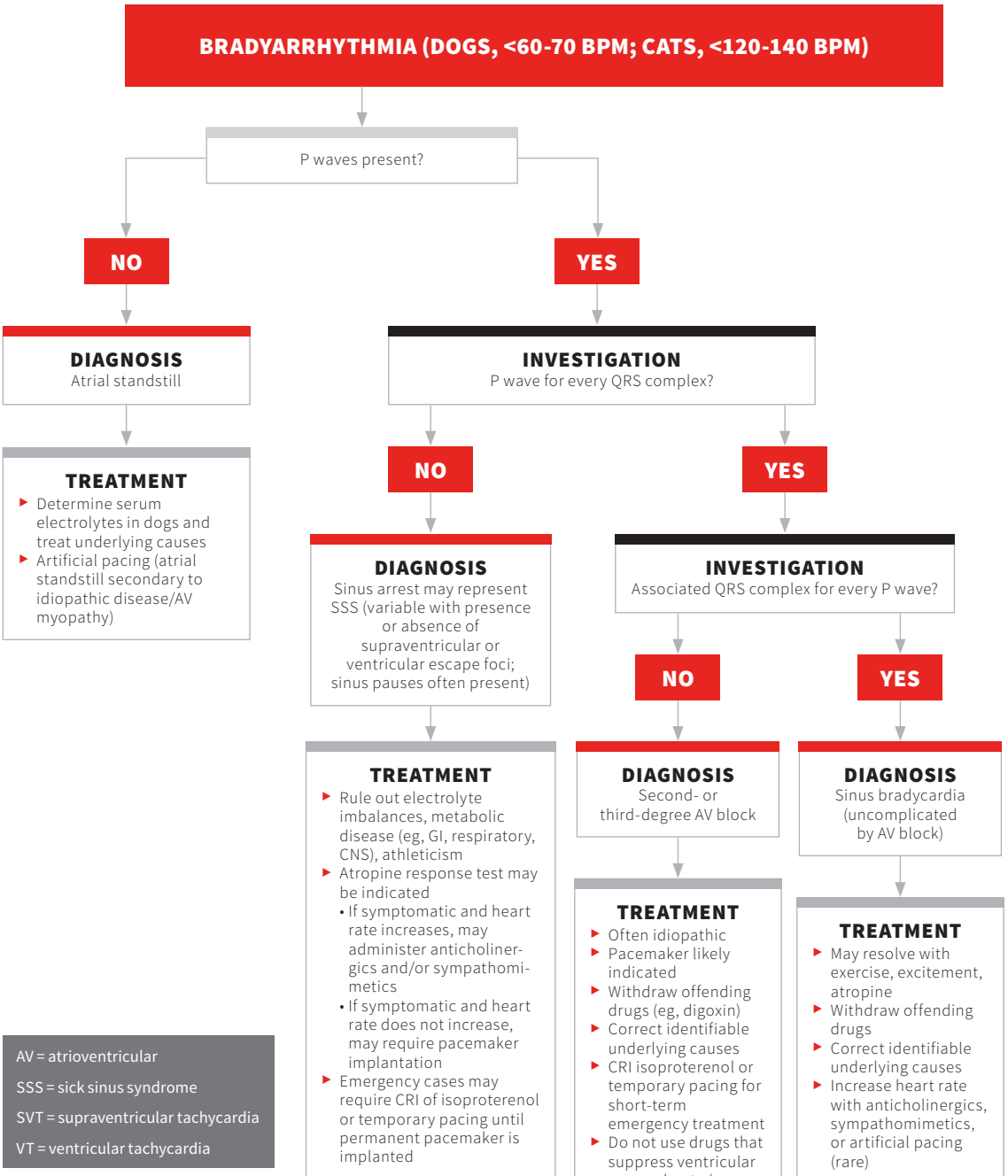
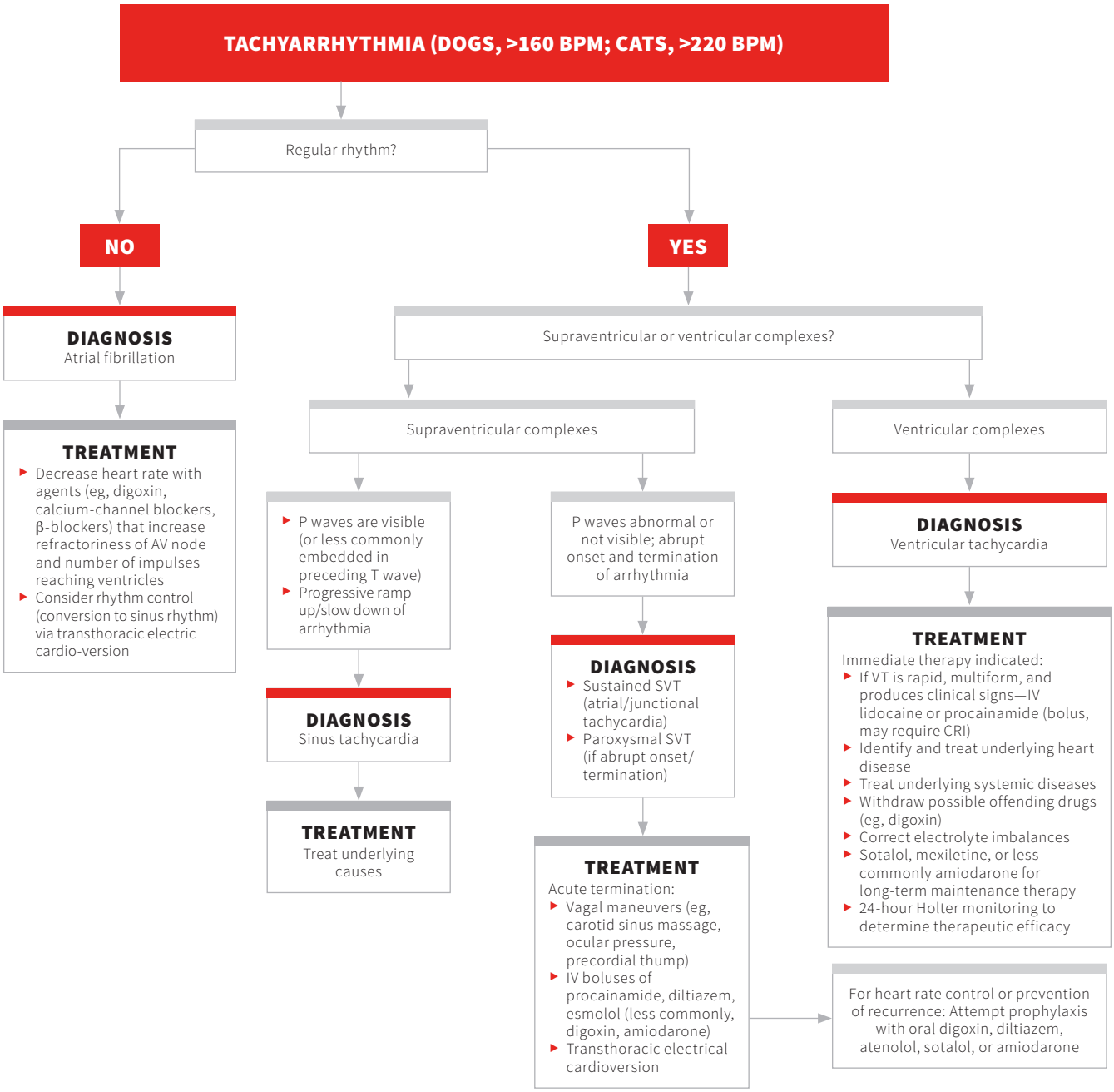
BRADYARRHYTHMIA & TACHYARRHYTHMIA

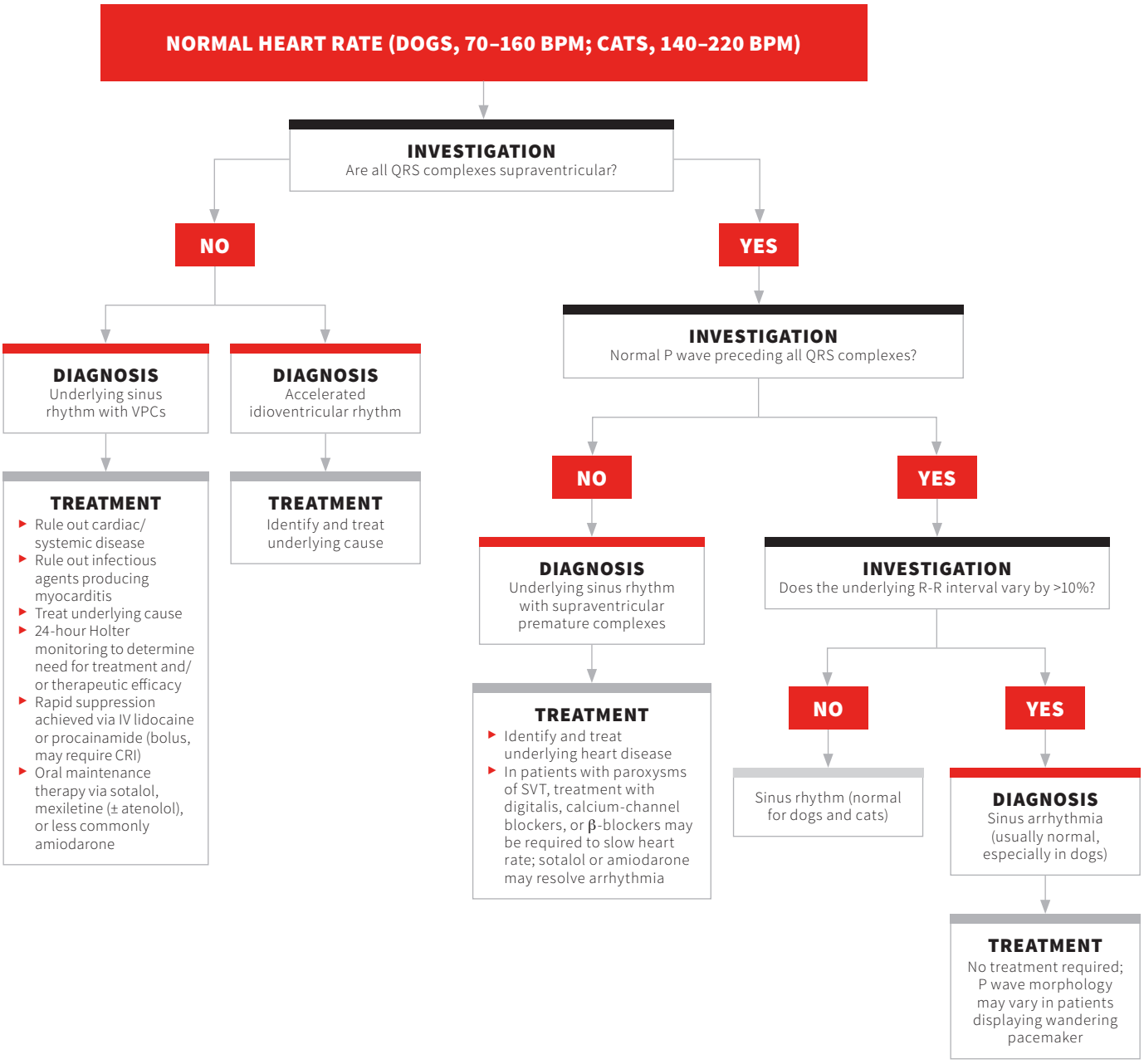
Barret Bulmer, DVM, MS, DACVIM (Cardiology)
Cummings School of Veterinary Medicine at Tufts University




TACHYARRHYTHMIA (DOGS, >160 BPM; CATS, >220 BPM)
Regular rhythm?
NO
YES
DIAGNOSIS
Atrial fibrillation
TREATMENT
Decrease heart rate with agents (eg, digoxin, calcium-channel blockers, β-blockers) that increase refractoriness of AV node and number of impulses reaching ventricles
Consider rhythm control (conversion to sinus rhythm) via transthoracic electric cardio-version
Supraventricular or ventricular complexes?
Supraventricular complexes
Ventricular complexes
P waves are visible (or less commonly embedded in preceding T wave)
Progressive ramp up/slow down of arrhythmia
DIAGNOSIS
Sinus tachycardia
TREATMENT
Treat underlying causes
P waves abnormal or not visible; abrupt onset and termination of arrhythmia
DIAGNOSIS
Sustained SVT (atrial/junctional tachycardia)
Paroxysmal SVT (if abrupt onset/termination)
TREATMENT
Acute termination:
Vagal maneuvers (eg, carotid sinus massage, ocular pressure, precordial thump)
IV boluses of procainamide, diltiazem, esmolol (less commonly, digoxin, amiodarone)
Transthoracic electrical cardioversion
DIAGNOSIS
Ventricular tachycardia
TREATMENT
Immediate therapy indicated:
If VT is rapid, multiform, and produces clinical signs—IV lidocaine or procainamide (bolus, may require CRI)
Identify and treat underlying heart disease
Treat underlying systemic diseases
Withdraw possible offending drugs (eg, digoxin)
Correct electrolyte imbalances
Sotalol, mexiletine, or less commonly amiodarone for long-term maintenance therapy
24-hour Holter monitoring to determine therapeutic efficacy
For heart rate control or prevention of recurrence: Attempt prophylaxis with oral digoxin, diltiazem, atenolol, sotalol, or amiodarone

COMMON CARDIAC ARRHYTHMIAS

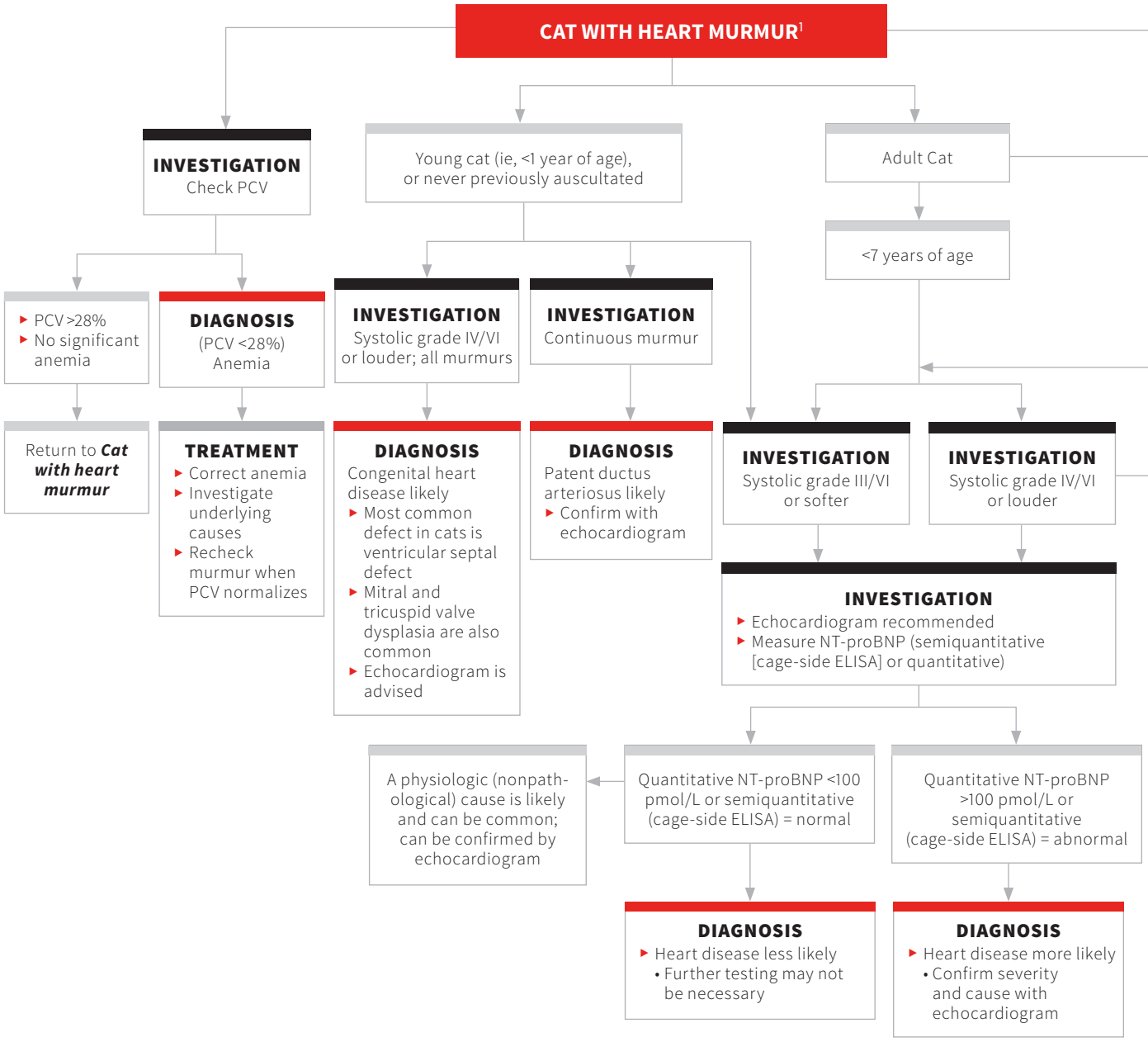
Barret Bulmer, DVM, MS, DACVIM (Cardiology)
Cummings School of Veterinary Medicine at Tufts University



