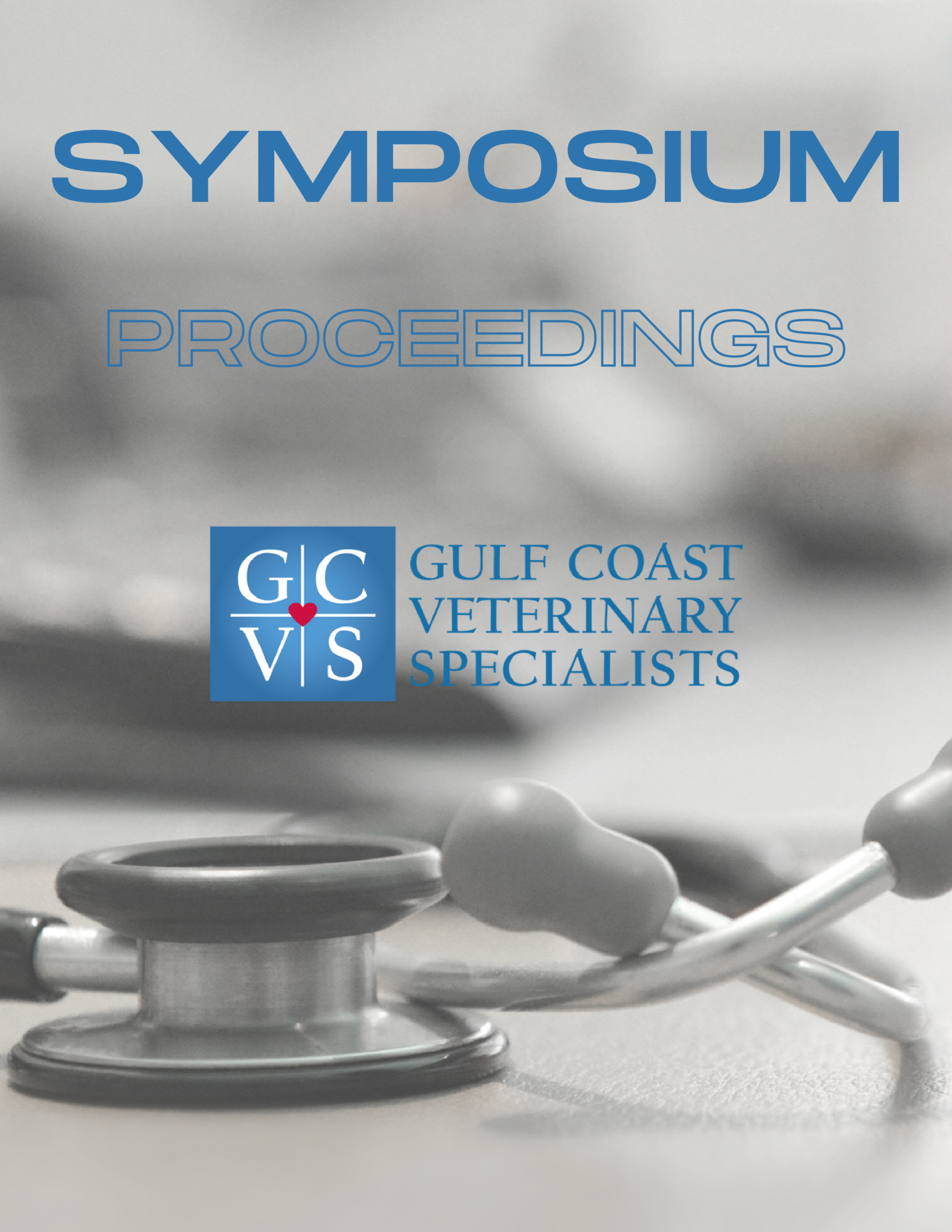
SYMPOSIUM

PROCEEDINGS

GULF COAST VETERINARY SPECIALISTS



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DVM SPEAKER SCHEDULE (8/19)

7:00 AM – 8:00 AM REGISTRATION & BREAKFAST | SPONSOR TABLES OPEN

8:00 AM - 8:10 AM
OPENING REMARKS
FREDRICK CANTOR | HOSPITAL DIRECTOR

8:10 AM - 9:05 AM
DISTAL LONG-BONE FRACTURES: R U READY TI-BI-AFIXED?
DR. PHILIP ALLEN| SURGERY

9:05 AM - 10:00 AM
BETWEEN TWO INTERNISTS: FREQUENTLY ASKED CONSULTS
DR. PAUL MANINO | INTERNAL MEDICINE

10:05 AM – 10:25 AM MORNING BREAK | SPONSOR TABLES OPEN

10:25 AM - 11:20 AM
BETWEEN TWO INTERNISTS: FREQUENTLY ASKED CONSULTS
DR. DANIEL JARDES| INTERNAL MEDICINE

11:20 AM - 12:15 PM
BACKYARD CHICKEN MEDICINE: WHAT THE CLUCK DO I NEED TO KNOW?
DR. SUSAN BALEY| AVIAN & EXOTICS

12:20 PM – 1:20 PM LUNCH | SPONSOR TABLES OPEN

1:20 PM - 2:15 PM
DISCOSPONDYLITIS: BRUCIE IS NOT YOUR FRIEND
DR. CARLEY GIOVANELLA | NEUROLOGY

2:15 PM - 3:10 PM
SEPSIS WON'T HESITATE. WILL YOU?
DR. HERNAN ANGULA-BRACHO| CRITICAL CARE

3:15 PM – 3:35 PM AFTERNOON BREAK | SPONSOR TABLES OPEN

3:35 PM - 4:30 PM
SURGICAL INTERVENTION OF BRACHYCEPHALIC [OBSTRUCTIVE] AIRWAY SYNDROME
DR. KRISTIN COLEMAN| SURGERY

4:30 PM - 5:15 PM
DIAGNOSING AND APPROACH TO TREATMENT FOR DIFFFERNT TYPES OF CORNEAL ULCERS
DR. ROBIN SANKEY | OPHTHALMOLOGY

5:15 PM – 5:30 PM CLOSING REMARKS

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DVM SPEAKER SCHEDULE (8/20)

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OPENING REMARKS
FREDRICK CANTOR | HOSPITAL DIRECTOR

8:10 AM - 9:05 AM
PRACTICAL TIPS FOR BLOOD TRANSFUSIONS
DR. MELISSA GARCIA-LACAZE | CRITICAL CARE

9:05 AM - 10:00 AM
FELINE PANCREATITIS
DR. MELANIE PUCHOT | INTERNAL MEDICINE

10:05 AM – 10:25 AM MORNING BREAK | SPONSOR TABLES OPEN

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CANINE AND FELINE PEMPHIGUS FOLIACEUS
DR. KARIN BEALE| DERMATOLOGY

11:20 AM - 12:15 PM
DEGENERATIVE VALVE DISEASE: PRACTICAL GUIDE MANAGEMENT
DR. ERIC OWENS | CARDIOLOGY

12:20 PM – 1:20 PM LUNCH | SPONSOR TABLES OPEN

1:20 PM - 2:15 PM
ORTHOPEDIC PROBLEMS IN GENERAL PRACTICE
DR. KYLE PFEIFER & DR. KATIE PANSINI | DIAGNOSTIC IMAGING

2:15 PM - 3:10 PM
CARING FOR THE MOST CRITICAL PATIENTS FIRST: THE ART OF TRIAGE
DR. RHONDA DIXON| EMERGENCY

3:15 PM – 3:35 PM AFTERNOON BREAK | SPONSOR TABLES OPEN

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OPIOIDS
DR. KATHERINE MCIVER | ANESTHESIA

4:30 PM - 5:15 PM
MEDIAL PATELLAR LUXATION
DR. NATHAN SQUIRE | SURGERY

5:15 PM – 5:30 PM CLOSING REMARKS

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SURGERY

PHILIP ALLEN

DVM, DACVS

Dr. Philip Allen obtained his Doctorate of Veterinary Medicine from Kansas State University. He then spent a year performing a rotating medicine/surgery internship at Tufts University. Following his internship, Dr. Allen traveled to Minneapolis to complete a three-year surgical residency at the University of Minnesota. Dr. Allen became board certified by the American College of Veterinary Surgeons in 2019. Prior to joining GCVS in 2019, Dr. Allen worked as a surgeon at a specialty hospital in Kansas City. Dr. Allen's surgical interests include minimally invasive surgery (laparoscopy, thoracoscopy, fracture repair), joint replacement therapy, and interventional radiology procedures.

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713-693-1111

Tibial Fractures & Surgical Approach to the Tibia

Philip Allen DVM, Diplomate ACVS-SA
Gulf Coast Veterinary Specialists
Houston, TX

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Incidence

- 10-20% fractures
- 50% occur in immature patients
- Majority are diaphyseal
- Concurrent fibular fracture
  - Rarely repaired

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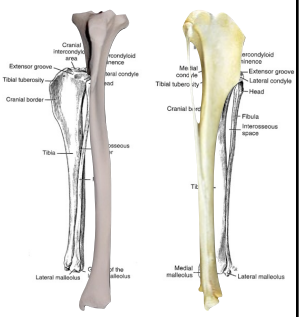
Tibial Anatomy

- Anatomy
  - Sigmoid shape on the frontal plane
  - Nominal soft tissue envelope medially
  - Proximally shaped like a triangle
  - Distally cylindrical
- Quadriceps insertion for stifle extension
- Fibula important for lateral collateral ligaments of stifle and tarsus



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Patient Assessment

- Complete physical exam
  - HBC common cause of tibial fx
  - Cardiovascular system
  - Urinary bladder
  - Other orthopedic injury
- 2 view radiographs affected bone
  - Craniocaudal & mediolateral

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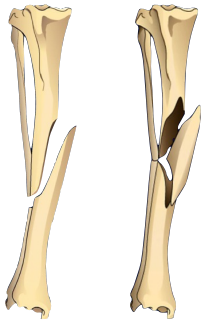
Fracture Pattern

- Simple Transverse
- Oblique
  - Short
  - Long
  - Spiral
- Butterfly
- Comminuted



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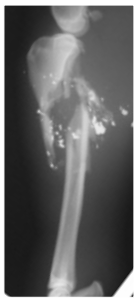
Open Fractures

- Common in the tibia
  - Limited surrounding soft tissue
- Contamination and vascular compromise
  - Increased infection risk
  - Delayed bone healing



Gun Shot Injury

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Methods of Fracture Stabilization

- Bone plate
- Bone plate with IM pin
- IM pin with cerclage wire
- External fixator
  - Linear
  - Circular
- Interlocking nail

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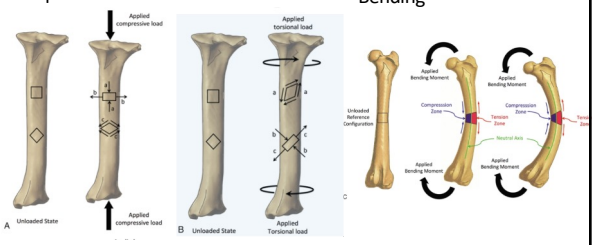
Forces Acting on a Fracture

Compression | Torsion | Bending



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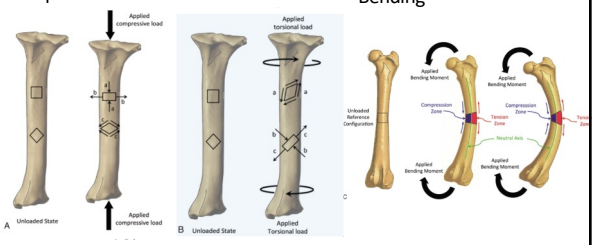
Forces Acting on a Fracture

Compression | Torsion | Bending



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Forces Acting on a Fracture

Compression | Torsion | Bending

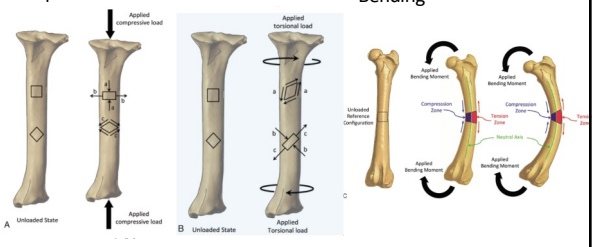

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Plate-Rod Construct

- Addition of an IM pin to a bone plate construct
- Decreases stress on plate
- Increases fatigue life of plate
- "Two-beam" system
- IM pin ~ 40% canal diameter
  - Interferes with screw insertion

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Reduction in Plate Strain by Addition of an Intramedullary Pin

Hulse D, Hyman W, Nori M, et al.
Veterinary Surgery, 1997

- Femoral gap model
  - Bridge plate
  - Bridge plate and IM pin
- Addition of rod decreased stress by 1/2 in plate-rod construct
- Implant fatigue life increased 10x

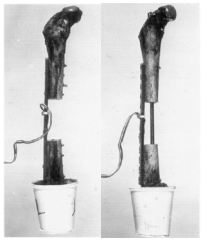

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Plate-Rod

- Commonly utilized for tibial fx
  - Pin resists bending
  - Plate resists axial collapse and rotation
- Increased construct stiffness
  - Increased AMI

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Decision Making

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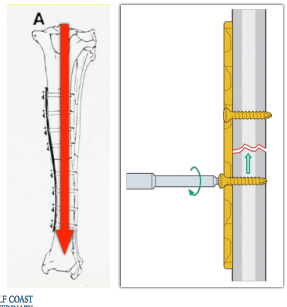
Compression Plating

- Application
  - Simple transverse
  - Short Oblique
- Bone segments compressed
- Reconstructed bone column resists the majority of forces acting on fracture



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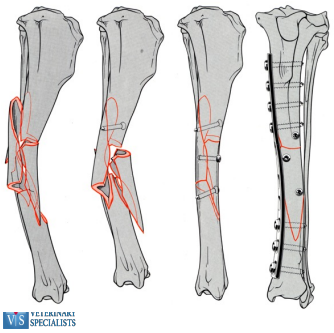
Neutralization Plating

- Application
  - Long oblique
  - Butterfly
- Compression provided by screws
  - Cerclage wire
- Bone splinting provided by plate.



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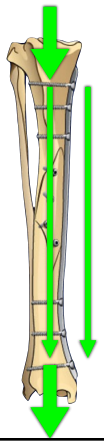
Neutralization Plating

- Load sharing
  - Bone plate
  - Reconstructed bone column



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Bridging Plate

- Application
  - Comminuted
- Major bone segments aligned and stabilized
- Gap at fracture site

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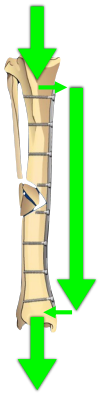
Bridging Plate

- Bone column not reconstructed
- Plate bears load across fracture site
  - No load sharing



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Pre-Op Planning

- Radiographs
- Implant selection
  - Size & system
- Plate pre-contouring

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Pre-Operative Radiographs

- Orthogonal views
- Affected & contralateral
- Positioning critical
  - Anatomic landmarks
- Magnification marker

Contralateral

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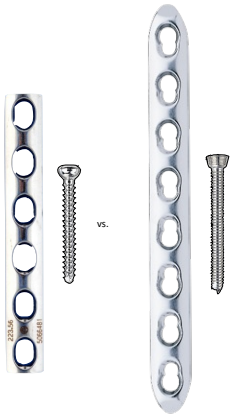
Implant Selection

- Locking vs. non-locking
  - Locking preferred for bridging
- Implant size
  - Screw diameter
  - Plate length
    - Span majority of bone
- IM pin
  - < 40% medullary diameter



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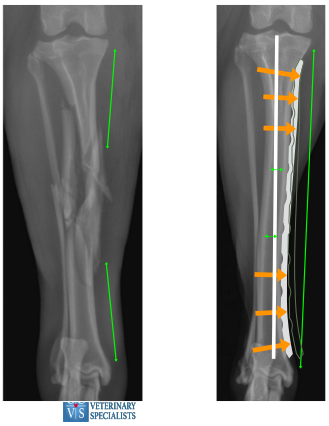
Pre-Operative Planning



VETERINARY SPECIALISTS

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Patient Positioning

- Dorsal recumbency
- Rear end at edge of table
- Table tilted down caudally
- Vacuum bag

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Patient Positioning

26

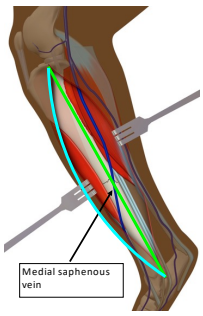
Patient Preparation

- Clip limb from hip to hock
- Aseptic scrub
- Duraprep
- Adhesive drape

27

Surgical Approach

- Medial approach for all fractures
- Landmarks
  - Medial tibial condyle
  - Medial malleolus
- Incision straight or curving cranially in middle
- Neurovascular bundle
  - Saphenous A,V,N



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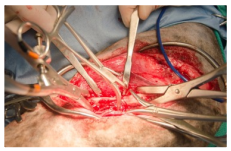
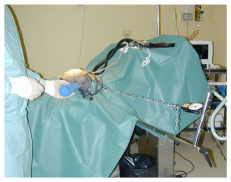
Reduction Techniques

- Direct, open reduction
  - Anatomic reconstruction
- Indirect reduction
  - Limb alignment
    - Joint above and below fracture
  - Fracture not anatomically reconstructed





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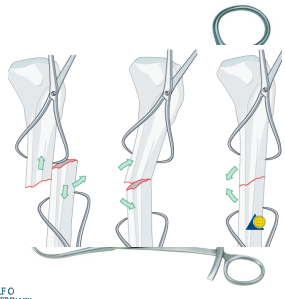
Direct Reduction Strategies

- Open surgical approach
- Bone holding forceps grip primary segments
- Distract and reduce
- In comminuted fracture work on 2 segments at a time



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Reduction Tools

Kern

Verbrugge

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Reduction Tools

AO Point to Point Reduction Forceps

Clamshell Reduction Forceps- Speedlock

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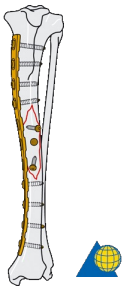
Direct Reduction

- Fracture has 2-4 fragments
- Repair 2 fragments at a time
- Interfragmentary compression
  - Lag screws, cerclage
- Primary fixation



Direct reduction

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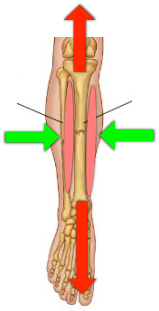
Indirect Reduction Strategies

- Principle: Traction restores length, soft tissue tension reduces fragments
  - Applicable to comminuted fractures
- Hanging limb technique
- IM pin reduction assistance
- Temporary external fixator +/- gear



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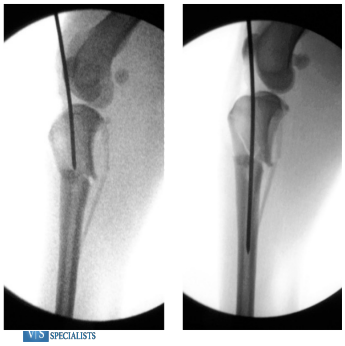
IM Pin Reduction

- Pin inserted normograde
- Guides fracture reduction
- Distract bone to proper length
  - Blunt distal pin tip
- Cut short or countersink for plate-rod



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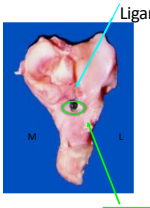
Tibia Technique

- Approach to tibial plateau
  - Medial or lateral
- Insert pin cranial to intermeniscal ligament
- Drive pin distally, medially, and slightly caudally
  - Reduction forceps on shaft
- Blunt pin tip to prevent cortical penetration



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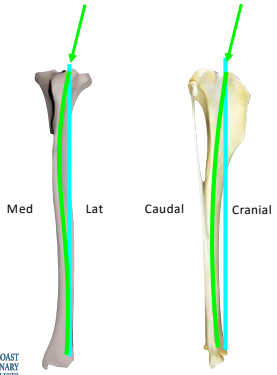
Tibial IM Pin Technique

- Insertion can be challenging
- Premature cortical penetration
  - Lateral
  - Cranial
- Tibial morphology
  - Recurvatum
  - Sigmoid shape



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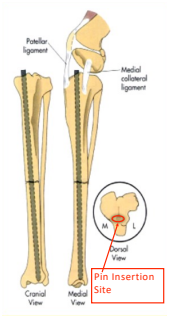
Tibia



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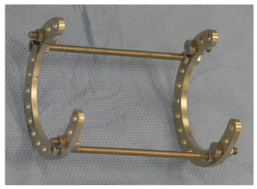
Temporary Ex-Fix

- Simple 2 ring circular fixator construct
- Single wire proximal and distal
- Wires parallel with joint surfaces
- Distract and align bone segments
- Remove after plate application



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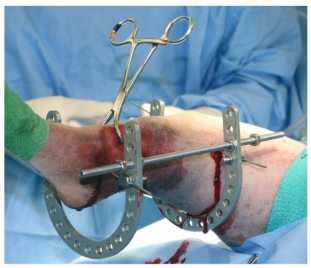
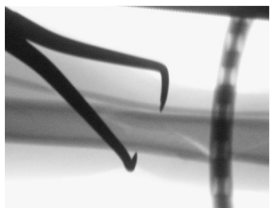
Temporary Ex-Fix





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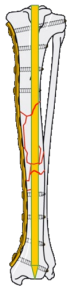
Stepwise Tibial Fx Repair

- Evaluate radiographs
  - Develop plan
- Surgical approach
- Reduce fracture
- Insert IM pin
- Apply bone plate
- Close



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Case Example: 10 y.o. M Beagle HBC Radiograph Evaluation

- Mid diaphyseal
- Comminuted
- Open
  - Subcutaneous emphysema
- Minimally displaced

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Surgical Plan

- Minimize disruption of fracture site
- Indirect reduction
  - Pre-contoured plate
- Bridge plate
  - Plate-rod
  - Possible delayed healing

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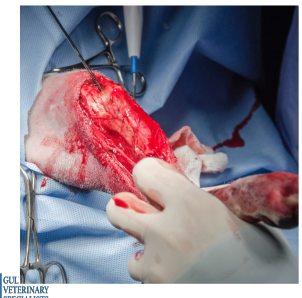
Surgical Approach & Reduction

- Full approach to medial shaft of tibia
- No disruption of fracture site
- Indirect reduction
  - Manual traction
  - Normograde IM pin insertion
  - Blunt pin tip



45

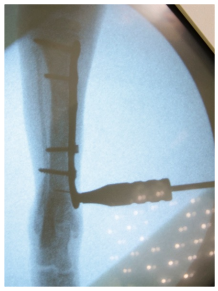
Bone Plate Application

- Locking plate selected
  - Pre-contoured
  - Contralateral limb radiographs
- Plate application improves fracture alignment

46

Temporary Stabilization With K-wires

47



Contoured plate
assists limb alignment

Drilling for
locking screws

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48

Post Op Radiographs

- Diaphysis straight
- Joint alignment good
- No screws in joint

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6 Wk Post

10 Wk Post

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50

Interlocking nails

- Initially used simple intramedullary nails
  - Current locked nails: union rates >90% in humans (Wiss, J Bone Joint Surg Am, 2001);
- Common characteristics
  - Solid intramedullary rods
  - Transverse holes (cannulations)
  - Locked in place with bone screws or partially threaded bolts

GULF COAST VETERINARY SPECIALISTS

51

Interlocking nails

- Initially used simple intramedullary nails
- Common characteristics
- Benefits:
  - Greater resistance to torsional forces than plates and standard IM pins (Kyle, Clin Orthop Rel Res 2001)
  - Superior fatigue resistance, bending stiffness compared with standard plate fixation (Muir, Vet Comp Orthop Traumatol 1995)
    - AMI of a solid 6-mm diameter ILN is almost 4 times that of a 3.5 mm dynamic compression plate (flat surface) (Bernarde, Vet Surg 2001)

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Concerns:

- Weaker in mediolateral bending d/t AMI
  - smaller in a bending plane parallel to the nail hole = stress concentration
- Screw failure - initially larger holes (3.5, 4.5), now 2.7, 3.5
  - Increased nail fatigue life 8x, decreased screw AMI by 40%
  - Switched to partially threaded bolts

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Interlocking nails

- Initially used simple intramedullary nails
- Common characteristics
- Currently available products (US):
  - Innovative Animal Products - Original Interlocking Nail System
  - BioMedtrix - I-Loc Angle Stable ILN

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Innovative ILN

- Modified Steinmann IM pin with single trocar point
  - 4-, 4.7-, 6-, 8-mm
- 3- or 4- hole design
- Initially used screws, now use partially/cis threaded bolts
  - 2-, 2.7-, 3.5-mm
  - fixed length, cut to size

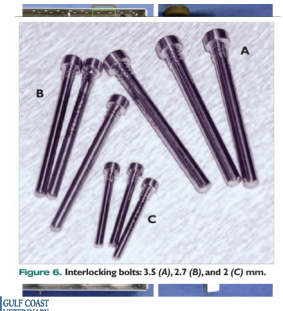

Figure 6. Interlocking bolts: 3.5 (A), 2.7 (B), and 2 (C) mm.

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Innovative ILN

- 83% initial success (105 dogs)
  - 10% nail breakage d/t fatigue failure

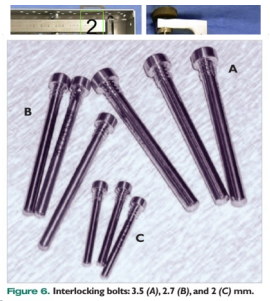

Figure 6. Interlocking bolts: 3.5 (A), 2.7 (B), and 2 (C) mm.

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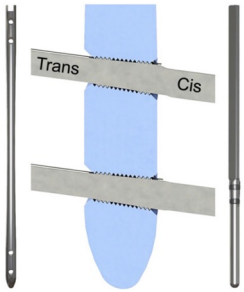
Biomedtrix I-Loc

- Hourglass profile IM pin
  - minimize damage to endocortices?
- Bullet tipped
  - minimize penetration into joint space
- Locking nail cannulations
  - self-centering, self-locking
  - threaded Morse taper
- Locking bolt
  - threaded conical central section
  - matches cannulation



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Implantation technique

- Use temporary smooth locking posts
  - creates rigid frame between alignment guide and nail
- Trial nail for accurate length estimate
- Threaded cannulation can confirm appropriate drill placement

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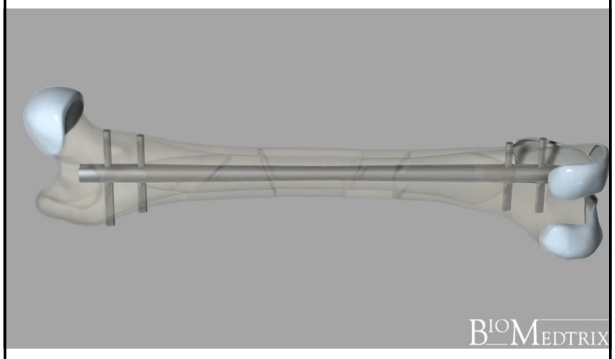

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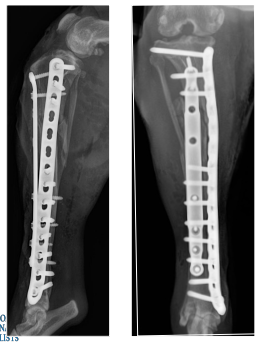
Toy Breed Considerations

- Relatively small medullary canal
- May be difficult to utilize IM pin/ plate-rod
- Small bone plates relatively weak
  - Stacked plates- not preferred
  - Double plating
- Consider bone graft



82

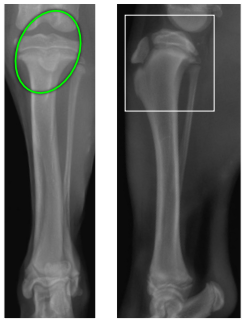
Proximal & Distal Tibial Fractures

- Philip Allen DVM, Diplomate ACVS
- Gulf Coast Veterinary Specialists
- Houston, TX

83

Immature Tibia

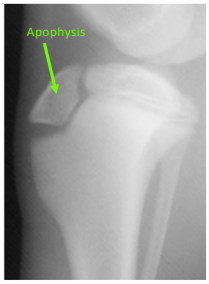
- Physes
  - Proximal
    - 40% growth in length
  - Distal
    - 60% growth in length
- Apophysis
  - Tibial tuberosity
  - Medial malleolus



85

Apophysis

- Normal bone outgrowth arising from a separate ossification center
  - Fuses with primary bone over time
- Apophysis is site of tendon or ligament attachment
  - Also called traction epiphysis
  - Epiphysis is a component of a joint



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Fracture Categories

- Immature patients
  - Physeal fractures
    - Salter Harris types
- Skeletally mature patients
  - Proximal & distal metaphyseal fractures

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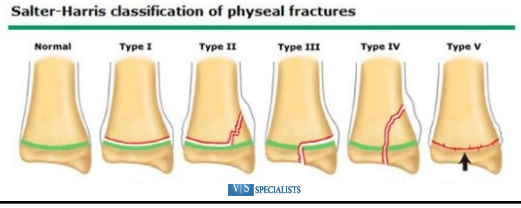
Physeal Fractures

- Salter Harris Classification
  - Five Types



88

Proximal Physis Fractures

- Salter Harris I
  - Slipped physis
  - Epiphysis & apophysis remain together
    - Displace proximally & caudally
- Anatomic reduction
  - Preferred if possible
  - Open or minimally invasive

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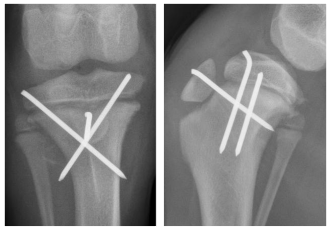
Proximal Physis Type I

- Stabilization options
  - Acute
    - Cross pins
    - Parallel pins through tuberosity
- Chronic
  - Cross pins
  - Lag screw(s)



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Reduction Techniques

- Open, direct reduction
  - Remove hematoma from fracture site
- Minimally invasive reduction
  - Acute time period
    - Often possible to obtain anatomic reduction

C-Arm Guided Reduction

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Proximal Physeal Fx Reduction

- Point to point forceps on tuberosity
- Hyperextend stifle joint

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Proximal Physis Stabilization

- Drive K-wire through apophysis
  - Center wire on segment
  - Direct caudal & distal
- Needle used to identify wire insertion site

Needle used to identify wire insertion site

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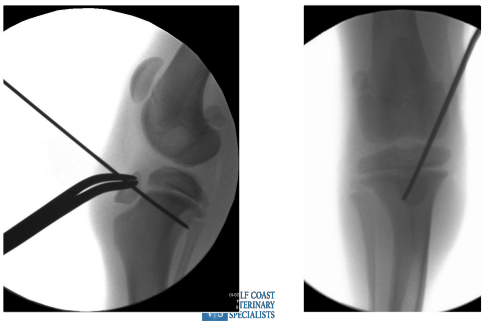
Proximal Segment Stabilization



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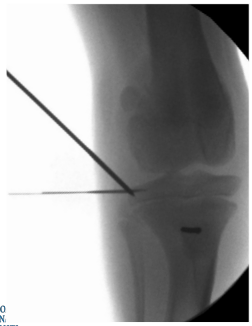
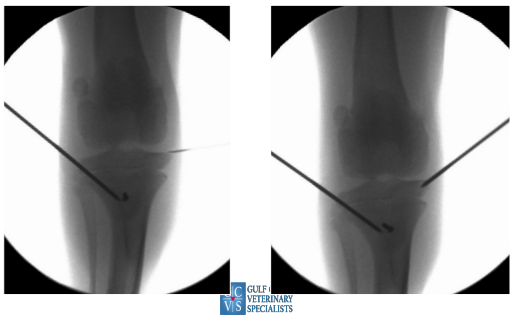
Insert Additional Wires

- Medial & lateral tibial plateau



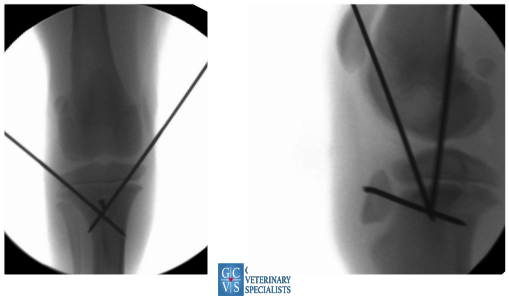
99

Additional Wire Insertion



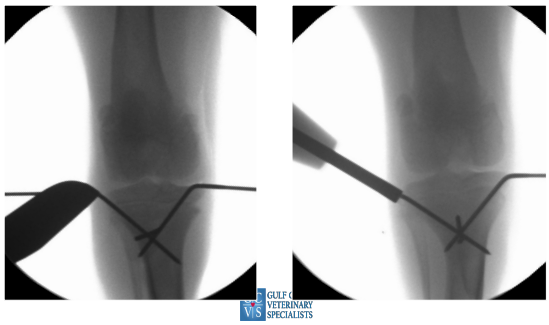
100

3 Wires Inserted



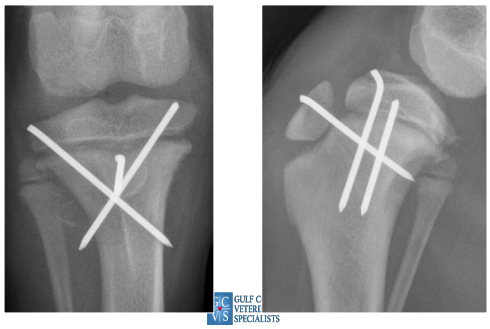
101

Cut Wires & Countersink



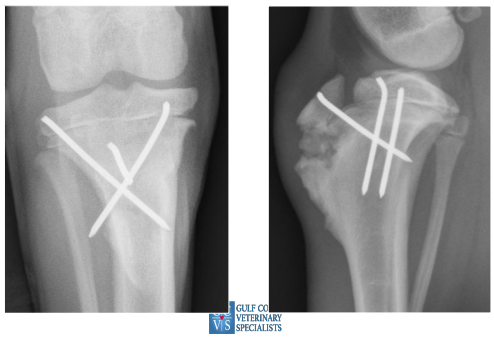
102

Post Op



103

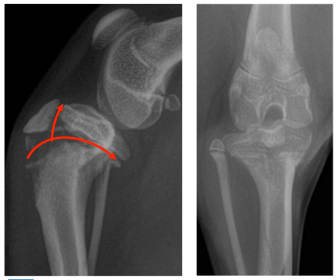
4 Wks



104

Proximal Physis Fracture

- Salter Harris type I
- Separation of apophysis from epiphysis



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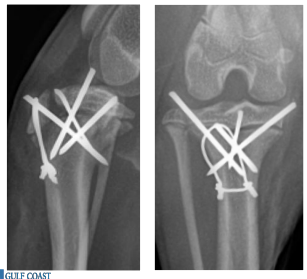
Post Op

- Divergent K-wires
  - K-wire through cranial epiphysis
- Tension band on tuberosity
  - Chronic injury
  - Patient almost skeletally mature



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4 Wk Post

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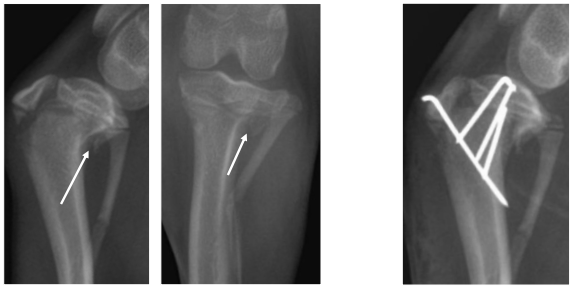
Explanted 6 Wk Post

107

Salter Harris II



Pre Op

Post Op

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Tibial Tuberosity Avulsion

- Apophysis avulsion
- Salter Harris I
- Quadriceps pull displaces tuberosity proximally
- Anatomically reduce

Normal

Avulsion

Avulsion

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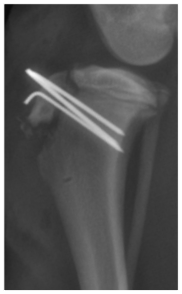
Tibial Tuberosity Avulsion

- Stabilization Options
  - K-wires only
    - At least two pins (Welsh et al Vet Surg)
    - Leave in situ immature dogs (Miller et al JAVMA)
  - K-wires & tension band
    - Wire vs. suture
  - Lag screw stabilization
    - Older patients



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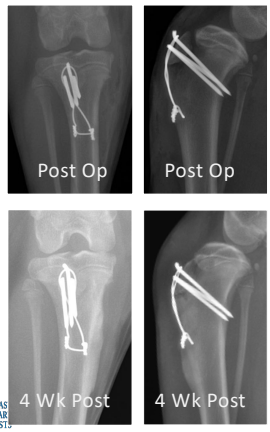
Tibial Tuberosity Avulsion

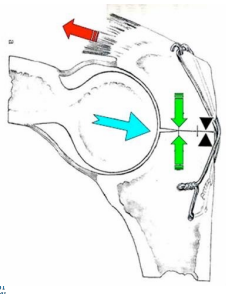
- Pins placed perpendicular to fracture line
- Tension band wire
  - Converts distraction forces to compression
- Closure of tuberosity growth plate



113

Biomechanics

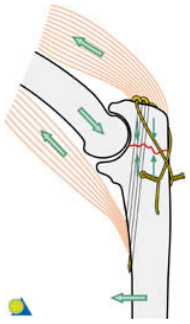
- Tension band applies opposing force to muscle pull
- Opposing forces create compression



114

Biomechanics

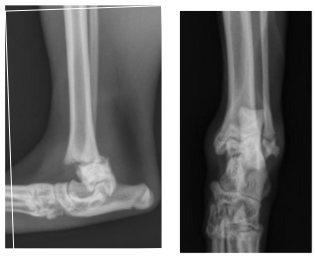
- K-wires
  - Prevent rotation
  - Compression force directed along K-wires
  - Insert perpendicular to fracture line
- Orthopedic wire
  - Counters tension loads



115

Distal Physeal Fractures

- Salter Harris I, II, III, IV
- Repair options
  - Cross pins
    - Type I
  - Lag screws +/- plate
    - Type II, III, or IV



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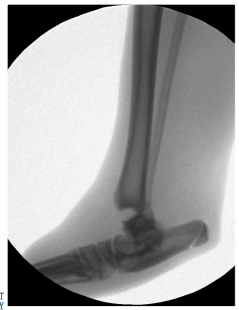
Distal Physeal Reduction

- Open, direct
- Minimally invasive



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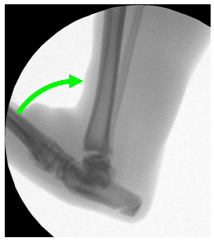
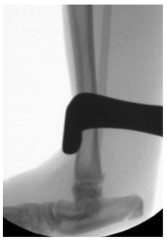
Reduction Techniques



Hyperflexion of tarsus helps achieve reduction



Army navy helps maintain reduction

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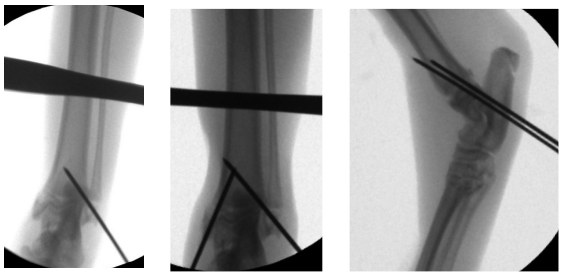
K-Wire Insertion



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Post Op 4 Wks

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Distal Physeal Fracture

- Metaphysis, physis and epiphysis affected
  - Salter Harris type IV
- Anatomic reconstruction
- Rigid stabilization
- Plate and lag screws

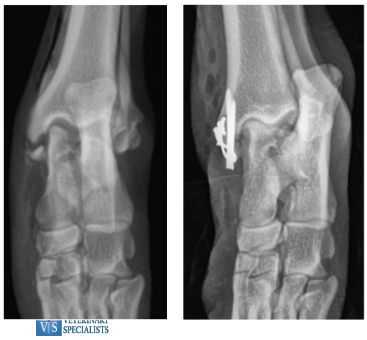
121

Healed at
4 Wks

122

Malleolar Fracture

- Avulsion fracture
  - Collateral attachment
    - Tibia or Fibula
- Pin & tension band stabilization
- Supplement with external coaptation



123

Metaphyseal Fractures

- Usually occur in skeletally mature patients
- Similar principles to shaft fractures
  - Stabilization can be challenging
- Limited bone available for implants
- Strategies
  - "T" plates
  - CBLO plates
  - Double plating

124

Specialized Function Plates

May be utilized for proximal or distal tibial fractures

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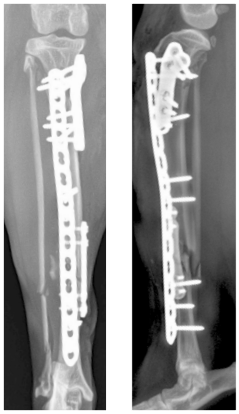
Post Op

- Double plating
  - At each fracture site
  - Increase number of screws per segment
- TPLO plate proximally



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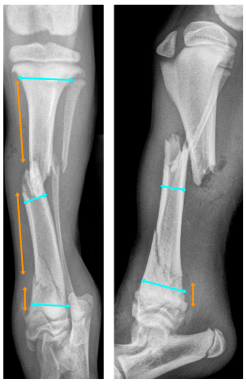
Pre Op Planning

- Midshaft diaphyseal fracture
- Distal metaphyseal minimally displaced fracture
- Implant type & location
- Determine location of physis



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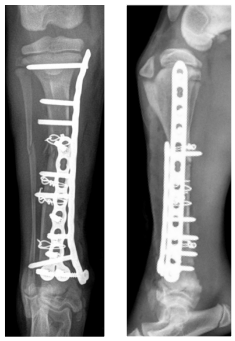
Post Op

- Double plating
  - Cranial notched head T plate
  - Medial LCP
    - Distal cortical screw
      - Angled to avoid growth plate
- Cerclage to control fissure lines



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4 Wk Post

- Fractures healed by 4 wks
- Physis still open proximally
- Distal physis?

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Thank you

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INTERNAL MEDICINE

PAUL MANINO
DVM, DACVIM

Dr. Paul Manino, DVM, completed his veterinary degree at Louisiana State University in 2003. Following this, he undertook a one-year small animal medicine and surgery internship at Carolina Veterinary Specialists, NC. With five years of experience in emergency/critical care, he pursued a three-year small animal internal medicine residency, becoming board certified by the American College of Veterinary Internal Medicine in 2012.

Dr. Manino's expertise lies in liver disease, fungal disorders, and advanced laparoscopy and endoscopic-guided laser procedures. He currently heads the internal medicine department at GCVS. Beyond his professional commitments, Dr. Manino enjoys family time, cooking, and philanthropic endeavors as a 501st legion member.

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DANIEL JARDES
DVM, DACVIM

Dr. Daniel Jardes received his bachelor's degree in organismal biology from the University of Kansas in 2007 and veterinary degree from Kansas State University in 2011. He completed a one-year rotating internship followed by a three-year residency in small animal internal medicine at Tufts University in Massachusetts. During his residency, his research was focused on acute kidney injury and chronic kidney disease. Dr. Jardes became board certified by the American College of Veterinary Internal Medicine in 2015. Dr. Jardes has a special interest in renal disease. In his spare time, he enjoys woodworking and assembling jigsaw puzzles with his family.

