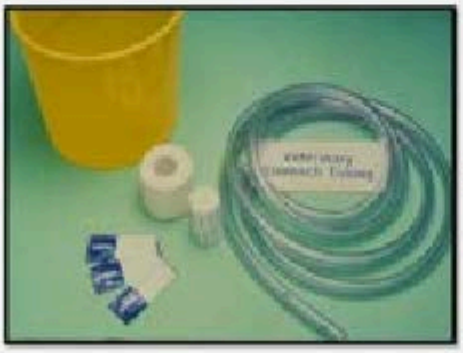
TREATMENT

TREATMENT: PRE-OPERATIVE

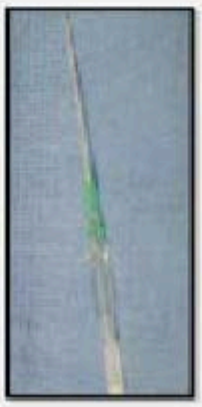
- Stabilization → IV isotonic crystalloids, hypertonic saline, colloids
  - 16 or 18 Ga IV Cath
  - Front limb
- Antibiotics
- Anti-arrhythmics
- Analgesia / Premeds
- Other: oxygen?
  free radical scavengers?



GASTRIC DECOMPRESSION IS CRITICAL!



And/Or



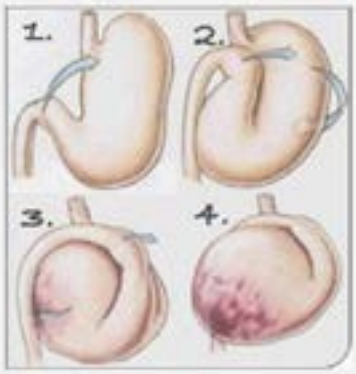
TREATMENT: INTRA-OPERATIVE

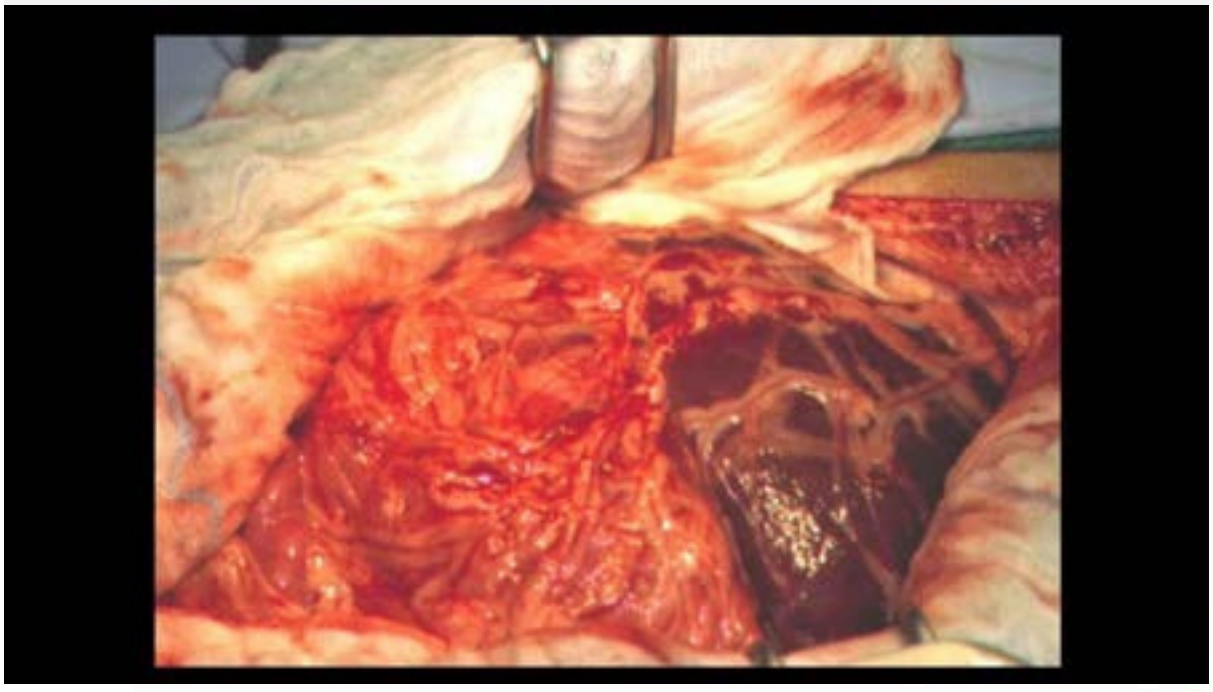
1. Gastric repositioning
2. Evaluate abdominal viscera
    1. If indicated, perform splenectomy
    2. If indicated, perform partial gastrectomy
3. Perform right sided gastropexy

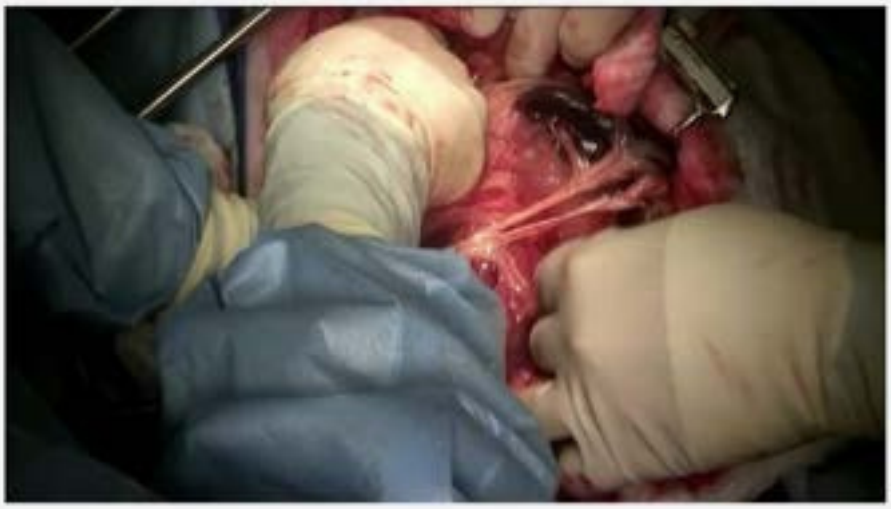
*** *Maintain vigilant monitoring of response to treatment and adjust (fluid) therapy accordingly – BP and EKG monitoring are essential intraop*

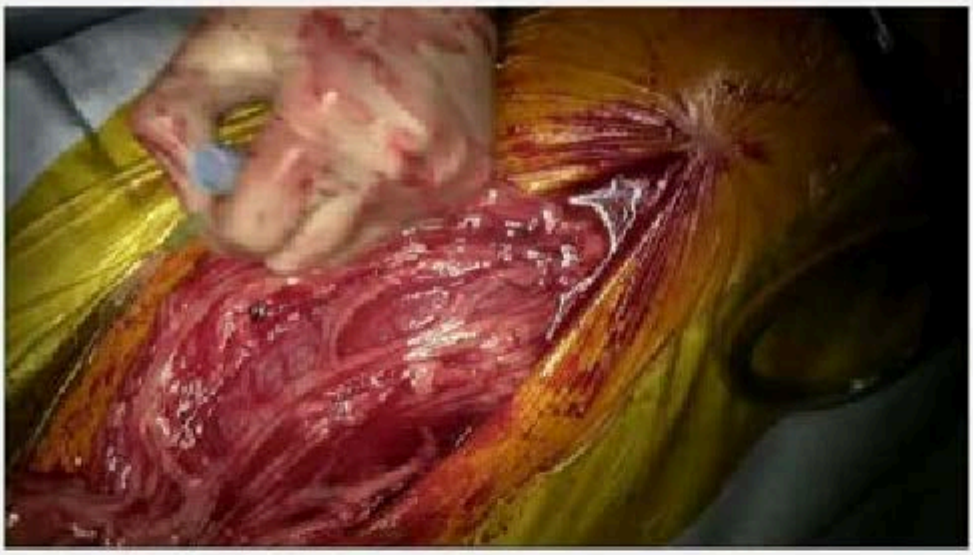
TREATMENT: GASTRIC REPOSITIONING

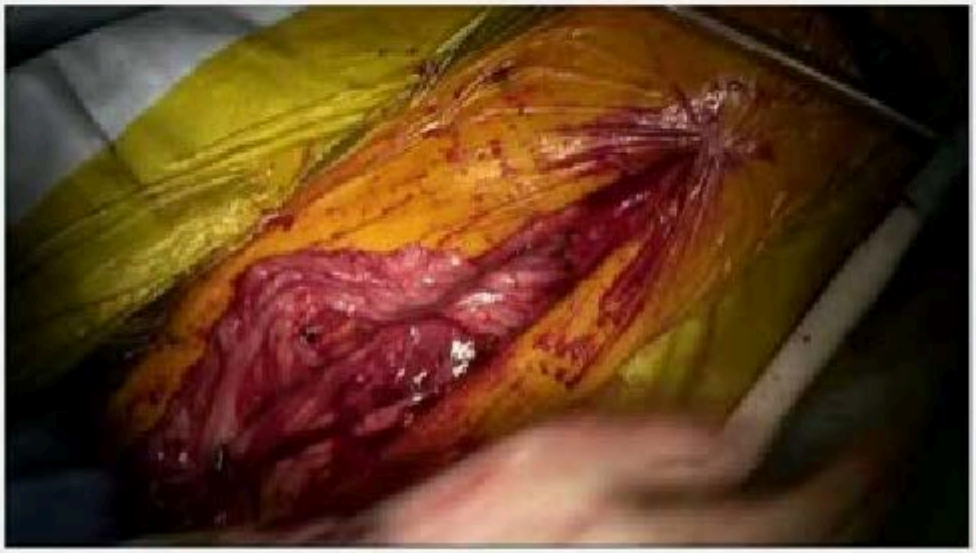
- In most cases, derotation can be accomplished by standing on the dog's right side and:
    - Pushing the fundus (which is nearest to you) dorsally and to the dog's left
    - Pulling the pylorus (which is away from you, near the dog's left side) ventrally and towards you
    - Use a gentle but confident application of force
    - Often helpful if the stomach is decompressed
- Verify correct positioning afterwards by carefully palpating esophageal hiatus
    - Some may have experienced a counter-clockwise volvulus



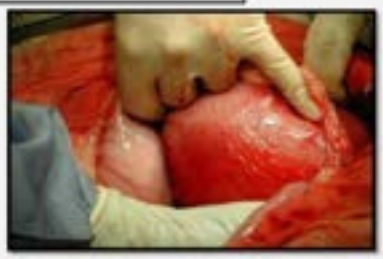


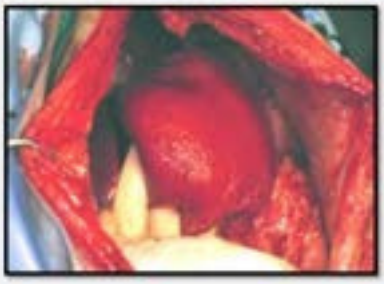






TREATMENT: ABDOMINAL EVALUATION



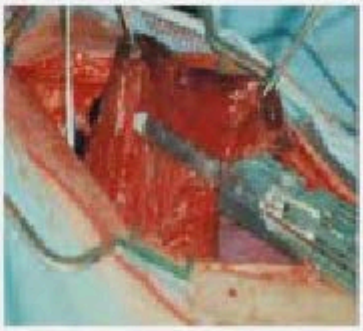


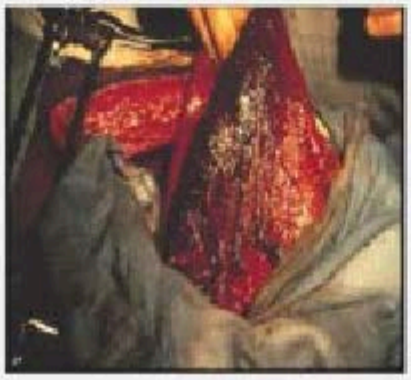
TREATMENT:
+/- SPLENECTOMY
+/- PARTIAL GASTRECTOMY

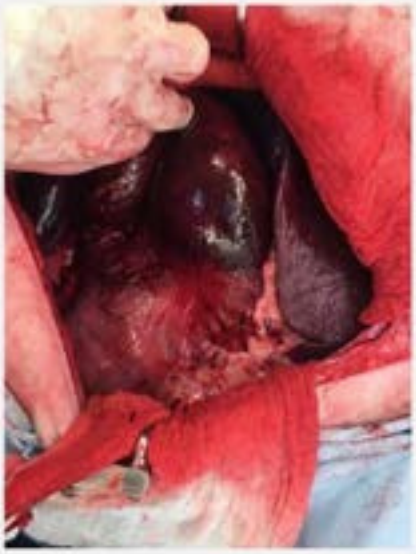
- Evaluate once completed remainder of abdominal explore
- If spleen is thrombosed or vascularly devitalized → splenectomy
- If stomach wall palpates thin, is excessively dark/black or white/grey → partial gastrectomy
- Gastric invagination? – has been associated with chronic recurrent bleeding gastric ulcers and clinical anemia

PARTIAL GASTRECTOMY

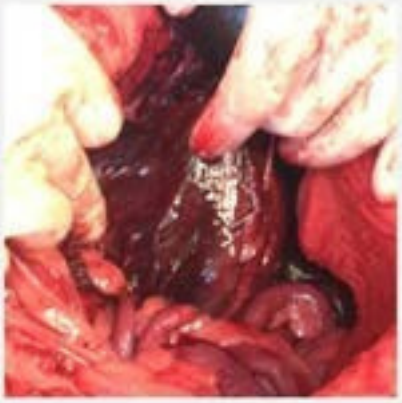
- Invagination
- Stapled – GIA stapler
- Hand sewn





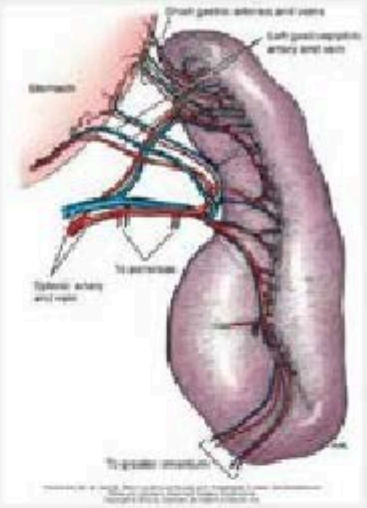


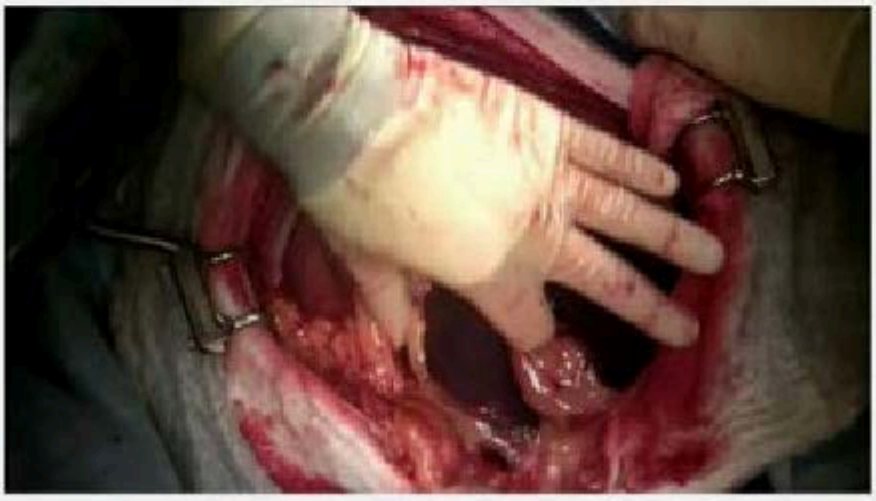
When do you say,
"I can't fix this."?



SPLEEN

- Hemoabdomen
  - Short gastric avulsion
- Splenic congestion
  - De-rotate stomach and see how it does!
  - Texture, color
  - Pulse





SPLENECTOMY?

- Evaluate once completed remainder of abdominal explore
- If spleen is thrombosed or vascularly devitalized → splenectomy

TREATMENT: RIGHT SIDED GASTROPEXY

Incisional
- Easiest; somewhat weaker but strength is still supra-physiologic

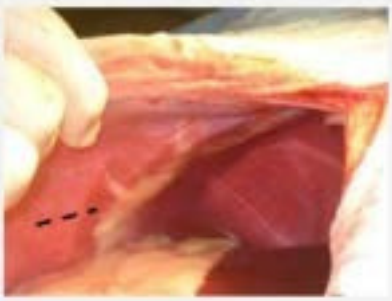
Belt-loop

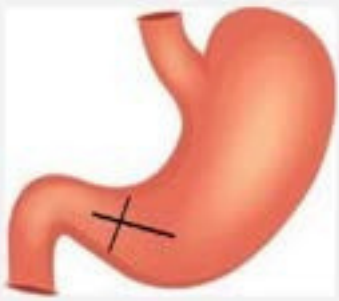
Circumcostal

Ventral incision
- May lead to inadvertent gastrotomy during future abdominal surgeries

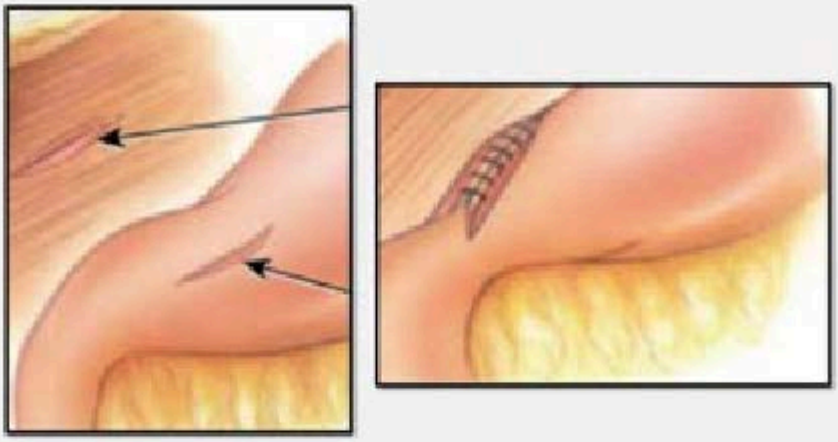
Tube
- Weakest, often stretches and may lead to recurrence

INCISIONAL GASTROPEXY

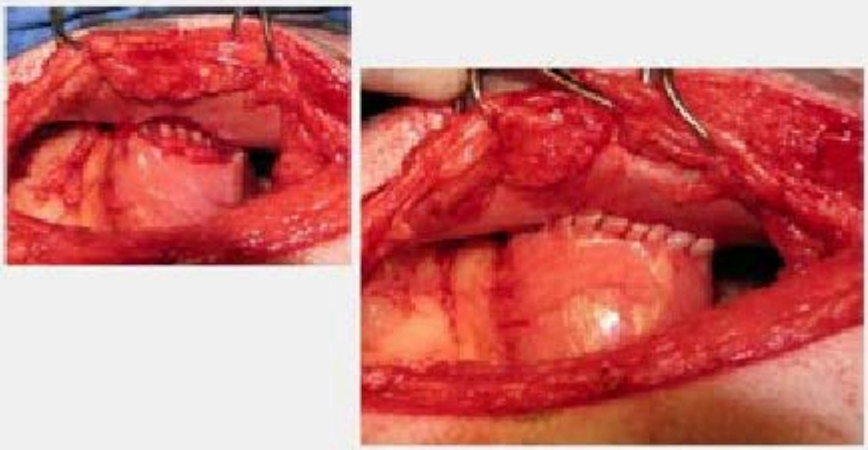


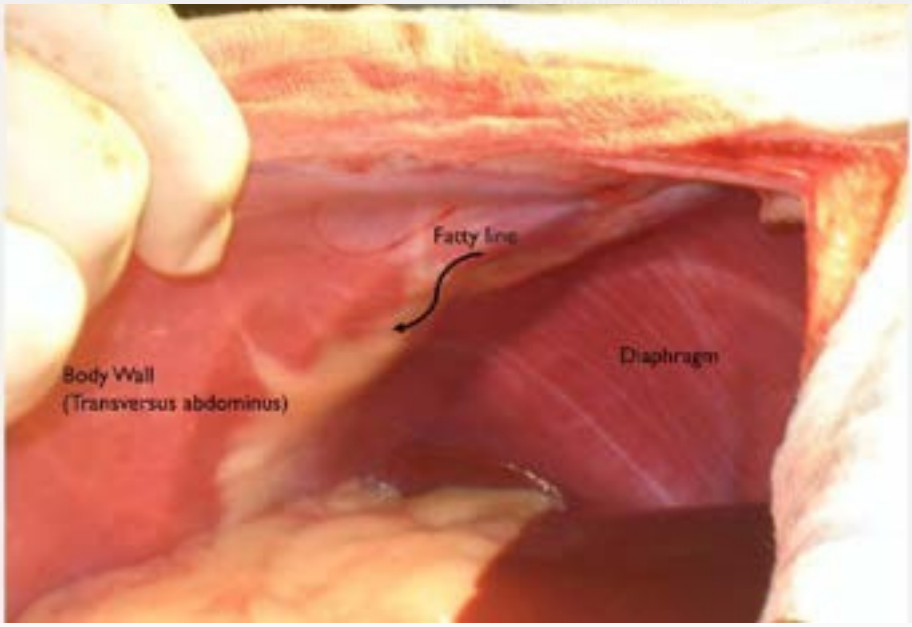


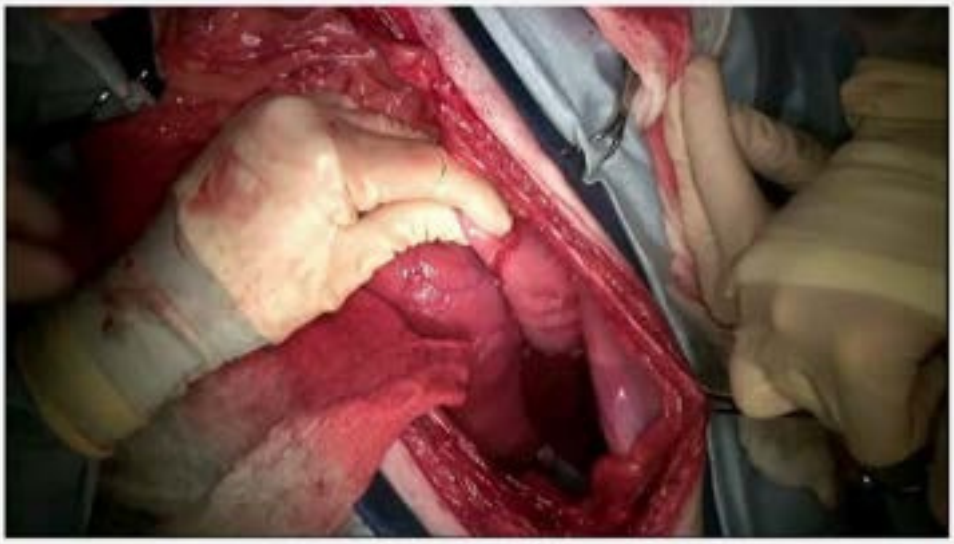
Incision in seromuscular layer of the stomach can be parallel or perpendicular to the long axis of the stomach

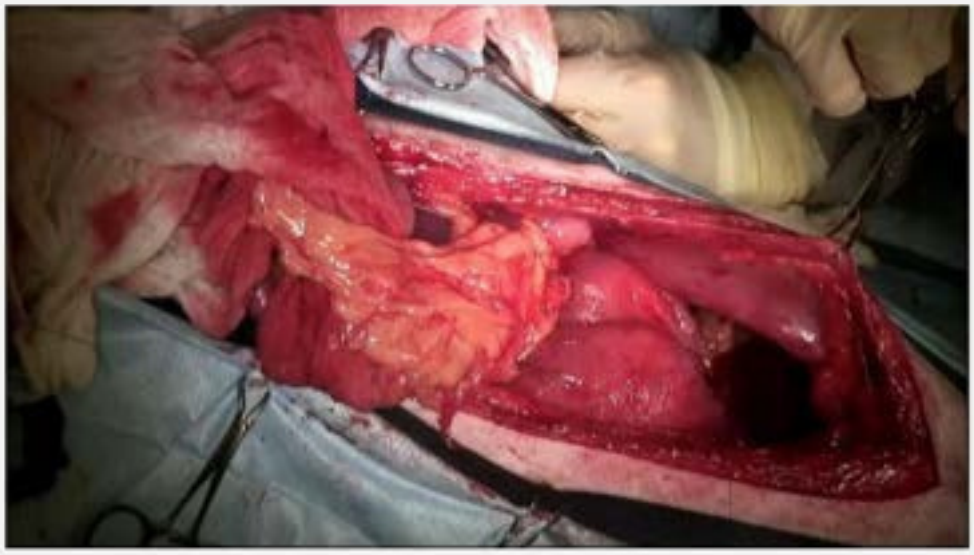


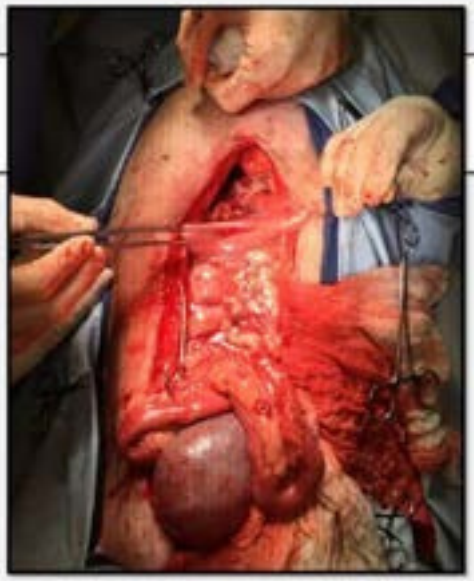
INCISIONAL GASTROPEXY

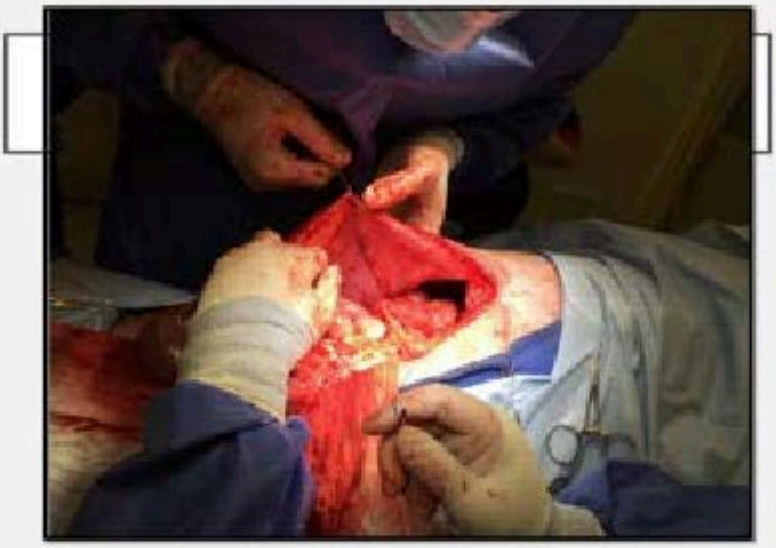


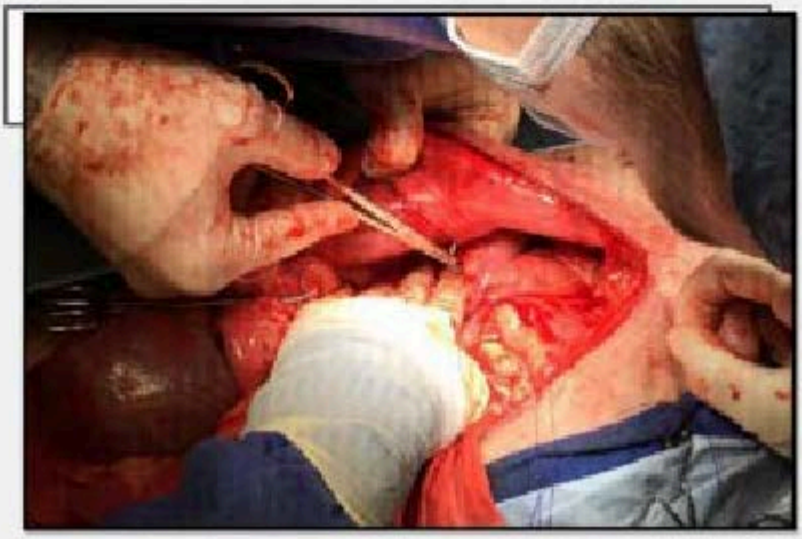


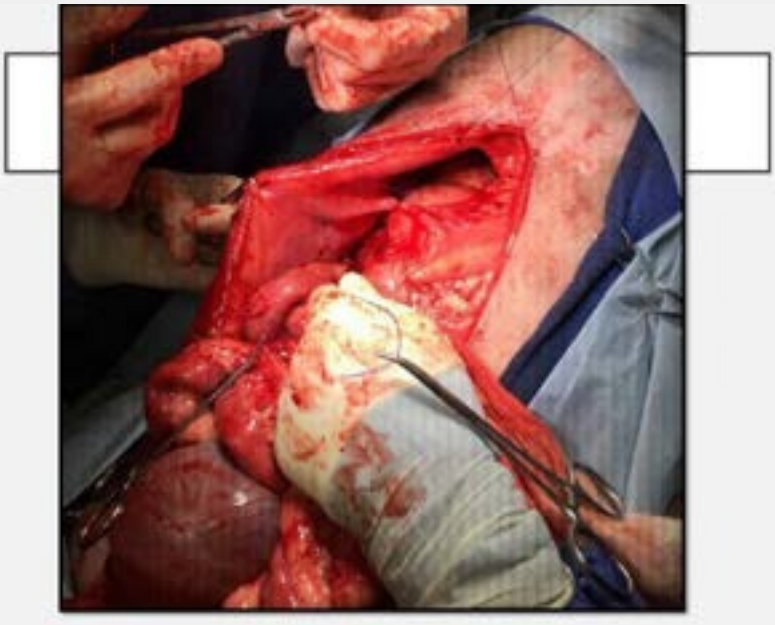


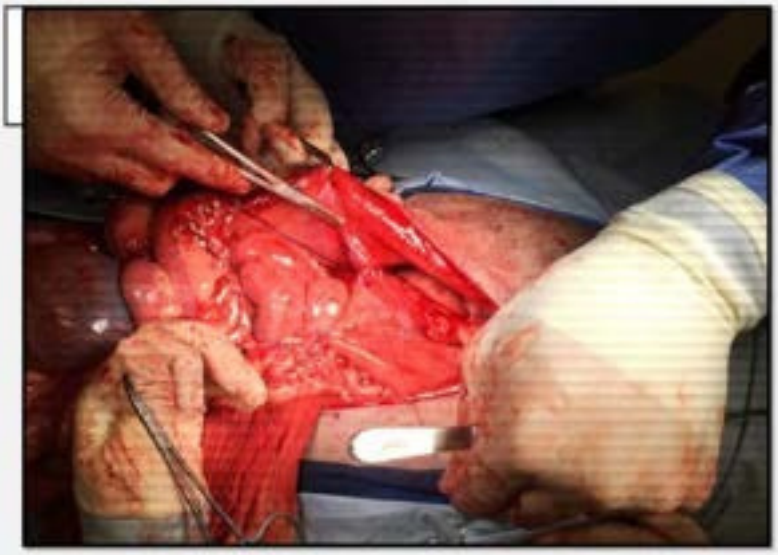


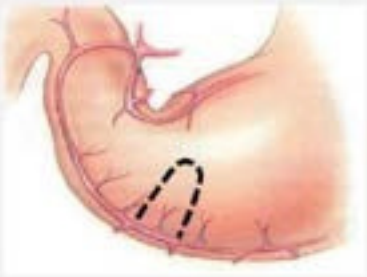


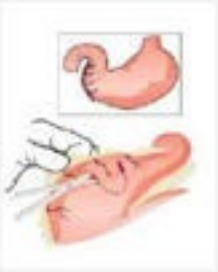




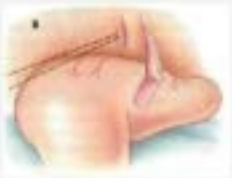




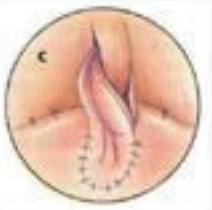




Abdominal Wall
~5 cm long
~3 cm apart

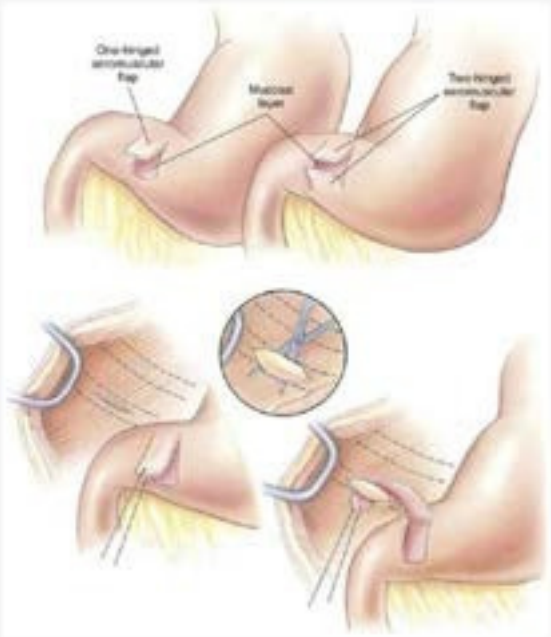


Seromuscular flap
based on greater
curvature
~4 cm long
~3 cm apart



Circumcostal Gastropexy

- Similar to a belt loop but passes behind a rib instead of through a soft tissue tunnel
- Behind the 11th or 12th rib
- Rib fracture, hemorrhage, and pneumothorax are reported complications.



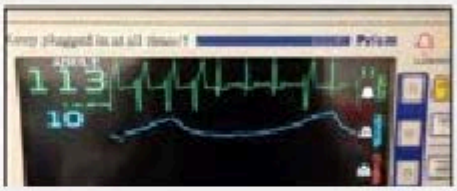
Efficacy of Incisional Gastropexy for Prevention of GDV in Dogs

Marian E. Benitez, DVM*, Chad W. Schmiedt, DVM, DACVS, MaryAnn G. Radlinsky, MS, DVM, DACVS, Karen K. Cornell, PhD, DVM, DACVS

- 61 dogs with incisional gastropexy
  - 34 had gastropexy at GDV surgery
  - 27 had prophylactic
- Median follow up was 700+ days
- Recurrence of GD alone in 3/34 patients (~9%) in the GDV group
- Occurrence of GD in 3/27 (~11%) in the prophylactic group

TREATMENT: POST-OPERATIVE

- Continuum of care from pre-op to intra-op to post-op
  - Aggressive volume expansion
    - Crystalloids 90-120 mL/kg/day
    - HES 10-30 mL/kg/day
    - +/- FFP or other blood products
- Monitoring
  - EKG, BP, emesis
  - Bloodwork and lytes



TREATMENT: POST-OPERATIVE

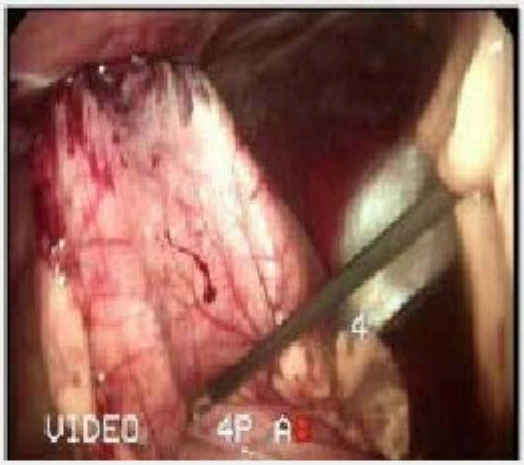
- Analgesics (opiods; avoid NSAIDs)
- Consider gastroprotectants (sucralfate +/- famotidine and/or omeprazole)
- Antibiotics usually indicated for presumed risk of bacterial translocation
- Consider anti-emetics/prokinetics if vomiting
- Encourage small frequent meals as soon as will tolerate

PROGNOSIS

- Guarded to fair
  - Mortality ~10- 27%
- Prognostic Factors
  - Long (>6 hrs) clinical signs
  - Concurrent gastrectomy or splenectomy
  - Gastric necrosis
  - Cardiac arrthymias
  - Peritonitis
  - Plasma Lactate (<6 mmol/L has a 99% survival)
  - Changes in lactate (> 4 mmol/L after resuscitation)

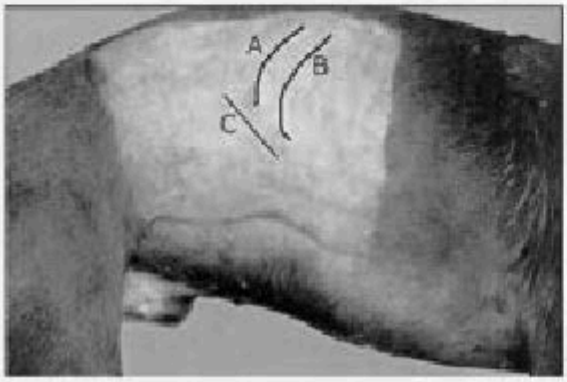
PROPHYLACTIC GASTROPEXY

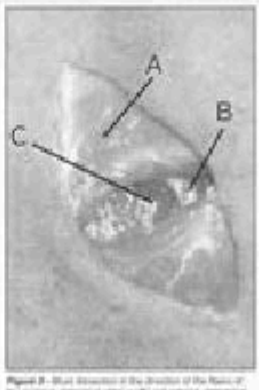
- Previous episode of GD?
  - Likelihood of GDV after episode of GD is ~ 85%
- Familial history of GDV?
- At risk breed?
- Working/valuable dog?

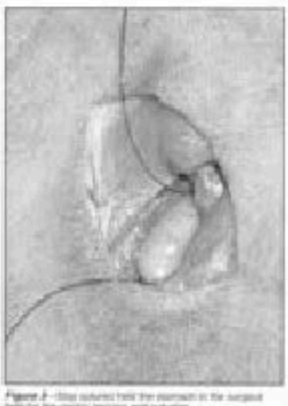


GRID GASTROPEXY

Clinical Evaluation of a Right-Sided Prophylactic Gastropexy Via a Grid Approach







J Am Anim Hosp Assoc 2003;39:397–402.

Spontaneous gastric dilatation-volvulus in two cats

Meredith L. Leary, VMD and Virginia Simnott-Stutzman, DVM, DACVECC

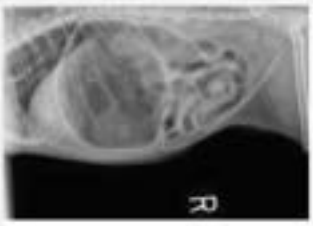
2 female Persian cats

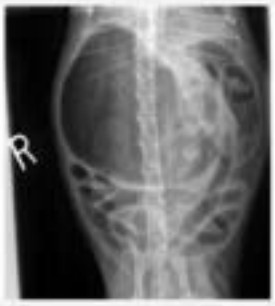
Only 1 had compartmentalization evident on preop rads

Both confirmed intraop

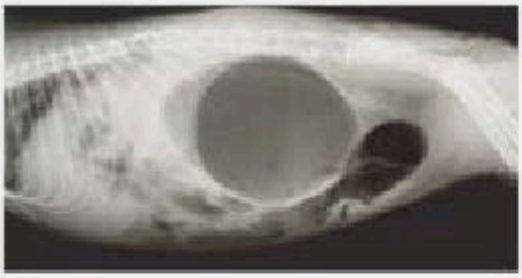
No history of trauma

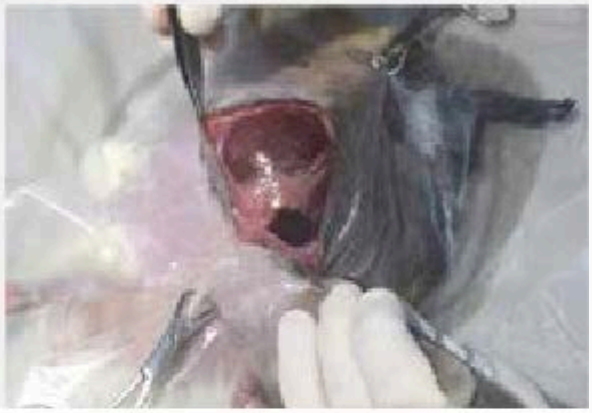
Others reported in association with diaphragmatic hernia





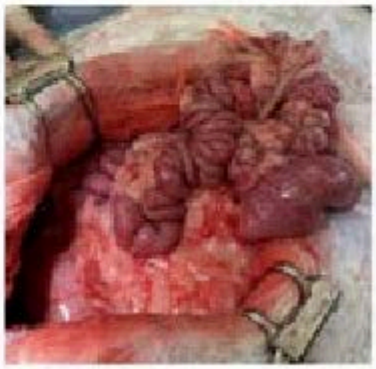
REVIEW OF GASTRIC TORSION IN EIGHT GUINEA PIGS (*CAVIA PORCELLUS*)





Considerations in Gastrointestinal Surgery:

Focus on GI Foreign Body Obstruction



Chad W. Schmiedt, DVM

College of Veterinary Medicine
UNIVERSITY OF GEORGIA

Objectives

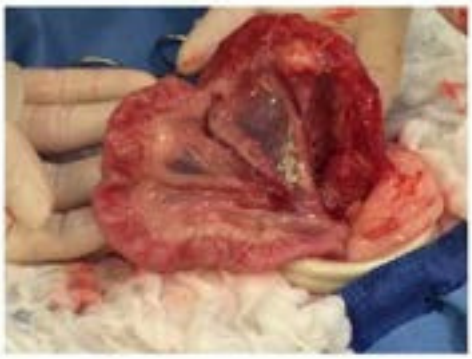
- Understand **preoperative diagnostics** and **medical stabilization** in dogs and cats with gastrointestinal foreign bodies
- Review **basic anesthetic considerations** for emergency foreign body surgery
- Review **basic surgical principles** of gastric and intestinal surgery
- Review **postoperative considerations and complications** in dogs and cats following gastrointestinal surgery

Preoperative Considerations and Diagnostics

Is there a functional obstruction?

Functional obstructions can present a similar way to mechanical obstructions

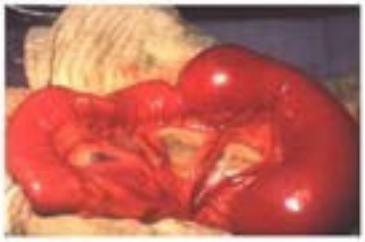
- Inflammation (IBD, peritonitis, pancreatitis)
- Infection (parvoviral)
- Iatrogenic (hypokinetic drugs, surgical manipulation)
- Electrolyte or metabolic imbalance
- Idiopathic

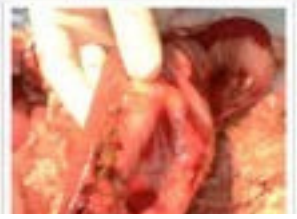


Pyogranulomatous jejunitis in a cat

Could this be a mechanical obstruction other than a foreign body?