SVT = supraventricular tachycardia
VPC = ventricular premature complex

HEART MURMURS IN CATS

Brian A. Scansen, DVM, MS, DACVIM (Cardiology)*
The Ohio State University



NT-proBNP = N-terminal pro-B type natriuretic peptide
PCV = packed cell volume
T4 = thyroid hormone assay

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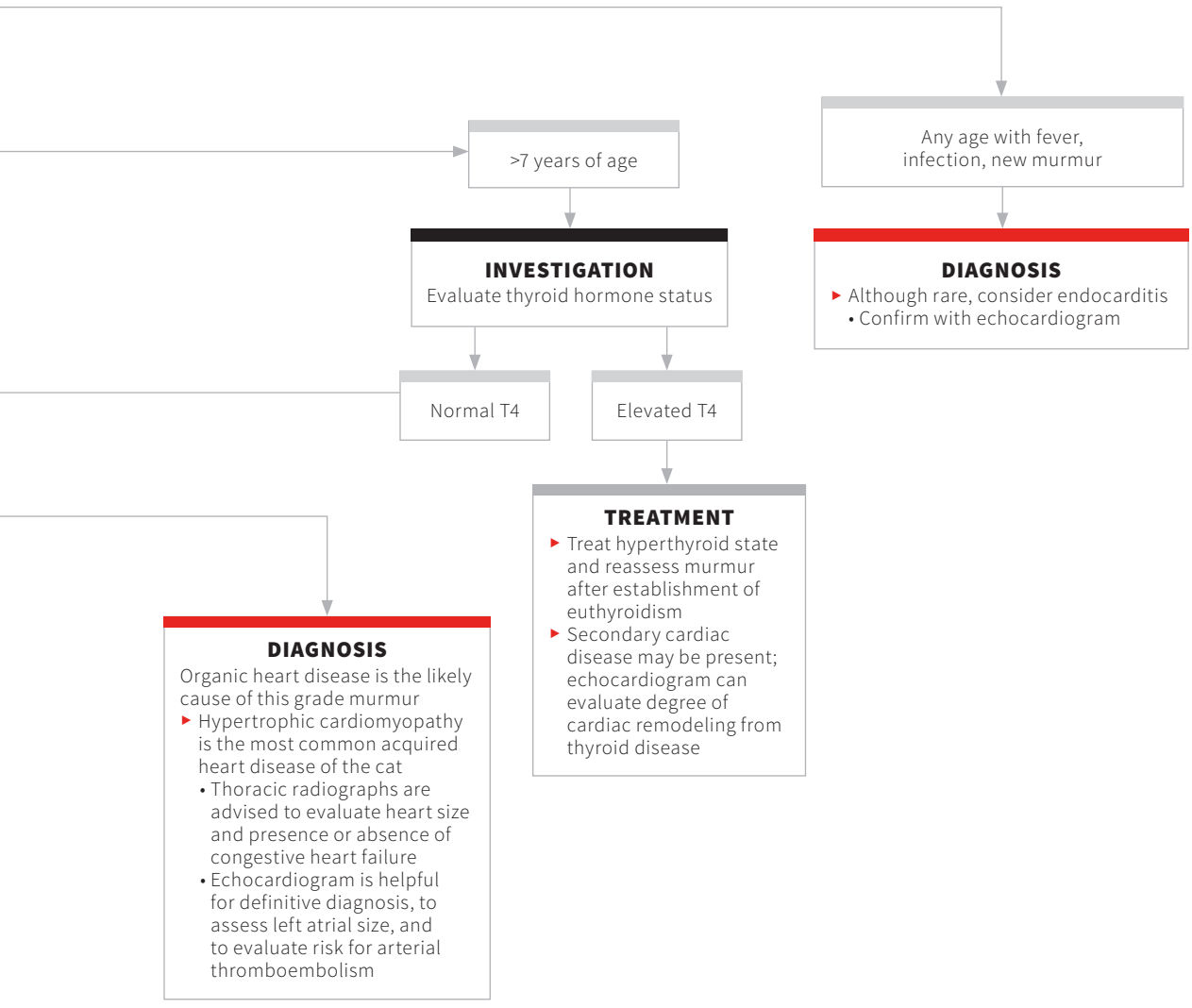
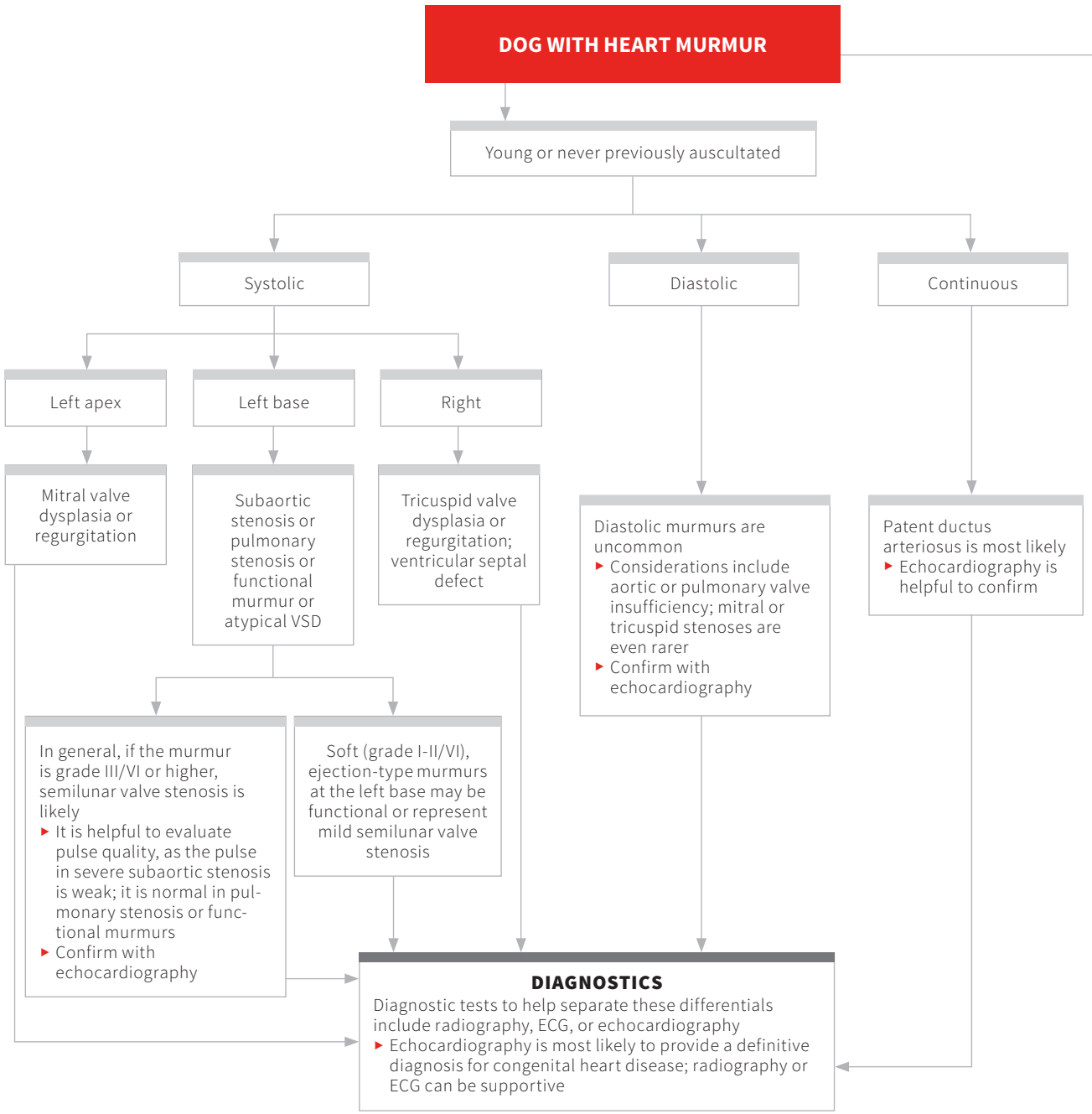


Reference

1. Côté E, Edwards NJ, Ettinger SJ, et al. Management of incidentally detected heart murmurs in dogs and cats. *J Am Vet Med Assoc*. 2015;246(10):1076-88.

HEART MURMURS IN DOGS

Brian A. Scansen, DVM, MS, DACVIM (Cardiology)
*Ohio State University**



VSD = ventricular septal defect

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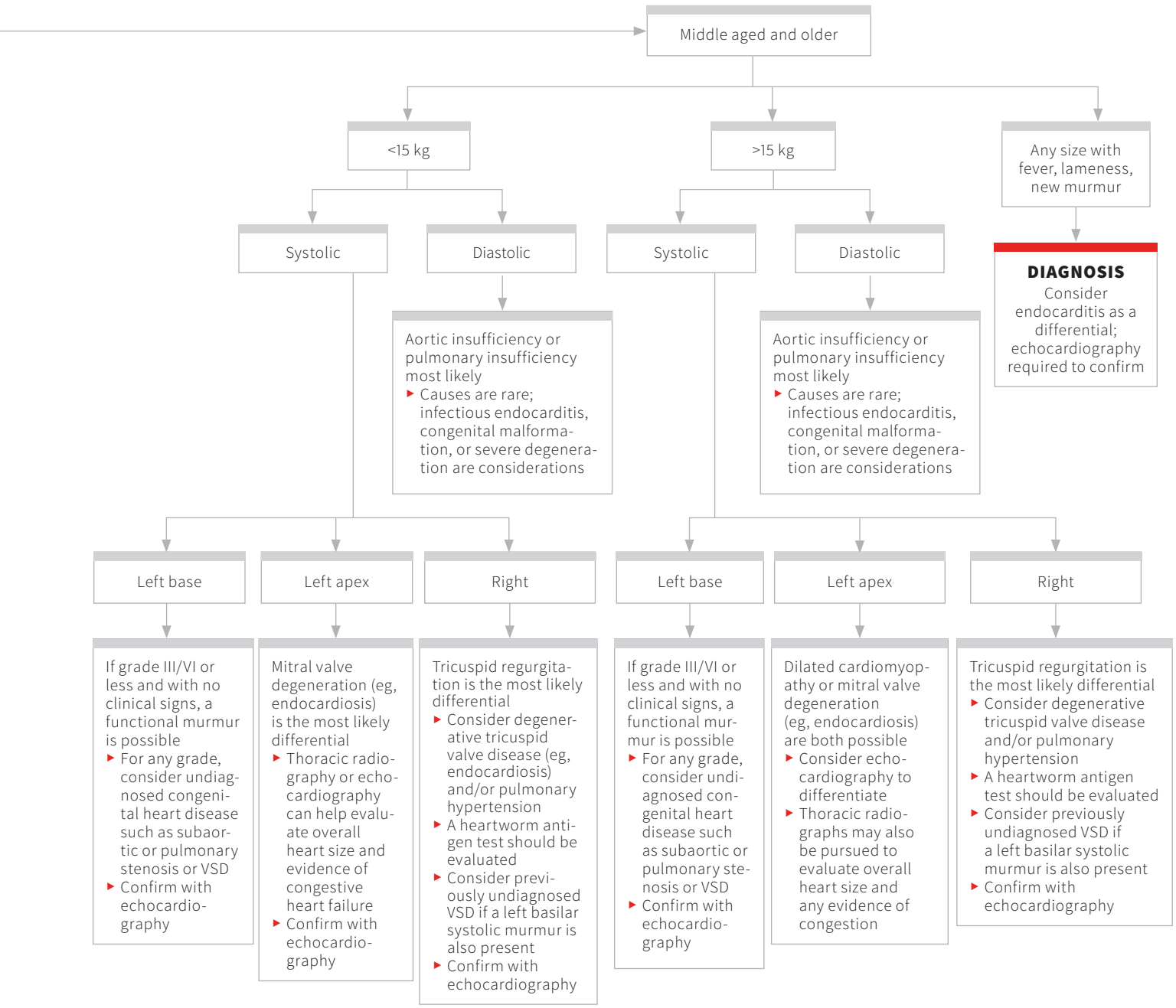
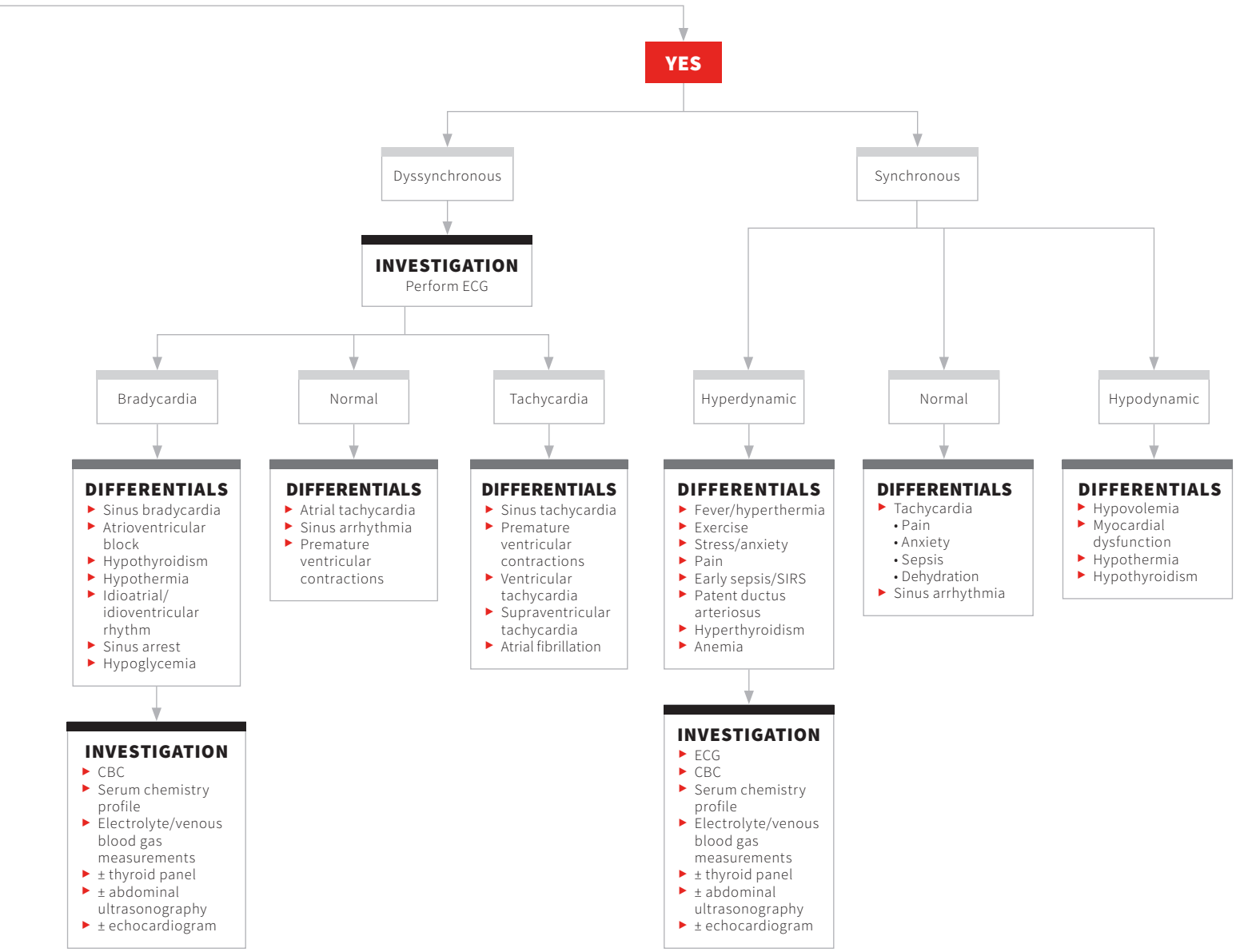