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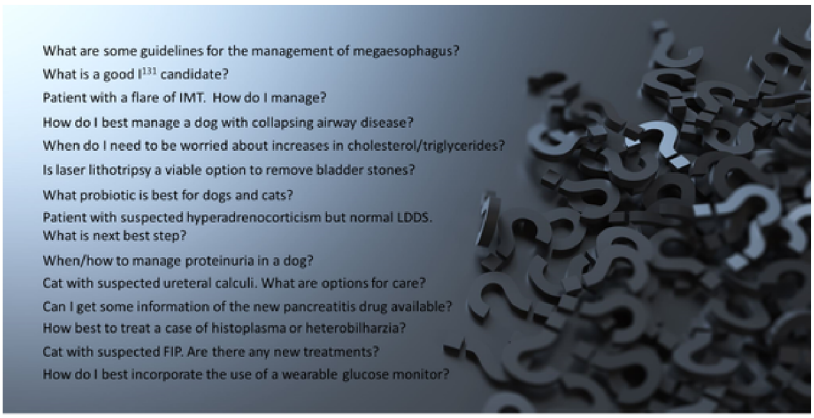
Paul M. Manino DVM DACVIM GULF COAST VETERINARY SPECIALISTS Daniel Jardes DVM DACVIM



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Questions on Proteinuria

- How and when do I screen for proteinuria?
- Does the degree of proteinuria help me determine the etiology?
- What are the most common causes of proteinuria?

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How and when do I screen for proteinuria?

- Typically clinically silent
  - Decreased appetite?
- An unexpected finding
  - Routine labs
  - During evaluation for an unrelated disease

Recommendations:

- All patients should have a urinalysis during their routine health screen every 6-12 months
- All patient should have a urinalysis performed at a time of illness
- Start with dipstick
- Urine protein to creatinine ratio for quantification (UPC)

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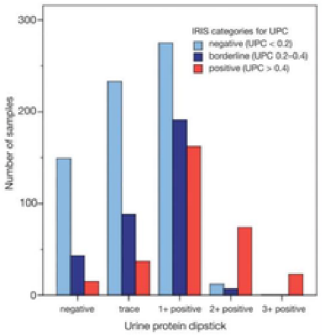
Dipstick evaluation vs. UPC



- Dipstick = semi-quantitative
- Not always significant
- Altered by *active* sediment

Recommendations:

- Run UPC if:
  - 2+-3+ proteinuria
  - Persistent 1+ proteinuria > 4wks
  - Concurrent disease with high risk for proteinuria

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Determining the cause of proteinuria

Three key factors:

- Persistence
  - Proteinuria on repeated testing
- Magnitude
  - The degree of proteinuria
- Localization
  - Where in the kidney?

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Determining the cause of proteinuria

Persistence

- Proteinuria on at least 2 occasions > 2 weeks apart
- Transient proteinuria
  - Fever
  - Steroids
  - Vasculitis

Magnitude (UPC)

- 0-0.2 normal
- 0.3-0.5 borderline
- >0.5 abnormal
- >2.0 more commonly seen with glomerular disease

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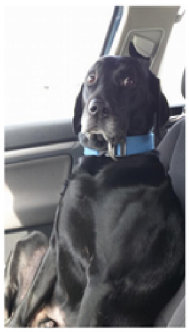
Determining the cause of proteinuria

Pitfalls of UPC measurement:

- Up to 40% day to day variation
- Altered by stress level
- Sensitive to sample handling

Recommendations

- Pooled sample collected over 3 days
- Collected at home in a glass container or sterile urine container
- Refrigerated until dropped off



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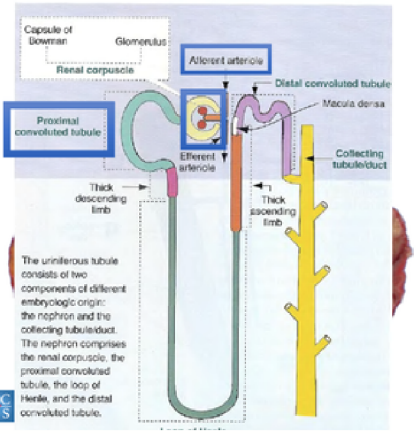
Determining the cause

Localization

- Pre-glomerular (systemic)
- Glomerular
- Post-glomerular (tubular)
  - Look for concurrent glucosuria



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Determining the cause of proteinuria

Pre-glomerular (afferent arterioles)
- Hypertension
- Marked hyperglobulinemia
- Fever
- Vasculitis

Glomerular
- Immune-complex glomerulonephritis
- Amyloidosis
- Podocytopathies
  - Minimal change disease
  - Glomerulosclerosis
  - Steroid administration

Post-glomerular (tubular)
- Leptospirosis
- Toxicity
  - Including jerky treats
- Fanconi's syndrome
- Chronic kidney disease
- Acute kidney injury

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Determining the cause of proteinuria

Diagnostic work up is ordered least to most invasive

Minimum database for all patients with elevated UPC
- Toxicity history (grapes, raisins, NSAIDs, jerky treats)
- Blood pressure
- CBC, Chemistry, Urinalysis
- Heartworm testing (causes immune-complex glomerulonephritis)
- Leptospirosis testing
- Protein electrophoresis if hyperglobulinemic

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Determining the cause of proteinuria

If all normal/negative you have glomerular localization

Additional diagnostics:
- Bicavitary imaging (chest radiographs/abdominal ultrasound)
- Vector-borne infectious disease testing (PCR and Serology; NCSU)
- Urine histoplasmosis testing
- Kidney biopsy

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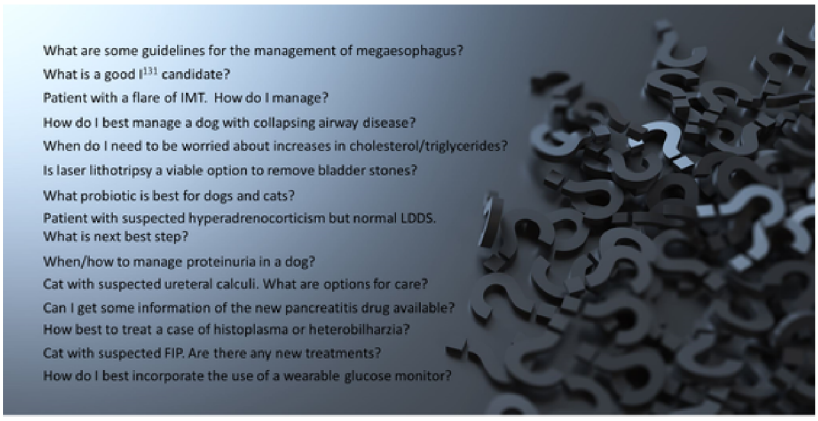

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Questions on Proteinuria Treatment

- What patients do I treat?
- Are there any recent changes in treatment?
- What are the best management strategies for patients with concurrent hypertension?
- Are there treatment options for presumed immune-complex glomerulonephritis for a patient who is unable to get a biopsy?

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What patients do I treat?

Persistence

- **Proteinuria on at least 2 occasions > 2 weeks apart**

Magnitude (UPC)

- 0-0.2 normal
- 0.3-0.5 borderline
- **>0.5 abnormal**
- **>2.0 more commonly seen with glomerular disease**

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Are there any recent changes in treatment?

Specific etiology treatment

Four mainstays of treatment:

- Dietary protein restriction
- Fatty acid supplementation
- Antithrombotic therapy
- *Inhibition of the Renin-Angiotensin-Aldosterone System (RAAS)*

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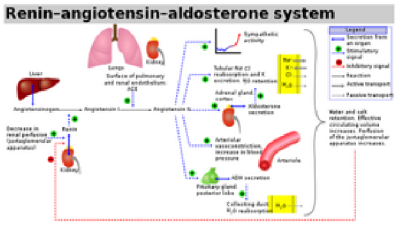
Are there any recent changes in treatment?

Inhibition of RAAS

- Used to reduce protein loss
- Renoprotective effects
- Anti-hypertensive effects

Two categories of medications

- Angiotensin-converting enzyme inhibitors (ACE-I)
- Angiotensin-receptor blockers (ARB)



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Are there any recent changes in treatment?

IRIS Canine GN Study Group 2013:

Drug	Indication	Initial Dose	Escalating Dose
Benazapril	Angiotensin converting enzyme inhibitor[a]	0.5 mg/kg PO q24h	Increase by 0.5 mg/kg/d to a maximum of 2 mg/kg PO per day. Can give q12h
Enalapril	Angiotensin converting enzyme inhibitor[a]	0.5 mg/kg PO q24h	Increase by 0.5 mg/kg/d to a maximum of 2 mg/kg PO per day. Can give q12h
Ramipril	Angiotensin converting enzyme inhibitor[a]	0.125 mg/kg PO q24h	Increase by 0.125 mg/kg/d to a maximum of 0.5 mg/kg PO per day. Usually give q24h
Imidapril	Angiotensin converting enzyme inhibitor[a]	0.25 mg/kg PO q24h	Increase by 0.25 mg/kg/d o a maximum of 5 mg/kg PO per day. Usually give q24h
Telmisartan	Angiotensin receptor blocker	1.0 mg/kg PO q24h	Increase by 0.5 mg/kg once daily up to 2 mg/kg/d
Losartan	Angiotensin receptor blocker[b]	0.125 mg/kg/d in azotemic dogs 0.5 mg/kg/d in nonazotemic dogs	0.25 mg/kg/d in azotemic dogs 1 mg/kg/d in nonazotemic dogs

Are there any recent changes in treatment?

Inhibition of RAAS- Side effects
- Decreased appetite
- Increase in creatinine
- Hyperkalemia

Management strategies
- Drug holiday
- ECG monitoring (K>6.0)
- Dose/medication reduction

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Are there any recent changes in treatment?

Dietary protein restriction
- Reduce the amount protein loss through the glomerulus and slow the progression of CKD
- Sodium restriction
- Works synergistically with RAAS inhibition to lower blood pressure

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Are there any recent changes in treatment?

Fatty acid supplementation
- Lower the degree of proteinuria
- Delay the progression of chronic kidney disease

Sources
- Commercial kidney diets
- Additional supplementation
- Tufts Heart Smart-> Diet -> Nutrients -> Omega-3

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Are there any recent changes in treatment?

Product Name	Company	Price/100 mg of EPA+DHA	Daily dose
Cats and Small or Medium-Sized Dogs			
CVS Fish Oil Concentrate Capsules 1000 mg (180 mg EPA and 120 mg DHA per softgel)	CVS Pharmacy	2¢	1 capsule per 10 pounds body weight
Large Dogs			
GNC Triple Strength 1500 (540 mg EPA and 360 mg DHA)	GNC	2¢	1 capsule per 30 pounds body weight
Nordic Naturals Ultimate Omega (325 mg EPA and 225 mg DHA)	Nordic Naturals	6¢	1 capsule per 18 pounds body weight
Simply Right Triple Strength Fish Oil (647 mg EPA, 253 mg DHA). Note: lower in DHA than other brands	Sam's Club	1¢	1 capsule per 35 pounds body weight
Solgar Omega-3 (360 mg EPA and 240 mg DHA per softgel)	Solgar	4¢	1 capsule per 20 pounds body weight
Twinlab Omega-3 (270 mg EPA and 180 mg DHA per softgel)	Twinlab	2¢	1 capsule per 15 pounds body weight
Liquids			
Vitamin Shoppe Omega-3 Fish Oil 800 EPA/500 DHA – liquid (800 mg EPA and 500 mg DHA per teaspoon). Note: lemon-flavored	The Vitamin Shoppe	2¢	1 teaspoon per 44 pounds body weight
Welactin Canine Liquid. Note: not independently tested	Nutramax	Variable	1 scoop per 48 pounds body weight
Welactin Feline Liquid. Note: not independently tested	Nutramax	Variable	1 scoop per 9 pounds body weight

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Are there any recent changes in treatment?

Antithrombotic therapy

- Thromboembolism in as high as 25% with GN
- Due to loss of antithrombin III
- Little evidence for recommendation

Widely used options

- Aspirin 1-5 mg/kg daily
- Clopidogrel 2-3 mg/kg daily

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Management of concurrent hypertension?

- Measurement of systolic blood pressure
- Risk of target organ damage
  - < 150 mmHg = no or minimal risk
  - 150-159 mmHg = low risk
  - 160-179 mmHg = moderate risk
  - >180 mmHg = high risk
- Multiple measurements needed to determine the risk

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Management of concurrent hypertension?

Goals for moderate to high risk patients:

- Blood pressure < 150 mmHg
- Gradual reduction over weeks (except with crisis)
- Individualized treatment plan based on risk

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Are there any recent changes in treatment?

Moderate risk

- Telmisartan at 1 mg/kg SID
- Low sodium diet

High risk

- If eating well Telmisartan at 1 mg/kg SID and
- Calcium channel blockage with Amlodipine at 0.2 mg/kg PO SID

Refractory cases: ACE-I or spironolactone

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Treatment for immune-complex glomerulonephritis without biopsy?

Very common predicament:

- Refractory proteinuria with glomerular localization
- Patient either isn't a candidate for biopsy or owner not consenting
- Most likely diagnosis is immune-complex glomerulonephritis
- Infectious disease testing has all returned negative
  - Leptospirosis
  - Histoplasmosis
  - Heartworm
  - Vector-borne disease (PCR and serology to infectious disease lab)

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Treatment for immune-complex glomerulonephritis without biopsy?

What options do we have?

- Mycophenolate at 10mg/kg BID
- Requires close monitoring for improvement of UPC
- Discontinue treatment if no benefit
- Should not be considered for patients with pre/post glomerular localization

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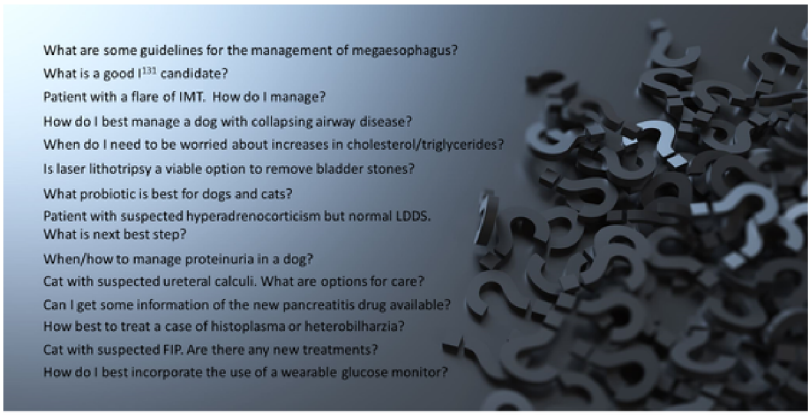

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I have a patient with a marked increase in serum triglycerides. How concerned do I need to be and what would be some treatment recommendations?

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Canine Hyperlipidemia

- Disturbance of lipid metabolism causing increase concentration of lipoprotein levels
  - Increased synthesis of lipids
  - Decreased degradation of lipids
- Hypertriglyceridemia/hypercholesterolemia
- Traditionally benign

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Classification of Lipoproteins

- Chylomicrons
  - Largest and least dense
  - Derived from dietary fat and contain the highest [TG]
  - Associated with most common lipid disorders in animals
- VLDL
  - Mediates transfer of endogenous TG
- LDL
  - Mediates transfer of endogenous cholesterol
- HDL
  - Facilitates removal of excess cholesterol
  - Contains highest amount of cholesterol in animals

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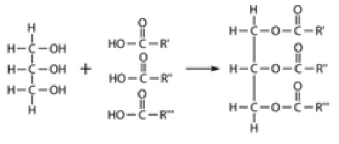
Triglycerides

- Ester derived from glycerol and three fatty acids
- Most abundant dietary lipid
- Uses
  - Source of stored energy in adipocytes
  - Cellular metabolism



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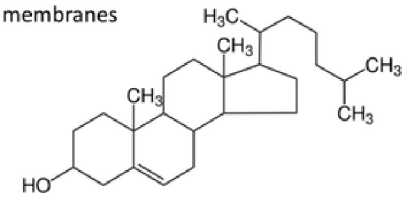
Cholesterol

- Principal sterol of all higher animals
- Primarily produced by hepatocytes
- Essential component of cell membranes
- Precursor of:
  - Steroid hormones
  - Bile acids
  - Cholecalciferol



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Causes of Hyperlipidemia

- Post-prandial
  - Most common
  - Rarely associated with increased cholestserol
  - Usually mild and lasts < 6-12 hrs
- Primary/Idiopathic
- Secondary/acquired

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Primary/Familial Hyperlipidemia

- Miniature Schnauzers
  - Seen in >30% of all MS in US
  - Characterized by HTG>>>HCH
  - Prevalence increases with age
  - Believed to be due to lipoprotein lipase deficiency
- Other breeds
  - HTG: Beagles*
  - HCH: Shelties*, Collies, Dobermans, Rottweilers, Briards

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Secondary/Acquired Hyperlipidemia

Common

- Hyperadrenocorticism (80%)
- Hypothyroidism (75%)
- Diabetes mellitus (50%)
- Pancreatitis (30%)
- Obesity (25%)
- Cholestasis
- Hepatic insufficiency
- Drugs
- Protein losing nephropathy*

Uncommon

- Lymphoma
- Parvoviral enteritis
- Leishmaniasis

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Clinical Significance of Hyperlipidemia

- Influence on lab parameters
  - Can falsely increase bilirubin, TP, and phosphorus
  - Can falsely decrease creatinine, tCO_2, and chloride
  - Can interfere with ALP, albumin, and BG at very high levels
  - Can cause hemolysis
- Increased risk of disease
  - Pancreatitis
  - Hepatobiliary disease (VH, lipidosis, biliary mucocele)
  - Diabetes mellitus
  - Ocular disorders (blindness, lipid keratopathy, uveitis)
  - Dermatologic abnormalities (xanthomas)
  - CNS disturbances (seizures, mentation changes, peripheral neuropathies)

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Clinical Signs of Hyperlipidemia

- Usually associated with the cause or effect of hyperlipidemia
- May include GI signs (V/D, hyporexia, abdominal discomfort)
- TG > 500mg/dL

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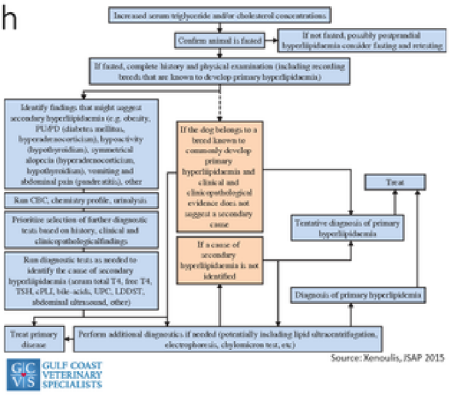
Diagnostic Approach to Hyperlipidemia

1. Ensure proper fasting
2. Assess for drugs/risk
3. Look for underlying disease



Source: Xenoulis, JSAP 2015

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Treatment Guidelines for Hyperlipidemia

- Indications for treatment
  - Hypertriglyceridemia >300mg/dL*
  - Hypercholsterolemia >500mg/dL
  - Presence of clinical signs or comorbidities
- Identify and treat underlying cause
- Principal treatments
  - Dietary modification/supplementation
  - Medical management

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Dietary Management of Hyperlipidemia

- First line treatment
- Low fat diets with <25g total fat/1000kcal recommended
- Commercial vs. home-made diets
  - RC GI LF preferred by author
  - Home-made diets with turkey and rice
- Recheck lipid levels after 4-6 weeks then q6months if adequate response

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Dietary Management of Hyperlipidemia

Received: 10 January 2020 | Accepted: 15 August 2020
DOI: 10.1111/jvim.15880

STANDARD ARTICLE

Journal of Veterinary Internal Medicine ACVIM

Effect of a low-fat diet on serum triglyceride and cholesterol concentrations and lipoprotein profiles in Miniature Schnauzers with hypertriglyceridemia

Panagiotis G. Xenoulis[1] | Paul J. Cammarata[2] | Rosemary L. Walzem[3] | Jan S. Suchodolski[1] | Jörg M. Steiner[1]

Conclusions

- Dogs (n=16) treated with RC GI LF for 8 & 12 weeks
- Serum cholesterol significantly reduced in all dogs
- Serum TG normalized in~25% of dogs
- 15/16 dogs with TG >500 had levels fall <500

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Dietary Supplements for Hyperlipidemia

- Chitosan
  - Fiber supplement derived from shellfish
  - Binds dietary TG decreasing absorption
  - No studies proving efficacy
  - No adverse effects
  - Recommended dose: 250-500mg q12hr 30min before meals
  - Not recommended to use Epakitin

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Dietary Supplements for Hyperlipidemia

- Omega-3 Fatty Acids (DHA/EPA)
  - Reduces lipogenesis of VLDL and LDL and increases lipoprotein lipase activity
  - Masaki-Tovi et. al 2014 showed that 20 healthy dogs given 220mg/kg daily for 30d had significant reductions in [TG]
  - Albuquerque et. al 2021 showed that MS given 730mg/d had a 36% and 20% reduction in [TG] and [CH] without a low fat diet
  - Little to no adverse effects
  - Recommended dose: 200-300mg/kg SID
  - Take care to use high quality products

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Medical Management of Hyperlipidemia

- Fibric acid derivatives
  - Increases fatty acid oxidation and lipoprotein lipase activity
  - Recommended when diet and omega-3 treatment fails
  - More effective at lowering triglyceride than cholesterol
  - Adverse effects: GI upset, hepatotoxicity, choleliths, myalgia/myopathy
  - Drugs: Fenofibrate*, Bezafibrate, Gemfibrozil
  - Recommended to recheck liver values and lipid levels after 30d

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Fenofibrate for Hyperlipidemia

- Recommended starting dose: 6.5-10mg/kg
- Generic vs. nanocrystal formulation (Tricor®)
- Very effective and well tolerated


Domestic Animal Endocrinology
Volume 74, January 2021, 106578

Fenofibrate treatment for severe hypertriglyceridemia in dogs

D.D. Micelli, V.P. Vidal, M.F.C. Blatter, O.P. Pignataro, V.A. Castillo


Received: 22 December 2020 | Accepted: 18 May 2021
DOI: 10.1111/jvim.16190
STANDARD ARTICLE
Journal of Veterinary Internal Medicine ACVIM

Efficacy of a micronized, nanocrystal fenofibrate formulation in treatment of hyperlipidemia in dogs

Matthew J. L. Munro | Sean E. Hulsebosch | Stanley L. Marks | Chen Gilor

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Medical Management of Hyperlipidemia

- Niacin
  - Acts primarily to reduce hepatic triglyceride synthesis
  - Studies lacking
  - Side effects: erythema, pruritus, restlessness, myotoxicity, hepatotoxicity
  - Use with caution in diabetic patients
  - Recommended dose: 50mg/d titrated up to 200-300mg/d

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Medical Management of Hyperlipidemia

- HMG CoA-reductase inhibitors or "Statins"
  - Most powerful cholesterol reducing medications available
  - Have little effect on lowering triglyceride levels
  - Side effects: lethargy, diarrhea, myopathy, hepatoxicity
  - Should not be used concurrently with fibrates
  - Recommended dose: Atorvastatin – 5mg/kd/d; Lovastatin 10-20mg/kg/d

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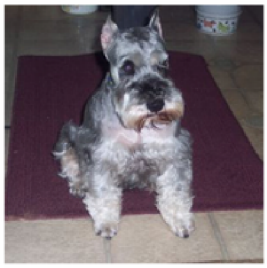
Canine Hyperlipidemia
"Vader"

- 8yr MN Miniature Schnauzer
- PPH: Atopic dermatitis
- Current hx
  - 5-7 days of lethargy and reduced appetite
  - RV labwork: ALT 158, ALP 1087, cholesterol 614
  - Referred for AUS and concern for pancreatitis and/or hyperadrenocoriticsm



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Canine Hyperlipidemia
"Vader"

- Physical Exam
  - BCS 7/9; normal vital parameters
  - Euhydrated
  - Normal thoracic auscultation; palpation hepatomegaly
- Diagnostics
  - CBC/Chemistry panel: ALT 162, ALP 1114, cholesterol 602, triglycerides 2356, wbc 18.4k, neutrophils 11k with mild left shift
  - Urinalysis: usg 1.029, 1+ protein; UPC 0.8
  - T4 1.4
  - Abdominal ultrasound: enlarged hyperechoic liver with few nodules, mild biliary sludge accumulation, moderately heterogenous pancreas with irregular margination mild surrounding peritonitis, normal adrenal glands
  - LDDS test: resting - 3.2, 4hr – 0.5, 8hr – 0.4

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Canine Hyperlipidemia
"Vader"

- Presumptive diagnosis: Pancreatitis associated with primary hyperlipidemia and suspected vacuolar hepatopathy
- Initial Treatment
  - Hospitalized for 3 days for pancreatitis
  - Discharged on clavamox, cerenia, and RC GI LF
- Follow-up @ 2 weeks
  - Much improved; eating 90%, no vomiting or diarrhea
  - Focused u/s noted resolution of peritonitis
  - Repeat bloodwork: ALT 202 (162), ALP 984 (1114), cholesterol 491 (602), triglycerides 1716 (2356)
  - Started omega-3 fatty acids and denamarin and continued RC GI LF

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Canine Hyperlipidemia
"Vader"

- Follow-up @ 6 weeks
  - Doing well at home; no issues
  - Repeat bloodwork: ALT 122 (202), ALP 1001 (984), cholesterol 449 (491), triglycerides 1262 (1716)
  - Added fenofibrate at 10mg/kg/d
- Follow-up @ 10 weeks
  - Still doing well at home
  - Repeat bloodwork: ALT 68 (122), ALP 532 (1001), cholesterol 216 (449), triglycerides 299 (1262)
  - Continued omega-3 FA, fenofibrate, and RC GI LF

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Canine Hyperlipidemia

- Seen on a daily basis and often underestimated
- Hypertriglyceridemia most common lipid disorder in dogs and cats
- Pancreatitis is major complication
- Diagnosis requires a 12 hour fast
- Identify and treat potential underlying causes
- Tx: low fat diet, omega-3 fatty acids, fibrates

The end you have reached. Wiser have you become

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What are some guidelines for the management of megaesophagus?
What is a good I^{131} candidate?
Patient with a flare of IMT. How do I manage?
How do I best manage a dog with collapsing airway disease?
When do I need to be worried about increases in cholesterol/triglycerides?
Is laser lithotripsy a viable option to remove bladder stones?
What probiotic is best for dogs and cats?
Patient with suspected hyperadrenocorticism but normal LDDS. What is next best step?
When/how to manage proteinuria in a dog?
Cat with suspected ureteral calculi. What are options for care?
Can I get some information of the new pancreatitis drug available?
How best to treat a case of histoplasma or heterobilharzia?
Cat with suspected FIP. Are there any new treatments?
How do I best incorporate the use of a wearable glucose monitor?

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I recently saw a cat for some respiratory problems and radiographs of his chest were read out as concerning for fungal disease. I recommended seeing a specialist but the owner has financial limitations and so I was looking for some guidance on what we might be able to do?

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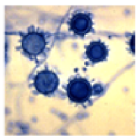
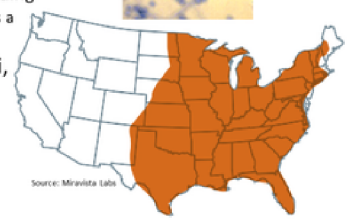
Histoplasmosis

- Causative disease of *Histoplasma Capsulatum*
- Saprophytic, dimorphic fungus
  - Free-living mycelium in nature
  - Yeast in host tissue that reproduces by budding
    - 2-4µm with a thin, clear cell wall that surrounds a round or crescent-shaped basophilic cytoplasm
- Endemic within the Mississippi, Missouri, and Ohio River valleys





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Histoplasmosis – Risk Factors

- Young animals (<4yrs)
- Exposure to large numbers of infective conidia
- Cats equally infected as dogs
- Correlation with immunosuppressive conditions not identified

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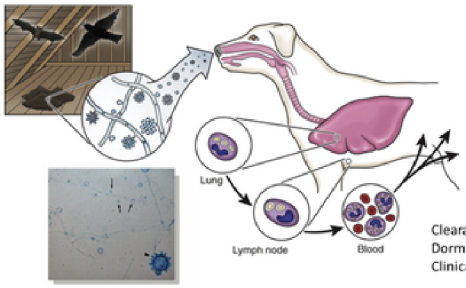
Histoplasmosis – Pathogenesis



Consequence of Exposure
Clearance due to cell-mediated immunity
Dormant Infection
Clinical disease

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Clinical Signs of Histoplasmosis in Dogs

- Gastrointestinal
  - Seen in 80% of clinically infected dogs
  - Weight loss and large bowel diarrhea with hematochezia
  - Vomiting is rare
- Pulmonary
  - Usually subclinical
  - Seen in around 17% of infected dogs
  - Dyspnea, tachypnea, coughing, abnormal lung sounds
- Ocular, dermatologic, orthopedic, cutaneous signs uncommon

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Clinical Signs of Histoplasmosis in Cats

- Pulmonary
  - Occurs in 38-93% of cases
  - Dyspnea, tachypnea, abnormal lung sounds
  - Coughing uncommon
- Ocular
  - Seen in~25% of cases
  - Anterior uveitis, chorioretinitis, and/or blindness
- Bone marrow and cutaneous signs possible
- Gastrointestinal signs rare
- Fever more prevalent than in dogs

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Diagnosis of Histoplasmosis

- Bloodwork
  - Often non-specific and/or unremarkable
  - Non-regenerative anemia, hypoalbuminemia, and hyperglobulinemia
  - Increased LE with liver involvement
  - Hypercalcemia uncommon
- Radiographs
  - Diffuse nodular or miliary interstitial pattern
  - Hilar lymphadenopathy common
  - Osteolysis and periosteal proliferation

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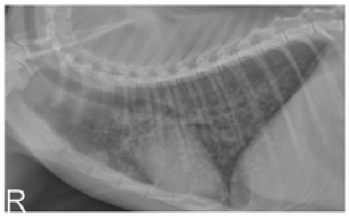
Diagnosis of Histoplasmosis



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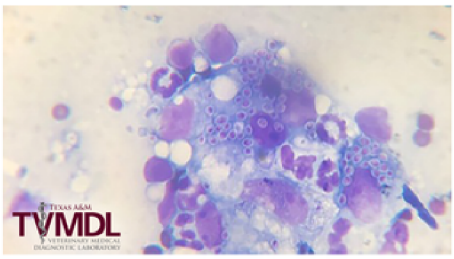
Diagnosis of Histoplasmosis

- Cytology/Histology
  - Historical gold standard
  - Bone marrow, lung, or lnn aspirates in cats
  - GI biopsies, rectal scrapings, or aspirates of liver, spleen, or lnn in dogs
  - Bx: Granulomatous to pyogranulamous inflammation



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Diagnosis of Histoplasmosis

- Serology
  - Unreliable and not recommended
  - Many false positives and negatives
- Antigen testing
  - Detects galactomannan in cell wall of fungi
  - Best performed on urine or CSF
  - EIA from Miravista preferred with Se of 95% and Sp of 99% (Clark, JVM 2021)
    - Cross reactivity with blastomyces and others possible
    - Higher rate of false negatives with renal insufficiency and primary ocular disease
  - Can be used to monitor response to therapy

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Treatment of Histoplasmosis

- Indicated in all clinical cases
- Standards of Care
  - Itraconazole or fluconazole +/- amphotericin B
  - Treat for minimum of three months and at least one month past resolution of clinical signs
  - Recheck liver values in one month and then q2-3 months
  - Recheck antigen level after 8-16 weeks then q2-3 months
  - Author's preferred treatment: Minimum of 16 weeks followed by two consecutive negative antigen tests taken 2-3 months apart
  - Recommended q3-4 months antigen tests for first year after stopping tx

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Treatment of Histoplasmosis - Itraconazole

- Drug of choice for both dogs and cats in most cases
- Generic vs. branded vs. compounded
  - ➢Higher rates of variable bioavailability with compounded versions
- Liquid vs. capsules
  - ➢Liquid products containing cyclodextrin (Itrafungol and Sporonox) have higher bioavailability (30%) than similar branded capsules
  - ➢Liquid itraconazole best given on an empty stomach
  - ➢Capsular intraconazole best given with fatty meal
- ➢Possible GI signs and hepatotoxicity
- ➢Dose: 5-10mg/kg BID

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Treatment of Histoplasmosis - Fluconazole

- May be better with ocular or CNS involvement
- Undergoes renal excretion so may be safer with hepatic insufficiency
- Direct comparisons lacking but anectedol evidence suggests equally as effective as itraconazole
- Treatment likely to be needed longer but generally less expensive
  - Author uses fluconazole for a minimum of 6 months
- Dose: 5-10mg/kg BID

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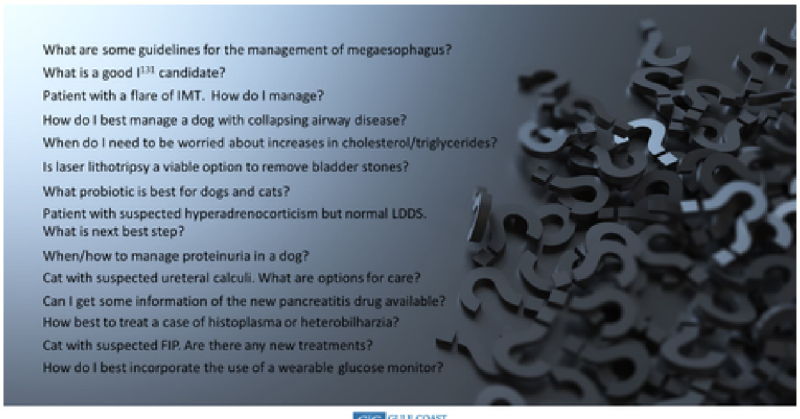

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AVIAN & EXOTICS

SUSAN BALEY

DVM, DABVP

Dr. Susan Baley received her Bachelor's degree in animal science with an emphasis in poultry management from the California Polytechnic State University in San Luis Obispo. She continued her education with a Doctor of Veterinary Medicine degree from the University of Minnesota in 2018. Upon graduation, she completed an avian and exotic animal medicine internship at Gulf Coast Veterinary Specialists. Dr. Baley completed a residency in avian medicine at Stahl Exotic Animal Veterinary Services in Fairfax, Virginia in 2021 and is actively working towards becoming board certified in avian practice.

Dr. Baley enjoys working with all kinds of exotics, but has a special interest in poultry medicine, endoscopy, and soft tissue surgery of exotic animals. Outside of the hospital, Dr. Baley enjoys hiking, playing video games, traveling, crafting, and spending time with her family and friends.

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Backyard Chicken Medicine:

What the cluck do I need to know?

Susan Baley DVM, Dipl ABVP (Avian)
Avian & Exotics Department
Gulf Coast Veterinary Specialists Symposium
August 19,2023

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The Popularity of Backyard Chickens

- Ownership of backyard chickens increased from 8% (2018) to 13%(2020).
- "Backyard chickens in the United States: A survey of flock owners"
  - 71% of respondents owned fewer than 10 chickens
  - Reasons for keeping chickens were as food (95%), gardening (63%), pets (57%)
  - Sources of info for owners: Internet (87.4%), books/magazines (62.5%), feed stores (40.2%), university poultry extension specialists/extension publications (28.2%).

Only 18.8% of respondents listed a general/avian veterinarian as a source of information

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NAHMS Backyard Animal Keeping Study

- Study to take place mid 2024
- Study will collect national estimates on the percentage of households that keep poultry, goats, rabbits, and pigs.
- All respondents will be asked questions about biosecurity, animal husbandry, and on their opinion on backyard animal keeping

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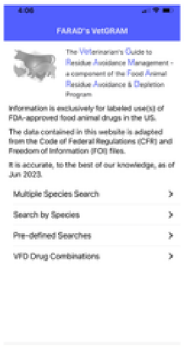
FARAD

- Chickens (and turkeys) = major producing food animals according to the FDA
- Be sure to check for withdrawal time for a drug
- There's an app for that!



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Prohibited and Restricted Drugs

- Group 1 Drugs: Not allowed for extra-label use in any food-producing animals
  - Chloramphenicol
  - Clenbuterol
  - Diethylstilbesterol (DES)
  - Fluroquinolone antibiotics
  - Glycopeptides- including vancomycin
  - Medicated Feeds
  - Nitroimidazoles- Metronidazole, Dimetridazole, etc.
  - Nitrofurans

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Prohibited and Restricted Drugs

- Group 2 Drugs: Restricted extra-label use in food-producing animals
  - Adamantane and Neuraminidase inhibitors – Prohibited in poultry- chickens, ducks, turkeys
  - Cephalosporins
  - Gentian Violet
  - Indexed drugs- prohibited in all MAJOR food producing animals
  - Phenylbutazone
  - Sulfonamide-class antibiotics – prohibited in lactating dairy cattle

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What medication can I use?

Drug Groups and Specific Examples that May be Used in Poultry in the United States

Drug Group	Examples
Macrolides	Tylosin, erythromycin
Tetracyclines	Oxytetracycline, chlortetracycline, doxycycline (extra-label)
Lincosamides	Lincomycin, clindamycin (extra-label)
Sulfonamides	Trimethoprim sulfa (extra-label)
Aminoglycosides	Gentamycin, novobiocin, streptomycin
Penicillins	
Spectinomycin	
Novobiocin	
Bacitracin	

Note: Prior to use, seek specific guidance on egg and meat withdrawal times.

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Biosecurity

- Avoid the spread of disease by practicing good biosecurity
- Wash hands before and after handling poultry
- Put control programs in place for pests
- Clean/disinfect equipment used for poultry and their care
- Check your flock daily and quarantine any sick birds
- Report sick birds

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Reportable Diseases in Texas

Poultry diseases

- Avian influenza - Orthomyxoviruse
- Avian infectious laryngotracheitis - Orthomyxovirus, herpesvirus
- Avian tuberculosis - Mycobacterium avium serovars 1,2
- Duck virus hepatitis - Picornavirus
- Fowl typhoid - Salmonella gallinarum
- Highly pathogenic avian influenza (fowl plague) – Orthomyxovirus (type H5 or H7)
- Infectious encephalomyelitis - Arbovirus
- Ornithosis (psitticosis) - Chlamydia psittaci
- Pullorum disease - Salmonella pullorum
- Newcastle disease (VVND) - Paramyxovirus-1 (PMV-1)
- Paramyxovirus infections (other than Newcastle disease) - PMV-2 to PMV-9

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Good resources for more information

- Texas Animal Health Commission
- https://www.tahc.texas.gov/regs/reportable.html
- Texas Department of State Health Services https://www.dshs.texas.gov/ For zoonotic diseases

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Physical Examination

- Start with a hands-off approach
  - Body posture, mentation
  - Respiratory rate/effort
- Eyes, nares, ears
- Head, neck, comb, wattles
- Oral cavity- tongue, choana, beak
- Feathers
- Coelomic palpation
- Auscultation- heart, lungs, air sacs
- Legs and feet
- Vent

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Common Backyard Chicken Emergencies

Reasons for evaluation on an emergency basis of and short-term outcomes for chickens from backyard flocks: 78 cases (2014–2017)

- Most common presentations ---- trauma followed by non-specific signs, and then reproductive disease.
- 50% of chickens that presented to ER service survived to discharge

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Trauma

- Clean and irrigate open
- Bandage wounds to help stabilize the patient
- Analgesia- butorphanol, ketamine, tramadol
- Anti-inflammatory- meloxicam, carprofen
- If trauma is from a predator, start antibiotics ASAP
- Fracture stabilization follows the same principles as in other avian species
- Orthopedic surgery

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RBG aka "Ruthie"

4 week old Easter egger

Attacked by a raccoon

Traumatic amputation of the left wing at the level of the humerus

Initial treatments

Analgesia – Butorphanol 2mg/kg IM

Meloxicam - 1mg/kg IM

Subcutaneous fluids- LRS 50ml/kg

Pip/Taz 100mg/kg IM

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"Ruthie"

Continued treatments:

- Wound debridement under general anesthesia
- Sedation: butorphanol (2mg/kg) + midazolam (2mg/kg) + gas anesthesia
- Debrided edge of distal humerus and apposed muscle bellies over remaining bone of the humerus.
- Skin closed in a simple continuous pattern
- Discharged after 24 hours with meloxicam, clavamox, and gabapentin.

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Reproductive Disease

- Common causes include: Egg binding, salpingitis, egg peritonitis, oviductal impaction or disease, ovarian/oviductal neoplasia
- Lethargy, dehydration, coelomic distension, increased resp. rate/effort, decreased egg production, abnormal eggs
- CBC/Chem panel, radiographs, U/S, cloacal endoscopy
- Tx: Antibiotics, anti-inflammatory medication, fluid therapy, +/- coelomocentesis
- Surgical exploratory and/or salpingohysterectomy

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Oviductal or Cloacal Prolapse

- Stress, age, obesity, and poor nutrition can contribute
- Edematous, inflamed, necrotic
- Use moistened CTAs to return tissue to normal anatomic position
- Swelling can be reduced with lubrication, 50% dextrose, or sugar
- Important to find underlying cause of the prolapse, can often recur
- Salpingohysterectomy/Cloacoplasty may be needed

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Respiratory Distress

- Potential causes: infectious disease, respiratory toxin, tracheal foreign body, severe esophageal trauma, organomegaly, ascites, neoplasia, cardiovascular disease
- Clinical signs:
  - Open beak breathing
  - Tail bobbing
  - Eyes closed
  - Hunched body position, keel sitting, or laying to the side
  - Pale or cyanotic or purplish comb/wattles

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Respiratory Distress

- O2 cage/oxygen supplementation
- Injectable butorphanol 1-2mg/kg
- Midazolam 1-4mg/kg
- Treat underlying cause
- Blood work, radiographs, ultrasound
- Coelomocentesis may be needed if free fluid is noted on palpation

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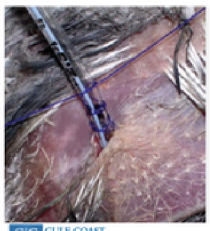
Respiratory Distress

- Place air sac cannula under gas anesthesia if indicated
- Obese chickens and poultry may have reduced air sac space



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Respiratory disease
Mycoplasma gallisepticum

- Vertical and horizontal transmission
- Respiratory rales, ocular and nasal discharge, swelling of intraorbital sinuses with caseous pus, conjunctivitis, decreased egg production
- Diagnosis: PCR, serology, isolation
- Tx: Tylosin- treats the clinical signs, but does not eliminate the infection or carriers

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Respiratory disease

Avian Influenza

- Orthomyxovirus
- High pathogenicity REPORTABLE
- High morbidity/mortality
- Transmission- Direct and indirect
- Diagnosis: Virus isolation, PCR of choana or cloacal swabs
- Treatment: None. Depopulation is mandatory

USDA file photo

Purple discoloration of the comb could indicate HPAI.

USDA file photo

Birds affected by HPAI could show swelling of the head, wattles, combs, and face.

USDA file photo

Hemorrhaging of the skin and legs is just one of the signs birds might exhibit when infected with the HPAI virus.

USDA file photo

Nasal discharge (a runny nose) can be a sign of HPAI.