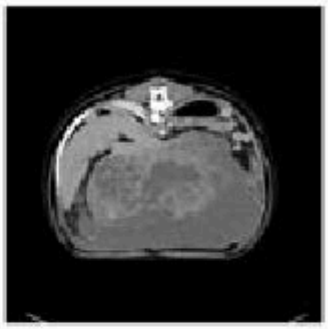
- Linear or Singular Foreign Body
- Intussusception
- Stricture
- Neoplasia
- Torsion





Other mental context for obstruction etiology

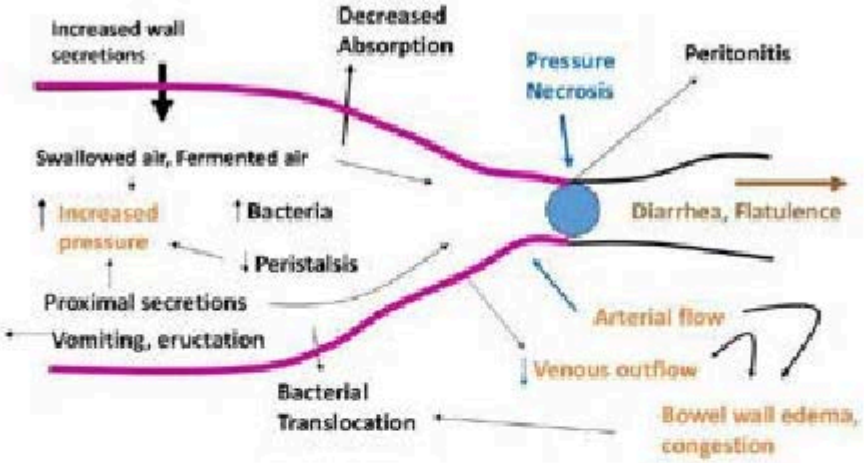
- Gastric vs. duodenal vs. jejunal
- Acute vs. chronic
- Partial vs. complete
- Intraluminal vs. intramural vs. extraluminal
- Strangulating vs. simple



Pathophysiology

- Excessive fluid secretion
  - Increased parasympathetic tone from stretch receptors
  - Lack of aboral transport
- Malabsorption
- Fluid, electrolyte, and acid base disturbance
- Bacterial proliferation +/- translocation
  - More problematic in distal obstructions
- Devitalized bowel
- Perforation

Pathophysiology



Preoperative Stabilization

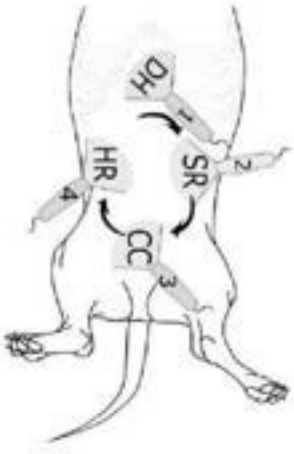
Based on abnormalities identified on physical and blood work

- Correct dehydration
- Correct electrolyte
- Reevaluate acid-base status

Other Diagnostics

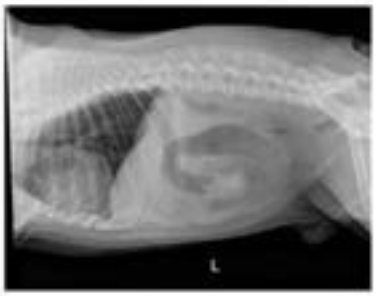
- aFAST (abdominal-**F**ocused **A**ssessment with **S**onography for **T**rauma)
  - Diaphragmaticohepatic
  - Splenorenal
  - Cystocolic
  - Hepatorenal

- Useful for identification of free abdominal fluid
- Free abdominal fluid will dramatically impact prognosis
- *May not be apparent in a dehydrated animal (recheck after rehydration)*

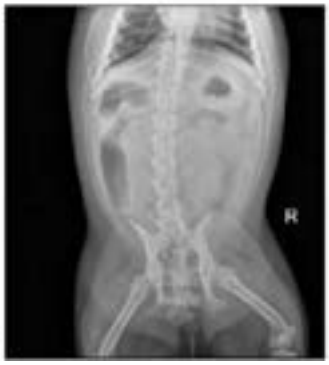


Lisciandro, J Vet Emerg Crit Care, 2011

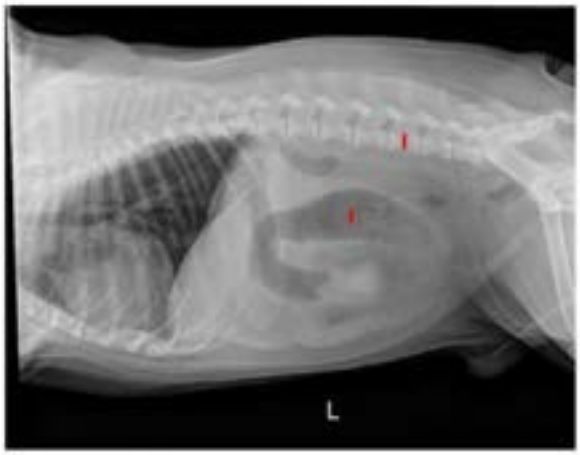
Radiography



- 3 view abdominal films
- Classic signs:
  - 2 populations of bowel
  - Radio-opaque foreign object
  - Abnormal shapes – plication, stacking
- Its not always classic though…
  - Intestinal dilation alone present in 45-55%
  - Active vomiting, poor detail, poor body condition, peritonitis can all make that evaluation difficult



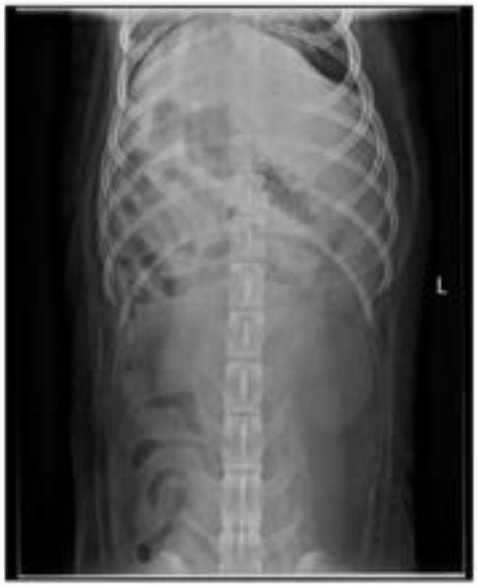
How big is too big?

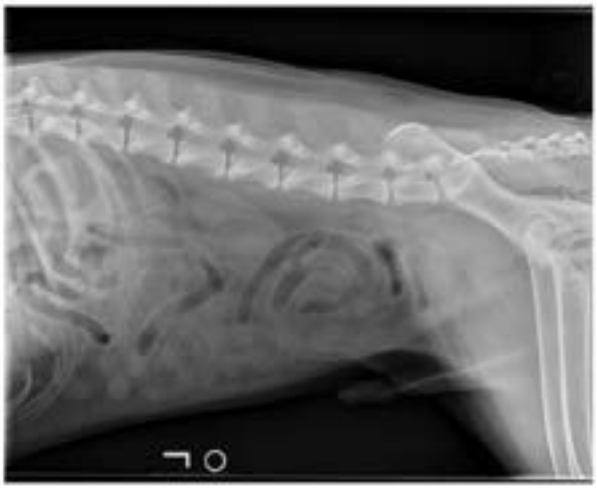


- Small intestine
  - Height of mid-body of L5 in dogs x 1.6 = 66% sensitive and specific
  - Greater than 2.4x = 74-92% sensitive, 74-84% specific
  - 1.6 -2.4x can be from nonobstructive causes – enteritis, nonobstructive ileus
  - Cats ~ 12 mm
- Large intestine
  - Length of body of L5

Radiographic findings need to pair with clinical signs

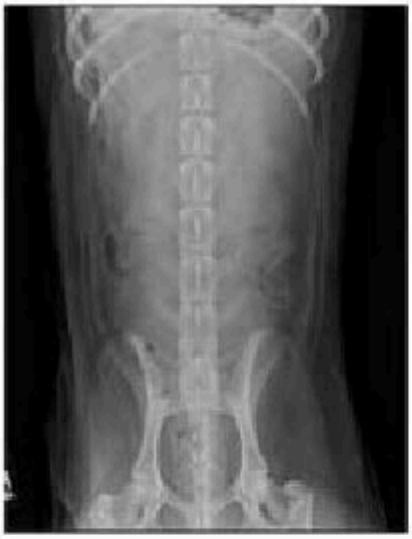
- 1 yr old, MN, Bernedoodle
- Previous history of eating foreign objects, previous gastrotomy ~ 6 months prior
- 1 week prior to presentation – ate toys, threw up in yard
- 3 days before presentation – threw up some boxer shorts, become progressively more lethargic and inappetent.
- At presentation dog appeared normal and had a nonpainful abdomen, NOVA was normal, lactate was 2.0 mmol/L, PCV -47%, TS – 6.6

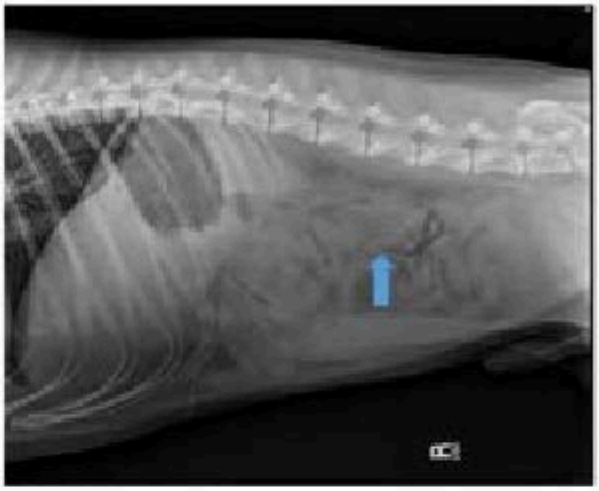




Admission:

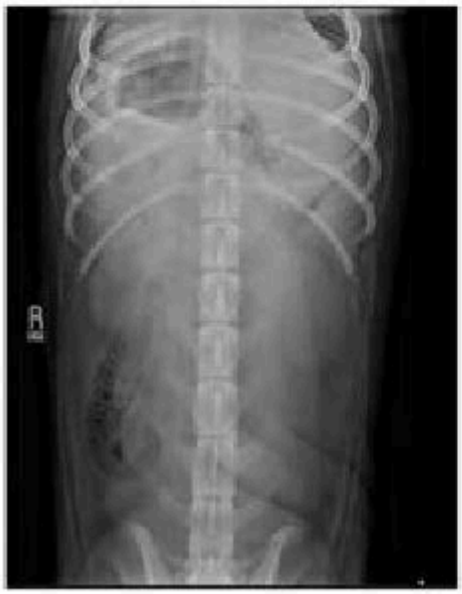
- Stomach, pylorus, duodenum are normal
- Right mid abdominal nonobstructive foreign body

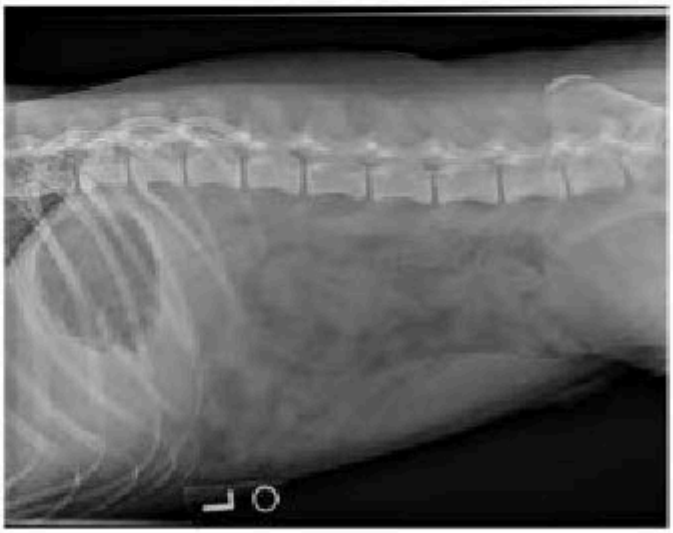




Day 2:

- Foreign material still present, still non obstructive
- Dog not vomiting, BAR, eating

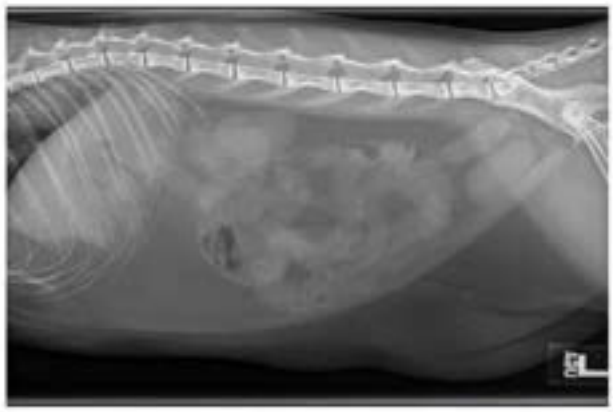


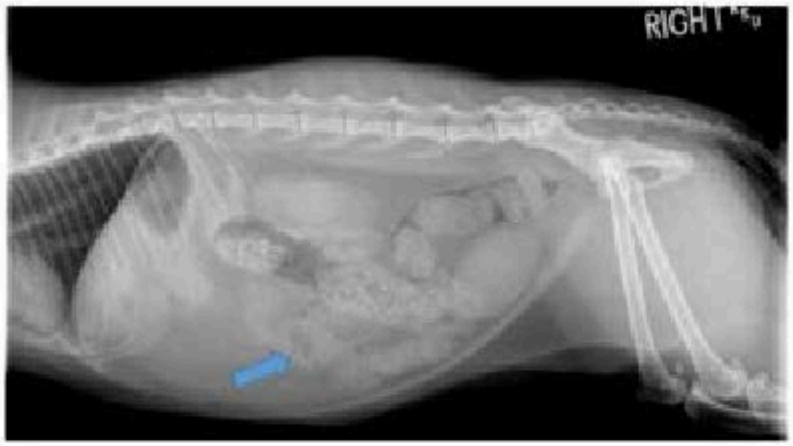


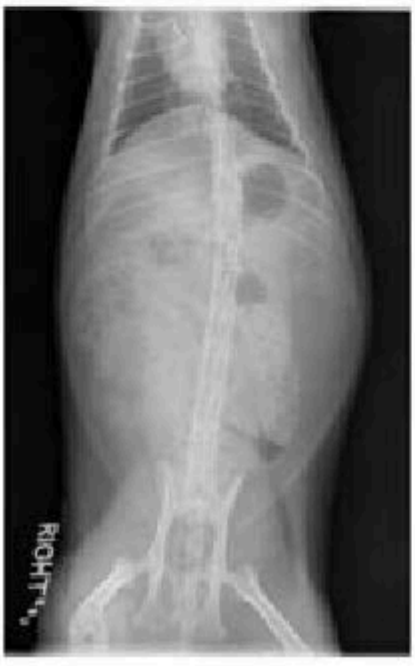
Day 3
Foreign material in colon
Dog discharged

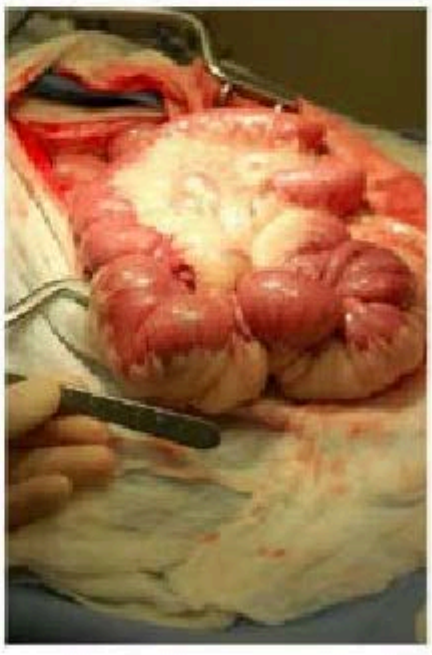
Radiographic appearance of linear foreign bodies

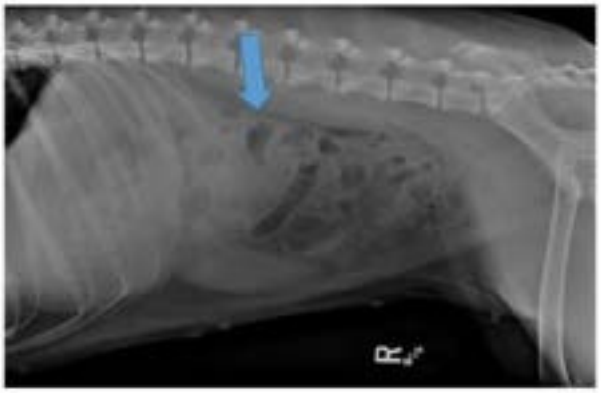
- Look at the stomach, duodenum, proximal jejunum
- Plication, comma shaped gas opacities
- Frequently no distention

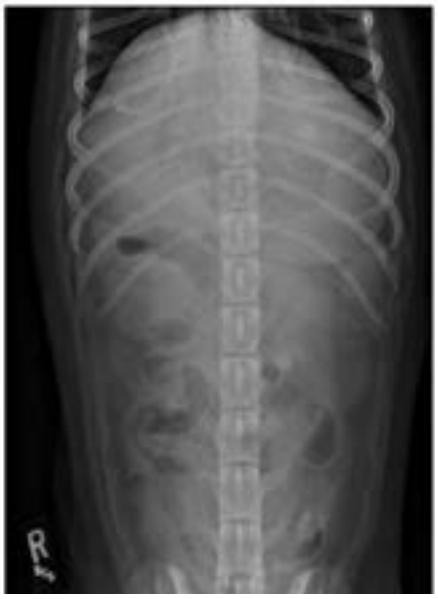










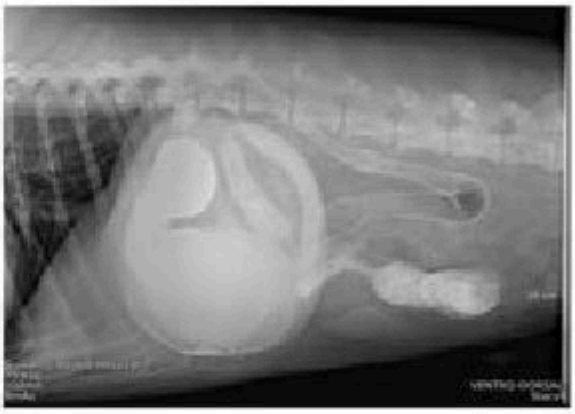


Comma shaped gas opacities

Bunched duodenum in the right cranial quadrant

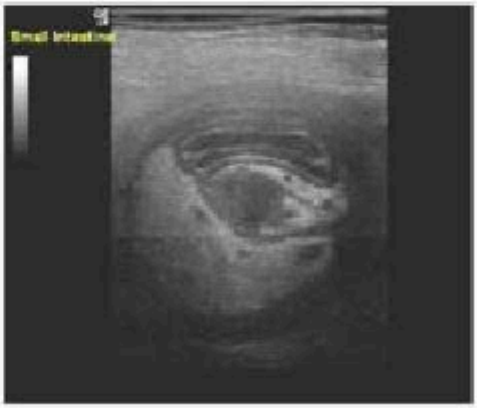
What if you can't tell?

- Wait, treat symptomatically, and take another series
- Barium
  - 5-7 ml/lb PO
  - Dog: 0, 0.25 -0.5, 1, 2, 3, 6 hrs
  - Cat: 0, 5, 15, 30, 60, 120 min
  - Problematic if perforation
- Iodinated contrast
  - 1 ml/lb
  - Radiographs Q 15 minutes
  - Typically takes about an hour
  - If perforation suspected or likely



Ultrasound vs. Radiology

- 82 dogs, 27 with confirmed foreign body
- Radiology produced a definitive result - 70% of the time
- Ultrasound produced a definitive result – 97% of the time
- 30% of obstructed dogs did not have radiographic evidence of obstruction, of which 50% were linear foreign bodies
- Jejunum >1.5 cm was a useful discriminatory finding



Sharma, VRU, 2011

Perioperative preparations

Timing of surgery?
Can this wait for tomorrow?

- Surgery <6 hours vs. surgery > 6 hours after presentation in 855 dogs
- Overall outcomes did not differ in immediate (584) vs. delayed (210)
- *Intestinal necrosis and perforation more common* in delayed cases.
- *Enterectomies more common* and duration of surgery and anesthesia longer in delayed cases.
- *Early surgery had earlier return to feeding and discharge.*
- ~5% of dogs had negative explore in both groups.

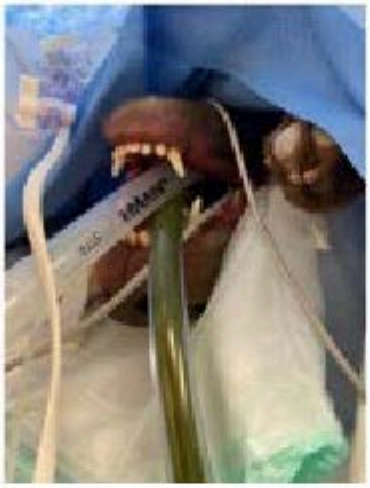
Maxwell, Vet Surgery, 2021

Anesthetic considerations

- Rehydrate prior to anesthesia
- Rapid induction
- Control and protect airway
- Ventilation maybe impaired if gastric distention
- Maropitant (Cerenia®) in the premeds (1 mg/kg)
- Regurgitation is common
- Consider an oro-gastric tube immediately after induction
- Perioperative antibiotics are indicated

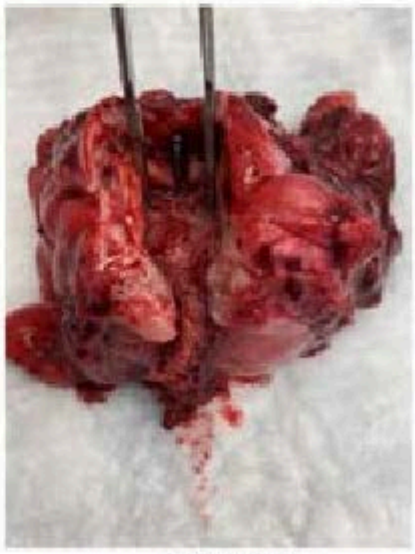
Orogastric tubes

- Measure to the last rib
- Mark the tube
- Gently feed down esophagus
- Twist or insufflate to get past LES
- Remove mouth from tube



Before you cut

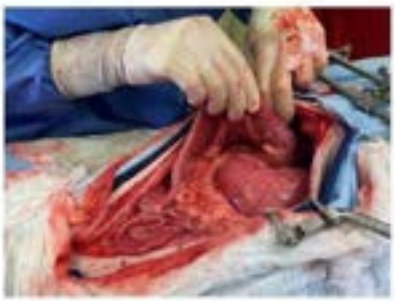
- Prep and drape more than you think you need
- Count your sponges
- Save clean instruments or have clean instruments for closure



Gossypiboma

Surgical exploration: incision length & retractors

- Do not cheat on exposure!
- Use Balfour retractors!
- Use Doyens!
- Complete abdominal explore!
  - ❖Satisfaction of search

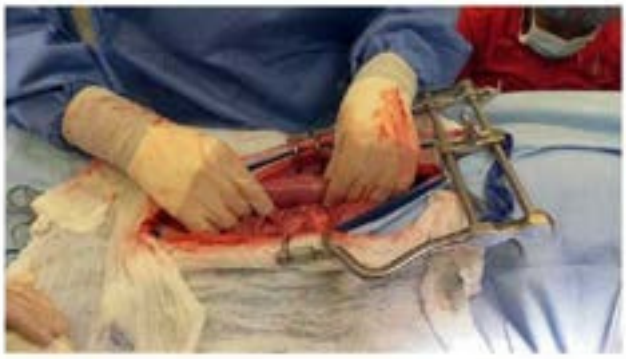


Can you milk it?

Into the stomach –
A gastrotomy is better than a enterotomy

Into the colon –
No need for anything

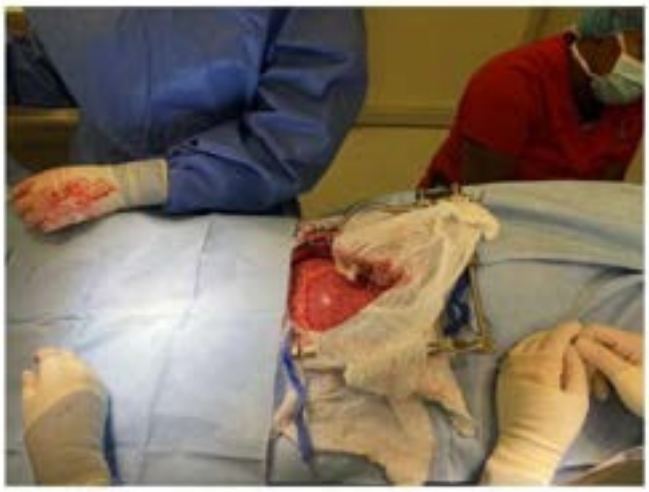
If moving foreign body will cause additional injury- don't do it



Specific surgical considerations: Stomach

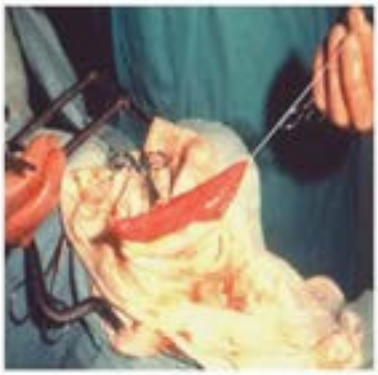
Gastrotomy

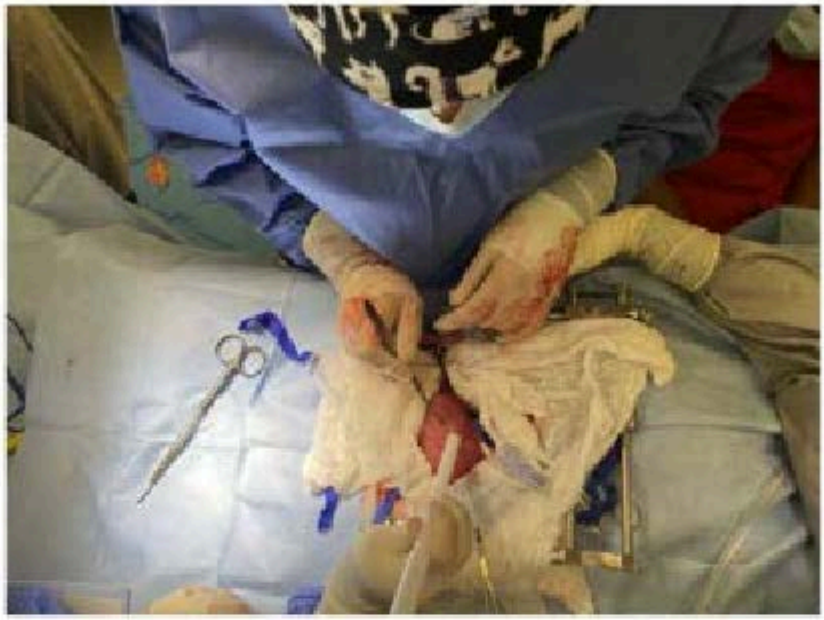
- Extend incision to xyphoid
- Pack it off
- Unable to completely isolate stomach outside of the abdomen
- Have a landing pad

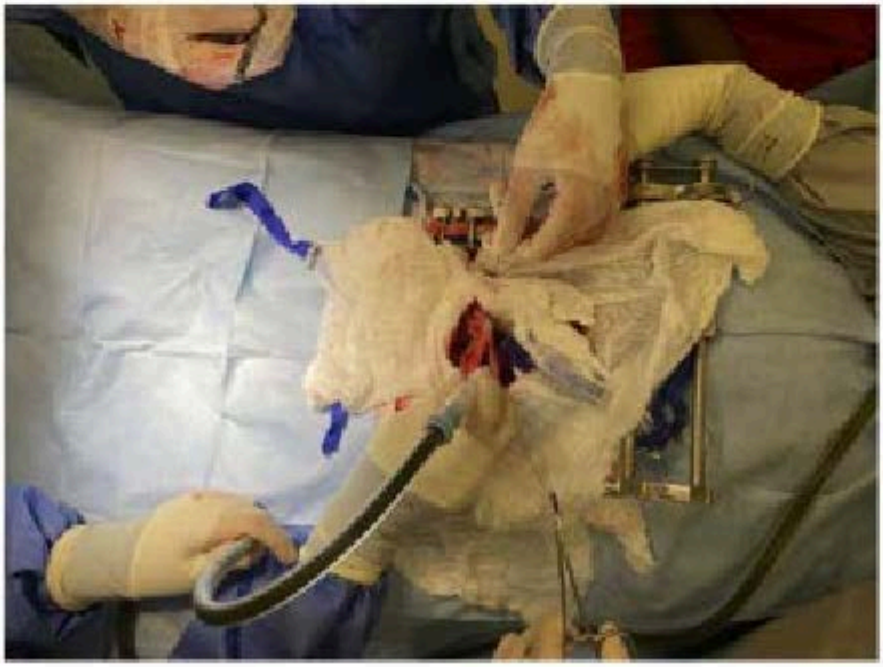


Gastrotomy

- Place stay sutures
- Stab incision into the body of the stomach
- Extend with scissors
- Between greater and lesser curvature

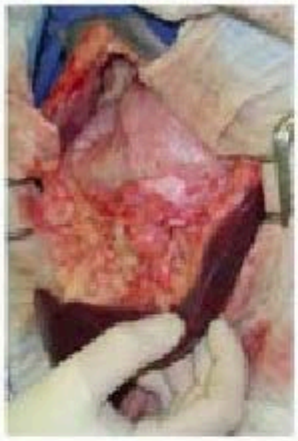


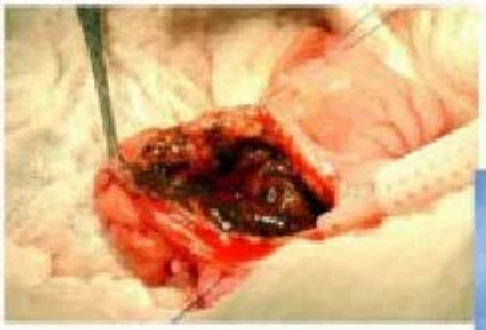


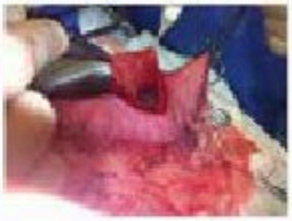


Make a big enough gastrotomy to do what you need to do.

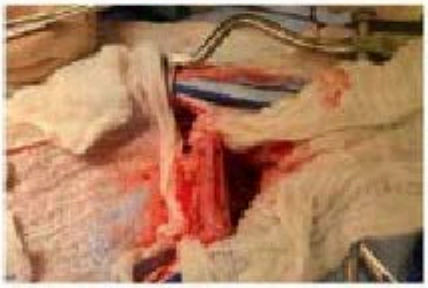
Occasionally, gastric ulcers can form with chronic or caustic foreign bodies

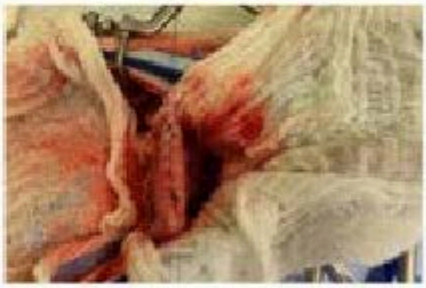




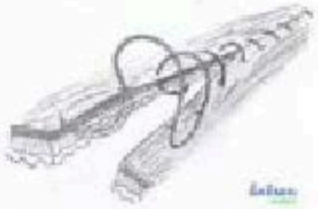


Closure





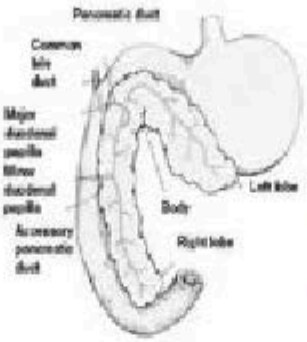
- 2 layers – appositional or inverting
- Hard to leak check
- Local/abdominal lavage

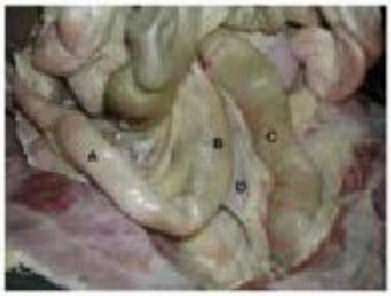


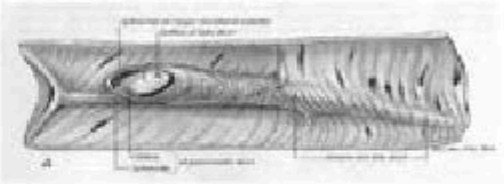
Specific surgical considerations: Duodenum

Problem Area

- Pylorus
- Common bile duct
- Pancreatic ducts
- Blood supply shared with pancreas
- Short mesentery
- Duodenal-colic ligament
- Potent secretions

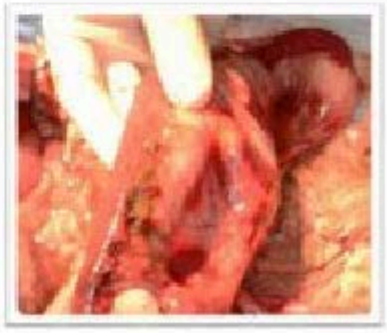






Strategies:

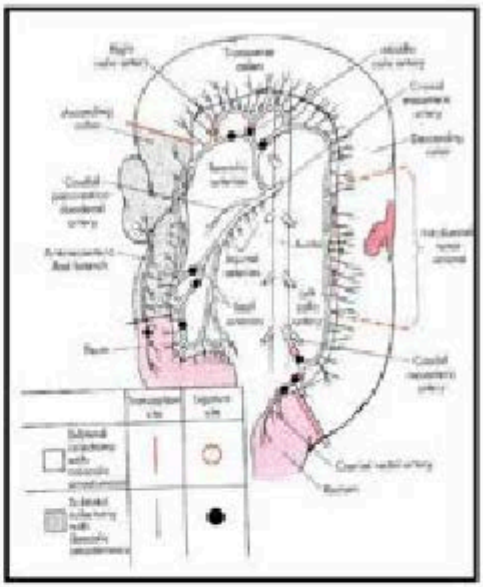
- Avoid if you can
- Milk into the stomach if possible
- Do not separate the pancreas from the duodenum
- Cannulate common bile duct
- Release the duodeno-colic ligament to improve exposure



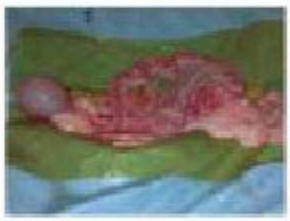
Specific surgical considerations: Colon

Problem area

- Rarely indicated to resect except for tumors or idiopathic megacolon
- Foreign material can be 'milked' out
- High bacterial load = high collagenase activity = prolonged healing
- Cannot be any tension on closure
- Poor blood supply
- High intraluminal pressure during defecation



Preservation of the Ileo-cecal-colic valve



- 166 cats, ICJ removed in about 25%
  - Median survival not reached – good long term survival
  - 77% of cats had good to excellent outcomes
  - 14% of cats died as a result of complications or treatment of megacolon, major complications in 10% of cats
  - Constipation recurrence occurred in 32% of cats at a median of 344 days
  - Not associated with removal or retention of the ICJ
  - Long term diarrhea associated with removal of the ICJ
  - Fair to poor outcome associated with removal of ICJ

Grossman, JAVMA, 2022

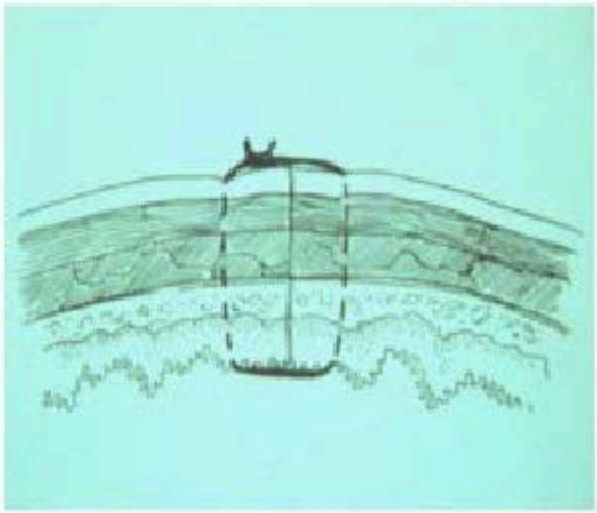
General Surgical Principles

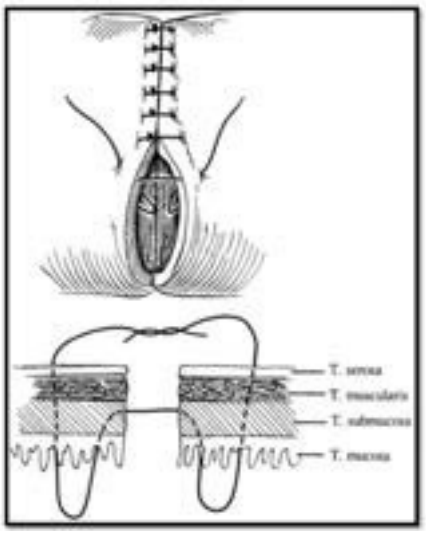
General Principles: Enterotomy

- Perform complete exploratory and palpate entire length of intestines
- Remove jejunum from abdomen
- Close Balfours to minimize contamination and preserve warmth
- Pack-off intestine with lap sponges
- Gently milk intestinal contents away from enterotomy site
- Handle gently with fingers or Doyen forceps

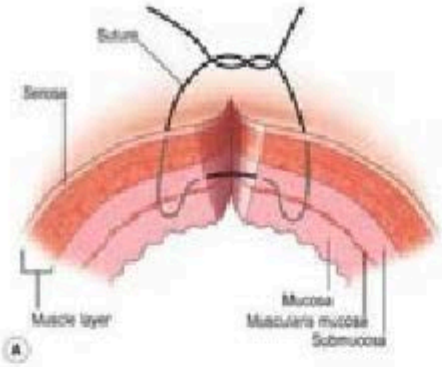
Closure

- Close with 3-0 or 4-0 monofilament, absorbable suture on taper needle
- Use SI or SC appositional pattern
- Avoid inverting or double layer closure
- Place sutures 4-5 mm away from edge and 3-4 mm apart

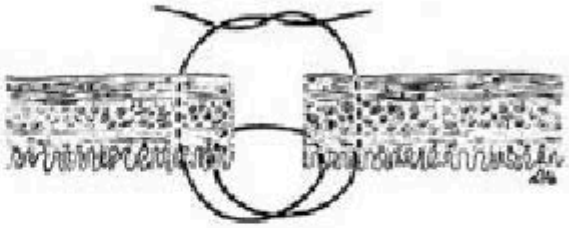




Gambee suture pattern helps to reduce mucosal eversion



Modified Gambee(s)



A Comparison of Ex Vivo Leak Pressures for Four Enterotomy Closures in a Canine Model

Nina R. Kisseb, DVM, DACVS-SA, DACVSMR, CCRT*, Alexander I. Kirsch, DVM, DACVS-SA†, Eric M. Zellner, DVM, DACVS-SA

Leak Pressure

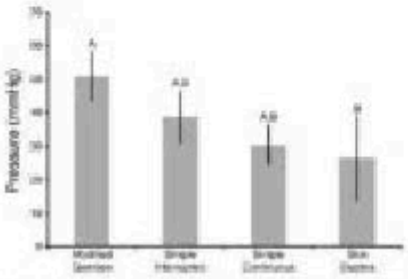


FIGURE 4 *Initial leak pressure (mm Hg) of the four enterotomy closures (mean ± SD). Means with common or shared letters indicate nonsignificant differences, whereas differing letters indicate significant differences (P < .05). SD, standard deviation.*

Time for placement

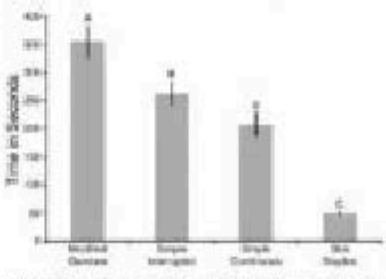


FIGURE 3 *Closure time in seconds for the four enterotomy closures (mean ± SD). Means with common or shared letters indicate nonsignificant differences, whereas differing letters indicate significant differences (P < .05). SD, standard deviation.*

J Am Anim Hosp Assoc 2018; 54:71–76. DOI: 10.5326/JAAHA-MS-6458

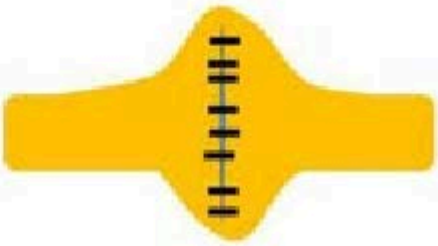
Preservation of lumen diameter

Classic anti-mesenteric enterotomy

- Reduces lumen diameter in small and medium size animals

Transverse Closure of a longitudinal incision

- Maintains diameter
- Creates dog ears



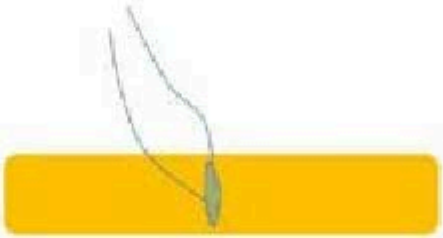
Transverse incision: Maintains lumen diameter

Negative Explore? It happens. Biopsy.

- Always biopsy on a 'negative' explore
- Biopsy multiple sites:
  - Gastric
  - Duodenal
  - Jejunal (proximal and distal)
  - Illeal
- Get full thickness samples
- Avoid crush artifact
- Don't forget lymph nodes

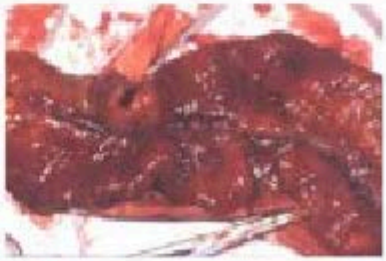
Intestinal Biopsy Technique

- Apply Doyen forceps or have an assistant hold off intestine
- Skin punch biopsy
- Stab incision with a 15 blade and scissors
- Suture technique



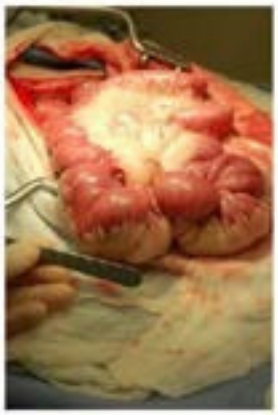
Linear Foreign Bodies

- Usually "anchor" under the tongue or at the pylorus
- Continued peristalsis causes sawing of the mesenteric border of the intestine
- Surgical treatment indicated



Linear vs. Non-linear Foreign Body

- 176 dogs linear vs. 323 non-linear
- LFB were more likely to have:
  - History of vomiting, anorexia, lethargy, pain on abdominal palpation
  - FB anchored in the stomach and extending into intestine
  - Intestinal necrosis, perforation, peritonitis
  - Require resection and anastomosis
  - Longer hospitalization
  - Increased cost of treatment
- Both groups have 96% survive to discharge

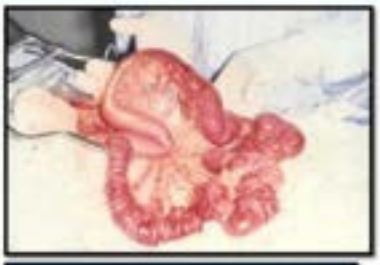


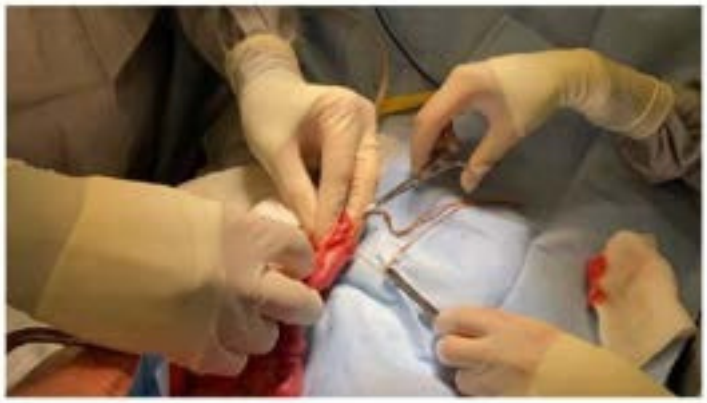
Journal of Small Animal Practice (2014) **55**, 560–565
DOI: 10.1111/jsap.12271

Linear Foreign Bodies

Strategy:

- Release anchor point with gastrotomy
- Remove remaining linear components through enterotomy (ies)





Linear Foreign Bodies

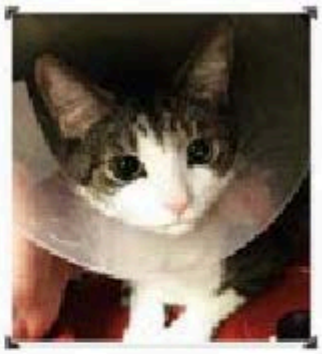
Alternate Strategy:

- Release anchor point with gastrotomy
- Tie foreign body to red rubber catheter and milk through intestines
- Better for cats than dogs with smaller, fine foreign bodies

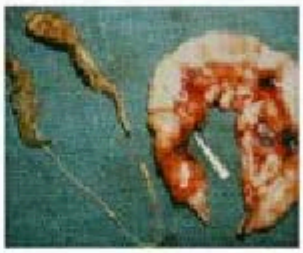
Not good for thicker linear foreign bodies

[illegible]

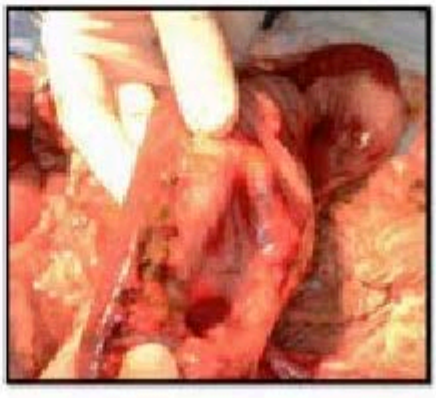
Conservative Management of Linear Foreign Bodies in Cats?