Middle aged and older
<15 kg
>15 kg
Any size with fever, lameness, new murmur
Systolic
Diastolic
Systolic
Diastolic
DIAGNOSIS
Consider endocarditis as a differential; echocardiography required to confirm
Aortic insufficiency or pulmonary insufficiency most likely
Causes are rare; infectious endocarditis, congenital malformation, or severe degeneration are considerations
Aortic insufficiency or pulmonary insufficiency most likely
Causes are rare; infectious endocarditis, congenital malformation, or severe degeneration are considerations
Left base
Left apex
Right
Left base
Left apex
Right
If grade III/VI or less and with no clinical signs, a functional murmur is possible
For any grade, consider undiagnosed congenital heart disease such as subaortic or pulmonary stenosis or VSD
Confirm with echocardiography
Mitral valve degeneration (eg, endocardiosis) is the most likely differential
Thoracic radiography or echocardiography can help evaluate overall heart size and evidence of congestive heart failure
Confirm with echocardiography
Tricuspid regurgitation is the most likely differential
Consider degenerative tricuspid valve disease (eg, endocardiosis) and/or pulmonary hypertension
A heartworm antigen test should be evaluated
Consider previously undiagnosed VSD if a left basilar systolic murmur is also present
Confirm with echocardiography
If grade III/VI or less and with no clinical signs, a functional murmur is possible
For any grade, consider undiagnosed congenital heart disease such as subaortic or pulmonary stenosis or VSD
Confirm with echocardiography
Dilated cardiomyopathy or mitral valve degeneration (eg, endocardiosis) are both possible
Consider echocardiography to differentiate
Thoracic radiographs may also be pursued to evaluate overall heart size and any evidence of congestion
Tricuspid regurgitation is the most likely differential
Consider degenerative tricuspid valve disease and/or pulmonary hypertension
A heartworm antigen test should be evaluated
Consider previously undiagnosed VSD if a left basilar systolic murmur is also present
Confirm with echocardiography

PULSE ALTERATIONS

Elisa Mazzaferro, DVM, MS, PhD, DACVECC
Cornell University Veterinary Specialists
Stamford, Connecticut

AFAST = abdominal focused assessment using sonography for trauma
CPR = cardiopulmonary resuscitation
SIRS = systemic inflammatory response syndrome
TFAST = thoracic focused assessment using sonography for trauma


YES
Dyssynchronous
Synchronous
INVESTIGATION
Perform ECG
Bradycardia
Normal
Tachycardia
Hyperdynamic
Normal
Hypodynamic
DIFFERENTIALS
Sinus bradycardia
Atrioventricular block
Hypothyroidism
Hypothermia
Idioatrial/ idioventricular rhythm
Sinus arrest
Hypoglycemia
DIFFERENTIALS
Atrial tachycardia
Sinus arrhythmia
Premature ventricular contractions
DIFFERENTIALS
Sinus tachycardia
Premature ventricular contractions
Ventricular tachycardia
Supraventricular tachycardia
Atrial fibrillation
DIFFERENTIALS
Fever/hyperthermia
Exercise
Stress/anxiety
Pain
Early sepsis/SIRS
Patent ductus arteriosus
Hyperthyroidism
Anemia
DIFFERENTIALS
Tachycardia
• Pain
• Anxiety
• Sepsis
• Dehydration
Sinus arrhythmia
DIFFERENTIALS
Hypovolemia
Myocardial dysfunction
Hypothermia
Hypothyroidism
INVESTIGATION
CBC
Serum chemistry profile
Electrolyte/venous blood gas measurements
± thyroid panel
± abdominal ultrasonography
± echocardiogram
INVESTIGATION
ECG
CBC
Serum chemistry profile
Electrolyte/venous blood gas measurements
± thyroid panel
± abdominal ultrasonography
± echocardiogram

ACUTE ABDOMINAL PAIN

Justine A. Lee, DVM, DACVECC*
Pet Poison Helpline
Minneapolis, Minnesota

SIGNS OF ACUTE ABDOMINAL PAIN

INVESTIGATION

Obtain complete history:

- Signalment
- Presenting complaint
- Previous history, surgery, trauma
- Diet (eg, normal diet, recent ingestion, fatty meals, table food)
- Vaccine and medication status
- Toxin exposure
- Travel history
- Progression
- Systemic and metabolic signs (eg anorexia, coughing, sneezing, vomiting, diarrhea, regurgitation, polyuria, polydipsia, polyphagia, weakness)
- Foreign body
- Indoor or outdoor status (cats)

INVESTIGATION

Perform physical examination:

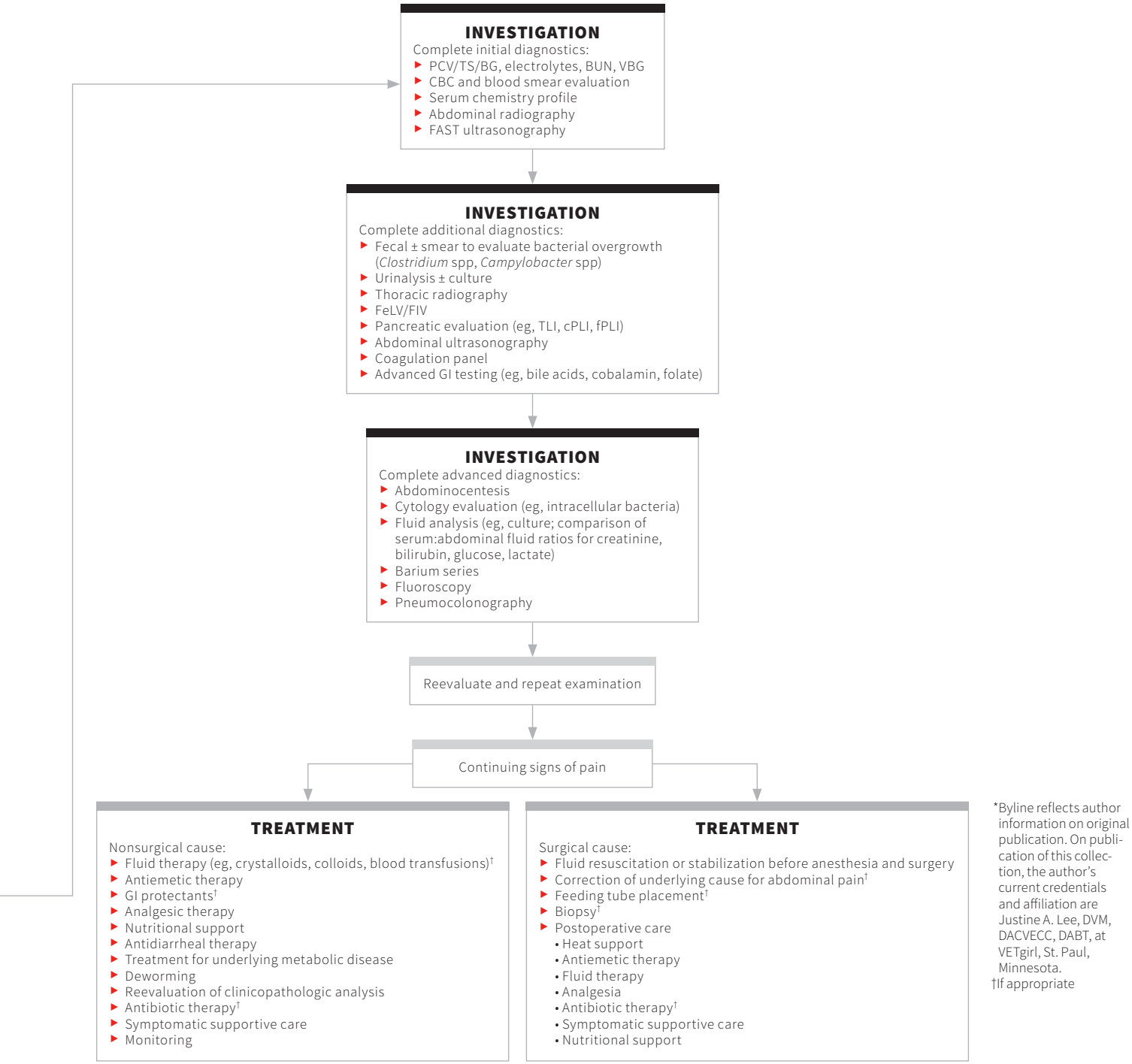
- **Hydration**: skin turgor, tacky mucous membranes, sunken eyes
- **Oral**: string under tongue, ulcerations, erosions, halitosis, foreign body, ptyalism
- **Cardiorespiratory**: lung sounds, heart rate, pulse quality, capillary refill time
- **Abdominal**: organomegaly, ascites, pregnancy, pain localization, fluid-filled loops, borborygmi, foreign body, masses
- **Integumentary**: petechiae, ecchymoses
- **Musculoskeletal/neurologic**: mentation, cranial nerves, ataxia, dysautonomia, decreased rectal tone, ambulatory
- **Rectal**: prostatomegaly, fecal evaluation, melena
- **Urogenital**: bladder evaluation, neuter status, vaginal discharge, pain on kidney palpation

Evaluate if signalment, history, presenting complaint, and examination findings help rule in or out differential diagnoses

DIAGNOSIS

- **Digestive**: gastric or duodenal ulcer, gastritis, gastroenteritis, GD, GDV, GI obstruction, intussusception, ileus, pancreatitis, intestinal parasitism, protein-losing enteropathy, inflammatory disease, neoplasia, hepatic disease
- **Metabolic**: acute renal failure, hepatopathy, hyperadrenocorticism, hypoadrenocorticism, diabetes
- **Peritoneal cavity**: trauma, septic peritonitis, GI tract perforation, foreign body, splenic torsion, ruptured abdominal abscess, uroabdomen, penetrating trauma, bile peritonitis, hemoabdomen, liver lobe torsion
- **Urinary**: ureteral, urethral, or cystic calculi; acute nephritis; pyelonephritis; urethral obstruction; acute renal failure; uroabdomen
- **Reproductive**: pyometra, labor or dystocia, uterine or testicular torsion, prostatic disease
- **Musculoskeletal**: intervertebral disk disease, abdominal muscular trauma, referred orthopedic pain
- **Infectious disease**: infectious canine hepatitis, leptospirosis, parvovirus, panleukopenia, FIP, *Giardia* spp infection, *Salmonella* spp infection, *Clostridium* spp infection, vector-borne diseases
- **Other**: toxicity, bezoar, caustic or corrosive ingestion, nausea secondary to ileus (opioid therapy, postoperative)

BG = blood glucose
FAST = focused assessment with sonogram for trauma
FIC = feline idiopathic cystitis
GD = gastric dilatation
GDV = gastric dilatation-volvulus
PCV = packed cell volume
PLI = pancreatic lipase immuno-reactivity
TLI = trypsin-like immunoreactivity
TS = total solids
VBG = venous blood gas

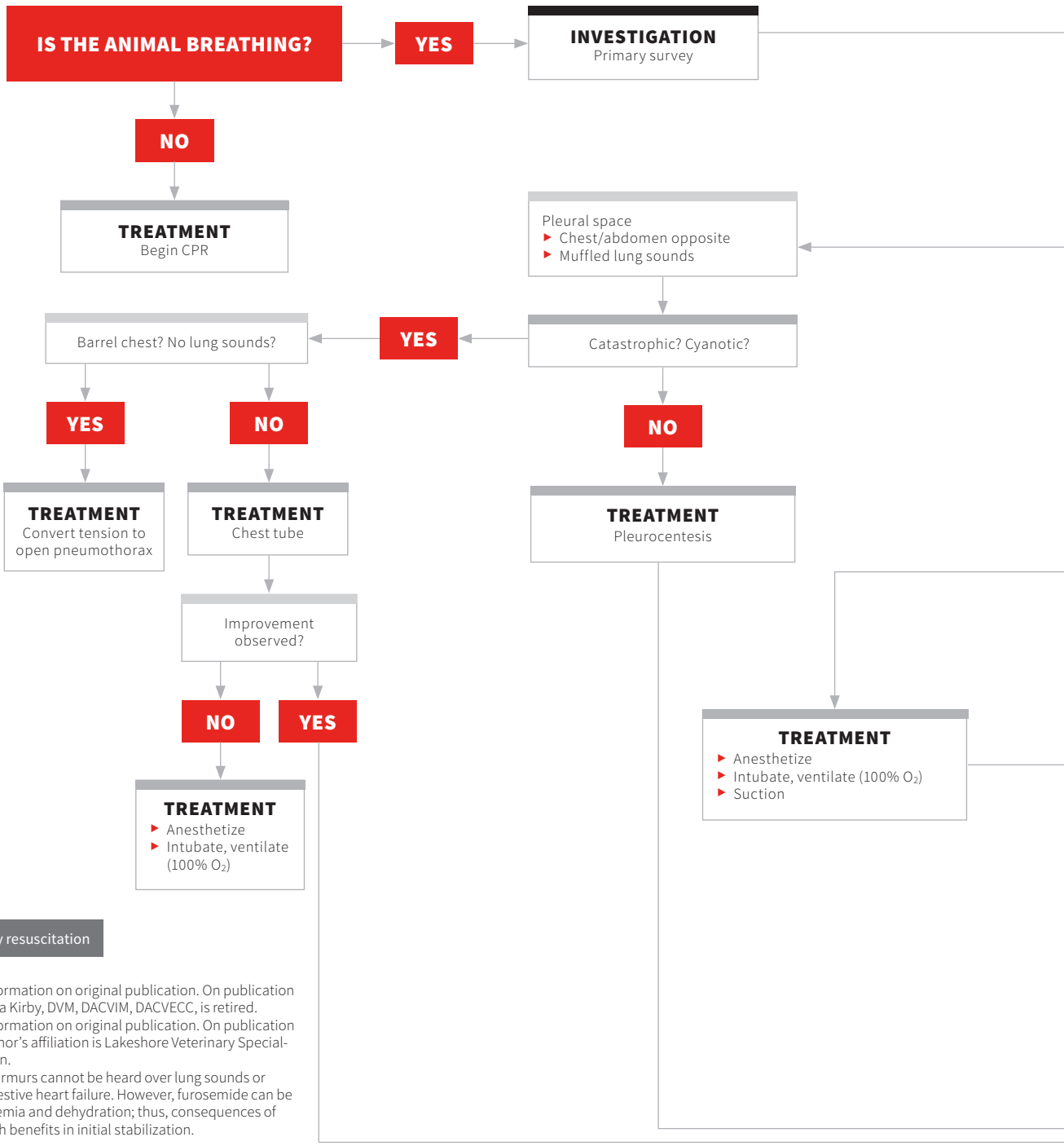


*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Justine A. Lee, DVM, DACVECC, DABT, at VETgirl, St. Paul, Minnesota.