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Neurologic Disease

- Possible causes: Marek's, AI, Exotic Newcastle, AE, lead poisoning, trauma, nutritional deficiencies, ischemia, stroke, toxin.
- Clinical signs: Ataxia, torticollis, seizures, paresis, paralysis, weight loss, weakness, bright green feces with heavy metal toxicosis
- Treatment: Dependent on cause

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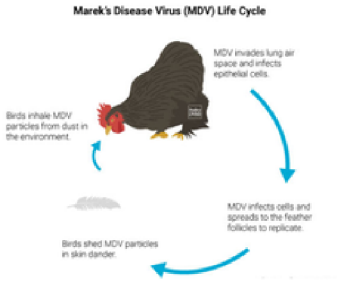
Neurologic Disease

Marek's Disease

- Gallid herpesvirus 2
- Mononuclear infiltration of nerves, organs, reproductive tract, iris, muscle and skin
- Cachexia, generalized weakness, diarreha, pale comb, unilateral paresis/paralysis, iris color change to grey, swollen feather follicles
- No treatment
- Vaccine available- does not prevent contraction of the virus



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Neurologic Disease

Exotic Newcastle Disease (END)

- Paramyxovirus type 1
  - Viscerotrpic velogenic form is END
- REPORTABLE
- High mortality
- Clinical signs: blue discoloration of shank, wattles & comb; hemorrhage, torticollis, paralysis, ataxia, piling.
- Diagnosis: serology, virus isolation, PCR
- Treatment: None. Depopulation.

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Maxine

- 1.5 yr hen
- Unable to stand, ataxia, torticollis
- Fed a laying hen diet
- Others in the flock were acting normal
- Differentials: Head trauma, otitis interna/media, toxin, nutritional deficiency, infectious (bacterial, viral, fungal)

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Maxine

- Treatments:
  - Butorphanol 2mg/kg IM q8-12hr
  - Meloxicam 1mg/kg PO q12h
  - Syringe feedings
  - Subcutaneous fluids
  - Vitamin B complex – 3mg/kg IM
  - Vitamin A/D/E – 0.15ml/kg IM

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Maxine

After 48 hours in the hospital, she is doing much better

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Diarrhea

- DDx: Coccidia, worms, bacteria,virus, high protein in the diet, kidney disease
- Diagnostics: Fecal float, direct , fecal cytology, culture/sensitivity
- Prevention: Proper litter management and yearly fecal examinations

Coccidia

- Species-specific. Multiple types affect chickens.
- Often seen in overcrowded flocks
- Clinical signs of cecal coccidiosis: bloody diarrhea, lethargy, dehydration, weight loss, death if left untreated
- Primarily seen in younger birds < 4mo, but can be seen in adults
- Tx: Amprolium

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Salmonellosis

4 main types in poultry: S.Pullorum, S.gallinarum , S.typhimurium & S.enteritidis

- S. pullorum and S. gallinarum - cause the greatest problem for poultry ***REPORTABLE***
- S. pollurum: fatal enteritis and septicemia
- Chicks infected from chronic carrier adults
- Prevention: Buy chicks from disease-free breeders

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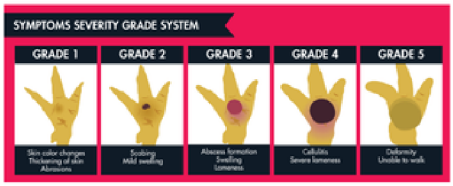
Pododermatitis



Bumblefoot

- Bandaging and "shoes"
- Epsom salt soaks
- Analgesics
- Antibiotics
- Culture/Sensitivity
- Radiographs to check for osteomyelitis
- Debridement of scabs or opening an abscess is often necessary

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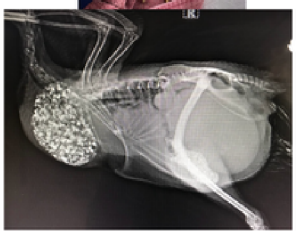
Crop Stasis



- Potential causes: many
- Clinical signs: regurgitation, weight loss, lethargy, distended/pendulous crop
- Diagnostics: CBC/Chem panel, radiographs, CT scan, crop cytology, fecal, +/- heavy metal testing
- Tx: SQ or IV fluids, analgesics, +/- prokinetics, crop aspiration, ingluviotomy

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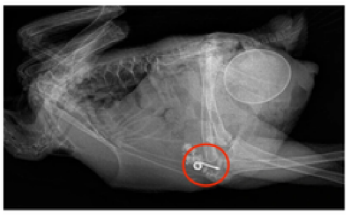
Foreign Body Ingestion



- Can be observed in the rhamphotheca, tongue, crop, and gastrointestinal tract.
- Imaging: x-ray, U/S, CT scan, other
- Endoscopy can be attempted; sometimes surgery is required.
- Treat similarly as in other species

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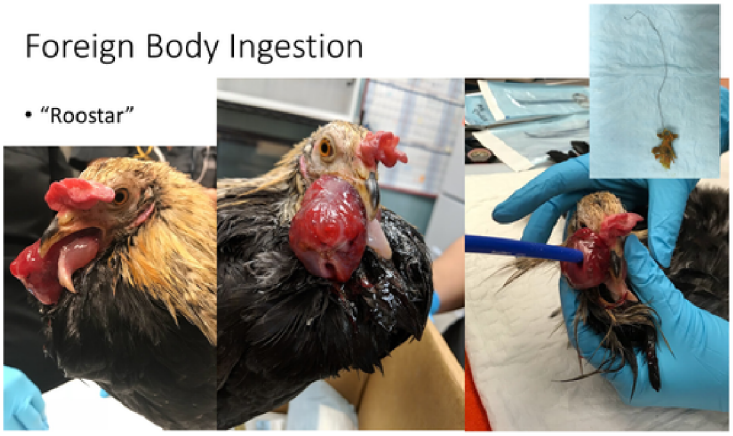
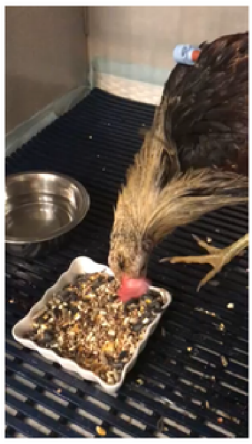
Foreign Body Ingestion

- "Roostar"



Foreign Body Ingestion

- "Roostar"



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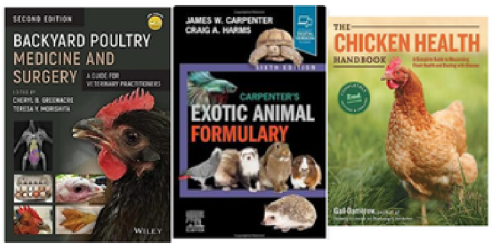
Other good resources for more information

- https://partnersah.vet.cornell.edu/avian-atlas/#/ Atlas of Avian Diseases by Cornell University
- https://posc.tamu.edu/texas-agrilife-poultry-extension-specialists/publications/ Department of Poultry Science Texas A&M



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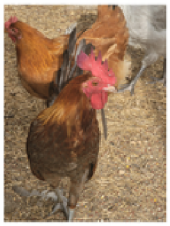
Thank you!

Contact information:
susan.baley@gcvs.com



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NEUROLOGY

CARLEY GIOVANELLA

DVM, DACVIM (NEUROLOGY)

Dr. Carley Giovanella received her doctor of veterinary medicine from Cummings School of Veterinary Medicine at Tufts University in 1999, followed by an internship at the University of Georgia. Her residency in neurology and neurosurgery was completed at the University of Wisconsin and the Animal Health Trust in Newmarket, England. Dr. Giovanella joined the faculty at The Ohio State University College of Veterinary Medicine in 2003 as an assistant professor and clinical neurologist/neurosurgeon. She became board certified by the American College of Veterinary Internal Medicine – Specialty of Neurology in 2004. She joined Gulf Coast Veterinary Specialists in 2005.

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DISCOSPONDYLITIS: "Brucie" is NOT your friend

Dr. Carley Giovanella, DVM, DipACVIM (Neurology)

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AVA

- Adopted in 2020 from as a puppy (assumed 3 months old)
- Spayed 3 months later
- Never wanted to jump
- Low back sensitivity
- Rear lameness in 2021

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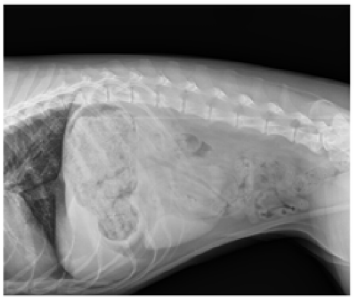
Additional Testing

- 3/2022- acute yelping in pain (21kg)
  - Rimadyl -50mg twice daily
  - Gabapentin -200mg twice daily
  - Ketamine -0.5mg/kg subQ
- Referral made to GCVS



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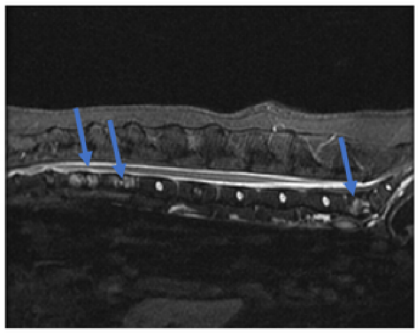
Referral 4/2022

Exam- Unremarkable, normal neurological evaluation

CBC and serum chemistry- unremarkable

Spinal MRI –



Urine culture

Blood culture

CSF analysis and culture

Brucella testing- IFA and AGID

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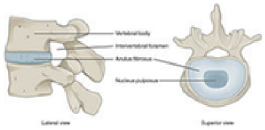
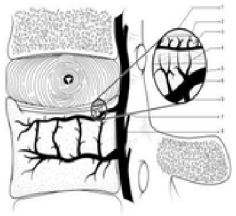
Intervertebral Disc Infection

Anatomy of the Disc



Vascular Supply of the Disc



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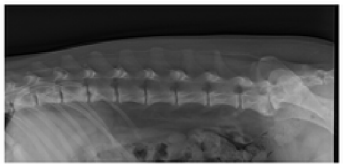
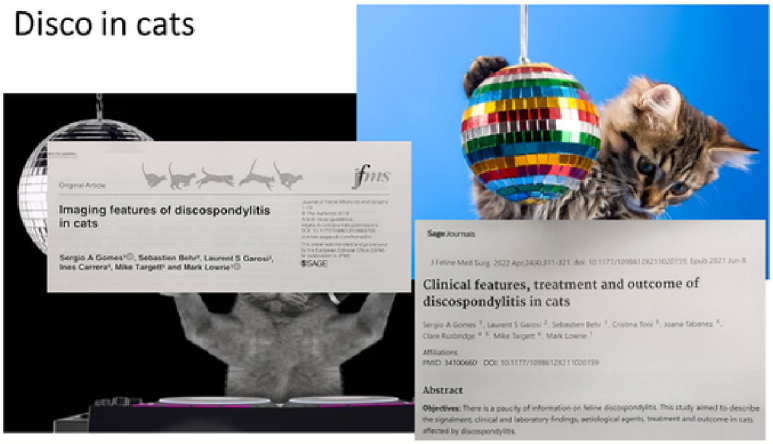


Where it comes from

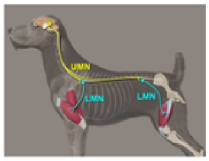
- Hematogenous –
  - bladder
  - skin
  - oral cavity
  - heart valves
- Foreign-body migration
- Cat bite/claw
- Iatrogenic infection
  - spinal surgery
  - paraspinal injection

GULF COAST VETERINARY SPECIALISTS

Disco in cats



Clinical Signs - Location



- Pain
- Lethargy
- Fever
- Late:
  - Lameness
  - Severe pain
  - Nerve root signature
  - Incontinence

GULF COAST VETERINARY SPECIALISTS

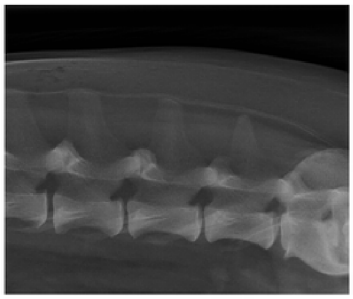
Organisms

- Staph pseudointermedius
- Staph aureus
- Strep
- E.coli
- Fungal (Aspergillus, Coccidioides, Actinomyces)
- *Brucella

GULF COAST VETERINARY SPECIALISTS

Workup

- Lab work (CBC, chemistry, tick panel)
- Spinal radiographs –
  - sedated
  - repeat 2-4 weeks later if suspect case
- Blood and Urine Cultures
- Fungal
- Brucella Testing
- ----------
- Referral (CT/MRI/CSF with cultures)
- Disc aspirates



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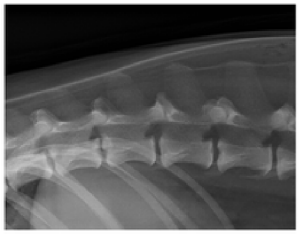
Imaging Findings

- Radiographs
  - Endplate sclerosis
  - Lysis of endplates
  - Narrowed disc space
  - May lag behind onset and resolution of disease



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Blood Cultures

- Shave and sterile prep site
- Clean top of culture bottle with alcohol and allow to dry
- Wear sterile gloves
- Collect 5-10ml of blood from each site
- Collect from 3* sites one hour apart.

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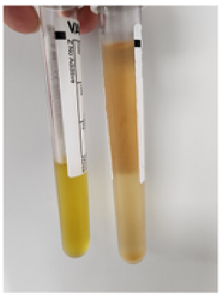
Urine Collection

- Urine collection:
  - Sterile cystocenteses



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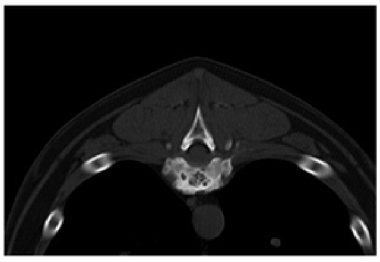
Additional Tests

- Fungal screen
  - Culture, serology, urine
  - Travel history
    - Penicillium purpurgenium (IT)
    - Blasto/crypto in US regions
- Brucella*
  - Two tests

GULF COAST VETERINARY SPECIALISTS

Advanced Imaging

- CT
  - Endplate erosion
  - Osteomyelitis
  - Fractures
  - Rapid acquisition and evaluation of whole spine



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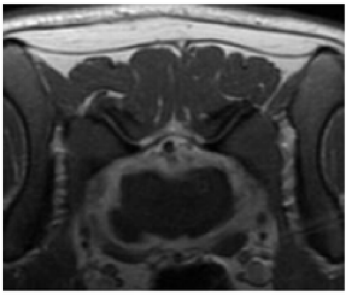
- MRI
  - Signal change of endplate
  - Disc bright on T2W and STIR
  - Paravertebral soft tissue edema/contrast
  - Nerves/meninges assessed
  - T1W endplate erosion seen later



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So, you got a diagnosis: the "easy" answer

1

Bacterial

Base on culture if possible

Start with Staph/Strep possibility

8 weeks minimum

3-4 weeks beyond radiograph resolution

Pain medications
Rest/confinement

2
3

Fungal

Based on typing
Itraconazole 10mg/kg/day
Fluconazole 10mg/kg/day

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Follow up

- Controlled activity at home
- Radiograph every 6-8 weeks
- Neurological exams every 6-8 weeks

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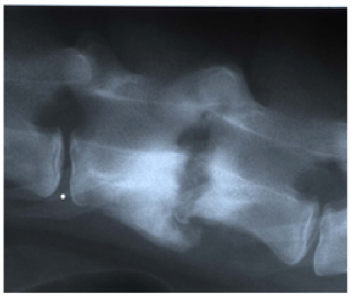
Complications

- ❑Meningitis, neuritis, spinal empyema
- ❑Spinal fracture
- ❑Sepsis
- ❑Antibiotic resistance
- ❑Persistent pain (QOL)



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AVA Diagnosis

- Urine culture – negative
- CSF – 5 cells/ul and 65 mg/dl protein (elevated protein and increased neutrophils noted on cytology)
- CSF culture - negative
- Brucella -
  - IFA – POSITIVE 1:1200
  - Slide Agglutination – POSITIVE
  - AGID II - NEGATIVE

GULF COAST VETERINARY SPECIALISTS

AVA Diagnosis

- Blood Cultures –
- Gram negative organism
- Possible Brucella – culture positive
- confirmed PCR positive for Brucella

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So, you got a diagnosis: the “difficult” answer

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Brucellosis (infection with a Brucella spp)

Bacterial organism

- Gram negative
- 12 strains
- Zoonotic, but host-preferred
- Attach to mucous membranes and reside in mononuclear cells
- Human signs: fever, large ln, miscarriage potential

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Public Health considerations

- B.canis first reported in humans in 1968.
  - Aborted fetal material
  - Lab aerosolization
- Reportedly (unreliable) 1% of all Brucella
- Immunocompromised/young/elderly
- Occupational hazard *

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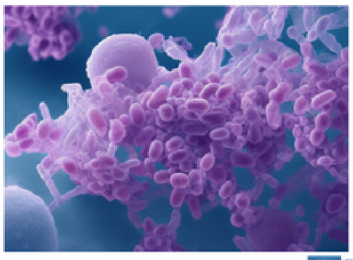
BRUCELLA CANIS



- Most common spp infecting dogs
- Symptoms:
  - DISCOSPONDYLITIS
  - Uveitis
  - Orchitis
  - Epididymitis
  - Abortion
- May be sub-clinical*

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Diagnostic Testing

- Difficult due to test sensitivity and specificity – 2 test confirmation
- Antibody tests are more sensitive.
- PCR specific but false negatives
- Rsat - screening
- ELISA
- IFA
- AGID – confirmation
- Culture

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Treatment protocols

- Euthanasia

- Enrofloxacin 10mg/kg/day + Doxycycline 10mg/kg/day

- Gentamycin – day 1-7 and 21-28 + Doxycycline 10mg/kg/day
  - 5mg/kg/day subQ
  - 20ml/kg subQ fluids
  - Renal profile, urine SpGravity and urine sediment day 1, 7, 21, 28

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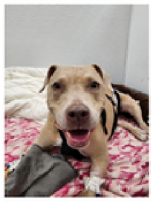
Follow up testing/management

- Test again one month after Gentamycin protocol.
- I test after 3 and every 6 months of Enrofoloxacin protocol
- RE-image at 3 or 6 months (rads vs MRI)
- Lifelong antibiotics
- Lifelong lifestyle adjustment



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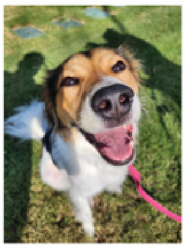
Ava update

- Enrofloxacin/Doxy for a year, still high IFA
- Gentamycin protocol completed one month ago – repeat titers pending



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Brucella canis

Do I report it?

- Not reportable in TX (it is in other states)
- Rare to see human infection
  - Immunocompromised or young/old
- Perception of increase
- Recent article shows Texas has high caseload

Why the increase?

- Travel/transport
- Natural disasters
- Foster/shelter shuffle

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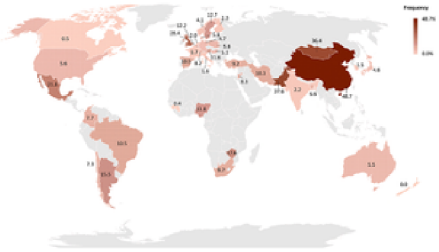

Canine Brucellosis: An Update

-Santos et al, frontiers in Veterinary Science 2021

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Kennel Management

- Euthanize
- Pre-test entry or breeding
  - Two tests, repeat if recent exposure
- If confirmed, deep clean

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Family Dog Management

- Euthanize
- Antibiotic course
- No daycare or parks
- Avoid small children and elderly
- Avoid immunocompromised
- Life long monitoring/risk

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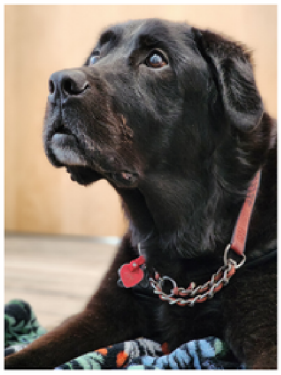
Future

- Test animals in shelter/rescues?
  - Funding
  - Protocol
  - Enforcement

Questions?



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Pathology

- Routes through digestive and respiratory mucosa
- Poor inflammatory stimulus in the host
- B canis mostly abortive in animals (febrile in humans)
  - Metritis, placentatitis/ abortion chorionic Villi necrosis and bacteria
  - Aborted fetus may have bronchopneumonia, myocarditis, renal hemorrhage, lymphadenitis and hepatitis.

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CRITICAL CARE

HERNAN BRACHO

DVM, MVZ, ESP

Dr. Bracho, originally from Mexico, earned his veterinary degree there. After a two-year Internal Medicine and Surgery residency, he practiced in Mexico City for 8 years. During his time at the Veterinary Teaching Hospital, he supervised Social Services and Practice Programs while also serving as an associate veterinarian.
In 2010, he moved to Houston, TX, practicing as a general practitioner and a key referring DVM for 6 years. Dr. Bracho even held the role of Chief of Staff at Banfield Pet Hospital. His desire for advanced training led him to pursue a three-year Emergency and Critical Care residency at the University of Wisconsin, culminating in July 2021.
With special interests in point-of-care ultrasound, cardiology, sepsis, and mentoring new veterinarians, Dr. Bracho is dedicated to medical excellence. Beyond his professional pursuits, he finds joy in soccer, fitness, photography, and travel. He shares his life with two rescue dogs, Nemi and Chip, who keep him active.

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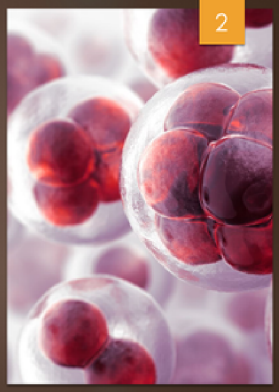
1

Sepsis won't hesitate, will you?

2

Outline

- Definitions
- Current statistics
- Criteria for SIRS & sepsis
- Pathophysiology & key factors for SIRS/sepsis
- Treatment: bundle concept
  - Lactate
  - Source identification & control
  - Antibacterial therapy
  - Fluid/vasopressor/inotropic therapies



3

Assessment of Global Incidence and Mortality of Hospital-treated Sepsis. Current Estimates and Limitations

Carolin Fleischmann [1,2], André Scherag [3], Neill K. J. Adhikari [4], Christiane S. Hartog [1,2], Thomas Tsaganos [5], Peter Schlattmann [6], Derek C. Angus [7]*, and Konrad Reinhart [1,2]*; on behalf of the International Forum of Acute Care Trialists

+ Author Affiliations

https://doi.org/10.1164/rccm.201504-0781OC PubMed: 26414292

Received: April 20, 2015 Accepted: September 25, 2015

Global estimates

- 31.5 million sepsis
- 19.4 million severe sepsis

Potentially 5.3 million deaths/year

Statistics….

4

- 783 articles assessed
- 170 studies included
- Septic shock mortality
  - ~ 30-d 34.7% (33.7% in NA)
  - 90-d 38.5%
- Sepsis mortality
  - 30-d 24.4%
  - 90-d 32.2%

Research | Open Access | Published: 19 May 2020

Mortality in sepsis and septic shock in Europe, North America and Australia between 2009 and 2019—results from a systematic review and meta-analysis

Michael Bauer, Herwig Gerlach, Tobias Vogelmann, Franziska Preissing, Julia Stiefel & Daniel Adam

Critical Care 24, Article number: 239 (2020) | Cite this article

Despite some evidence of lower mortality rates over the past decade, continuous reduction was not observed

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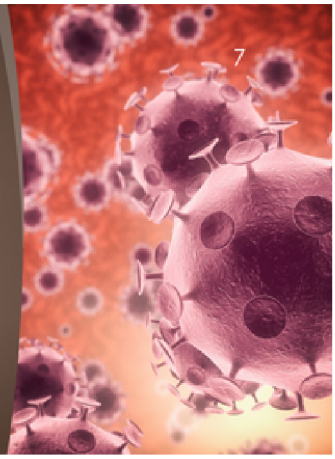
Vetmed statistics…

5

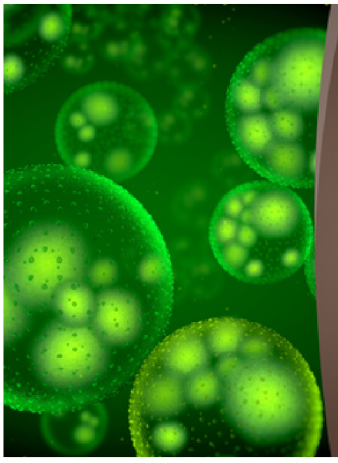
6

Statistical conclusions in vetmed

- Incidence is unknown
- Mortality ***appear*** to be high, (20 to 70% ***or more***)

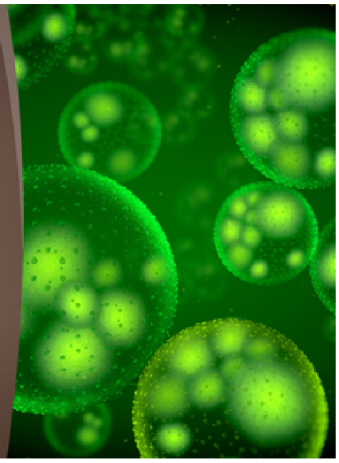


Definitions



- **Bacteremia**
  - Live bacteria in the bloodstream
- **Systemic inflammatory response syndrome (SIRS)**
  - Exaggerated inflammatory response to stressors

- **Sepsis**
  Life threatening organ dysfunction caused by a dysregulated host response to infection
- **Septic shock (SS)**
  - Sepsis + circulatory failure



11

Where to start?

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SIRS criteria

1. Hypo or hyperthermia
2. Leukocytosis or leukopenia
   1. Bands %
3. Tachycardia/ Bradicardia C
4. Tachypnea

Table 6-1 Proposed Criteria for the Diagnosis of SIRS in Dogs and Cats

	Dogs (2/4 Changes Required)*	Cats (3/4 Changes Required)
Temperature (°C)	<100.6 or >102.6	<100 or > 104
HR (beats/min)	>120	<140 or >225
RR (breaths/min)	>20	>40
WBC (x10^3/µl); % bands	<6 or >16; >3%	>19 or <5

HR, Heart rate; *RR*, respiratory rate; *SIRS*, Systemic inflammatory response syndrome; *WBC*, white blood cells.
*Proposed criteria for the diagnosis of SIRS include at least two (in dogs) or three (in cats) of the changes listed. Criteria described for dogs were found to have a sensitivity of 97% and a specificity of 64% for the diagnosis of SIRS.[35]

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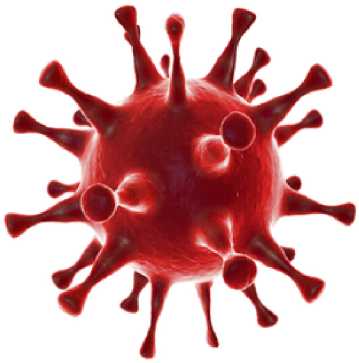

Sepsis criteria

- SIRS + documented or suspected infection

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Abstract

Objective—To evaluate the ability of various individual criteria and grouped criteria to diagnose sepsis in dogs.

Study Design—Prospective acquisition of clinical data.

Animals or Sample Population—Client-owned dogs; 30 septic and 320 nonseptic.

Methods—Rectal temperature, heart rate, respiratory rate, white blood cell (WBC) count with percent bands, platelet count, and serum glucose concentration were obtained on day 0. True sepsis was determined on days 0 to 3 according to the following criteria: (1) histological, microbiological, and/or gross confirmation of infection, and (2) systemic illness caused by infection. Data were analyzed by the Mann-Whitney U test and multiple logistic regression. Sensitivity and specificity were calculated.

Results—The mean temperature, heart rate, WBC count, and percent bands were greater, whereas the mean platelet count was less in septic compared with nonseptic dogs. There was no difference in respiratory rate or glucose concentration. WBC/bands were the best individual criterion for the diagnosis of sepsis (sensitivity 87%; specificity 69%). The sensitivity and specificity of the grouped criteria (two of four; temperature, heart rate, respiratory rate, WBC) varied according to ranges of normal used. Multiple logistic regression resulted in little improvement in the sensitivity/specificity of these diagnostic criteria for the diagnosis of sepsis.

Conclusions—These criteria are useful for the diagnosis of sepsis when limits are used that result in a high sensitivity (eg, 97%). The high sensitivity was associated with a low false-negative and a high false-positive rate; sepsis was overdiagnosed with these grouped criteria.

Clinical Relevance—These criteria may be used for a sensitive, but nonspecific, diagnosis of sepsis in dogs.

Evaluation of the Sensitivity and Specificity of Diagnostic Criteria for Sepsis in Dogs

J. G. HAUPTMAN DVM, MS, Diplomate ACVS, R. WALSHAW BVMS, Diplomate ACVS, N. B. OLIVIER DVM, PhD, Diplomate ACVIM

- Temp, HR, WBC & bands higher in sepsis vs non-sepsis
- Platelet ct less in sepsis vs non-sepsis
- WBC/bands best individual criterion

These criteria is highly Se, poor Sp

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Identify an infectious source

Common septic foci in D&C

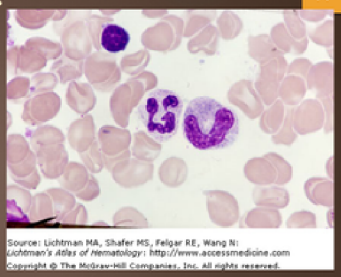
16

SITE	DOG	CAT
Respiratory/pleural	20%	24%
Peritoneal cavity	35%	47%
Gastrointestinal	4%	5%
Reproductive	25%	
Urinary	4-10%	7-8%
Soft tissue	29%	16%
Cardiovascular		14%

Is my patient septic?

17

- SIRS criteria (1 suspicion)
- Laboratory findings
  - WBC abnormalities
    - Bands!! >2%
    - Toxic Neu
  - Thrombocytopenia
  - Coagulopathies



Confirmation (or high suspicion) of a septic foci

Is my patient in septic shock?

18

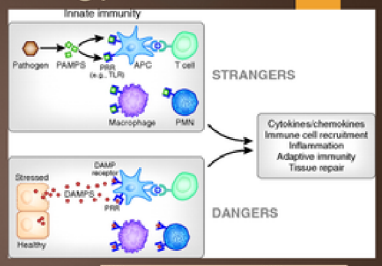
Meets sepsis criteria AND…

- Hyperlactatemia >2 mmol/L
- Hypotension
  - MAP<60-65 mm Hg
  - Doppler BP <90 mmHg
- Dysglycemia?

SIRS-sepsis Pathophysiology

SIRS-sepsis pathophysiology

1. Infectious or non-infectious insult
2. Expression of pathogen-associated molecular patterns (PAMPs) or damage-associated molecular patterns (DAMPS)
3. Stimulation of pathogen recognition receptors (PRRs), toll-like receptors (TLRs) or other systems in immune host cells



DAMPs	PAMPs
ATP	lipopolysaccharide
Uric acid (monosodium urate)	flagellin
High-mobility group box 1	viral double-stranded RNA
Albumin	viral single-stranded RNA
Mitochondrial DNA	lipoteichoic acid
Cytosolic RNA	α-hemolysin
Heat-shock proteins	β-hemolysin
Mitochondrial cardiolipin	
Lysosomal cathepsin	
Azurokin	
Calcium oxalate	

Pathophysiology

5. Proinflammatory mediators circulate & cause:
   1. peripheral vascular dilation, increased capillary permeability, & depressed cardiac function

   3 forms of shock occur simultaneously
6. Shock → MODS → death

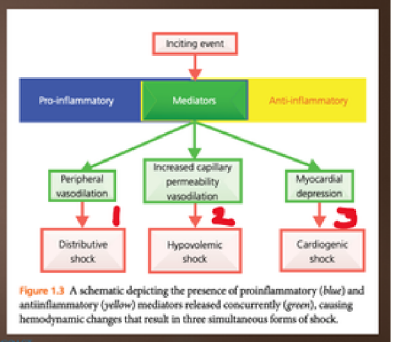


Figure 1.3 A schematic depicting the presence of proinflammatory (*blue*) and antiinflammatory (*yellow*) mediators released concurrently (*green*), causing hemodynamic changes that result in three simultaneous forms of shock.

Key pathophysiologic factors

1. Microbial
   1. PAMPS
2. Host response
   1. Cytokines, exaggerated CARS!
3. Loss of hemostasis
   1. Loss of vasomotor tone
   2. Inflammatory/coagulation dysregulation
   3. Endothelial, microcirculatory & mitochondrial abnormalities

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Approach to the septic/SS patient

Bundle concept

Group of therapies that, together, have better outcomes than individual its components alone

Bundle elements

1. Lactate
2. Source identification (blood , tissue & fluid cultures)
3. Early source control & antimicrobial administration
4. Septic shock treatment (fluids, vasopressors)

Lactate

- Bioproduct of anaerobic metabolism
  - Hypoperfusion
  - ↑ catecholamines
  - Underlying disease
- Measure within 1st hour of admission
- Recheck 2-4 h after volume resuscitation
- Continue resuscitation until normal

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Bundle elements

1. Lactate
2. Source identification (blood , tissue & fluid cultures)
3. Early source control & antimicrobial administration
4. Septic shock treatment (fluids, vasopressors)

Source identification

- Blood cultures: standard of care in humans (30-50% positive)
  - vetmed< 49%
- Promotes antibacterial selection (DEESCALATION!)
- Sampling MUST not delay tx

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Table 2. **Species of bacteria isolated from the blood of 140 dogs and 39 cats with positive blood cultures**

Bacterial isolates	Gram stain	Dogs	Cats
Staphylococcus species	+	26·4% (n=42)	2·5% (n=1)
Streptococcus species	+	13·8% (n=22)	7·5% (n=3)
Enterococcus species	+	5·7% (n=9)	5·0% (n=2)
Micrococcaceae	+	0·6% (n=1)	0·0% (n=0)
Escherichia coli	–	29·6% (n=47)	30·0% (n=12)
Pasteurella species	–	4·4% (n=7)	5·0% (n=2)
Enterobacter species	–	3·8% (n=6)	15·0% (n=6)
Pseudomonas species	–	2·5% (n=4)	7·5% (n=3)
Klebsiella species	–	2·5% (n=4)	2·5% (n=1)
Serratia marcescens	–	1·3% (n=2)	0·0% (n=0)
Campylobacter jejuni	–	0·6% (n=1)	0·0% (n=0)
Salmonella species	–	0·0% (n=0)	7·5% (n=3)
Yersinia pseudotuberculosis	–	0·0% (n=0)	2·5% (n=1)
Anaerobic bacteria	+ or –	8·8% (n=14)	15·0% (n=6)

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JSAP JOURNAL OF SMALL ANIMAL PRACTICE BSAVA

A retrospective study of the clinical presentation of 140 dogs and 39 cats with bacteraemia

M. Greiner, G. Wolf, K. Hartmann

First published: 24 July 2008 | https://doi.org/10.1111/j.1748-5827.2008.00546.x | Citations: 39

GULF COAST VETERINARY SPECIALISTS

Bundle elements

1. Lactate
2. Source identification (blood , tissue & fluid cultures)
3. Early source control & antimicrobial administration
4. Septic shock treatment (fluids, vasopressors)

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Early source control

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Early source control

- Mostly surgical
  - Infectious peritonitis
  - Pyometra
  - Bite wounds
  - Pyothorax
  - Necrotizing fascitis

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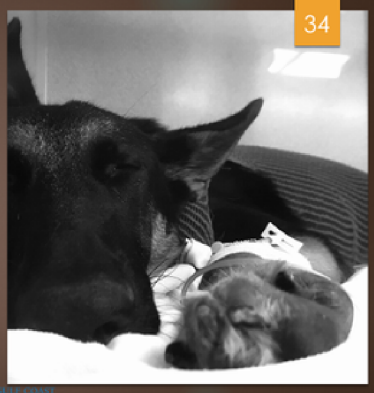
Antibacterial administration

Recommendations SS21

- Low likelihood, defer antibacterials, keep looking
- Possible sepsis, no shock
  - 3h window
- High likelihood or septic shock
  - Within 1 hr



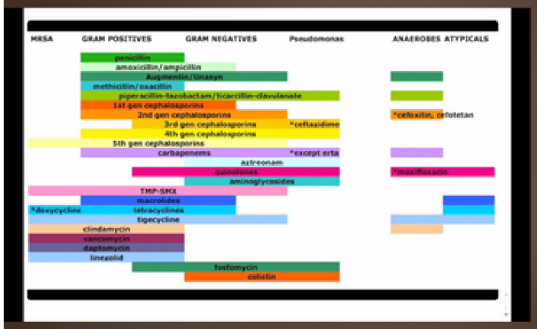
Antimicrobial approach

Sepsis/septic shock	Antibacterial coverage
"Simple"	Broad spectrum, gram neg
Risk MDR+sepsis/septic shock	Double gram neg
Risk MRSA+sepsis/septic shock	MRSA
Fungal	Antifungal
Virus	□□

"Simple" sepsis

- Ampicillin/sulbactam
- Ampicillin/enrofloxacin
- 2nd/3rd cephalosporin
- Trimetoprim/sulfas

37



MDR

- Ampicillin/sulbactam +
  - Enrofloxacin
  - Ceftazadime
  - Amikacin
  - TMS
- Piperacillin/tazobactam

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MRSA

- Carbapenem
- Amikacin/Vancomycin
- Chloramphenicol
- TMS

Optimization of β-lactams

39

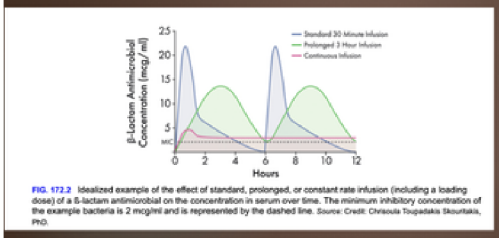

FIG. 172.2 Idealized example of the effect of standard, prolonged, or constant rate infusion (including a loading dose) of a ß-lactam antimicrobial on the concentration in serum over time. The minimum inhibitory concentration of the example bacteria is 2 mcg/ml and is represented by the dashed line. Source: Credit: Chrisoula Tsugadakis Skountakis, PhD.

- β-lactams can be used for all septic groups, except MRSA
- Time-dependent
- Standard infusion, extended infusion or CRI
- CONS:
  - Technically challenging vetmed
  - Augmented renal clearance
  - Lack of studies

Bundle elements

1. Lactate
2. Source identification (blood , tissue & fluid cultures)
3. Early source control & antimicrobial administration
4. Septic shock treatment (fluids, vasopressors)

Fluid therapy

- 3 shock types, only ONE responds to fluid therapy

41

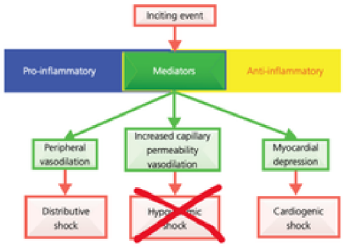

Figure 1.3 A schematic depicting the presence of proinflammatory (*blue*) and antiinflammatory (*yellow*) mediators released concurrently (*green*), causing hemodynamic changes that result in three simultaneous forms of shock.

Initial recommendations

42

1. Bolus (at least) 30 ml/kg balanced crystalloids in first 3h- SS21
   - 10-20 "rule"
   - 10-20 ml/kg over 10-20 min
2. Calculate deficit + maintenance & administer
3. Fluid responsiveness assessment
4. Bolus #2
   2. Start

Is there evidence of vasodilatory or cardiogenic shock?

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Vasopressor therapy

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- Early use improves outcome
- Norepinephrine (NE) 1st, Vasopressin (AVP) 2nd
- NE starting dose depends on SS severity
  - Early, NE 0.05 mcg/kg/min
  - High suspicion, non-critical 0.1 mcg/kg/min
  - Severe SS 0.3-0.5 mcg/kg/min
- Max dose: 1 mcg/kg/min, then add 2nd

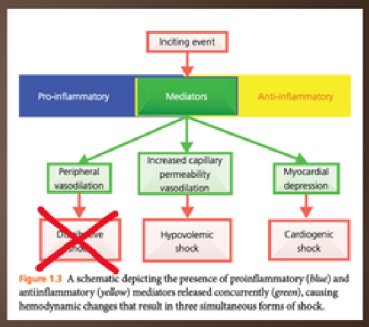

Figure 1.3 A schematic depicting the presence of proinflammatory (*blue*) and antiinflammatory (*yellow*) mediators released concurrently (*green*), causing hemodynamic changes that result in three simultaneous forms of shock.

GULF COAST VETERINARY SPECIALISTS

Vasopressor therapy

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- Higher dose have multiple detrimental effects
  - ↑ myocardial O2 demand
  - ↓ CO
  - ↓ regional blood flow
    - Renal!!!
  - Immunosupression

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Vasopressor therapy

45

Arginine Vasopressin (ADH)

- Vasopressin (AVP) 1-4 mU/kg/min
- AVP is degraded/depleted during sepsis/ss
- Faster SS resolution vs monotherapy
- Reduced catecholamine dose, & easier wean-off

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Dobutamine

Why?

Septic cardiomyopathy

- Cytokines
- LPS
- NO
- Complement

- Synthetic dopamine analog
- Increase forward cardiac flow

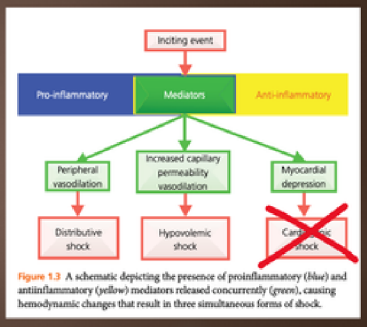

Figure 1.3 A schematic depicting the presence of proinflammatory (*blue*) and antiinflammatory (*yellow*) mediators released concurrently (*green*), causing hemodynamic changes that result in three simultaneous forms of shock.

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Key points

- Sepsis/SS: high morbidity/mortality
- Sepsis can be challenging to diagnose
- Act early , don't wait
- Antibacterial selection is important
- There are 2-3 shock types, not all is solved with IV crystalloids
- Septic/SS patients requires ICU specialized care
- Initial interventions can be life saving

GULF COAST VETERINARY SPECIALISTS

Thank you!

GULF COAST VETERINARY SPECIALISTS

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- Sepsis main cause of neonatal death in 1st 3wk age
- Mortality rate 25.6%
  - Early mortality (0–2 d) 69%
- Morbidity & mortality of neonatal septic dogs are high

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- 78% dogs had 1+ dysfunctional organ systems
- 50% dogs had MODS
- Mortality increased w/ number of dysfunctional organ systems
- Mortality 70% for dogs in MODS, 25% without.

SURGERY

KRISTIN COLEMAN

DVM, MS, DACVS

Dr. Coleman graduated from Auburn University in 2008, earning her Bachelor of Science degree in Microbiology with the Biomedical Sciences Dean's Medal. Remaining in the Southeast, she continued her education and received her Doctor of Veterinary Medicine degree from Auburn University in 2012, earning the Small Animal Surgery proficiency award.

Afterward, she completed a general rotating medicine and surgery internship at the Animal Medical Center in Manhattan in 2013. This was followed by a combined Small Animal Surgery residency and Master of Science degree at Colorado State University Veterinary Teaching Hospital in Fort Collins, Colorado.

In 2016, Dr. Coleman embarked on her career journey by joining a bustling private practice in Brooklyn, New York, and within months, achieved board certification from the American College of Veterinary Surgeons.