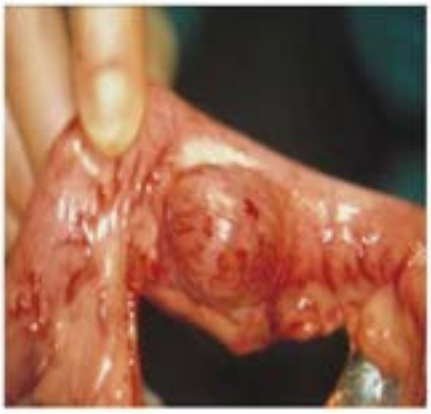
- About 50% will pass in 1 – 3 days if the anchor is released.
  - Study of 19 cats with LFB treated conservatively – 9 were successfully managed without surgery, 10 required surgery after conservative surgery failed. All cats survived.
- Those not passed will progress to septic peritonitis....
- Maybe an option for some owners

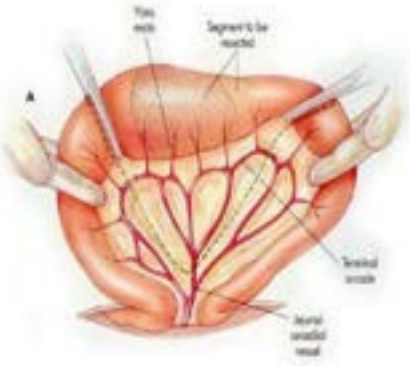


- Carefully inspect mesenteric border for pathology
- If perforation, culture, copious lavage, +/- open abdomen or abdominal drains

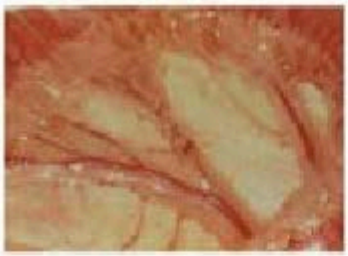


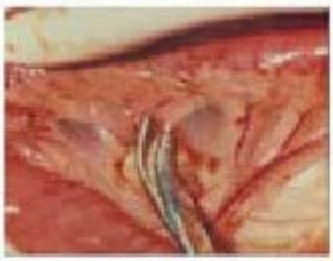
RESECTION-ANASTAMOSIS

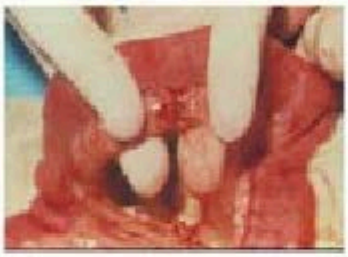


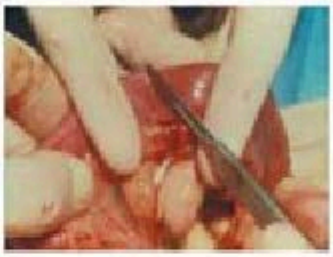


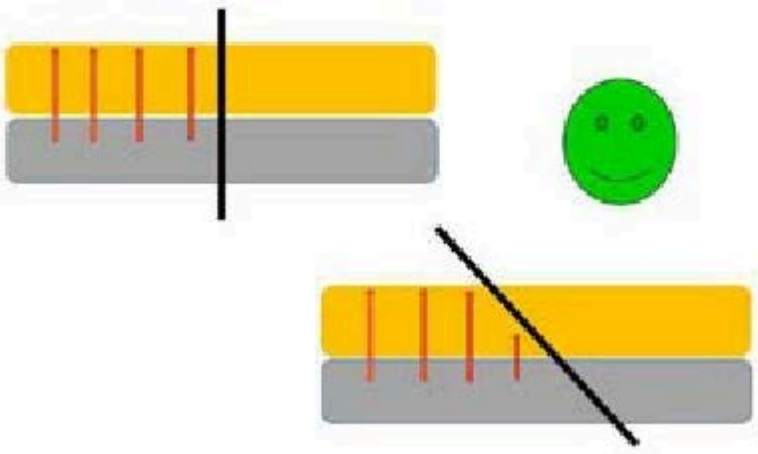
RESECTION-ANASTAMOSIS

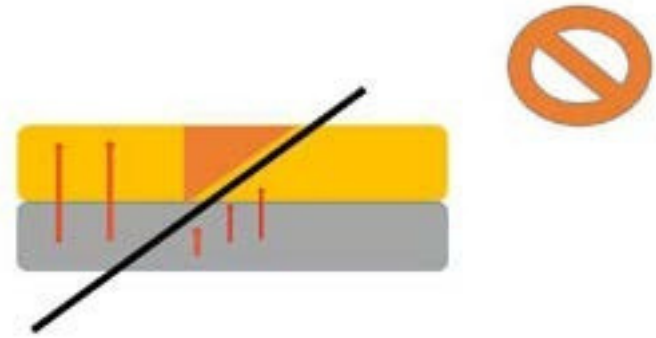






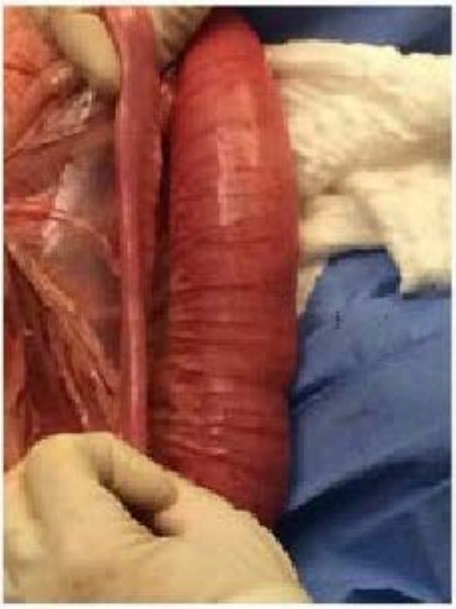


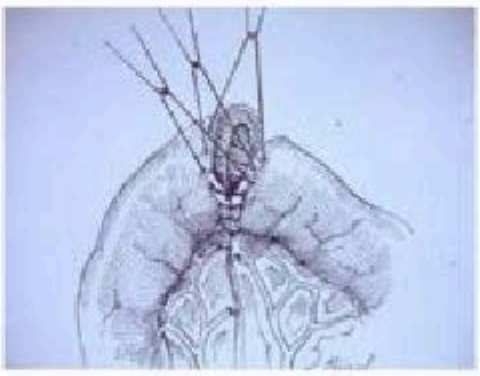




Management of Luminal Disparity

- Can be significant
- Chronic partial obstructions
- Can resect some oral intestine to a location with a more normal diameter
- Back cutting the antimesenteric surface
- Variable spacing of suture
- Partial over sew

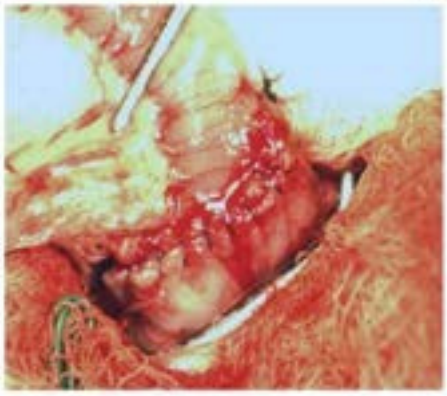




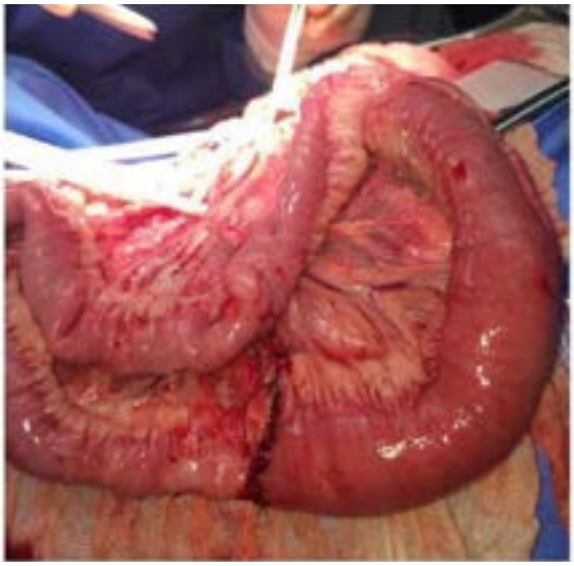
Disparity in luminal size can be accommodated by incising the smaller bowel at an oblique angle (left) or by placing sutures slightly further apart through the larger bowel (right).

Luminal Disparity:

Over sew



Danger!



Identification of risk factors for septic peritonitis and failure to survive following gastrointestinal surgery in dogs

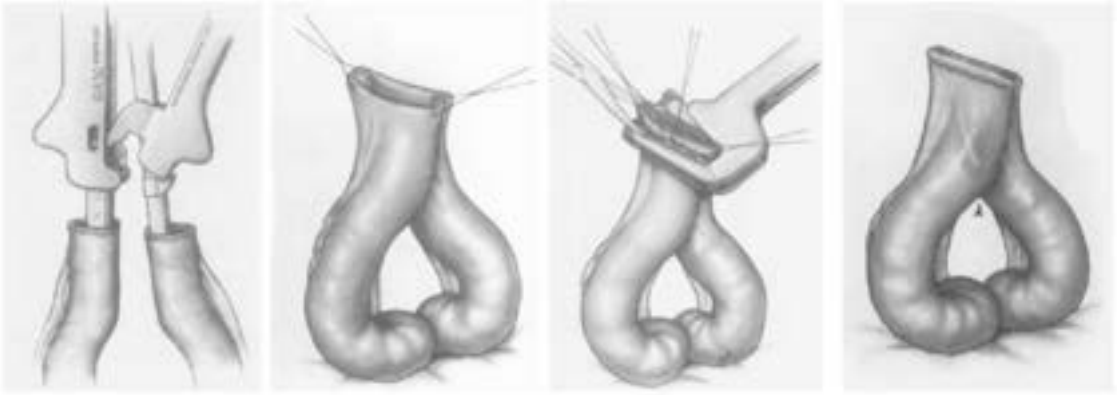
Janet A. Grimes, DVM, Chad W. Schmiedt, DVM, DACVS,
Karen K. Cornell, DVM, PhD, DACVS, MaryAnn G. Radlinsky, DVM, MS, DACVS

- 197 dogs with 225 surgeries
- 16% died prior to discharge
- 12% developed septic peritonitis
  - About 1/3 had continued peritonitis
  - About 1/3 of those dogs died
- 180 surgeries in dogs without septic peritonitis
  - 6% developed peritonitis

Risk factors for septic peritonitis

- Preoperative septic peritonitis
- Low serum albumin
- Low plasma proteins
- Intraoperative hypotension
- Foreign body was protective

Functional end to end stapled anastomosis (FEESA)



Why bother?

Frequency of Dehiscence in Hand-Sutured and Stapled Intestinal Anastomoses in Dogs

Significantly reduced surgery time

Technique	N	Dehiscence	Surgery Time
Hand sutured	142	21/134 (16%)	140 minutes
FEESA	72	8/71 (11%)	108 minutes
		P=0.389	p<0.001

Intra-abdominal complications following intestinal anastomoses by suture and staple techniques in dogs

Significantly lower dehiscence rates in dogs with stapled anastomosis vs. hand sutured technique

Technique	N	Dehiscence
Hand sutured	93	13%
FEESA	87	5%
		p=0.04

JAVMA 2018

Influence of preoperative septic peritonitis and anastomotic technique on the dehiscence of enterectomy sites in dogs: A retrospective review of 210 anastomoses

Daniel J. Davis, DVM | Ryan M. Doniniak, DVM, DACVS-SA | Jan Mosser, DVM | Maria Podniedlik, DVM | Joe Hoopman, DVM, DACVS-SA

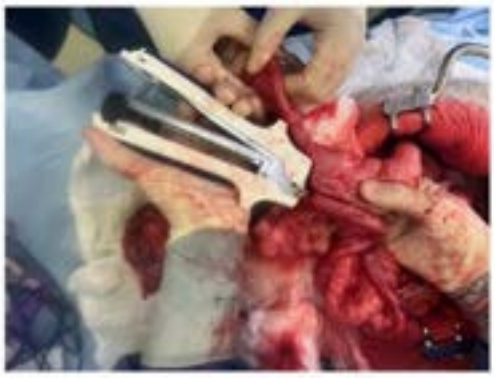
Significantly less risk of dehiscence in the face of septic peritonitis

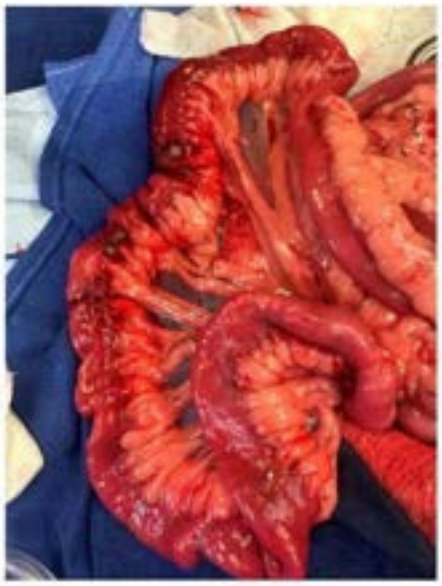
	WITHOUT Pre-op Septic Peritonitis		WITH Pre-op Septic Peritonitis	
	FEESA	Sutured	FEESA	Sutured
Dehiscence rate	4.2%	8.1%	9.7%	28.9%
	p = 0.38		P = 0.015	

**** When all dogs are considered together (n = 198) risk factors for dehiscence were preoperative septic peritonitis and hand sewn technique**

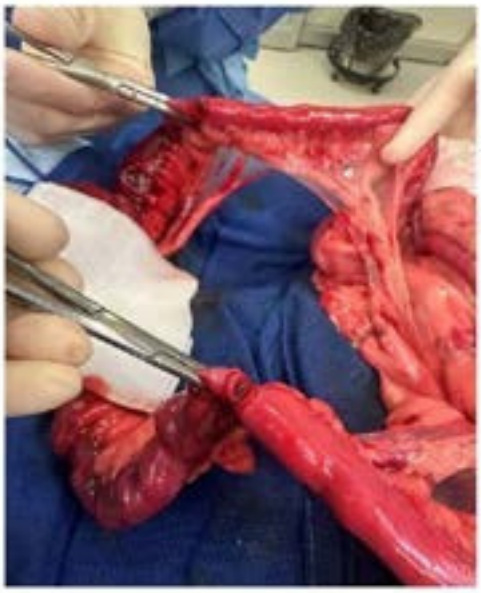
Vet Surg 2018

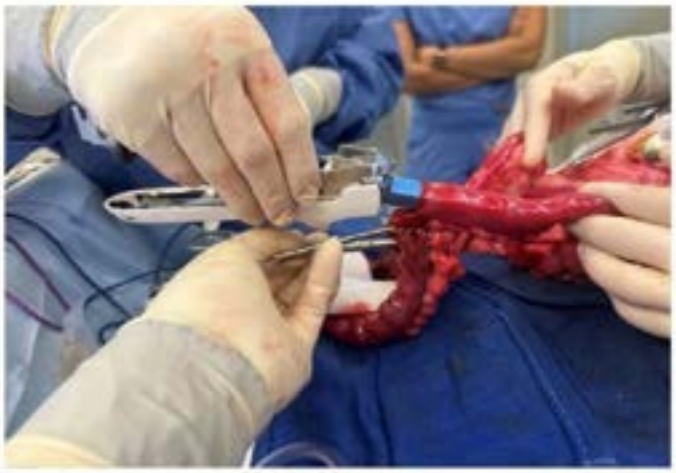
FEESA Technique

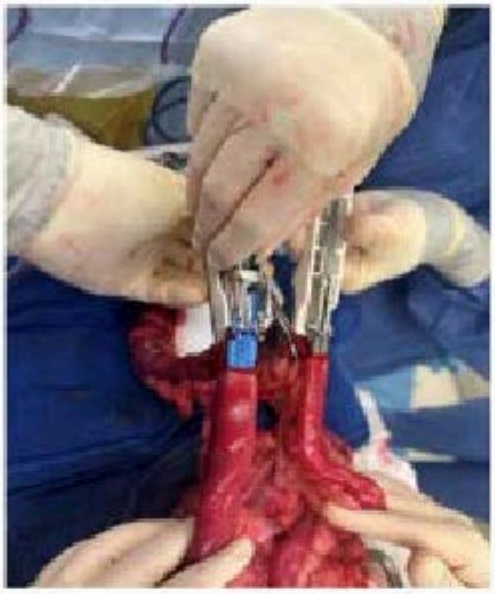


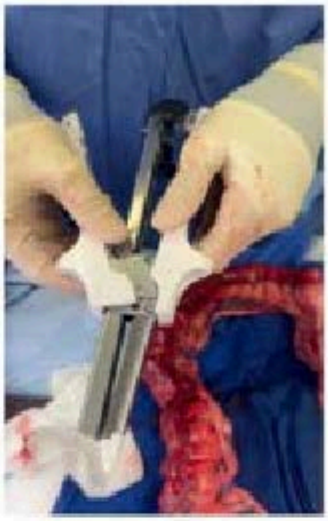


Aftermath of a linear foreign body

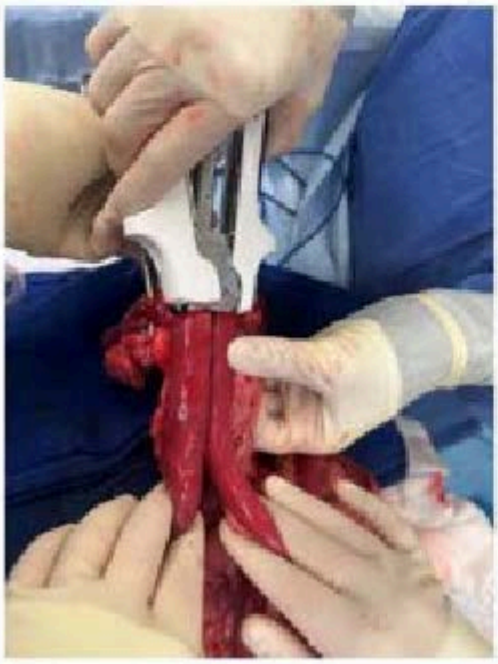






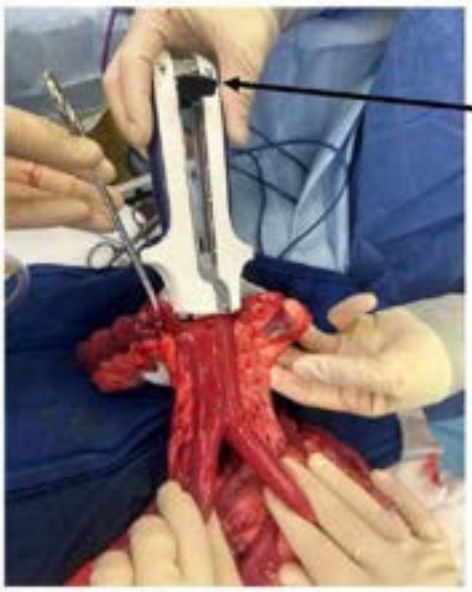


Assemble stapler



Antimesenteric surfaces are apposed
± Stay sutures can be useful

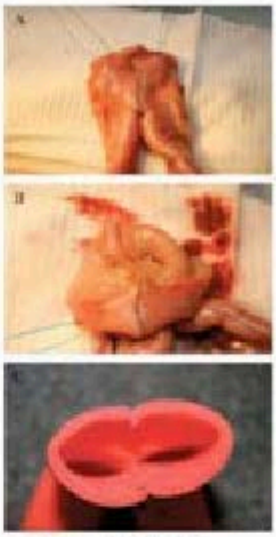
White, JSAP, 2008



Staple and blade control

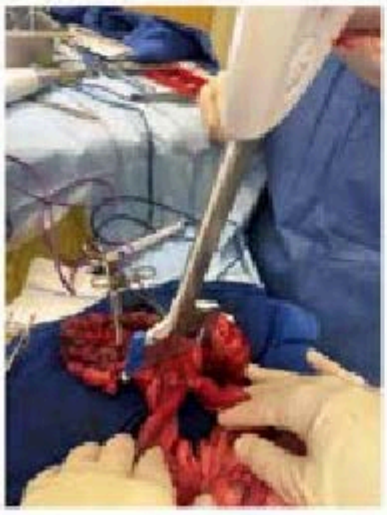
Stapler is closed

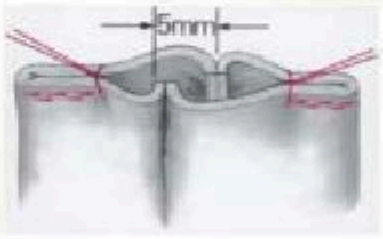
Blade is advanced

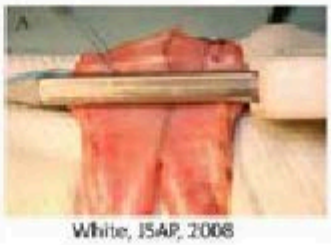


White, JSAP, 2008

GIA staple lines are offset prior to TA stapler

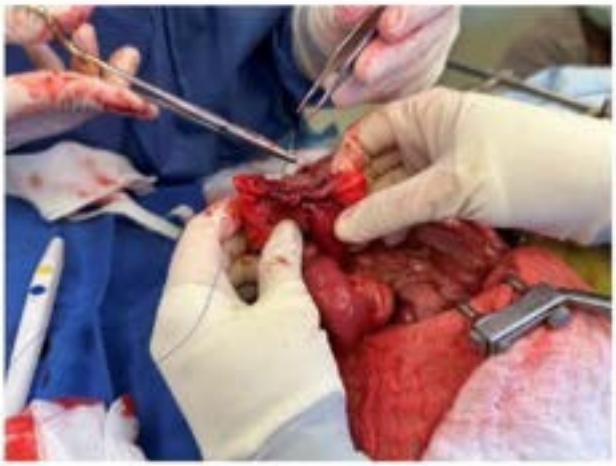




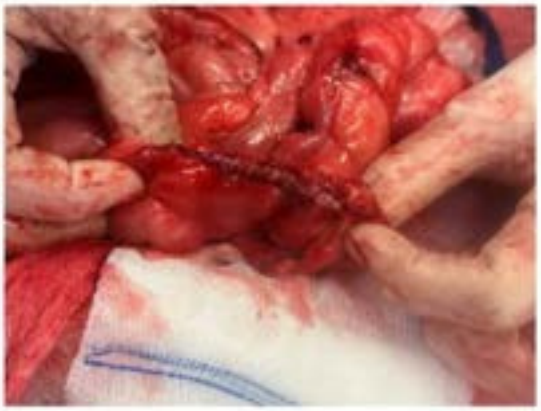


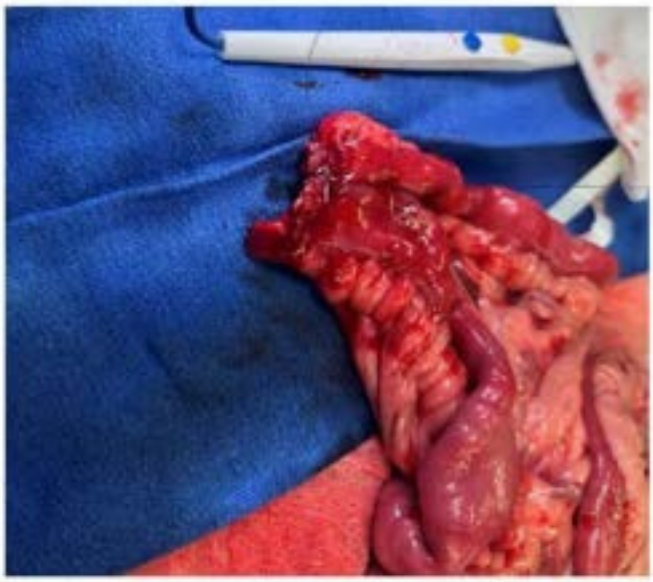
Can also use another GIA cartridge

White, JSAP, 2008

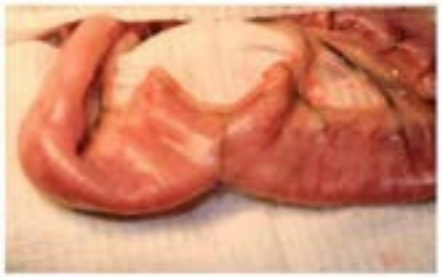


TA stapler line
is oversewn

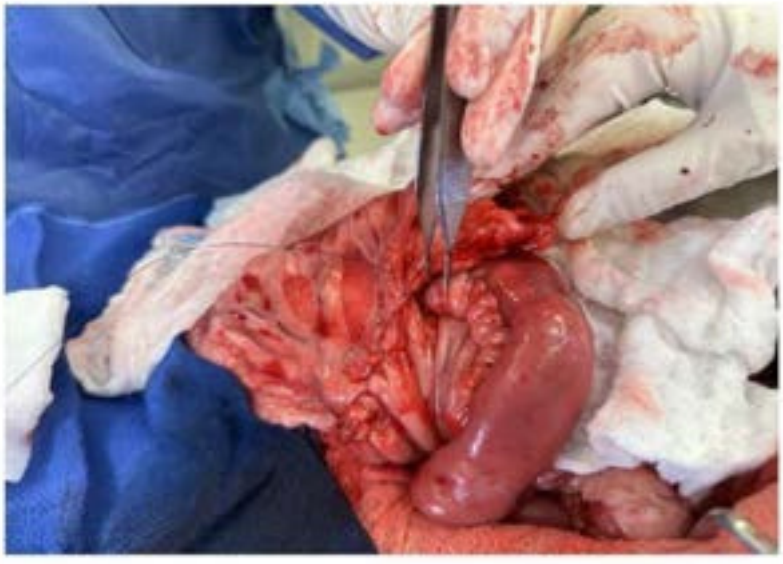




"Crotch" suture
reinforces staple line



White, JSAP, 2008



Mesentery
is closed

Cost Comparison

	Stapler (Infinity)	Stapler (Synergy Surgical (formerly suture.com)	Suture
Materials	$348 (1 new stapler = $248, 1 reload = box of 5 for $500)	$310 (1 new stapler = $145, 1 reload = (box of 6 for $989)	$30 (2 packs)
Time	$540 1.8 x $300/hr	$540 1.8 x $300/hr	$690 2.3 x $300/hr
Total	$888	$850	$720

Endo-GIA staplers

- Linear staple cutters
- Made for laparoscopic use
- Useful in open surgery for smaller dogs or cats

Stapled functional end-to-end intestinal anastomosis with endovascular gastrointestinal anastomosis staplers in cats and small dogs

Small dogs (n = 10) and cats (n = 15)
Median bodyweight = 4.6 kg
No major complications reported

POSTOPERATIVE CARE

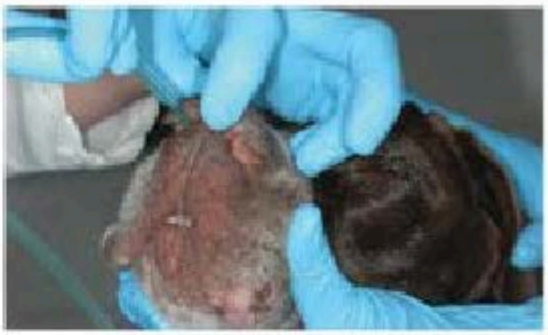
- Analgesia
- Feeding
- Antibiotics
- Prokinetics

Analgesia

- Local blocks can help (Nocita)
- NSAIDS contraindicated because of reduced GI mucosal blood flow
- Acetaminophen a good option for dogs
- Opiods are great, except...
  - Can cause nausea, ileus, reduced appetite
  - Methadone, Fentanyl
  - Switch to buprenorphine 1 day post operative
- Gabapentin
- Tramadol

Feeding post operative

- Enterocytes need the food in the gut to heal
- Feeding increases strength of anastomosis
- Feed when awake, typically that night or the next day
- Intraoperative NG tubes
- Perioperative esophagostomy tubes



Antibiotics

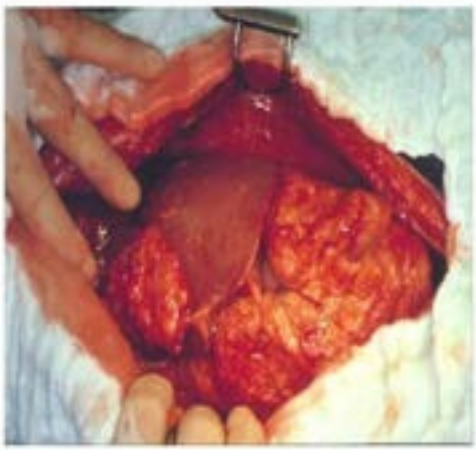
- Perioperative antibiotics are indicated
  - 30 minutes prior to incision
  - Q90 minutes after that
  - Cefazolin for most surgery (targeting G+ Staph and Strep)
  - Cefoxitin if colonic/rectal to increase anaerobic coverage
- Post operative antibiotics only indicated if contamination preop or intraop
  - Encourage resistance
  - Mask leakage

Prokinetics

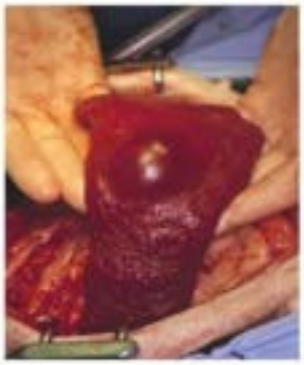
- Ileus can be a problem post op
- Worse in chronic obstructions, proximal (duodenal obstructions?), LFB
- NG Tube to reduce gastric residual volume
- Metoclopramide
- Lidocaine
- Ranitidine
- Erythromycin

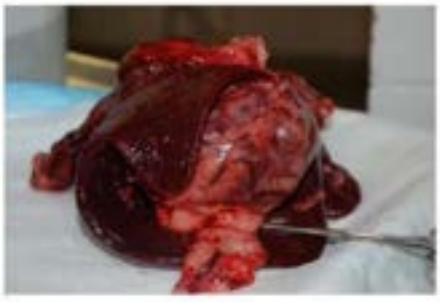
COMPLICATIONS

- Vomiting
- Dehiscence- most likely in the first 2-5 days
- Stricture
- Short bowel syndrome- greater than 80% of SI removed



Management of Hemoperitoneum and Principles of Splenic Surgery

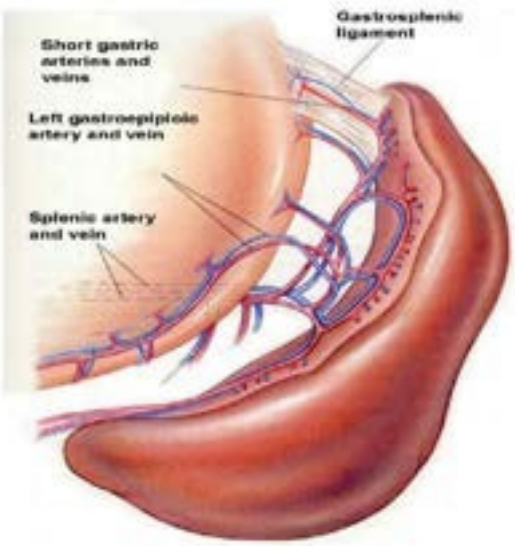


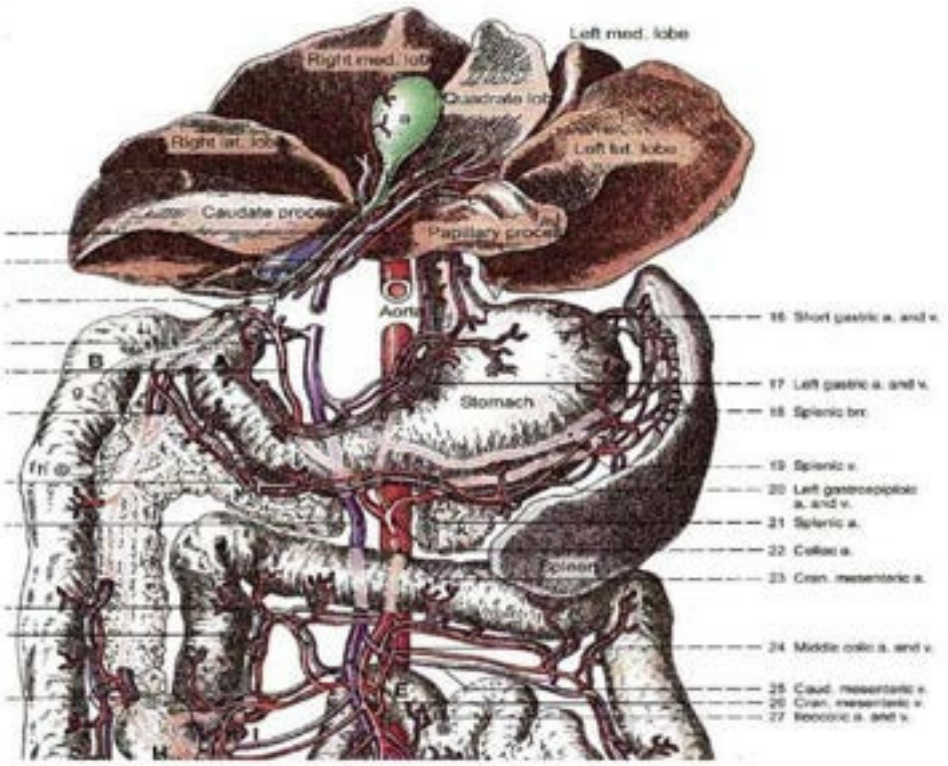


Chad Schmiedt DVM, DACVS
Professor, Small Animal Surgery
University of Georgia

SURGICAL ANATOMY

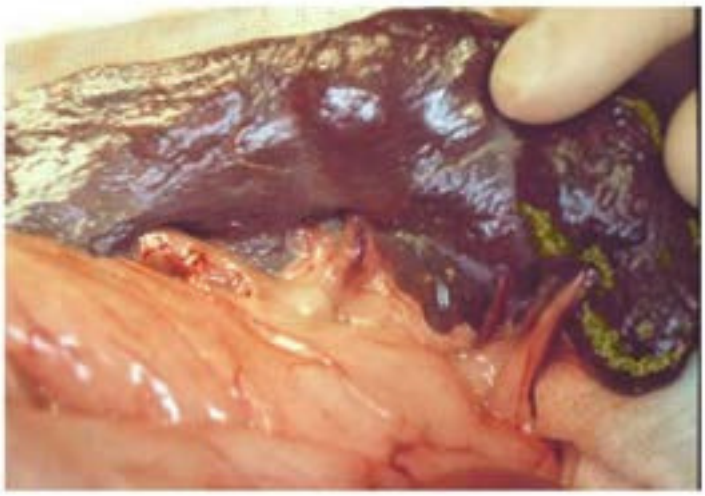
- Parenchymal organ
- Thin capsule of elastic fibers and smooth muscle
- Blood supply- splenic artery and vein
- Vagus and celiac nervous innervation





SURGICAL ANATOMY

- Splenosis, siderosis and hyperplastic nodules normal



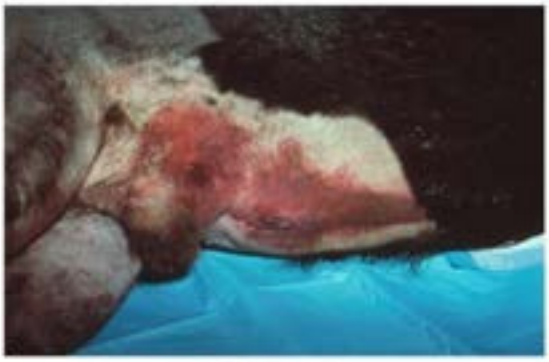
FUNCTION

- Blood storage –
  - Red pulp and venous sinuses
- Blood filter –
  - Damaged red cells are removed
  - Recticuloendothelial function
- Hematopoesis
- Iron Metabolism
- Immunoglobulin production – white pulp

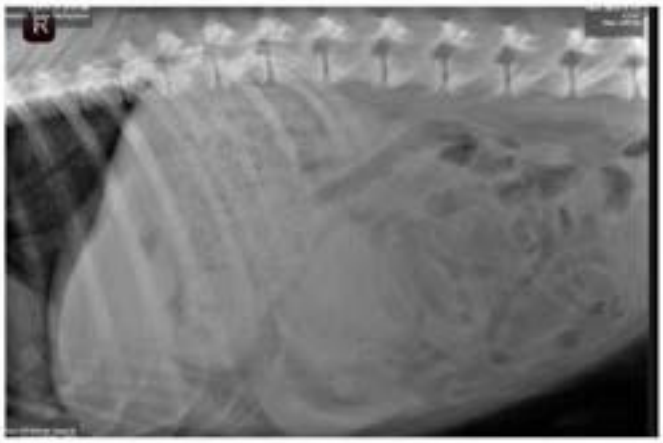
Physical Examination and Preoperative Work up

Clinical Signs of Splenic Disease

- Cranial abdominal mass
- Abdominal pain, distention
- Lethargy, weakness
- Hypotensive shock
- Anemia, thrombocytopenia, icterus
- Cardiac arrhythmias
- Coagulopathy
- GI - vomiting
- Nothing



SURVEY RADIOGRAPHS



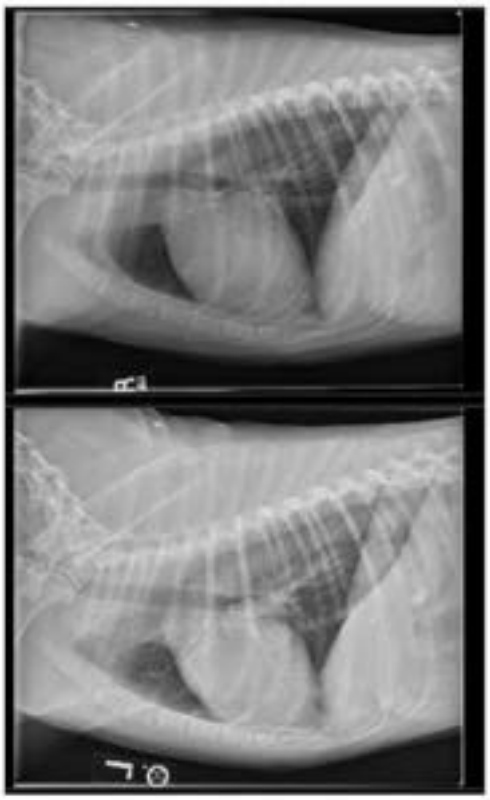
Can provide valuable screening information

Frequently omitted in favor of more sensitive imaging, particularly with emergent cases or with hemoperitoneum

Maybe difficult to identify origin of large mass

Thoracic Imaging

- Thoracic radiographs/CT to look for pulmonary metastasis
- Cardiac ultrasound to look for heart base or right atrial tumors that might be a source of a primary tumor



Should an echocardiogram always be part of your staging?

Metastatic pattern in dogs with splenic haemangiosarcoma: Clinical implications

D. J. WATERS, D. D. CAYWOOD, D. W. HAYDEN, and J. S. KLAUSNER

- Necropsy study of 25 dogs presenting for hemoperitoneum/splenic mass
- 6 dogs (25%) had right atrial HAS
- Most common metastatic sites in dogs liver, omentum, mesentery

J. small Anim. Pract. (1988) **29**, 805–814

Concurrent Splenic and Right Atrial Mass at Presentation in Dogs with HSA: A Retrospective Study

Sarah E. Boston, DVM, DVSc, DACVS, Geraldine Higginson, BSc, MSc, Gabrielle Monteith, BSc

- Group 1: 23 dogs with splenic HSA
  - 2 dogs (8.7%) had concurrent cardiac masses (both Goldens), neither had pericardial effusion, 7 dogs (30%) had distant metastasis
- Group 2: 31 dogs with cardiac HAS
  - 9 dogs (29%) had concurrent splenic HAS and 13 (42%) had distant metastasis
- Dogs with splenic HAS staged with abdominal US had a low incidence of concurrent cardiac mass

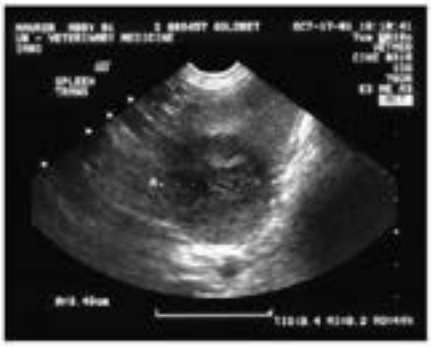
(*J Am Anim Hosp Assoc* 2011; 47:336-341. DOI 10.5326/JAAHA-MS-5603)

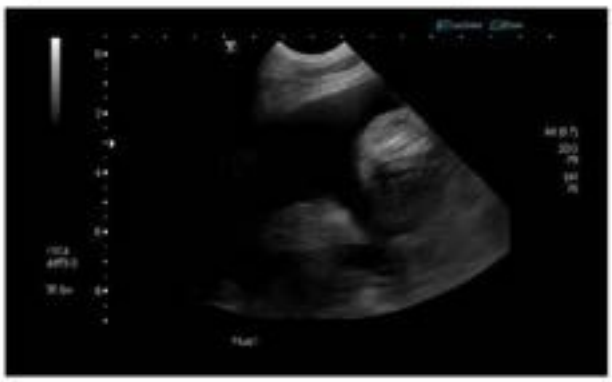
Concurrent Splenic and Right Atrial Mass at Presentation in Dogs with HSA: A Retrospective Study

Sarah E. Boston, DVM, DVSc, DACVS, Geraldine Higginson, BSc, MSc, Gabrielle Monteith, BSc

- Group 1: 23 dogs with splenic HSA
  - 2 dogs (8.7%) had concurrent cardiac masses (both Goldens), neither had pericardial effusion, 7 dogs (30%) had distant metastasis
- Group 2: 31 dogs with cardiac HAS
  - 9 dogs (29%) had concurrent splenic HAS and 13 (42%) had distant metastasis
- Dogs with splenic HAS staged with abdominal US had a low incidence of concurrent cardiac mass

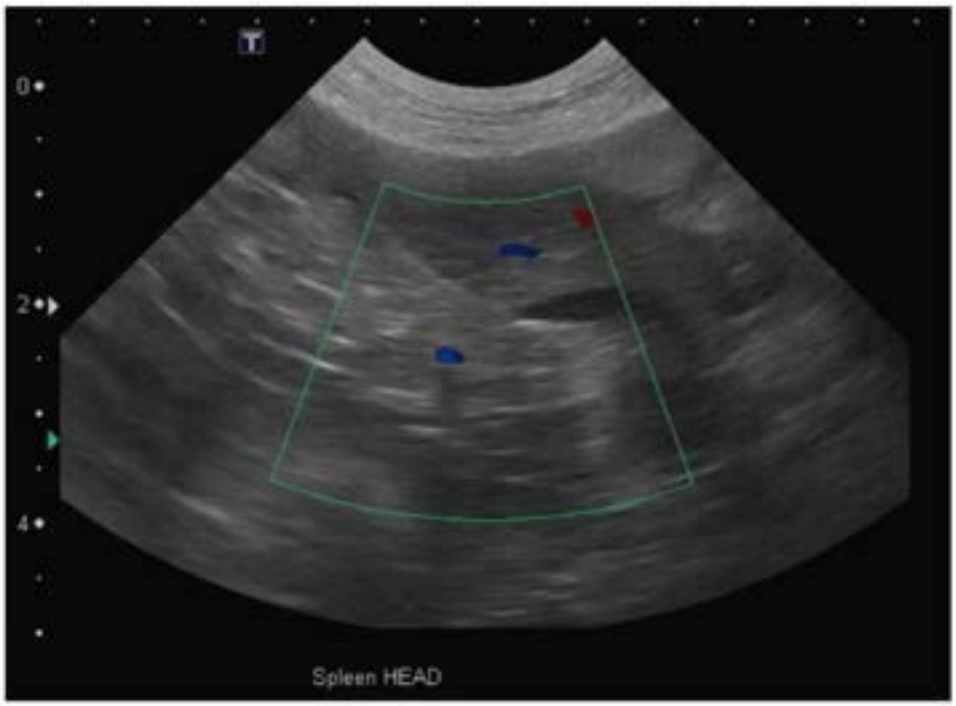
(*J Am Anim Hosp Assoc* 2011; 47:336-341. DOI 10.5326/JAAHA-MS-5603)



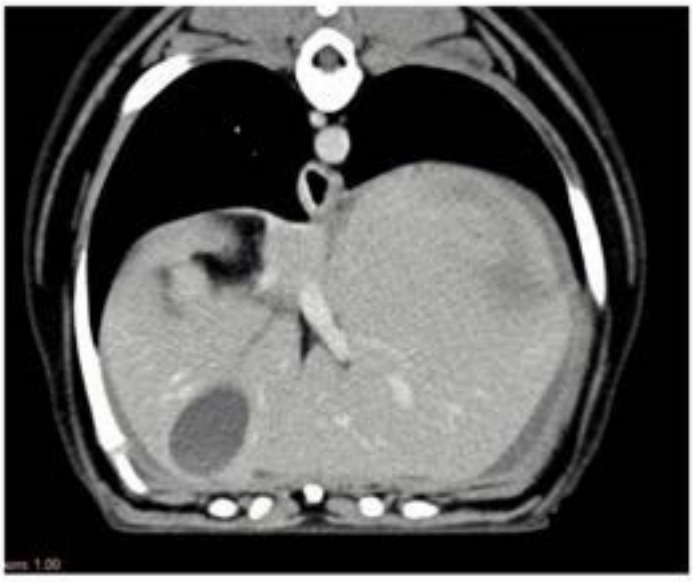


ULTRASOUND

- Blood flow?
- Evidence of lesions in other organs?
- Free abdominal fluid?
- Other abdominal concerns?



CT scan – greater sensitivity for metastatic lesions, can be easier in larger dogs

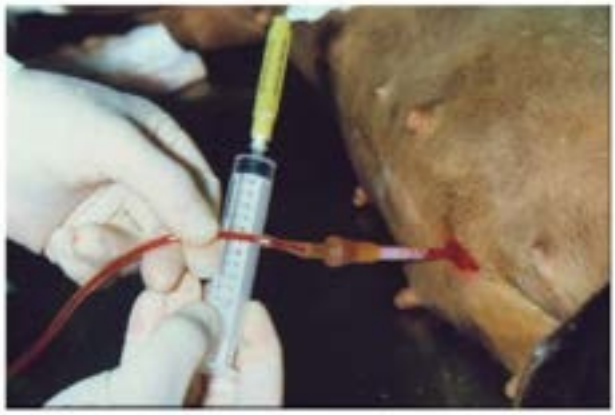


Abdominal US ~$350

Abdominal CT ~$600

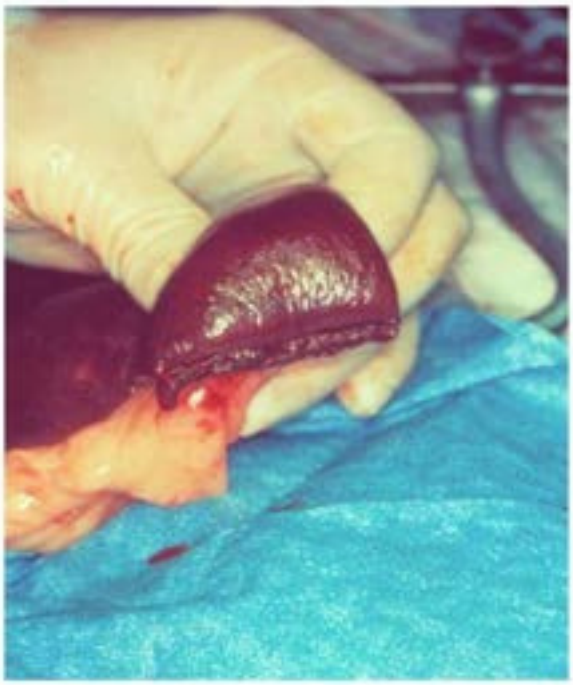
Abx and Tx CT ~$1200

Abdominocentesis



- Frequently US guided aspirates of abdominal fluid if present
- Measure PCV/TS
- Evaluate microscopically
- Can track overtime (every 1-2 hr) and compare to blood to determine bleeding activity

Splenic Aspirate or Surgical Biopsy



Splenic aspirate or biopsy are rarely performed as part of work up procedures in dogs.