†If appropriate

ACUTE BREATHING DIFFICULTY: INITIAL MANAGEMENT

Rebecca Kirby, DVM, DACVIM, DACVECC*
Elke Rudloff, DVM, DACVECC†
Animal Emergency Center
Glendale, Wisconsin



CPR = cardiopulmonary resuscitation

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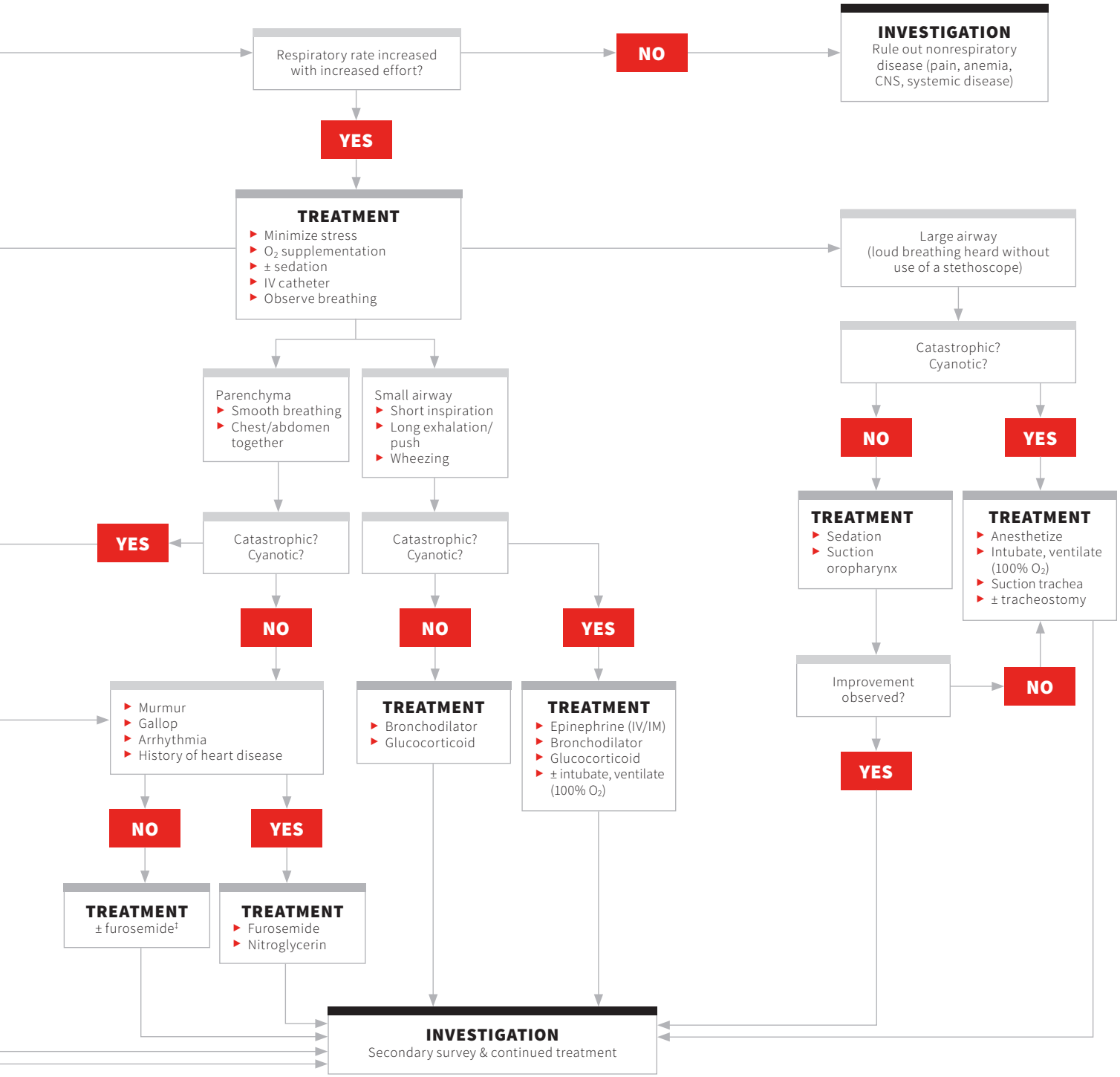
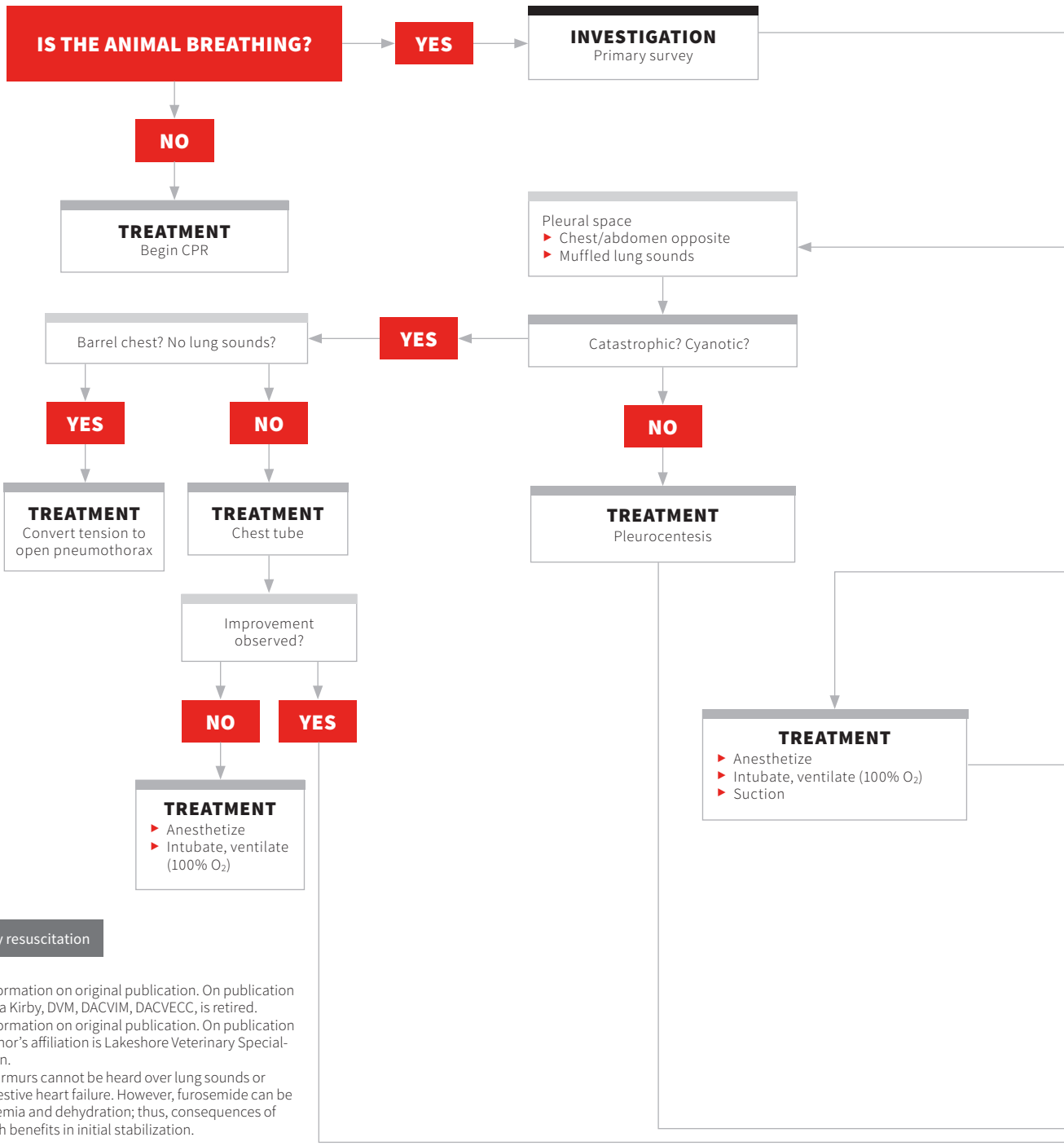
†Byline reflects author information on original publication. On publication of this collection, the author's affiliation is Lakeshore Veterinary Specialists in Glandale, Wisconsin.
‡In some cases, extant murmurs cannot be heard over lung sounds or because of true left congestive heart failure. However, furosemide can be associated with hypovolemia and dehydration; thus, consequences of furosemide may outweigh benefits in initial stabilization.


Respiratory rate increased with increased effort?
NO
INVESTIGATION
Rule out nonrespiratory disease (pain, anemia, CNS, systemic disease)
YES
TREATMENT
Minimize stress
O_2 supplementation
± sedation
IV catheter
Observe breathing
Large airway (loud breathing heard without use of a stethoscope)
Catastrophic? Cyanotic?
NO
YES
TREATMENT
Sedation
Suction oropharynx
TREATMENT
Anesthetize
Intubate, ventilate (100% O_2)
Suction trachea
± tracheostomy
Improvement observed?
NO
YES
Parenchyma
Smooth breathing
Chest/abdomen together
Small airway
Short inspiration
Long exhalation/ push
Wheezing
Catastrophic? Cyanotic?
YES
Catastrophic? Cyanotic?
NO
NO
YES
Murmur
Gallop
Arrhythmia
History of heart disease
TREATMENT
Bronchodilator
Glucocorticoid
TREATMENT
Epinephrine (IV/IM)
Bronchodilator
Glucocorticoid
± intubate, ventilate (100% O_2)
NO
YES
TREATMENT
± furosemide‡
TREATMENT
Furosemide
Nitroglycerin
INVESTIGATION
Secondary survey & continued treatment

ACUTE BREATHING DIFFICULTY: INITIAL MANAGEMENT

Rebecca Kirby, DVM, DACVIM, DACVECC*
Elke Rudloff, DVM, DACVECC†
Animal Emergency Center
Glendale, Wisconsin



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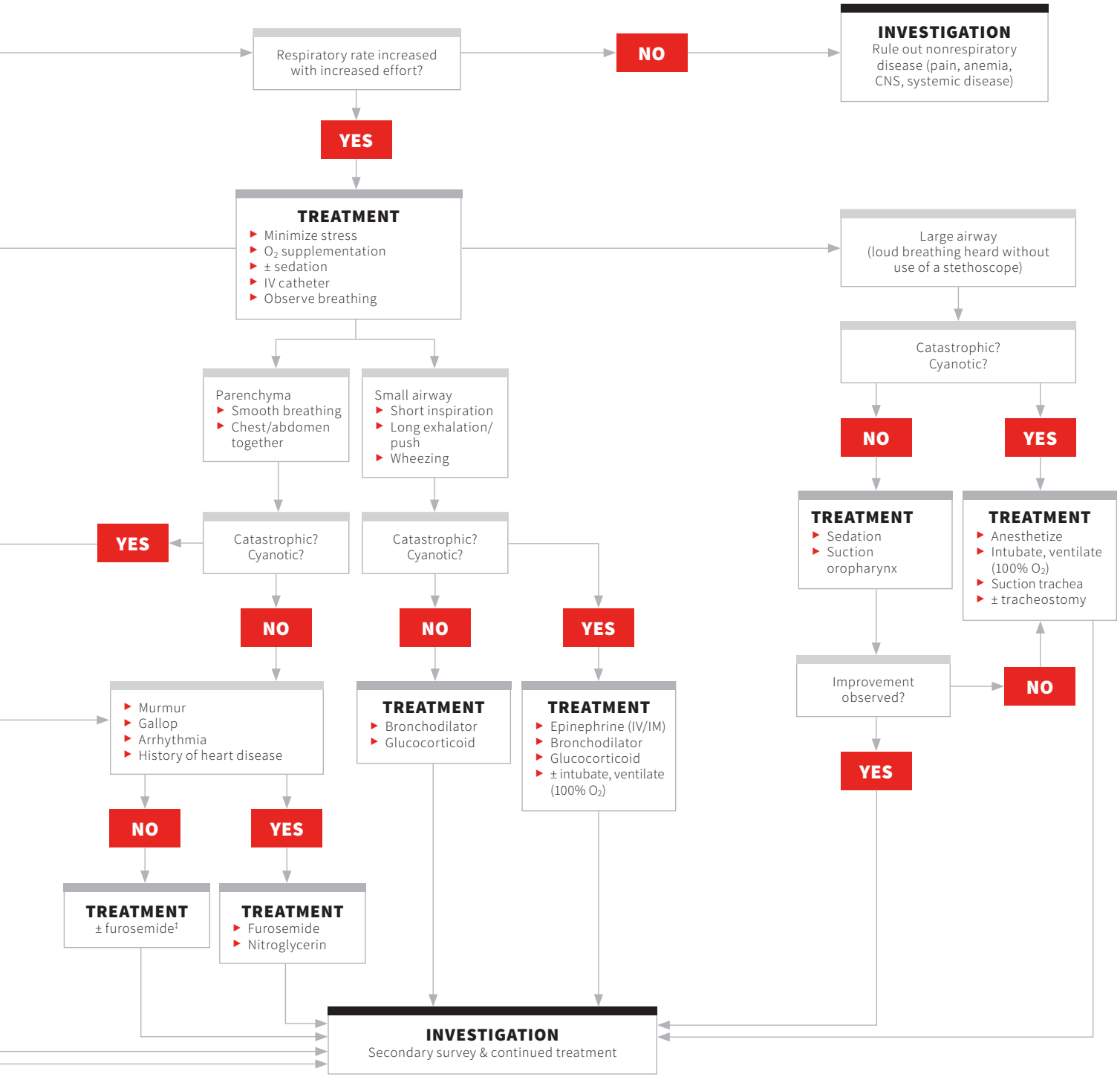
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± intubate, ventilate (100% O_2)
NO
YES
TREATMENT
± furosemide‡
TREATMENT
Furosemide
Nitroglycerin
INVESTIGATION
Secondary survey & continued treatment

BLOOD GAS ANALYSIS

Lori S. Waddell, DVM, DACVECC
University of Pennsylvania

This algorithm reflects canine normals. For cats, substitute feline normals for pH, BE (or HCO_3^-), PCO_2, and PO_2 values (*Table 1*).

SELECT ANALYZER

INVESTIGATION
Determine if arterial or venous
- Venous: Jugular sample provides the best idea of whole-body status. Peripheral samples may represent local tissue bed and not whole body.
- Arterial: Dorsal metatarsal artery, femoral artery, auricular artery, or caudal artery

INVESTIGATION
Arterial sample (to assess respiratory function)
- $PaCO_2$ assesses ability to ventilate
- PaO_2 assesses ability to oxygenate

INVESTIGATION
Venous sample (pulse oximetry can be used with venous blood gas to assess oxygenation)
- $PvCO_2$ can suggest ventilation (usually about 5 mm Hg higher than $PaCO_2$)

INVESTIGATION
Evaluate pH to determine if acidemia or alkalemia present

INVESTIGATION
Acidemia = pH <7.35

INVESTIGATION
Alkalemia = pH >7.45

INVESTIGATION
Determine if metabolic or respiratory in origin

INVESTIGATION
Evaluate PCO_2 and BE for masked disturbances if pH = 7.35-7.45

INVESTIGATION
Determine if metabolic or respiratory in origin

Respiratory = $PaCO_2$ >45 mm Hg

Metabolic = BE <−4 mmol/L (or HCO_3^- <21 mEq/L)

INVESTIGATION
Assess for compensation (see ***Rules of Compensation***)

Respiratory = $PaCO_2$ <35 mm Hg

Metabolic = BE >4 mmol/L (or HCO_3^- >27 mEq/L)

DIFFERENTIAL
Respiratory acidosis
- Depressed respiratory center
- Cervical spinal cord disease
- Neuromuscular disease
- Pleural space disease
- Airway obstruction
- Rarely severe pulmonary parenchymal disease
- Respiratory muscle fatigue

TREATMENT
- Correct underlying problem
- Relieve airway obstruction/ restrictive disease (pleural space)
- Intubate, begin PPV

DIFFERENTIAL
Metabolic acidosis
- Increased anion gap:
  - Ketones
  - Lactate
  - Uremia
  - Toxicity (eg, ethylene glycol, salicylates)
- Normal anion gap:
  - HCO_3^- loss through kidneys or intestinal tract

TREATMENT
- Correct underlying cause
- Lacate: Improve oxygen delivery to the tissues
- Ketones: Insulin therapy
- $NaHCO_3$ (if needed)
  - HCO_3^- deficit = BE × body weight (kg) × 0.3
  - Give 1/4 to 1/3 of dose and recheck blood gas

DIFFERENTIAL
Respiratory alkalosis
- Hypoxemia
- Pulmonary disease
- CNS disease (eg, stimulating respiratory center)
- Exercise, pain, stress

TREATMENT
Correct underlying disease

DIFFERENTIAL
Metabolic alkalosis
- GI obstruction with loss of H^+, K^+, and especially Cl^- in vomitus
- Loop diuretics
- $NaHCO_3$ administration

TREATMENT
- Hypokalemic hypochloremic metabolic alkalosis: Volume expansion with 0.9% NaCl
- Loop diuretic or HCO_3^- therapy: No treatment, usually self-limiting

TABLE 1

NORMAL VALUES FOR BLOOD GASES

	Arterial	Venous
CANINE		
pH	7.35-7.45	7.35-7.45
PO_2 (mm Hg)	90-100	30-42
PCO_2 (mm Hg)	35-45	40-50
HCO_3^- (mmol/L)	20-24	20-24
BE (mmol/L)	−4-+4	−4-+4
FELINE		
pH	7.34 ± 0.1	7.30 ± 0.08
PO_2 (mm Hg)	102.9 ± 15	38.6 ± 11
PCO_2 (mm Hg)	33.6 ± 7	41.8 ± 9
HCO_3^- (mEq/L)	17.5 ± 3	19.4 ± 4
BE (mmol/L)	−6.4 ± 5	−5.7 ± 5

TABLE 2

EXPECTED COMPENSATORY CHANGES

Disorder	Primary Change	Compensatory Response
Metabolic acidosis	↓ HCO_3^-	0.7 mm Hg decrease in PCO_2 for each 1 mmol/L decrease in HCO_3^-
Metabolic alkalosis	↑ HCO_3^-	0.7 mm Hg increase in PCO_2 for each 1 mmol/L increase in HCO_3^-
Acute respiratory acidosis	↑ PCO_2	1.5 mmol/L increase in HCO_3^- for each 10 mm Hg increase in PCO_2
Chronic respiratory acidosis	↑ PCO_2	3.5 mmol/L increase in HCO_3^- for each 10 mm Hg increase in PCO_2
Acute respiratory alkalosis	↓ PCO_2	2.5 mmol/L decrease in HCO_3^- for each 10 mm Hg decrease in PCO_2
Chronic respiratory alkalosis	↓ PCO_2	5.5 mmol/L decrease in HCO_3^- for each 10 mm Hg decrease in PCO_2

Continues ▶

Rules of Compensation

1. Change in respiratory or metabolic component of the acid-base status will normally induce opposite, compensatory change in the other to return pH toward normal.
2. Lungs compensate rapidly by changing minute ventilation (eg, respiratory rate/tidal volume/both) within minutes.
3. Metabolic compensation occurs via the kidneys and is much slower, starting after a few hours and requiring 4 to 5 days for maximum compensation.
4. Absence or presence and degree of compensation for respiratory disturbance can give an idea of chronicity (***Table 2***).
5. Overcompensation does not occur.
6. If expected compensation is absent, a mixed disturbance is present. For example, if metabolic acidosis is not accompanied by compensatory respiratory alkalosis (CO_2 is normal or increased), a mixed disturbance is occurring with both metabolic acidosis and respiratory acidosis.