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713-693-1111

SURGICAL TREATMENT OF BRACHYCEPHALIC OBSTRUCTIVE AIRWAY SYNDROME

KRISTIN COLEMAN, DVM, MS, DACVS
GULF COAST VETERINARY SPECIALISTS (GCVS), HOUSTON, TX
GCVS SYMPOSIUM – AUGUST 2023

GULF COAST VETERINARY SPECIALISTS

1

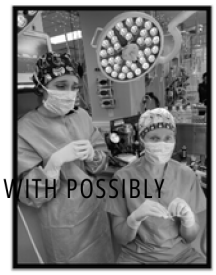
CONFLICT OF INTEREST DISCLOSURE

I HAVE NO RELEVANT FINANCIAL INTEREST, ARRANGEMENT OR AFFILIATION WITH ANY COMPANY OR ORGANIZATION.

2

GOAL OF TODAY

- BECOME COMFORTABLE WITH BOAS ANATOMY AND WHAT CAN BE DONE TO HELP THESE ANIMALS BREATHE
- EITHER BECOME COMFORTABLE WITH POSSIBLY PERFORMING THESE PROCEDURES OR DISCUSSING WITH CLIENTS BEFORE REFERRING



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BOAS

- ENGLISH BULLDOG
- FRENCH BULLDOG
- PUG
- BOSTON TERRIER
- (SOME) SHIH TZU
- (SOME) CAVALIER KING CHARLES SPANIEL

- CLINICAL SIGNS
- UPPER AIRWAY OBSTRUCTION
- EXERCISE INTOLERANCE
- COLLAPSE
- CYANOSIS
- +/- GASTRO-ESOPHAGEAL REFLUX
- +/- SLIDING HIATAL HERNIA

5

OUTLINE

- ANATOMY OF THE AIRWAY
  PRIMARY & SECONDARY COMPONENTS
- PATHOPHYSIOLOGY
- COMPONENTS OF BOAS
- SURGICAL INTERVENTION OF SOME COMPONENTS
- PRE- & POST-OP CONSIDERATIONS
- WHEN TO TRACH & OPTIONS

6

ANATOMY OF THE AIRWAY

- CONCHAL GROWTH
- NASOPHARYNGEAL COMPRESSION
- ABERRANT NASAL TURBINATES
- SOFT PALATE HYPERPLASIA
- MACROGLOSSIA

PRIMARY COMPONENTS:

- STENOTIC NARES, ELONGATED SOFT PALATE, HYPOPLASTIC TRACHEA

- SECONDARY COMPONENTS:
- EVERTED LARYNGEAL SACCULES OR TONSILS, TONSILLAR ENLARGEMENT, LARYNGEAL COLLAPSE AND EDEMA

7

PATHOPHYSIOLOGY OF BOAS

- NARES CAN COLLAPSE ON INSPIRATION
- ELONGATED SOFT PALATE CAN PROTRUDE INTO THE RIMA GLOTTIS
- INTERFERES WITH LAMINATE AIRFLOW
- REDUCED AIRWAY DIAMETER
- INCREASED UPPER AIRWAY RESISTANCE
- GREATER NEGATIVE INTRATHORACIC PRESSURE TO COMPENSATE FOR ADEQUATE FLOW

8

PATHOPHYSIOLOGY OF BOAS

- INCREASED WORK OF BREATHING WITH DYNAMIC PRESSURE CHANGE
- FURTHER EXACERBATION OF COLLAPSIBLE AIRWAY PASSAGES
- FURTHER INCREASE IN AIRWAY RESISTANCE
- CAN LEAD TO INFLAMMATION AND EDEMA OF THE PHARYNGEAL TISSUES

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COMPONENTS OF BOAS

- HYPOPLASTIC TRACHEA
- STENOTIC NARES
- ELONGATED SOFT PALATE
- EVERTED LARYNGEAL SACCULES
- REDUNDANT NASOPHARYNGEAL TURBINATES / ABNORMAL CONCHAE
- ENLARGED TONGUE BASE
- EVERTED OR ENLARGED TONSILS LARYNGEAL COLLAPSE

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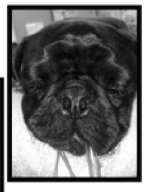
PRE-OPERATIVE EVALUATION

- ENSURE OWNERS ARE INFORMED OF RISKS ASSOCIATED WITH BOAS SURGERY:
  - TEMPORARY TRACHEOSTOMY TUBE
  - ASPIRATION PNEUMONIA
  - CONTINUED GI SIGNS



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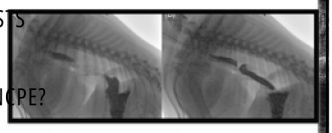
PRE-OPERATIVE EVALUATION

- PRE-OPERATIVE DIAGNOSTIC TESTS
- PHYSICAL EXAMINATION
- THORACIC RADIOGRAPHS
- PNEUMONIA? HIATAL HERNIA? NCPE?
- TRACHEAL SIZE
- BLOODWORK
- +/- VIDEOFLUOROSCOPIC CONTRAST SWALLOW STUDY
- +/- HEAD CT SCAN



Journal of Veterinary Internal Medicine

Retrospective analysis of esophageal imaging features in brachycephalic versus non-brachycephalic dogs based on videofluoroscopic swallowing studies

Caroline Elvers, Rocio Chicon Rueda, Tiziana Liuti, Silke Salavati Schmitz

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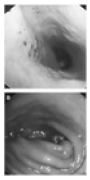
PRE-OPERATIVE CONSIDERATIONS

- 97% OF BOAS DOGS HAVE GI LESIONS
- ENDOSCOPY + BIOPSIES OF STOMACH & DUODENUM
  - SOME DO NOT EVEN HAVE CLINICAL SIGNS OF GI ISSUES!
- 80% OF DOGS HAD IMPROVEMENT IN GI SIGNS POST-OP
- AFTER UPPER AIRWAY SURGERY PROKINETICS AND ACID SUPPRESSION RECOMMENDED
  - PANTOPRAZOLE
  - METOCLOPRAMIDE
- SEVERITY OF AIRWAY DISEASE CORRELATED WITH SEVERITY OF DIGESTIVE SIGNS
- SIGNIFICANT IN FRENCHIES, MALES, AND HEAVY DOGS



JSAP BSAVA

Prevalence of gastrointestinal tract lesions in 73 brachycephalic dogs with upper respiratory syndrome

Poncet, JSAP, 2005

13

PRE-ANESTHETIC PLANNING

RISK OF ANESTHESIA-RELATED COMPLICATIONS IN BRACHYCEPHALIC BREEDS

JAVMA 2018 - GRUENHEID M, AARNES TK, MCLOUGHLIN MA, ET AL.

- AROUND 50% PERI-ANESTHETIC COMPLICATIONS FOR BOTH GROUPS
- POST-ANESTHETIC COMPLICATIONS: 8.7% BAS, 3.6% NON-BAS
- 1.57X MORE LIKELY TO EXPERIENCE AN INTRAOPERATIVE COMPLICATION
  - RISK INCREASED WITH EVERY 30 MINUTE INCREASE OF ISOFLURANE ANESTHESIA
- 4.33X MORE LIKELY TO HAVE A POST-OPERATIVE COMPLICATION
  - RISK INCREASED WITH EACH 30 MINUTE INCREASE OF ISOFLURANE ANESTHESIA

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PRE-ANESTHETIC PLANNING

RISK OF ANESTHESIA-RELATED COMPLICATIONS IN BRACHYCEPHALIC BREEDS

- ORTHOPEDIC CASES APPEARED SAFER COMPARED TO SOFT TISSUE CASES
- INVASIVE PROCEDURE WERE 5X AS LIKELY TO CAUSE POST-OP COMPLICATIONS VS NON-INVASIVE
- MOST COMMON POST-OP COMPLICATION WAS ASPIRATION PNEUMONIA
- KETAMINE AND BENZODIAZEPINE LEAD TO MORE POST-ANESTHETIC COMPLICATIONS THAN IF JUST PROPOFOL WAS USED

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PRE-ANESTHETIC PLANNING

ANESTHETIC RISK DURING SUBSEQUENT ANESTHETIC EVENTS IN BRACHYCEPHALIC DOGS THAT HAVE UNDERGONE CORRECTIVE AIRWAY SURGERY: 45 CASES (2007-2019)

JAVMA 2020

DOYLE CR, AARNES TK, BALLASH GA, ET AL.

- ODDS OF COMPLICATIONS DURING POST-ANESTHETIC PERIOD FOLLOWING SUBSEQUENT ANESTHETIC EVENTS DECREASED BY 79% IN DOGS THAT HAD PREVIOUS AIRWAY SX
- INTRA-ANESTHETIC BRADYCARDIA INCREASED ODDS OF DEVELOPING POST-ANESTHETIC COMPLICATION BY 4.5X
- EVERY 15-MINUTE INCREASE IN ANESTHETIC DURATION INCREASED ODDS OF POST-ANESTHETIC COMPLICATION BY 12% & INTRA-ANESTHETIC COMPLICATION BY 11%

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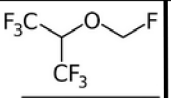
PRE-ANESTHETIC PLANNING

- PREOXYGENATION
  - •INCREASE OXYGEN STORES VIA FUNCTIONAL RESIDUAL CAPACITY
- RAPID INTUBATION
- RAPID SMOOTH RECOVERY à AVOID LONG-ACTING DRUGS
- USE INHALANTS WITH FASTER RECOVERY TIME
  - •SEVOFLURANE AND DESFLURANE
  - •REMEMBER ALL INHALANTS PRODUCE A DOSE-DEPENDENT REDUCTION OFàINOTROPY, SYSTEMIC VASCULAR RESISTANCE, AND PRE-LOAD



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PRE-OPERATIVE PREPARATION

- NO FOOD FOR 24 HRS PRIOR TO SURGERY
- LIGHT PLANE OF ANESTHESIA + FLOW-BY O2
  - •0.3 MG/KG METOCLOPRAMIDE SC
  - •1 MG/KG PANTOPRAZOLE
  - •1 MG/KG MAROPITANT
- PROPOFOL TO EFFECT
- SEDATED OROPHARYNGEAL / LARYNGEAL EXAMS
- DOXAPRAM 1 MG/KG IV BOLUS

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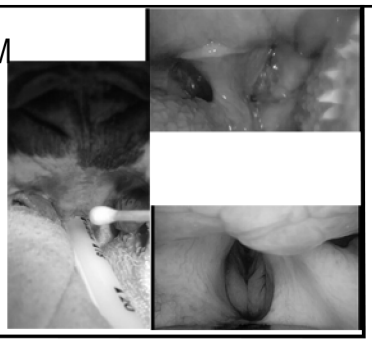
LARYNGEAL EXAM

- SOFT PALATE LENGTH &
- THICKNESS SACCULES & TONSILS
- LARYNGEAL COLLAPSE
- LARYNGEAL FUNCTION
- NASOPHARYNX
- TONGUE
- OTHER MASSES OR LESIONS



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Oral Prep (0.12% Chlorhexidine Diacetate)
(Modified after CET Oral Hygiene Rinse)
940 mls Tap Water & 60 mls of Nolvasan

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COMPONENTS OF BOAS

- HYPOPLASTIC TRACHEA
- STENOTIC NARES
- ELONGATED SOFT PALATE
- EVERTED LARYNGEAL SACCULES
- REDUNDANT NASOPHARYNGEAL TURBINATES / ABNORMAL CONCHAE
- ENLARGED TONGUE BASE
- EVERTED OR ENLARGED TONSILS
- LARYNGEAL COLLAPSE

22

- Hypoplastic
  Ratio <0.16

- Normal trachea
  Ratio >0.20
- Most common dog breed to experience this:
  English Bulldog

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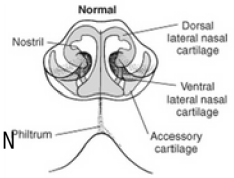
SURGERY: STENOTIC NARES

- TRADER'S TECHNIQUE
- PUNCH TECHNIQUE
- AL APEXY
- VERTICAL ALAR WEDGE RESECTION
  - HORIZONTAL WEDGE RESECTION



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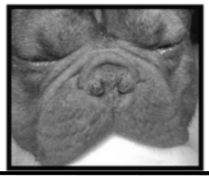
TRADER'S TECHNIQUE

- ORIGINALLY REPORTED IN SHIH TZU PUPPIES
- NEED PRESSURE OR TOPICAL EPINEPHRINE TO CONTROL BLEEDING



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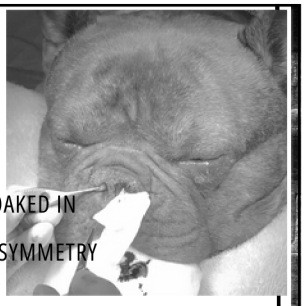
TRADER'S TECHNIQUE

#11 BLADE POINTED VENTROLATERALLY

AMPUTATE VENTRAL ALAR WING

- APPLY PRESSURE WITH DRY GAUZE STRIP
  - REPLACE WITH GAUZE STRIP SOAKED IN DILUTE EPINEPHRINE
  - TRIM ADDITIONAL TISSUE FOR SYMMETRY



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PUNCH TECHNIQUE

- 2MM – 6MM PUNCH
- GRASP PLUG OF TISSUE WITH FORCEPS
- EXCISE BASE WITH METZENBAUM SCISSORS
- GOAL IS TO LEAVE 2-3MM RIM OF TISSUE FOR SUTURING

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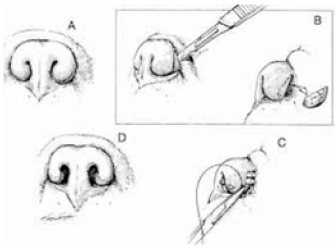
ALAPEXY

- TWO SMALL ELLIPTICAL INCISIONS
  - ABDUCT ALAE
- MAY BE MORE DIFFICULT THAN OTHER WEDGE RESECTIONS
- OPTION IF OTHER TECHNIQUES HAVE FAILED



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VERTICAL ALAR WEDGE RESECTION

- OTHER OPTION: HORIZONTAL WEDGE
- MAKE SURE INCISION IS DEEP TO LEVEL OF NASAL CONCHA
- FIRST SUTURE APPOSES ROSTROVENTRAL MARGIN

Tobias, Manual of Soft Tissue Surgery

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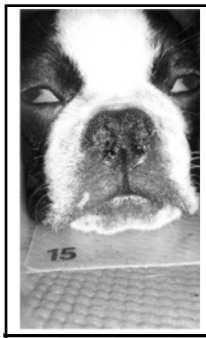
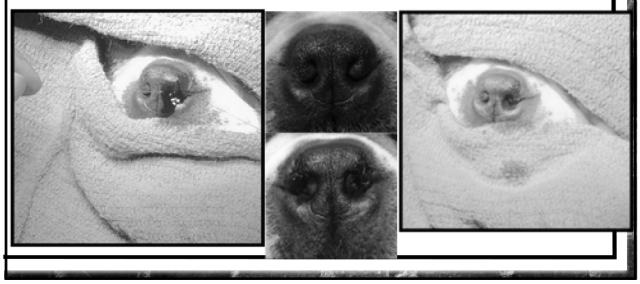


WHAT CAN GO WRONG?

- INFECTION!
- HINT: DON'T USE CATGUT SUTURE ON THE NARES.

30

VERTICAL ALAR WEDGE RESECTION



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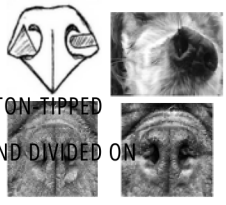
VERTICAL ALAR WEDGE RESECTION

- LEAVING A TAG AND HOLDING YOUR FIRST SUTURE DORSALLY AS STAY SUTURE ALLOWS INTRA-NASAL SUTURING
- +/- TOPICAL EPINEPHRINE ON COTTON-TIPPED APPLICATOR FOR HEMORRHAGE
- 0.1ML DILUTED 1:1 WITH SALINE AND DIVIDED ON 2 Q-TIPS FOR BOTH NOSTRILS



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ALAR FOLD VESTIBULOPLASTY / MODIFIED TRADER'S TECHNIQUE

- FIRMLY GRASP ALAR FOLD WITH A CURVED HEMOSTAT, #11 BLADE USED TO SHARPLY INCISE AT BASE TO RESECT
- MODIFIED TRADER'S IS PERFORMED TO EXCISE DORSAL WALL OF ALAR FOLD TO LEVEL OF NASAL VESTIBULE
- HEMOSTASIS VIA PRESSURE WITH CTA + PHENYLEPHRINE-SOAKED SPONGES

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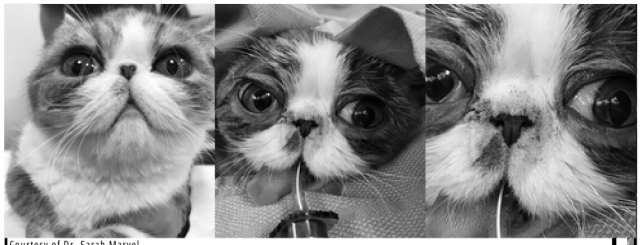

Courtesy of Dr. Sarah Marvel

VERTICAL WEDGE RESECTION IN A CAT

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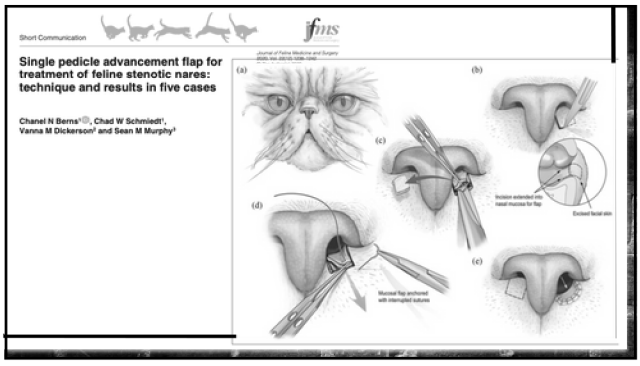

Short Communication

jfms

Journal of Feline Medicine and Surgery

Single pedicle advancement flap for treatment of feline stenotic nares: technique and results in five cases

Chanel N Berns[1], Chad W Schmiedt[1], Vanna M Dickerson[2] and Sean M Murphy[3]

35

SURGERY: ELONGATED SOFT PALATE

- "STAPHYLECTOMY"
- SOFT PALATE EXTENDS PAST CAUDAL ASPECT OF TONSILS
- MAY ALSO BE ABNORMALLY THICK
- EVIDENCE THAT SOFT PALATE MAY HAVE DENERVATION IN DOGS WITH BOAS

Tobias, Manual of soft tissue surgery

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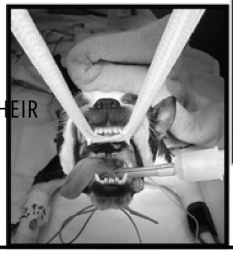
STAPHYLECTOMY: OPTIONS

- CUT-AND-SEW
- *MY FAVORITE*
- L ASER
- BIPOLAR ELECTROSURGERY
- • LIGASURE™
- FOLDED FLAP PALATOPLASTY
  - *GREAT FOR THOSE MEATY PALATES!*

37

STAPHYLECTOMY

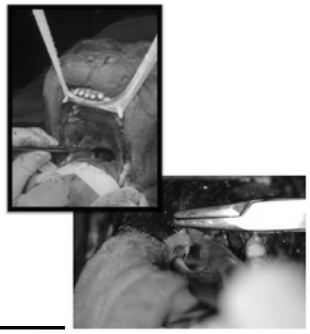
- STERNAL RECUMBENCY
- ENSURE STRAIGHTNESS
- ELEVATE HEAD
  - BE MINDFUL OF THEIR NECK!
- TIE ET TUBE TO MANDIBLE
- HEAD LAMP



38

STAPHYLECTOMY

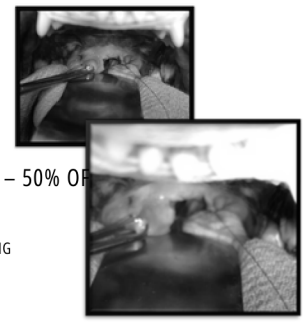
- PLACE STAY SUTURES AT PLANNED RESECTION SITE
- ALWAYS VISUALIZE ORAL AND NASOPHARYNGEAL MUCOSA
- TAPING TONGUE OUT OF MOUTH AND DOWN OR USE MALLEABLE RETRACTOR



39

STAPHYLECTOMY

- ONLY CUT 50% OF SOFT PALATE AT A TIME
- OR, CUT IT ALL AT ONCE!
- DISTANCE TO CUT:
- NO MORE CRANIAL THAN 1/3 – 50% OF LENGTH OF TONSILLAR CRYPT
- ALWAYS VISUALIZE ORAL AND NASOPHARYNGEAL MUCOSA WHILE SUTURING



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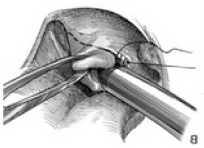
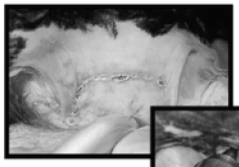


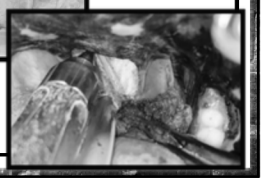
Tobias & Johnston
Small Animal Veterinary Surgery 2011

41

STAPHYLECTOMY – CO2 LASER

- PRODUCES FOCUSED INFRARED LIGHT TO ABLATE AND VAPORIZE THE TISSUES
- WAVELENGTH: 10,600 NM
- HIGHLY ABSORBED BY WATER





42

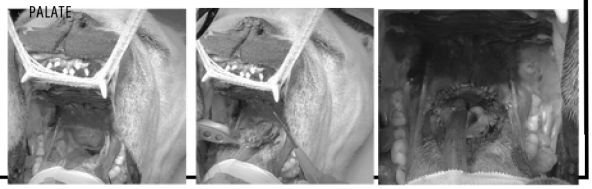
STAPHYLECTOMY – BIPOLAR VSD

- USES ELECTROTHERMAL ENERGY TO DENATURE COLLAGEN AND ELASTIN IN BLOOD VESSEL WALLS AND SEAL VESSELS
- SAME STEPS AS CUT-AND-SEW UNTIL THE CUT AND SEW PORTIONS…

43

FOLDED FLAP PALATOPLASTY

- DESCRIBED IN 2007 BY 2 FRENCH SURGEONS
- (DUPRE) PURPOSE: TO SHORTEN AND THIN THE PALATE



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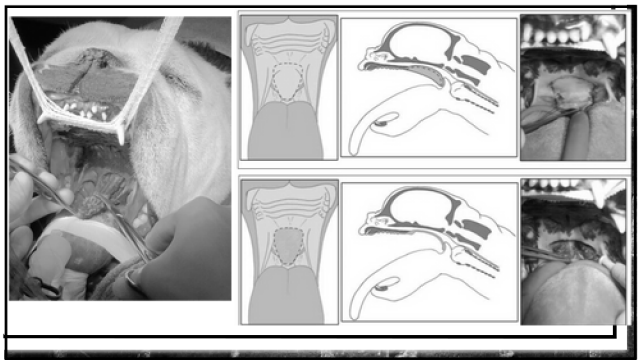

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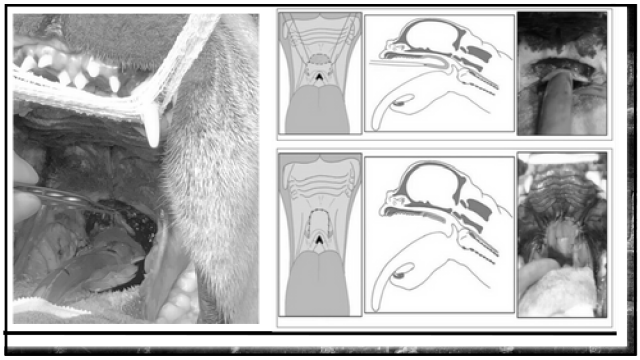
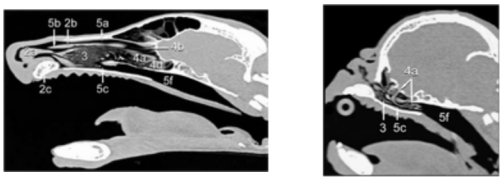


47

NASOPHARYNGEAL TURBINATES

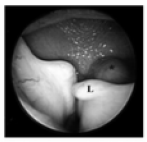
T. H. Oechtering et al.: CT imaging of the nose in brachycephalic dog breeds 2007



48

ODDITY: NORWICH TERRIERS

- ENLARGED SUPRAARYTENOID
- FOLDS NOT SURGICAL
- CAN FIX OTHER BOAS PROBLEMS



Johnson, JVIM, 2013

50

EVERTED TONSILS

- TO REMOVE OR NOT TO REMOVE?
- MILDLY EVERTED + SOFT?
  - PROBABLY LEAVE.
- MARKEDLY EVERTED + FIRM?
  - TAKE THEM OUT!

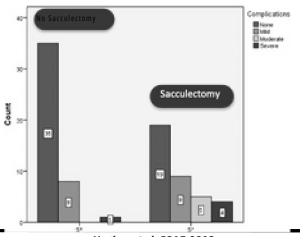
51

EVERTED LARYNGEAL SACCULES

- WHITE SHINY DOME STRUCTURES
- SOFT?
  - LEAVE THEM ALONE.
- FIRM?
  - CONSIDER REMOVING.

52

EVERTED LARYNGEAL SACCULES



Hughes et al. JSAP 2018

Fossum et al. 2012

Complications:

None

Mild: <48 h regurg, mild stertor or stridor w/o dyspnea

Moderate: >48 h regurg, mild cough, episode of spontaneously resolving dyspnea

Severe: dyspnea, regurgitation requiring tx, temp trach, tx for pneumonia, ventilator, euthanasia, death

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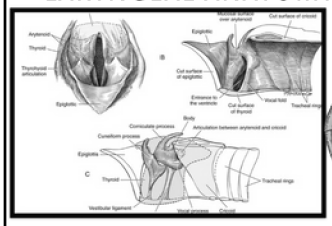
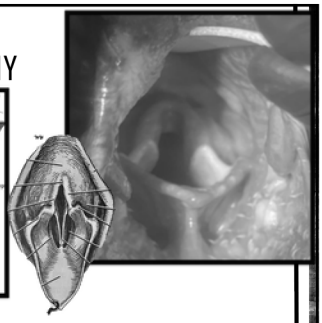
LARYNGEAL ANATOMY





Tobias and Johnston, Veterinary Surgery Small Animal

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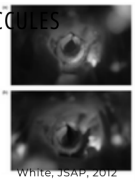
LARYNGEAL COLLAPSE

- STAGE 1
  - •EVERTED LARYNGEAL SACCULES
- STAGE 2
  - •MEDIAL DISPLACEMENT CUNEIFORM
- STAGE 3
  - •MEDIAL DISPLACEMENT CORNICUL ATE



White, JSAP, 2012

56

POST-OPERATIVE CARE

- HEAD-UP WITH CONTINUED MONITORING
- STERNAL POSITION
- ADDITIONAL INDUCTION AGENTS AND TRACHEAL TUBES
- PREPARE FOR A TEMPORARY TRACHEOSTOMY
- COOL ENVIRONMENT
- MINIMIZE RESTRAINT
- PLAN SURGERY AND SEDATION IN THE AM
- HOSPITALIZE FOR "RESPIRATORY WATCH" OVERNIGHT

62

POST-OPERATIVE CARE

- SINGLE DOSE OF ANTI-INFLAMMATORY STEROID à 0.15MG/KG DEX SP
- SEDATION AS NEEDED
  - •DEXMED CRI 0.5 – 2 UG/KG/HR
- OVERNIGHT MONITORING RECOMMENDED
- MAJOR COMPLICATION RATE ~7%
  - •ASPIRATION PNEUMONIA ASSOCIATED WITH MAJOR COMPLICATIONS

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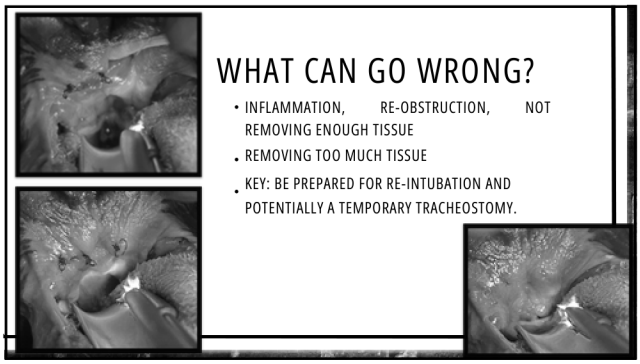
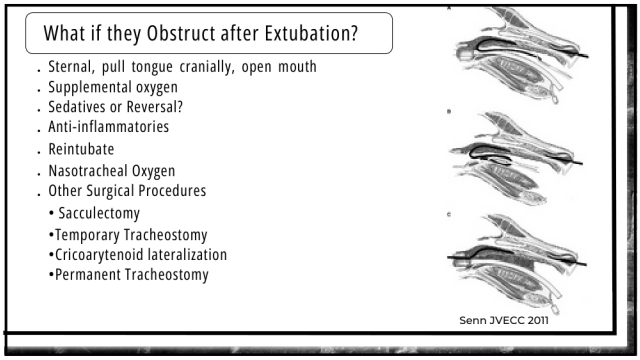
SURGERY: POTENTIAL COMPLICATIONS

- •RESPIRATORY DISTRESS / OBSTRUCTION IN THE IMMEDIATE POST-OP PERIOD
  - • RE-INTUBATE.
  - •WHAT TO DO IF DOG CANNOT BE EXTUBATED?! TEMP TRACH.
- RESPIRATORY
  - •CONTINUED STERTOR/STRIDOR
  - •UPPER AIRWAY OBSTRUCTION
  - •ASPIRATION PNEUMONIA
  - • RHINITIS
- GASTROINTESTINAL
  - • REGURGITATION
  - • VOMITING
- INCISIONAL COMPLICATIONS
- DEATH USUALLY <5%

66


WHAT CAN GO WRONG?
• INFLAMMATION, RE-OBSTRUCTION, NOT REMOVING ENOUGH TISSUE
• REMOVING TOO MUCH TISSUE
• KEY: BE PREPARED FOR RE-INTUBATION AND POTENTIALLY A TEMPORARY TRACHEOSTOMY.

67


What if they Obstruct after Extubation?
• Sternal, pull tongue cranially, open mouth
• Supplemental oxygen
• Sedatives or Reversal?
• Anti-inflammatories
• Reintubate
• Nasotracheal Oxygen
• Other Surgical Procedures
• Sacculectomy
•Temporary Tracheostomy
•Cricoarytenoid lateralization
•Permanent Tracheostomy
Senn JVECC 2011

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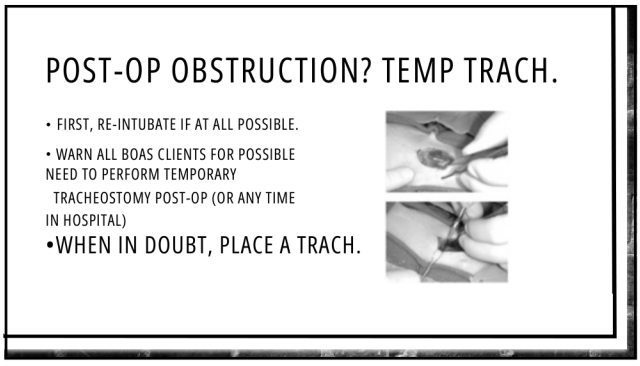
POST-OP OBSTRUCTION? TEMP TRACH.
• FIRST, RE-INTUBATE IF AT ALL POSSIBLE.
• WARN ALL BOAS CLIENTS FOR POSSIBLE NEED TO PERFORM TEMPORARY TRACHEOSTOMY POST-OP (OR ANY TIME IN HOSPITAL)
•WHEN IN DOUBT, PLACE A TRACH.

69

SURGERY: POTENTIAL COMPLICATIONS

- ALWAYS BE READY TO RE-INTUBATE
- READY FOR A TEMPORARY TRACHEOSTOMY

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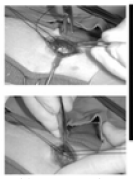
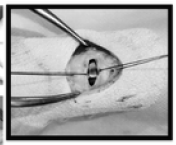
TEMPORARY TRACHEOSTOMY

- INCISE BETWEEN TRACHEAL RINGS 3-5 ON THE VENTRAL SURFACE
- PLACE STAY SUTURES AND MARK "UP" AND "DOWN"
- DON'T INFLATE TRACH TUBE CUFF UNLESS VENTILATING





Tobias, Manual of soft tissue surgery

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TRACHEOSTOMY TUBES

- INNER CANNULA ALLOWS FOR LESS STRESSFUL CHANGES
- SMALLEST SIZES DO NOT COME WITH THIS CANNULA

72

TRACHEOSTOMY TUBES - HOMEMADE

- REMOVE END THAT ATTACHES TO
- OXYGEN CUT THE PLASTIC TO CREATE
- FLAPS
- REPLACE CAP AT DESIRED LENGTH

PLACE HOLES IN FLAPS TO ATTACH UMBILICAL TAPE TO TIE TO NECK OF ANIMAL

73

LARYNGEAL COLLAPSE

- SURGICAL INTERVENTION
  - MODIFIED TIE-BACK
  - TEMPORARY TRACHEOSTOMY

74

NASOTRACHEAL INTUBATION

75

76

77

WHAT IF THEY DON'T IMPROVE AFTER SURGERY?

- REASSESS AND TREAT THE TREATABLE
- THINGS SEVERE LARYNGEAL COLLAPSE
- ABERRANT NASAL TURBINATES
- SLEEP APNEA
- WEIGHT LOSS
- THICKENED SOFT PALATE

83

SUMMARY

- TREAT EARLY ~~PREVENT~~/MINIMIZE LARYNGEAL COLLAPSE TREAT
- THE TREATABLE PROBLEMS
- KEEP IT SIMPLE
- GRADE 3 LARYNGEAL COLLAPSE—GUARDED PROGNOSIS

84

84

THANK YOU!

~~T~~HANK ALL OF YOU!

- THANK YOU TO THE CLIENTS, PATIENTS, AND WONDERFUL TECHNICIANS!

****

•SURGICAL DESCRIPTIONS AND TECHNIQUES AVAILABLE IN:

TECHNIQUES IN SMALL ANIMAL SOFT TISSUE, ORTHOPEDIC, AND OPHTHALMIC SURGERY

TEXTBOOK COMING SOON IN 2024

85

QUESTIONS?

MIEL

LILLIPUTIAN&LUDWIG

KELEV

86

OPHTHALMOLOGY

ROBIN SANKEY

DVM, DACVO

Dr. Robin Sankey is a board-certified veterinary ophthalmologist. She earned her doctor of veterinary medicine degree from Colorado State University in 2001. Dr. Sankey owned and managed a small animal practice in New Mexico for 13 years. In 2014, she was chosen for a coveted residency position in Comparative Veterinary Ophthalmology at the Animal Ophthalmology Clinic in Dallas.

While Dr. Sankey is proficient in all aspects of ophthalmology, her clinical focus revolves around ocular manifestations of systemic diseases. Beyond her professional dedication, she finds joy in hiking, exploration with her husband and children, engaging in community service projects, gardening, and indulging in crafting during her free time.

ROBIN.SANKEY@GCVS.COM
713-693-1111

A Practical Approach to Corneal Ulcers and Treatment

Robin Sankey, DVM, DACVO

GULF COAST VETERINARY SPECIALISTS

OUTLINE

- Corneal Anatomy and Properties
- Corneal Ulcers
  - Symptoms
  - Depths and Types
  - Medical Therapy
  - When to Debride
- Indolent Ulcers
- Cases

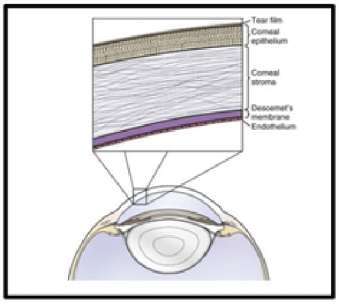
GULF COAST VETERINARY SPECIALISTS

Corneal Properties and Anatomy

- The cornea is the transparent anterior portion of the fibrous tunic of the globe
- 4-5 cell layers thick
- Corneal thickness varies among species
- CCT in dogs and cats ~0.5- 0.6 mm

GULF COAST VETERINARY SPECIALISTS

Corneal Anatomy



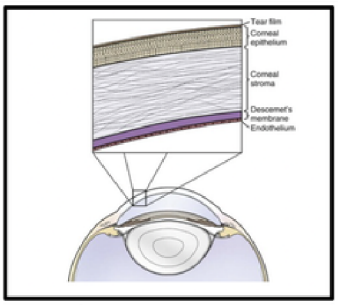
- **Corneal Epithelium:**
  - Nonkeratinized, stratified squamous epithelium
  - 25-40 micrometers thick (dogs and cats)
- **+/- Bowman's Layer:**
  - Humans and Non-human Primates
- **Corneal Stroma:**
  - Makes up 90% of corneal thickness
  - Made up of transparent lamellae of collagenous tissue
  - The precise arrangement is important for maintaining corneal clarity

(Source: Veterinary Ophthalmology 6th ed)

Source: https://veteriankey.com/corneal-ulcers/

GULF COAST VETERINARY SPECIALISTS

Corneal Anatomy



- **Descemet's Membrane**
  - **Basement membrane for the endothelium**
- **Endothelium**
  - **A single layer of cells lining the inner cornea**
  - **Loss leads to corneal edema, up to 500x thicker (corneal endothelial degeneration)**

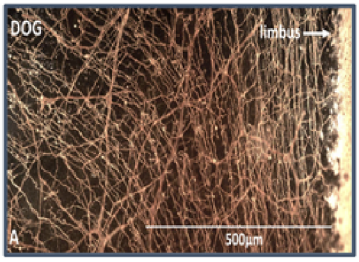
(Source: Veterinary Ophthalmology 6th ed)

Source: https://veteriankey.com/corneal-ulcers/

GULF COAST VETERINARY SPECIALISTS

Corneal Properties and Anatomy

- **Richly supplied by nerves**
- **Innervated by the long ciliary nerve, which is derived from the ophthalmic branch of the trigeminal nerve**
- **Superficial layers are innervated primarily by pain receptors, while the stromal layer is primarily innervated by pressure receptors**
- **Nutrition supplied by the tear film and aqueous humor**
- **Protection supplied by the eyelids and nictitating membrane**



GULF COAST VETERINARY SPECIALISTS

What Makes the Cornea Transparent?

- Lack of blood vessels
- Nonkeratinized surface epithelium maintained by the pre-corneal tear film
- Lack of pigmentation (melanin)
- Relative dehydration (deturgescence)
- Size and organization of stromal collagen fibrils
- The goal is to promote healing in a way that will allow the cornea to return to as close to transparent as possible

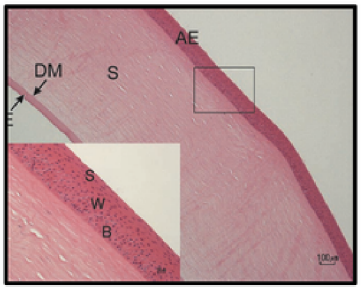
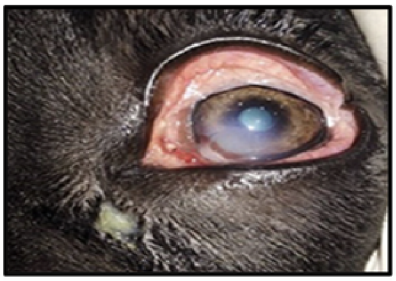


Image and info from Gellatt's Veterinary Ophthalmology, 6th ed

GULF COAST VETERINARY SPECIALISTS

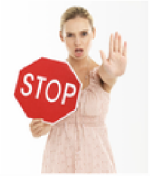
Signs of Corneal Ulceration

- Blepharospasm (Squinting)
- Conjunctival hyperemia
- Discharge
- Corneal vascularization (4-7 day lag period)
- Corneal edema
- +/- Hypopyon



GULF COAST VETERINARY SPECIALISTS

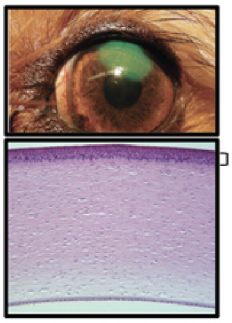
https://doi.org/10.3389/fvets.2021.641265



Depth Matters

GULF COAST VETERINARY SPECIALISTS

Superficial Ulcers



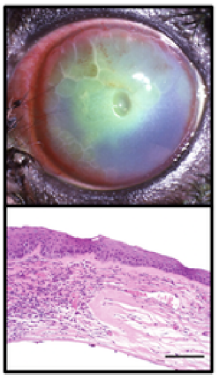
- Fluorescein adheres only to the ulcer floor
- Distinct edges are visible
- Epithelium migrates at ~1 mm/day from both sides of the ulcer bed
- Repithelialization occurs in 4-7 days (mitosis)
- Longer for full strength formation of basement membrane and stromal attachment to occur

(Slatters Fundamental of Veterinary Opthalmology and Gelatt's Veterinary Opthalmology, 5th ed)

GULF COAST VETERINARY SPECIALISTS

Stromal Ulcers

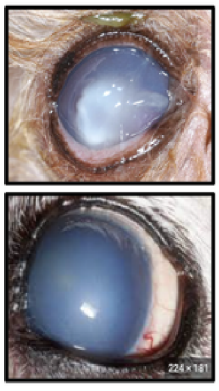
- Fluorescein retention along floor and walls of the ulcer with deeper ulcers
- Fluorescein may diffuse into surrounding stroma (less defined edges) esp. if infection is present
- Deep corneal defects heal by fibrovascular granulation tissue and non-vascular keratocytes
- Takes weeks, sometimes years to heal
- May epithelialize before the stroma fills in (facet)
- More scarring due to irregular arrangement of new stromal tissue (lack of organized collagen)



GULF COAST VETERINARY SPECIALISTS

Infected "Melting" Ulcers

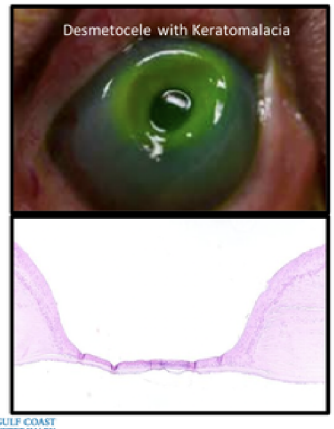
- The cornea has a greyish, gelatinous appearance
- Differentiate from edema
- Bacteria, fungi, and inflammatory cells can produce matrix metalloproteinases (MMP's) that break down corneal collagen
- Can progress from superficial to rupture in 24 hours
- Topical steroids increase the risk of corneal melting



GULF COAST VETERINARY SPECIALISTS

Desmetocele

- Destruction of the corneal epithelium and stroma down to the level of Descemet's membrane
- Often black in appearance
- Desmetocele will not take up fluorescein, but the sides of the ulcer typically stain positive

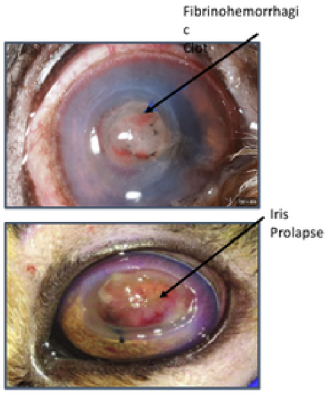


Images: A. VETERINARY NURSING IN ACTION | June/July 2019; B. COPLOW
Text: Slatter's Fundamentals of Veterinary Ophthalmology and Veterinary Ophthalmology, 6th ed

GULF COAST VETERINARY SPECIALISTS

Corneal Rupture

- Rupture through Descemet's membrane
- Can be secondary to:
  - Progressive deepening of a corneal ulcer
  - Traumatic
  - Foreign body
  - Hairs (distichia/ectopic cilia, entropion)



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How to Approach Treatment

- Always perform a STT if the ulcer does not look deeper than anterior stromal
  - Value should be > or = 15 mm/min
  - Increases risk of recurrent ulceration and delays healing
  - If tears < 15 mm/min, treat concurrently for KCS
- Apply proparacaine, but evaluate eyelids before application if entropion is suspected
- Perform tonometry (as long as ulcer does not look deep/ruptured)
- Look for concurrent corneal diseases (i.e. lipid/mineral), look for periocular issues (i.e. eyelid masses, extra hairs, entropion), look for foreign bodies, evaluate intraocular structures for abnormalities (i.e. lens luxation, intraocular tumors)

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How to Approach Treatment

- Consider Age
  - Indolent Ulcers (7 years old or older)
  - Ectopic Cilia (2 years or less, or recurrent ulcers while young)
- If superficial, topical antibiotic therapy can be started TID to QID
  - NPB, tobramycin, or ofloxacin, +/- atropine (normal tears, normal IOP), +/- serum
  - +/- Bandage contact lens
  - Expect healing within a week

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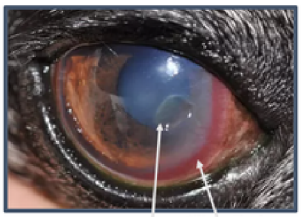
How to Approach Treatment

- Stromal Ulcers
  - Look for a mechanical irritant or FB
  - Look for signs of infection (soft cornea, edema, intense blepharospasm, ciliary flush)
  - Culture +/- cytology if an infection is suspected
  - Topical antibiotics q 1-2 hours
  - +/- Serum or amniotic extract
  - +/- Atropine



Stromal ulcer with early keratomalacia

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Corneal Cytology

- Cytobrush
- Kimura Spatula
- Blunt end of #10 blade handle

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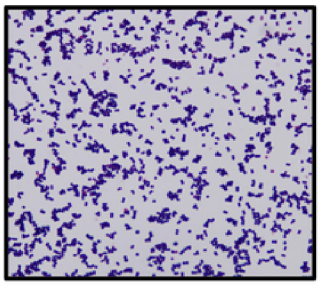
Corneal Cytology



Staphylococcus pseudointermedius

Pseudomonas aerugenosa

GULF COAST VETERINARY SPECIALISTS

veterinary sciences MDPI

Article

Antibiotic Recommendations for Treatment of Canine Stromal Corneal Ulcers

Milan Joksimovic [1], Bradley A. Ford [2], Tatjana Lazic [1,3], Ivan Soldatovic [4], Sergey Luzetsky [3] and Sinisa Grozdanic [1,3,*]

A variety of bacterial species were identified wherein the most common specimens were represented by Staphylococcus pseudintermedius (22%), Staphylococcus epidermidis (12%), Staphylococcus capitis (11%), and Pseudomonas aeruginosa (10%). Based on the overall antibiotic susceptibility data, neopolybac alone (96%) or a combination of neopolybac with either ofloxacin or amikacin (each 99%) showed the best coverage for commonly isolated bacterial organisms from canine corneal stromal ulcers. Results of this study support the use of the combined antibiotics as the first-line response for the treatment of canine corneal stromal ulcers.