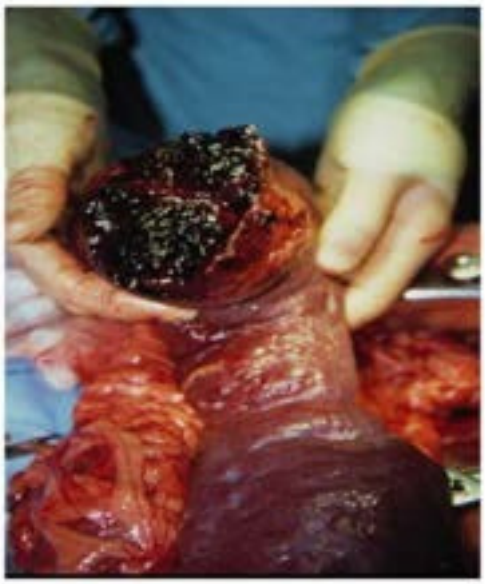
Preoperative Prognostication

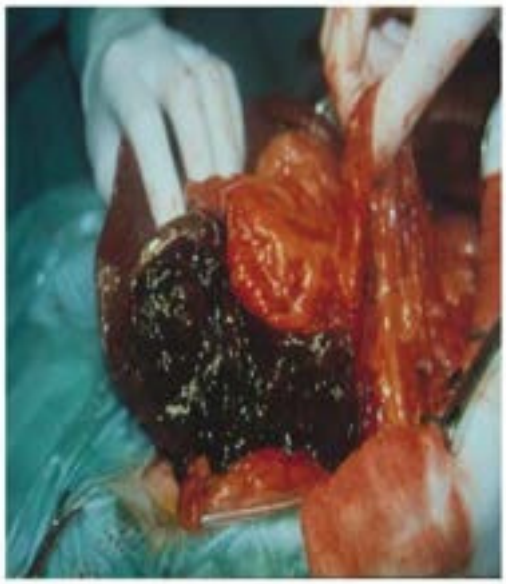
Dogs with hemoabdomen go from normal to abnormal very quickly, owners have little time to digest

Your dog has cancer, I'm sorry. But wait...

- Roughly 1/3 of splenic masses are **benign**.
  - Anecdotally, the larger masses are more likely benign. A small bleeding mass is more like HAS
- Most dogs survive surgery and those that do have an good quality of life (if its HAS though it is probably short).

What is this ??





General Statistics

Evaluation of the validity of the double two-thirds rule for diagnosing hemangiosarcoma in dogs with nontraumatic hemoperitoneum due to a ruptured splenic mass: a systematic review

Ashley R. Schick, DVM, and Janet A. Grimes, DVM, MS, DACVS-SA*

Department of Small Animal Medicine and Surgery, College of Veterinary Medicine, University of Georgia, Athens, GA

*Corresponding author: Dr. Grimes (jgrimes@uga.edu)

doi.org/10.2460/javma.22.08.0369

- "Double 2/3^{rds}" rule is reasonably accurate but likely an underestimation
- About 73% of splenic masses are neoplastic.
- About 87% of the neoplastic masses are hemangiosarcoma
- If a dog has a non-traumatic hemoabdomen with a splenic mass – about risk of neoplasia increases

Perioperative outcome in dogs with hemoperitoneum: 83 cases (2005–2010)

Cassie N. Lux, DVM; William T. N. Culp, VMD, DACVS; Philipp D. Mayhew, BVMS/MS, DACVS; Kim Tong, DVM; Robert B. Rebhun, DVM, DACVIM; Philip H. Kass, DVM, PhD, DACVMP

Objective—To characterize the clinical course of dogs with hemoperitoneum in the perioperative setting and to determine risk factors that may affect short-term outcome.

Design—Retrospective case series.

Animals—83 client-owned dogs.

Procedures—The medical records of dogs with hemoperitoneum that underwent surgery between 2005 and 2010 were reviewed. Data were analyzed to determine risk factors associated with perioperative outcome. The perioperative period was defined as the time from admission to the hospital for treatment of hemoperitoneum until the time of discharge or euthanasia (within the same visit).

Results—13 of 83 (16%) dogs died or were euthanized in the perioperative period. The median hospitalization time for surviving dogs was 2 days (range, 1 to 5 days). The requirement for a massive transfusion with blood products was a negative prognostic indicator for hospital discharge. The source of bleeding was isolated to the spleen in 78 of 83 (90%) dogs; a splenic source of hemorrhage was determined to be a positive predictor of survival to discharge from the hospital.

Conclusions and Clinical Relevance—In the present study, factors associated with death and failure to be discharged from the hospital included tachycardia, a requirement for massive transfusion with blood products, and the development of respiratory disease secondary to suspected pulmonary thromboembolism or acute respiratory distress syndrome. The presence of disease within the spleen was positively associated with survival to discharge. Surgical intervention for treatment of hemoperitoneum, regardless of etiology, resulted in discharge from the hospital for 70 of the 83 (84%) dogs in this series. (*J Am Vet Med Assoc* 2013;242:1385–1391)

Do the more abdominal lesions identified on ultrasound mean its more likely cancer? *Not really*

Prevalence of malignancy when solitary versus multiple lesions are detected during abdominal ultrasonographic examination of dogs with spontaneous hemoperitoneum: 31 cases (2003–2008)

Joshua G. Levinson, DVM, Jennifer L. Bouzna, VMD, DACVR, Gary C. Althouse, DVM, MS, PhD, DACT and Teresa M. Rieser, VMD, DACVECC

Spontaneous hemoabdomen, US-detected lesions

- 1 lesion = 80% malignant
- More than 1 lesion = 81% malignant

Journal of Veterinary Emergency and Critical Care **19**(5) 2009, pp 496–500
doi: 10.1111/j.1476-4431.2009.00466.x

Owner assessment of dogs' quality of life following treatment of neoplastic haemoperitoneum

June 2, 2012 | Veterinary Record

A. H. Crawford, M. S. Tivers,
S. E. Adamantos

The study found that despite a significant perioperative mortality, dogs treated surgically for neoplastic haemoabdomen encountered minimal serious perioperative complications, a short duration of hospitalisation and a rapid recovery postoperatively. Although surgically treated dogs had a short survival time, this was associated with a significant improvement in QOL and good owner satisfaction. Thus, surgical management can achieve temporary remission and give the owner valuable time with their pet, with a good short-term outcome and high owner satisfaction, despite a poor long-term prognosis.

Spontaneous hemoperitoneum in cats: 65 cases (1994–2006)

William T. N. Culp, VMD, DACVS; Chick Weisse, VMD, DACVS; Melissa E. Kellogg, DVM; Ira K. Gordon, DVM, DACVR; Dana L. Clarke, VMD; Lauren R. May, VMD, DACVS; Kenneth J. Drobatz, DVM, MSCE, DACVECC, DACVIM

Objective—To describe the clinical signs, physical examination findings, clinical laboratory abnormalities, etiology, and outcome in cats with spontaneous hemoperitoneum.

Design—Retrospective case series.

Animals—65 client-owned cats with spontaneous hemoperitoneum.

Procedures—Medical records of cats with spontaneous hemoperitoneum at 7 large referral clinics were reviewed. Cats were included if a definitive diagnosis of spontaneous hemoperitoneum could be obtained from review of the medical records.

Results—65 cats met inclusion criteria. The most common historical findings were lethargy, anorexia, and vomiting. Common findings on physical examination included inadequate hydration status and hypothermia. The most common clinicopathologic abnormalities were high serum AST activity, anemia, prolonged prothrombin time, and prolonged partial thromboplastin time. Forty-six percent (30/65) of cats had abdominal neoplasia, and 54% (35/65) had nonneoplastic conditions. Hemangiosarcoma was the most often diagnosed neoplasm (18/30; 60%), and the spleen was the most common location for neoplasia (11/30; 37%). Eight cats survived to be discharged from the hospital. Cats with neoplasia were significantly older and had significantly lower PCVs than cats with nonneoplastic disease.

Conclusions and Clinical Relevance—Spontaneous hemoperitoneum in cats often results in debilitating clinical consequences. In contrast to dogs with hemoperitoneum, the cause of hemoperitoneum in cats is approximately evenly distributed between neoplastic and nonneoplastic diseases. Although only a few cats were treated in this study, the prognosis appears poor. (*J Am Vet Med Assoc* 2010;236:978–982)

When to do surgery?
Is it elective, urgent, emergent?

- Just a mass?
- Actively bleeding mass?
- Had a bleed, but now stable?
- Actively bleeding, temporarily responsive to fluid resuscitation?
- Your schedule?
- Comfort level (Could it be a liver met bleeding)?
- No mass? Traumatic?

Preoperative Stabilization

Fluid resuscitation

Blood replacement

Fibrinolysis inhibitors

Monitoring

IV Fluid Resuscitation

- Shock Dose = 90 ml/kg,
  give 1/3 to 1/2 and reassess

- Limited volume resuscitation?
  - Hypertonic saline, hyperoncotic fluids (hetastarch)

A Pilot Comparison of Limited Versus Large Fluid Volume Resuscitation in Canine Spontaneous Hemoperitoneum

Tara N. Hammond, DVM, DACVECC, Jennifer L. Holm, DVM, DACVECC, Claire R. Sharp, BVMS, DACVECC

ABSTRACT

Treatment for hemorrhagic shock secondary to a spontaneous hemoperitoneum includes restoration of IV volume and surgical control of hemorrhage. This study was designed to determine if limited fluid volume resuscitation (LFVR) with hypertonic saline (HS) and hyperoncotic fluids (hydroxyethylstarch [HES]) results in more rapid cardiovascular stabilization in dogs with spontaneous hemoperitoneum versus conventional resuscitation (CR) with large volume resuscitation. Eighteen client-owned dogs presenting in hemorrhagic shock with a spontaneous hemoperitoneum were enrolled. Dogs were randomized to be fluid resuscitated with up to 90 mL/kg of an isotonic crystalloid (CR group) or up to 8 mL/kg of 7.2% Na chloride (i.e., HS) combined with up to 10 mL/kg of 6% HES. Measurements of vital signs, lactate, packed cell volume (PCV), total solids (TS), and blood pressure were made at standard time points. The primary end point was time to stabilization of hemodynamic parameters (measured in min). Dogs in the LFVR group achieved hemodynamic stabilization significantly faster (20 min; range, 10–25 min) than those in the CR group (35 min; range, 15–50 min; $P = .027$). Future studies are warranted to further investigate potential benefits associated with LFVR in dogs with spontaneous hemoperitoneum. *J Am Anim Hosp Assoc* 2014; 50:159–166. DOI 10.5326/JAAHA-MS-6086)

Autotransfusion?

- Perfectly fine for dogs with traumatic hemoperitoneum, hemothorax
- Be sure there is no possibility of sepsis
- Cancer?
  - Is there other blood available?
  - Will they die without it?
  - Not really any evidence that it reduces long term survival
  - Tumor has likely already spread

Autologous blood transfusion in dogs with thoracic or abdominal hemorrhage: 25 cases (2007–2012)

Veronica A. Higgs, DVM; Elke Rudloff, DVM, DACVECC; Rebecca Kirby, DVM, DACVIM, DACVECC and Andrew K.J. Linklater, DVM, DACVECC

Abstract

Objective – To describe the use and outcome following autologous blood transfusion (ABT) in dogs.
Design – Retrospective study (January 2007–July 2012).
Setting – Private veterinary referral center.
Animals – Twenty-five dogs that underwent ABT secondary to thoracic or abdominal hemorrhage.
Interventions – None.
Measurements and Main Results – The hospital transaction database was searched using the keyword "autotransfusion" from January 2007 to July 2012. Data collected included signalment, body weight, etiology of hemorrhage, source and method of collection, volumes and method of ABT administration, use of anticoagulant, reported complications, and outcome. Twenty-five dogs were included for a total of 27 ABTs. Causes of hemorrhage included vascular trauma (14/25 dogs, 56%), ruptured tumor (6/25, 22%), and coagulopathy attributed to brodifacoum toxicosis (3/25, 12%). Autologous blood was collected from the abdominal (19/25, 76%), thoracic (5/25, 20%), or abdominal and thoracic cavities (1/25, 4%). Anticoagulant was added to the ABT blood in 13 of 25 (52%) cases. A median ABT volume of 20.3 mL/kg (range 2.9–406.9 mL/kg) was infused through either a 250 μm blood administration filter (21/27, 78%) or an 18 μm hemostatic filter (6/27, 22%). Reported complications that may have been associated with ABT included hypocalcemia (4/17, 24%), hemolyzed serum (5/19, 26%), and prolonged coagulation times (4/5, 80%). These complications were considered of minimal clinical significance. Additional blood products were administered in 17 of 25 (68%) dogs. Seventeen (68%) dogs survived to discharge. Cause of death in the remaining cases was euthanasia or cardiac arrest secondary to uncontrollable hemorrhage.
Conclusions – ABT is an adjunct to volume replacement in dogs with thoracic or abdominal hemorrhage secondary to vascular trauma, ruptured tumor, or anticoagulant rodenticide toxicosis. ABT may be used as a bridge to definitive hemorrhage control, particularly when other blood products are not available or affordable. Complications may include hypocalcemia, prolonged coagulation times, and hemolysis.

(J Vet Emerg Crit Care 2015; 25(6): 733–740 doi: 10.1111/vec.12338)

Autologous Blood Collection/Administration

Collection:

1. Into syringe by butterfly catheter
2. Into catheter tip syringe
3. Pool suction tip into sterile container
4. ± anticoagulant

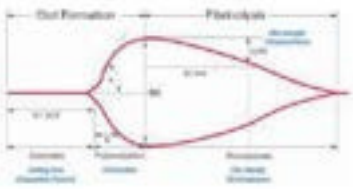
Transfer into separate sterile syringe for administration through blood administration filter

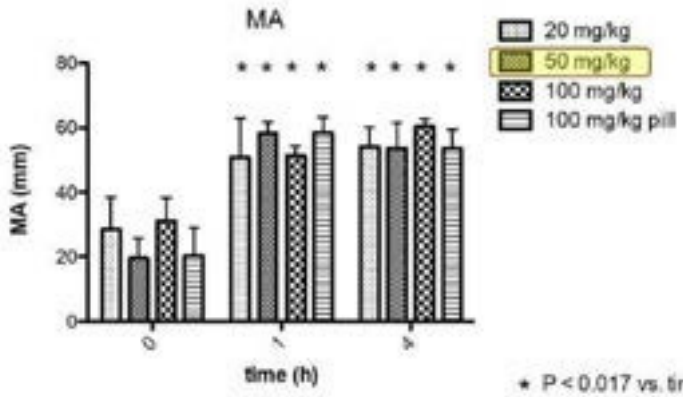
(J Vet Emerg Crit Care 2016; 26(6): 766–774 doi: 10.1111/vec.12476)

(J Vet Emerg Crit Care 2009; 19(5): 496–500 doi: 10.1111/j.1476-4431.2009.00466.x)

Fibrinolysis Inhibitors

- Aminocaproic acid, Tranexamic acid
- Stabilizes clot formation

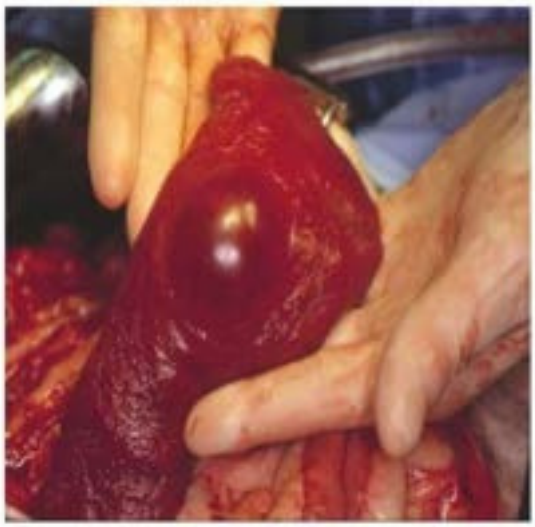




MA is "maximum amplitude" and is a measure of overall clot strength on a TEG

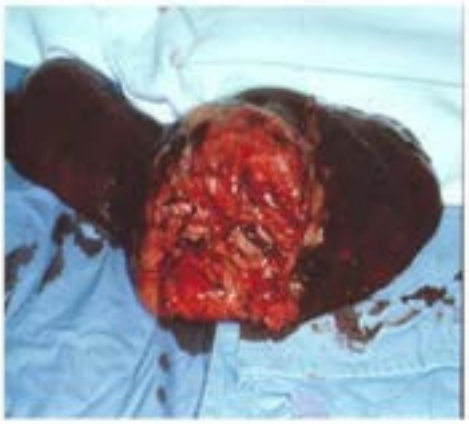
SURGICAL DISEASES

- Neoplasia
- Hematoma
- Traumatic rupture
- Torsion
- Abscess



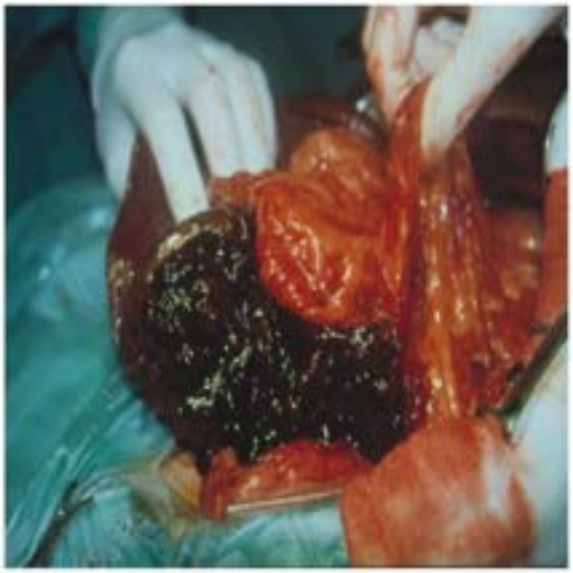
NEOPLASIA

- Hemangiosarcoma
- Hemangioma
- Mast cell tumor
- Lymphosarcoma
- Histiocytoma
- Sarcoma



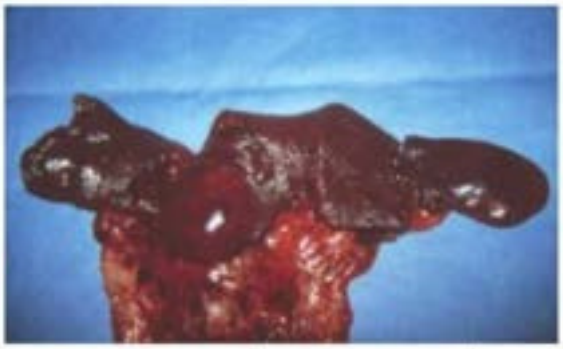
HEMATOMA

- **May cause very similar clinical signs as a neoplastic mass**
- **Gross appearance may be the *same* as a neoplastic mass**
- **Histopathology necessary to determine the difference**
- **Provide pathology with large tissue sample**



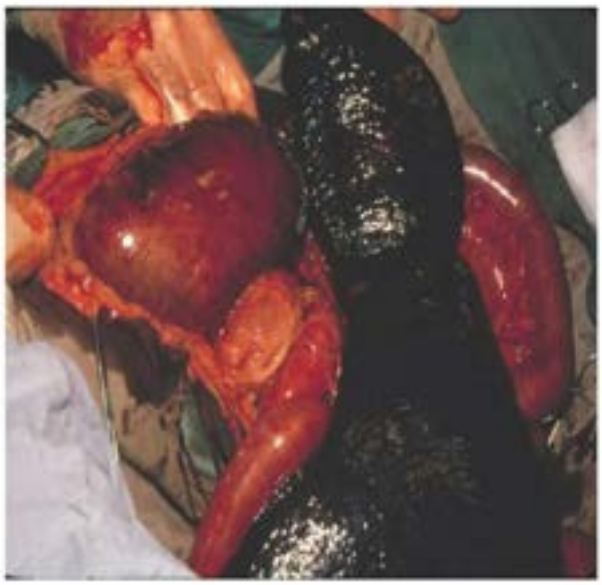
TRAUMATIC RUPTURE

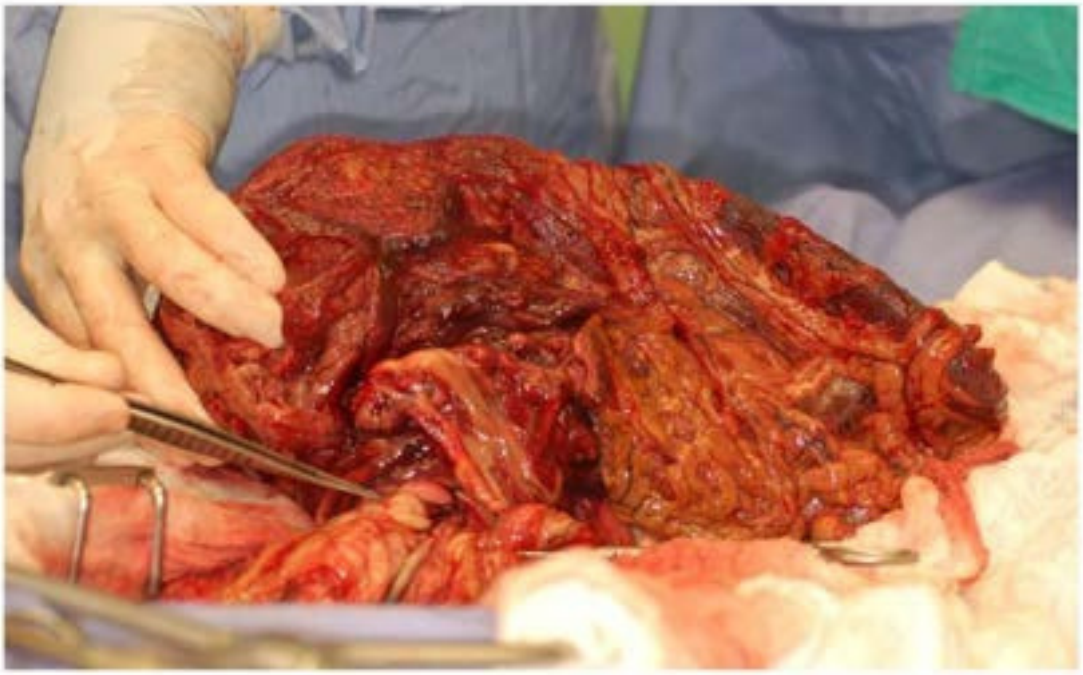
- **Usually secondary to severe blunt trauma**
- **Hemorrhage can be significant but usually stops without surgical intervention**
- **Evaluate spleen**
- **Suture lacerations or remove all or portions that are devitalized**

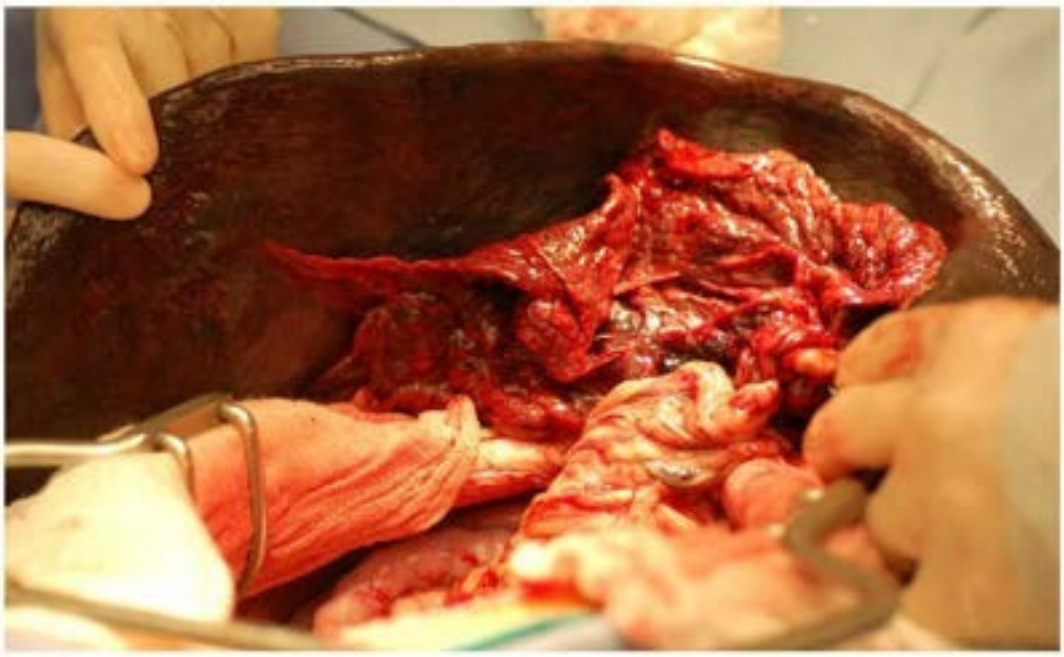


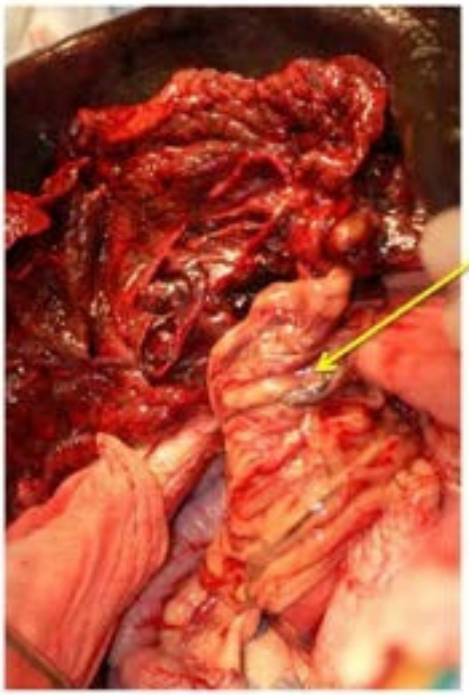
TORSION

- May be spontaneous or secondary to GDV
- Occlusion of vessels leading to ischemia and necrosis
- Generally requires splenectomy unless diagnosed early and spleen still viable and can be de-rotated



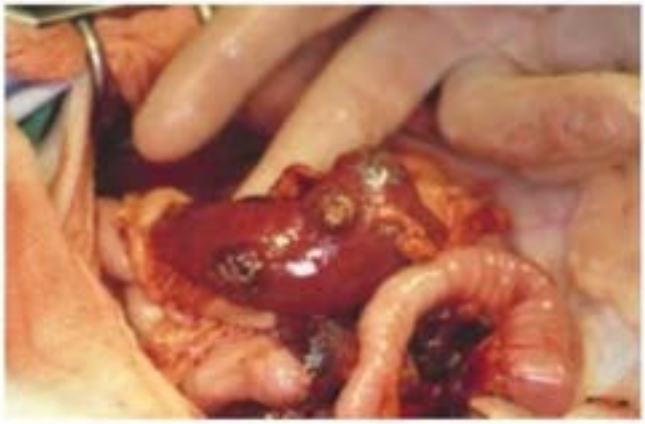






ABSCESS

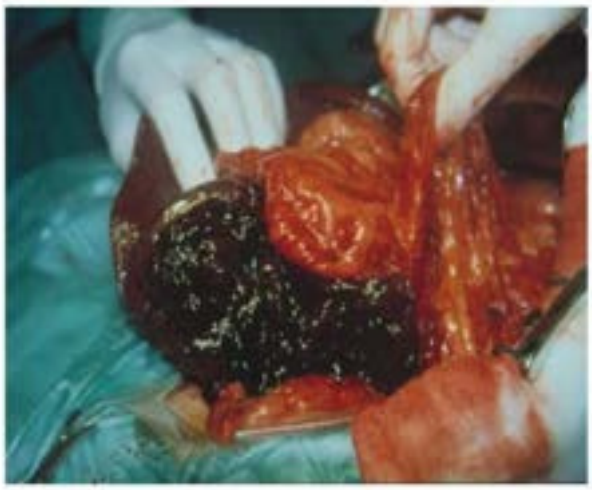
- Vague clinical signs
- Hematogenous or direct spread of bacteria
- Diagnose with US and FNA
- Splenectomy
- Culture and continue systemic antibiotics



Splenic Surgery

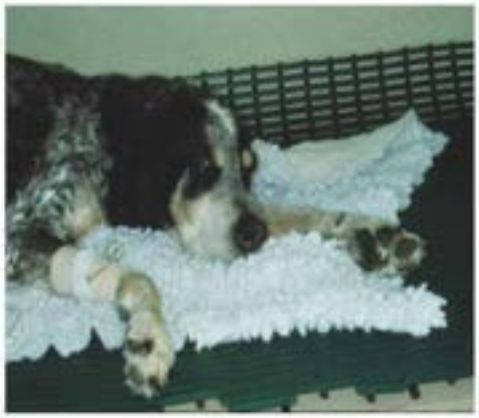
SURGICAL PRINCIPLES

- Handle spleen carefully to avoid rupture
- Have plenty of hemostats available – use them on the spleen side or both sides if active bleeding
- Perform a gastropexy
- Provide multiple tissue samples for biopsy



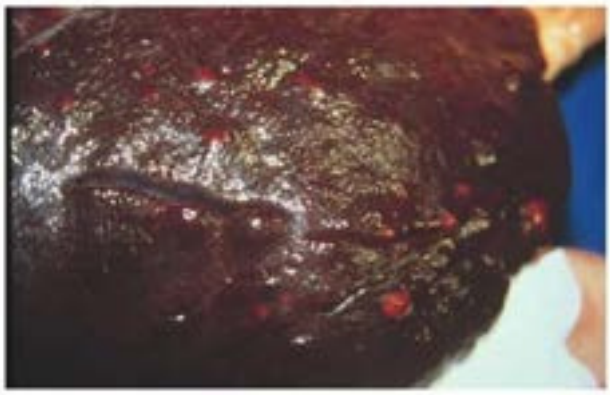
SURGICAL PRINCIPLES

- Make adequate size laparotomy for thorough exploratory
- Have suction to remove hemorrhage
- Pedicle-ize and remove omental adhesions
- Stay close to the spleen



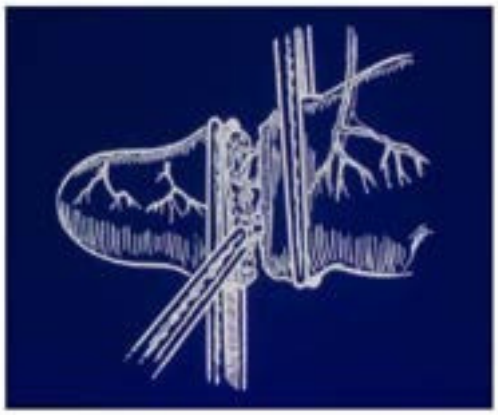
SURGICAL PROCEDURES

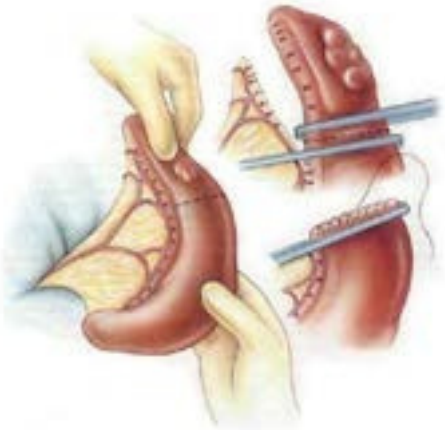
- Partial splenectomy
- Total splenectomy



PARTIAL SPLENECTOMY

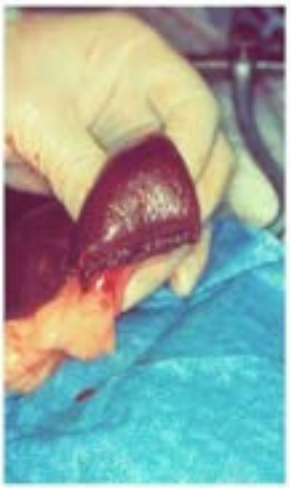
- Double ligate vessels that supply portion of spleen to be removed
- Gently compress spleen before clamping
- Transect spleen and and oversew cut edge with two continuous rows
- Use 3-0 or 4-0 absorbable suture on taper needle
- Monitor suture line for bleeding





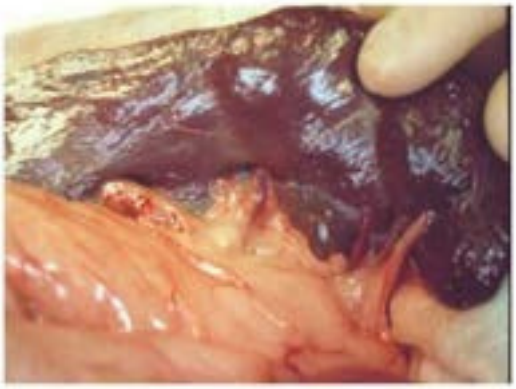
PARTIAL SPLENECTOMY

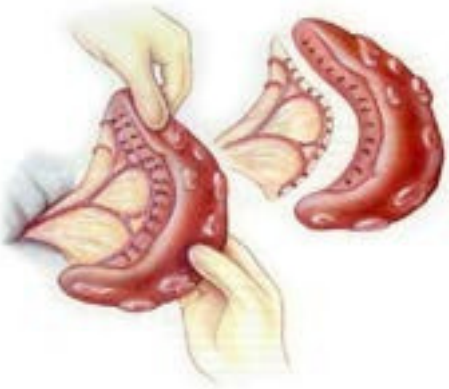
- Automatic stapling devices can be used for partial splenectomy
- Two staggered row of staples are placed
- Must ensure that the tissue is not too thick and staples are secure



SPLENECTOMY

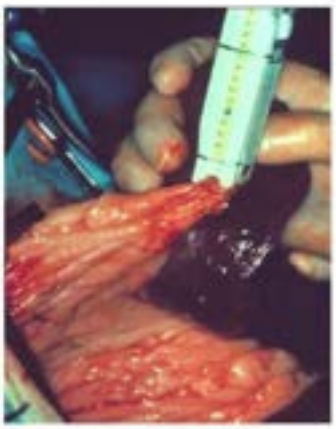
- Isolate spleen and identify vascular pedicle
- Pack-off spleen with moist lap sponges
- Double ligate vessels with absorbable or nonabsorbable material



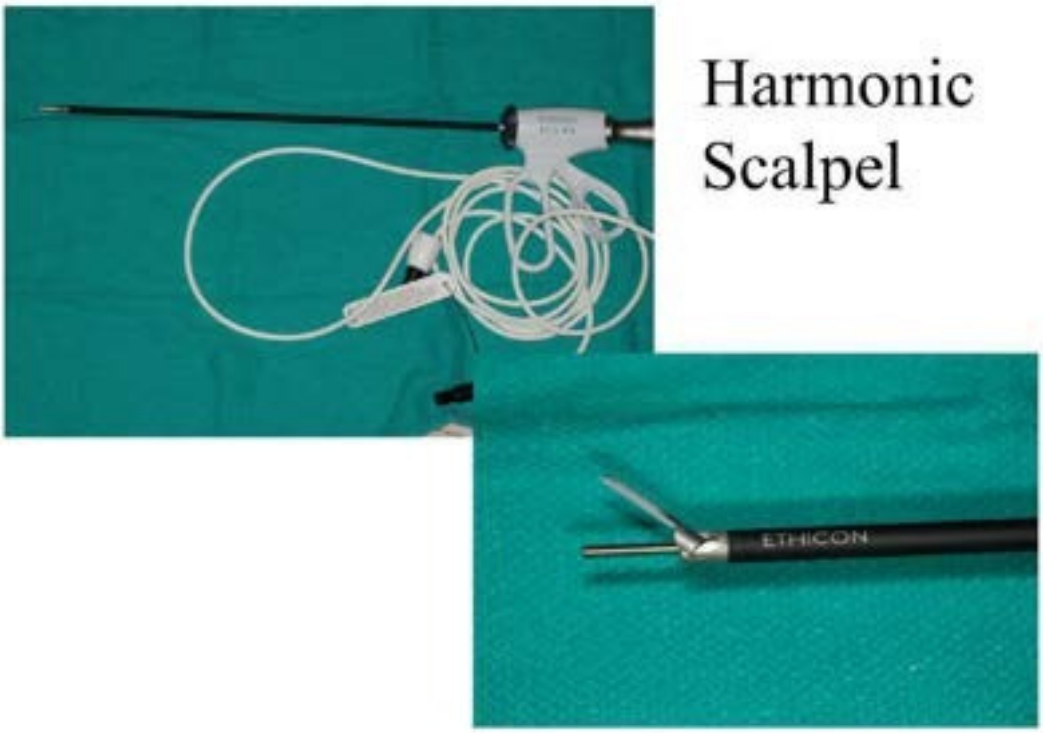


SPLENECTOMY

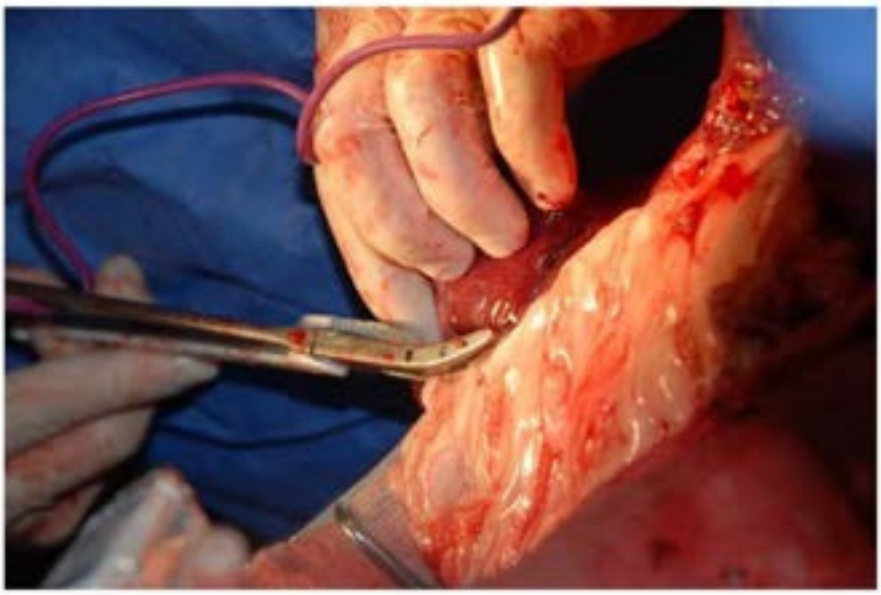
- **Hemoclips may be used to save time but must be secure**
- **LDS stapling device (Ligate – Divide – Staple)**
- **LigaSure or Harmonic Scalpel**

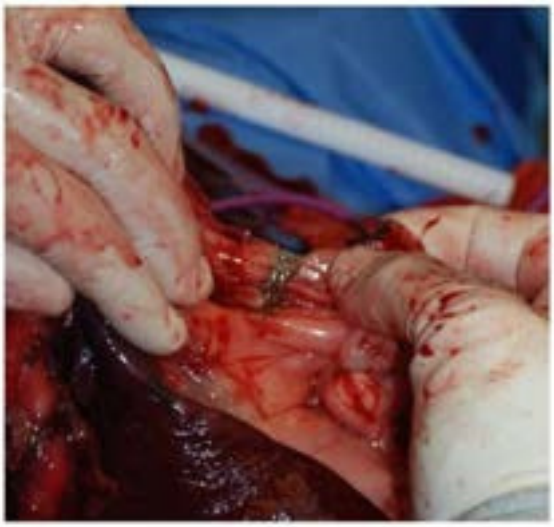


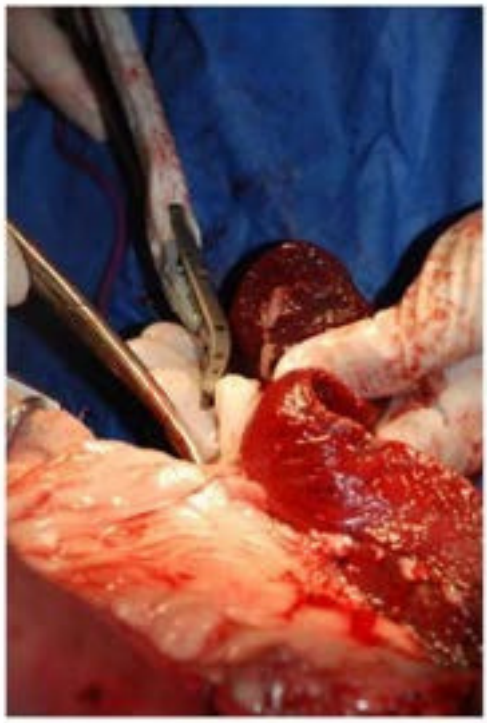
Harmonic Scalpel

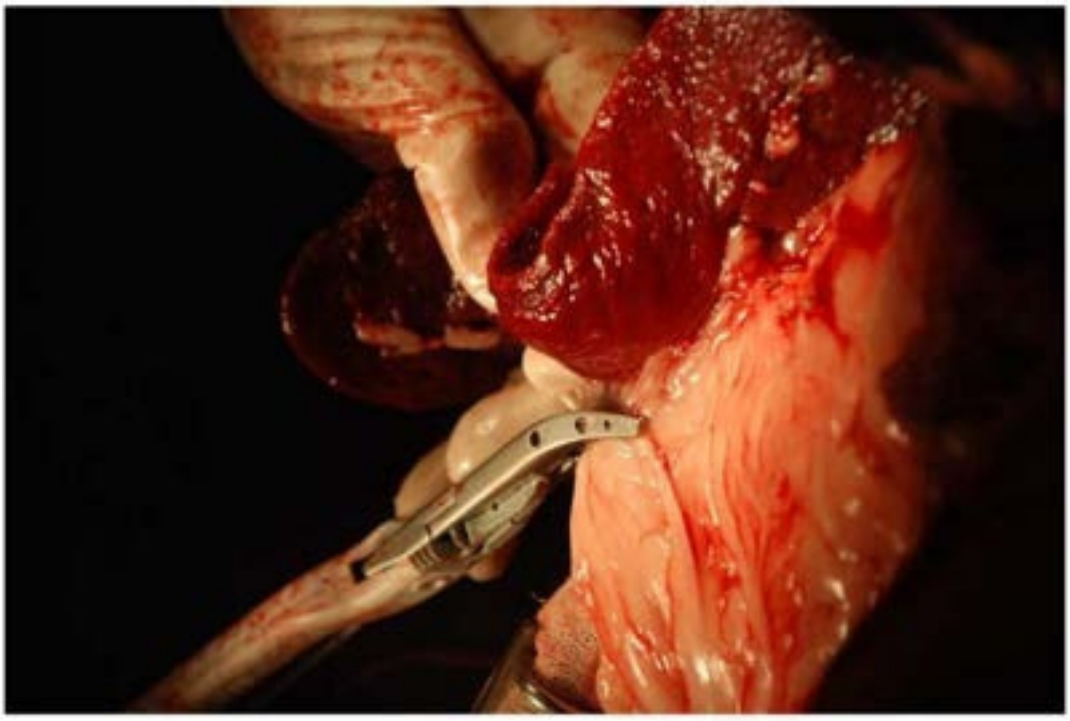


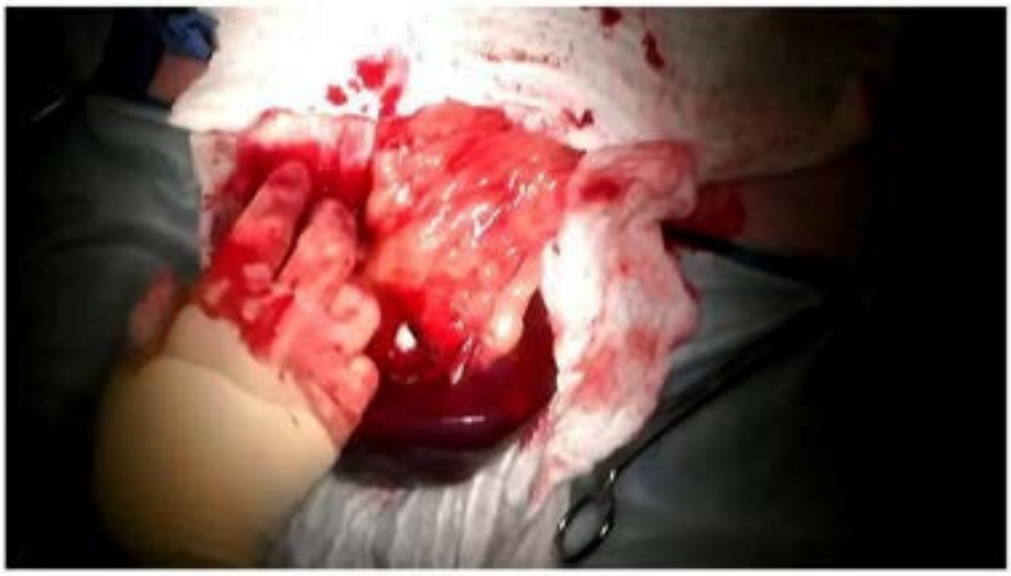
Ligasure





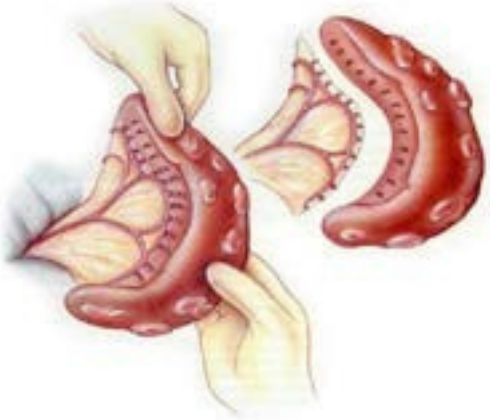






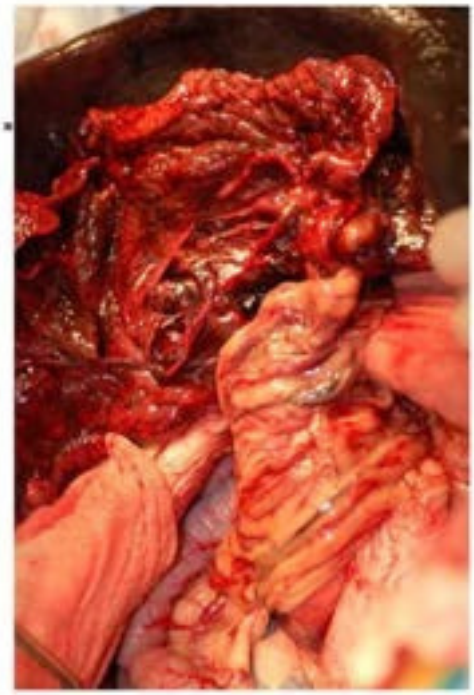
Or... you can just ligate....