BE = base excess
HCO_3^- = bicarbonate
NaCl = sodium chloride
$NaHCO_3$ = sodium bicarbonate
$PaCO_2$ = partial pressure of arterial carbon dioxide
PaO_2 = partial pressure of oxygen
PCO_2 = partial pressure carbon dioxide
PO_2 = partial pressure oxygen
PPV = positive-pressure ventilation
$PvCO_2$ = partial pressure of venous carbon dioxide

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BLOOD GAS ANALYSIS ALTERNATIVES

If blood gas analysis is not available, some information can be obtained through other tests. Pulse oximetry can be used to assess a patient's oxygenation. It must be remembered that pulse ox saturation and PaO_2 are not directly correlated; a pulse ox of 93% corresponds with a PaO_2 of 80 mm Hg and a pulse of 90% corresponds with a PaO_2 of 60 mm Hg. If the patient is intubated, end tidal CO_2 ($ETCO_2$) can be used to assess for hypercarbia or hypocarbia. $ETCO_2$ usually corresponds well to $PaCO_2$, with $ETCO_2$ ≈5 mm Hg lower than $PaCO_2$ in normal patients. In medium and large dogs, the end tidal tubing can be placed just inside the nostril of an awake, compliant patient to estimate $ETCO_2$.

If anion gap measurement is available on a chemistry screen, it can be used to detect some causes of metabolic acidosis, including those caused by ketones, lactate, and exogenous acids (eg, toxins, phosphates, sulfates). These will cause an increase in the anion gap. If the anion gap is normal but the corrected chloride is elevated (see equation below), loss of bicarbonate via the kidneys or the large bowel may be causing a metabolic acidosis.

In addition, the patient's ketones may be measured via either ketone strips for use with urine or a ketometer to measure serum ketones. A handheld lactate meter can be used to measure ketones. These are relatively inexpensive, small pieces of equipment similar to a glucometer and give results in a few minutes. Although they do not give information about pH, they can help guide fluid therapy and potentially help prognosticate in patients that are presented with a high lactate and do not respond to therapy. To detect metabolic alkalosis, the patient's chloride concentration should be evaluated. If the corrected chloride is low, a metabolic alkalosis is likely.

$$\textbf{Corrected } Cl^- = (\textbf{normal } Na^+ / \textbf{measured } Na^+) \times \textbf{measured } Cl^-$$

This equation helps determine if the chloride concentration is abnormal in comparison to the sodium concentration, as normally the 2 ions will change in the same direction and to the same degree. Changes in the corrected chloride can indicate metabolic derangements.

Cl = chloride

$ETCO_2$ = end tidal CO_2

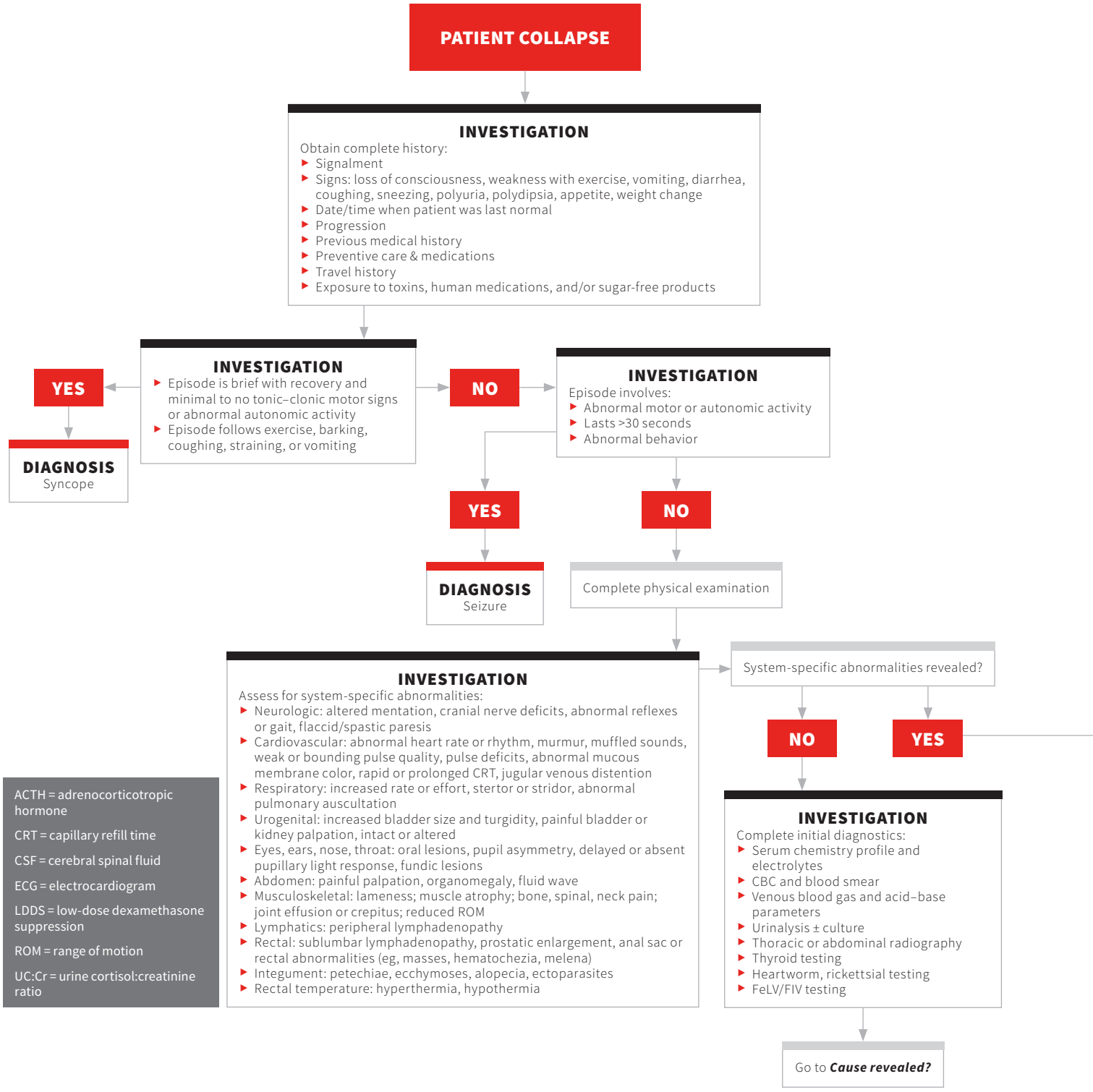
Na = sodium

$PaCO_2$ = partial pressure of arterial carbon dioxide

PaO_2 = partial pressure of oxygen

COLLAPSE

Vincent Thawley, VMD*
Deborah Silverstein, DVM, DACVECC
University of Pennsylvania



ACTH = adrenocorticotropic hormone

CRT = capillary refill time

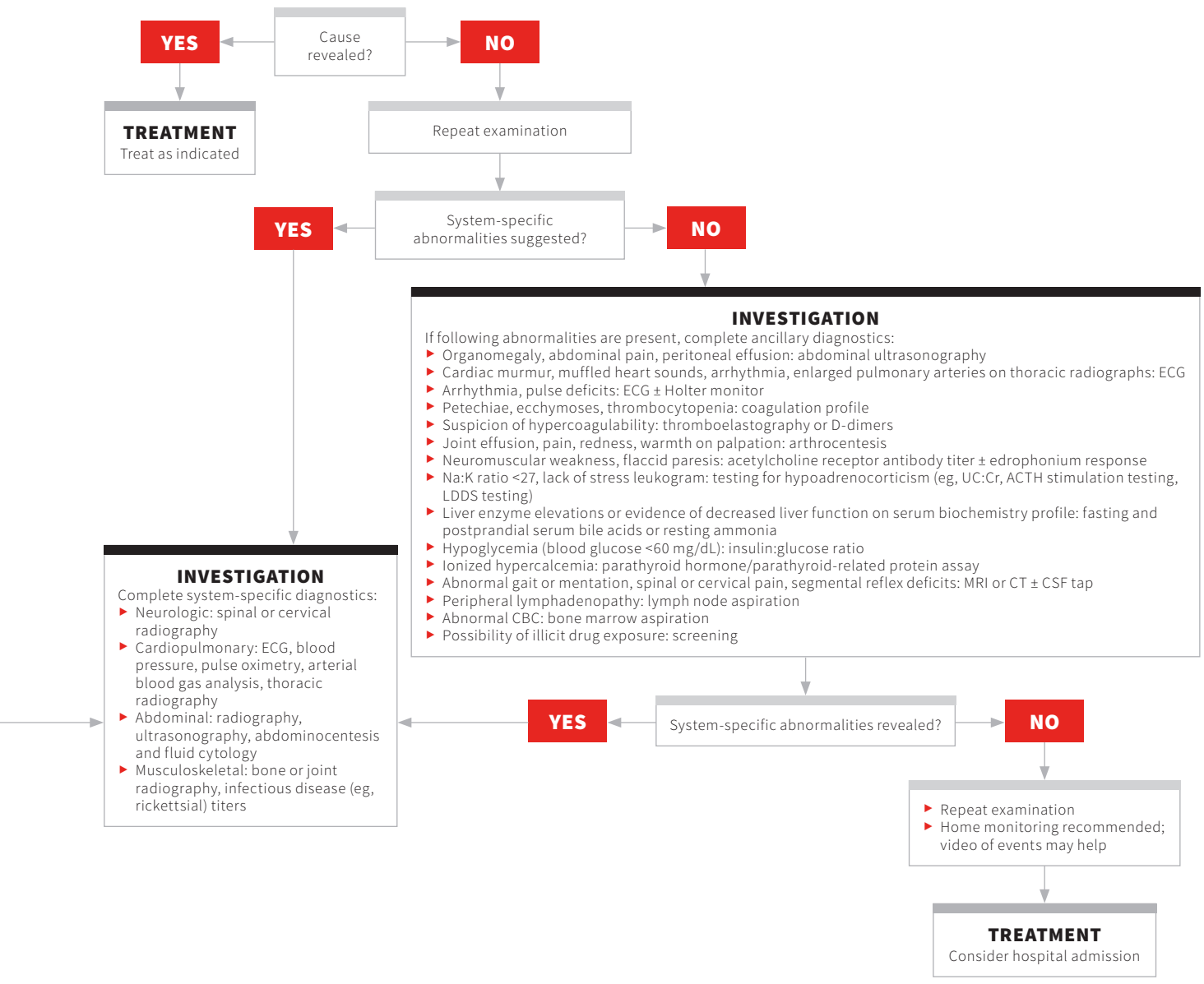
CSF = cerebral spinal fluid

ECG = electrocardiogram

LDDS = low-dose dexamethasone suppression

ROM = range of motion

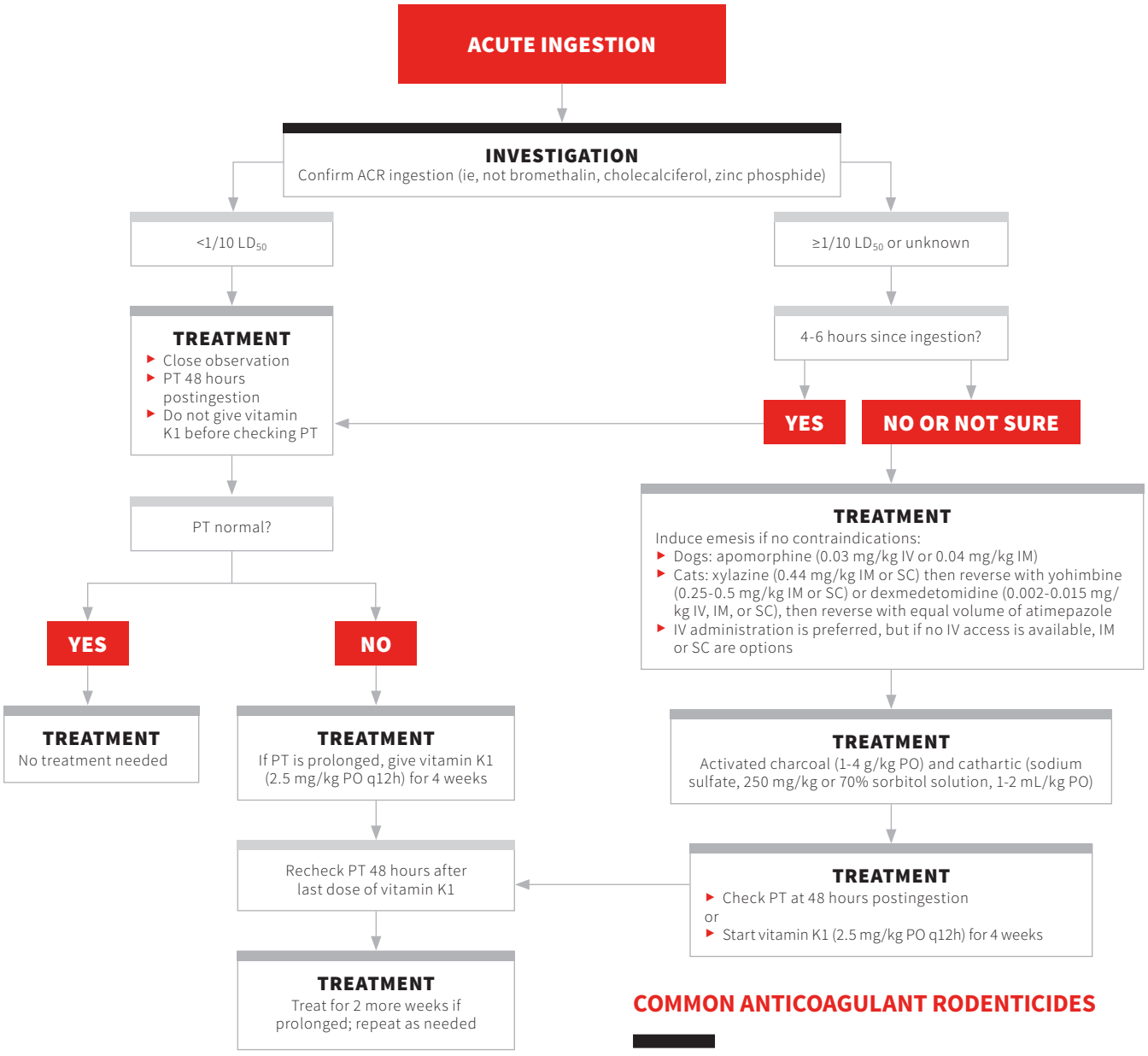
UC:Cr = urine cortisol:creatinine ratio



*Byline reflects author information on original publication. On publication of this collection, the author's current credentials are Vincent Thawley, VMD, DACVECC.