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Amniotic Extract

- Morselized Amniotic Membrane
- Anti-inflammatory Against Innate Inflammation
- Contains High Molecular Weight Hyaluronic Acid
  - Superior Ocular Lubrication
  - Prevent Microtrauma to the ocular surface from blinking
- TID Dosing
- 18-Month Shelf Life/ 1 Month Once Reconstituted

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Crosslinked HA Hydrogels

- Most contain 0.75% of a cross-linked hyaluronic acid
- Great lubricant for the corneal surface
- Reduces shear effects of the eyelids on the corneal surface during blinking
- Studies show an improvement in corneal epithelialization
- No anti-inflammatory properties
- Does not stop the break-down of the cornea

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Mid to Deep Stromal Ulcer, Keratomalacia, Desmetocele, or Corneal Perforation…

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If Unable to Refer Immediately….

- Ofloxacin and Neo-Poly-Bac/Neo-Poly-Gram every 2 hours
- Amniotic Extract if available/Serum
- +/- Atropine (not if ruptured, glaucoma, or low tears)
- E-collar
- Harness only (no collar) if deep or ruptured and limit activity
- Systemic antibiotic if ruptured or worried about rupture

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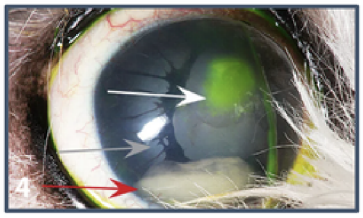
Reflex Uveitis and Hypopyon

- Hypopyon is an accumulation of leukocytes in the anterior chamber
- Can occur due to corneal ulcers, corneal abscesses, uveitis, and systemic illnesses
- Stimulation of corneal nerves can cause the release of prostaglandin, which can lead to reflex anterior uveitis
- Due to leaking of the normally tight junctions in the iris/ciliary body of the eye secondary to intraocular inflammation which allows increased leaking of protein and potentially WBC's, which leads to hypopyon



White arrow: indolent ulcer
Grey arrow: Severe iris atrophy
Red Arrow: Hypopyon

Image from DJ Haeussler, Jr. DVM, MS, DACVO, The Animal Eye Institute, Cincinnati, Ohio
OPHTHALMOLOGY: CLINICIAN'S BRIEF
|JULY 2016|

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Only Debride If You Can Answer YES To One Of Two Questions…

1. IS THIS A SUPERFICIAL INDOLENT ULCER WITH NO INFECTION PRESENT?

2. IS THIS A SUPERFICIAL NON-HEALING FELINE HERPETIC ULCER WITH LOOSE EDGES THAT HAS ALREADY UNDERGONE TREATMENT?

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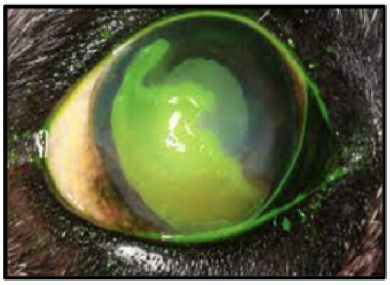
Indolent Ulcers

- Spontaneous Chronic Corneal Epithelial Defect (SCCED), Boxer Ulcer, Recurrent Erosion
- Often spontaneous and only found in middle-aged to older dogs (at least 7 years old)
- Indolent ulcers are superficial, so if it is not superficial, it either is not an indolent ulcer or it is an indolent ulcer that has become infected or there is mechanical trauma (i.e. eyelid mass, distichia, foreign body)
- Loose epithelial edges
- Epithelium cannot adhere normally to the anterior stroma, so the epithelial cells migrate but cannot "stick" in place normally



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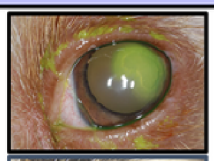
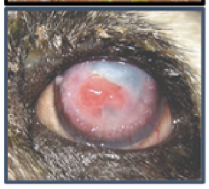
Indolent Ulcers

- Typically, the fluorescein diffuses under the loose epithelial edges
- CTA debridement alone allows for healing in about 30-50%
  - May repeat
- A Bandage contact lens can be used to improve comfort and it may aid in healing (May fall out)
- Often requires a Diamond Burr Keratoplasty (DBK) or Grid Keratotomy
  - DBK about 92% healing rate at 2 weeks
  - Least corneal scaring
- With chronicity, dense granulation tissue and vascularization can develop
- May occur in other eye or even in other locations on the cornea





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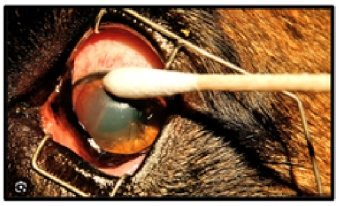
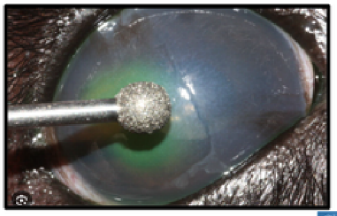
Indolent Ulcer Treatment





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Indolent Ulcer Treatment

- Infection and deepening is the biggest concern
- Ofloxacin and Neo-Poly-Gram QID (after treatment)
- Oral Pain Medication
  - Gabapentin +/- Carprofen
- E-collar
- Recheck in 2 Weeks
- Warn may need to repeat a debridement procedure
- Can take months to heal without a debridement procedure

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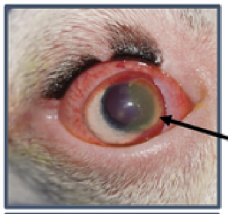
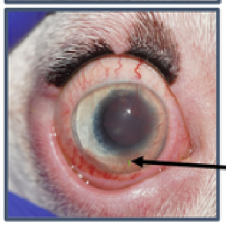


Initial Exam:
OD Indolent
Ulcer
Before DBK



Epithelial
Break
(2 weeks post
DBK)

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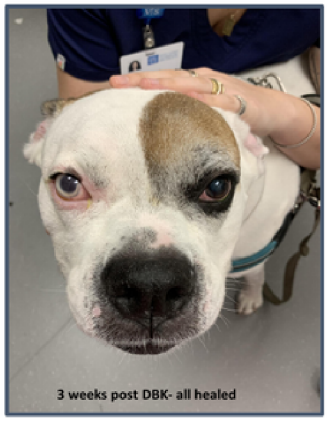
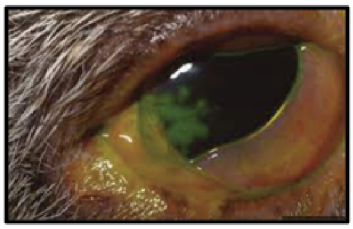


3 weeks post DBK- all healed

Feline Herpetic Ulcers

- Topical antibiotic therapy
  - Preferable 0.5% erythromycin ointment (Chlamydia and Mycoplasma)
  - Avoid Neomycin and Polymyxin B in cats – possible hypersensitivity reaction
- Topical anti-viral therapy
  - 0.5% Cidofovir
    - BID dosing
  - 0.1% Idoxuridine
    - QID or more dosing
- With persistent corneal ulcers with obviously loose epithelial edges, CTA debridement can be performed
  - Rarely needed



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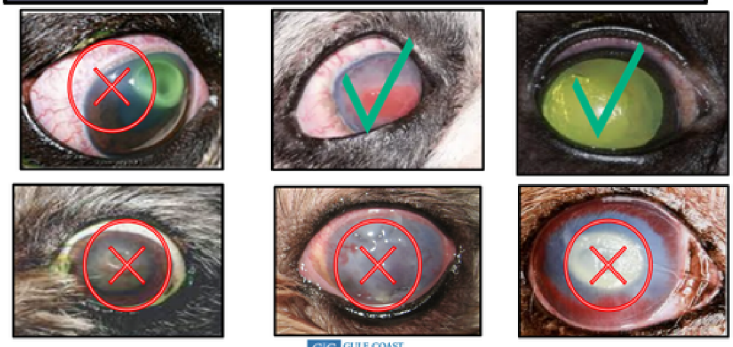
To Debride or Not to Debride???



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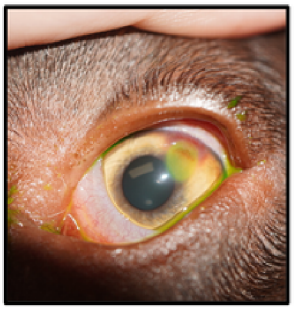
The Case of a Non-Healing Ulcer

- SIGNALMENT: 10-month-old Intact Male GSP
- PRESENTING COMPLAINT: Non-healing ulcer x 3 weeks
- TREATMENTS: NPB, Gentamicin, Deramaxx
- EXAM: Blepharospasm, Mild third eyelid protrusion, Superficial ulcer along dorsal temporal cornea



What are your differentials? GULF COAST VETERINARY SPECIALISTS

In A Young Dog Consider:

- Ectopic cilia
- Distichia
- Foreign Body

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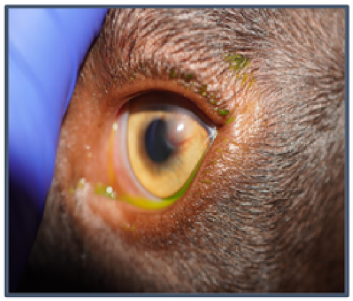
The Outcome of the Non-Healing Ulcer



- 1.5 Weeks Post Foreign Body Removal
- Residual vascularization and mild edema
- Expect full recovery with no residual corneal inflammation or edema

GULF COAST VETERINARY SPECIALISTS

Robin Sankey, DVM, DACVO

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Houston, Tx 77024

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CRITICAL CARE

MELISSA LACAZE

DVM, DACVECC

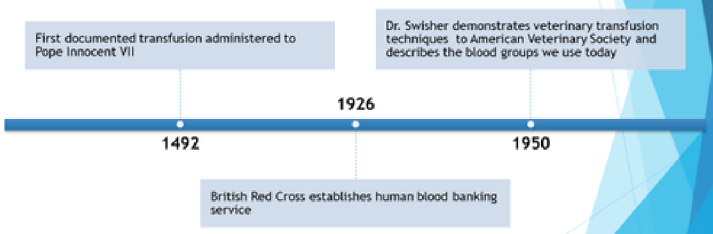
Dr. A. Melissa Garcia-Lacaze received her doctor of veterinary medicine from Texas A&M University in 1998. She then completed a one-year emergency and critical care internship, followed by a three-year emergency and critical care residency at the Animal Emergency Center in Glendale, Wisconsin. Dr. Lacaze joined Gulf Coast Veterinary Specialists in 2002, and became board certified by the American College of Veterinary Emergency and Critical Care in 2003. Dr. Lacaze has a mixed- breed dog, Mungo, and two cats, Nimo and Diamond.

MELISSA.LACAZE@NVA.COM
713-693-1111

Practical Blood Transfusions

Melissa Garcia-Lacaze, DVM, DACVECC
Gulf Coast Veterinary Specialists - Critical Care

History of Transfusion Medicine



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History of Transfusion Medicine

- Veterinarians start using in house donors as crossmatching and typing cards become more available
- Veterinary blood banks are established
- Association of Veterinary Hematology and Transfusion medicine are established to set guidelines

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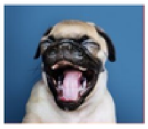
Canine transfusion medicine

DEA 1.1, 1.2, 2, 3, 4, 5, 6, 7 and 8

No naturally occurring antibodies - so reactions to first time transfusions are rare



DEA 1.1, 1.2 and 7 are the most antigenic

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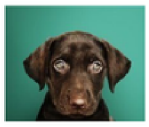
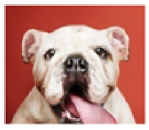
Canine transfusion medicine



Blood type

Crossmatch to determine if unit if compatible



Only perform if patient is stable!

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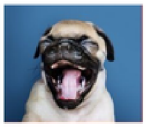
TRANSFUSION PEARL - life threatening hemorrhage

Large bore, short catheter

Crossmatch type not necessary



If patient has had previous transfusion and no type/crossmatch can be done, use negative

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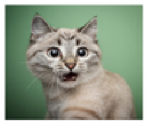
Feline transfusion medicine

Type A, Type B, Type AB (rare)



Cats have naturally occurring antibodies

There is no universal blood type for cats!

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Feline transfusion medicine

Blood type to determine if Type A, Type B or Type AB

Crossmatch

Type A cat given Type B blood - rapid hemolysis
Type B cat given Type A blood - death can occur

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Cornish Rex

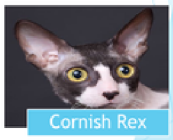

British Shorthair


Somali Cat


Abyssinian


Persian


Himalayan


Cornish Rex

Typical Type B cats = exotic breeds

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Sometimes Type B cats are not exotic breeds

ALWAYS make sure to type cats
prior to a transfusion!

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TRANSFUSION PEARL

Most cats are Type A.....easy to remember
cAt

Standard of care to type and crossmatch all feline blood transfusions

Type cats for plasma transfusions, administer appropriate type - concerns for RBC fragments

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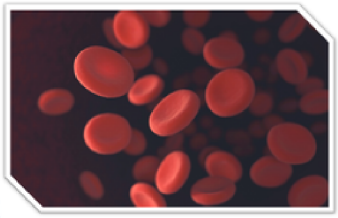
What do I give?

Rule of thumb: Give what the pet is losing!



- Blood loss: whole blood
- Hemolytic anemia: pRBC
- Coagulopathy: plasma OR whole blood

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Transfusion Pearl

Anti-coagulant Rodenticide Ingestion

- Bleeding due to lack of active clotting factors
- Antidote: vitamin K
- Transfusion treatment: fresh frozen plasma OR whole blood
- If you only have frozen plasma - it will still provide vitamin K dependent clotting factors!

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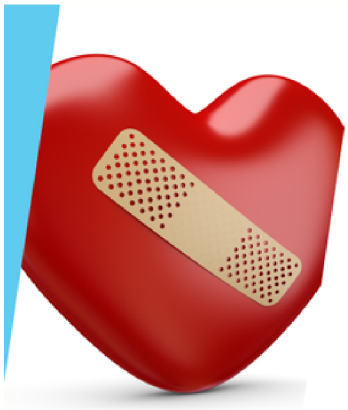

Transfusion Pearl

Plasma to replace albumin

- Not long-term solution
- Questionable improvement in outcome
- Very expensive
- Focus on treating the cause of the loss

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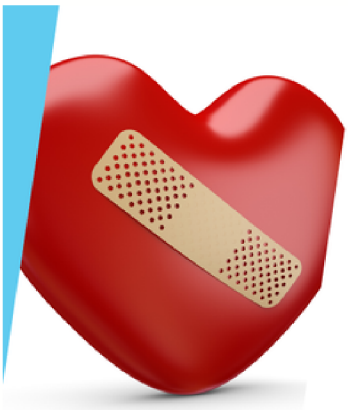

Transfusion Pearl

Platelets to treat IMTP

- Very expensive
- Same process destroys transfused platelets
- Lyophylized platelets - no longer available
- Platelet rich plasma for life threatening bleeding
- Focus on treating the cause of the loss

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When do I give a transfusion?

There is no magic number!

- Clinical appearance
- Age of patient
- How quick was the loss
- Continued blood loss
- Anticipated blood loss
- At 15%, concerns for tissue oxygenation

Transfusion Pearl

- In patients with suspected blood loss:
  - Always evaluate BOTH PCV/TP
  - Splenic contraction causes PCV to remain elevated while TP drops
  - Be proactive - don't wait for the PCV to drop!

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How Fast Do I Give a Transfusion?

- How fast was the loss?
- Current clinical condition
- Concurrent health disease
- Patient's fluid status
- Can bolus from the bag
- Can bolus in a syringe with filter
- If given as an infusion - do not leave unrefrigerated for longer than 4-6 hours

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How much do I give?

Whole blood and pRBC

- Patient size
- Amount of blood lost
- Is there ongoing loss?

Plasma: typically start at 20ml/kg/day and re-evaluate

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How much do I give?

Volume of WHOLE BLOOD to administer:

$$kg \times \frac{\text{desired PCV} - \text{recipient PCV}}{\text{donor PCV}} \times \begin{matrix} 90 \text{ (dogs)} \\ 70 \text{ (cats)} \end{matrix}$$

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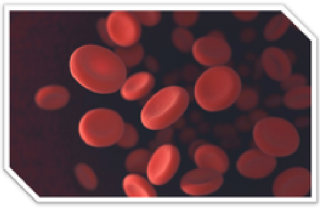
Transfusion Pearl

1ml per lb. will increase the PCV by 1%



5lb. Patient

Current PCV 10% - goal is 25%
That is an increase of 15%

It will take 5ml to increase the PCV 1%

5ml * 15 = 75ml WHOLE BLOOD needed

pRBC (50% volume needed) = 37.5ml

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Transfusion Reactions

Types of reactions:

- Acute immunologic reaction
- Acute non immunologic reaction
- Delayed immunlogic reaction
- Delayed non immunologic reaction

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Transfusion Reactions
Acute Immunologic

- Incompatible unit
- Typically occurs with "second" transfusions in dogs
- Can occur with first transfusions in cats
- Can be seen with plasma/cryoprecipitate transfusions

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Transfusion Reactions
Acute Immunologic

Acute hemolysis

- Respiratory difficulty
- Vomiting
- Diarrhea
- Arrhythmias
- Severe cardiovascular collapse can occur
- Hemoglobinemia / Hemogloburia

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Transfusion Reactions Acute Immunologic

Acute hemolysis - Treatment

- Slow or stop the transfusion
- Support HR, blood pressure with IV fluids
- Corticosteroids are not indicated

Transfusion Reactions Acute Immunologic

Allergic Reaction

- Type 1 reaction: mast cell degranulation resulting in release of vasoactive substances
- Reactions to plasma are more severe

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Transfusion Reactions Acute Immunologic

Allergic Reaction

- Vomiting/diarrhea
- Urticaria
- Erythema
- Pruritis
- Facial swelling
- Can result in anaphylactic shock (hypotension, tachycardia, bronchoconstriction)Lawrence that anything how

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Transfusion Reactions
Acute Immunologic

Febrile Reactions

- Most common type of reaction
- Reaction of donor antigen to recipient antibodies
- Typically self-limiting or responds to slowing the transfusion

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Transfusion Reactions
Acute Immunologic

Transfusion Related Acute Lung Injury (TRALI)

- Typically within 1-6 hours of transfusion, not known to occur in cats
- Bilateral pulmonary infiltrates, fever +/- hypotension
- Adherance of leukocytes to pulmonary endothelium
- Cannot distinguish from ARDS
- Low mortality rate with supportive care
- Must differentiate from transfusion associated circulatory overload (TACO)

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Transfusion Pearl

TACO

- Physical exam: jugular distension
- Chest radiographs: pulmonary venous distension
- TFAST: enlarged caudal vena cava or intrahepatic venous distension
- Protein content of fluid: low (<3.0)
- Treatment: stop fluids / furosemide

TRALI

- Physical exam: no jugular distension
- Chest radiographs: no pulmonary venous distension
- TFAST: no enlarged caudal vena cava or intrahepatic venous distension
- Protein content of fluid: high (>3.5-4)
- Treatment: supportive care / oxygen

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Transfusion Reaction
Acute Non immunologic

- TACO (fluid overload)
- Hypothermia
- Hypocalcemia
- Bacterial contamination

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Transfusion Reaction
Acute non-immunologic

Transfusion Associated Circulatory Overload (TACO)

- Result of incresed blood volume from the transfusion
- Dogs ~5%, Cats 3%
- Normovolemic anemia
- Rapid transfusion
- Concurrent conditions:
  - Heart disease
  - Renal disease

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Transfusion Reaction
Delayed immunologic

Delayed hemolysis

- Occurs when patient develops antibodies to recently transfused antigen
- Typically mild
- PCV falls 1-4 days post transfusion due to early destruction
- Extravascular hemolysis / no hemoglobinuria

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Transfusion Reaction
Delayed Non-Immunologic

Transmission of Infectious Disease

- Risk is significantly decreased with proper donor screening

Dogs
- CBC/chemistry, TT4 and urinalysis
- Anaplasma, Babesia sp, Brucella, Ehrlichia sp., RMSF, heartworm Ag, Lyme AB, vWF

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Transfusion Reaction
Delayed Non-Immunologic

Transmission of Infectious Disease

- Risk is significantly decreased with proper donor screening

Cats
- CBC, chemistry, TT4, FeLV/FIV PCR, FeLV Ag, FIV Ag and urinalysis
- HW panel, toxoplasma titers (IgG/IgM), Anaplasma, Bartonella Sp., Cytauzoon felins, Ehrlichia sp., Feline Coronavirus, Feline hemotropic mycoplasma

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Summary

- Transfusions can be life threatening
- Give the components that are being lost
- There are risks with transfusions
- Knowledge about how to use blood products and how to monitor for reactions is vital for asuccessful outcome!

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INTERNAL MEDICINE

MELANIE PUCHOT

DVM, DACVIM

Dr. Melanie Puchot is a Texas native who received her veterinary degree from Texas A&M University College of Veterinary Medicine in 2013. After graduation, she completed a small animal rotating internship at Cornell University in Ithaca, NY. She then returned to Texas A&M University and completed a 3-year residence in small animal internal medicine finishing in 2017. Dr. Puchot then joined a specialty hospital in New Jersey for four years before coming back to her hometown of Houston. She has a particular interest in endocrinology, nephrology, and interventional endoscopy/radiology.

During her free time, she enjoys spending time with her wife, Diane, camping, and working towards the goal of visiting all the national parks. She has two pets: a dog named Onyx and Cat named Sprite.

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Feline Pancreatitis

Melanie L Puchot, DVM, DACVIM (SAIM)

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Outline

- Pancreatic anatomy and physiology
- Pathophysiology
- Etiology
- Clinical presentation
- Diagnostics
- Treatments

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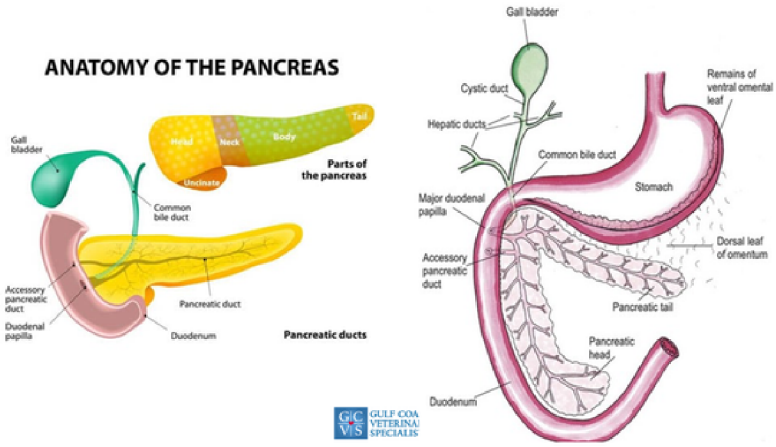
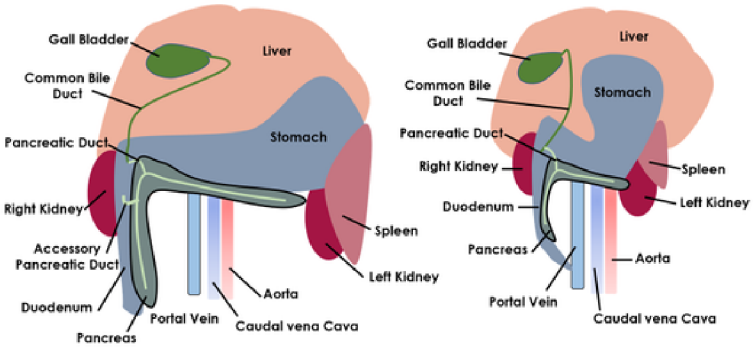
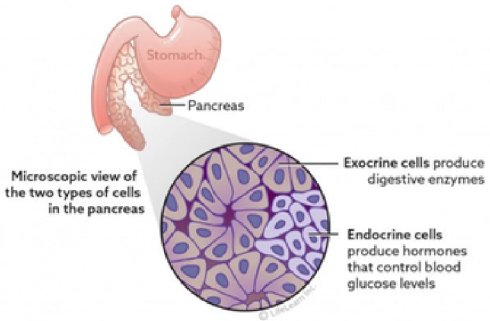




Brief review

- Endocrine: 2%
  - Islets of Langerhans
  - Alpha: Glucagon
  - Beta: Insulin
  - Delta: Stomatostatin
  - PP: Pancreatic polypeptide
- Exocrine: 98%
  - Tubuloacinar gland
  - Digestive enzymes
    - Proteases
    - Amylase and lipase
    - Trypsin inhibitors
  - Inactive zymogens
  - Ductal cell secretions



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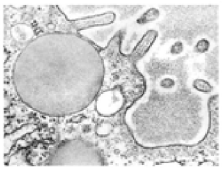
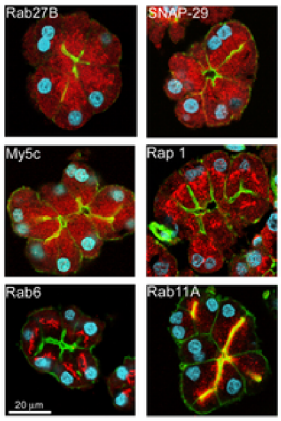
Pathophysiology

- No longer rare
- Premature activation of digestive enzymes
- Pancreatic autodigestion
- Acute vs chronic
- Inflammation in surrounding organs





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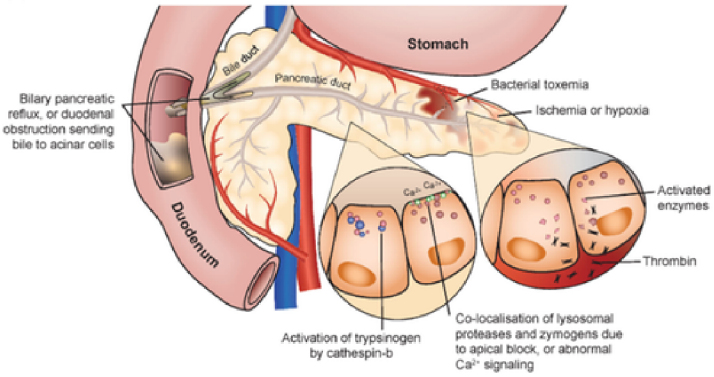

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Pathophysiology

Acute

- Trypsinogen activation
- NFkB pathway
- Influx of neutrophils
- Hypoperfusion and thrombosis
- Biliary reflux / circulation
- Bacterial cholangitis

Chronic

- Acute can lead to chronic
- Pancreatic stellate cells
  - Fibrosis
- Trypsin activation independent
- Cholecystokinin and oxidative stress
- Ductal obstruction or ductal epithelial damage

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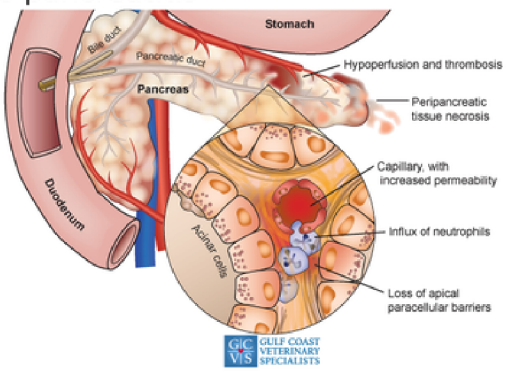
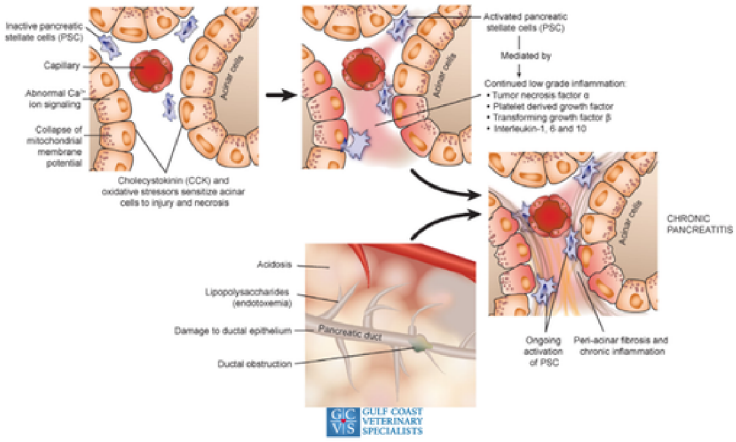
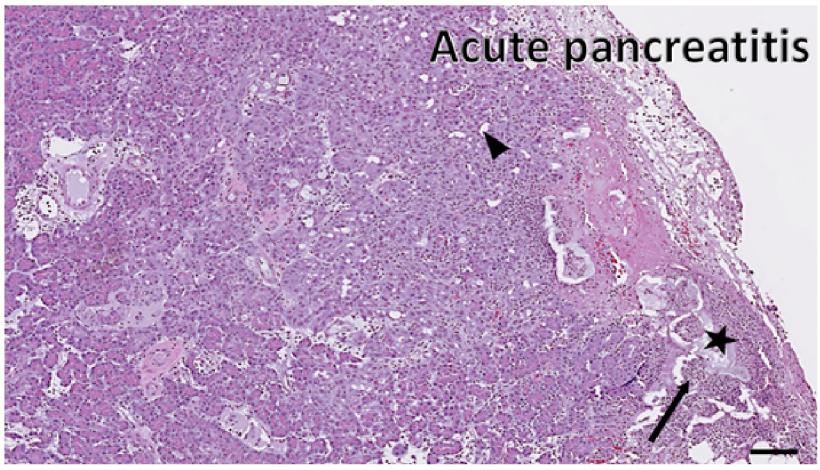
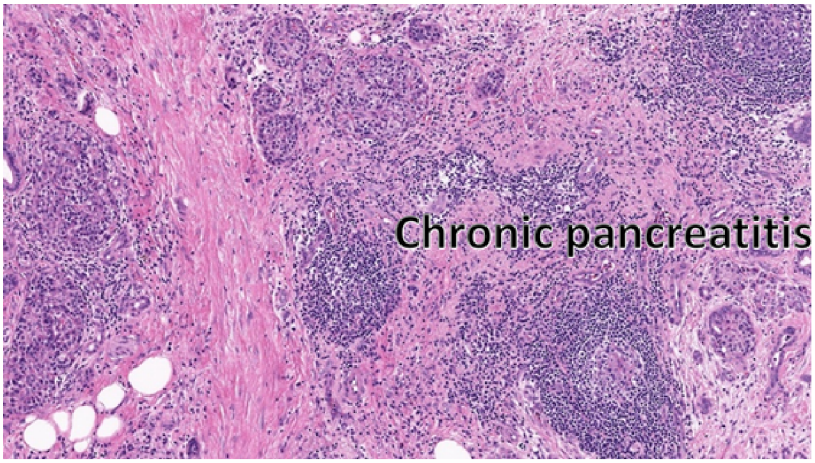
Acute pancreatitis



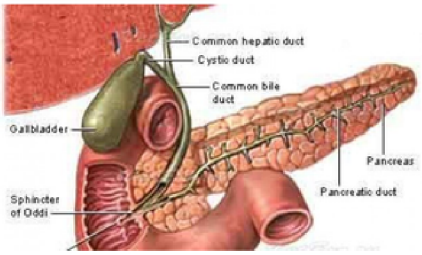






Chronic pancreatitis

Etiology



- Idiopathic
- 'Triaditis'
- Hypotension
- Ischemia
- Trauma
- Immune mediated
- Infection: Toxoplasma, viral
- Neoplasia
- Drugs/toxins
- NOT DIETARY INDISCRETION

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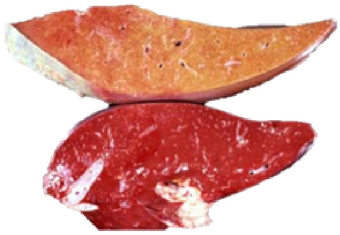
Concurrent illnesses

- Inflammatory bowel disease
- Cholangiohepatitis
- Diabetes mellitus
- Hepatic lipidosis
- Nephritis
- Immune mediated hemolytic anemia



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Role of Bacteria

- FISH identified bacteria in 35% of pancreas
  - Moderate to severe pancreatitis
  - Acute >> Chronic
- Reflux from duodenal papilla
- Intestinal translocation
- No evidence supports clinical relevance
- No antibiotics unless concurrent illness
- Rare pancreatic abscess

Clinical Signs

- Vague
- Lethargy
- Hyporexia
- Vomiting
- Weight loss
- Icterus
- Diarrhea
- Dyspnea
- Rarely, abdominal pain

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Physical exam findings

- Dehydration
- Hypothermia
- Icterus
- Abdominal mass effect
- Pallor
- Tachypnea
- Fever
- Abdominal pain

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Clinical Signs Differences

Acute Pancreatitis
- Young to middle aged
- Hypothermia
- Acute vomiting and anorexia
- Abdominal mass effect
- DIC/Shock
- Dyspnea

Chronic Pancreatitis
- Middle aged to older
- Weight loss
- Polyuria/polydipsia
- Diarrhea

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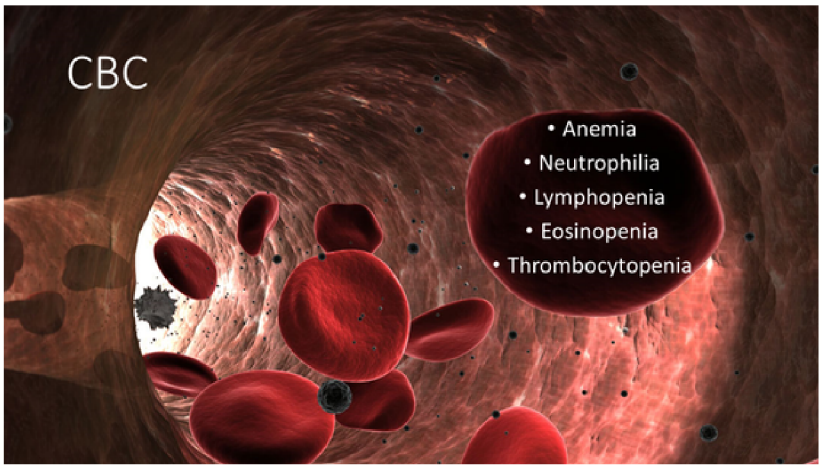
Diagnostics

- CBC
- Chemistry Panel
- Urinalysis
- Pancreatic specific lipase
- Abdominal Ultrasound
- FNA or Biopsy

GULF COAST VETERINARY SPECIALISTS



Chemistry Panel

- Variable
- Liver enzyme elevation
  - ALT, AST, and bilirubin
- Azotemia
- Isosthenuria
- Hypoglycemia or hyperglycemia
- Electrolyte abnormalities: hypokalemia, hyponatremia, and hypocalcemia
- No dyslipidemia expected

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Pancreatic enzyme testing

- Enzymes normally leaked into vascular
- Increased inflammation → increased leakage
- Amylase / Lipase
  - Not specific; DON'T USE
- Pancreatic specific lipases
  - DGGR (PSL; Antech)
  - fPLI (Idexx / TAMU GI Lab)
- Trypsin-like immunoreactivity

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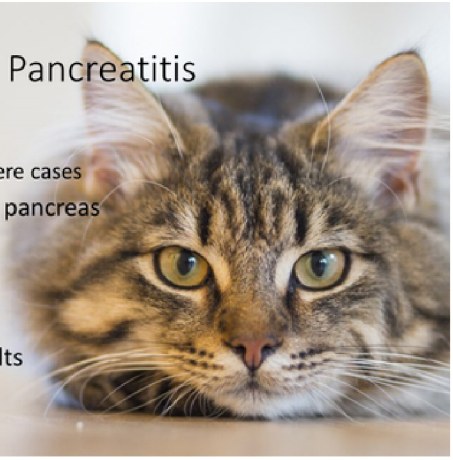
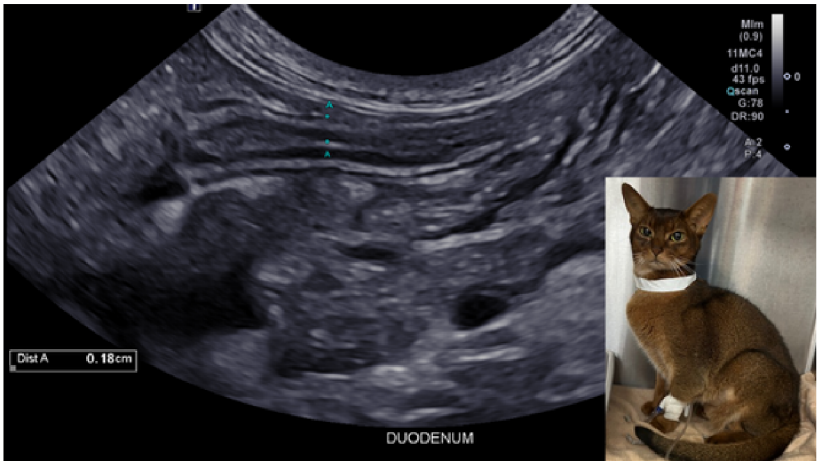
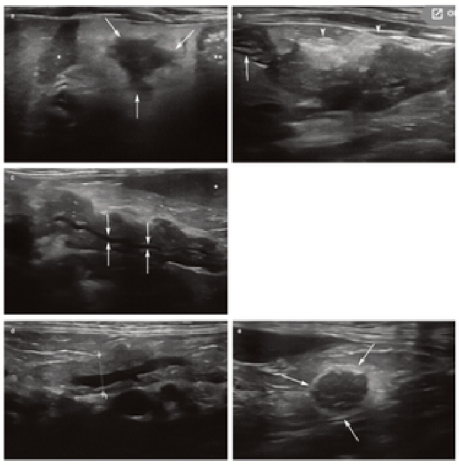
Ultrasound: Acute Pancreatitis

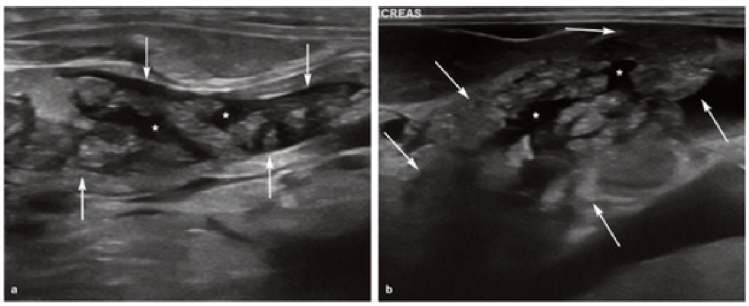
- Low sensitivity
  - Better in moderate to severe cases
- Hypoechoic and enlarged pancreas
- Hyperechoic mesentery
- Focal abdominal effusion
- Duodenitis
- Operator dependent results







Griffin S. Feline abdominal ultrasonography: What's normal? What's abnormal? The pancreas. *Journal of Feline Medicine and Surgery*. 2020;22(3):241-259. doi:10.1177/1098612X20903599



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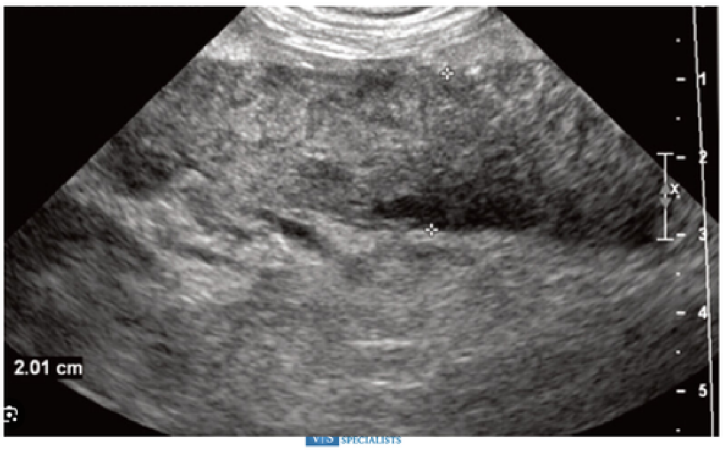
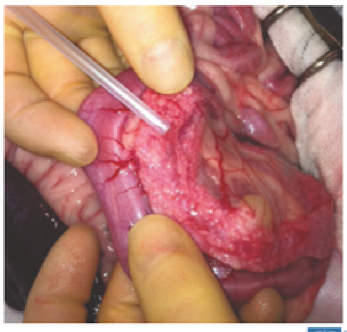
Ultrasound: Chronic Pancreatitis

- Harder in chronic pancreatitis
- Equivocal changes common
- Overlap with acute pancreatitis
- Hyperechoic or mixed echogenicity pancreas
- Dilated common bile duct
- Enlarged pancreas and irregular margins
- Pancreatic nodule hyperplasia – incidental



Pancreatic sampling



- FNA of focal abnormalities
- Laparoscopic or surgical biopsies
- Less commonly done in acute cases
- Unlikely to exacerbate pancreatitis
- Acute: Neutrophilic and fat necrosis
- Chronic: Fibrosis and lymphocytic
- Evaluate for concurrent illnesses

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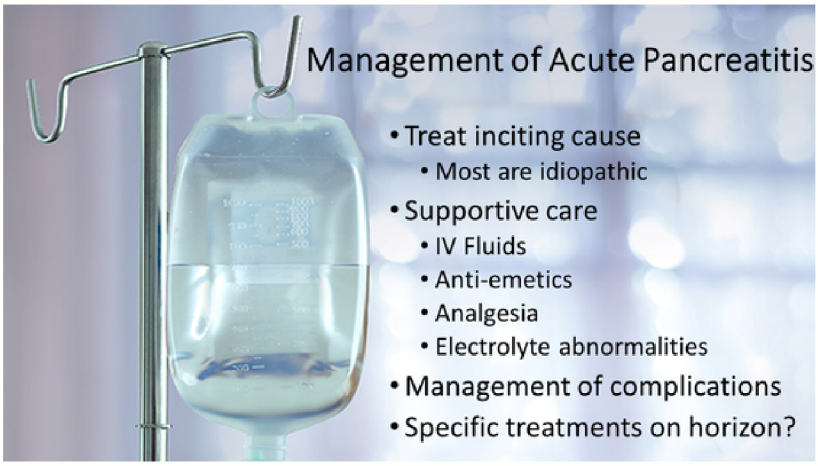

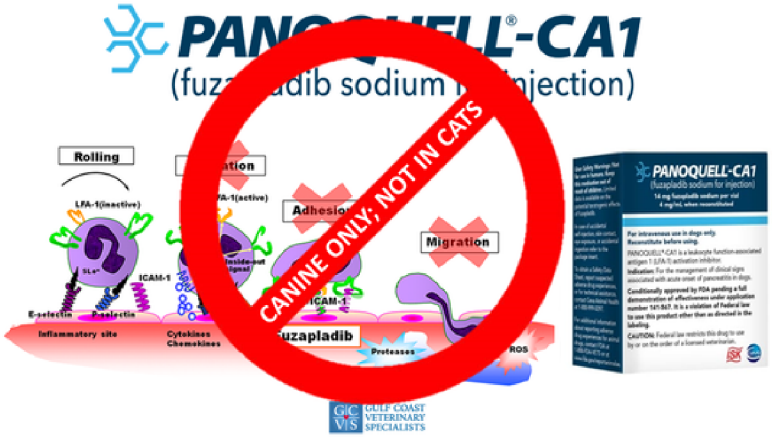

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Enteral Nutrition is VITAL

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Acute Pancreatitis: Other treatments

- Antibiotics?
  - Not recommended in uncomplicated
  - Cholangitis, abscess, sepsis
  - FISH identified bacteria in 35%
- Corticosteroids?
  - Not generally recommended
  - Anti-inflammatory
  - Pancreatitis-associated protein
  - Not enough evidence

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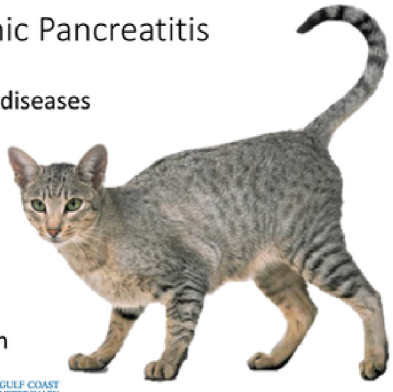
Management of Chronic Pancreatitis

- Treat underlying/concurrent diseases
- Nutrition:
  - No concern about fat content
  - Gastrointestinal diets
  - Hypoallergenic diets
- Anti-emetics
- Analgesics
- Vitamin B12 supplementation



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Chronic Pancreatitis: Immunosuppressives

- Chronic inflammatory condition
- Progressive fibrosis
- Prednisolone
  - Anti-inflammatory doses
  - Concurrent cholangitis or IBD: Immunosuppressive doses
- Cyclosporine
  - Less risk of diabetes
- Chlorambucil
  - Not well investigated
  - Concurrent IBD

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Prognosis

Acute Pancreatitis

- 10-40% mortality rate
- Hypocalcemia
- Hypoglycemia
- Azotemia
- Delayed care / prolonged illness

Chronic Pancreatitis

- Better prognosis
- Long-term management
- Concurrent illnesses

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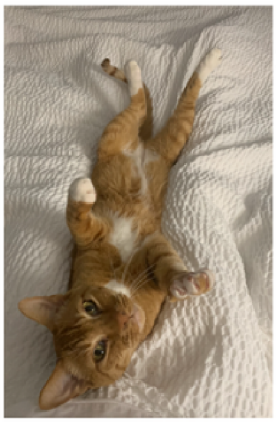
Summary

- Two related but different forms
- Different etiologies than other species
  - Not related to dietary fat or dyslipidemia
  - Idiopathic most common
- Vague and variable clinical signs
- Mainstays of treatment
  - Hydration
  - Nutrition
  - Anti-inflammatory drugs (?)