- Hand ties or instrument ties
- Ligature with the dog, hemostats with the spleen
- Suture type?



The one exception...

- Splenic torsions should be removed at the twist.
- Suture or staple



Association between previous splenectomy and gastric dilatation-volvulus in dogs: 453 cases (2004–2009)

Angela J. Sartor, DVM; Adrienne M. Bentley, DVM, DACVS; Dorothy C. Brown, DVM, MSCE, DACVS

- Odds of GDV 5.3x higher in dogs with previous splenectomy vs. those without splenectomy

Results—6 (4%) dogs in the GDV group and 3 (1%) dogs in the control group had a history of previous splenectomy. The odds of GDV in dogs with a history of previous splenectomy in this population of dogs were 5.3 times those of dogs without a history of previous splenectomy (95% confidence interval, 1.1 to 26.8).

Conclusions and Clinical Relevance—For the patients in the present study, there was an increased odds of GDV in dogs with a history of splenectomy. Prophylactic gastropexy may be considered in dogs undergoing a splenectomy, particularly if other risk factors for GDV are present. *J Am Vet Med Assoc* 2013;242:1381–1384)

POSTOPERATIVE CARE

- Analgesia and supportive care
- Monitor closely for signs of hemorrhage
- Repeat PCV/TS as necessary
- Provide blood products if necessary to replace losses
- Continue ECG if arrhythmias persistent
- Continue IV fluids until eating and drinking

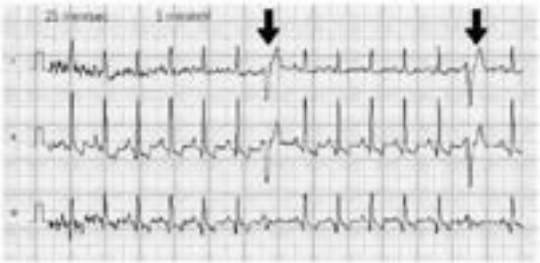
Complications

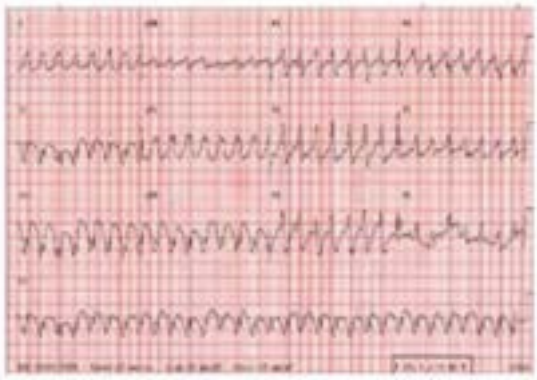
Hemorrhage

- Mostly commonly – its your fault – bad ligature, missed pedicle, etc.
- Coagulopathy
- Another mass – check the liver!

Arrythmias

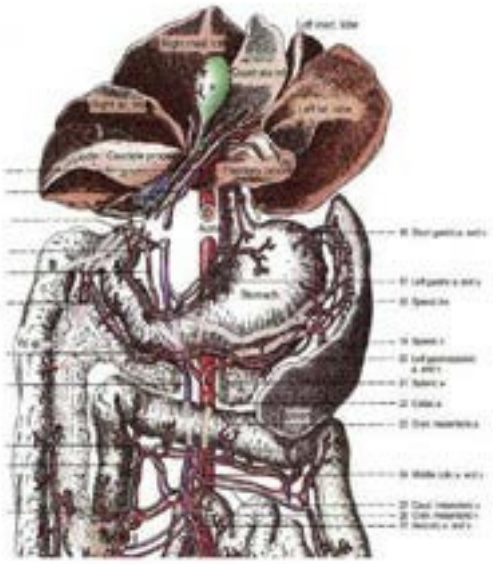
- Most common post operative complication
- Usually not pathologic and usually doesn't require treatment
- Treat if: tachycardic, R on T, multiform, reducing cardiac output
- Lidocaine (2 mg/kg bolus then 50 µg/kg/min) or Sotalol (1 – 2 mg/kg BID)





Pancreatitis or Gastric Necrosis

- Stay close to the spleen!!



Babesiosis in Pit Bulls

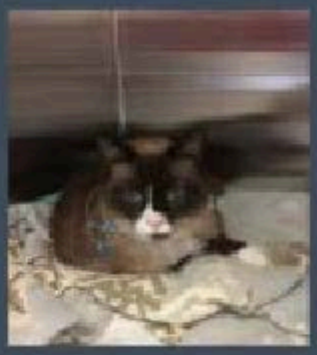
- Pit Bull Terriers and related may develop Babesiosis when immunosuppressed and potentially after removal of the spleen
- Vertical or horizontal transmission
- Discuss risk with owners
- Consider a partial splenectomy

CHYLOTHORAX IN DOGS AND CATS

Chad Schmiedt DVM, DACVS
Professor, Soft Tissue Surgery
College of Veterinary Medicine
University of Georgia

Outline

- Overview of chylothorax
  - *Pathophysiology*
  - *Diagnosis*
  - *Treatment*
  - *Prognosis*



PATHOPHYSIOLOGY

Signalment

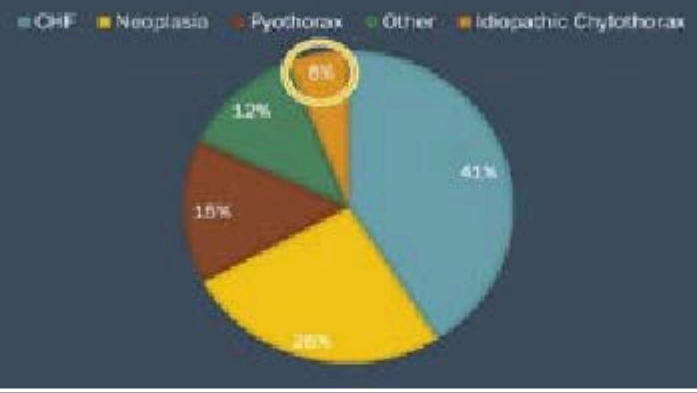
- Oriental cats
- Dogs
  - *Afghan hound*
  - *Mastiff*
  - *Shetland sheepdog*
  - *Shiba Inu (young)*
- Any age possible
- No sex predisposition

Current Techniques in SA Surgery 2014.
Singh, et al. Compendium 2012.

Prevalence

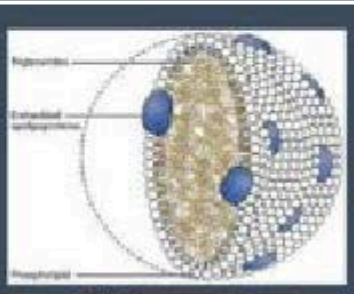
Characterization of and factors associated with causes of pleural effusion in cats

Ruiz, et al. JAVMA 2018.



What is Chyle?

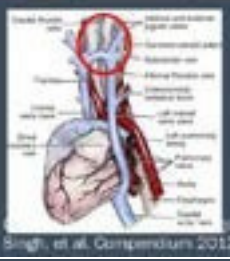
- Modified transudate composed of lymph and chylomicrons
- Proteins between 2.5 – 4 g/dL
- Lymphocytes, chronically mere nondegenerate neutrophils
- Normally returned to systemic circulation by the thoracic duct



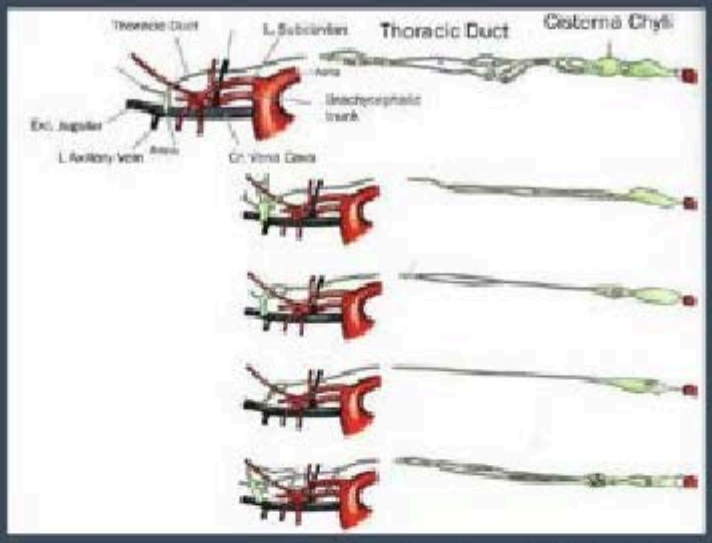
Current Techniques in SA Surgery 2014.
Singh, et al. Compendium 2012.

The Lymphatic System

- Cisterna chyli (CC)
  - *Abdominal lymphatic reservoir*
  - *Fat from SI absorbed as chylomicrons → collected by villous lacteals → empties in CC → TD*
- Thoracic duct (TD)
  - *Largest lymphatic vessel in the body*
  - *Dorsal to the aorta*
  - *Lymphaticovenous junction*

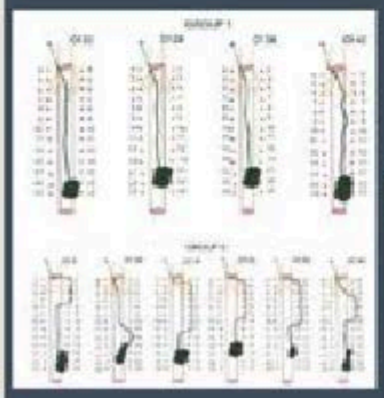


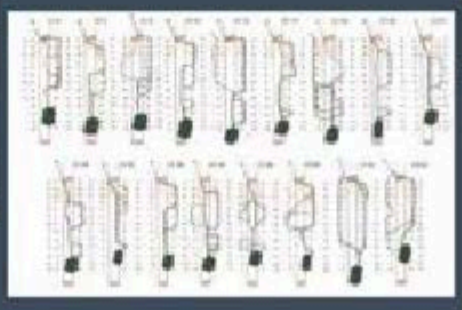
Singh, et al. Compendium 2012.



Anatomical variations of the thoracic duct in the dog

- Variations in 39/43 dogs
- Originates as 1 vessel in 83.7% of dogs, 2 vessels in 14%, and 1 vessel in 2.3%





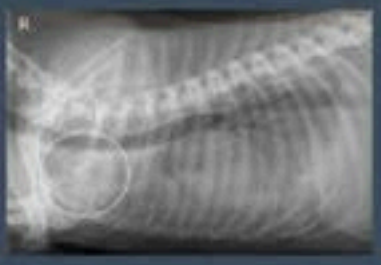
How Does Chylothorax Occur?

- Anything that impedes TD flow or obstructs its lymphaticovenous junction
- Differentials
  - *Idiopathic ***
  - *Cardiac disease – restrictive pericarditis, RCHF, Heartworm*
  - *Mediastinal mass – neoplasia (lymphosarcoma, thymoma) or fungal (blastomycosis)*
  - *Diaphragmatic hernia*
  - *Lung lobe torsion*
  - *Thoracic trauma – tear or rupture of the TD*

Current Techniques in SA Surgery 2014.
Singh, et al. Compendium 2012.

Idiopathic Chylothorax

- Theories
  - *Lymphangectasia leading to leakage of lymph*
  - *Functional obstruction of the lymphaticovenous junction*
  - *Lymphatic hypertension*
- Fibrosis pleuritis develops
  - *Especially problematic in cats*



Small Animal Soft Tissue Surgery 2013.
Singh, et al. Compendium 2012.

DIAGNOSIS

History and Physical Exam

- History
  - *Coughing*
  - *Difficulty breathing*
  - *Exercise intolerance*
  - *Weight loss*
- Physical Exam
  - *Tachypnea or dyspnea*
  - *Quiet lung sounds*
  - *+/- murmur, arrhythmia, caval syndrome*

Singh, et al. Compendium 2012.

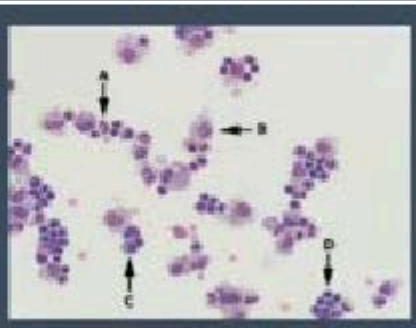
Thoracocentesis

- Both diagnostic and therapeutic
  - *Sedation as needed*
  - *Get as much as you can*
  - *Save some for cytology, biochemical analysis, and culture*
- Chyle
  - *Milky appearance*
  - *May be clear in anorexic patients*
  - *No odor*

Singh, et al. Compendium 2012.

Fluid Analysis

- *Lots of lymphocytes*
- *Some macrophages*
- *Some non-degenerate neutrophils*
  - Especially if chronic – intrapleural inflammation and lymphocyte depletion
- *Lipid droplets may also be present*



Current Techniques in SA Surgery 2014.
Singh, et al. Compendium 2012.

Fluid Analysis

- Triglycerides
  - *Higher in pleural fluid than serum*
  - *Can be pronounced*
- Cholesterol
  - *Lower in pleural fluid than serum*

Table 43-2. Characterization of Chylous Pleural Fluid in Dogs and Cats

	Dogs	Cats
Specific gravity	1.022-1.037	1.019-1.039
Total Protein (g/dl)	2.5-6.2	3.5-7.8
Average nucleated cells/µL	6,127	11,919

Triglycerides	
Serum	189 mg/dl
Effusion	250 mg/dl

Current Techniques in SA Surgery 2014.

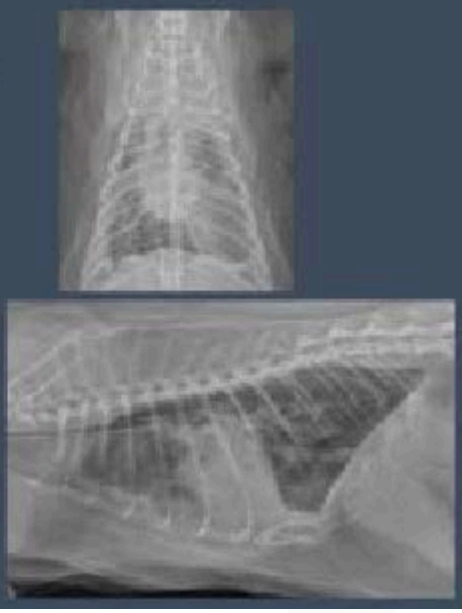
Bloodwork

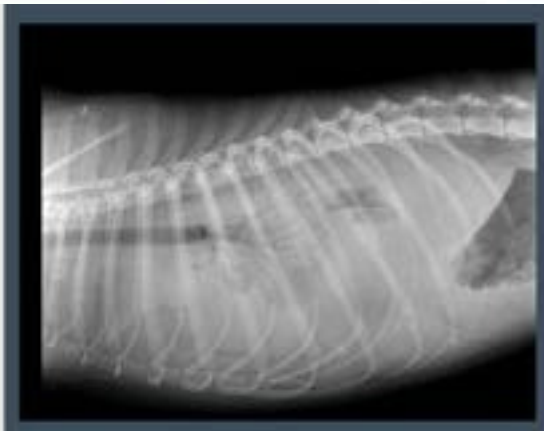
- Chemistry Panel
  - *May see hyponatremia and hyperkalemia*
- CBC
  - *May see lymphopenia*
- Heartworm,
- FeLV, FIV testing

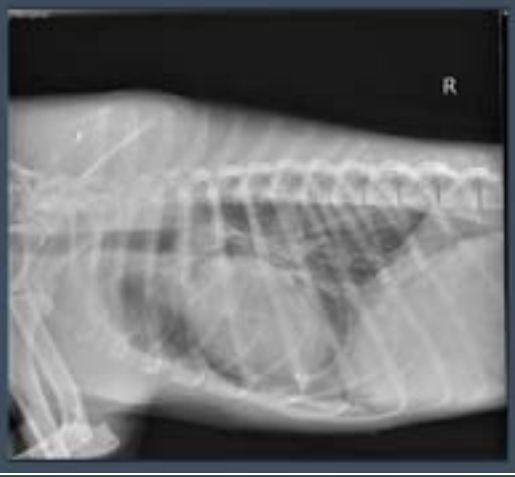
Singh, et al. Compendium 2012

Imaging: Radiographs

- Thoracic radiographs
  - *Pleural effusion?*
  - *Cardiomegaly?*
  - *Mass?*
  - *Rounding of the lungs?*
  - *Lung lobe torsion?*
- Thoracocentesis first, or repeat films afterwards

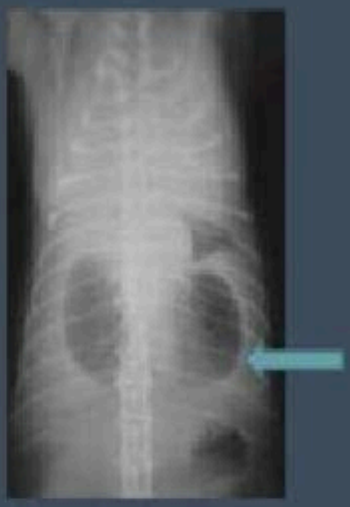






Fibrosing Pleuritis

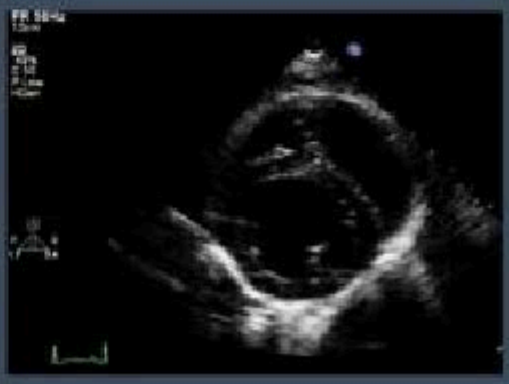
- Chronic exposure of the pleura to chyle → altered fibrin production and degradation
  - *Fibrosis of the visceral pleura can result*
  - *Restricts pulmonary expansion*
- More common in cats
  - *Poorer prognosis*



Small Animal Soft Tissue Surgery. 2013.
Singh, et al. Compendium 2012.

Imaging: Echocardiogram

- Recommended in all patients with chylothorax
  - *Regardless of auscultation*
- Rule out cardiac causes of chylothorax
  - *Impacts treatment!*

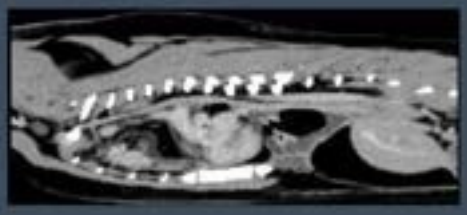


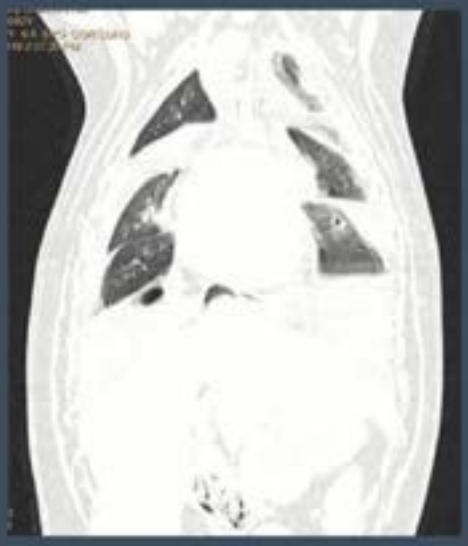
Thoracic CT scan

Definitively rule out mediastinal mass lesions, other pathology

Further evaluate lungs

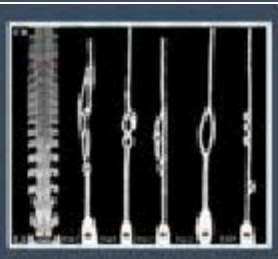
Lymphangiography can be performed to assess thoracic duct anatomy

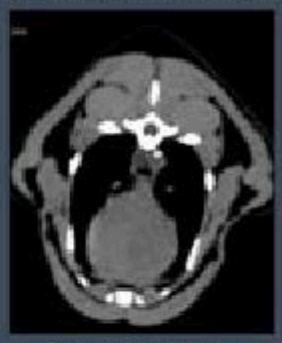




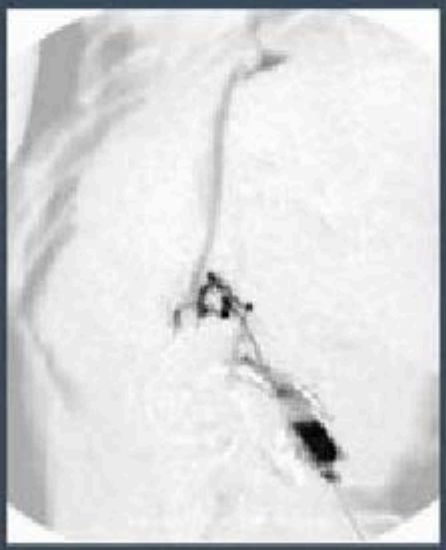
Imaging: Lymphography

- Many methods described
  - *CT*
  - *Fluoroscopy*
  - *Fluorescent imaging*
  - *Catheterization and direct injection*
  - *Intranodal – popliteal, ileocecal colic*
  - *SQ rectal, metatarsal*
- Surgical planning
  - *Can also help diagnose lymphangiectasia*
- Confirmation of ligation
- Investigation of recurrence or failure to resolve
- As part of embolization procedures





Lee, et al, VRU 2011.



Lymphangiogram 1 month after CCA and TDL

Contrast shunting directly into caudal vena cava

MEDICAL TREATMENT AND PROGNOSIS

Etiology is Important

- Treat any underlying cause if identified
- Will not guarantee complete resolution of chylothorax
  - *May consider concurrent chylothorax surgery if already performing thoracic surgery*

General Medical Treatment

- Periodic thoracocentesis
  - *As needed to prevent dyspnea*
  - *Risk of introducing bacteria – sterile technique!*

- Low fat diet
  - *Reduce lipid concentration in chyle to improve absorption of effusion*

Singh, et al. Compendium 2012

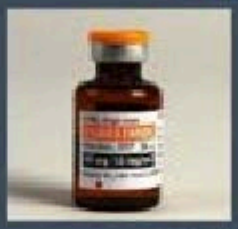
Medical Treatment: Rutin

- OTC Benzopyrene
- Possible Mechanism of Action:
  - *Stimulation of macrophage function to remove protein and promote fluid reabsorption*
  - *Decreased vascular leakage*
- Case reports in cats show possible efficacy
  - *Minimal information on its utility in dogs*

Small Animal Soft Tissue Surgery 2013.
Singh, et al. Compendium 2012.

Medical Treatment: Miscellaneous

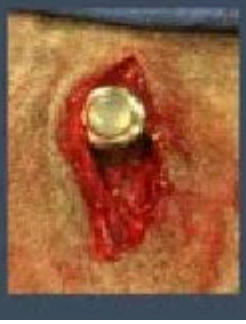
- Octreotide
  - *Somatostatin analogue*
  - *May reduce TD flow and aid in healing*
  - *Little information in the veterinary literature*
  - *Injectable only, $$$*
- Steroids?
- Furosemide?

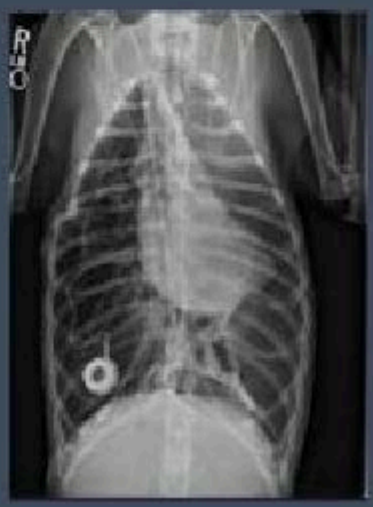


Small Animal Soft Tissue Surgery 2013.
Singh, et al. Compendium 2012.

Pleuroport as part of medical management

Reduce risk and discomfort of regular thoracocentesis





Prognosis with Medical Treatment

- Spontaneous resolution of chylothorax is possible
  - *May take weeks to months*
- Exposure to chyle likely increases risk of fibrosing pleuritis
- Other sequela
  - *Electrolyte imbalances*
  - *Hypoproteinemia*
  - *Dehydration*
  - *Immunodeficiency?*

Small Animal Soft Tissue Surgery 2013.
Singh, et al. Compendium 2012.

SURGICAL TREATMENT AND PROGNOSIS

Surgical Treatment

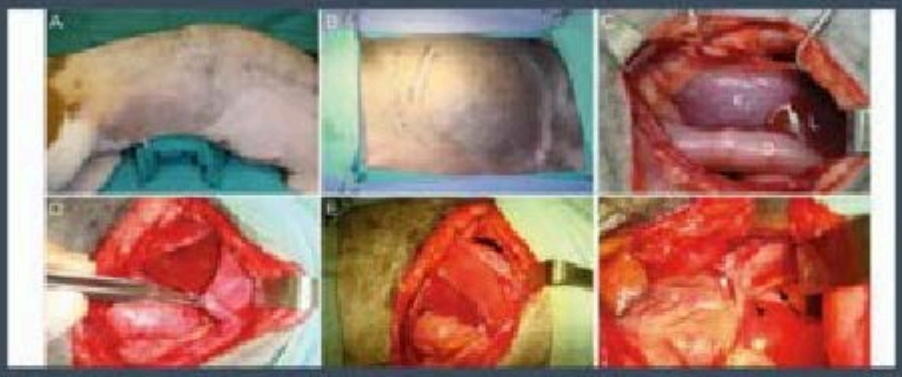
- When?
  - *Persistent effusion despite treatment of a predisposing condition*
  - *Idiopathic etiology*
  - *Persistence for >4 weeks (?)*
  - *Consider patient*
- Several techniques described

Small Animal Soft Tissue Surgery 2013.
Singh, et al. Compendium 2012.

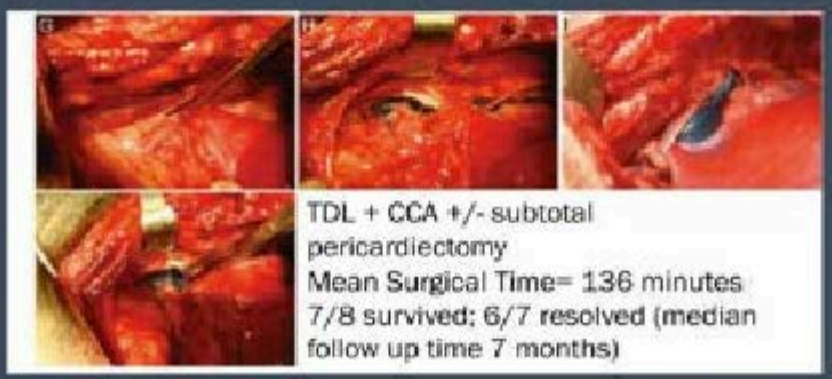
Open surgical strategy

- Approach the thoracic duct by a paracostal incision at the 12th intercostal space
- Inject abdominal LN with methylene blue
- Identify and ligate thoracic duct
- Ablate the cisterna chyli
- Remove the pericardium through a separate incision

Single Paracostal Approach



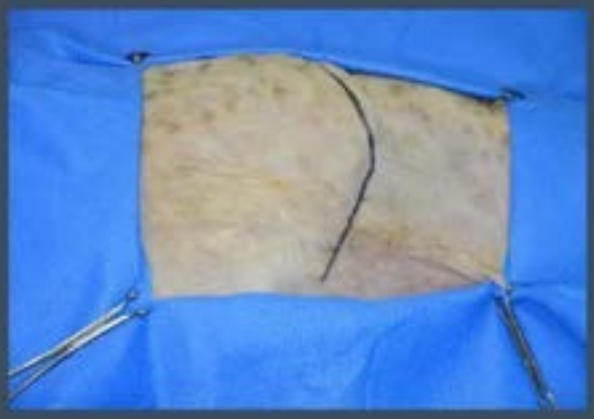
Single Paracostal Approach

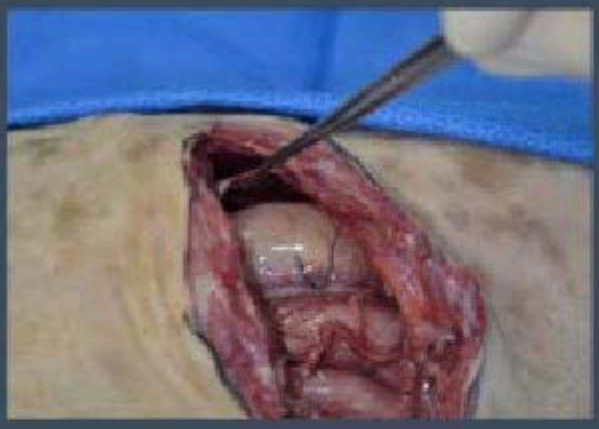


TDL + CCA +/- subtotal pericardiectomy
Mean Surgical Time= 136 minutes
7/8 survived; 6/7 resolved (median follow up time 7 months)

Small Variation:

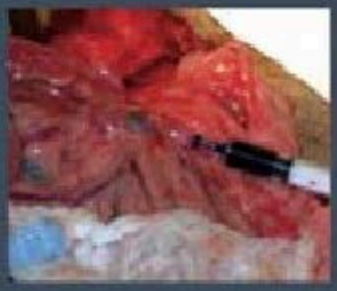
Use of 12th intercostal space allows for easier access to caudal thorax





Intraoperative Identification of Lymphatics

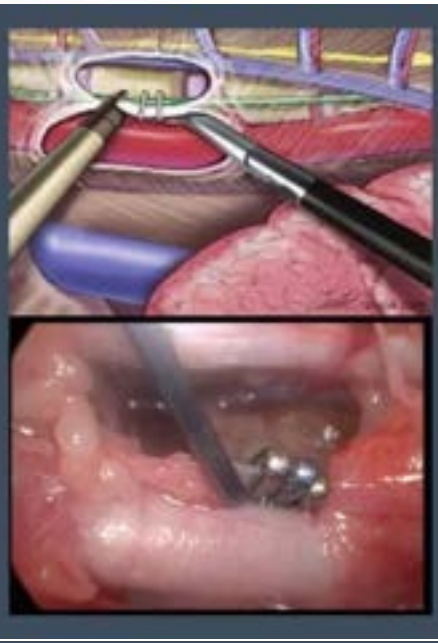
- Methylene blue
  - *Diluted with 10 – 20x saline*
  - *Injected into mesenteric LN typically the ileocecocolic*
  - *One shot at good injection, 25 gauge needle, slow injection*
  - *Visible within 10 min, lasts ~5 – 60min*
- Tripan Blue can also be used – less visible
- Heavy cream administered PO before surgery



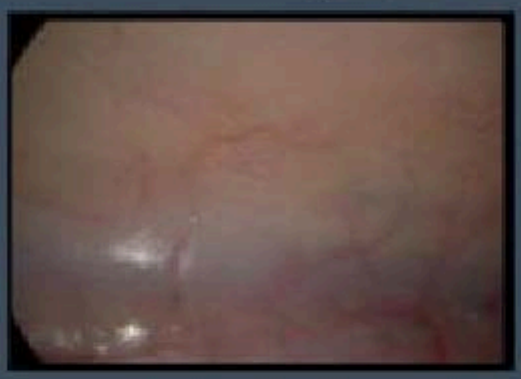
Small Animal Soft Tissue Surgery 2013.
Singh, et al. Compendium 2012

Thoracic Duct Ligation

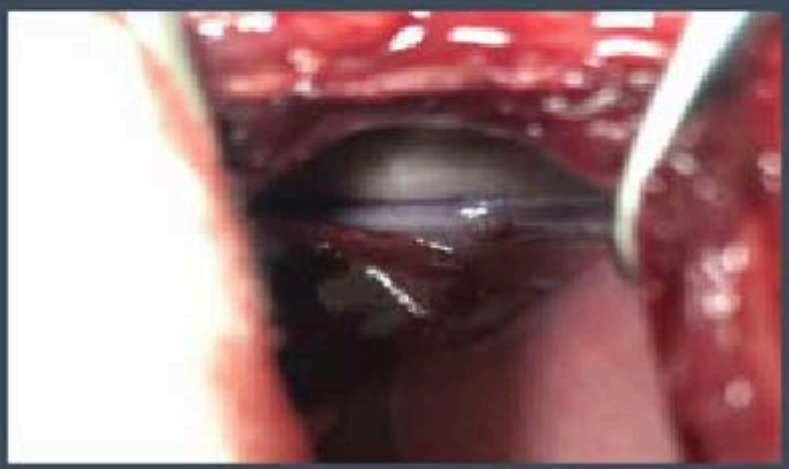
- TD occluded at its entry point in the thorax just past the cisterna chyli
- Typically double ligated with non-absorbable suture or clips
  - *Thoracoscopic or open*
  - *Branches common!*
  - *Dog – right sided*
  - *Cat – left sided*
- Ligasure can be used to seal and divide

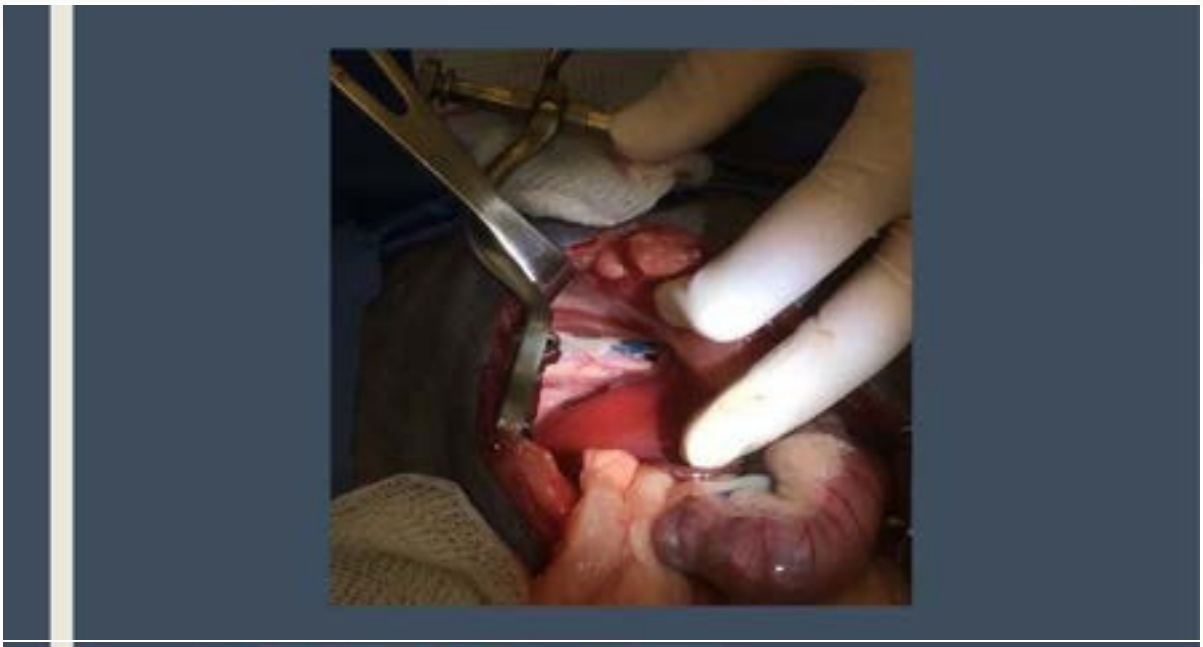


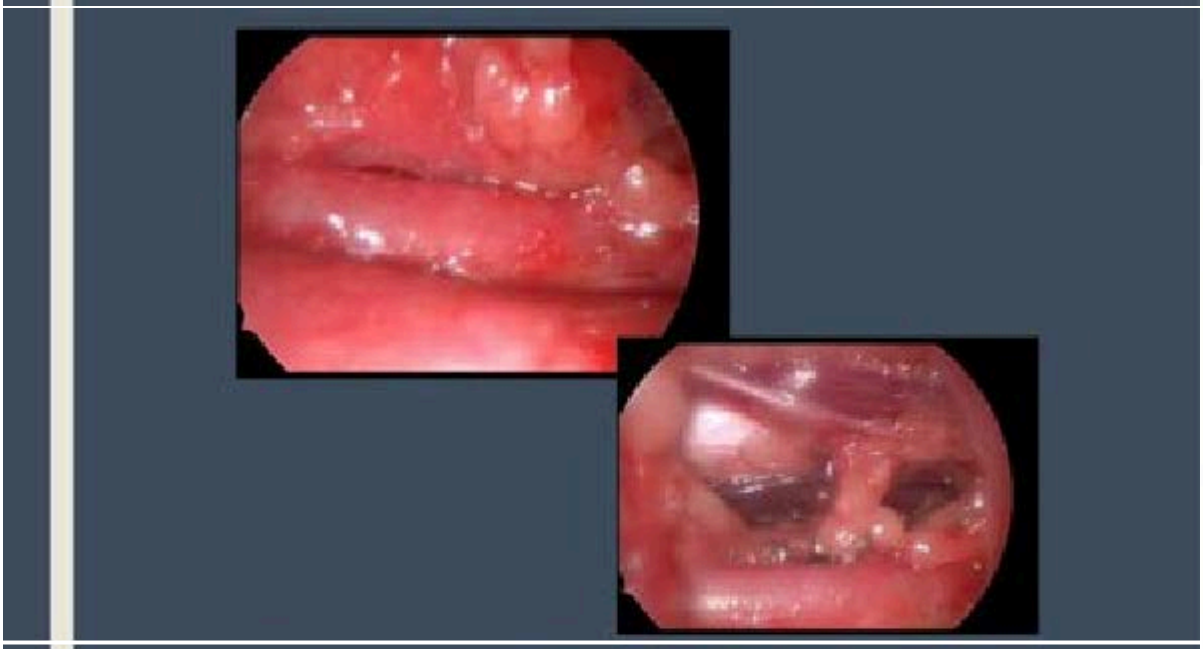
The thoracic duct can be difficult to see under the thickened pleura

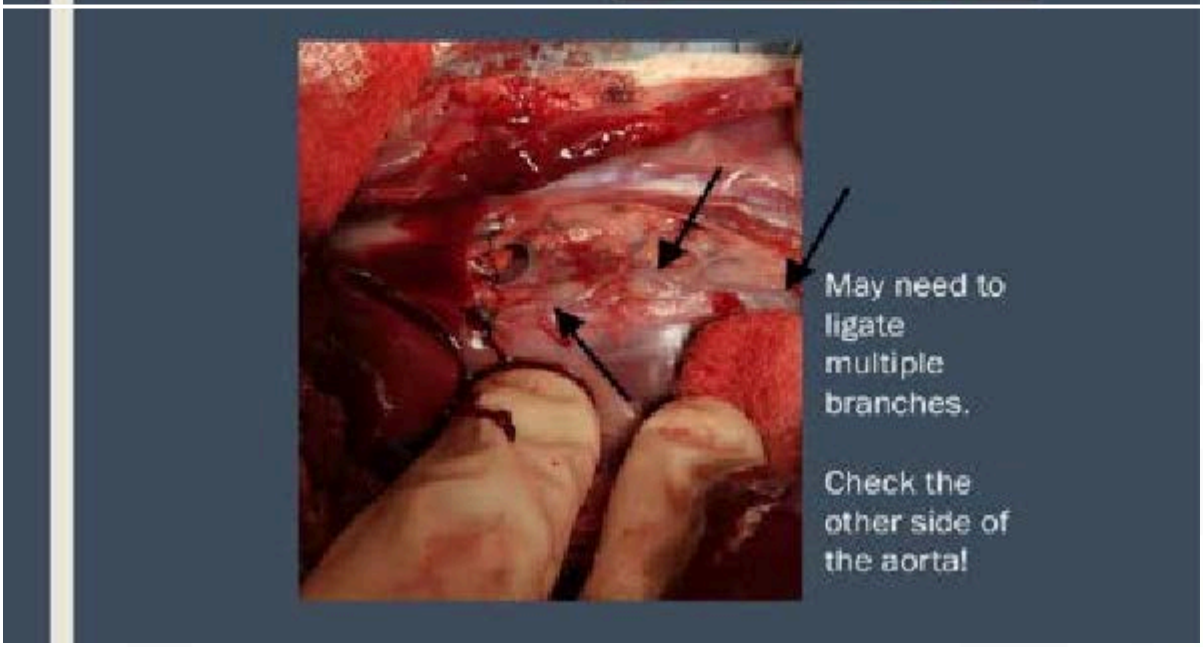


Prior to injection of the LN, the pleura should be gently removed from the aorta and dorsal



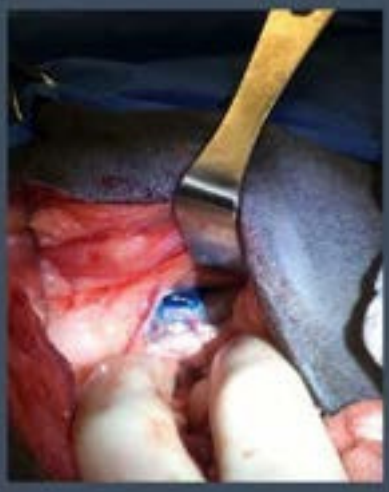





May need to ligate multiple branches.
Check the other side of the aorta!

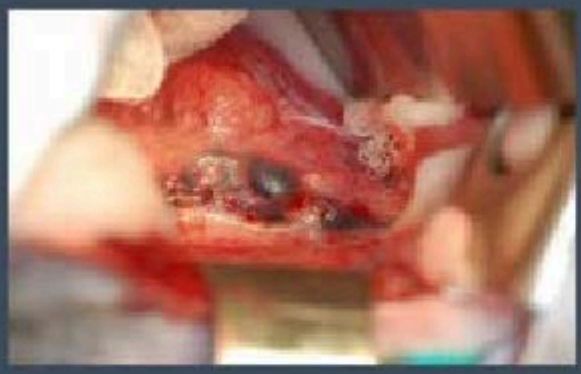
Cisterna Chyli Ablation

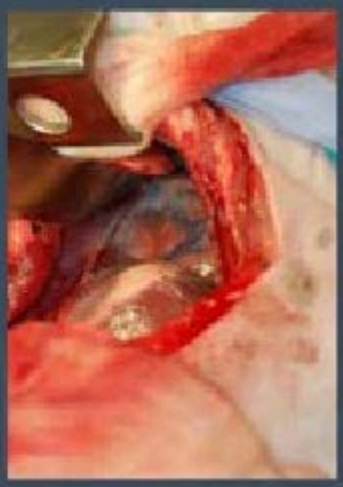
- Cisterna chyli identified and broken open
  - *Must be after TDL*
  - *Dorsal to aorta at the level of the renal arteries*
  - *Walls are grasped and debrided*
- Relieves increased lymphatic pressure caudal to TD ligation site
  - *Encourages intra-abdominal lymphaticovenous anastomosis formation*



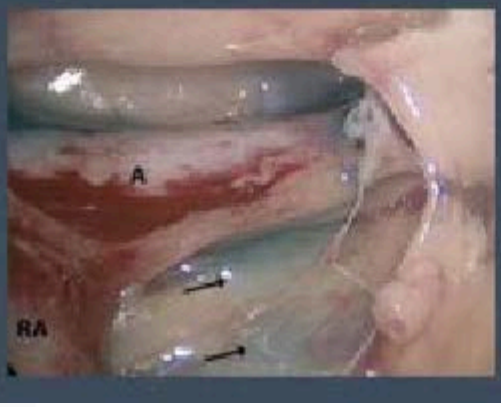
Small Animal Soft Tissue Surgery 2013.
Singh, et al. Compendium 2012.

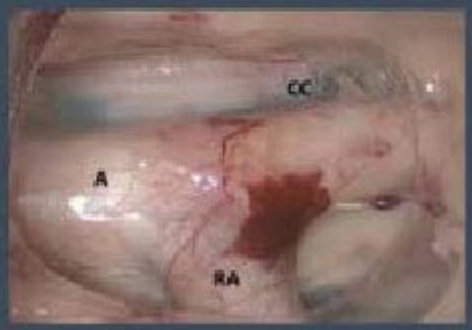
Feline cisterna chyli before and after ablation.