ANTICOAGULANT RODENTICIDE INGESTION

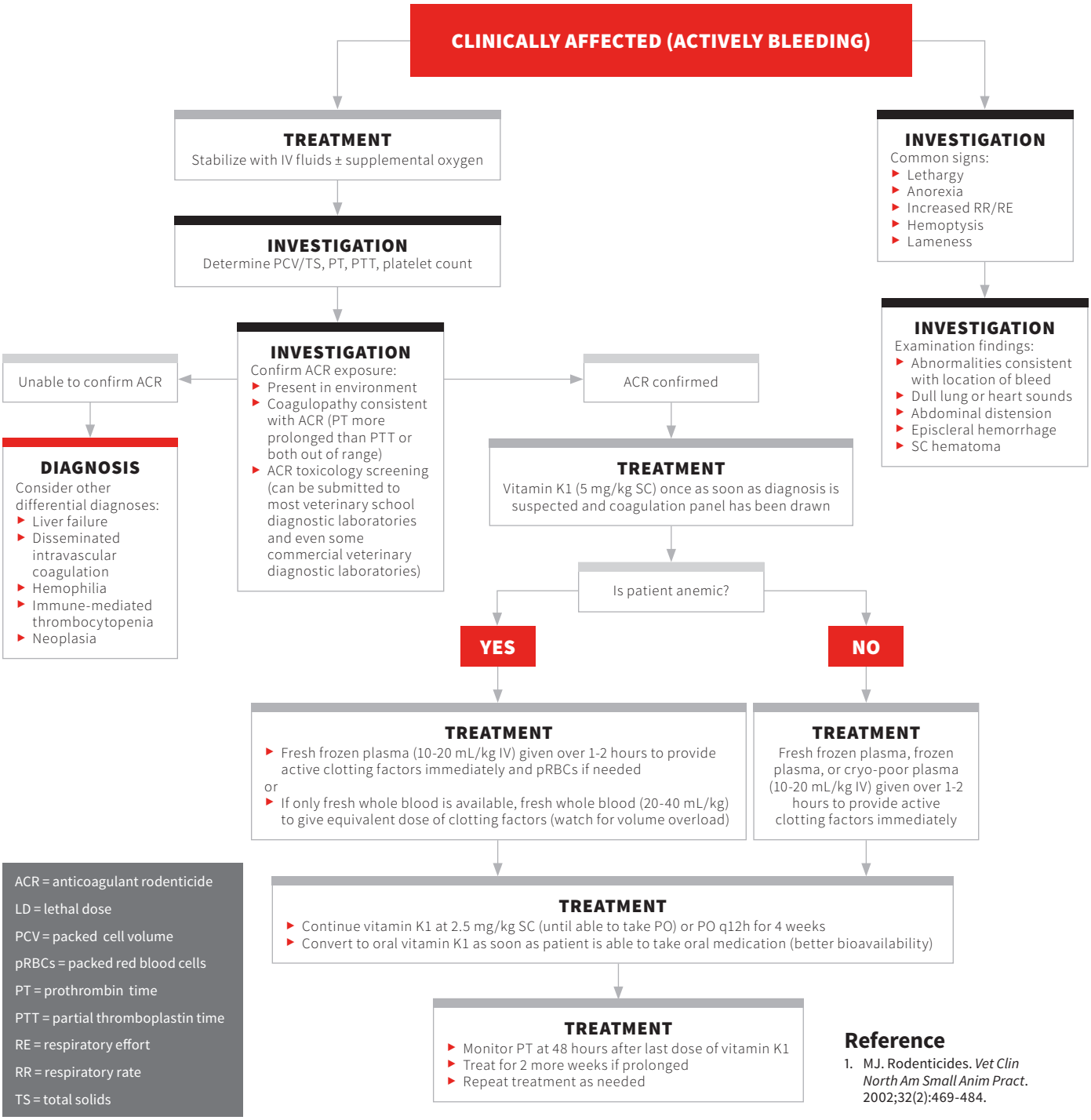
Lori S. Waddell, DVM, DACVECC
University of Pennsylvania



COMMON ANTICOAGULANT RODENTICIDES

Agent	LD_{50} (mg/kg) in Dogs[1]	LD_{50} (mg/kg) in Cats[1]
Brodifacoum	0.2-4	25
Bromadiolone	11-15	>25
Chlorophacinone	3-20	15
Diphacinone	0.9-8	15

The EPA has banned public use of brodifacoum, bromadiolone, difenacoum, and difethialone. These products were manufactured for home use until December 31, 2014, and shipped until March 2015. The newer products contain diphacinone, and 4 weeks of vitamin K1 therapy is recommended at the time of this publication to treat this anticoagulant rodenticide (ACR). These changes may decrease ACR toxicities and increase cases of other rodenticide toxicities, particularly bromethalin, which can cause neurotoxicity.



ACR = anticoagulant rodenticide
LD = lethal dose
PCV = packed cell volume
pRBCs = packed red blood cells
PT = prothrombin time
PTT = partial thromboplastin time
RE = respiratory effort
RR = respiratory rate
TS = total solids

Reference

1. MJ. Rodenticides. *Vet Clin North Am Small Anim Pract*. 2002;32(2):469-484.

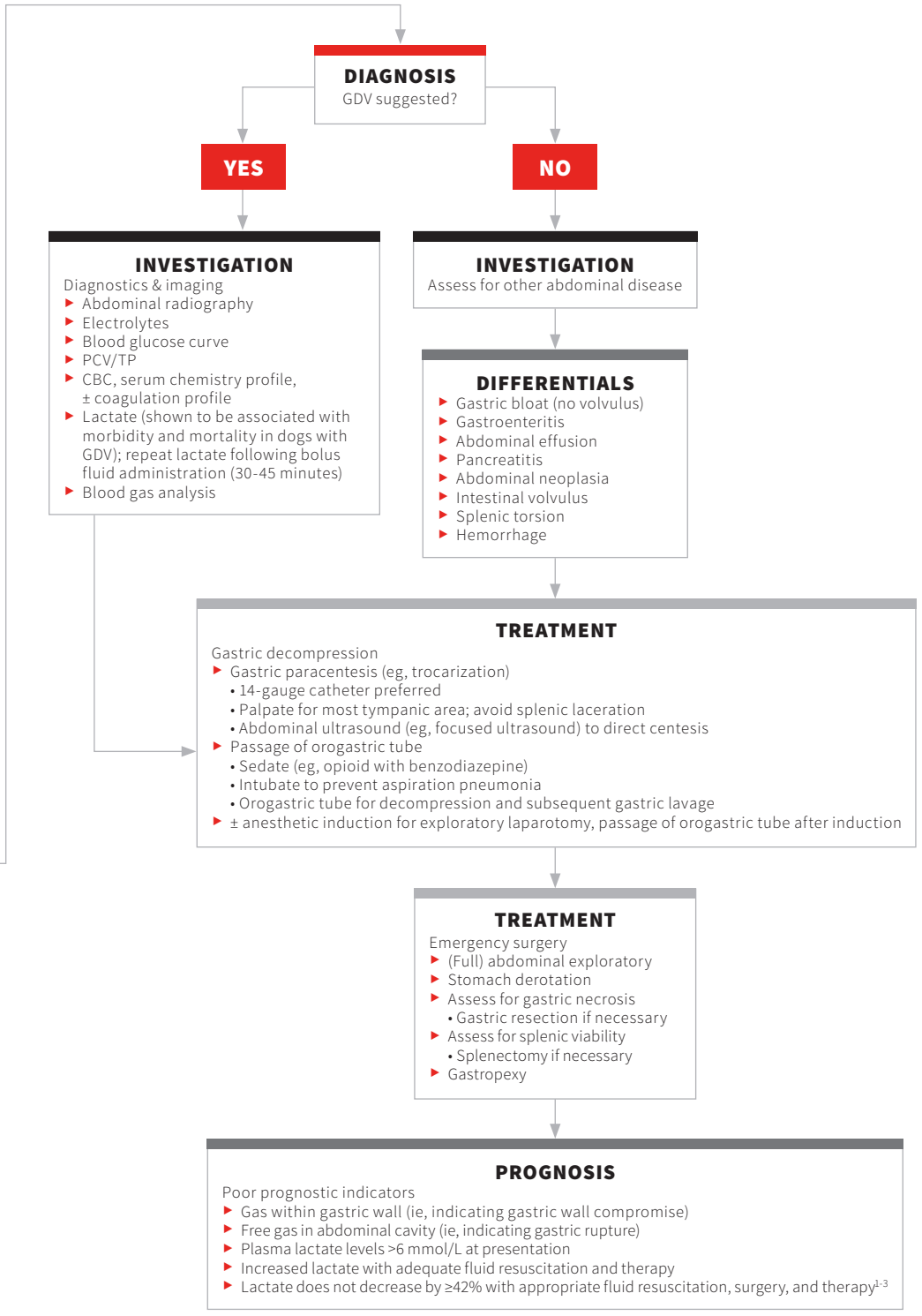
GASTRIC DILATATION-VOLVULUS

Lisa L. Powell, DVM, DACVECC*
University of Minnesota

POSSIBLE COMPLICATIONS

- Cardiac arrhythmias often begin within 24 hours of surgery. Continuous ECG monitoring is ideal. Factors include poor myocardial perfusion, electrolyte disturbances, acidosis, myocardial-depressant factor, and DIC. Antiarrhythmic drugs should only be considered if volume has been adequately replaced and arrhythmia is life threatening or causing poor perfusion.
- DIC can persist after surgery. Factors include blood pooling in portal circulation and the caudal vena cava, sepsis, vascular thrombosis, endotoxemia, acidosis, tissue hypoxia, and splenic congestion. Diagnosis may be confirmed by prolonged activated clotting time or abnormalities in platelet, FSP, and PTT values. Treatment should target underlying cause; microvascular thrombosis should be prevented with adequate tissue perfusion via IV fluid therapy.
- Sepsis can occur postoperatively; aspiration pneumonia is a frequent cause (vs gastric leakage). Thoracic radiography and peritoneal lavage can help identify origin.
- Recent studies have shown that lactate clearance is a much better indicator of prognosis versus a single, baseline lactate at presentation.[1-3] A lactate decrease of ≥42% was found to be an indicator of improved outcome.[1-3] A baseline lactate should be obtained, then serial lactates after adequate fluid resuscitation, and postoperatively, should be performed and compared.

*Byline reflects author information on original publication. On publication of this collection, the author's current affiliation is BluePearl Pet Hospital, Eden Prairie, Minnesota.
†Abdomen may vary from remarkable, distended, and firm to tympanic. If stomach is contained in rib cage or if there is gastric torsion with minimal air, visual bloat (ie, distention) may not be present.



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2. Zacher LA, Berg J, Shaw SP, et al. Association between outcome and changes in plasma lactate concentration during presurgical treatment id dogs with gastric dilatation-volvulus: 64 cases (2002-2008). *J Am Vet Med Assoc* 2010:236:892-897.
3. Green TI, Tonozzi CC, Kirby R, Rudloff E. Evaluation of initial plasma lactate values as a predictor of gastric necrosis and initial and subsequent plasma lactate values as a predictor of survival in dogs with gastric dilatation-volvulus: 84 dogs (2003-2007). *J Vet Emerg Crit Care* 2011;21(1):36-44.

CRT = capillary refill time

DIC = disseminated intravascular coagulation

FSP = fibrin split products

GDV = gastric dilatation-volvulus

MDB = medulloblastoma

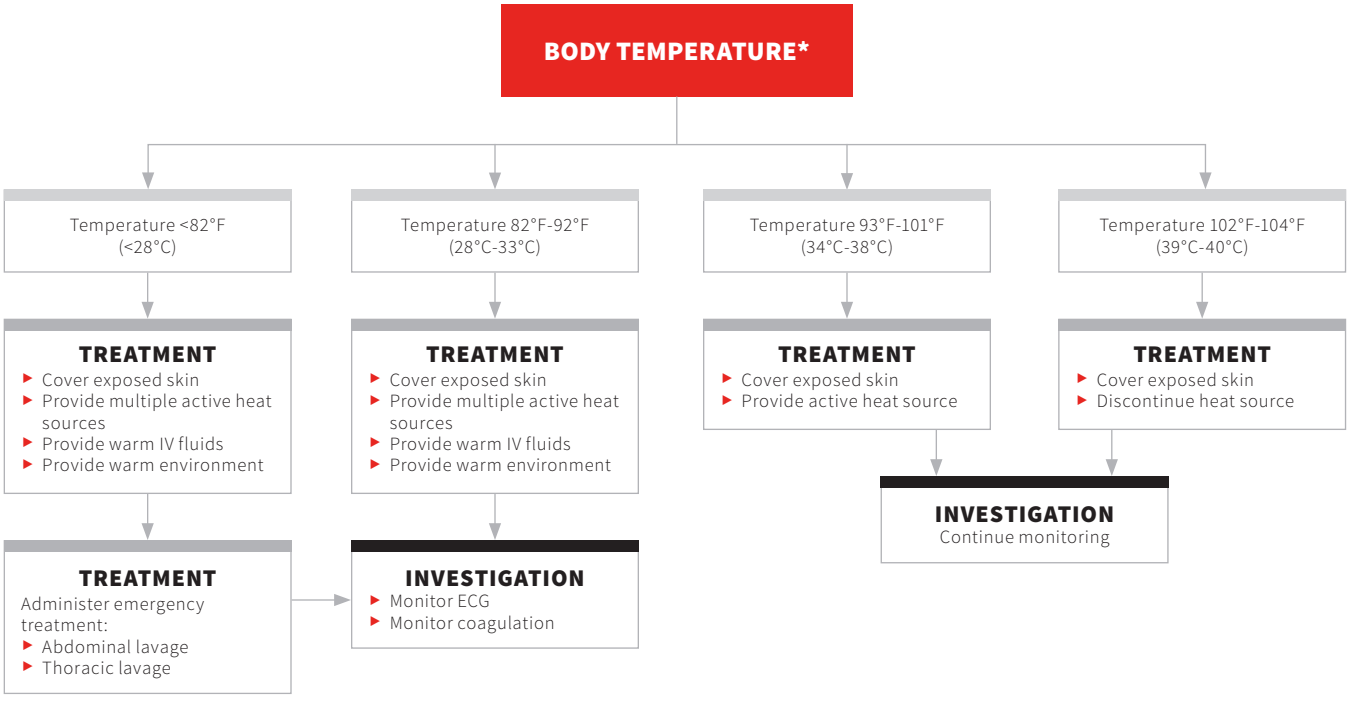
PCV = packed cell volume

PTT = activated partial thromboplastin time

TP = total protein

HYPOTHERMIA

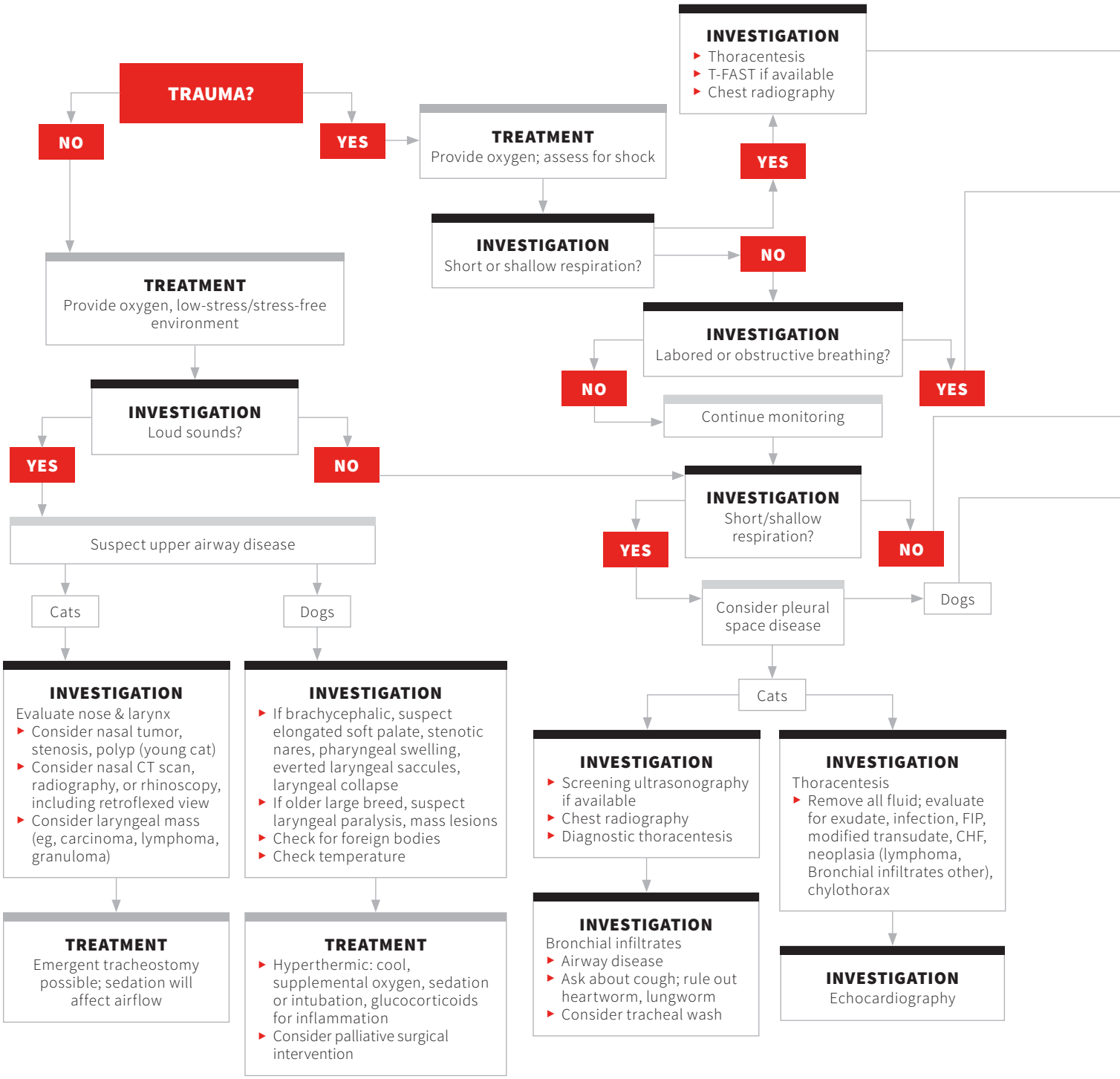
Lysa Posner, DVM, DACVAA
North Carolina State University