Questions??

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DERMATOLOGY

KARIN BEALE

DVM, DACVD

Dr. Karin Beale earned her Bachelor of Science in biology and Doctor of Veterinary Medicine degrees from the University of Florida in 1985. After a year of small animal private clinical practice in Washington, D.C., her passion for dermatology emerged. She pursued a residency in veterinary dermatology at the University of Florida, achieving board certification.

For four years, she served as an assistant professor of veterinary dermatology at the University before establishing Gulf Coast Veterinary Dermatology and Allergy. Dr. Beale's expertise extends to infectious skin and ear conditions, as well as managing allergic patients. Beyond her professional commitments, she shares her home with her youngest daughter and two Golden Retrievers. Dr. Beale is fluent in French as a second language and enjoys travel, both for personal enjoyment and educational opportunities. In her leisure time, she channels her creativity into artistic ventures, such as painting and crafting jewelry.

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Canine and Feline Pemphigus Foliaceus

Karin Beale, DVM, Diplomate ACVD
Gulf Coast Veterinary Specialists
Houston, TX

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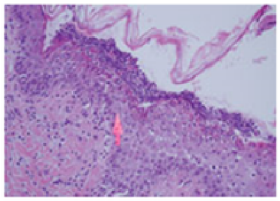
Pemphigus

- Autoantibodies target desmosomal proteins in KC, separation of KC follows, with blistering.
- With PF, the blister occurs in the superficial layers of the skin – typically a subcorneal pustule.



Subcorneal pustule of feline PF

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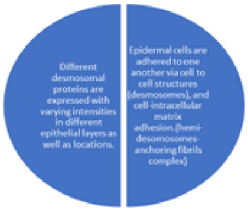
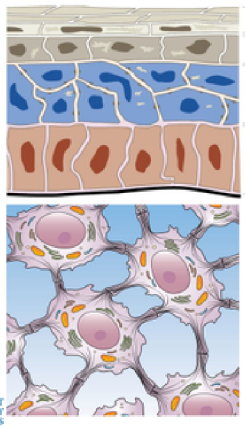
Pemphigus foliaceus





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Pemphigus auto-antigens

- Desmocollin-1 is the major autoantigen in dogs with PF.
- This protein is not found in the mucosa, explaining lack of mucosal lesions.
- An anti-keratinocyte IgG has been demonstrated in cats with PF with different immunoreactivity compared to the canine anti-kc IgG. Potentially explaining some of the differences in clinical signs.

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Pemphigus foliaceus

- Most common autoimmune dermatopathy in dogs and cats.
- Can occur at any age. Median age 5-6 years.
- Canine: Akitas, Chows, Labs, cockers, shepherds, Eng. Bulldogs over-represented.
- Feline: DSH, Siamese, Persians, Burmese and Himalayans are over-represented.

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Pemphigus risk factors

Most cases presumed to be Spontaneous

Risk factors:

- allergic skin disease
- Drugs: sulfonamides, penicillins, cephalosporins, cimetidine
- Topical parasiticides with metaflumizone, fipronil, amitraz, s-methoprene, dinitofuran, pyriproxyfen or permethrin
- Neoplasia (paraneoplastic pemphigus)

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Certifect® induced PF

Robot
Photos courtesy of Dr. Valerie Fadok, DVM, PhD, DACVD

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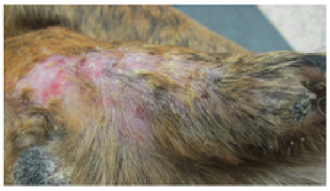
PF dermatologic changes

Primary lesion: pustule, vesicles (often spanning several hair follicles)

Pustules progress to crusts, erosions, alopecia.



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PF: clinical signs

- Lethargy
- Anorexia
- Pyrexia
- Decreased appetite
- Pain
- Pruritus

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Feline pemphigus foliaceus

Pustular, crusting disease.

- Head, face, pinnae
- Trunk
- peri-areolar
- Purulent paronychia
- footpads

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Feline PF

Primary lesion is a pustule.

Crusting predominates

Symmetry in lesions

Pruritus

Clinical signs can mimic feline allergic disease!

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Feline Pemphigus foliaceus

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Feline PF

- Pinnae 85%
- Dorsal muzzle 85%
- Peri-areolar 10%
- Foot pads 22%
- Nail beds 56%

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Canine PF

- Pustular to crusting disease
  - Head, face and pinnae
  - Footpads
  - Trunk
  - Limbs
  - Feet
- Lesions themselves resemble those of pyoderma.

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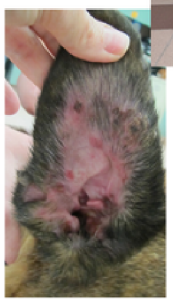
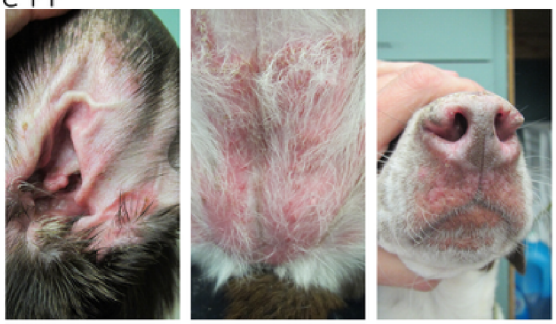
Canine PF



Canine PF



Lady Gaga, 6-year-old Brittany Spaniel

GULF COAST VETERINARY SPECIALISTS

DDX of crusting dermatitis in cats (+/- pruritus)

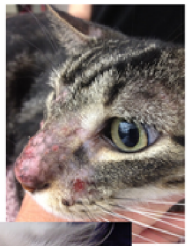
- Dermatophytosis
- Notoedres
- Allergic dermatitis

Notoedres photo courtesy Gail Kunkle, DVM, DACVD

GULF COAST VETERINARY SPECIALISTS

DDX of crusting dermatitis in cats (+/- pruritus)

- Mosquito bite hypersensitivity
- Pemphigus foliaceus

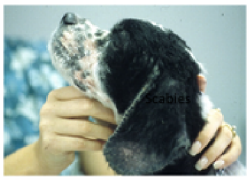


- Idiopathic facial dermatitis of persian cats
- Pyoderma

GULF COAST VETERINARY SPECIALISTS

DDX of pustular to crusting canine skin disease

- Bacterial pyoderma
- Pemphigus foliaceus
- Parasitic skin disease
- dermatophytosis



GULF COAST VETERINARY SPECIALISTS

DDX of pustular to crusting canine skin disease

- Drug reaction
- Hepatocutaneous syndrome (SND)
- Other autoimmune skin diseases (PE, DLE, SLE)

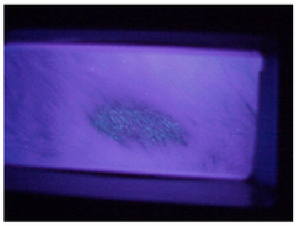
GULF COAST VETERINARY SPECIALISTS

Evaluating the patient

- Thorough history:
  - Indoor/outdoor (cat)?
  - Previous treatments/response?
  - Contact with other animals?
  - Progression of disease?
  - Seasonality?
  - Intensity of pruritus?
  - Drug history in relation to development of disease?

GULF COAST VETERINARY SPECIALISTS

Diagnostics



- Skin scrapings to R/O ectoparasites
- Woods lamp/DTM/ Dtp PCR (always in cats)
- Cytology
- C/S if bacteria noted on cytology
- Minimum data base (CBC, biochemical profile, UA)

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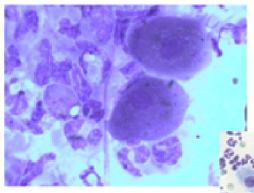
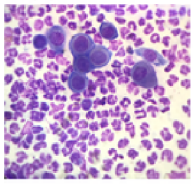
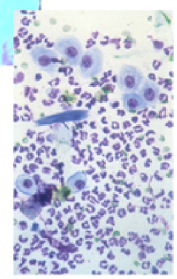
Where to collect samples for cytology

PF cytology

- Neutrophils
- +/- eosinophils
- Acantholytic cells
- Bacteria (?) culture indicated.







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Definitive diagnosis: histopathology

- Collect 3 or more biopsy samples
- If pustules are available, they are ideal to sample.
- If not, biopsy freshly crusted over lesions (exudation underneath the crust)
- Or biopsy heavily crusted areas.

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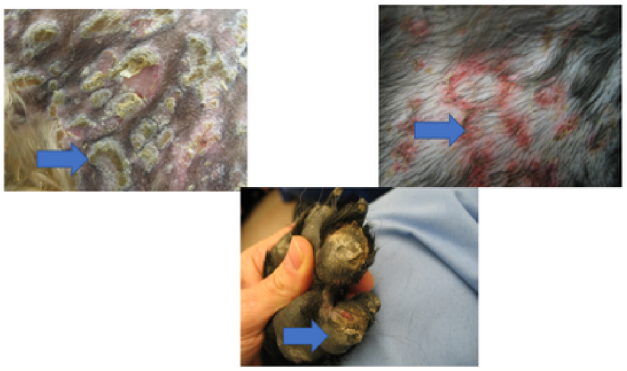
Biopsies for PF

- 6mm punch or larger
- 4-5mm punch if biopsying footpads
- Intact pustules if present!
- Thick, crusty footpads are great areas to biopsy

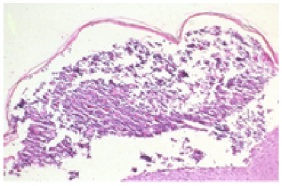
GULF COAST VETERINARY SPECIALISTS

PF biopsies



histopathology

- Subcorneal or intracorneal pustules with neutrophils, +/- eosinophils, acantholytic cells.
- There may be outer root sheath involvement.



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Treatment of feline PF

Glucocorticoids

- Prednisolone 2-4mg/kg induction qd
- Triamcinolone 0.6-2mg/kg induction qd

Immunomodulatory therapy

- Steroid sparing effects
- Chlorambucil 0.1-0.2mg/kg qd-qod
- Modified CsA 7-10mg/kg +
- Oclacitinib 1mg/kg q 12

GULF COAST VETERINARY SPECIALISTS

CsA is a good steroid sparing agent in cats with PF

- Cyclosporine provided greater steroid-sparing effects than chlorambucil
- 6/8 patients managed with CsA were ultimately managed with no GCC. This was only true in 1/7 patients with chlorambucil.
- Starting dose 7-10 mg/kg qd
- Median maintenance dose 5.4mg/kg twice weekly to daily.

Vet Dermatol. 2012 Oct;23(5):403-e76. doi: 10.1111/j.1365-3164.2012.01069.x. Epub 2012 Jun 25.

Use of modified ciclosporin in the management of feline pemphigus foliaceus: a retrospective analysis

Katherine E Irwin [1], Karin M Beale, Valerie A Fadok

Oclacitinib has also been described in the treatment of feline pemphigus foliaceus


Veterinary Dermatology

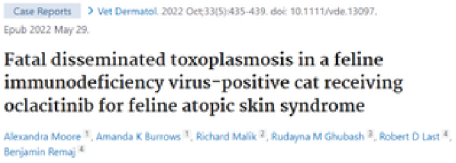
Beneficial effect of oclacitinib in a case of feline pemphigus foliaceus

Isaac Carrasco* Marta Martínez* and Gloria Allbinyana†

GULF COAST VETERINARY SPECIALISTS

Immunomodulatory therapy in cats

- Discussion of recrudescence of latent toxoplasmosis (or novel)
- Outdoor hunting cats?
- Avoid raw diets


Case Reports > Vet Dermatol. 2022 Oct;33(5):435-439. doi: 10.1111/vde.13097. Epub 2022 May 29.

Fatal disseminated toxoplasmosis in a feline immunodeficiency virus-positive cat receiving oclacitinib for feline atopic skin syndrome

Alexandra Moore [1], Amanda K Burrows [1], Richard Malik [2], Rudayna M Ghubash [3], Robert D Last [4], Benjamin Remaj [4]

Steroid monotherapy in cats with PF

Prednisolone as monotherapy with a median induction dose of 2mg/kg daily led to remission in 97% of cats within 8 weeks.

Monotherapy with triamcinolone is more likely to induce remission sooner than prednisolone.

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My "go-to" treatment protocol in cats with PF

- Induction with prednisolone 2mg/kg bid.
- Initiate modified CsA 7-10mg/kg qd.
- Recheck at 7-10 days. Begin a slow steroid taper.
- Recheck week 3, taper further if indicated.
- Ultimately try to discontinue steroids altogether

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CsA side effects in cats

- GI- vomiting, loose stool
- Gingival hyperplasia

- Activation of infectious agents:
  - Mycobacterium
  - Increased shedding *toxocara cati*
  - Activation of clinical syndromes of FIV, FeLV
  - Toxoplasmosis activation

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CsA

- Cats with high CsA levels when first exposed to *T. gondii* can develop fatal infections.
- Seronegative cats either should be kept indoors, or not treated with CsA.
- Check toxo, FIV prior to treatment

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Anabelle, 3 yr FS DSH pre and post treatment with prednisolone and CsA.

2 months CsA (9mg/kg qd), no longer on prednisolone

Treatment of canine PF

- Glucocorticoids:
  - Prednisone 1-2.5mg/kg BID induction
  - (prednisolone)
  - Dexamethasone 0.1-0.3mg/kg qd induction

Additional immunomodulatory therapy induction dose

- Azathioprine 1.5-2.6mg/kg qd-qod
- Modified CsA 5-10mg/kg qd
- Oclacitinib 0.6-1 mg/kg bid
- Mycophenolate mofexitil 10-20mg/kg bid
- Tetracycline/niacinamide 250-500mg each TID
- Doxycycline/niacinamide 5mg/kg bid/250-500mgBID

Azathioprine side effects:

- vomiting
- Hepatotoxicity
- Myelosuppression (median onset 14 days)
- NEVER USE IN CATS

GULF COAST VETERINARY SPECIALISTS

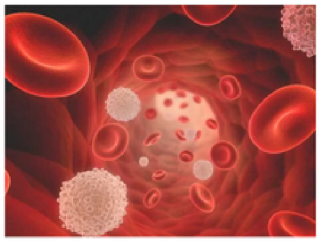
Modified cyclosporine side effects

- Gastrointestinal upset (reduced by freezing capsules)
- Gingival hyperplasia
- Papillomas
- Lichenoid psoriasiform plaques
- Atypical infections

GULF COAST VETERINARY SPECIALISTS

Mycofenolate side effects:

- Diarrhea
- vomiting
- Myelosuppression
- Hepatotoxicity



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Oclacitinib use for PF

Off label use of the medication

0.6-1mg/kg bid initial dose and then taper (label is 0.4-0.6mg/kg qd for maintenance)

Monitor for:

- Leukopenia
- Hepatotoxicity
- Demodicosis (use isoxazoline for flea/tick)
- papillomas

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Monitoring patients

- First recheck 7-10 days
- Recheck again around 3 weeks
- Recheck 2 months
- Following rechecks depend on response to therapy
- Patients receiving immunosuppressant therapy should have routine CBC,Chem, UA when initiating therapy, 3 week recheck and then every 4-6 months.

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How to taper steroids in dogs and cats

- 7-10 day recheck reduce dose by 25% if there are no new lesions and old lesions are 80% healed.
- Recheck every 2-3 weeks initially and continue to reduce dose until plateau is reached.
- Don't reduce steroid sparing agents at the same time.

GULF COAST VETERINARY SPECIALISTS

Calhoun, 7 yr MC black lab

- No prior history of skin disease.
- Recent (4 week) onset of painful, crusty footpads.
- Current medications
  - Carprofen (osteoarthritis)
  - Heartguard
  - Nexguard

GULF COAST VETERINARY SPECIALISTS

Calhoun

- Malaise
- Fever 103.1F
- Pain
- Decreased appetite
- No other lesions other than on pads

- DDX:
  - Pemphigus foliaceus
  - SND/hepatocutaneous syndrome

- Plan:
  - Biopsy
  - CBC, Chem, UA

GULF COAST VETERINARY SPECIALISTS

Calhoun

- Biopsy: subcorneal pustular dermatitis compatible with PF.
- Crusts contain layers of neutrophils, acantholytic cells
- Neutrophilia, normal BUN, albumin, ALT.

- Therapy initiated with prednisone 2mg/kg BID for 10 days
- Phone follow up: significant improvement! Reduce to 1.5mg/kg bid, then 1.5 mg/kg qd

GULF COAST VETERINARY SPECIALISTS

Calhoun 5 weeks after initiation of therapy

- Currently taking 1.5mg/kg qd prednisone.
- Ultimately tapered to maintenance dose of 1mg/kg qod with only occasional flares.

GULF COAST VETERINARY SPECIALISTS

Why just prednisone?

- Well controlled with a moderate dose of prednisone.
- HUNTING DOG!
- I avoid use of CsA if at all possible.

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What happened eventually.....

- Calcinosis cutis.....
- We started modified CsA at 8mg/kg daily.
- Eventually tapered him off prednisone with intermittent low dose use for flares.
- Topical DMSO for calcinosis cutis.
- Retired from hunting.

GULF COAST VETERINARY SPECIALISTS

Gunner, 6 year old Australian Shepherd

- Recently adopted with dermatitis.
- Owner applying topical steroid/Ab/Af ointment.
- Marked alopecia and thick crusting dorsal muzzle, with similar lesions on concave pinna and pinnal margins.
- Mild crusting some footpad margins
- Two focal areas of alopecia and crusting on trunk.

GULF COAST VETERINARY SPECIALISTS

Gunner plan:

- Cytology: neutrophils with scant acantholytic cells, no bacteria seen.
- Biopsy: 3 sites, 2 dorsal muzzle and one trunk.
- MDB: mild neutrophilia, mild increase ALP
- Sent home on prednisone 1mg/kg bid while awaiting biopsy results.

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Gunner 10 days

- VERY PU/PD
- Multiple emails exchanged with owner in the 10 day period. ☺
- Currently on 1 mg/kg qd.
- Discussed other therapy options

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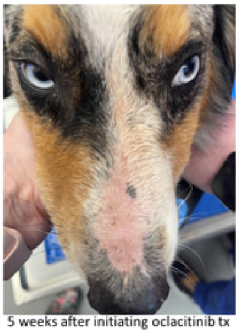
Gunner's options:

- Switch to dexamethasone from pred.
- Add in an additional immune modulator to reduce dose of steroids.
  - CsA
  - oclacitinib
- Additional therapies
  - Sun avoidance, sunscreen

GULF COAST VETERINARY SPECIALISTS

Gunner

- Oclacitinib 1mg/kg bid
- Prednisone slowly tapered to ½ mg/kg QOD over 4 weeks.
- Ultimately managed with oclacitinib 1 mg/kg qd and topical tacrolimus qd on dorsal muzzle



5 weeks after initiating oclacitinib tx

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PF….you never know when the next case will walk in your door!

GULF COAST VETERINARY SPECIALISTS

Questions?

GULF COAST VETERINARY SPECIALISTS

CARDIOLOGY

ERIC OWENS

DVM, DACVIM

Dr. Eric Owens is a board-certified cardiologist from Chicago. He studied at the University of Notre Dame and then earned his Doctor of Veterinary Medicine from St. George's University, with clinical training at North Carolina State University. Dr. Owens completed a small animal internship at The Ohio State University. Thereafter, he completed a cardiology internship and a cardiology residency at Oregon State University.

His clinical interests include interventional cardiology and congenital cardiac disease, and his research interests include advanced cardiac imaging modalities, utilization of endovascular and minimally invasive therapies, and cardiomyopathic disease in dogs.

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713-693-1111

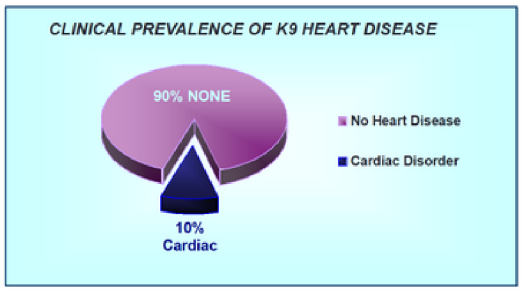
Degenerative Valve Disease: Practical Guide to Management

Eric Owens, DVM, DACVIM (Cardiology)
Gulf Coast Veterinary Specialists

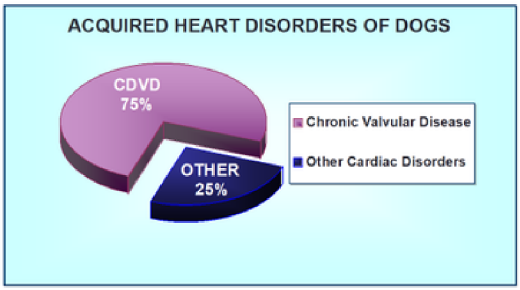
Outline

- Why does CDVD matter?
- Review mitral valve disease
- Clinical course
- Medical therapy
- Complications and outcomes
- New therapy

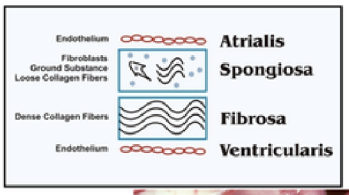
Importance

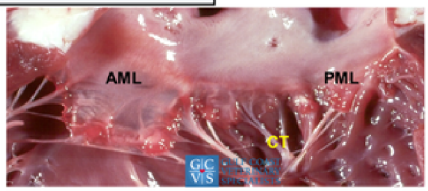


Prevalence

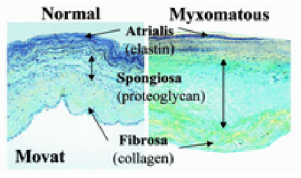


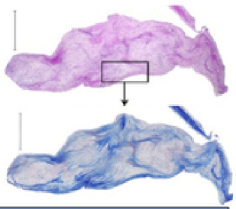
Mitral Valve Leaflets





Myxomatous Degeneration

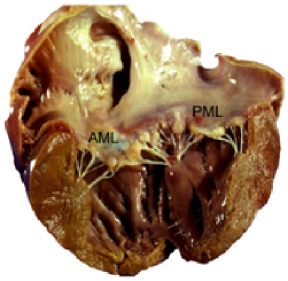


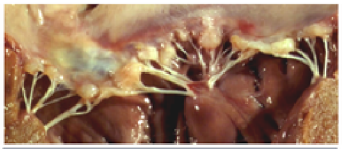


Fox. JVC 2012. 14;1:103-126

1. Degeneration of the fibrosa
   -collagen and elastin dissolution/disorganization
2. Thickened spongiosa
   -hyaluronic acid, chondroitin sulfate accumulations
3. Endothelial damage
4. Hemorrhage, edema

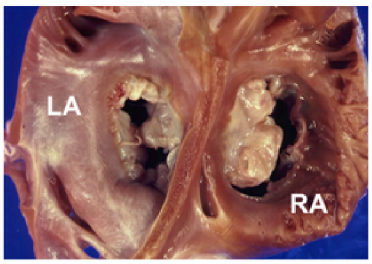
Pathology of CDVD





Grade 1 - opaque, thickened
Grade 2 - small nodules
Grade 3 - large nodules, plaques thickened chordae
Grade 4 - leaflets grossly distorted, hemorrhage, edema, ruptured chordae

GULF COAST VETERINARY SPECIALISTS



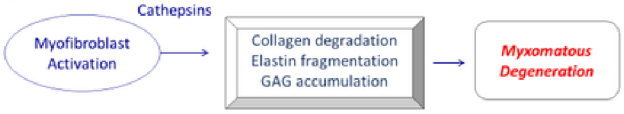
Distribution of Lesions

• Mitral valve alone	62.0%
• Mitral and tricuspid	32.5%
• Mitral and aortic	2.5%
• Tricuspid valve alone	1.5%

GULF COAST VETERINARY SPECIALISTS

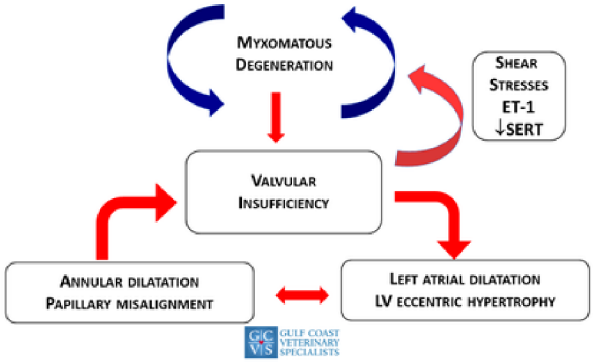
Pathogenesis and Etiology

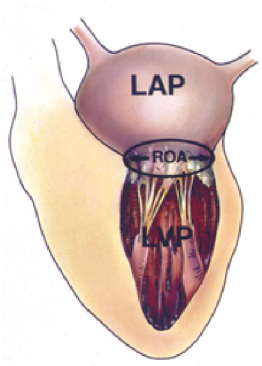
- Etiology still unknown
- Suspected to be genetic due to breed associations
- May be one or more inciting causes
  - NOT associated with prior infection or bacteremia
- Endothelial damage may activate and suppress substances involved in cell growth



GULF COAST VETERINARY SPECIALISTS

Progression

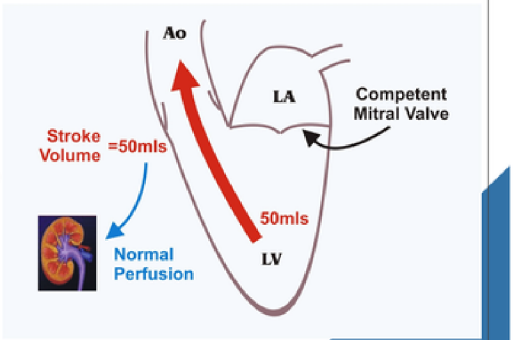




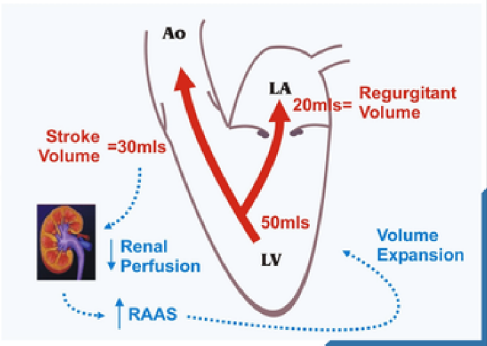
Regurgitant Volume

- Determinants of Regurgitant Volume
  - Regurgitant orifice area (ROA)
  - LV-LA pressure gradient (SPG)
    - Duration of systole (RT)

Pathophysiology

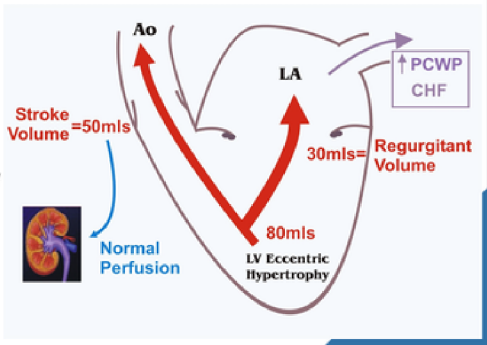


Pathophysiology



GULF COAST VETERINARY SPECIALISTS

Pathophysiology



GULF COAST VETERINARY SPECIALISTS

Signalment
- Small breed
- Older Age
- Males

GULF COAST VETERINARY SPECIALISTS

Physical Exam

Grade 1-6 Scheme	Simplified Scheme
Grade 1/6	Soft
Grade 2/6	
Grade 3/6	Moderate
Grade 4/6	Moderate / loud
Grade 5/6	Loud / thrilling
Grade 6/6	

GULF COAST VETERINARY SPECIALISTS

Murmur Intensity and Severity

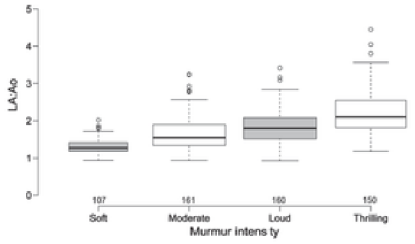
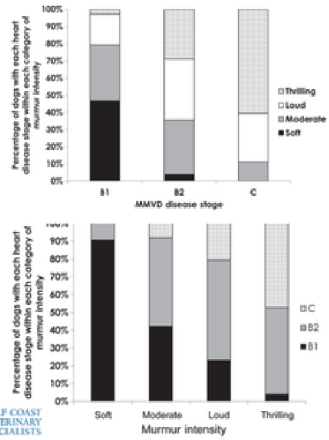


FIG 1. Increasing murmur intensity is associated with increasing left atrial size, as determined by the left-atrial-to-aortic ratio (LA:Ao). The black line denotes the median value, the box denotes the interquartile range (IQR (25th to 75th percentile)), the circles denote values greater than 1·5 times the IQR greater than the 75th percentile. Numbers above the category labels denote the sample sizes in each category. All groups were different from each other (P<0·05)

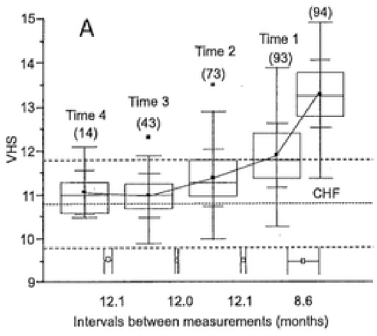
- Ljungvall I, et al. *J Sm Anim Pract.* 2014;55:545-550

GULF COAST VETERINARY SPECIALISTS

Grade 1-6 Scheme	Simplified Scheme
Grade 1/6	Soft
Grade 2/6	
Grade 3/6	Moderate
Grade 4/6	Moderate / loud
Grade 5/6	Loud / thrilling
Grade 6/6	



GULF COAST VETERINARY SPECIALISTS



Lord PF, et al. *JVIM* 2011;25:1312-1319

Radiographic Monitoring

- VHS increases slowly in early disease
- % increase goes up in later stages

GULF COAST VETERINARY SPECIALISTS

Thoracic radiographs

Three hallmarks of cardiogenic pulmonary edema:

1. Left atrial enlargement
2. Pulmonary venous distension
3. Interstitial or alveolar lung pattern

Differentiate causes of coughing: cardiac vs. pulmonary

Physical exam: Is there a loud, left apical systolic murmur? Heart rate?

Chest x-rays: Are all hallmarks present? Crucial for management

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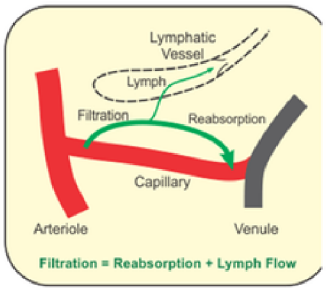
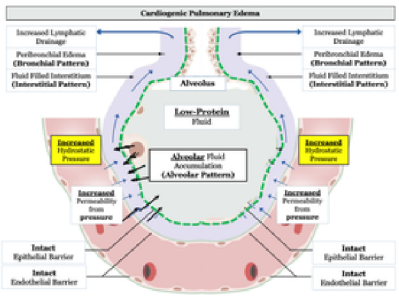
Congestive heart failure



The interstitial volume (bounded area) depends on the rates of filtration, reabsorption and lymph flow.



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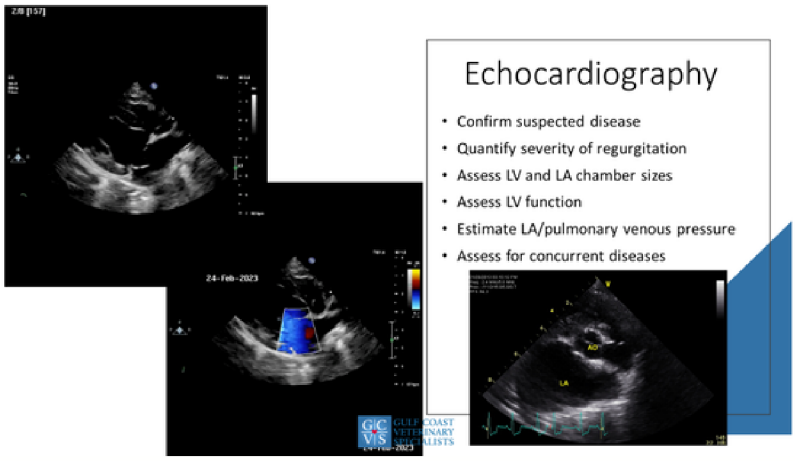


Echocardiography

- Confirm suspected disease
- Quantify severity of regurgitation
- Assess LV and LA chamber sizes
- Assess LV function
- Estimate LA/pulmonary venous pressure
- Assess for concurrent diseases

Monitoring Disease Progression

- Best diagnostic test = Thoracic radiographs
  - Monitor increase in left ventricular and left atrial size
- Soft murmur/Mild LAE: Recommend every 12-24 months
- Moderate murmurs/LAE: Recommend every 6-9 months
- Loud murmurs/Severe LAE: Recommend every 3-4 months

Therapy

Therapy of CDVD

- Asymptomatic dogs
  - Without heart enlargement
  - With enlargement
- Symptomatic dogs
  - Congestive Heart failure
    - Left sided: Pulmonary Edema
    - Right sided: Pleural effusion, Ascites
  - Complications

GULF COAST VETERINARY SPECIALISTS

Review CDVD staging

Stage A - no murmur or echocardiographic abnormality	Stage B – heart murmur or echocardiographic abnormality	Stage C – congestive heart failure	Stage D – advanced heart failure
• Higher than average likelihood of development of disease	• B1 – range from no heart enlargement to enlargement not meeting B2 criteria • B2 – VHS >10.5, LA:Ao 1.6, LVIDdN 1.7, grade 3/6 murmur or louder	• Furosemide • ACEi • Pimobendan • Spironolactone	• > 8 mg/kg/day furosemide • Torsemide

GULF COAST VETERINARY SPECIALISTS

Preclinical therapy

- Stage A
  - No specific therapy
  - Annual evaluation for high-risk breeds
- Stage B1
  - No specific therapy
  - Recheck every 6-12 months
- Stage B2
  - Pimobendan 0.25-0.3 mg/kg BID
  - Dietary
  - ACEi – no consensus
  - Beta-blockers, spironolactone, amlodipine in specific circumstances
  - Surgical intervention

ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs

Bruce W. Keene[1] | Clarke E. Atkins[2] | John D. Bonagura[3,4] | Philip R. Fox[5] | Jens Häggström[6] | Virginia Luis Fuentes[3] | Mark A. Oyama[4] | John E. Rush[7] | Rebecca Stepien[8] | Masami Uechi[9]

GULF COAST VETERINARY SPECIALISTS

ACVIM stage B2

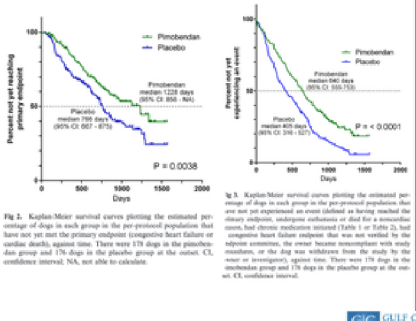


Fig 2. Kaplan-Meier survival curves plotting the estimated percentage of dogs in each group in the per-protocol population that have not yet met the primary endpoint (congestive heart failure or cardiac death), against time. There were 178 dogs in the pimobendan group and 176 dogs in the placebo group at the outset. CI, confidence interval; NA, not able to calculate.

Fig 3. Kaplan-Meier survival curves plotting the estimated percentage of dogs in each group in the per-protocol population that are not yet experienced an event (defined as having reached the primary endpoint, undergone euthanasia or died for a noncardiac reason, had chronic medication initiated (Table 1 or Table 2), had congestive heart failure endpoint that was not verified by the adjcom committee, the owner became noncompliant with study procedures, or the dog was withdrawn from the study by the owner or investigator), against time. There were 178 dogs in the pimobendan group and 176 dogs in the placebo group at the outset. CI, confidence interval.

Effect of Pimobendan in Dogs with Preclinical Myxomatous Mitral Valve Disease and Cardiomegaly: The EPIC Study—A Randomized Clinical Trial

GULF COAST VETERINARY SPECIALISTS

Preclinical ACE inhibitors

- Preclinical ACE inhibitors are controversial
  - DELAY (2020) study, SVEP (2002) trial did not demonstrate benefit
  - VETPROOF (2007), Puchelon et. al (2008) demonstrated benefit
- Some clinicians start ACE-inhibitor before CHF, others do not
- ACVIM consensus statement: 5 (of 10) panelists recommend treatment with ACEi (weak level of evidence)

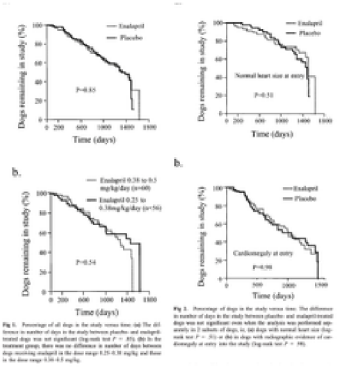


Fig 1. Percentage of all dogs in the study versus time. (a) The difference in number of dogs in the study between placebo- and enalapril-treated dogs was not significant (log-rank test $P = .85$). (b) In the treatment group, there was no difference in number of days between dogs receiving enalapril in the dose range 0.25–0.58 mg/kg and those in the dose range 0.58–0.5 mg/kg.

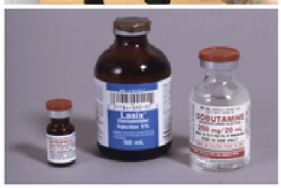
Fig 2. Percentage of dogs in the study versus time. The difference in number of dogs in the study between placebo- and enalapril-treated dogs was not significant even when the analysis was performed separately in 2 subsets of dogs, ie, (a) dogs with normal heart size (log-rank test $P = .51$) or (b) in dogs with radiographic evidence of cardiomegaly at entry into the study (log-rank test $P = .98$).