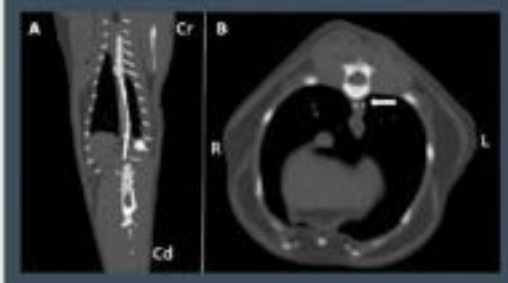




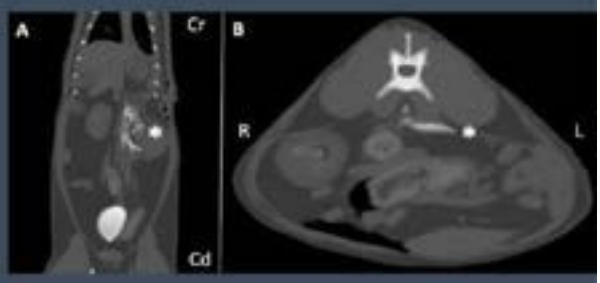
Cisterna can be accessed by open approach or by transdiaphragmatic laparoscopy





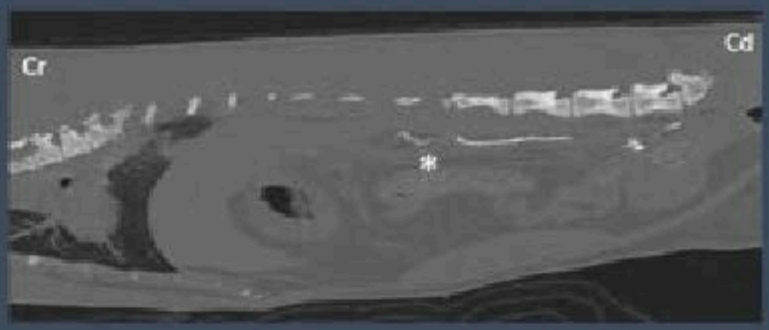


Lymphangiogram in a cat immediately prior to TDL and CCA; popliteal injection



Lymphangiogram in a cat immediately after to TDL and CCA; popliteal injection

Dickerson, AJVR, 2019

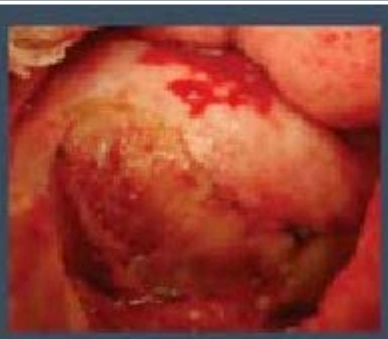


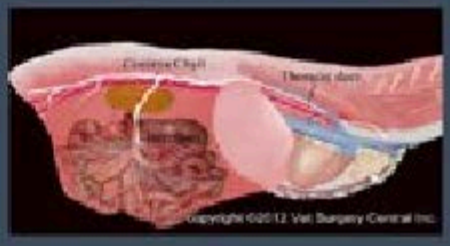
Lymphangiogram in a cat 30 days after TDL and CCA; popliteal injection; * = new lymphatic anastomosis

Dickerson, AJVR, 2019

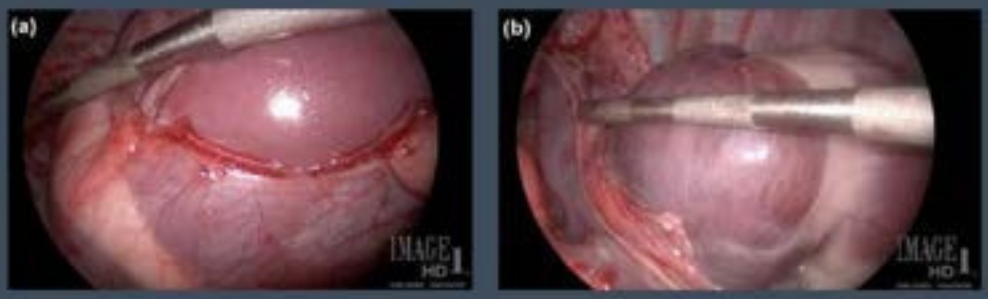
Subtotal Pericardectomy

- Pericardium removed, preserving the phrenic nerve
- Thickened pericardium secondary to chyle irritation vs primary condition
- If thickened, may be restrictive
  - *Increased right sided venous pressure*
  - *May impede chyle drainage*
- Could evaluate intracardiac pressures to determine need for pericardectomy



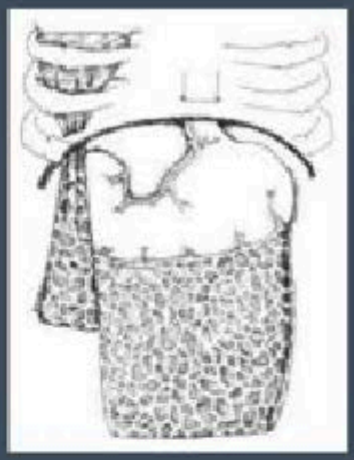


Need to fully release the pericardium – this is not a "window"



Omentalization

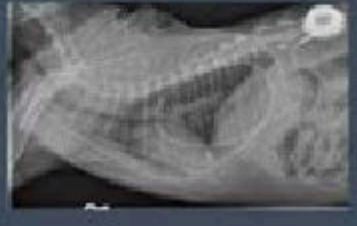
- Omentum pulled through a slit in the diaphragm
- Proposed benefits
  - *May function as a physiologic lymphatic drain*
  - *May promote healing of the TD through neovascularization and fibroplasia*



Stewart, et al. JAAHA 2010.
Singh, et al. Compendium 2012.

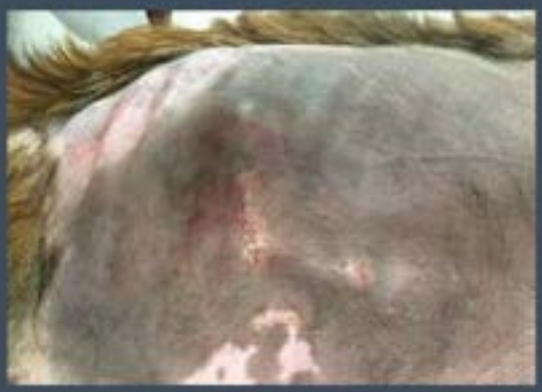
Pleural Port

- May be placed at time of original surgery, or in the event of persistent effusion
- Long term chest tube with silicone subcutaneous port
- Potential risks:
  - *Port obstruction*
  - *Infection*
  - *Suboptimal positioning*



Singh, et al. Compendium 2012.

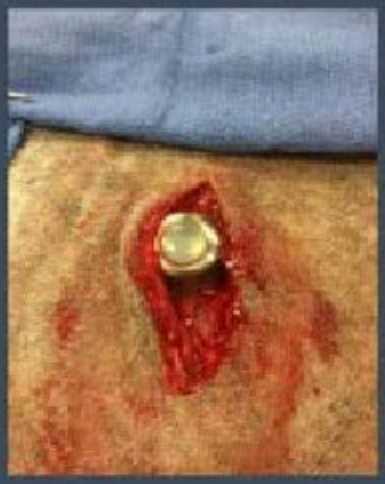
Normal immediately post placement



Painful, swollen, purulent = infected Pluralport

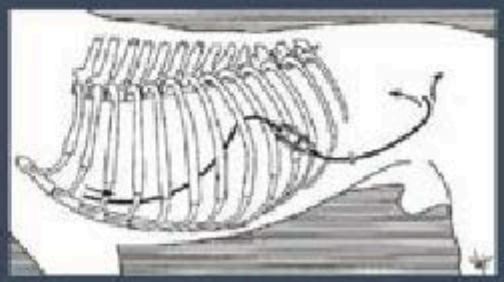
Indications of Pleural Port

- Prior to surgery
- At the time of surgery
- After surgery if the chylothorax does not resolve



Pleuroperitoneal Shunt

- Fenestrated catheters shunt fluid from the pleural to the peritoneal cavity
  - *One-way valve under the skin is pumped daily*
- Potential risks:
  - *Obstruction*
  - *Dislodgement*
  - *Infection*
  - *Pain at pump site*
- Complication in over 50% of dogs
- Median disease free interval – 27 months



Smeak: 2001 JAVMA

Postoperative Strategies

General expectations

- Give at least 30 days for response to allow for lymphaticovenous anastomoses formation
- In dogs – 70 – 80% response rate for surgery
- In cats – 50% response rate to surgery
  - *More likely to get restrictive pleuritis*
- Dogs or cats may have persistent non-chylous (serosanguinous) effusion
  - *Lymphatic drainage from the head/cranial thorax?*
  - *Secondary to chronic inflammation?*

Outcome After Surgery in Cats

- TDL alone
  - *Fossum 1991 – 15 cats – 20% resolution*
  - *Kerpsack 1994 – 19 cats – 68% resolution, 9 died early post-op*
- TDL + CCA
  - *Not reported in cats*
- TDL + SP
  - *Stockdale 2018 - 15 cats – 73% resolution, 1 died early post-op*
- TDL + CCA + SP
  - *Stockdale 2018 - 7 cats – 14% resolution, 3 died early post-op*
  - *UGA records review – 8 cats – 38% resolution, 4 died early post-op*

Reported outcomes in dogs

- TDL and PC
  - *McAnulty 2011 – 11 dogs – 60%*
- CCA and TDL –
  - *Hayashi 2005 – 8 dogs – 88%*
  - *McAnulty 2011 – 12 dogs - 83%*
  - *Staiger 2011 – 8 dogs (4 with pericardiectomy) – 75%*
- Video assisted TDL and pericardiectomy
  - *Mayhew 2018 – 39 dogs – 89%*
  - *Mayhew 2012 – 6 cases – 85%*

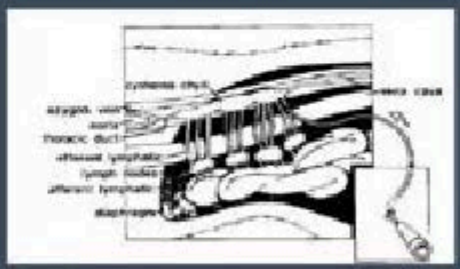
Outcome After Surgery

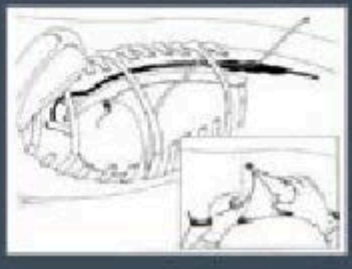
- Options if not successful
  - *Try steroids*
  - *Lymphangography +/- repeat surgery*
  - *Pleural port*
  - *Continued medical management*
  - *Euthanasia*

Small Animal Soft Tissue Surgery 2013.
Singh, et al. Compendium 2012.

Lymphatic embolization

- Used as a first-line treatment or as a salvage procedure
- Open approach recommended, percutaneous approach is technically difficult
- Advantages:
  - *reduces chyle flow into the chest.*
  - *occludes branches which maybe missed in surgery or "silent or sleeping" branches*
  - *may reduce the likelihood of collateralization*
- Sternal position
- 3:1 mixture of Lipiodol® and N-butyl cyanoacrylate glue
  - *Starting volume of 0.1 ml/kg*
  - *Catheterized efferent abdominal lymphatic*

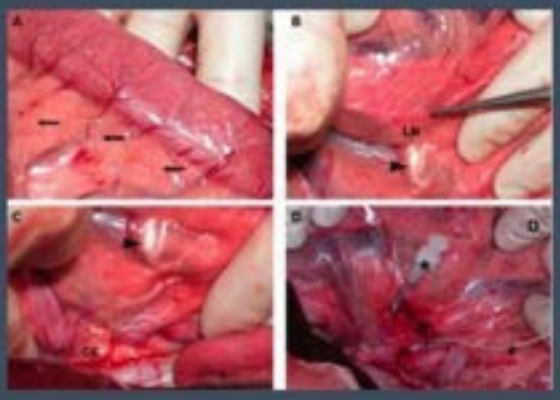


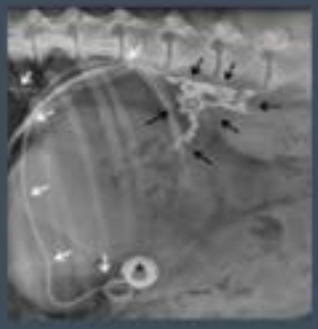


Singh, AJVR, 2011; Clendaniel JVIM, 2014, Pardo Vet Surg, 1989

Salvage Cisterna Chyli and Thoracic Duct Glue Embolization in 2 Dogs with Recurrent Idiopathic Chylothorax

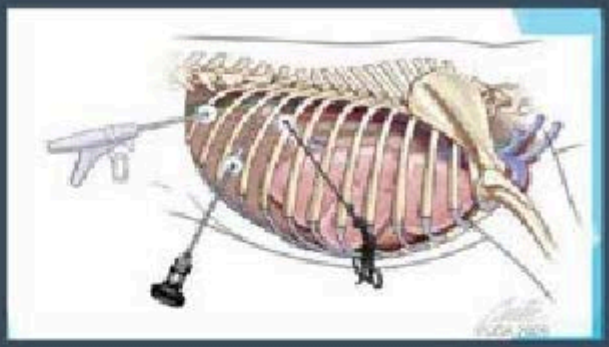
D.C. Clendaniel, C. Weisse, W.T.N. Culp, A. Berent, and J.A. Solomon





Thoracoscopic approach

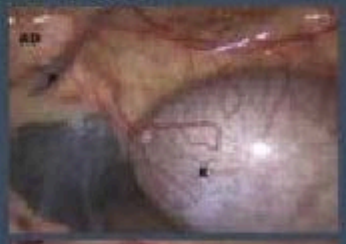
- Sternal recumbancy
- Camera – mid 10th intercostal space
- Instruments –
  - Dorsal 9th or 10th intercostal space
  - Dorsal 11th or 12th

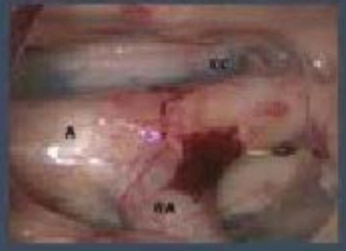


Allman, Vet Surg 2010

Laparoscopic cisterna chyli ablation

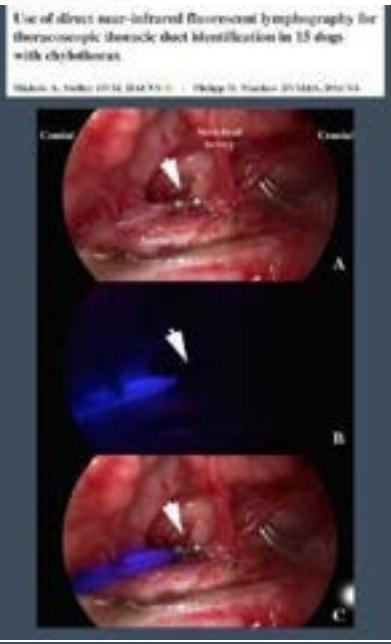
- Transdiaphragmatic
  - *Long threaded port*
  - *Use camera portal from thoracoscopic TDL*
  - *Establish new abdominal instrument portals*
- Transabdominal
  - *Establish 3 new portals in abdominal wall*





Direct near-infrared fluorescent lyphography using indocyanine green (ICG)

- ICG is the fluorophore
- Given intranodal in the popliteal or intrahepatic
- Requires special light and filter
- Used for intraoperative identification of the thoracic duct and cisterna chyli



Leo – UGA MR 247381

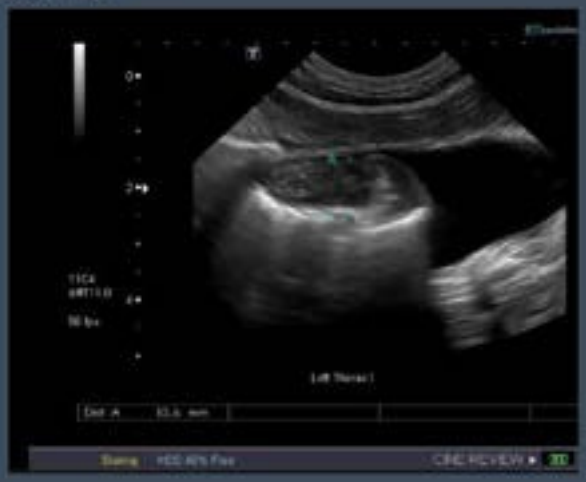
- 4 year old, castrated male, domestic shorthair cat
- Presented for respiratory distress
- Bloodwork – mild thrombocytopenia, all serum chemistry was within normal limits
- Heartworm, FIV, FeLV negative
- Up to date on vaccines, indoor only, 1 other cat in the house

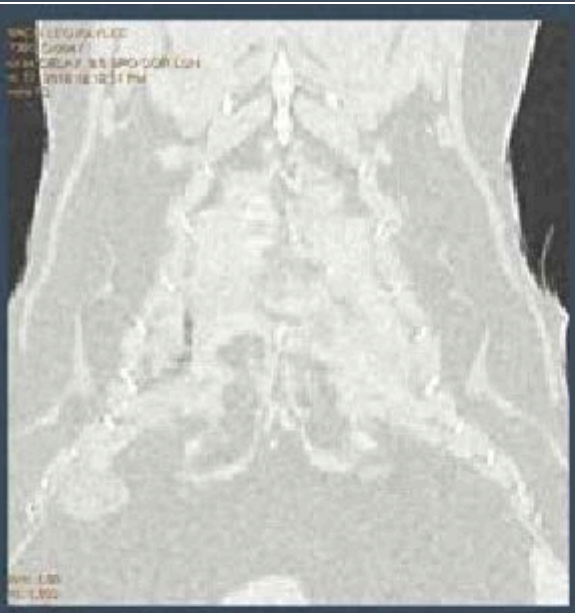
Bloodwork and Initial Diagnostics

- Blood pressure - 150- mmHg
- PCV – 36%, Total solids – 6.8 g/dL
- Mild hyperlactatemia, mild azotemia
- Fluid cytology:
  - *No infectious organisms*
  - *Non-degenerate, neutrophil rich exudate*
  - *Lightly eosinophilic proteinacious background*
- Cardiology consultation:
  - *Ventricular rhythm rate of 160 – 180 during exam, consider electrolyte changes*
  - *No cardiac cause of chylothorax*

Abdominal Ultrasound

- Normal abdomen
- Left caudal lung lobe mass
- Non-diagnostic aspirate – mixed inflammation





Thoracic CT scan:

- Left caudal lung lobe mass
- Pleural effusion
- Sternal Lymphadenopathy

Surgical procedure

- 5th intercostal thoracotomy
  - *Left caudal lung lobectomy with a TA-30 vascular stapler*
  - *Pericardectomy*
- 12th intercostal thoracotomy
  - *Trypan Blue used to identify thoracic duct, injection in ileocecocolic lymph nodes, 0.5 ml*
  - *Thoracic duct ligated*
  - *Cisterna chyli ablated*
  - *Chest tube placed*

Recovery

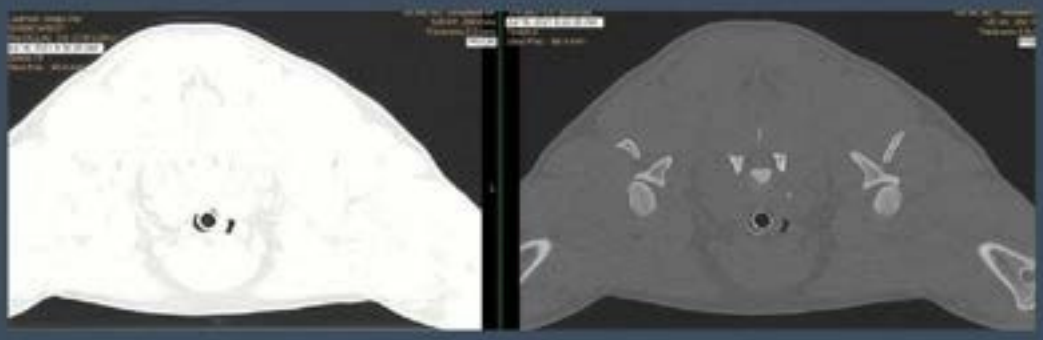
- Uneventful except developed ventricular tachycardia post op
- Oral Sotalol (20 mg Q12 hours) resolved ventricular rhythm
- Discharged 4 days post op
- No neoplasia noted – lung 'mass' was fibrosing restrictive pleuritis.
- Chylothorax resolved

Jengo – UGA MR 312092

- 3 yr old male neutered Pit Bull Terrier
- Thoracic effusion noticed incidentally on radiographs
- 600 mL of milky pink fluid was aspirated from the thorax
- Bloodwork prior to referral was unremarkable
- Started Rutin 500 mg Q 8 hours

At presentation

- Normal physical examination
- BP – 200 mmHg
- Pleural effusion on thoracic ultrasound (TFAST)
- Paired triglycerides – Blood 44 mg/dL, Thoracic Fluid – 1558 mg/dL
- Thoracic fluid cytology – pinkish white, opaque, nucleated cells 3.3 x10^3/µL, lymphocyte rich
- Echocardiograph – No cardiac abnormalities
- CBC – mild lymphopenia
- Serum Chemistry – no remarkable abnormalities



Thoracic CT scan: pleural effusion,
no other significant abnormalities

Surgical treatment

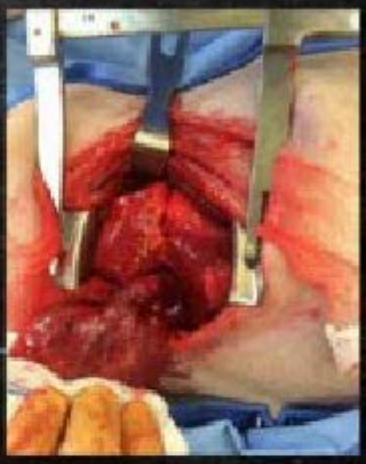
- Right sided, 12th intercostal thoracotomy
- Trypan Blue used for direct visualization of the thoracic duct and cisterna chyli
- Thoracic duct ligation
- Cisterna chyli ablation
- Pericardectomy

Post-operative

- Hydromorphone for 24 hours, then a NSAID and gabapentin
- IV fluids
- Management of chest tube – fluid amount significantly reduced after 48 hours, removed after 72 hours
- Discharged about 5 days after surgery
- No recurrence of chylothorax

Approaches to the Thorax

Chad Schmiedt DVM, DACVS
Professor, Small Animal Surgery
University of Georgia

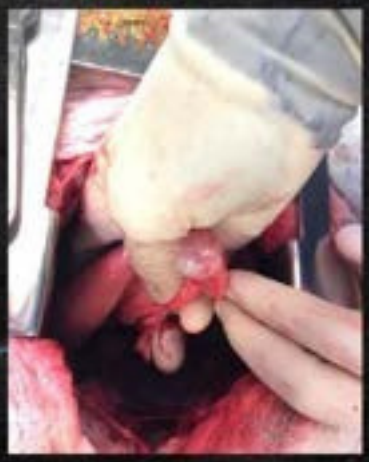


Summary

- Thoracic anesthetic considerations
- Intercostal thoracotomy
- Median Sternotomy
- Recovery and post operative care

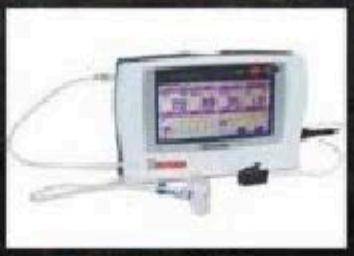
Anesthetic Considerations

Thoracic Surgery



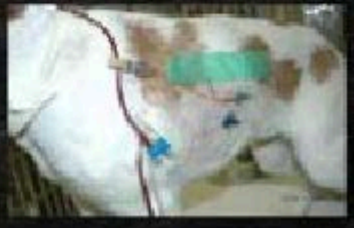
Anesthetic Considerations

- Ventilation
- Capnography
- Painful procedures
  - Systemic opoids
  - Local blocks
- Preoperative thoracocentesis
- Preoxygenate prior to induction



Additional anesthetic considerations

- Pressure for ventilation will be less once thorax is open
- Lung lobes may be packed off – gently re-expanded prior to closure
- Be wary of tension pneumothorax, especially during closure – keep chest tube open until thorax is closed
- Consider small bore chest tubes whenever possible
- Be sure any post operative wraps are not too tight



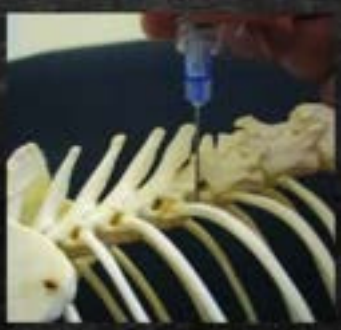
Local Blocks – Intercostal Blocks

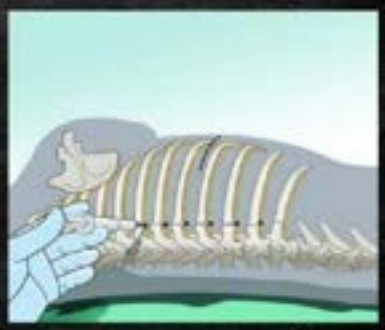
Can be done preoperative or intraoperative

Just off caudal border of the rib

Bupivicaine has best duration of action

Up to 2 mg/kg





https://todaysveterinarynurse.com/articles/locoregional-anesthesia-for-small-animal-patients/

Preoperative considerations

- Surgery guided by preoperative imaging and goals
- **Lateral intercostal thoracotomy** exposure of about 1/3 of ipsilateral thorax
- **Median Sternotomy** – Explore the most of the thorax, poor exposure to the dorsal vessels, pulmonary hilus, thoracic duct

Intercostal Thoracotomy

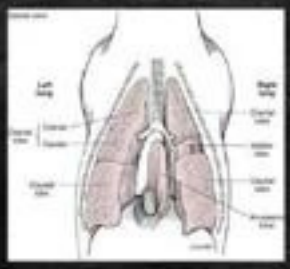
Considerations and Technique

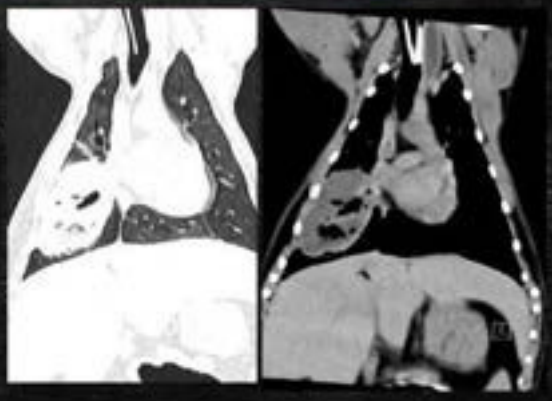
Intercostal Thoracotomy

Rib space can by guided by imagining

Additional exposure can be gained by rib resection

Target is the hilus of the lobe – not where the tumor is located





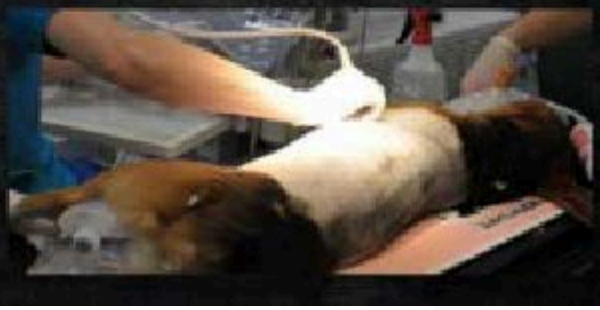
https://veterinarykey.com/surgery-of-the-lower-respiratory-system-lungs-and-thoracic-wall/

Recommended sites

Target	Left	Right
Heart/pericardium	4, 5	4, 5
PDA, PRAA	4	
Cranial lung lobe	5, 4	5, 4
Middle lung lobe		5
Caudal lung lobe	5, 6	5, 6
Cranial esophagus	3, 4	3, 4, 5
Caudal esophagus	7, 8, 9	7, 8, 9
Thoracic duct (dog)		10
Thoracic duct (cat)	10	

Preparation and Positioning

- Lateral recumbency
- Consider towel underneath the patient
- Shave entire thorax



Skin and SQ are incised

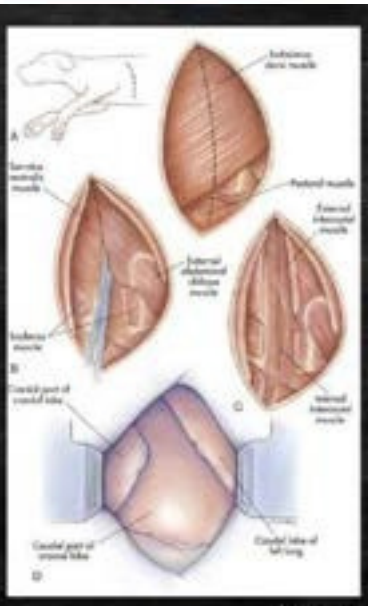
Hypaxial muscles to sternum

Latissimus dorsi is undermined and divided or preserved

Scalenus identified and transected

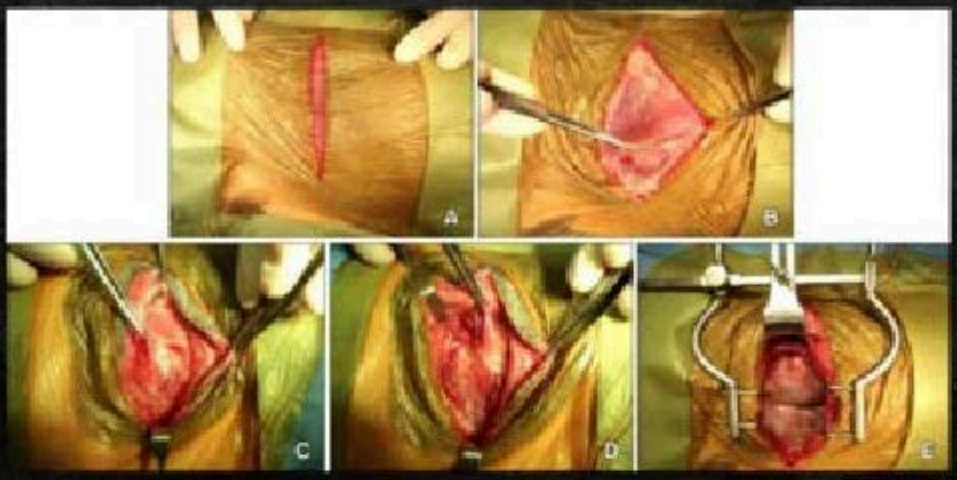
External and Internal intercostals are divided

Pleura is incised – sharp or blunt



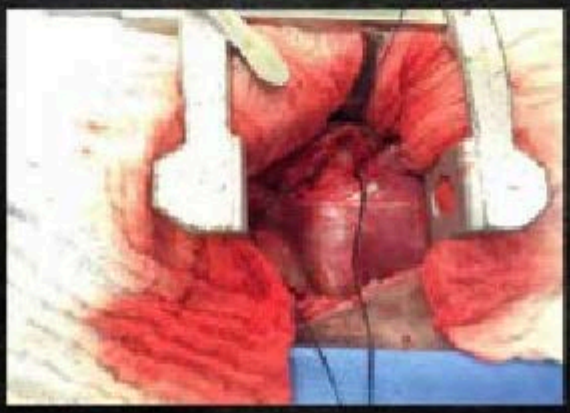
https://veterinkey.com/surgery-of-the-lower-respiratory-system-lungs-and-thoracic-wall/

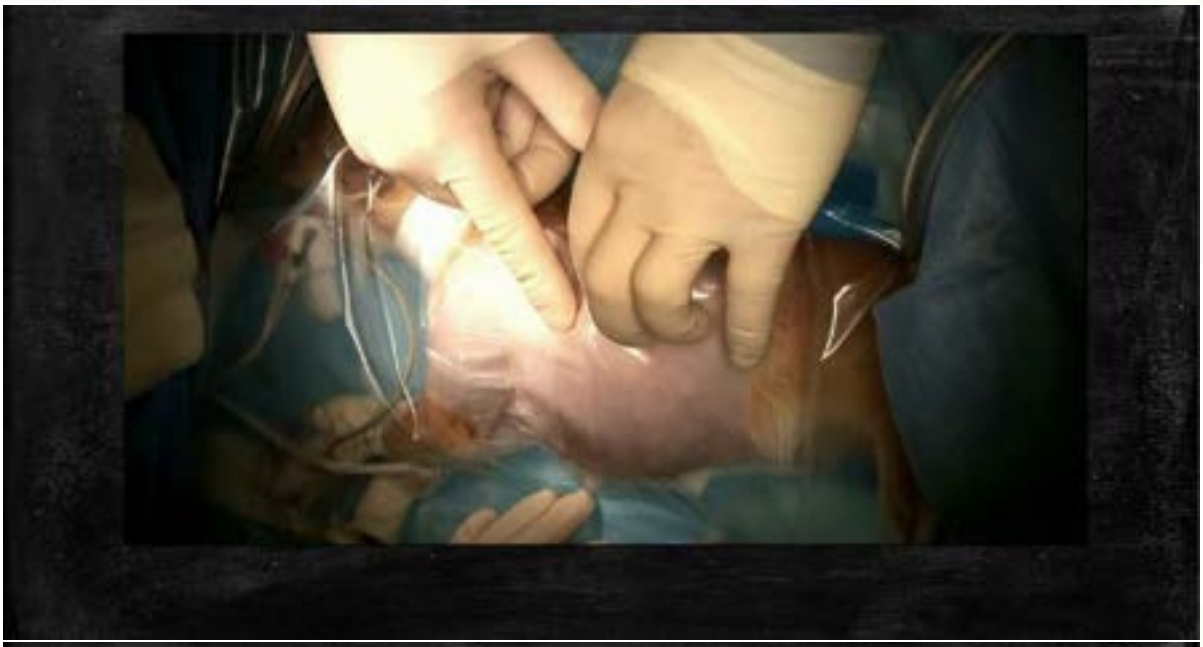
Muscle Sparing Approach – Latissimus dorsi preserved



Yoon, J Vet Sci, 2015

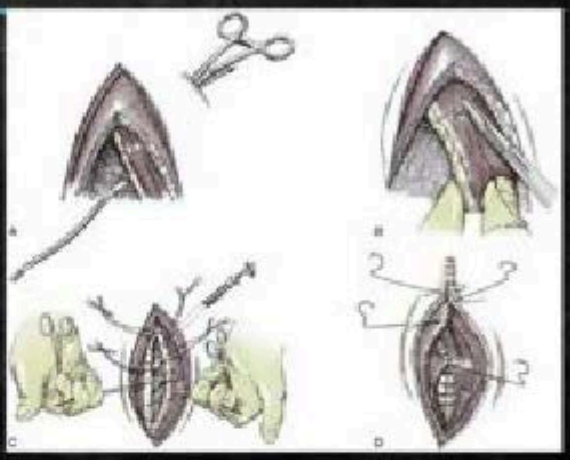
Laparotomy pads and Finocchieto retractors are placed

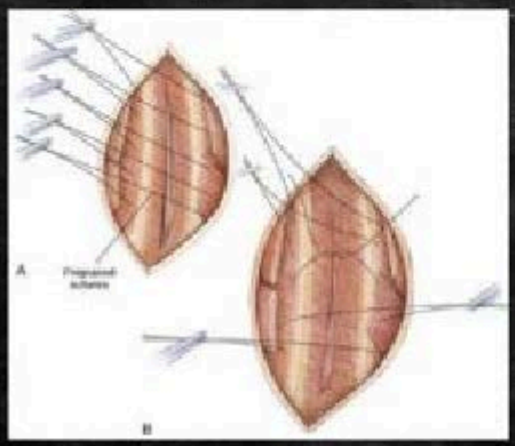


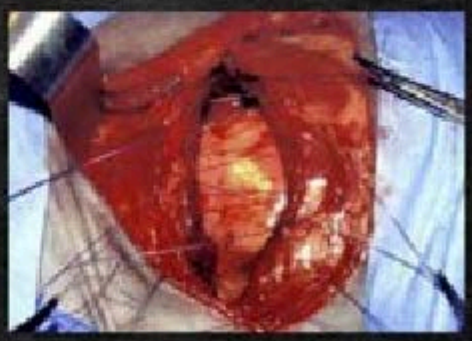


Closure of Intercostal Thoracotomy

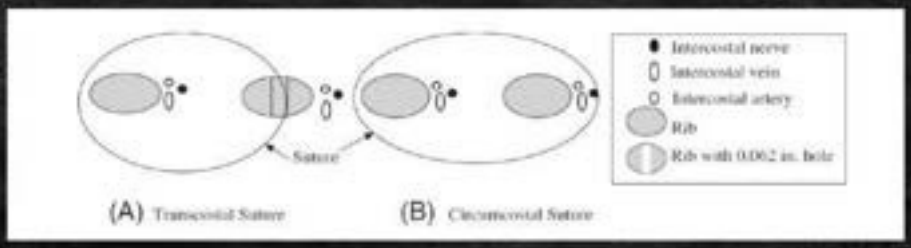
- Flush with warm saline
- Place chest tube prior to closing
- Circumcostal sutures



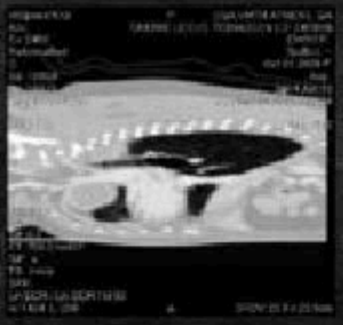


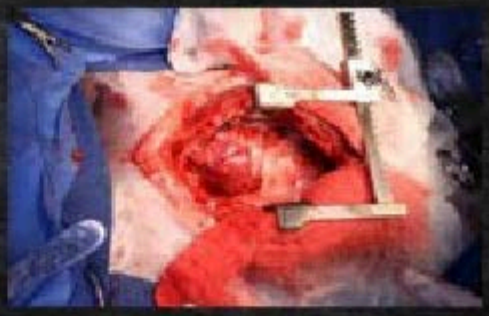


Transcostal suture technique



Rooney, Vet Surg, 2004



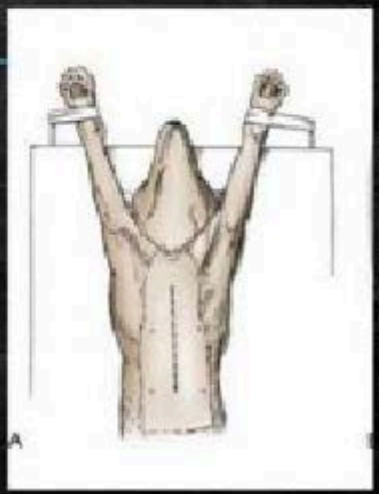


Median Sternotomy

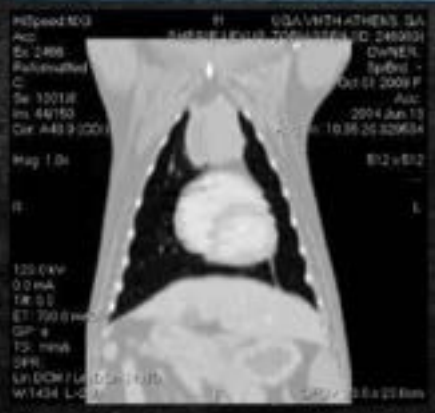
Technique and considerations

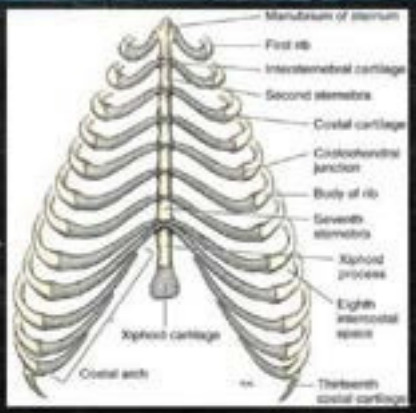
Positioning and Preparation

- Dorsal recumbency
- Shave and prepare entire thorax and cranial abdomen
  - Mid cervical area
  - Umbilicus
- Shave and drape laterally for thoracostomy tube



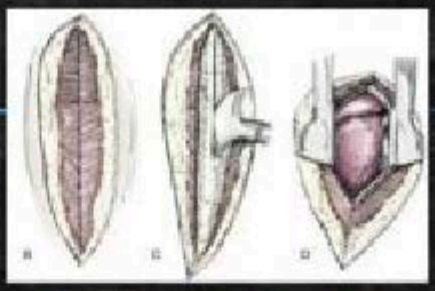
Cranial or Caudal approach?