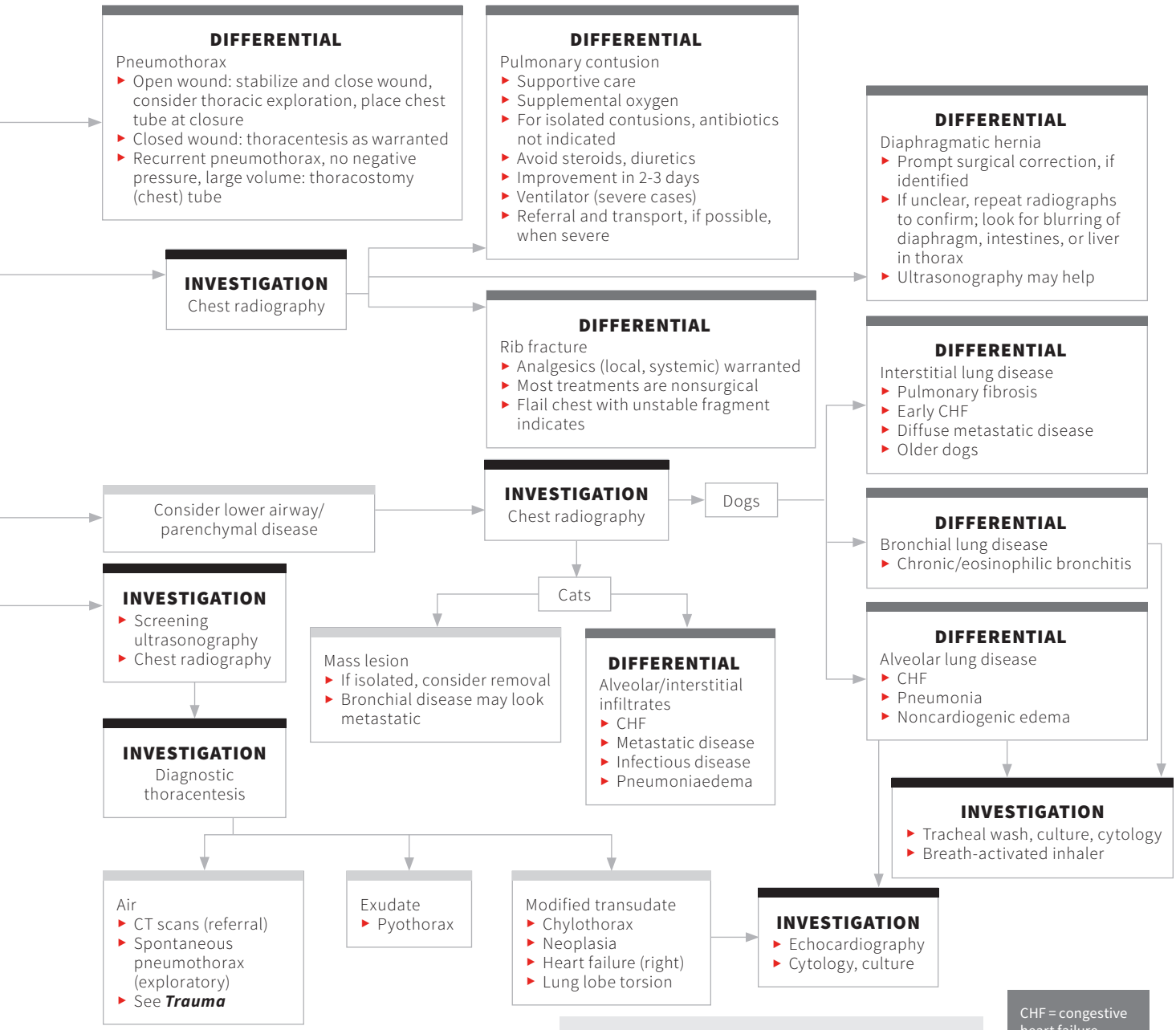


*As patient warms, treatment strategies change to suit current body temperature.

RESPIRATORY DISTRESS

Elizabeth Rozanski, DVM, DACVIM (SAIM), DACVECC
Cummings School of Veterinary Medicine at Tufts University





CANINE RESPIRATORY DISTRESS TIPS

- Loud murmur and weight loss often signal heart disease.
- Eosinophilic disease may be idiopathic or parasitic.
- Fever may be present with pneumonia.
- Dorsal caudal infiltrates are commonly noncardiogenic.
- Consider endemic fungal disease based on geographic location and travel.

FELINE RESPIRATORY DISTRESS TIPS

- Hypothermic cats often have heart disease.
- History of cough is likely signal of airway disease, but not all cats with airway disease cough.
- Severe heart disease may exist with soft/absent murmur; loud murmur may be innocent flow murmur.
- Isolated lung masses should not cause shortness of breath; if breathing labored, interstitial fibrosis possible.

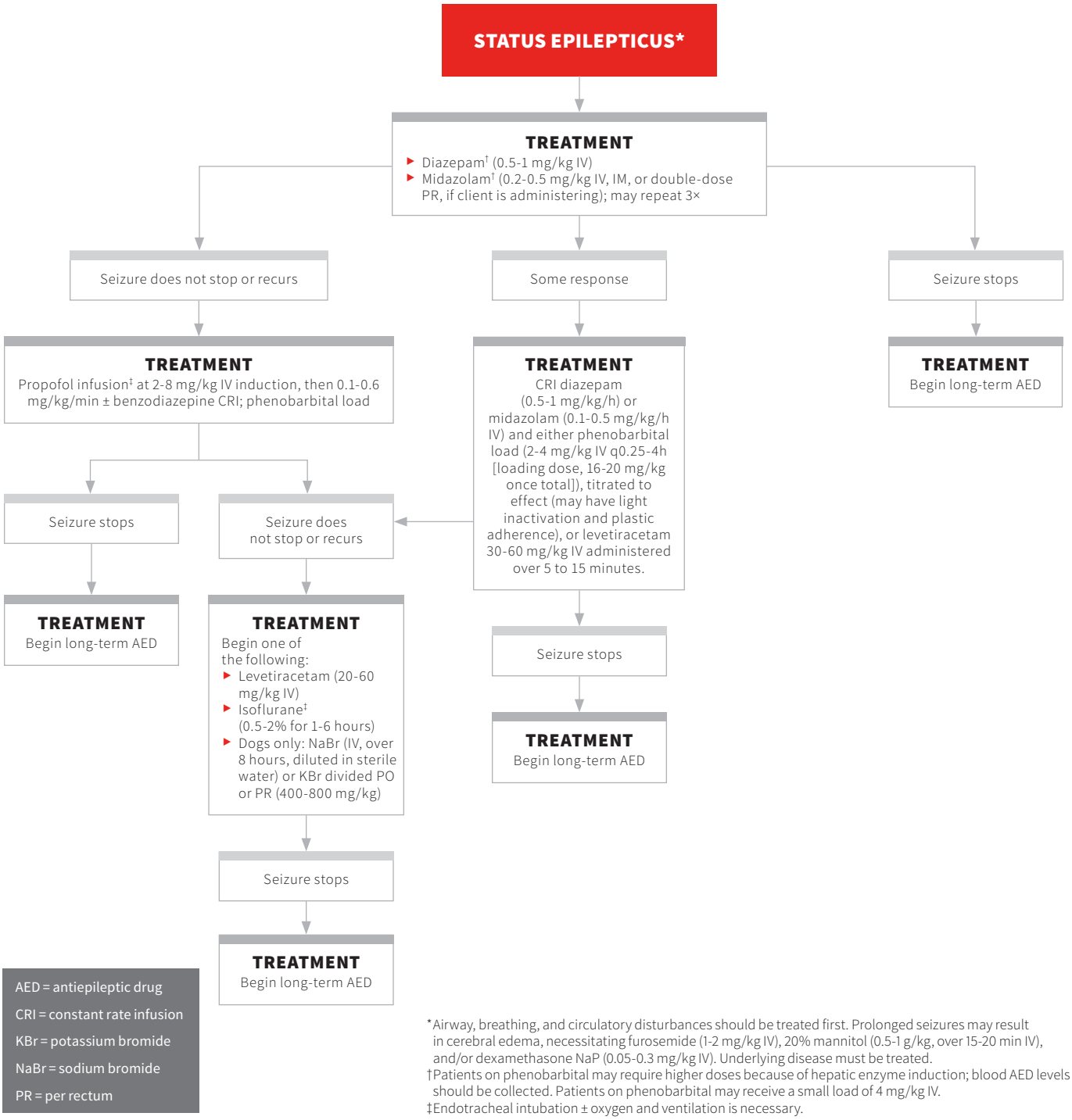
CHF = congestive heart failure

FIP = feline infectious peritonitis

T-FAST = thoracic-focused assessment with sonogram for trauma

STATUS EPILEPTICUS: EMERGENCY MANAGEMENT

Andrew Linklater, DVM, DACVECC
Lakeshore Veterinary Specialists
Glendale, Wisconsin

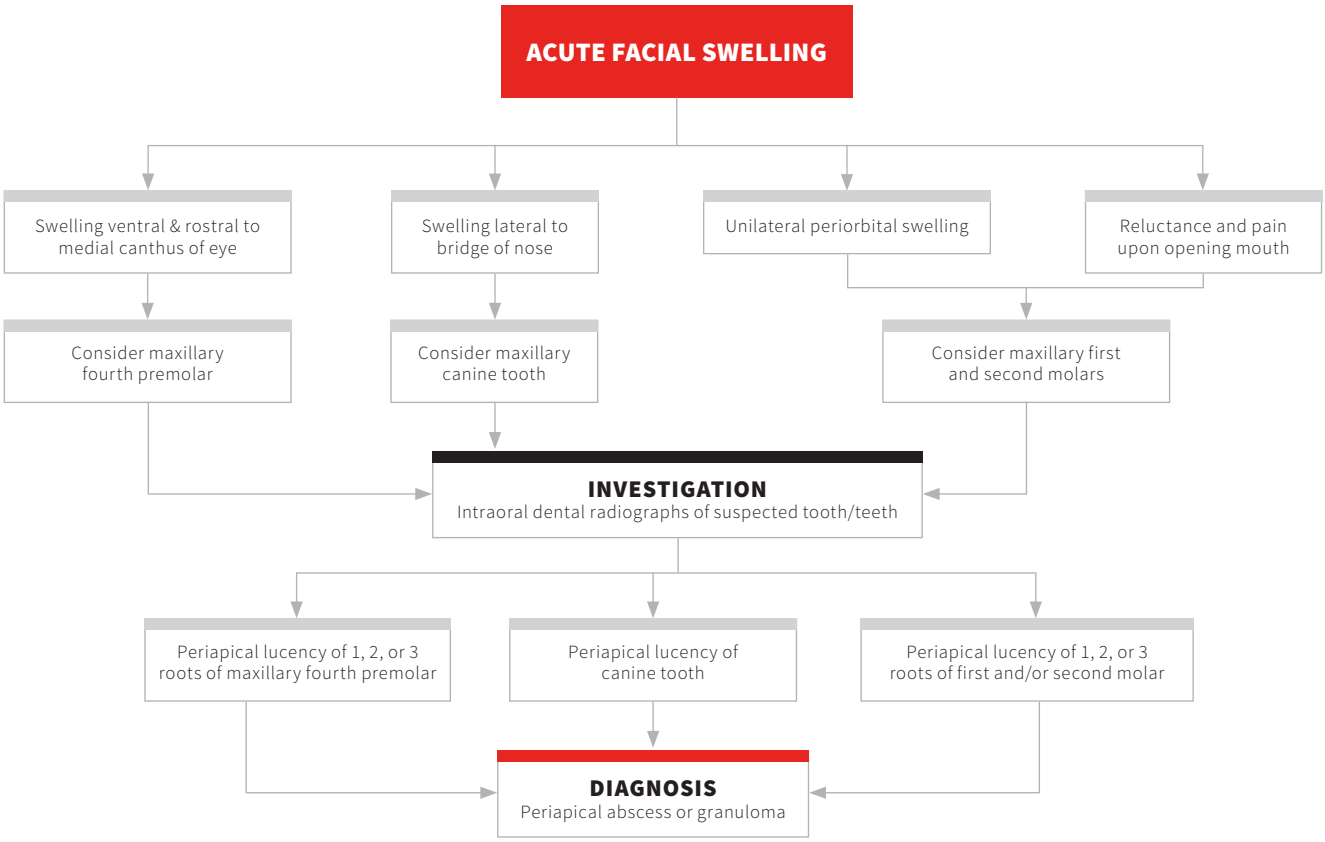


AED = antiepileptic drug
CRI = constant rate infusion
KBr = potassium bromide
NaBr = sodium bromide
PR = per rectum

*Airway, breathing, and circulatory disturbances should be treated first. Prolonged seizures may result in cerebral edema, necessitating furosemide (1-2 mg/kg IV), 20% mannitol (0.5-1 g/kg, over 15-20 min IV), and/or dexamethasone NaP (0.05-0.3 mg/kg IV). Underlying disease must be treated.
†Patients on phenobarbital may require higher doses because of hepatic enzyme induction; blood AED levels should be collected. Patients on phenobarbital may receive a small load of 4 mg/kg IV.
‡Endotracheal intubation ± oxygen and ventilation is necessary.