GULF COAST VETERINARY SPECIALISTS

Acute congestive heart failure

- Most common signs: tachypnea, dyspnea, cough
- Other signs:
  - Reduced appetite
  - Exercise intolerance
  - Syncope
- Diagnosis?
  - History
  - Physical exam, **HR**
  - Chest radiographs

GULF COAST VETERINARY SPECIALISTS



Acute CHF

Pulmonary edema:

- Oxygen
  - 40-50% inspired oxygen desired
  - Oxygen cage (small dogs)
  - Nasal canulas (large dogs)
- Furosemide
  - IV is best
  - IM, SQ if unstable
- Pimobendan – if possible
- +/- nitroprusside
- +/- nitroglycerin
- +/- dobutamine

GULF COAST VETERINARY SPECIALISTS

Management of acute CHF

- Lasix protocol:
  - Mildly decompensated dog: 2 mg/kg IV initial dose
  - Moderately decompensated dog: 2-4 mg/kg IV initial dose
  - Severely decompensated dog: 4-6 mg/kg IV initial dose
- Reassess respiratory rate and effort every 30 minutes
  - Try to reduce dose on subsequent injections
  - May repeat dose every 1-2 hours and then spread out frequency …q 4 hours, q 6 hours, q 8 hours
  - Max ~ 12-16 mg/kg/day

GULF COAST VETERINARY SPECIALISTS

Management of acute CHF

- When dog is stable:
  - IV catheter placement
  - Draw blood to assess renal values
  - Thoracic radiographs to assess severity of pulmonary edema
- Debate over repeated boluses vs. CRI of furosemide
  - Pros: More consistent diuresis, less potassium loss
  - Cons: Requires concurrent fluid administration
    Requires syringe pump
    No reported treatment benefit to CRI
- My personal preference is intermittent bolus dosing

GULF COAST VETERINARY SPECIALISTS

Acute respiratory distress

- Sedation
  - Sedation may be necessary….and life saving.
    - Butorphanol: 0.05-0.4 mg/kg IM or SQ
      - Usual dose 0.1-0.2 mg/kg
    - Acepromazine: 0.005-0.02 mg/kg
      - Usual dose: 0.01 mg/kg

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Management of acute CHF

<u>Monitoring and discharge</u>

- Hourly respiratory rate and effort
- Heart rate every 4-6 hours

- Avoid any extra fluid administration
- Free access to water unless profound distress
- Repeat imaging 12-24 hours into therapy
- Once breathing normally and drinking = able to discharge
  - I like to have them eating too
- Repeat chest x-rays prior to discharge (ideal)
  - Or can send home and repeat them at 1 week recheck

GULF COAST VETERINARY SPECIALISTS

Chance of success?

- High survival rate to discharge for 1st time Acute CHF

- Open conversation with owners regarding:
  - Need for life-long oral medications
  - Financial and emotional investment
  - Possibility and expectation of recurrent HF
  - Possibility of arrhythmia complications
  - Possibility of sudden death

- Long term prognosis: Average of ~ 1 year

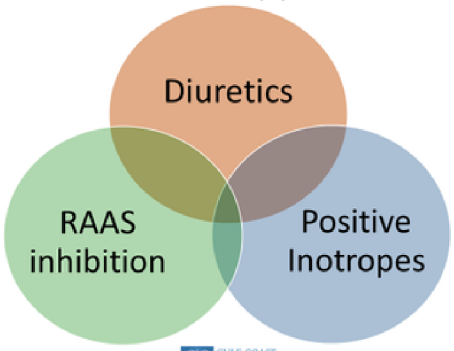
GULF COAST VETERINARY SPECIALISTS

Transition from acute to chronic therapy

- Frequently withhold ACE-inhibitor until eating well at home
- Recheck in about 1-2 weeks after discharge:
  - Renal values
  - Chest x-rays
- Long term recheck plan: Once every 3-4 months

GULF COAST VETERINARY SPECIALISTS

Chronic therapy of CHF



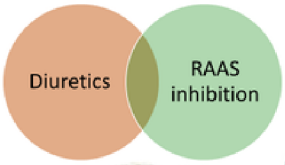
GULF COAST VETERINARY SPECIALISTS

Chronic CHF Treatment

- Furosemide:
  - Starting dose: 1-2 mg/kg PO SID-BID
  - Chronic doses: 2-4 mg /kg PO BID; uptitrate with recurrent CHF
  - If > 5-6 mg /kg BID needed, switch to TID admin or torsemide
  - Assess electrolytes more frequently as dose goes up

GULF COAST VETERINARY SPECIALISTS

Chronic Therapy of CDVD



GULF COAST VETERINARY SPECIALISTS

Chronic CHF treatment

- Furosemide
- ACE-inhibitor
  - Enalapril or benazepril
    - Starting dose: 0.3-0.4 mg/kg PO BID
    - Assess renal values before and 1 week post
    - If CREA inc > 15%:
      - Reduce dose to 0.2 mg/kg PO BID

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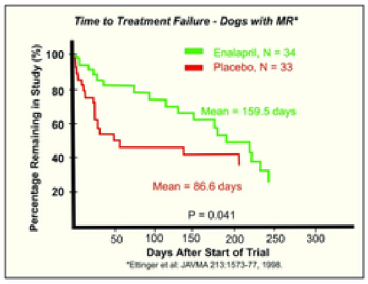
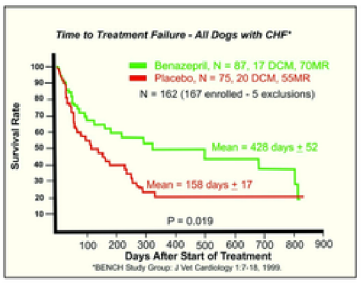
ACE Inhibitors



Enalapril



Benazepril

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Chronic CHF treatment

- Furosemide
- ACE-inhibitor
- Pimobendan
  - All dogs with documented CHF
  - Dose: 0.3 mg/kg PO BID
  - Up to 1 mg/kg TID for advanced, refractory cases

GULF COAST VETERINARY SPECIALISTS

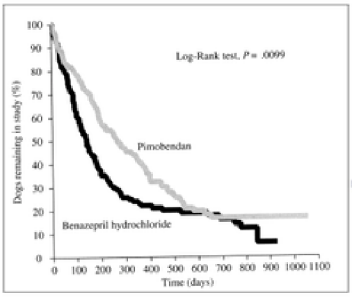
Pimobendan

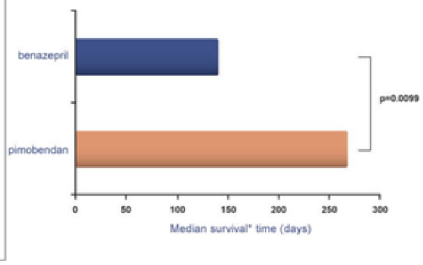
Inodilator

Benefits:
- Improves clinical signs
- Increases exercise tolerance
- Improves survival

GULF COAST VETERINARY SPECIALISTS

Pimobendan





Häggstrom J, et al. The QUEST Study. J Vet Int Med 2008 22:1124-1125

GULF COAST VETERINARY SPECIALISTS

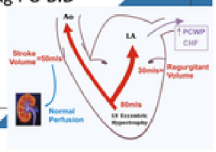
Chronic CHF treatment

- Furosemide
- ACE-inhibitor
- Pimobendan
- Spironolactone
  - Dose: 0.5-4 mg/kg PO SID-BID
  - Used as a second diuretic with furosemide
  - Sequential nephron blockade (collecting duct)
  - May help manage hypokalemia

GULF COAST VETERINARY SPECIALISTS

Refractory CHF therapy

- Furosemide, pimobendan, ACEi, spironolacone
- Still having episodes of CHF….what now??
  - Change: Switch from furosemide to torsemide
    - More potent loop diuretic (10x), but less info known
    - Dose: 0.2 mg/kg SID-BID
  - Change: SQ admin of furosemide
  - Add: Hydrochlorothiazide: uptitrate to 2-4 mg/kg PO BID
  - Add: Amlodipine (0.1 mg/kg SID)
    or Hydralazine (0.5-2 mg/kg BID)



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Chronic or refractory CHF therapy

What else can we do that is helpful?

- Intermittent fluid removal: pleural or peritoneal
- Optimization of all arms of therapy
  - Diuretics: Sequential nephron blockade
  - RAAS inhibition
- Cough suppressants for "intractable cough"
  - Ensure no active pulmonary edema
  - Hydrocodone: 0.2 mg/kg PO q6-12 hours
  - Lomotil 0.2 mg/kg q8h
  - Butorphanol: 0.05-1.0 mg/kg PO q 6-12 hours

GULF COAST VETERINARY SPECIALISTS

Chronic or refractory CHF therapy

What can the client do that is helpful?

- Medication administration compliance
  - Pill pockets, gelatin capsules, compounded meds
- Resting respiratory rate log
  - < 36 breaths per minute, resting or asleep
  - Smart phone apps
- Diet modification:
  - Salt-restriction, be wary excessive much sodium restriction
  - 50 – 100 mg/100kcal
  - Tufts - https://heartsmart.vet.tufts.edu/nutrition/

Heart Disease Breathing Rate Monitor for Dogs

Long-term low salt diet increases blood pressure by activation of the renin-angiotensin and sympathetic nervous systems

Jialiang Wang, Yi Deng, Xue Zou, Hao Luo, Pedro A. Jose, Chunjiang Fu, Jian Yang & Chunyu Zeng

GULF COAST VETERINARY SPECIALISTS

Prognosis

- Progression of CDVD can be slow....
- May have a murmur for years before CHF develops
- Generally once CHF develops, most dogs will live (on meds) for about 1 year
- If complications occur, prognosis shortens

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Complications of CDVD

1. Arrhythmias: Atrial fibrillation
2. Pulmonary hypertension
3. Ruptured chordae tendineae
4. Left atrial tear
5. Syncope

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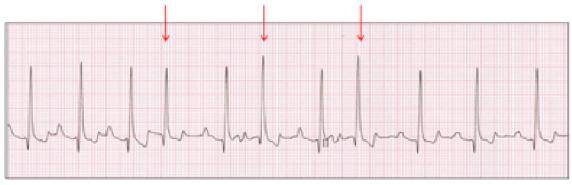
Arrhythmias



Atrial Premature Contractions (APCs)

- No specific therapy required or initiated for APCs
- But….important clue that more severe arrhythmias may be develop in near future

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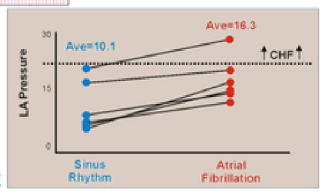
Atrial fibrillation

1. No P waves
2. Irregularly irregular
3. Supraventricular
4. Tachycardic



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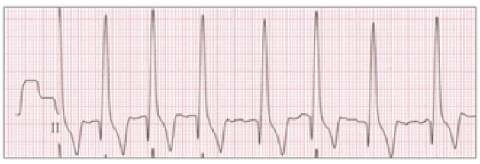
Atrial fibrillation



- Loss of atrial kick + tachycardia → Decreased cardiac output
- Therapy: Digoxin: 0.003-0.005 mg/kg PO BID
  Diltiazem: 1-2 mg/kg PO TID
  or extended-release diltiazem: 2-3 mg/kg PO BID

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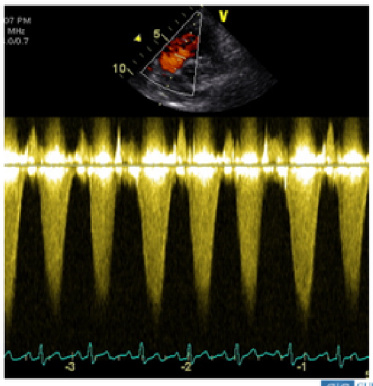

Pulmonary hypertension

- Collapse, ascites
- R apical murmur
- Pulmonary disease

GULF COAST VETERINARY SPECIALISTS

J Vet Intern Med 2000;14:430–434

Clinical Efficacy of Sildenafil in Treatment of Pulmonary Arterial Hypertension in Dogs

A.J. Brown, E. Davison, and M.M. Sleeper

Clinical efficacy of tadalafil compared to sildenafil in treatment of moderate to severe canine pulmonary hypertension: a pilot study☆

J.A. Jaffey, DVM, MS[a], S.B. Leach, DVM[a], L.R. Kong, DVM[a], K.E. Wiggen, DVM[a], S.B. Bender, PhD[b,c,d], C.R. Reinero, DVM, PhD[a,*]

Pulmonary hypertension

- Sildenafil, tadalafil
- PDE 5 inhibitor
- Pulmonary arterial vasodilator
- Sildenafil 1-2 mg/kg TID
- Tadalafil 1-2 mg/kg SID ($$$)

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Pulmonary Hypertension

When should we add sildenafil?

1. If the PHT is severe (> 75 mmHg)
   - Requires echocardiography
2. If the patient is exhibiting syncope, exercise intolerance
3. If the patient has signs of right-sided CHF

- If there is left-sided CHF present, I address that first

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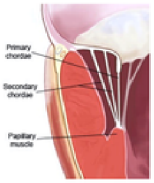
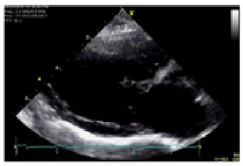


Ruptured Chordae Tendineae

- Acute pulmonary edema
  - Often without commensurate left-heart enlargement
- Collapse, cyanosis
- Variable murmur
- Sudden death

GULF COAST VETERINARY SPECIALISTS



Ruptured Chordae Tendineae

- Require aggressive therapy and hospitalization
- Survival rate to discharge: ~50%
- May shorten prognosis

***Chordae tendineae* Rupture in Dogs with Degenerative Mitral Valve Disease: Prevalence, Survival, and Prognostic Factors (114 Cases, 2001–2006)**

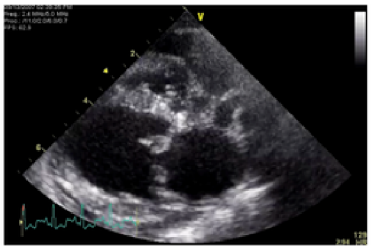
GULF COAST VETERINARY SPECIALISTS

Treatment of Congestive Heart Failure with Intravenous Nitroglycerin in Three Dogs with Degenerative Valvular Disease

Ron Achiel, DVM, Andy Carver, DVM, DACVECC, Robert A. Sanders, DVM, DACVIM

- Nitroprusside
  - 2 -10 µg/kg/minute
  - Start low and increase if needed
  - Potent mixed vasodilator
- Nitroglycerin
  - 1-6 µg/kg/minute
  - Start low and increase if needed
  - Venous vasodilator
- Hydralazine – onset 1 hour, peak 3 hours
- Amlodipine – peak effect 4-7 days, not recommended
- Furosemide
  - High doses needed initially
  - ~4 mg/kg boluses

GULF COAST VETERINARY SPECIALISTS



Left Atrial Tear

- Collapse, ascites, weak pulse
- Diminished murmur
- Sudden death

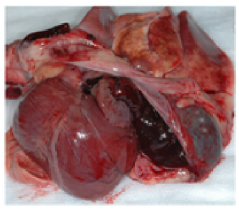
- Therapy:
  - May be futile
  - Vasodilators and inotropes
  - +/- Pericardiocentesis
  - Surgical closure

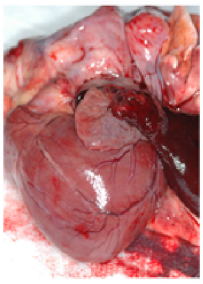
Debutamine infusion without pericardiocentesis as a treatment for cardiac tamponade due to left atrial rupture in a dog

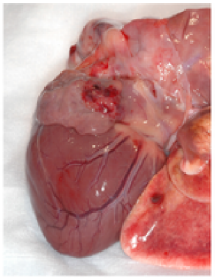
Noriko Isoyama | Yusuke Uchimura | Erika Maeda

GULF COAST VETERINARY SPECIALISTS

Left Atrial Tear







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Left Atrial Rupture Secondary to Myxomatous Mitral Valve Disease in 11 Dogs

Nakamura et al. *J Am Anim Hosp Assoc* 2014; 50:405-408

- 10/11 dogs survived > 24 hours after LA tear
- Median survival = 203 days (range 0-760 days)
- Survival improved if no previous CHF

- Treatment Plan:
  - Vasodilation to lower afterload, hopefully lower LA pressure
  - Amlodipine: 0.1 mg/kg PO SID

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Syncope and Weakness

- Arrhythmias- Tachycardia
  - SVT, atrial fibrillation: Try to slow heart rate
  - Ventricular tachycardia: uncommon with CDVD
- Non-arrhythmia causes
  - Pulmonary hypertension: add sildenafil
  - Low forward flow: Add positive inotrope
- Vasovagal syncope
  - No effective therapy

Holter Monitoring of Small Breed Dogs with Advanced Myxomatous Mitral Valve Disease with and without a History of Syncope

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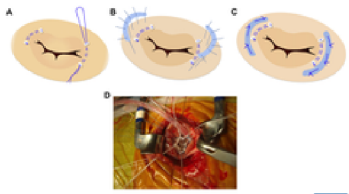
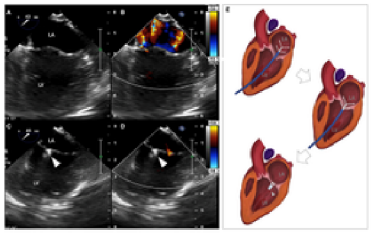
New therapies

- Mitral valve repair surgery – UK, Tokyo
- Transcatheter repair – CSU, TAMU, others





Preliminary Outcome of a Novel Edge-to-Edge Closure Device to Manage Mitral Regurgitation in Dogs

Bo Liu[1], Stacey B. Leach[2], Wenzhi Pan[1], Fangyu Zheng[1], Liujun Jia[1], Xueying Zhou[1] and Jing Li[1]

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Summary

- CDVD is slowly progressive disease
- Murmur intensity correlates reasonably with disease severity
- Best monitoring tool: Chest x-rays
- Best definitive diagnosis: Echocardiography … but not always necessary
- Pimobendan is great
- Uncomplicated cases, including CHF, can be managed without cardiologist
- Surgical intervention is here!

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DIAGNOSTIC IMAGING

KATIE PANSINI
DVM

KYLE PFIEFER
DVM

Orthopedic Problems in General Practice: Radiograph Physics, Positioning, and Common Diseases

Dr. Katie Pansini and Dr. Kyle Pfeifer

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Table of Contents

- General physics of radiography
  - Heel effect, plate locations
- Developmental diseases
  - Positions for best interpretation
  - Osteochondrosis/ osteochondritis dissecans
  - Panosteitis
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  - Humeral intracondylar fissures
- Degenerative diseases
  - Degenerative joint disease – Hips and elbows
  - Intervertebral disc disease and degenerative spinal changes
  - Feline capital physeal fractures
  - Feline metaphyseal osteopathy
- Trauma
  - Cranial cruciate ligament tears
  - Fractures
  - Physeal fractures
  - Fracture healing

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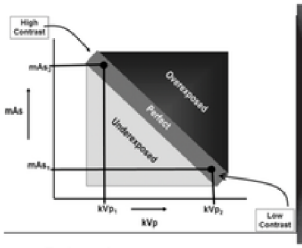
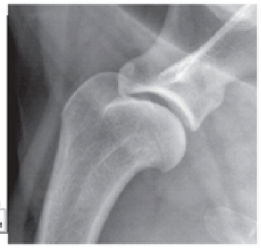
Radiographic technique



Typical mAs:
- Extremities = 10
- Spine = 40

Allows for kVp in the 40-60 range
Increasing kVp by 15% = 2x mAs



Low kVp High mAs
High contrast
Low latitude

High kVp Low mAs
Low contrast
High latitude

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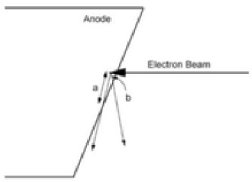
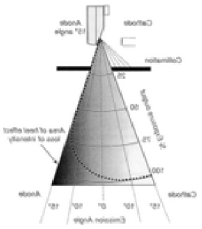
The Heel Effect





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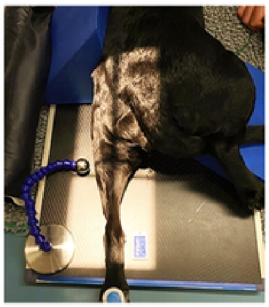
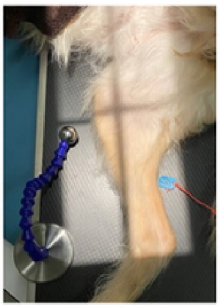
Increasing resolution for small body parts





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Positioning aids

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Radiographic Views

- At least 2 orthogonal radiographs
  - 90° from each other
  - Often a craniocaudal or dorsopalmar/plantar and mediolateral views.
- Additional views
  - Oblique views
  - Flexed/extended
  - Stressed views
- Collimated to the region of interest

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Radiographic Views

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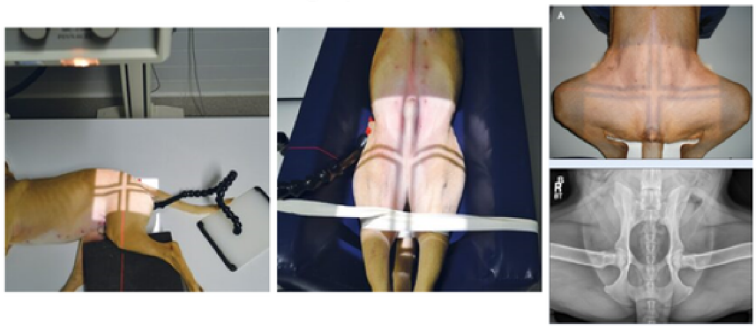
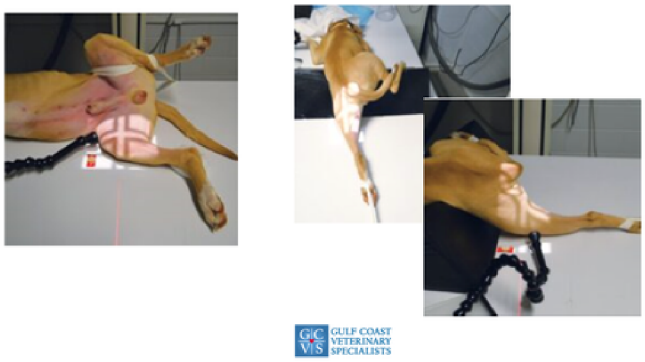
Radiographic Views

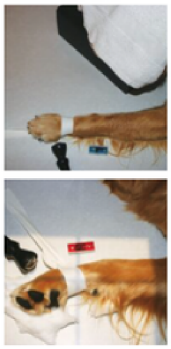
Radiographic Views

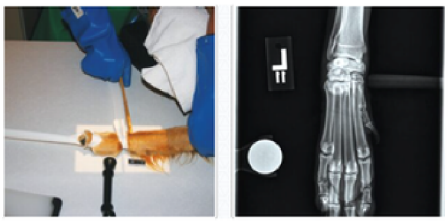


Radiographic Views



Radiographic Views





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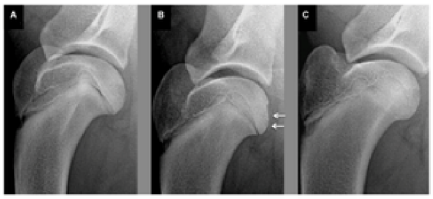
Osteochondrosis / Osteochondritis Dissecans

- One of the most common causes for canine thoracic limb lameness
- Often bilateral
- Young, rapidly growing dogs
- Failure of endochondral ossification
  - Caudal humeral head - most common
  - Trochlea of the talus
  - Femoral condyle
  - Humeral condyle – part of elbow dysplasia
  - Spine
- Image acquisition tip: supinate the shoulder on the mediolateral view



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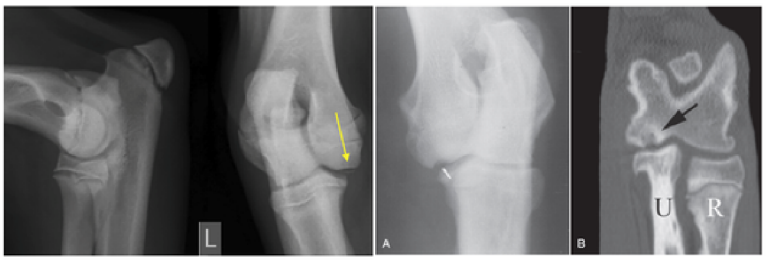

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Osteochondrosis / Osteochondritis Dissecans

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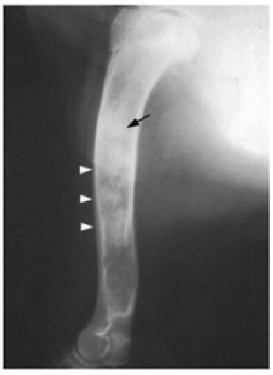
Panosteitis

- Self-limiting
- Males > Females
- Large and giant breeds
- Typically between 5 and 12 months of age

- Shifting limb lameness
- Subtle signs, difficult to differentiate from joint pain

- Early phase: Increased medullary opacity near nutrient foramen, with blurring of the medullary trabecular pattern
- Middle phase: Medullary opacities begin to coalesce. Can see a smoothly marginated periosteal reaction.
- Late phase: Resolution of medullary opacities. May see reduced trabecular pattern.



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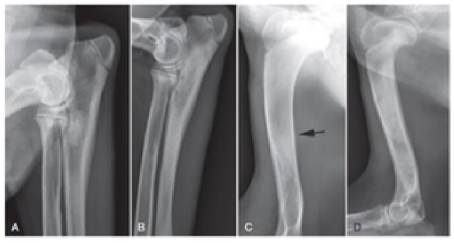
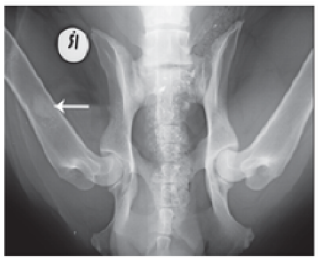
Panosteitis





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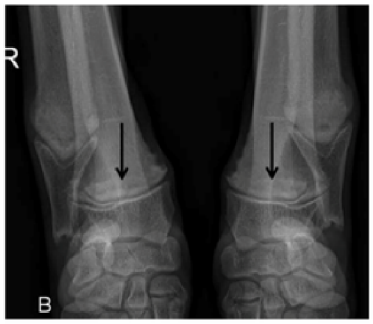
Hypertrophic Osteodystrophy (Metaphyseal Osteopathy)

- Young, rapidly growing, large dogs
- Swelling of the metaphases of long bones, pain, lameness, pyrexia, depression, and inappetence
- Etiology is still incompletely understood.
- Typically bilaterally symmetric and progressive.



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Hypertrophic Osteodystrophy (Metaphyseal Osteopathy)

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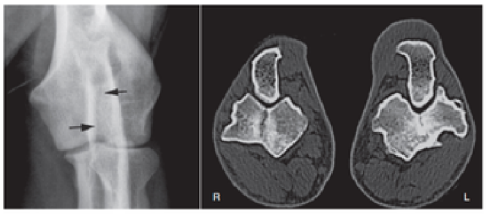
Incomplete Ossification of the Humeral Condyle

- Also known as humeral intracondylar fissure
- Predominantly in spaniel breeds, though many brachycephalics and other breeds are also affected
- Etiology is not completely understood, and is subject to much debate.
- Most commonly diagnosed by CT, or when the fissure ultimately leads to a fracture
- Occasionally identified on radiographs



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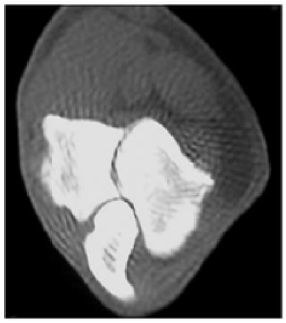
Incomplete Ossification of the Humeral Condyle



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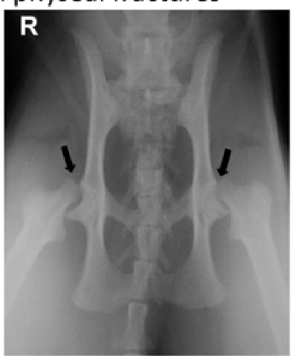
Spontaneous femoral capital physeal fractures

- Occurs in the absence of trauma
- Affected cats:
  - Male
  - Neutered
  - Obese
  - Younger than 2 years of age
- Testosterone and estrogen facilitate cartilage maturation in the growth plate, so reduced levels leads to delay of cartilage maturation and closure
- Age of neutering does not seem to influence the degree of growth plate closure
  - No difference found between cats neutered at 7 weeks and 7 months



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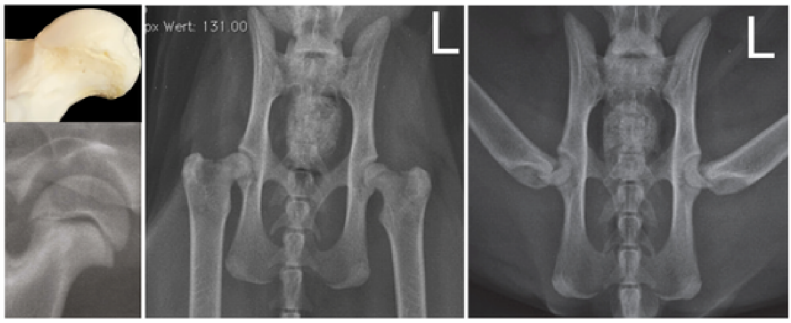
Growth plate closure at 30-40 wks of age in intact cats

Spontaneous femoral capital physeal fractures



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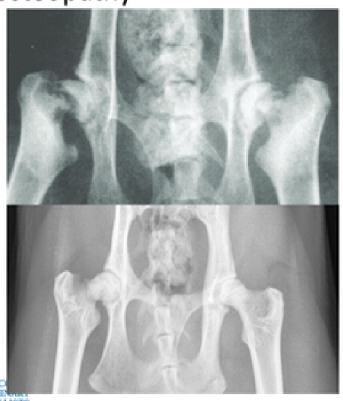
Metaphyseal osteopathy

- Similar signalment and history to spontaneous capital physeal fractures
- Etiopathogenesis is not completely understood
  - Necrosis and collapse of the femoral neck can occur with either an increase or decrease in vascularization of the femoral neck
- Unclear if related to spontaneous femoral capital physeal fractures
  - Often chronic lameness with acute worsening
  - Osteolysis and sclerosis of the femoral neck occurs following capital physeal fracture
- Epiphysis is not affected, possibly due to blood supply through the round ligament



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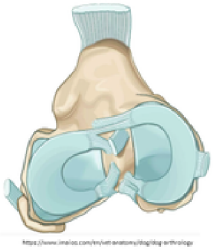
Meniscal ossicles

- Occurs along the cranial horn of the medial meniscus and to some extent the cranial intermeniscal ligament
- May be seen alone or with concurrent degenerative changes
- Not typically associated with pain on physical exam
- Was considered an incidental finding



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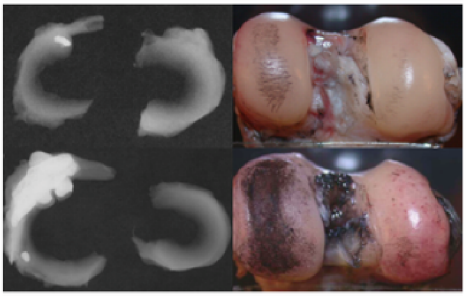
Meniscal Mineralization in Domestic Cats

Mila Freire[1], DVM, James Brown[2], DVM, Diplomate ACVR, Ian D. Robertson[2], BVSc Diplomate ACVR, Anthony P. Pease[2], DVM, MS, Diplomate ACVR, Jonathan Hash[1], BA, Stuart Hunter[3], DVM, Diplomate ACVP, Wendy Simpson[4], DVM, Andrea Thomson Sumrell[1], RVT, and B. Duncan X. Lascelles[1], BSc, BVSc, PhD, DSAS(ST), Diplomate ACVS & ECVS

- Cats with meniscal mineralization were significantly older (10.5 years vs 7.46 years), weighed less (4.57kg vs 5.5kg), and had lower BCS (2/5 vs 3/5)
- Prevalence of 46% in one or both stifles (100 cats)
- No significance in pain scores
- Clear relationship between medial meniscal mineralization and cartilage damage on the medial femoral condyle and medial tibial plateau
  - Unknown if meniscal mineralization is a cause or result of cartilage damage



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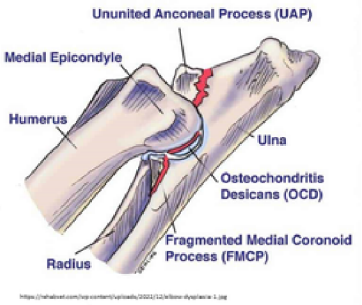
Elbow dysplasia

Optimal positioning (4 views)
- Mediolateral (in a relaxed standing position)
- Flexed mediolateral
- Craniocaudal
- Craniolateral-15°-caudomedial oblique



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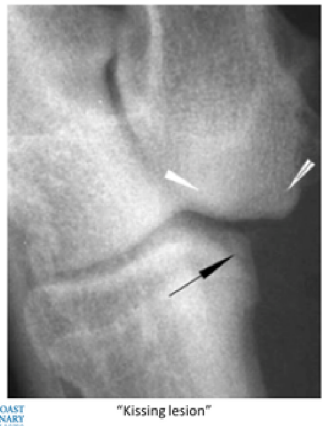
Ununited anconeal process

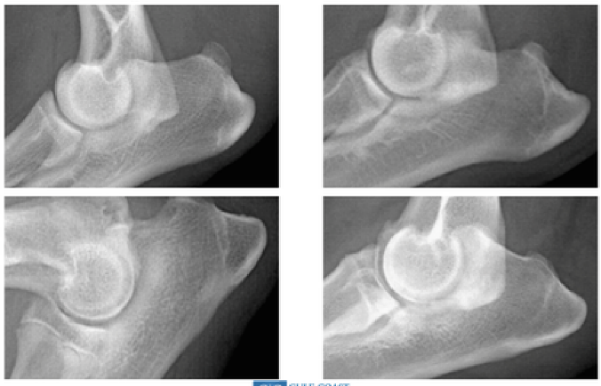
- Anconeal process physis closes around 20-22 wks
- Humeral condyle physis closes around 20-32wks

Osteochondritis dissecans (OCD)



"Kissing lesion"

Changes to the medial coronoid process



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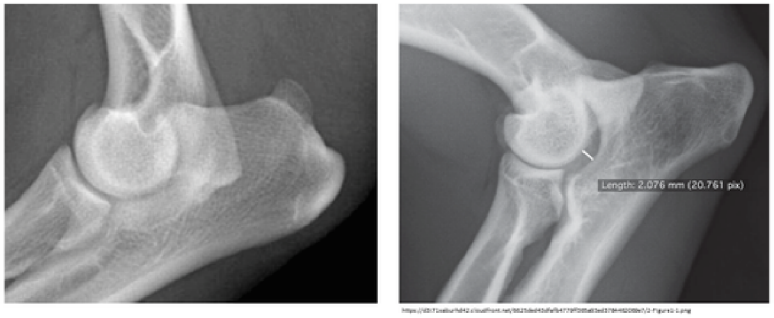
Congruency



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Early detection and OFA grading

- Anconeal process is often the first place for osteoarthritis secondary to dysplastic lesions
- Orthopedic Foundation for Animals (OFA) grade
  - Grade 0: No proliferation on anconeal process
  - Grade 1: Up to 3mm
  - Grade 2: 3-5mm
  - Grade 3: >5mm
- Proliferation on the anconeal process may be a normal variant or enthesopathy in some cases

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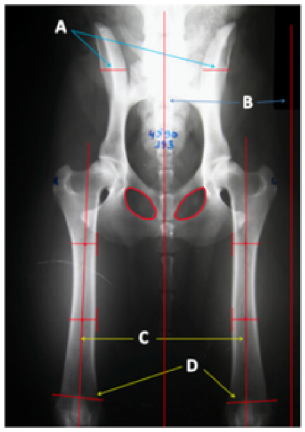
Hip dysplasia - positioning



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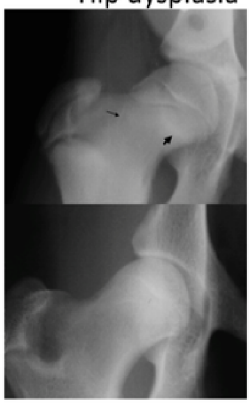
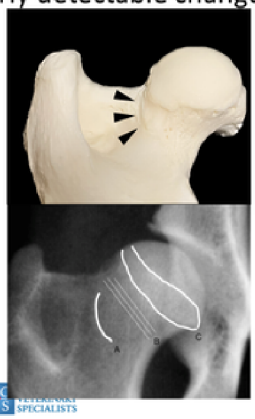
Hip dysplasia – early detectable changes





Caudal joint capsule attachment

- A: Morgan line
- B: Indistinct metaphyseal sclerosis
- C: Circumferential osteophyte

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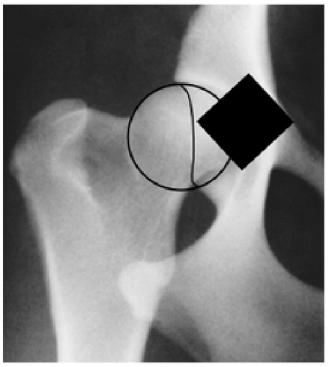
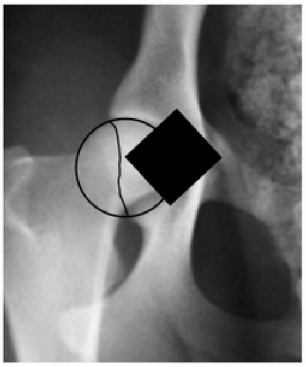


Early hip dysplasia



Normal

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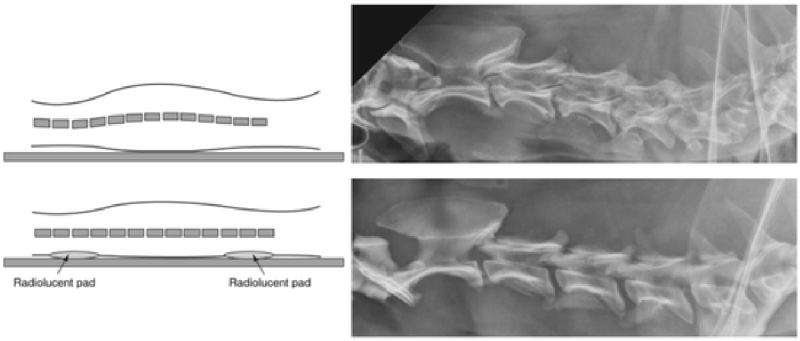
Degenerative changes of the spine - positioning



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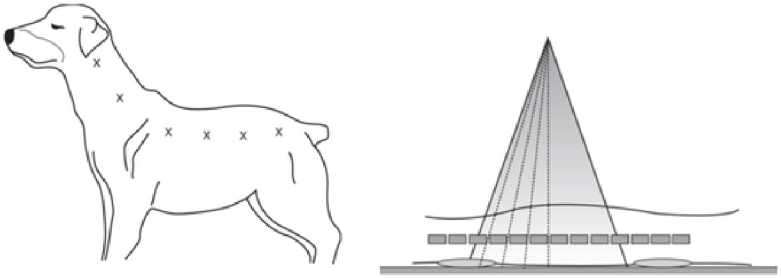
Degenerative changes of the spine - positioning



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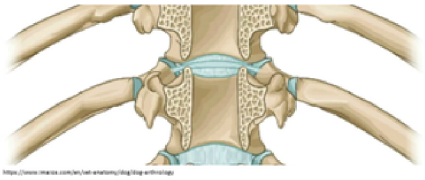
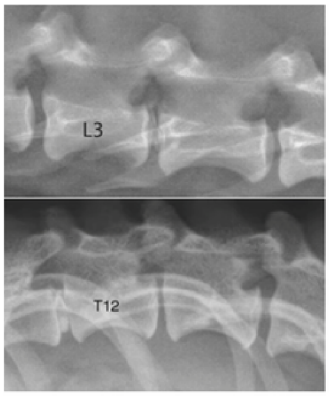
Intervertebral disc disease

- T10-T11 and L5-L6 disc spaces are normally narrower than adjacent disc spaces
- Intercapital ligaments from T2 to T11
- Mineralized disc *in situ* – indicative of degeneration but often incidental finding





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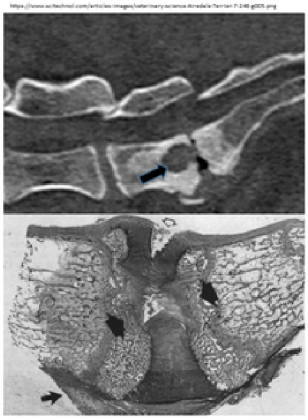
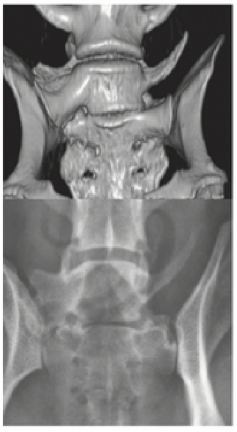
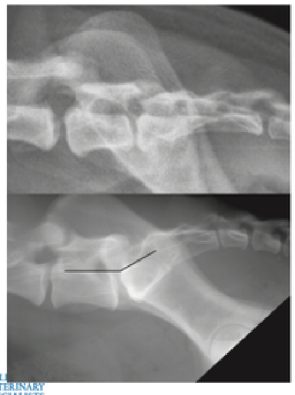
Schmorl's nodes

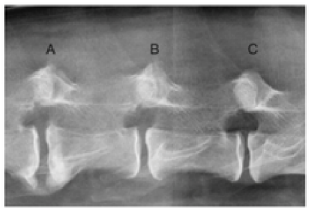


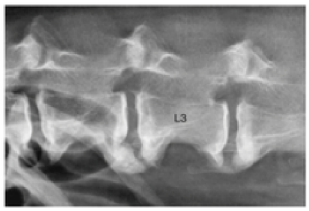


Sacralization of L7

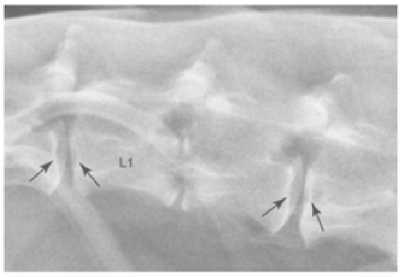


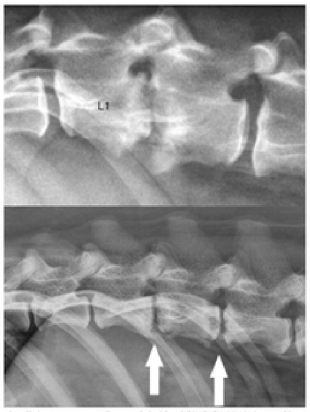
Spondylosis deformans





Discospondylitis





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Cranial Cruciate Ligament Tears

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Fractures

- Make clinical decisions and prognoses with healing complications
- Classified according to:
  - Anatomical location
  - External wounds
  - Extent of bone damage
  - Direction of the fracture line
  - Displacement of fracture fragments
  - Stability

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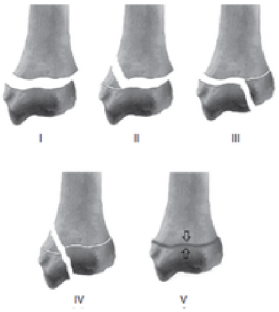
Physeal Fractures



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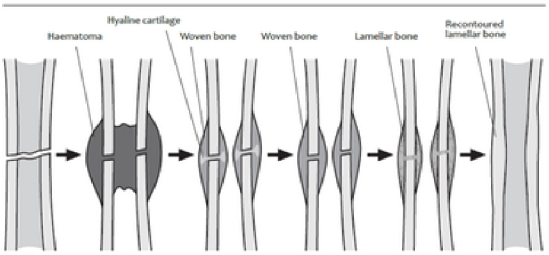
Fracture Healing



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Fracture Healing Complications

- Delayed union
- Non-union
- Malunion
- Implant failure
- Infection

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Fracture Healing Complications

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Thank you.