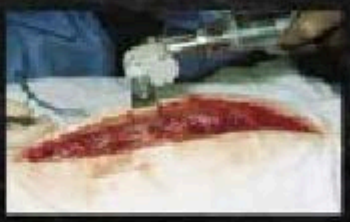


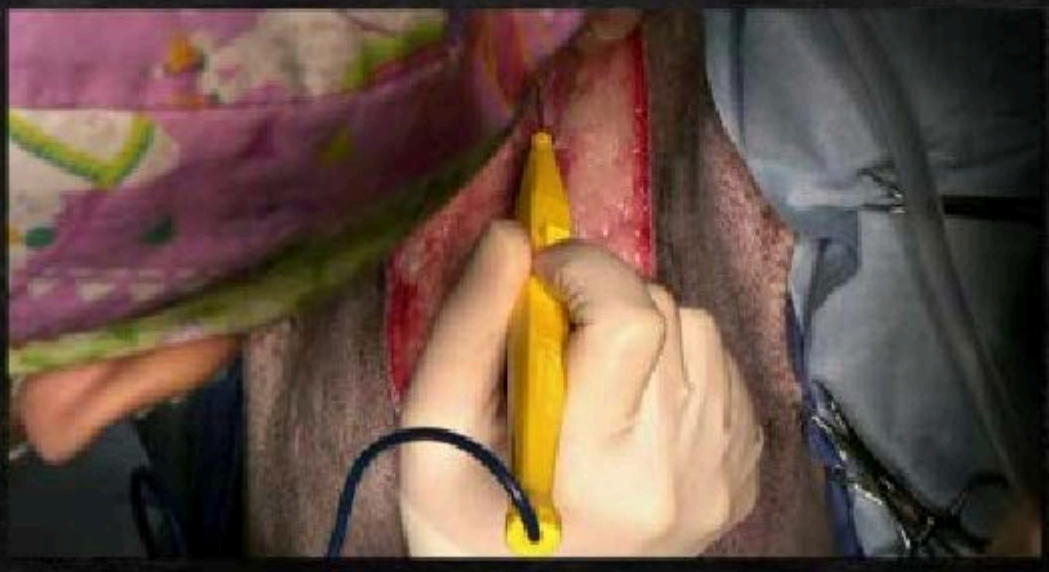


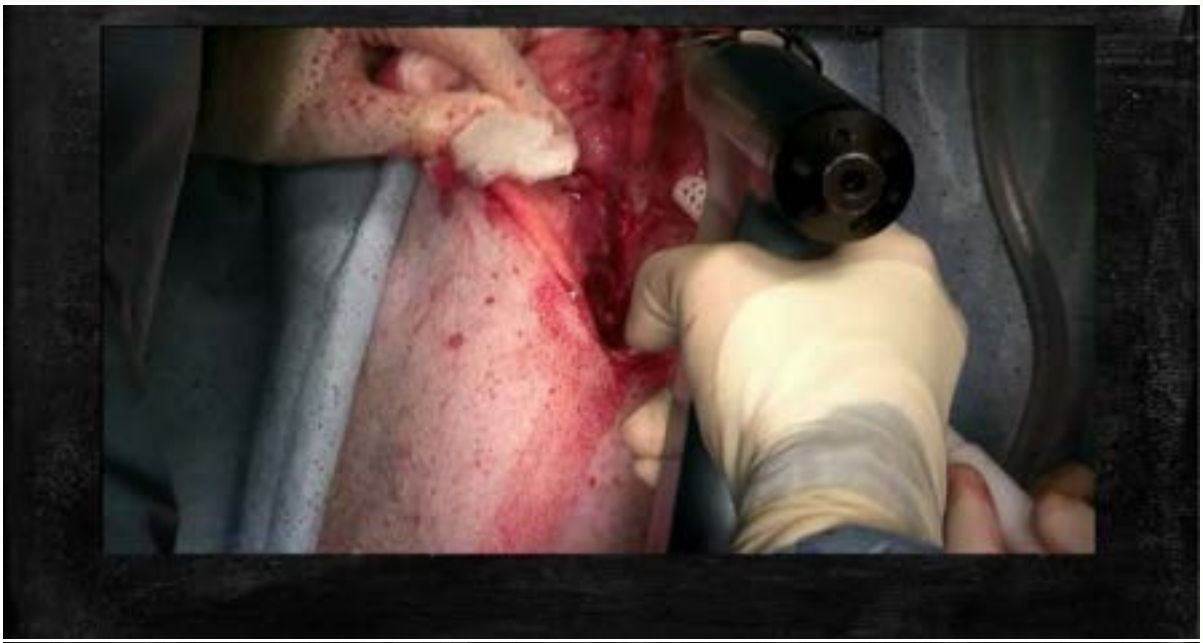
Technique

- Skin and SQ incised on midline
- Sternum is exposed and scalpel is used to create a groove for oscillating saw
- Freer elevator is used to help define the sternum
- An oscillating saw is used to split sternum on midline – leaving cranial or caudal sternebrae intact
- Can be challenging to stay on midline in small patients



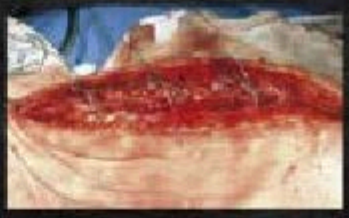


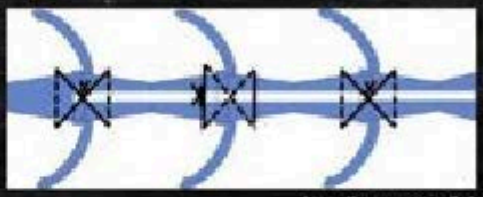


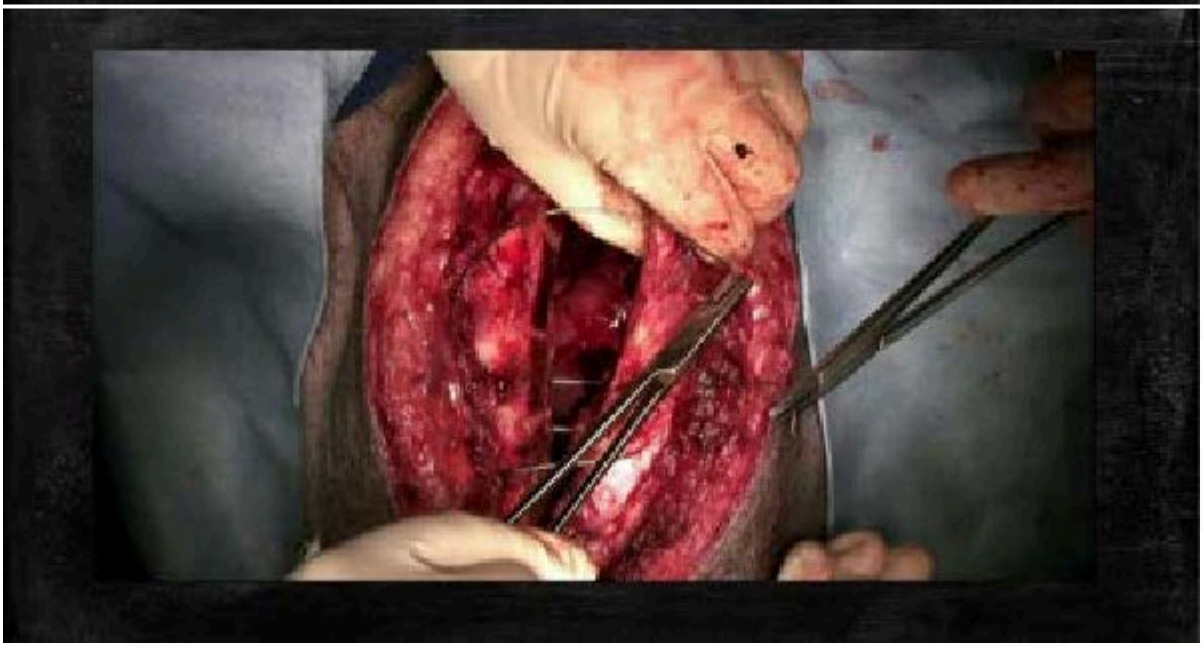


Closure of Median Sternotomy

- Flush thorax, place chest tube
- Paracostal figure of 8 pattern
- Orthopedic wire recommended 18 – 22 g
- Can use suture in smaller animals

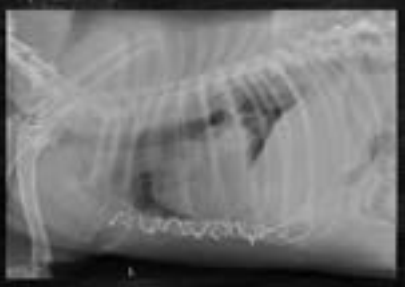


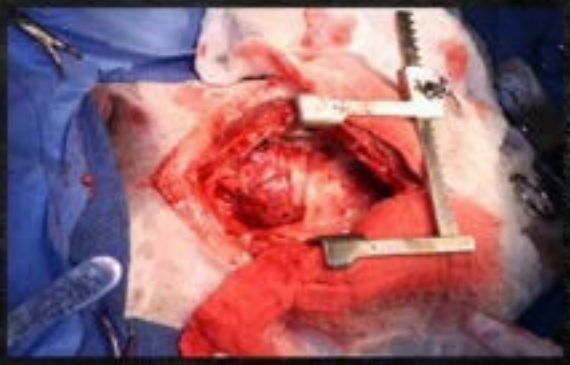




Closure – Median Sternotomy

- Robust closure of muscle and SQ
- Padded bandage



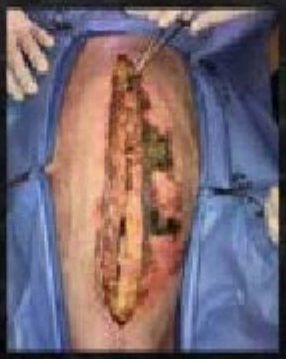


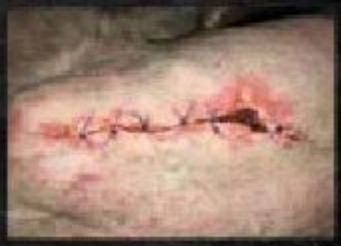
Post operative management

- Hypoventilation – support with oxygen
- Pain control
  - Continue treatment with NSAIDs and Opioids
  - Consider intrathoracic bupivacaine lavage - 1.5 mg/kg Q 8 hours
  - Remove thoracic drain when appropriate
- Keep wound covered

Median Sternotomy Complications

- Seroma
- Infection
- Hypoventilation

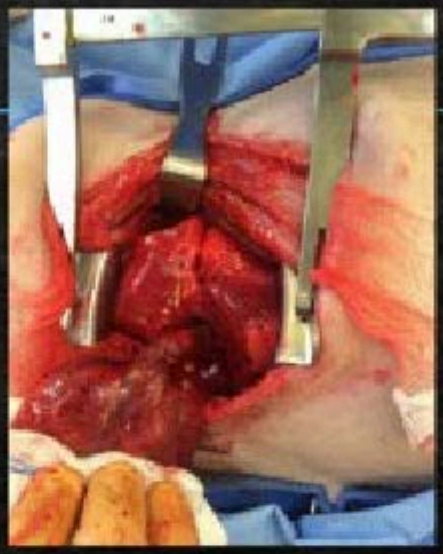




Select Procedures: Lung lobectomy

Lung Lobectomy

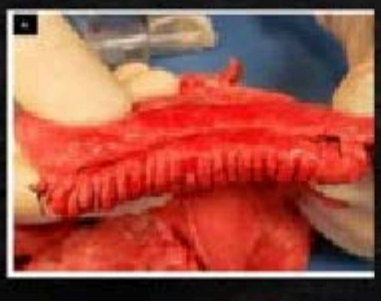
- Common indications
  - Lung tumor
  - Abscess
  - Torsion
- Hand Sutured vs. Stapled

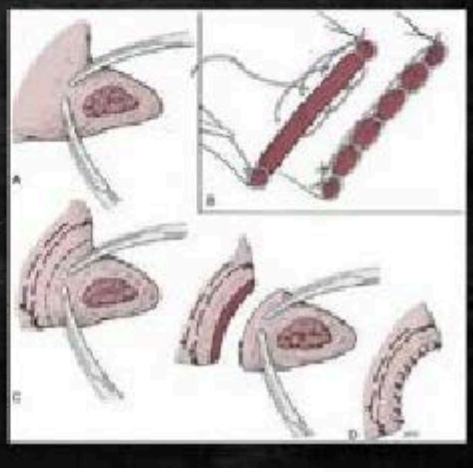


Hand Sutured Lung Lobectomy - Partial

Overlapping continuous mattress pattern

Oversew cut end

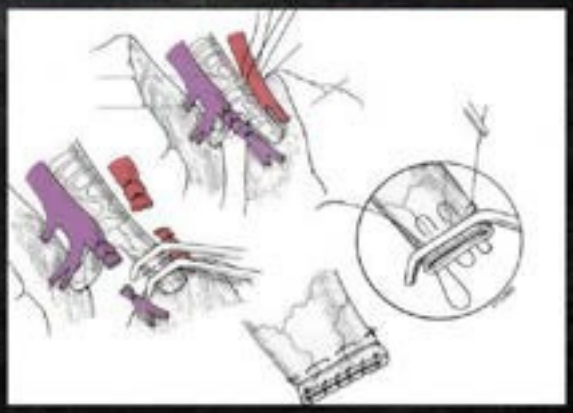




Hand Sutured Lung Lobectomy - complete

Individual ligation of

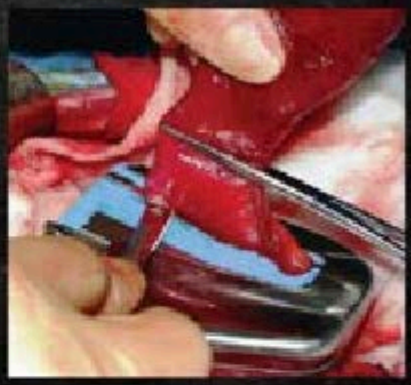
1. Pulmonary artery
2. Pulmonary vein
3. Bronchus



Small Animal Veterinary Surgery, 2nd ed

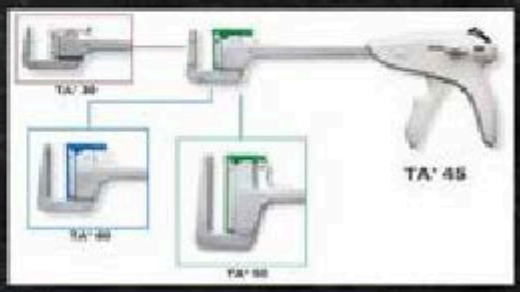
Stapled Lung Lobectomy

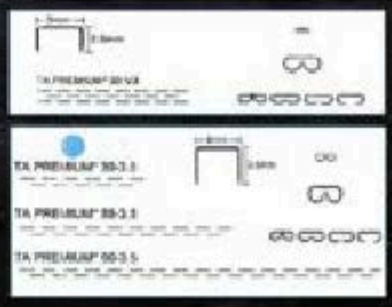
- One staple line ± oversew bronchus
- Typically with TA (thoraco-abdominal) linear stapler or GIA (gastrointestinal anastomosis)
- White or blue staples



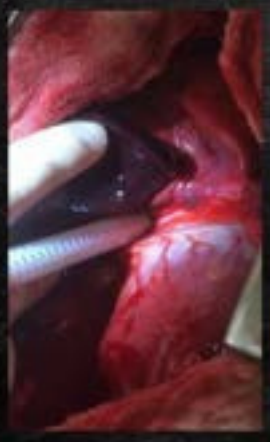
General technique

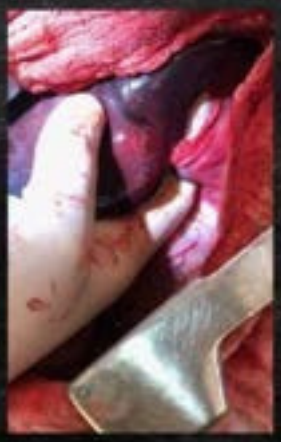
- Prepare stapler if needed
- Isolate smallest pedicle possible
- Apply stapler across pedicle
- Fire stapler
- Cut the tissue off with stapler in place
- Remove stapler
- ± Oversew (bronchi, lung parenchyma, intestinal wall, liver parenchyma)





Right middle lung lobe torsion
V3 cartridge – 'Snow White'





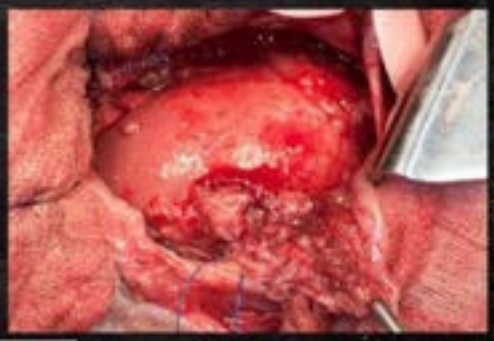
Prior to closure

- Consider lymph node biopsy
- Leak check with saline
- Count sponges

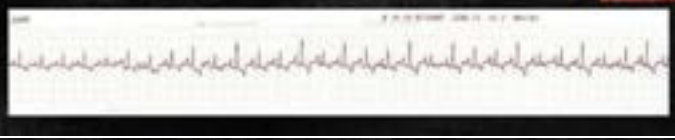
Select procedures:
Pericariectomy

Indications for pericardiectomy

- Pericardial effusion
- Chylothorax/restrictive pericarditis
- Pericardial neoplasia
- Pericardiectomy vs. pericardial window?

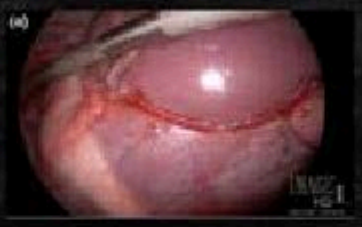


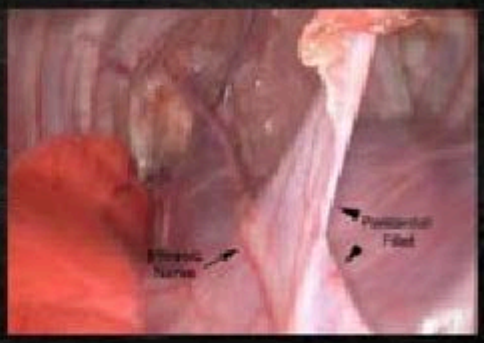
Pericardial mesothelioma



Pericardectomy technique

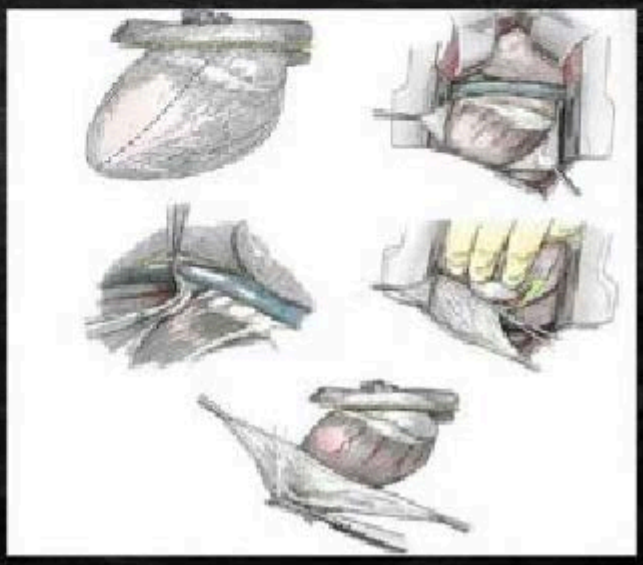
- Approach through right sided ICT or median sternotomy
- Subtotal vs. complete
- Fillet
- Window

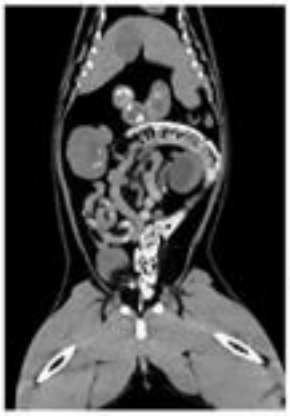




Subtotal Pericardiectomy

- Sharp dissection
- Bipolar cautery
- Caution with monopolar cautery!
- Pericardium is removed to the level of the phrenic nerve



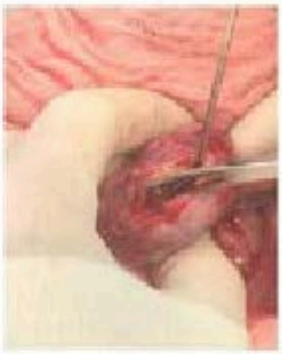


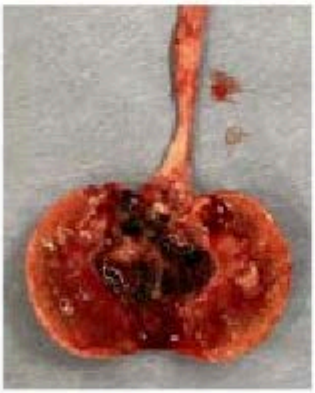
Surgery of the Urinary Track

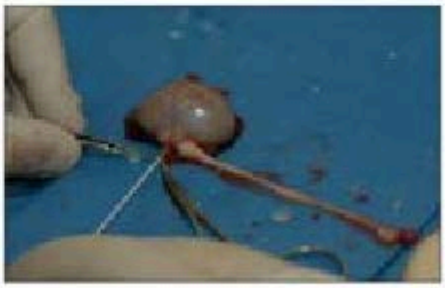
Chad Schmiedt DVM, DACVS-SA
Professor, Small Animal Surgery
Alison Bradbury Chair of Feline Health
University of Georgia, College of Veterinary Medicine

Lecture Outline

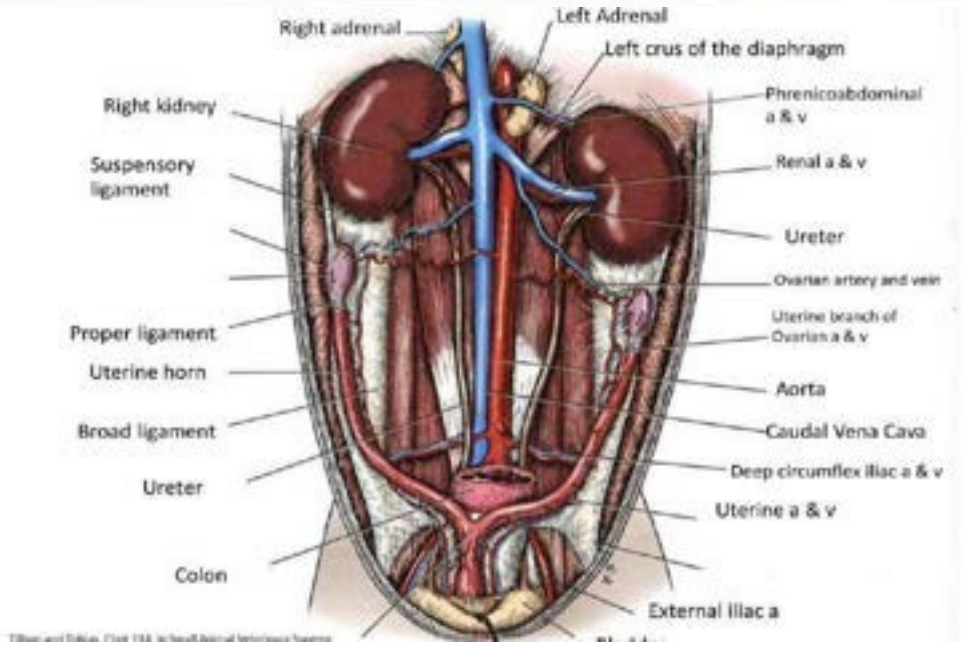
- Surgery of the kidney
  - Uretero-nephrectomy
  - Renal biopsy
  - Nephrotomy
- Surgery of the ureter
  - Ureterotomy
  - Ureteral reimplantation
- Surgery of the bladder
  - Cystotomy



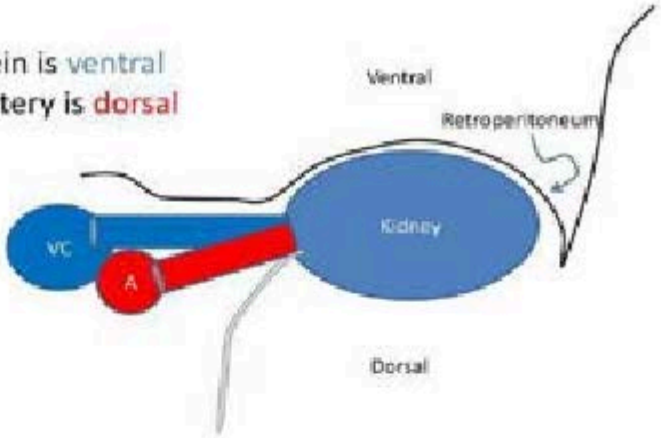




SURGERY OF THE KIDNEY

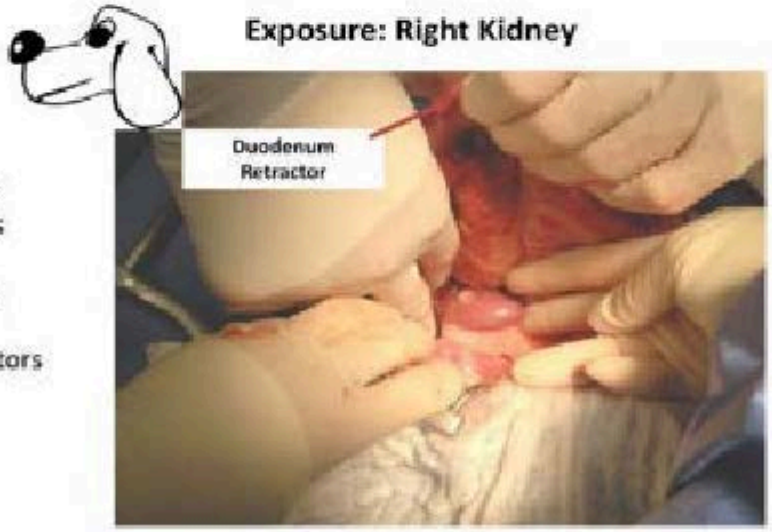


The renal vein is ventral
The renal artery is dorsal

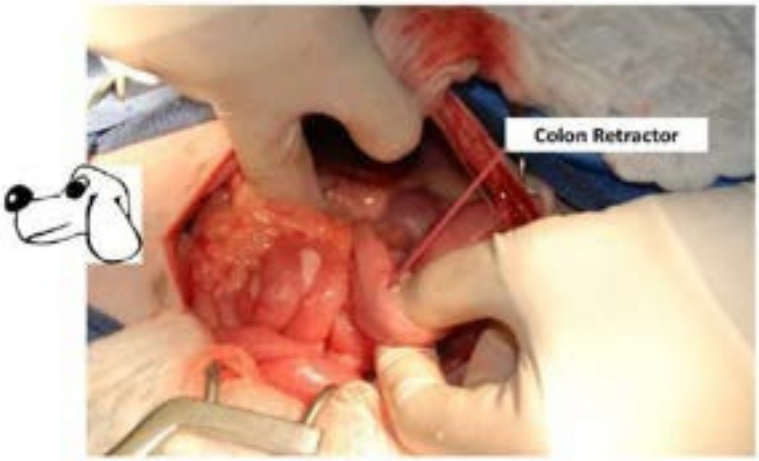


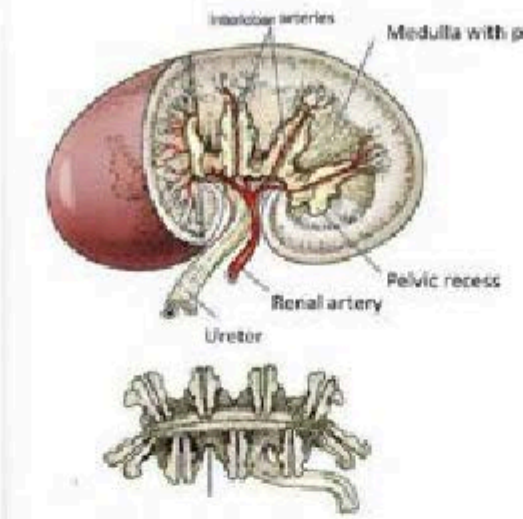
Exposure of the kidneys requires

1. Long incision
2. Use of retractors



Exposure: Left Kidney





Opportunities for bleeding and urine leakage

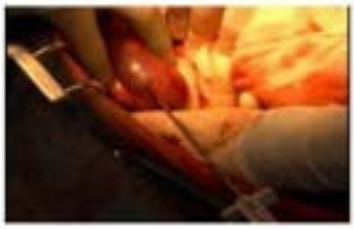
Renal Biopsy

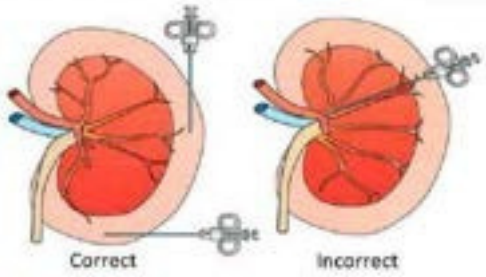
- Indications:
  - Renal neoplasia
  - Nephrotic syndrome
  - Renal cortical disease – protein losing glomerulopathy
  - Acute renal failure of unknown cause
- Contraindications:
  - Coagulopathy
  - Pyelonephritis
  - Ureteral obstruction
  - Hydronephrosis

Do the benefits outweigh the risks?

Will is change what you recommend?

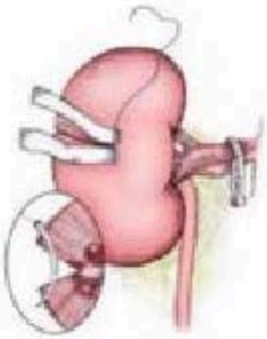
Ultrasound or laparoscopic guided renal biopsy

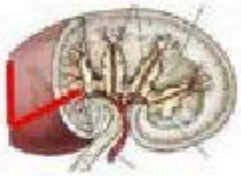




Open Renal Wedge Biopsy

1) Occlude the vasculature
   1) Fingers
   2) Vascular clamps
2) Small superficial sample
3) Close with a horizontal mattress
4) Sutures pulls through very easily
5) Apply pressure for hemostasis





Types of Renal Biopsy

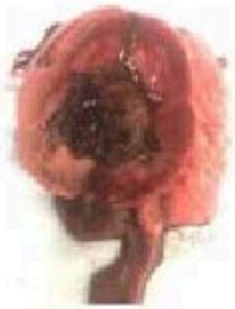
- Open wedge biopsy – best sample, ability to treat hemorrhage, most invasive
- Laparoscopic – good sample (14 g), insufflation reduced hemorrhage, specialized equipment
- Ultrasound guided – small sample (18g), unable to treat hemorrhage, least invasive

Complications of Renal Biopsy

- Bleeding!
  - Stay in cortex
- Small sample size – may not represent focal or multifocal lesion
- Crush artifact
- Urine leakage
  - Stay in cortex
- Results may not influence therapy

Ureteronephrectomy

- Preoperative considerations
  - Will the patient survive on 1 kidney?
  - Difficult to determine single kidney function
    - Nuclear scintigraphy
  - Remove ureter and kidney
  - Need excellent exposure
  - Need retraction

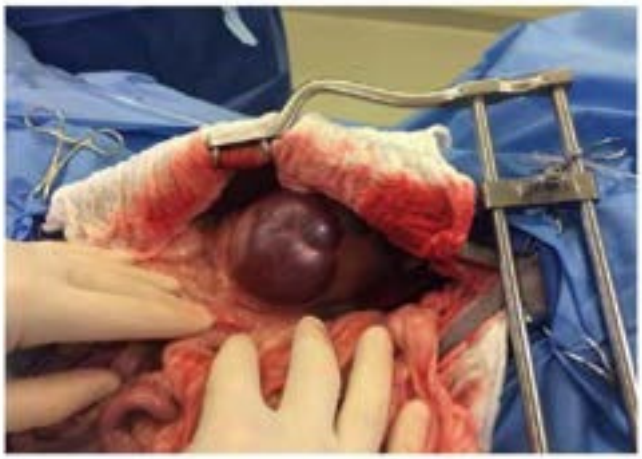


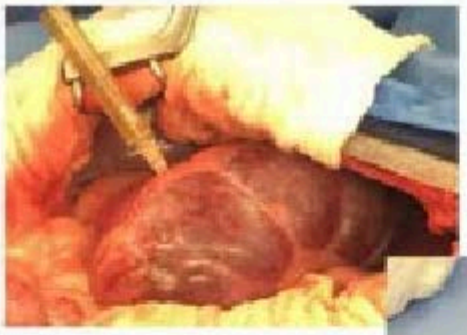
Ureteronephrectomy

- Indications
  - Severe infection (pyelonephritis)
  - Traumatic injury
  - Hydronephrosis

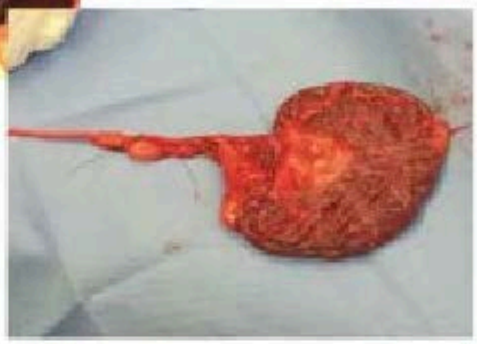
- Partial Nephrectomy (nephron sparing) an option in patients with reduced renal function

End-stage
Hydronephrosis

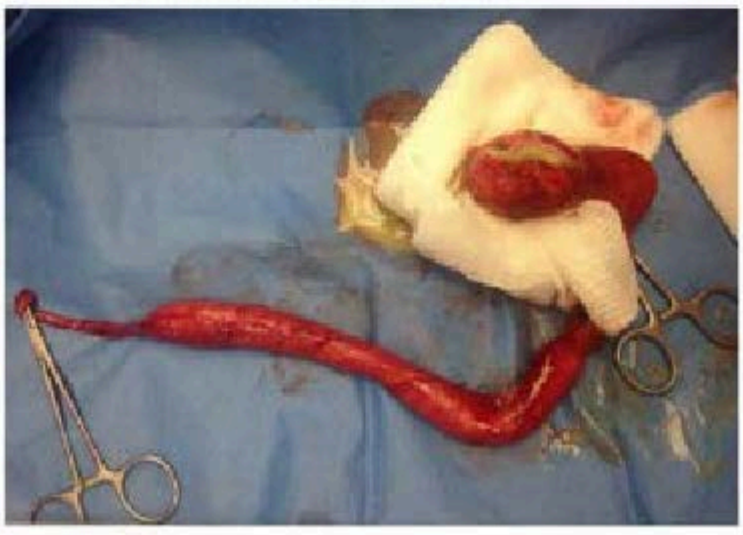


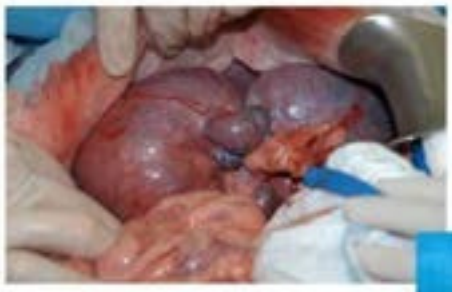


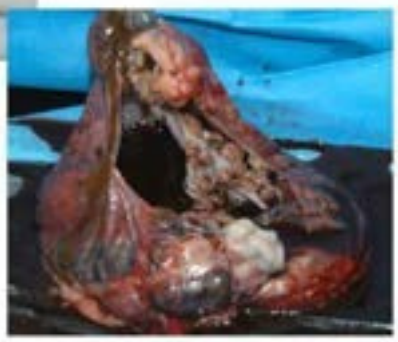
Hydronephrosis



Pyelonephritis and pyoureter

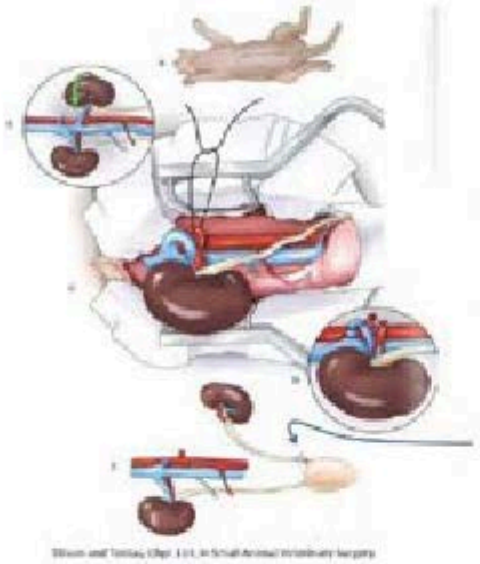






Renal papillary adenocarcinoma

Ureteronephrectomy: Technique



1) Ventral midline incision
2) Free retroperitoneal attachments and flip kidney medially to expose artery
3) Ligate artery
4) Ligate vein
5) Ligate ureter near UVJ

Why take the whole ureter?

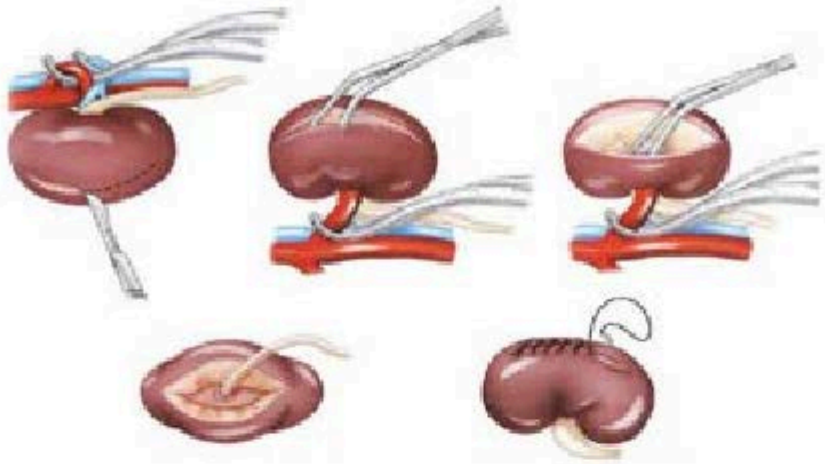
Complications of Ureteronephrectomy

- Bleeding!
  - Large vessels need good ligatures
  - Exposure!
- Leakage of urine
- Chronic infection
  - Remove all of ureter
- Renal failure

Nephrotomy

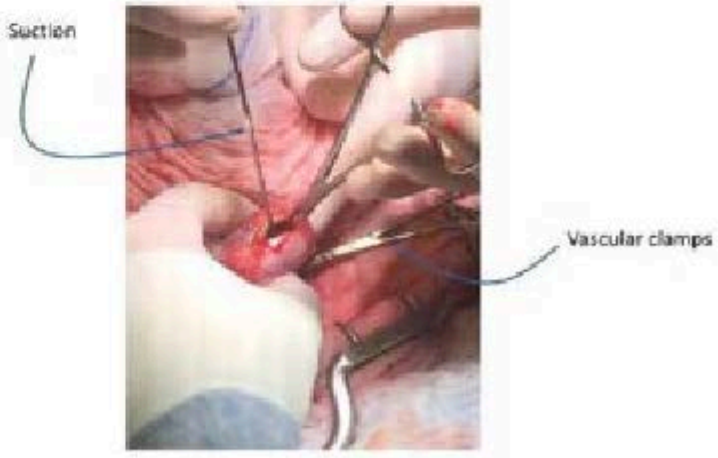
- Midline incision through the kidney to the renal pelvis
- Nephrotomy vs. nephrostomy?
- Indication: Removal of nephrolith that one cannot get through the renal pelvis
- Nephrolith removal indicated when:
  - Obstructive
  - Chronic/recurrent infection
  - Causing other clinical signs (hematuria)

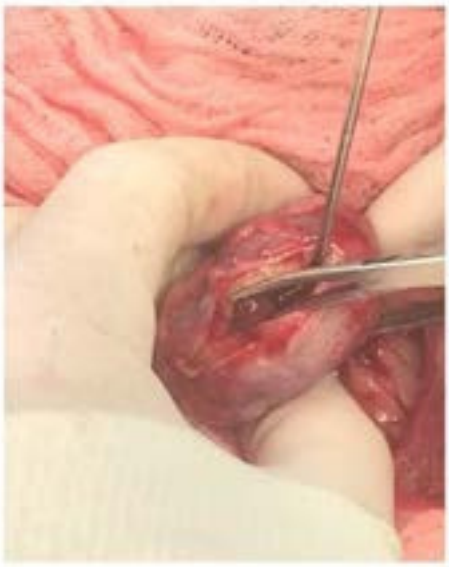
When would that be?



Consider pretreatment with Mannitol (0.5 g/kg) IV over 15 minutes just prior to placing vascular clamp.

Limit clamp time to – 15 – 20 minutes





Remove stone

Flush pelvis

Close just the capsule

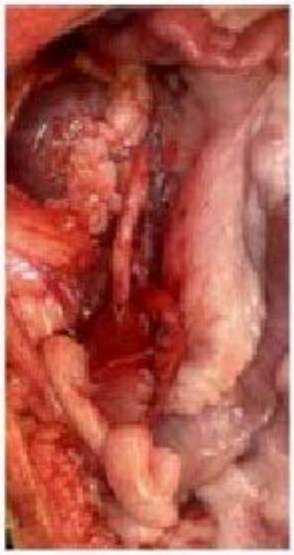
Release the vascular clamps

Complications of Nephrotomy

- Acute kidney failure
  - Ischemic injury
  - Parenchymal injury
  - Bilateral?
- Acute kidney injury
  - GFR is effected temporarily, may be minimal
    - Technique
    - Ischemia time
    - Hemorrhage
    - Anesthetic protocols
- Bleeding
- Urine leakage
- Ureteral obstruction
  - Debris from the stone flushed into ureter

What could go wrong?

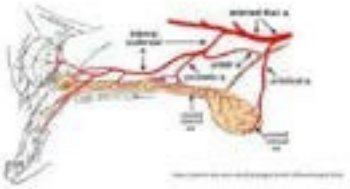
Ureteral Surgery

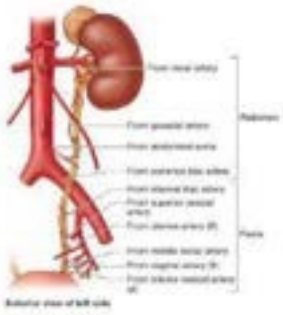


Ureteral Blood Supply

In people, blood supply is segmental with branches from renal, gonadal, vesical, vaginal, and uterine

In cats and dogs?
- Renal artery
- Prostatic or vaginal
- Cranial vesical





Circumcaval/Retrocaval Ureters

Present in 106/301 (35%) cadaver cats evaluated

Right side most common
- Right side = 30.6%
- Left 1.3%
- Bilateral 3.3%

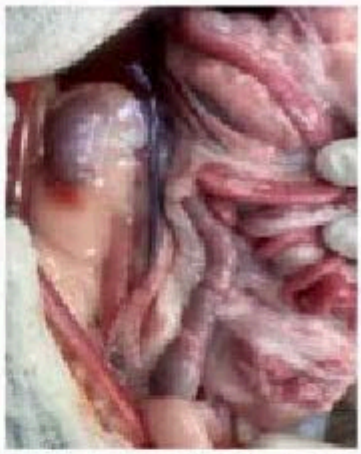
Double caudal vena cava = 7%

No sex predilection

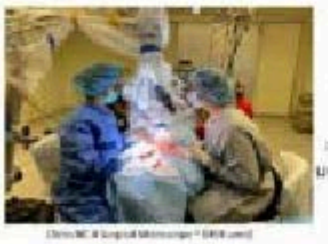
In cats with right circumcaval ureter, right kidney was longer (4.39 vs. 4.16 cm), but no evidence of hydronephrosis

Strictures more common cause of ureteral obstruction in cats with circumcaval ureters

Rarely reported in dogs



Ureters are Small



Surgical microscope

Variable magnification up to about 10×

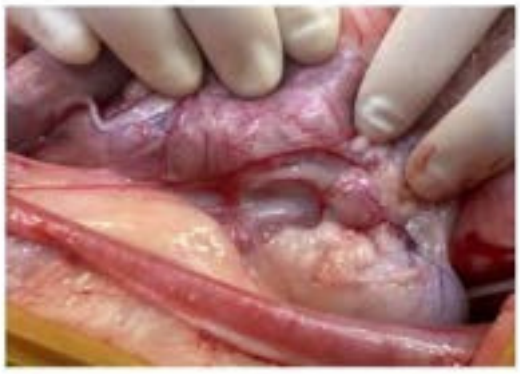
Surgical loupes

2.5-4.5 x magnification

Ureterotomy

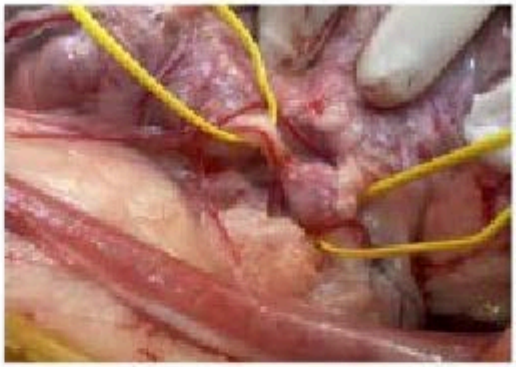
Good for-

- Proximal obstruction
- Bilateral obstruction
- Dilated ureter
- Patent ureter

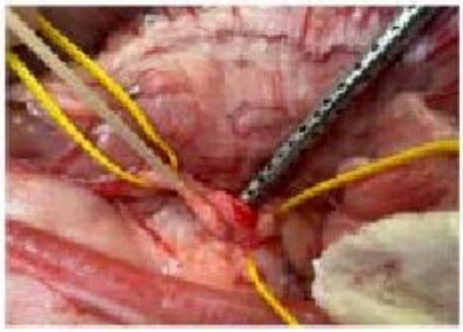


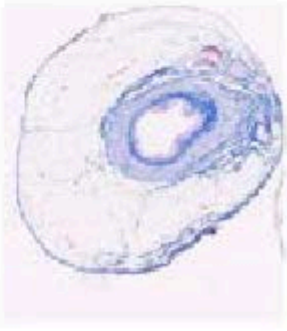
Isolate the ureter

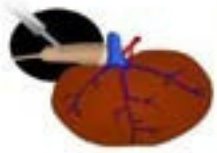
- Palpate the stone
- Careful dissection and preservation of the ureteral blood supply
- Isolate area with vessel loops

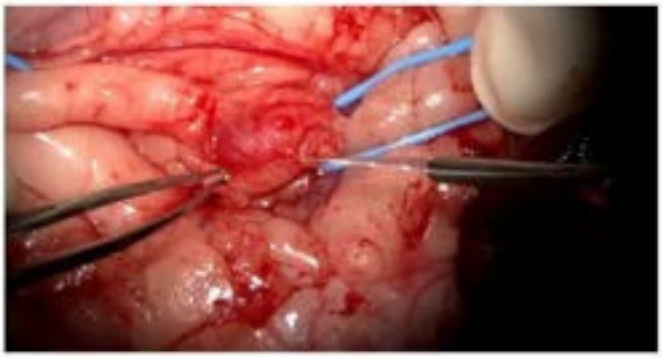


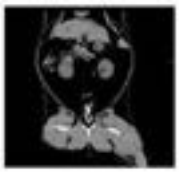
Incise through the fat to the ureter just proximal to the obstruction

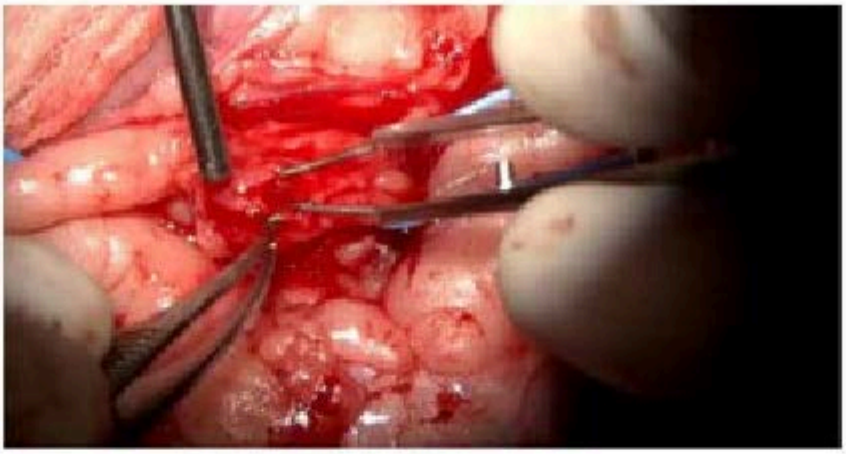


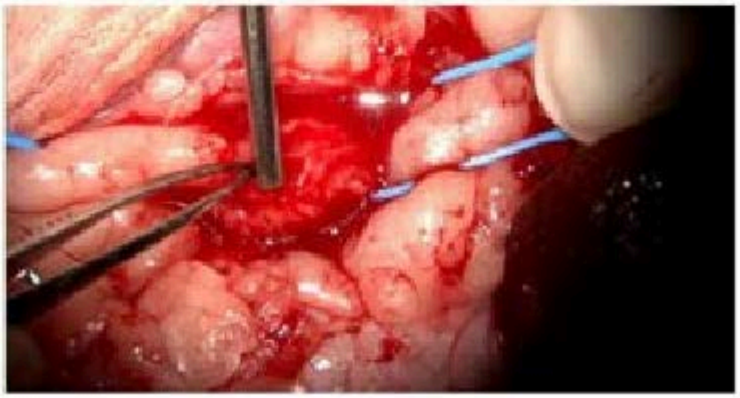




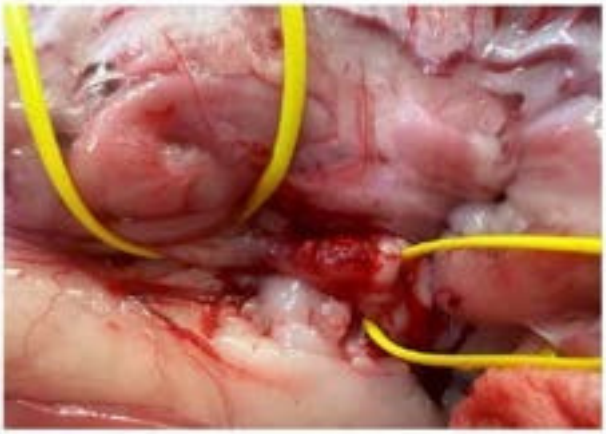








Closure, SI or SC over a stent or suture.