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GULF COAST VETERINARY SPECIALISTS

EMERGENCY

RHONDA DIXON

DVM

Dr. Rhonda Dixon received her doctor of veterinary medicine degree from Texas A&M University in 1999, having previously graduated with a bachelor's degree in biomedical science and animal science from Texas A&M in 1994. Dr. Dixon has practiced emergency/critical care medicine in the Houston and Sugar Land areas for more than 15 years.

Her special interests include trauma medicine, feline medicine, and endoscopy. Time away from work is spent with her wife, their multiple cats/dogs, and a bird. Outside of work, she enjoys watching football, spending time at her lake house in east Texas, and going off road in her 4WD FJ Cruiser.

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Caring For The Most Critical Patients First: The Art of Triage

Rhonda Dixon, DVM
Gulf Coast Veterinary Specialists
Emergency Department Lead

Triage:

- Derived from the French word "trier"—to sort, pick out or separate from others
- In a medical setting
  - The sorting of and allocation of treatment to patients according to a system of priorities designed to maximize the number of survivors
  - The sorting of patients (as in an emergency room) according to the urgency of their need for care

TRIAGE CLASSIFICATION CHART

Triage Classification	Description	Examples:
First Priority	Patient Critical Treatment must be initiated within seconds to minutes	Cardiopulmonary arrest, Major bleeding, breathing problems, altered mentation, shock, history of toxin ingestion, active seizures, anaphylaxis
Second Priority	Patient Urgent Currently stable, but may become 1st priority patient, need to be reassessed, or have treatment initiated within minutes to hours	History of major trauma, history of unsuccessful urination, repeated vomiting or diarrhea, some toxin ingestions, some fever patients
Third Priority	Patient Stable Pressing problem that is non-critical Treatment initiated within hours	Fever, lacerations, open fractures, vomiting, diarrhea (no shock or altered state of mentation)
Fourth Priority	Patient Completely Stable Needs evaluation, requires action in the next 24+ hours	Minor skin issues, ear infections, most limping patients

The Golden Hour

- First 1+ hours after presentation
- Identify life threatening problems and start treatments to mitigate consequences
- BE PROACTIVE
- ANTICIPATE

PATIENT ASSESSMENT

- Started by technician/triage team
  - Quick history; more thorough history later
  - Obtain vitals
- Concurrent assessment by DVM
- ID life threatening problems/injuries
- Airway, Breathing, Circulation, Neurological

AIRWAY

AIRWAY
- Open? Obstructed?
- INTUBATION—if you are thinking about it, do it.

BREATHING

- Normal vs labored
- Increased vs decreased rate
- Localization of problem—upper airway vs lower airway vs pleural space vs parenchymal
- Look Alikes/Honorable Mentions
- Always OK to give oxygen

RECOGNIZING RESPIRATORY PATTERNS

UPPER AIRWAY:

- Exaggerated or prolonged inspiration effort with little to no air movement
- O2 supplementation is not effective, therefore immediate invasive measures are indicated (sedation/anesthesia, intubation, positive pressure ventilation)
- Emergency tracheostomy may be indicated if intubation not feasible
- Laryngeal paralysis, brachycephalic airway obstructive syndrome, collapsing trachea, foreign body, neoplasia

RECOGNIZING RESPIRATORY PATTERNS

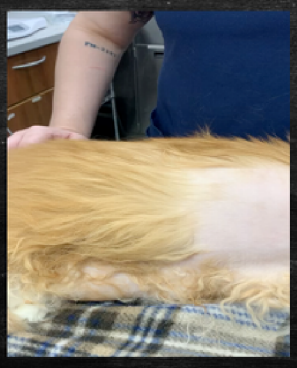
SMALL AIRWAY/LOWER AIRWAY

- Wheezes or whistles may be heard
- Most likely to be a cat vs dog
- Cats will present with a pronounced expiratory push, normothermic and normotensive
- Oxygen supplementation, bronchodilators (albuterol inhaler, terbutaline), steroids, sedation?
- Examples: Feline asthma, chronic bronchitis (dog)

RECOGNIZING RESPIRATORY PATTERNS

PLEURAL SPACE DISEASE

- Asynchronous or restrictive pattern
- Dull or absent lung sounds
- TFAST—fluid vs. air vs. organs vs mixture
- Pleural fluid (pyothorax, chylothorax, hydrothorax, neoplasia)
- Air—pneumothorax (trauma, spontaneous)
- Diaphragmatic hernia—recent trauma?



Recognizing Respiratory Patterns

PARENCHYMAL DISEASE

- Diseases that affect the terminal and respiratory bronchioles, interstitium, alveoli, or vasculature of the lung
- Infiltration with microorganisms, inflammatory or neoplastic cells
- Airspaces may be filled with edema, or foreign material
- Or Lung tissue may be replaced by fibrotic tissue
- Examples: Infectious pneumonia, aspiration pneumonia, pulmonary fibrosis, CHF, NCPE, pulmonary contusions, or neoplasia

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RECOGNIZING RESPIRATORY PATTERNS

LOOK ALIKE/HONORABLE MENTIONS

- Decreased O_2 delivery—-secondary to poor cardiac output
  - Cardiac arrythmias
  - Hypovolemia
  - Pericardial effusion
- Stress/anxiety/pain
- Reverse Sneeze
- Acid-base disturbances—metabolic acidosis (uremia, ketoacidosis, ethylene glycol intoxication)
- Hyperthermia
- Neurological Disease – intracranial diseases affecting brain stem, or cerebral cortex; Cheyne-Stokes, gasping/chaotic respirations, central neurogenic hyperventilation

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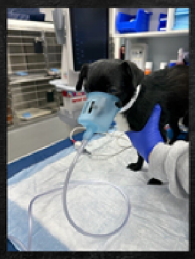
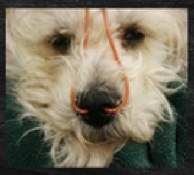
OXYGEN DELIVERY METHODS

	FiO_2 (%)	Flow rate	Advantages	Limitations	Indications
Low flow					
Flow by	25–45	6–8 L/min	▪ Utilizes readily available equipment	▪ Not appropriate for prolonged therapy ▪ Wasteful	▪ Triage and procedures ▪ Initial stabilization
Oxygen cage	21–60		▪ Well tolerated ▪ Allows eating and drinking	▪ Reduced access to patients ▪ FiO_2 rapidly decreases when doors opened ▪ Larger patients	▪ Patients that will not tolerate nasal oxygen or in which nasal oxygen is contraindicated
Face mask	35–55	5–6 L/min	▪ Utilizes readily available equipment ▪ Rebreathing at low rates	▪ Not appropriate for prolonged therapy ▪ FiO_2 depends on fit of mask	▪ Triage and procedures ▪ Initial stabilization ▪ Risk of rebreathing
Nasal prongs		50–150 ml/kg/min	▪ Easy to place ▪ Well tolerated	▪ Poor patient tolerance at high flow rates ▪ Not suitable for some facial conformations	▪ Ongoing oxygen support in hospital
Nasal catheter	30–60	50–150 ml/kg/min	▪ Well tolerated	▪ Poor patient tolerance at high flow rates ▪ Harder to place	▪ Ongoing oxygen support in hospital
High flow					
CPAP	21–100		▪ Reliable FiO_2 ▪ Delivers PEEP ▪ Humidifies inhaled gases	▪ Often requires heavy sedation ▪ Specific equipment	▪ Hypoxaemia despite oxygen support ▪ Upper airway obstruction
HFNOT	21–100	10–60 L/min	▪ Reliable FiO_2 ▪ Delivers PEEP ▪ Humidifies inhaled gases	▪ Specific equipment	▪ Hypoxaemia despite conventional oxygen therapy ▪ Increased work of breathing
Mechanical ventilation	21–100		▪ Reliable FiO_2 ▪ Delivers PEEP ▪ Humidifies inhaled gases	▪ Specific equipment ▪ High complication rate ▪ High cost	▪ Hypoventilation ▪ Hypoxaemia despite oxygen support ▪ Increased work of breathing (fatigue)





High Flow Oxygen Therapy

CIRCULATION

Heartrate

MM color

- Can use vulva or prepuce
- CRT

Pulse Quality

Extremity/body temperature

Blood pressure—not sensitive test for diagnosis of shock

- Compensatory shock—will be normotensive

TABLESIDE DIAGNOSTICS DURING TRIAGE ASSESSMENT

- VPOCUS (AFAST/TFAST)
- BG
- LACTATE
- PCV/TP
- SpO_2
- EKG

Shock vs Dehydration

SHOCK

- Tissue hypoperfusion
  - Hypovolemic (hemorrhage, dehydration, 3rd spacing)
  - Distributive/Vasodilatory (sepsis, SIRS, anaphylaxis)
  - Cardiogenic (CHF, arrythmia)

DEHYDRATION

- Loss of water from interstitial space
- Untreated/unresolved dehydration can lead to shock, but they are not interchangeable

HYPOVOLEMIC SHOCK

- Most common shock seen in dogs/cats
- Dogs tend to have more predictable progressive changes in their cardiovascular status in response to volume depletion
  - heart rate
  - mucous membranes color
  - capillary refill time
  - pulse pressure profile
- Hemorrhage, fluid losses (e.g., from GI tract) or third spacing
- Goal of therapy is to restore perfusion by increasing intravascular volume

STAGES OF HYPOVOLEMIA (DOG)

	Mild (compensatory)	Moderate	Severe (decompensatory)
Heart rate per min	130–150	150–170	170–220
Mucous membrane colour	Normal to pinker than normal	Normal to slightly pale	Grey, muddy, white
Capillary refill time	Vigorous, <1s	Reduced vigour, 1.5s	>2s or absent
Pulse height	Tall	Normal	Short
Pulse duration	Mild decrease	Moderate decrease	Severe decrease
Metatarsal pulse	Easily palpable	Just palpable	Absent
Plasma lactate concentration (mmol/l)	3–5	5–8	>8

**GUIDELINES ONLY

HYPOVOLEMIC SHOCK--CATS

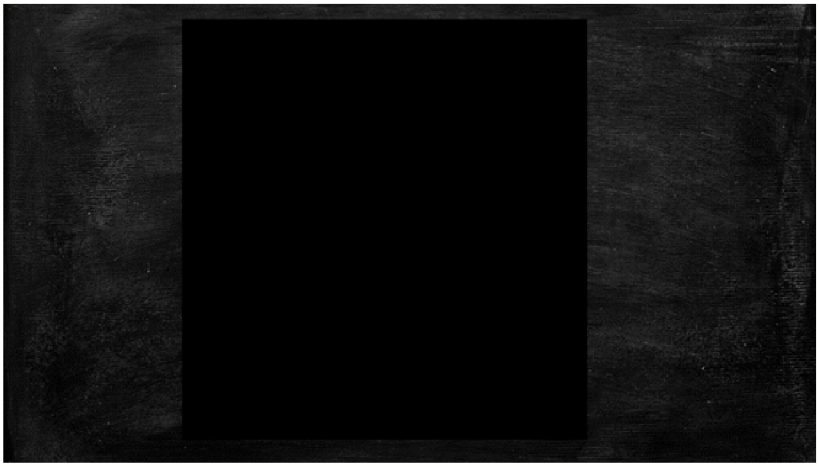
- Do not have same cardiovascular response as dogs
- Not as predictable signs of improvement with resuscitation
- Bradycardia
- Hypothermia
- Slowly warm feline patients AND restore volume

LACTATE MEASUREMENT DURING TRIAGE

- Global hypoperfusion – most common pathological reason for increased lactate
- In general: how severe the hypoperfusion but NOT if reversible or not
- If not normalizing with fluids, start to think of other conditions
  - Under-resuscitation
  - Ongoing cause of hypoperfusion—fluid loss?
  - Increase not due to hypoperfusion—sepsis, SIRS?
- Numerous studies on using lactate as prognostic indicators for sepsis, GDV

CARDIOGENIC SHOCK

- Results in a low-forward flow state due to cardiac derangements
  - Poor myocardial contractility
  - Poor preload
- Examples:
  - DCM
  - HCM
  - Valvular Disease
  - Pericardial Tamponade
  - Severe arrhythmias
- Important to identify because it is NOT treated by fluid resuscitation
  - Patients can look very similar to patients in hypovolemic shock
  - Hallmarks: Murmur, arrythmia, pulmonary edema, ascites, respiratory distress



NEUROLOGICAL STATUS

- MENTATION
  - Alert
  - Obtunded
  - Stuporous
  - Comatose
- MOTOR FUNCTION/GAIT/BALANCE
- CRANIAL NERVES
  - Pupil size—equal vs anisocoria
  - Menace Response
  - Facial sensation

NEUROLOGICAL STATUS

- VESTIBULAR DISEASE
  - Rolling behavior, ataxia, head tilt, nystagmus
- DULL OR COMATOSE: Could indicate serious intracranial disease
- OPISTHOTONUS POSTURE (stiff forelimbs and head extended)-- increased cranial pressures
- NON-AMBULATORY (paresis or paralysis)
  - Trauma?
  - IVDD?
  - Neoplasia of spinal cord

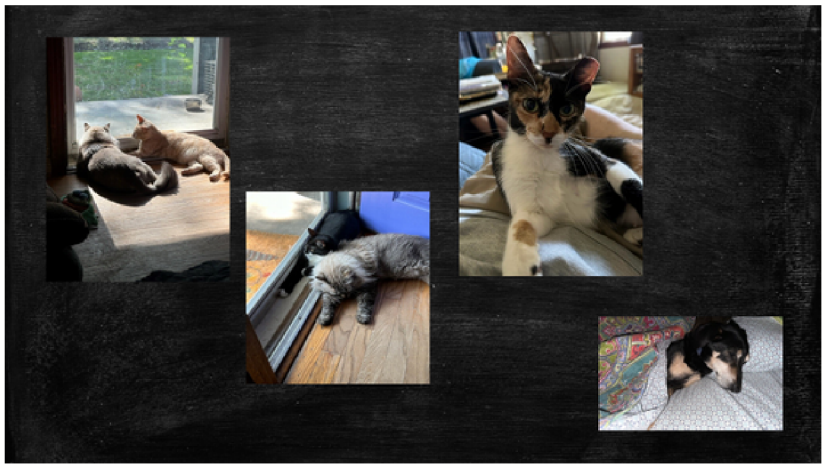
A FEW POINTS TO REMEMBER

During TRIAGE, patients should be re-assessed frequently
- Determine response to treatment
- Monitor for decompensation and need for more emergent intervention

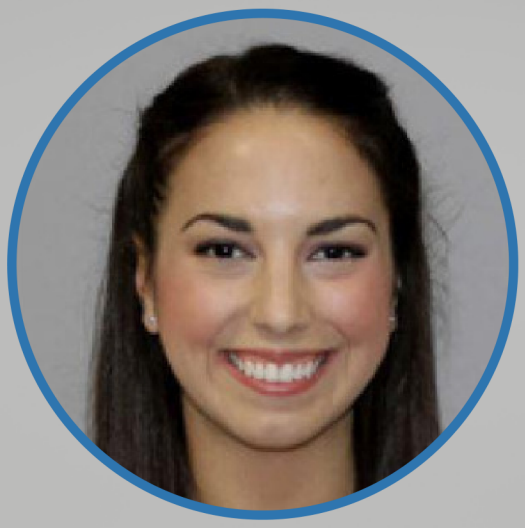
HYPOVOLEMIC SHOCK– Most common in dogs/cats

CARDIOGENIC SHOCK-- Important to identify because it is NOT treated by fluid resuscitation
- Patients can look very similar to patients in hypovolemic shock

SECONDARY ASSESSMENT—more thorough PE after life threatening issues have been addressed



ANESTHESIA



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GULF COAST VETERINARY SPECIALISTS

Pain Management

Katharine A. McIver, DVM, MPH

Pain Management

Objectives

- Pain physiology and pathophysiology
- Acute pain
- Chronic pain
- Pharmacological intervention of pain
- Non-pharmacologic modalities for pain
- Closing thoughts

Pain :

An unpleasant sensory and emotional experience associated with actual or potential damage, or described in terms of such damage

Lumb and Jones et all. Veterinary Anesthesia and Analgesia 5th Edition. Ames Iowa, 2015

Nociception :
Neuronal process of encoding noxious stimuli

Lumb and Jones et all. Veterinary Anesthesia and Analgesia 5th Edition. Ames Iowa, 2015

Physiology

Types of Pain

- **Temporal**
  - Acute vs chronic
- **Physiological**
  - Peripheral vs central
  - Inflammatory vs neuropathies
    - Physical damage vs nerve damage
- **Somatic vs visceral**
  - Well localized vs poorly localized

Physiology

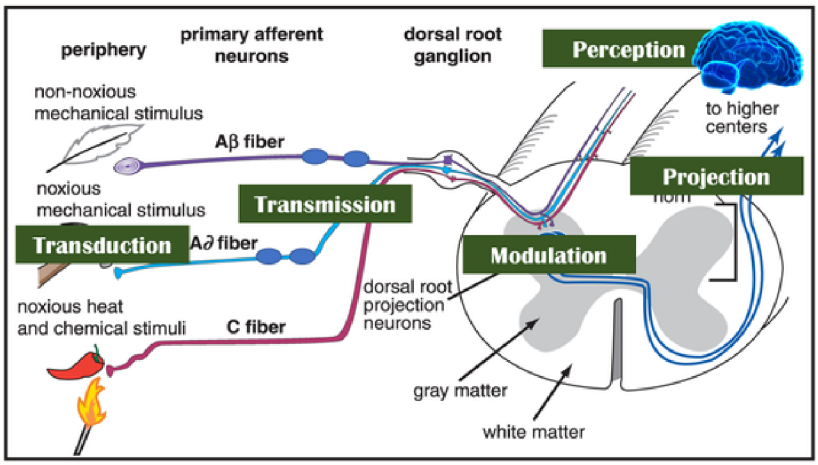
Nociceptors

- **Free endings on primary sensory neurons**
- **Detect signals from damaged tissue**
- **Found in skin, muscle, joints, bones, and viscera**
  - Not present in the CNS
- **Several categories of nociceptors**
  - Mechanical
  - Thermal
  - Chemical
  - Polymodal
  - Silent

Physiology

Nociceptors

- Several factors activate nociceptors
  - Damaged tissues release a variety or substances from lysed cells
  - New substances are made at the site of injury
- Nociception has a minimum threshold and is not an all or none
  - Increased stimulation = increased sensation of pain
- The release of local inflammatory factors can cause sensitization



Physiology

Acute Pain

- Acute pain is caused from the following:
  - Surgery, traumatic injury, tissue damage, and inflammatory processes
- Useful biological purpose with a rapid onset
- Can be self-limiting and should be resolved ≤ 3 months
- Treatment of acute pain:
  - Interrupting nociceptive signals and treating the underlying cause
- Inadequate controlled of acute pain can be a factor in the development of chronic pain

Physiology

Chronic Pain

- Pain that is present for >6 month
- Pain that continues after the initial injury or illness has healed
- Chronic pain can be considered a disease
- Chronic pain has no biological purpose
- Requires a multidisciplinary approach
- Often time requires more than one therapeutic modality
- Complex and frustrating

Pharmacological Treatment

- Alpha- 2 adrenergic receptor agonist
- Local anesthetics
- N-Methyl-D-aspartate receptor antagonist (NMDA)
- Non-steroidal ant-inflammatory drugs (NSAIDs)
- Opioids
- Adjunctive analgesics
  - Gabapentin
  - Neurokinin (NK)-1 receptor antagonists
  - Cannabis
  - Others

Pharmacological Treatment

Alpha-2 Agonist

- Wide range of effects on the body
  - Cardiovascular, respiratory, central nervous system, endocrine, GI, urogenital
- Dexmedetomidine is alpha-2 agonist of choice
- Site of analgesic activity
  - Peripherally
  - Spinally and supraspinal
    - Induces action via the presynaptic terminal of primary afferent neurons
    - Direct inhibition of spinal cord neurons

Pharmacological Treatment

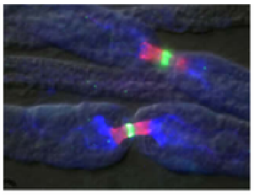
Dexmedetomidine

- Pre-medication 0.5-2.0 mcg/kg IV or IM
- Post-operative analgesia 0.5-2.0 mcg/kg IV
- Can run as a CRI
  - 0.5-2.0 mcg/kg/hr
- Reversible drug
  - Will reverse the analgesia
- Works synergistically with opioids
- Caution in patients with decreased cardiovascular function

Pharmacological Treatment

Local Anesthetics

- Prevents nociceptive transmission
- Can produce dose dependent anti-inflammatory effects
- May reduce free radical formation
- Good for visceral pain

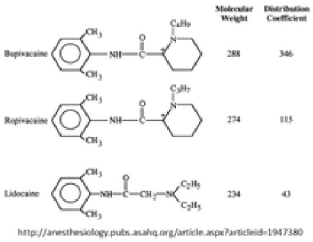


https://www.bcm.edu/research/labs/matthew-rasband

Pharmacological Treatment

Local Anesthetics

- Lidocaine
  - Loading dose 2mg/kg
  - CRI 25-35 mcg/kg/min
  - Nerve block 1-2mg/kg
- Bupivacaine/ Ropivacaine
  - Peripheral nerve block 1-2mg/kg
  - Epidural 0.8mg/kg
  - Never give IV
- Local anesthetic doses are additive
- Toxic effects seen at 8mg/kg in dogs and 6mg/kg in cats



http://anesthesiology.pubs.asahq.org/article.aspx?articleid=1947380

Pharmacological Treatment

Local Anesthetics

- **Liposomal Bupivacaine→ Nocita®**
  - Up to 72 hours of pain control
  - Dose is 5.3mg/kg
  - Can dilute with NaCl 0.9%
  - FDA approved
    - TPLO in dogs
    - Onychectomy in cats

Pharmacological Treatment

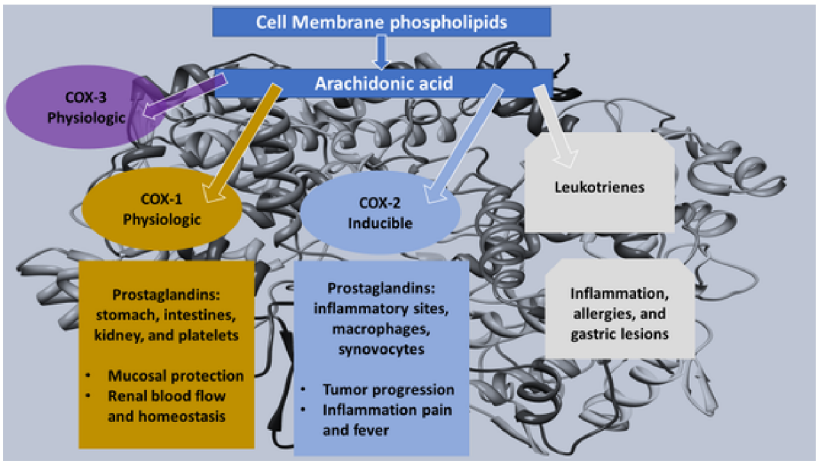
N-Methyl-D-aspartate receptor antagonist

- **Ketamine**
  - Somatic pain >> visceral
  - Adjunctive analgesia
  - Loading dose 1-2 mg/kg
  - CRI 0.5-0.8 mg/kg/hr
  - Potential side effects

Pharmacological Treatment

N-Methyl-D-aspartate receptor antagonist

- **Amantadine**
  - Oral NMDA antagonist
  - Can be helpful with chronic pain
  - Can take up to 21 days to be effective
  - Dogs and cats 3-5mg/kg PO q. 12
- **Methadone**
  - Opioid with NMDA antagonist properties
  - Works synergistically with other drugs
  - Less hyperthermia in cats
  - More consistent sedation when compared to hydromorphone



Pharmacological Treatment

Non-Steroidal Anti-Inflammatory

- Decrease COX enzyme conversion
  - Mild reduction of central pain transmission
- Non-controlled substance
- Oral and injectable forms
- Standard medication after elective procedures
- Avoid in patients with:
  - Dehydration, hypovolemia, and hypotension
  - Certain renal, hepatic, and cardiovascular insufficiency
- Use with caution in patients:
  - Receiving corticosteroids, aminoglycosides, and polymixins , furosemide, and ACEi

Pharmacological Treatment

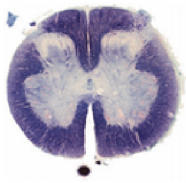
Acetaminophen

- Potential central mediation of COX-3
- Possible activation of serotonin receptors
- K9 studies does not produce anti-inflammatory but, is an effective analgesic
- Can cause liver toxicities therefore monitor liver values
- Often combined with codeine
- Dogs 15mg/kg PO q. 8
- DO NOT USE IN CATS

Pharmacological Treatment

Opioid Receptors

- Types
  - Mu
  - Kappa
  - Delta
- Receptors located
  - Peripherally
  - Spinally
  - Supraspinal
- Certain peripheral tissues can generate receptors
  - Articular
  - Periarticular
  - Corneal tissue



Pharmacological Treatment

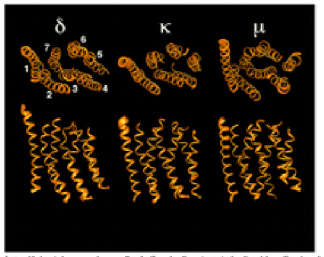
Opioids

- Caution in patients with head trauma
- Various interactions between opioids
- Interaction with drug clearance
  - Rely more on hepatic blood flow
- Interactions with other analgesics and antidepressants
  - Potential interaction with selegiline, amitraz, tramadol, and trazadone

Pharmacological Treatment

Opioid Pharmacodynamics

- Analgesia
- Sedation and or excitement
- Antitussive
- Respiratory depression
- Cardiovascular effects
- Nausea, emetic, and antiemetic effects
- Pupil diameter
- Decreased GI motility
- Urinary retention
- Thermoregulatory effects
- Immunomodulator effects
- Reduction in MAC



http://physiology.med.cornell.edu/faculty/hweinstein/online/dan_fluc.html

Pharmacological Treatment

Pure Mu Opioids

- **Morphine**
  - 2-4 hour intra-op duration
  - Administer slowly IV (histamine release)
- **Hydromorphone**
  - 1-2 hour intra-op duration
- **Methadone**
  - 2-4 hour intra-op duration
  - NMDA antagonist activity
  - Least likely to cause vomiting
- **Fentanyl – Remifentanil**
  - 15 – 30 min and 6 min duration respectively
  - Usually administered as a CRI

★ Different Potency but Equal Efficacy!

Pharmacological Treatment

Mixed Opioids

- **Buprenorphine**
  - ~ 45 min intra-op duration
  - Partial mu agonist
  - Preferential binding
  - "Ceiling effect"
- **Butorphanol**
  - ~ 30-60 min duration
  - Kappa agonist and mu antagonist
  - May be used to reverse pure mu opioid incase of overdose

Pharmacological Treatment

Reversal

- **Naloxone**
  - Used to reverse all opioids
  - ~ 30- 60 min duration
    - Redosing may be necessary in long-acting opioids
  - May be used incases of opioid dysphoria
  - Administer via titration to avoid acute pain
    - 0.02 mg diluted into 5 ml sterile saline
    - Administer in 1 ml increments until desired effect achieved

Pharmacological Treatment

Oral Opioids for post-op pain

- **Codeine**
  - Low oral bioavailability
- **Tylenol 3**
  - Acetaminophen + codeine
  - Schedule III drug
- **Tramadol**
  - Low efficacy in dogs and horses
  - Good efficacy in cats – tastes bitter

Pharmacological Treatment

Gabapentin

- **Structurally similar to inhibitory neurotransmitter GABA**
- **Not an agonist and does not effect GABA uptake or degradation**
- **Presynaptic inhibition of calcium channels**
- **Neuropathic pain treatment**
- **Great adjuvant with NSAID**
- **Can take several days to establish effectiveness**
- **Dogs 10-20 mg/kg PO q. 6-8 hr**
  - Need frequent dosing due to short half life
- **Cats 5-10 mg/kg PO q. 8-12 hr**

Pharmacological Treatment

Maropitant

- **Neurokinin (NK)-1 Receptor Antagonist**
- **Blockade of substance P**
- **NK-1 receptors have been identified in the CNS and PNS**

Pharmacological Treatment

Other Non-opioid Options

- Grapiprant (Galliprant®)
  - Prostaglandin E2 receptor antagonist
- Corticosteroids
- Tricyclic antidepressants
  - Amitriptyline
- Bisphosphonates
  - Bone cancer
- Nutraceuticals
- Polysulfated glycosaminoglycan
- Magnesium Sulfate

Pharmacokinetics, Safety, and Clinical Efficacy of Cannabidiol Treatment in Osteoarthritic Dogs

Lauri-Jo Gamble, Jordyn M. Boesch, Christopher W. Frye, Wayne S. Schwark, Sabine Mann, Lisa Wolfe, Holly Brown, Erin S. Berthelsen and Joseph J. Wakshlag

Front. Vet. Sci., 23 July 2018

Pharmacological Treatment

Cannabidiol

- Cannabidiol (CBD) is now being used for pain management
- Tetrahydrocannabinol (THC) is a class I substance that is found in marijuana
- CBD is an extract of the hemp and has THC levels < 0.3%
- CBD affects the endocannabinoid receptors
  - Located peripheral and central
  - The receptors are important toward hemostasis
- Other known actions include downregulation of anxiety, noise phobia, epilepsy, inflammation, emesis, and anorexia

Pharmacological Treatment

Cannabidiol

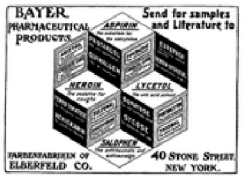
- Difficult to know the purity of products and exact CBD concentrations
- Peak Performance Veterinary Group – Colorado
- CBD concentration 100mg/mL
  - Has additional cannabidiol substances
  - www.peakvets.com
- Google CHO pet
- Current dose recommendations 0.02 - 0.1mg/kg PO q. 12
  - Half life is about 200 minutes
- Has caused increases in ALP
- Seems oil based products have better pharmacokinetics

Non-Pharmacological Pain management

- Weight optimization
- Acupuncture
- Physical rehabilitation
- Thermal modification
- Environmental modifications
- Chiropractic care
- Homeopathy

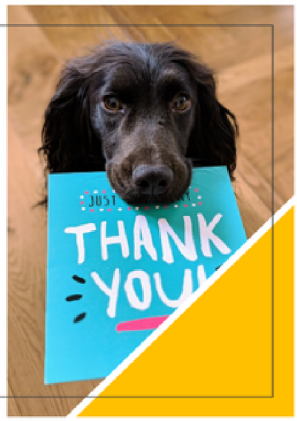
Closing Thoughts

- Preparation for addressing pain : surgical or trauma
  - Can I sedate this patient?
  - Can I give an NSAID?
  - Can I provide a peripheral nerve block and or an epidural?
- Choose an appropriate analgesia
  - Bolus or CRI
- Additional analgesic CRIs
  - Lidocaine
  - Ketamine
  - Dexmedetomidine
  - Additional opioid



Special Thanks

- Rebecca S. Salazar DVM, DACVAA
- GCVS
- NVA Compassion First



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Questions?

Please feel free to reach out to kam0174@auburn.edu for further questions

SURGERY

NATHAN SQUIRE

VMD, MS, DACVS-SA

Dr. Nathan Squire obtained his Veterinariae Medicinae Doctoris (VMD) from the University of Pennsylvania in 2017, before traveling to Tufts University for a rotating internship in medicine and surgery. From there, he headed south to the Veterinary Orthopedic and Sports Medicine Group for a surgical internship, before heading south yet again to the University of Tennessee for a 3-year residency in small animal surgery, during which he also earned his Master of Science degree.

Dr. Squire's surgical interests include minimally invasive surgery (including soft tissue and orthopedic procedures), fracture repair, and joint replacement.

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Patellar Luxation in Dogs

Nathan Squire, VMD, MS, DACVS-SA
Small Animal Surgery

GULF COAST VETERINARY SPECIALISTS

Objectives

- Anatomy review
  - Quadriceps mechanism
- Predisposing factors
- Clinical signs and diagnostics
- Management
  - Surgical vs conservative
- Complications

GULF COAST VETERINARY SPECIALISTS

Objectives

- **Anatomy review**
  - **Quadriceps mechanism**
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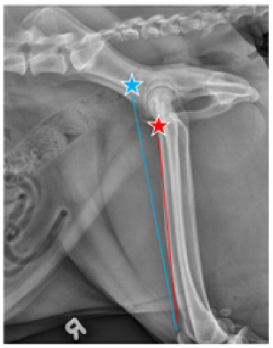
GULF COAST VETERINARY SPECIALISTS



Quadriceps mechanism

- Quadriceps = "four headed"
  - Rectus femoris – originates on ventral ilium
  - Vastus medialis, lateralis, and intermedius – originate on proximal femur
- Common tendinous insertion on patella

Quadriceps mechanism

- Patellar tendon
- Patella
  - Sesamoid bone within tendon of insertion
- Patellar ligament
- Tibial tuberosity
  - Insertion composed of Sharpey's fibers
  - Separate ossification center in juveniles (closure typically at 10-12 months)

Quadriceps mechanism

- Trochlear groove of the femur
  - Area between femoral condyles in which patella should glide
  - Covered by articular cartilage
- Tibial tuberosity

Objectives

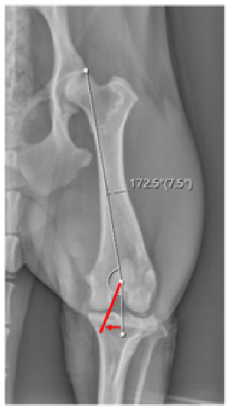
- Anatomy review
  - Quadriceps mechanism
- **Predisposing factors**
- Clinical signs and diagnostics
- Management
  - Surgical vs conservative
- Complications

Developmental condition

- Patellar luxation typically *not* present at birth, but predisposing factors often *are*
- Medial patellar luxation is the most common form, regardless of dog size[1]
  - Medial, lateral, or bi-directional possible

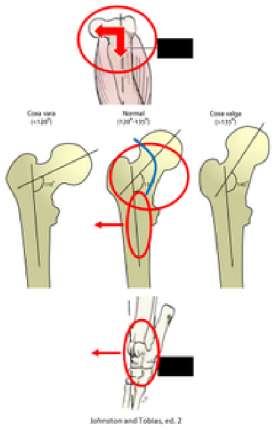
"Q-angle"

- Quantitative measure of deviation of quadriceps mechanism
- Directly related to horizontal force of quadriceps on patella
- In unaffected dogs, average reported is 10.5° (+/- 5.6°)[1]
- Increase in Q angle can result in *medial* patellar subluxation or luxation[2]



What causes increased Q-angle?

- Coxa vara
- Distal femoral varus
- Medial femoral condylar hypoplasia
- Internal rotation of the tibia
- Medial displacement of the tibial tuberosity
- Internal rotation of the hock



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Development

- During skeletal growth, trochlear groove develops secondary to pressure from patella
- Patellar luxation can lead to shallow/absent trochlear groove and hypoplasia of one or both of the femoral trochlear ridges
  - Predisposes to further or more severe luxation

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Traumatic patellar luxation

- Can also occur secondary to trauma – much less common
  - Frequently entails damage to medial or lateral parapatellar joint capsule/fascia
  - Reported to occur in 18% of cases of patellar luxation[3]

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Objectives

- Anatomy review
  - Quadriceps mechanism
- Predisposing factors
- **Clinical signs and diagnostics**
- Management
  - Surgical vs conservative
- Complications

Clinical signs

- Highly dependent on *grade* of luxation…

Grading system

- Grade I
  - Patella can be manually luxated with stifle in extension
  - Bony deformities not present
- Clinical signs:
  - Typically absent
- "In-in"

Grading system

- Grade II
  - Patella luxates spontaneously during range of motion, but also spontaneously reduces
  - Mild skeletal abnormalities may be present
    - Internal rotation of tibia
  - May progress to grade III as trochlear ridge is worn down
- Clinical signs:
  - Often a non-painful, "skipping" gait in one or both hindlimb(s)
  - +/- crepitus during stifle ROM
- "In-out"

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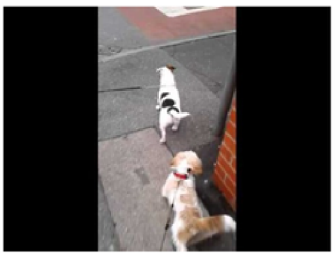
Grade II



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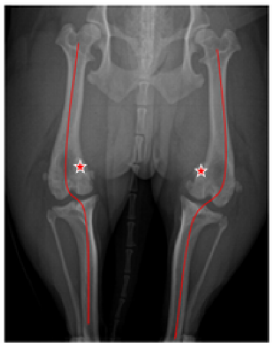

Grading system

- Grade III
  - Patella is luxated from trochlear groove, but can be manually reduced
  - More significant bony abnormalities
    - Internal tibial rotation, "S" shape of distal femur and proximal tibia
- Clinical signs:
  - Intermittent hindlimb lameness (mild to severe) possible
  - "Crouched" gait may be noted by owner if medial luxation
  - "Knock-kneed" appearance with LPL
- "Out-in"

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Grading system

- Grade IV
  - Permanent, non-reducible patellar luxation
  - If not corrected early, will lead to severe skeletal abnormalities, making repair challenging
  - Tibia often rotated 60-90 degrees
- Clinical signs:
  - Similar to grade III luxations
  - More likely to have persistent gait changes, such as "crouched" stance
- "Out-out"

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Grade IV MPL

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Acute onset lameness?

- **CHECK FOR CCL RUPTURE!!**
  - Also consider potential for traumatic patellar luxation
    - May be accompanied by asymmetric subcutaneous swelling
  - Concurrent CCLR present in 15-20% of mature dogs with MPL[4]
- **When assessing for tibial thrust or cranial drawer, important to reduce patella first
- Stifle effusion often more severe with CCLR +/- meniscal injury

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Orthopedic Exam

- Performed with animal standing, then in lateral
- Can trace patellar ligament from tibial tuberosity to isolate patella
- MPL typically demonstrated by:
  - Internally rotating tibia
  - Flexing stifle – often a "popping" sensation palpated
  - Extending stifle
- Palpation of trochlear groove to note depth/erosion
- Check for tibial thrust and cranial drawer

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Diagnostics

- Orthogonal stifle radiographs sufficient for low-grade luxations
  - Patella may reduce spontaneously during positioning
- Higher grade (III or IV) with concurrent skeletal abnormalities:
  - Consider independent, orthogonal views of femur and tibia
  - CT scan with 3-D reconstruction highly useful for angular deformities and surgical planning

Brower et al, 2016

GULF COAST VETERINARY SPECIALISTS

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- Anatomy review
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- Clinical signs and diagnostics
- **Management**
  - **Surgical vs conservative**
- Complications

GULF COAST VETERINARY SPECIALISTS

To cut or not to cut?

- Grade I
  - Surgery not indicated
  - Continue to monitor for clinical signs, reevaluate if noted
- Grade II?
  - Degree/frequency of lameness, progression of OA, $$ commitment
- Grade III or IV
  - Early surgical intervention indicated to prevent progressive skeletal abnormalities and osteoarthritis
- Concurrent stifle pathology (e.g. CCLR)
- Immature patients?
  - Staged procedures possible, with early interventions aimed at sparing physes

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Conservative management

- Weight loss
- Joint supplements
  - Glucosamine and chondroitin sulfate
  - Omega-3 fatty acids
- +/- analgesics as needed
  - NSAID (Carprofen, meloxicam, Onsior), gabapentin
  - Methocarbamol if muscle spasm suspected
- Can consider joint injections in later stages of refractory OA

GULF COAST VETERINARY SPECIALISTS

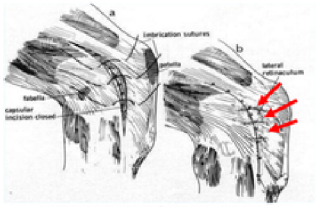
Surgical management

- Goals:
  - Restore alignment of quadriceps mechanism
  - Restore (or create) functional trochlear groove of femur
- Typically entails both bony and soft tissue reconstruction

GULF COAST VETERINARY SPECIALISTS

Soft Tissue Reconstruction

- **Soft tissue reconstruction**:
  - Joint capsule imbrication +/- capsulectomy – performed on side opposite luxation
  - Retinacular release or partial capsulotomy – performed on side of luxation
- Goal is to alter tension of soft tissues such that patellar luxation is less likely
- Rarely used as lone corrective procedures
  - Exceptions: traumatic luxations, juvenile patients



https://www.slideshare.net/management-of-ruptured-cruciate-ligament-in-dogs-29548715

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Tibial Tuberosity Transposition (TTT)

- Tibial tuberosity osteotomized, translated laterally or medially (opposite side of luxation)
- Secured in new position using K-wires +/- tension band wire
  - Recovery period ~6-8 weeks
  - Tension band wire results in significantly stronger construct[7]

Rossanese et al 2019

GULF COAST VETERINARY SPECIALISTS

Trochl

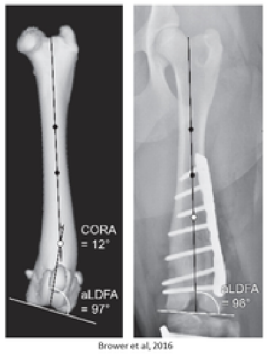
- Troch
  remov
- Articu
- Press-
  patell
  opera
- (Patel

https://www.kyon.ch/knowledge-solutions/vet-patellar-groove-replacement/

GULF COAST VETERINARY SPECIALISTS

Femoral or Tibial Corrective Osteotomies

- Excess femoral varus drawing more attention as underlying cause for patellar luxation[5]
  - Proposed as cause for re-luxation in large breeds
- Torsional deformities
  - Best recognized with CT[6]
- Pre-surgical planning important for accurate corrective osteotomy



Brower et al, 2016

GULF COAST VETERINARY SPECIALISTS

Objectives

- Anatomy review
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- Predisposing factors
- Clinical signs and diagnostics
- Management
  - Surgical vs conservative
- **Complications**

GULF COAST VETERINARY SPECIALISTS

Complications

- Complication rates reported between 18-37%[5,8-10]
  - Majority considered minor
- Most common complications:
  - Re-luxation of patella (0-48.1%[5,8-10])
    - Often grade I re-luxation, may not require further surgical intervention
  - Implant-associated complications (particularly pins for TTT):
    - Infection
    - Seroma
    - Migration
  - Delayed union of osteotomy sites (2-5%[5,10])

GULF COAST VETERINARY SPECIALISTS

Take-Home Points

- Grade I – surgery not indicated
- Grade III + IV – early surgical intervention indicated to prevent skeletal changes and minimize progression of OA
  - Juvenile patients may benefit from staged procedures
- Acute onset lameness MUST raise suspicion for CCL rupture!
- High grade luxations require recognition of all skeletal abnormalities, including angular/torsional deformities of femur and/or tibia (separate radiographs, CT scan)
- Complication rate variable depending on severity
  - Most dogs have acceptable function post-operatively

GULF COAST VETERINARY SPECIALISTS

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