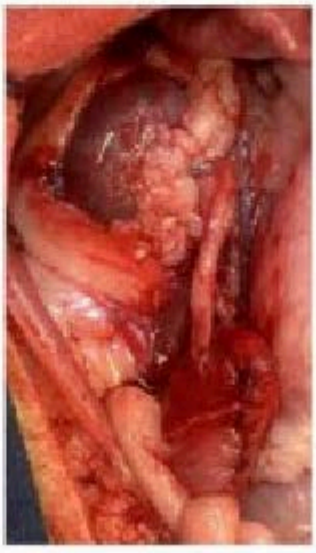


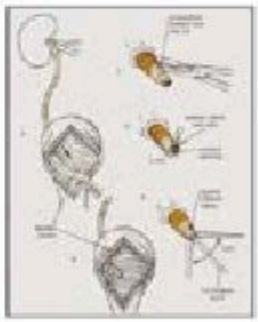
Complications of a ureterotomy

- Underlying kidney disease almost always present
- Urine leakage
- Ureteral stricture
- Re-obstruction
- Much better outcomes in dogs vs. cats.

Ureteral reimplantation

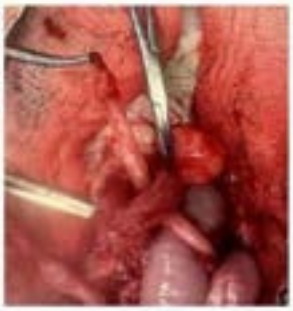
- Intravesicular
  - Smaller ureters
- Extravesicular
  - Larger ureters
- Extravesicular side-to-side
  - Proximal ureters
- Tension relieving techniques
  - All ureters
    - Renal dicalmus
    - Psoas hitch
    - Nephropexy
    - Nephrocystopexy



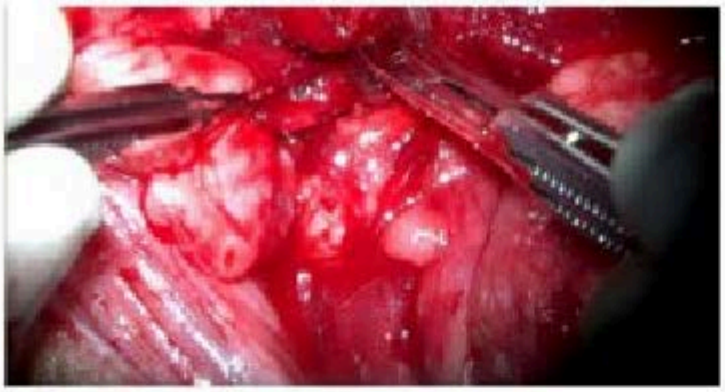


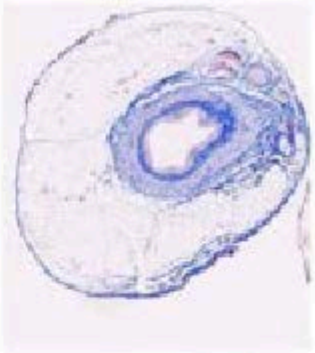
Intravesicular technique

- Ligate and divide ureter more distal than you intend to reimplant
- Push hemostats through bladder apex
- Pull ureter back through

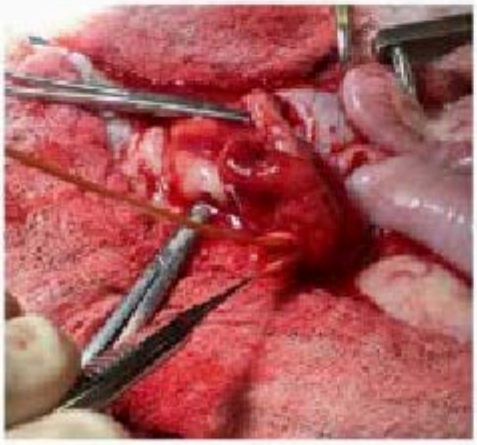


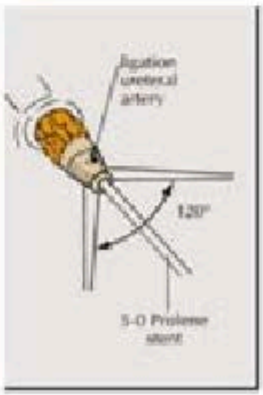
Debride peri-ureteral fat



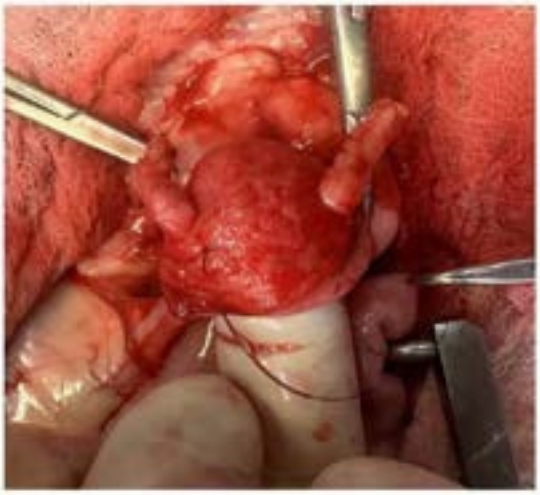


Spatulate the end of the ureter and ligate the ureteral vessel

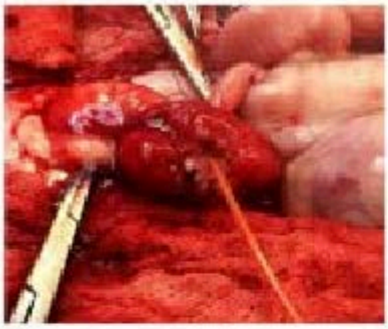


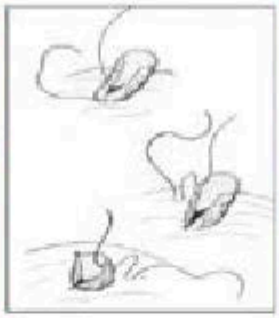


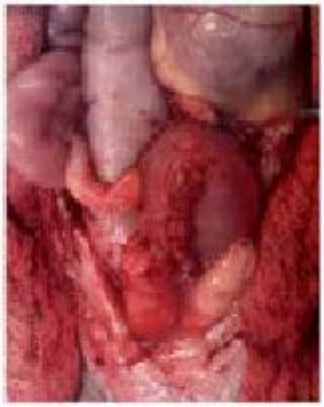
Invert the bladder so the mucosa is facing out



Suture the mucosa of the ureter to the bladder mucosa



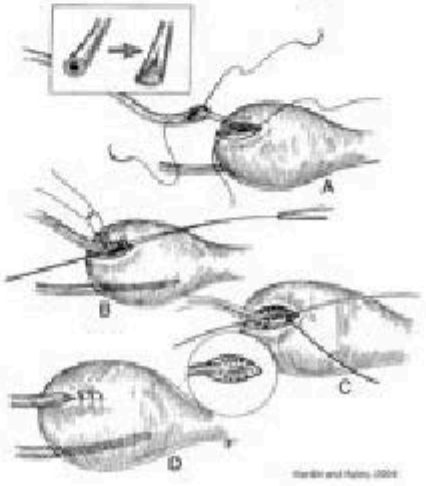




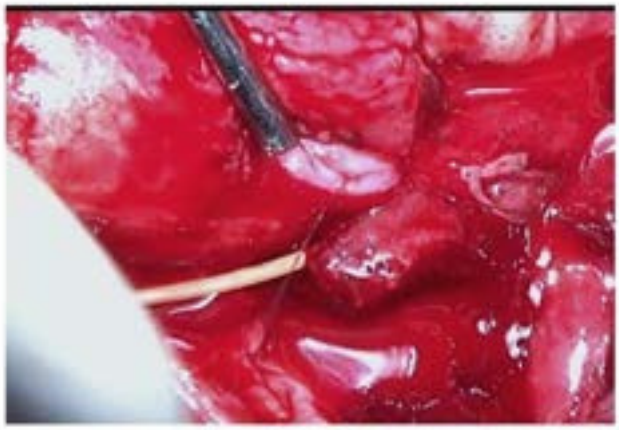
- superficial external sutures

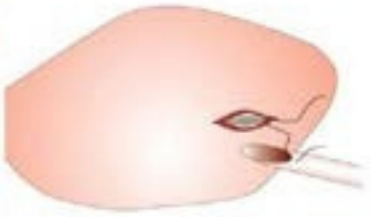
Extravesicular Ureteroneocystostomy

- Better for larger ureters (for me, anyway)
- Avoids a cystotomy



Extravesicular Ureteroneocystostomy

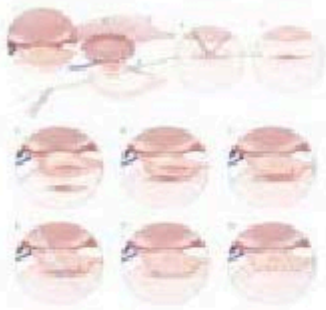




Side-to-Side Ureteroneocystostomy

Extravesicular, two-layer, side-to-side ureteroneocystostomy combined with tension-relieving techniques for feline proximal ureteral obstruction: A retrospective study

Kazuhiko Oyamada DVM[1] | Michiko Inoue DVM[1] | Kosuke Sato-Takeda DVM, Diplomate ACVIM[1] | Tsuyoshi Mizuno DVM, PhD[1] | Makoto Fujiki DVM, PhD[1]

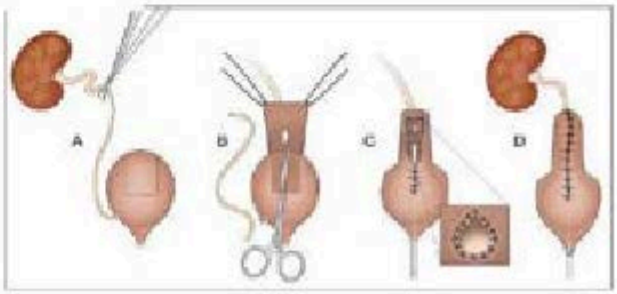


- 10 cats, all discharged
- Perioperative complications – ureteral catheter dislodgement (3), pollakiuria (2), dysuria (1)
- UTI post op – 3/10 cats
- Median follow up 648 days
- 7/10 alive without recurrent ureteral obstruction

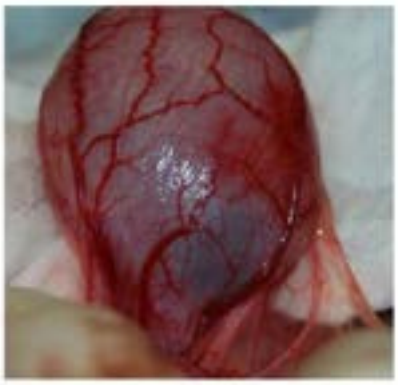
Use of a modified Boari flap for the treatment of a proximal ureteral obstruction in a cat

Lillian R. Aronson VMD, DACVS[1] | Ambrosine Clarson DMV, IPSAV[1] | Chloe Warnaar VMD, DACVS[1]

- 3 year old Russian Blue cat
- Proximal ureteral obstruction (mild ureteral edema, edema, periureteritis)
- One year follow up, normal imaging and biochemical profile



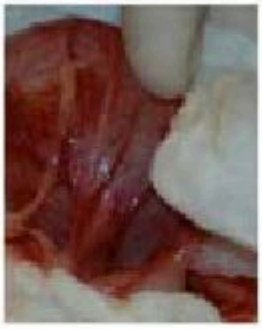
Aronson 2018

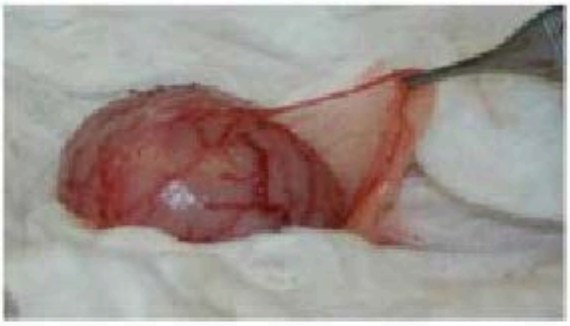


SURGERY OF THE BLADDER

Bladder Anatomy: Ligaments

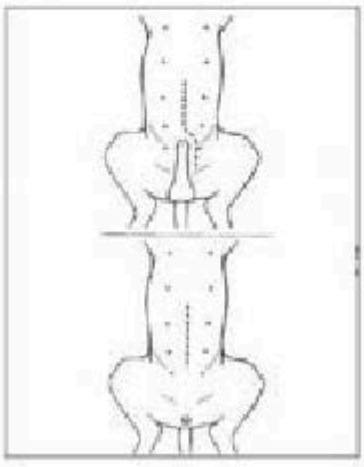
- Lateral ligaments – innervation, blood supply
- Median ligament – nice landmark





Surgical Approach: Cystostomy

- Standard ventral midline
- Paraprepucial – branches of Cd. superficial epigastric vessels
  - Prepucial ligament, vein, and muscle
- Think about whether a urethrotomy or urethrostomy may be required

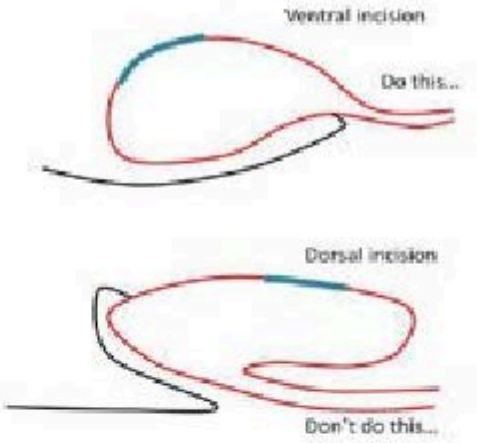


Principles

- Pack off the abdomen
- Stay sutures to manipulate the bladder
- Aspirate urine / use suction
- Catheterize the urethra
- Monofilament, absorbable suture
- One layer or two
- Holding layer – SUBmucosa
- Leak test

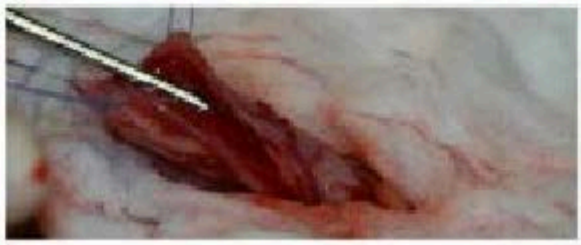
Cystotomy Dorsal vs Ventral?

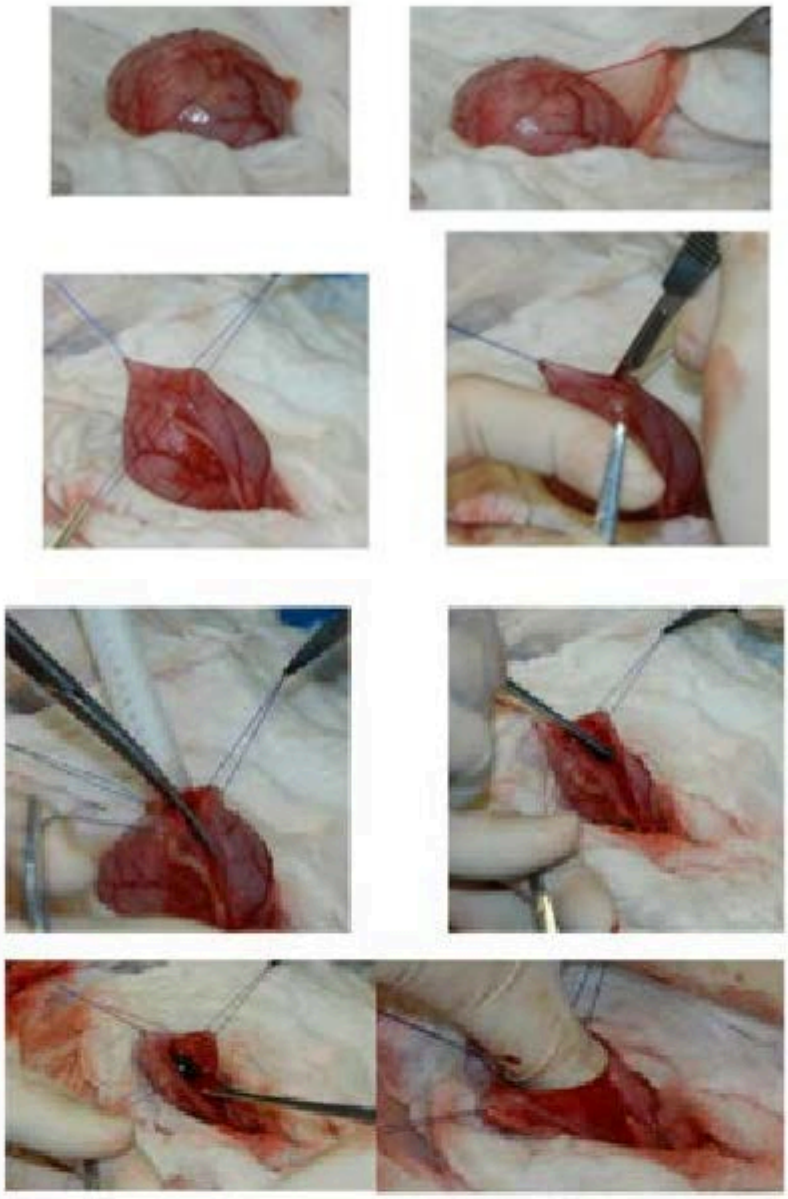
- Fears: gravity → leakage, adhesions, stones
- Pressure spreads out the force, so equal risk of leakage
- No difference in adhesions, use omentum
- Absorbable suture ↓ risk of stones greatly



Dorsal vs Ventral

- Dorsal cystotomy puts the hidden course of the intramural ureter at risk!
- Calculi roll down into the urethra – much easier to access with ventral cystotomy



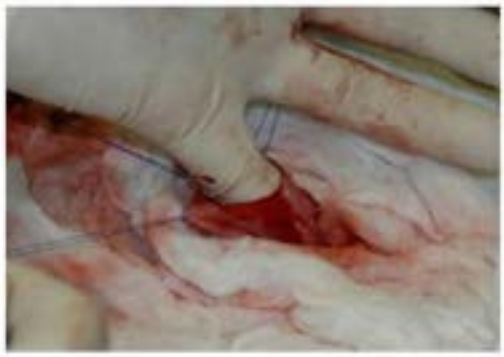


Evaluate the Urethra

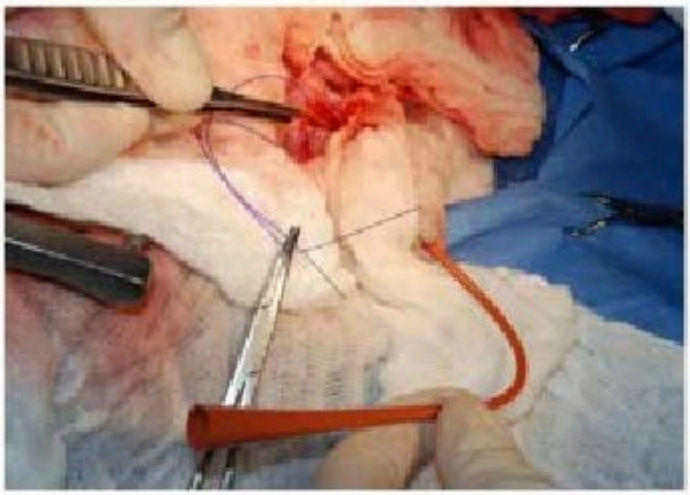
- Pass a catheter retrograde first!
- Flush retrograde – remember to "pressurize" the urethra
- Flush normograde & evaluate the stream

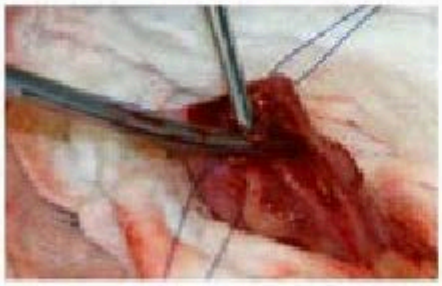
Make Sure ALL Stones are Out

- Palpate



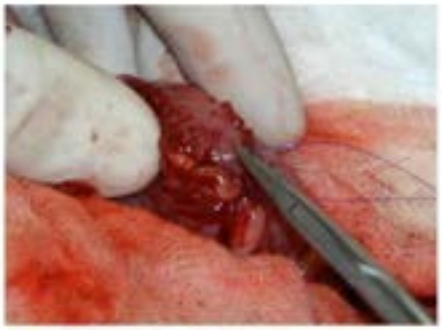
Urethral Catheterization

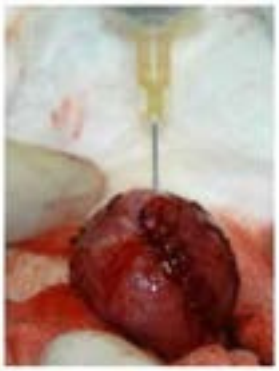


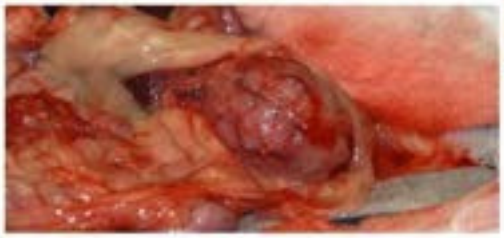


Submit samples

1. **Bladder mucosa – best culture**
2. **Stones – analysis ±culture**
3. **Bladder wall - histopathology**



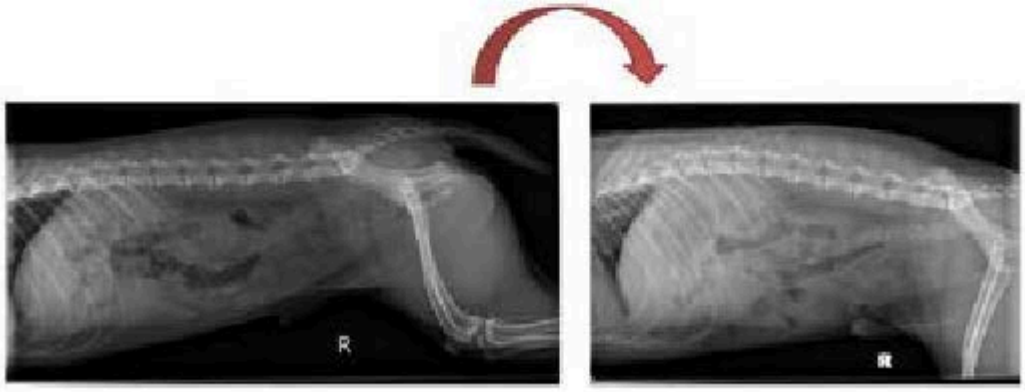




Single vs Double Layer Closure

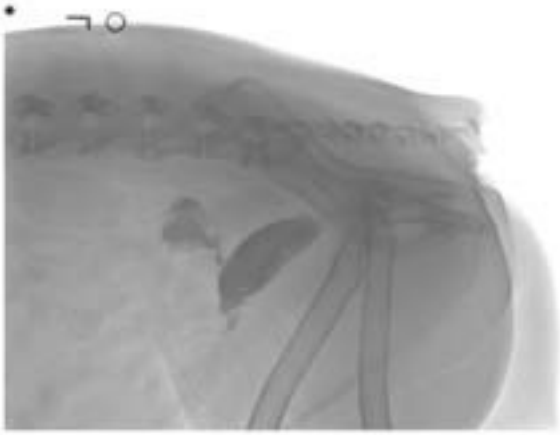
- Surgeon's preference
- Holding layer is SUBmucosa
- Options: full thickness simple continuous, continuous cruciate, 2 layers of simple continuous, simple continuous with an inverting oversew
- Small bladders = 1 layer

Always take post operative radiographs to confirm stone removal!



Complications of Cystostomy

- Urine leakage
- Stranguria/pollakiuria
- Hematuria
- Ureteral injury



Dr. Yathiraj Sreenivasa

BVSC, MVSc, PhD., Professor of Medicine,
Former Dean, veterinary college, Hebbal,
Regional Coordinator - Asia,
Commonwealth veterinary association,
Chief Executive, Lakeside Veterinary Hospital
and Research Centre, Bengaluru.

Dr. Yathiraj sreenivasa was born on 15.03.1955. Dr. Yathiraj is a doctorate in veterinary medicine and has more than 38 years of teaching experience. Has worked as Head of Department, Dean, Director of Research and Officer on Special Duty for establishing a veterinay college.

In the profession he has made his mark as top notch small animal practitioner and an academician of standing and repute. Has served as a member in WSAVA, Global Nutrition Committee. Has published very many Research articles in peer reviewed and referral Natanal/International Journals. Has participated in more than 45 TV programme and 35 Radio programme. Has delivered very many lectures in State. National and international conference/meetings and various forums. Recipient of more than 54 National/International awards.

Has participated in many training programmes.

Has worked ds member in various statutory authority/Universities, ICAR and VCI Institutions/Profesional bodies.

Area of Specialty:

Canine internal medicine, canine cardilogy, canine Nephrology, Dermatolo. Gastroenterology, Neonatal care and Alternate and Complimentary Veterinary Medicine practice.

Is Laser Therapy Useful In Veterinary Practice

S.Yathiraj
Former Dean (Vety) And Chief Executive,
Lakeside Veterinary Hospital and Research Centre
South End Road, Basavanagudi, Bangalore 560004, Karnataka, INDIA

Historical Background:

The therapeutic effects of light is known since the 6th century B.C. Treatment of diseases utilizing phototherapy won the first of many Nobel Prizes awarded in this field in 1903. In 1960, the first laser was developed. The first documentation of accelerated healing in veterinary medicine was done in 1967. By the early 1970's, laser therapy was recognized as a physical therapy modality in Eastern Europe, the Soviet Union, and China. The first appearance of laser therapy in the United States wasn't until 1977.

The World Association for PhotobiomoduLation Therapy (WALT) was formed in 1994 in Barcelona, Spain at the joint Congress of the International Laser Therapy Association (ILTA) and the International Society for Laser Application in Medicine (ISLAM) when these two international groups merged and WALT became the leading world body for promoting research, education and clinical applications in the field of photobiomodulation with lasers and other light sources.

Since receiving FDA approval in 2002, laser has gained widespread scientific and clinical evidence-based acceptance, accompanied by advances in the technology of the equipment utilized to deliver it. In India laser therapy has been put into practice for treatment of various conditions in companion animal practice since 2016.

Veterinary laser therapy is an innovative treatment that has gained popularity in recent years as veterinarians discover its benefits for pets. Used similarly to acupuncture, massage therapy, and other alternative therapies, laser treatment can be used in conjunction with, or instead of, medication to manage pain, inflammation, and wound healing.

Laser is an acronym of light amplification of stimulated emission of radiation (Laser). Laser when passed on cell or tissue alters physiology of cells and tissue by means of photons (light). Laser therapy lessens pain, relaxes muscle and improves circulation by altering the physiology of cells by means of light (photons). The effect of laser on mitochondria, cells and tissues is called photobiomodulation. Low level laser therapy (LLLT) refers to use of light at a much lower level than those used for tissue ablation or photocoagulation.

“Laser”—an acronym for “light amplification of stimulated emission of radiation”- refers to a unit that emits focused, penetrating light beams in three forms and is the unique property of laser.

- **Monochromatic:** Light that is a single wavelength (as opposed to natural light, which is emitted as a range of wavelengths)
- **Coherent:** Photons (i.e., tiny particles of light or electromagnetic radiation) that travel in the same phase and direction (tightly aligned)
- **Collimated:** Photons that travel in a single straight beam (parallel)

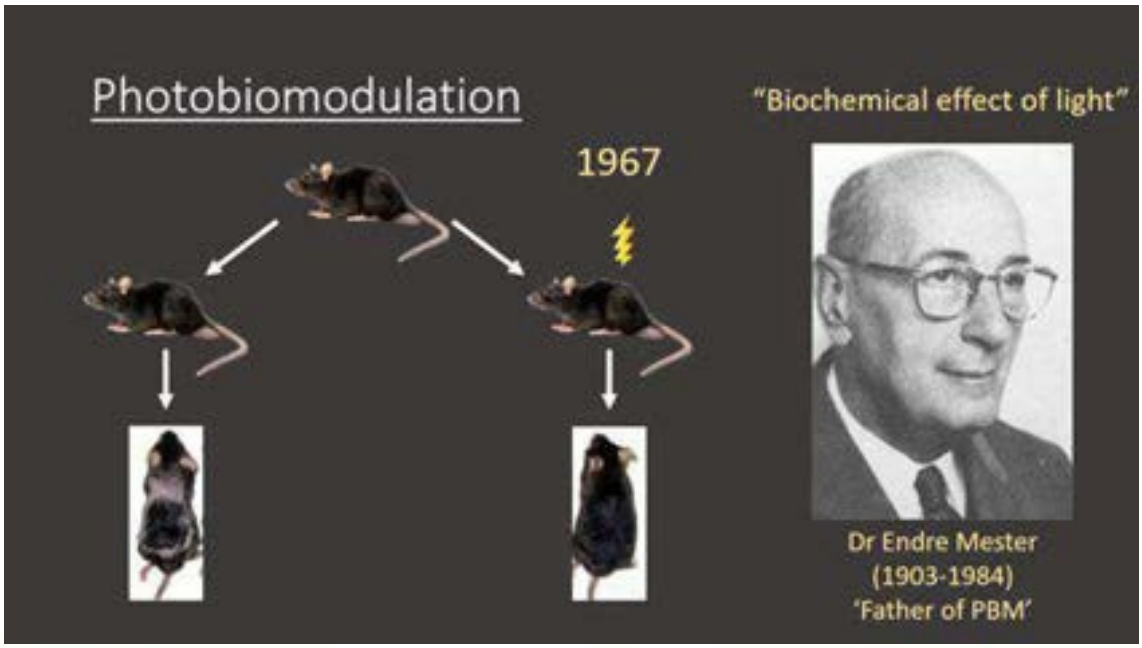
Coherence and collimation give a laser penetrating power to a restricted area so that nearby tissues are unaffected.
Lasers are classified based on their wavelength and potential energy output, with four classes currently recognized:

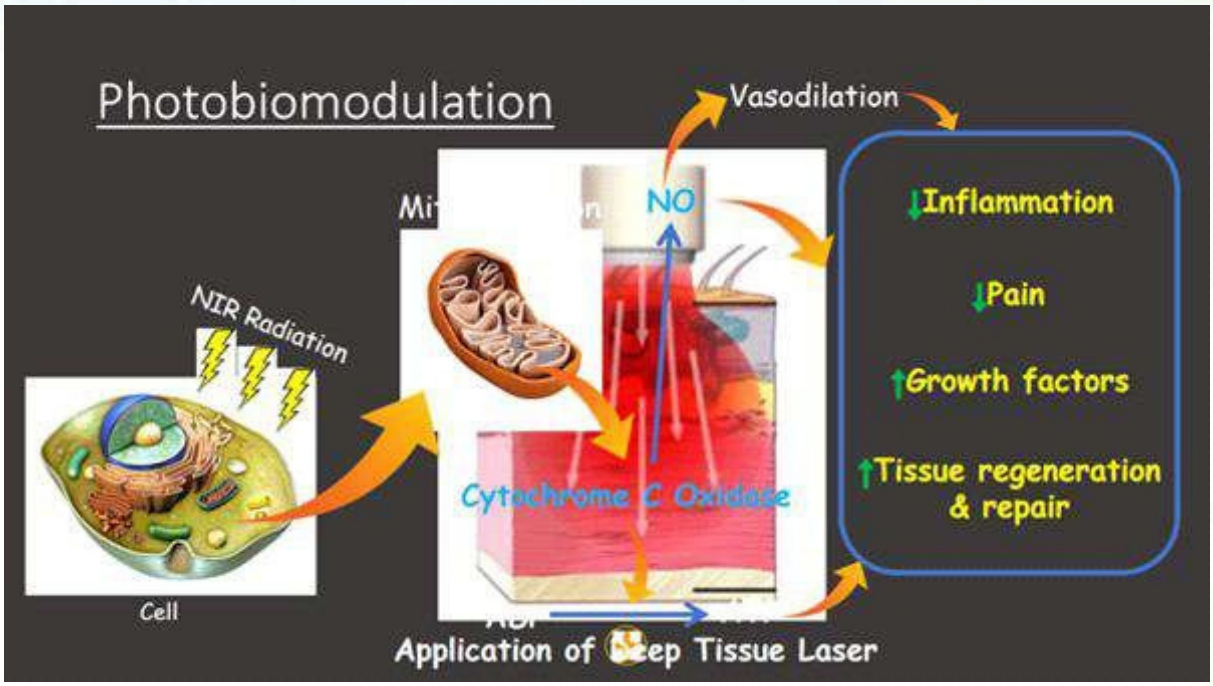
- **Class 1** lasers, such as barcode scanners used in supermarkets, are used safely every day
- **Class 2** lasers, which include laser pointers and some therapeutic lasers, produce a beam in the visible spectrum (400–700 nanometers)
- **Class 3** lasers include the most commonly used therapeutic lasers
- **Class 4** lasers are currently , most commonly used for therapeutic purpose and includes surgical lasers used to cut and cauterize tissue during surgical procedures

Laser therapy helps tissue repair by causing the following:

- Endorphin release
- Vasodilation, which increases blood flow to bring in oxygen and cells involved in the healing process
- Muscle relaxation
- Decreased inflammation
- Faster healing and repair

The main clinical benefits of laser use in pets include decreased inflammation, decreased pain, and improved wound healing.
Many units use red or near infrared light (600 to 1070 nm) but blue, green and violet light (around 400nm wavelength) are also becoming popular. Most commonly used therapeutic lasers in Veterinary Medicine are Class III (deliver energy from 1 to 500mW) and IV (delivers energy more than 500mW) lasers. One watt is one Joule of energy that is delivered per second and the laser dose is expressed as Joules/cm2.





Physiologic Effects

Tissue Regeneration and wound healing	Analgesic/anti-inflammatory effect
• ↑ATP production – needed for cellular metabolism	• Endogenous opioid release
• ↑ROS and RNS – downstream signalling from mitochondria to nuclei – regulate nucleic acid synthesis, protein synthesis, enzyme activation and cell cycle progression	• ↓secretion of endogenous noxious substances like bradykinin and histamine
• ↓apoptosis; ↑cell proliferation, adhesion, and migration	• ↓NCV of pain transmitting fibers
• ↑fibroblast activity with accelerated collagen deposition	• ↑pain threshold
• Neoangiogenesis	• NO release from CCO binding site – vasodilation
• Modulation of prostaglandin secretion	

Laser increases platelet-derived growth factor (PDGF) and basic fibroblast growth factor (bFGF), among other growth factors, which have a stimulating effect on the growth of fibroblasts. The magnitude of this increase is about threefold to sixfold compared to control cultures in some studies. There will be more fibroblasts, and they will be working more efficiently, producing more collagen. LT improves tensile strength by increasing the amount of type I collagen.

Benefits of Therapeutic Laser:
Neovascularisation
Angiogenesis
Collagen synthesis which enhances wound healing
Stimulation of nerve healing
Enhanced healing of tendons, cartilage and bones
Reduced swelling from injury
Modulation of degenerative tissue changes
Mitigation of CNS damage following traumatic brain injury and spinal cord injury

Therapeutic laser has been used to treat conditions like:
Superficial skin wounds/ Aural Hematoma/ pyotraumatic dermatitis
Post surgical wounds
Gingivitis/periodontal disease/stomatitis (closed mouth)
Deep tissue and musculoskeletal conditions
Rhinitis/Sinusitis (closed mouth)

Abdominal disorders
Tendon and ligament injury
Trigger points
Edema
Anal sacculitis
Lick granuloma
Muscle injuries
Neurological conditions
Osteoarthritis
Pododermatitis
Otitis

Laser therapy is particularly useful for pets with limited medical treatment options, such as:

- Pets with liver disease who cannot take medications
- Cats, for whom only a few pain-control medications are approved
- Exotic pets for whom medication administration is difficult or impossible
- Older pets with diminished organ function

Preparation for Laser Therapy for Dogs
Most dogs do very well in laser therapy and do not need preparation before treatment. If the pet has a lot of anxiety, administer anti-anxiety medications one to two hours before the session. Staff handling the animal should wear goggles and patient should also wear goggles when necessary.

How are laser treatments administered?
Success of laser treatment depends upon administraion of appropriate target dose of laser. Select appropriate accessory treatment head (small non contact up to 3W, s,all contact up to 3W, large contact up to 15W, large non contact up to 15W)

Calculation of therapeutic dose of Laser

As per WALT cells need 5 to 7 joules/cm2 to stimulate a photochemical response in human and the same is being practice in animals. Generally 5 joules/cm2 is used in animals. One watt gives 1 joule per second.

$$\frac{\text{Power(W) x Time (seconds)}}{\text{Area of treatment (cm2)}} = \text{Joules/cm2}$$

Area calculation: Example

1.Pyotraumatic dermatitis: let us say length is 6 cms amd width is 8 cms. Then area of treatment will be 6x8 = 48cms. Laser required will be 48 x 5 (5 joules/cm2) ie 240 joules
Duration of treatment if set at 6W. One minute will give 60x6 joules ie 360 joules/minute
Duration of treatment at 6W will be Total joules/360 i.e 240/360 = 0.66 minutes
2.Labrador, lumbar area pain:
Width of lumbar area 15 cms, length of lumbar area 25 cms
Total area 15 x 25 = 375 cms , laser required 375x5 ie 1875 joules
If set at 6W duration will be 1875/360 = 5.20 minutes
If set at 8W duration will be 1875/480(60x8=480)= 3.90 minutes.

General treatment principles:

Do not treat through casting or bandage material
Utilise a grid like pattern, evenly treating an area, perpendicular to the target tissue
Continuously move the hand piece at all times (1-3 /sec) regardless of protocol
Utilise appropriate treatment settings concerning skin and coat color
Maintain proper distance from treatment area for non contact treatments
Utilise small accessory treatment heads only at power settings of 3W Or Less
Utilise small and /or large cones as non contact hand piece attachments
Any topical sprays, ointments, or lotions should be rinsed off from treatment area prior to initiating laser application

The most important point to remember prior to initiating laser application is to calculate the dose of laser and duration of treatment. This depends on the weight, condition of the animal, length of hair, color of hair and condition being treated (post procedure/acute/chronic)

During a treatment session, the handheld laser wand is slowly moved back and forth over the damaged tissue, producing a warm, pleasant sensation that most pets seem to enjoy and find relaxing. Sessions duration and frequency depends on the condition and severity being treated. For treating superficial conditions duration is usually 1- 2 -5 -10 minutes and deep conditions 2 - 15 minutes. While treating large area/deep conditions in giant breeds, sometimes it may extend up to 15 to 30 minutes). Initial treatment is on daily basis or alternate day basis for few days to weeks, depending on the improvement. Post procedure conditions require normaly 1 or 2 sessions daily. However if there is more extensive tissue disruption additional treatments (2-6) on alternate days. Acute conditions (pyotraumatic dermatitis, mild otitis, abscess with out surrounding cellulitis) are treated once daily or multiple times till the condition resolves. Sometimes more sessions are required on alternate days (2-4). Chronic conditions may be treated daily or on alternate days until improvements are seen or otherwise and subsequently at weekly intervals based on need. Weekly.

Is laser therapy safe for pets?

Laser therapy is safe if performed correctly, using the proper settings and treatment durations. Higher-powered units can cause thermal burns to tissues if used incorrectly. Also, laser beams directed at an eye can cause permanent retinal damage, so patients and all veterinary staff must wear protective goggles during treatment.

Contraindications for laser therapy

Tattoos,
Carcinomas,
Thyroid gland conditions

Do not apply laser over an active haemorrhage area , abdomen of pregnant patient , implanted cardiac device , thorax of heart patient , testicles and autonomic nerve centres. In immature patients (less than one year) it may stimulate premature closure of epiphyses. In patients where immune stimulation is not desired including those with lymphoma or on immunosuppressant medications.

Controversies

1. The unproven claims related to pulse therapy
2. advantages of low or high powered units
3. long term safety of high powered treatment.
4. Duration and frequency of treatment

VET EXPERT

THE BEST CHOOSE BEST

VET EXPERT

SPECIALISED SUPPLEMENTS FOR DOGS AND CATS

- comprehensive support
- ingredients with scientifically proven effect
- convenient application **(Twist off capsule)**

Imported and Marketed By:

Vet Planet India Private Limited

Ph: 9711140505, 7827836303

UNIQUE DIETS VET EXPERT ELIMINATION

DEDICATED FOR DOGS WITH FOOD INTOLERANCE

MONOPROTEIN
GLUTEN FREE
HIGHLY PALATABLE

OPUSPET
PASSION UNITES INNOVATION

Mervue
LABORATORIES - Est. 1986

BEST in class IMMUNITY builder & booster

MultiBoost

Specially formulated for immunity & general well-being of Dogs

NON GMO

Presentation: 150 ml

A MUST FOR

- New born / growing pups
- Improved response to vaccination
- Pregnant dogs
- Lactating dogs
- Post surgical recovery

HIGHLY BIOAVAILABLE FORMULATION

1st ever Multivitamins & Multiminerals FORMULATION FOR CATS

MultiBoost

Specially formulated for immunity & general well-being of Cats

NON GMO

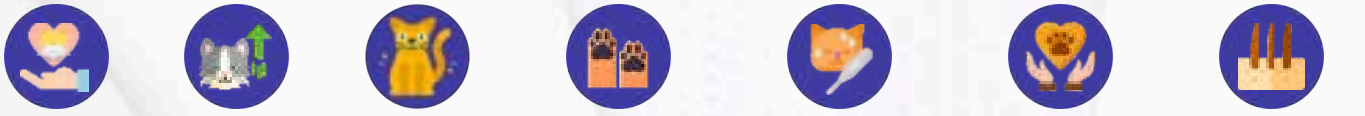
Presentation: 150 ml

A must ✓ have for Every Life Stage of Cats

1. New Born
2. Post weaning
3. Growth & Development
4. Motherhood
5. Prime years
6. Senior years

CONTAINS Chelated Minerals | Taurine | Carnitine | Omega 3 & 6 | Vitamin B | Vitamin C

✓ Safe for all Ages ✓ Safe for all Cats ✓ High Palatability



Marketed by: OPUS PET PVT. LTD.
Office No. 8, Sunrise Apartment, SB Road, Pune - 411 016, India
Customer Care No. +91 20 4676 6676 Email : info@opuspet.in www.opuspet.in
OpusPet opus.pet

With best
compliments from:

Boehringer
Ingelheim

OriHeal
LIFESCIENCES

Happy Pet Solutions

"Health to Happiness"

Importer & Distributer of
High Quality Imported Products
For Small & Large Animals

Unique Muzzles

Digital Ultrasound

Tubular Bandage

Catheter

Joint Protector

Two fold
& Three fold
Strature

Microchips

Splints

Examination
Bag

ICU Cage

Our Business Associates

BUSTER

KRUUSE

For any enquiry contact at:

Happy Pet Solutions

S-101, Okhla Industrial Area, New Delhi-110020 **Ph.**: 011-41403221, +91-9667081783 E-mail: happypetsolutions@gmail.com

With best
compliments from:

FUJIFILM

Vekō

INNOVATION, CARE & ANIMAL HEALTH

CMI+

Protection & Defence
for your Pet

- For General Resistance
- To enhance cell meditated immunity
- Reduces Vaccination failures
- Adjuvant in the treatment for viral diseases
- Enhanced Immunity against parvo, distemper, kennel cough, hepatitis

APOCARE

- Increase response to cancer treatment
- Speed up recovery after surgical removal of mammary gland tumors or other tumors
- Promotes long term health & Improves quality of life

Vetfood PROFESSIONAL

Complementary feed
PREMIUM
NTS Immunactiv

health

Vet care+

INDICATIONS:

- Cancer
- Cancer Cachexia
- Chemotherapy
- Decreased Immunity
- Convalescence Period
- Body Weight Deficiency
- Muscle Mass Loss.

VET EXPERT

BASED ON EVIDENCE

🎉 We're Live! Welcome to **vetexpertorder.in**

– Your Trusted Pet Care Partner! 🐾

www.vetexpertorder.in

WEBSITE LAUNCH
FEBRUARY 26, 2025

We're thrilled to announce the launch of vetexpertorder.in, vet-go-to destination for premium pet healthcare products! 🐶🐱

🌟 WHAT WE OFFER:

✅ SPECIAL DISCOUNTS FOR VET

✅ HASSLE-FREE SHOPPING & FAST DELIVERY

EXCLUSIVE FOR VETS ONLY!

www.vetexpertorder.in

OUR INDUSTRY PARTNER

Prime Sponsor

drools
Feed Real. Feed Clean.

VET EXPERT
SAVAVET
Shaping the Future
TAGO
The Animal Care Organization

ROYAL CANIN
INCREDIBLE IN EVERY DETAIL
ENAVANT PETCARE
SKY EC
Committed to Quality

veteducation
zoetis
Globalvets

VENKY'S PET
The expression of loyalty
PURINA
Your Pet, Our Passion.
FUJIFILM

INDIAN IMMUNOLOGICALS LIMITED
SIGNATURE
GRAINZERO | FREEDOM
drools VET-PRO
Farmina Pet Foods
Happy pet. Happy You.

scientific remedies
Mankind's PetStar V Diet
OPUSPET
PASSION UNITES INNOVATION
OriHeal LIFESCIENCES

Vetrina Animal Nutrition & Health
MSD Animal Health
Happy Pet
VIVALDIS Animal Health

Veko
Virbac
Boehringer Ingelheim
WELLCON Animal Health

NVF VETERINARY FOUNDATION ®

Regd. Office : 5/39, Old Rajinder Nagar, New Delhi-110060
E-mail: nvfworld@gmail.com • Website: www.nvf.world