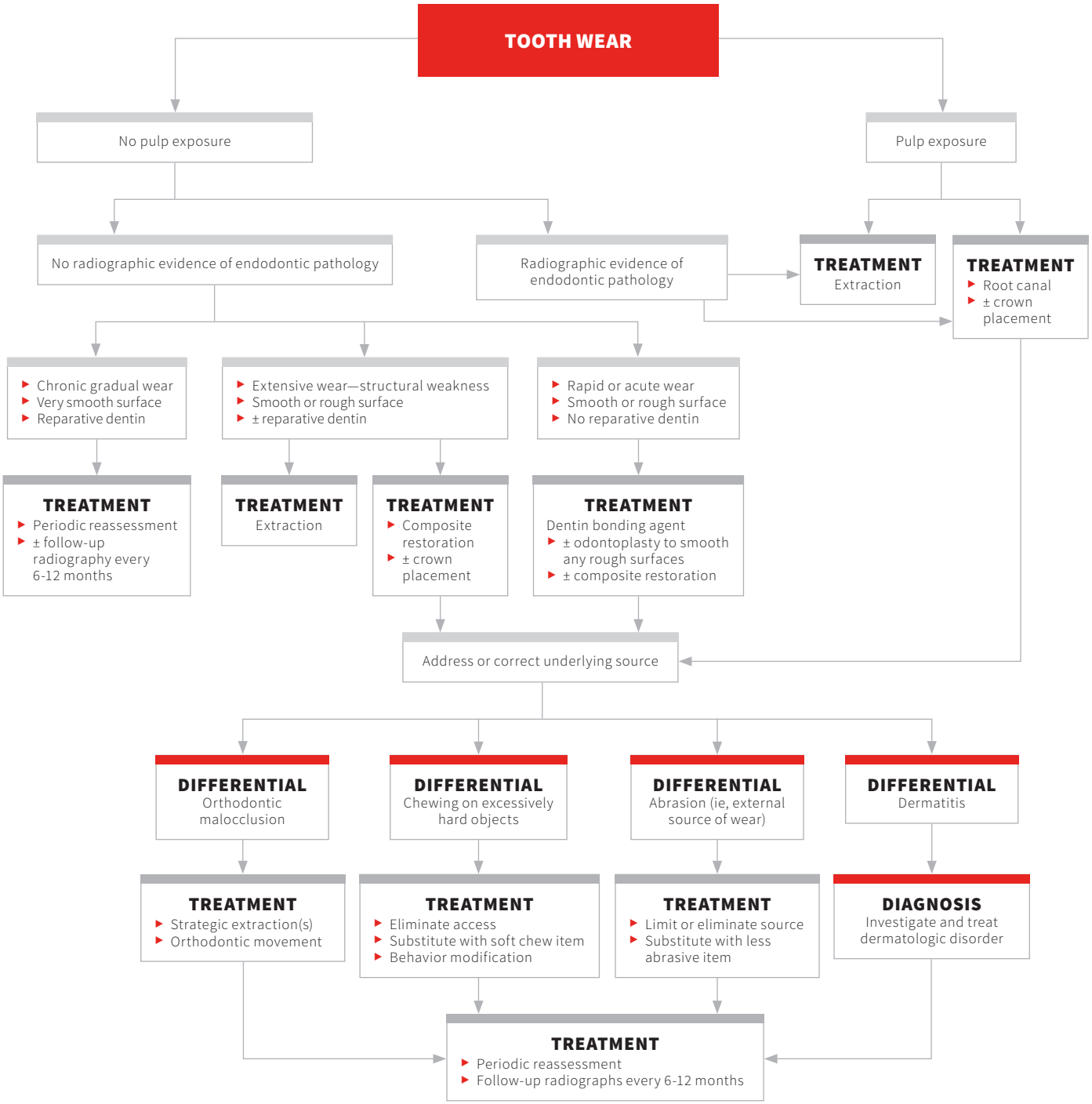
ACUTE FACIAL SWELLING & DENTAL DISEASE

Linda J. DeBowes, DVM, MS, DACVIM (SAIM), DAVDC



DENTAL WEAR: ATTRITION & ABRASION

Donald E. Beebe, DVM, DAVDC
Apex Dog & Cat Dentistry
Englewood, Colorado



TOOTH WEAR TYPES

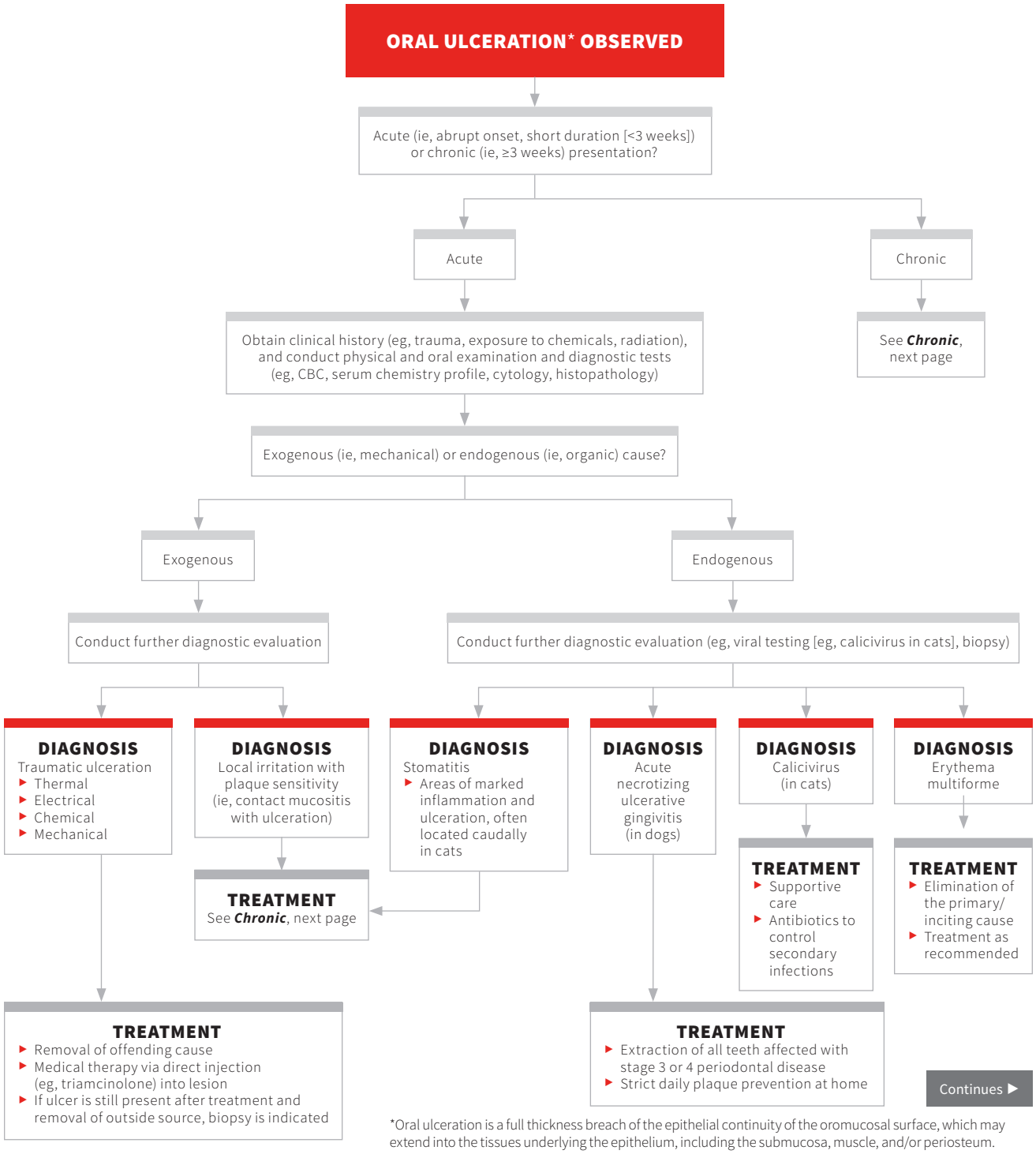
- Abrasion is mechanical wear of teeth from external forces (eg, brushing, dental instruments), also defined as wear from chewing on abrasive objects (eg, tennis balls, hair).
- Attrition is gradual physiologic wear resulting from natural mastication.
- Pathologic attrition is excessive wear caused by heavy chewing, biting, or grinding against other teeth (eg, orthodontic malocclusion).

FAST FACTS

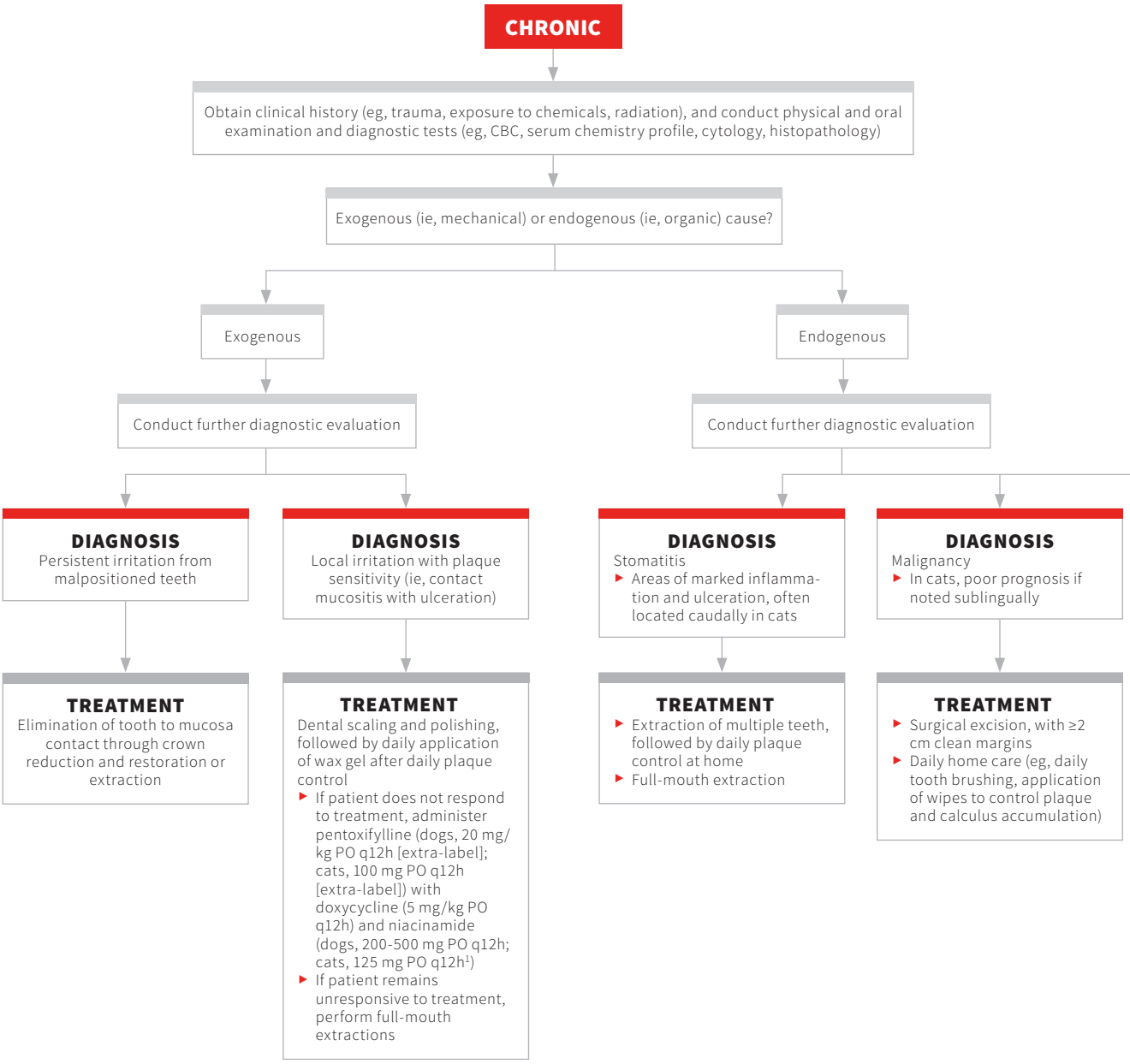
- Dental explorers have a sharp tip that helps determine whether a worn surface is smooth from gradual wear or rough from minor fractures; it can also probe for pulp exposure. Its use is mostly reserved for anesthetized patients; diligent caution should be used with nonanesthesized patients.
- Reparative dentin is denser than regular dentin, lacks organized tubules, is produced during tooth wear, and acts as a protective barrier. Pulp recedes behind the deposited mineralized layer and remains shielded from exposure.
- Intraoral radiographs are essential for evaluating compromised teeth.
- Odontoplasty is the adjustment of tooth contours. Sharp edges can be smoothed with hand or powered instrumentation. Small surface defects can be restored with dental composite.
- Signs of endodontic pathology may include wider-than-normal pulp canals from odontoblast death and delayed maturation, strictured or obliterated pulp canals from accelerated calcification (can occur during pulpitis), periapical radiolucency, or internal or external root resorption.

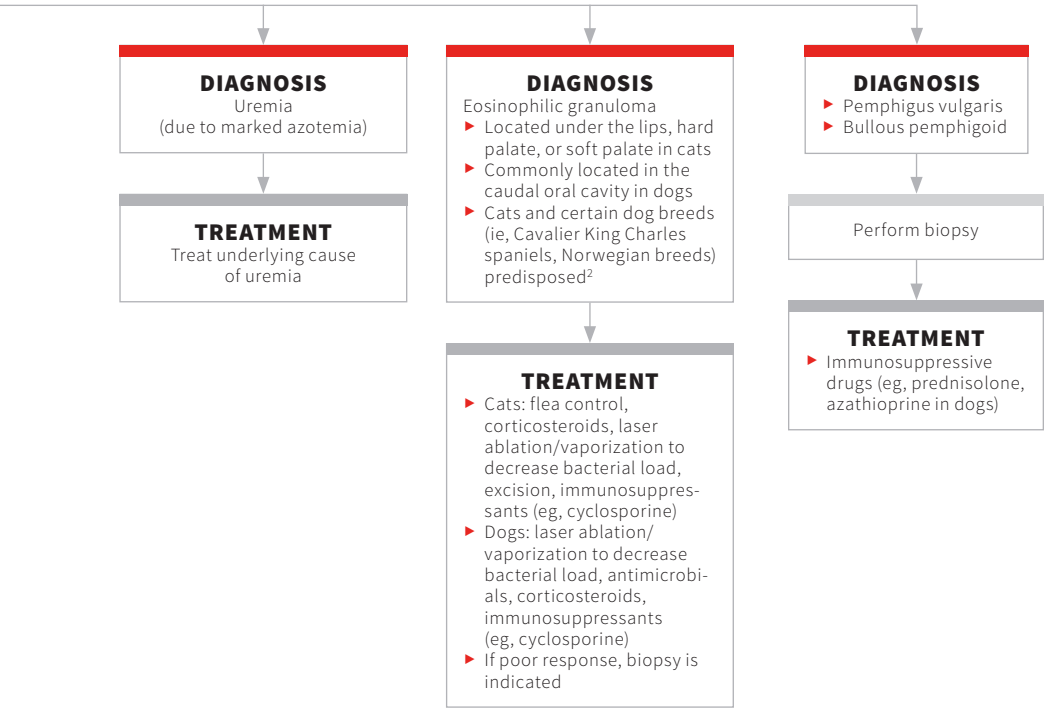
DIAGNOSING ORAL ULCERATION IN DOGS & CATS

Jan Bellows, DVM, FAVD, DAVDC, DABVP
All Pets Dental
Weston, Florida



*Oral ulceration is a full thickness breach of the epithelial continuity of the oromucosal surface, which may extend into the tissues underlying the epithelium, including the submucosa, muscle, and/or periosteum.


CHRONIC
Obtain clinical history (eg, trauma, exposure to chemicals, radiation), and conduct physical and oral examination and diagnostic tests (eg, CBC, serum chemistry profile, cytology, histopathology)
Exogenous (ie, mechanical) or endogenous (ie, organic) cause?
Exogenous
Endogenous
Conduct further diagnostic evaluation
Conduct further diagnostic evaluation
DIAGNOSIS
Persistent irritation from malpositioned teeth
DIAGNOSIS
Local irritation with plaque sensitivity (ie, contact mucositis with ulceration)
DIAGNOSIS
Stomatitis
Areas of marked inflammation and ulceration, often located caudally in cats
DIAGNOSIS
Malignancy
In cats, poor prognosis if noted sublingually
TREATMENT
Elimination of tooth to mucosa contact through crown reduction and restoration or extraction
TREATMENT
Dental scaling and polishing, followed by daily application of wax gel after daily plaque control
If patient does not respond to treatment, administer pentoxifylline (dogs, 20 mg/kg PO q12h [extra-label]; cats, 100 mg PO q12h [extra-label]) with doxycycline (5 mg/kg PO q12h) and niacinamide (dogs, 200-500 mg PO q12h; cats, 125 mg PO q12h[1])
If patient remains unresponsive to treatment, perform full-mouth extractions
TREATMENT
Extraction of multiple teeth, followed by daily plaque control at home
Full-mouth extraction
TREATMENT
Surgical excision, with ≥2 cm clean margins
Daily home care (eg, daily tooth brushing, application of wipes to control plaque and calculus accumulation)



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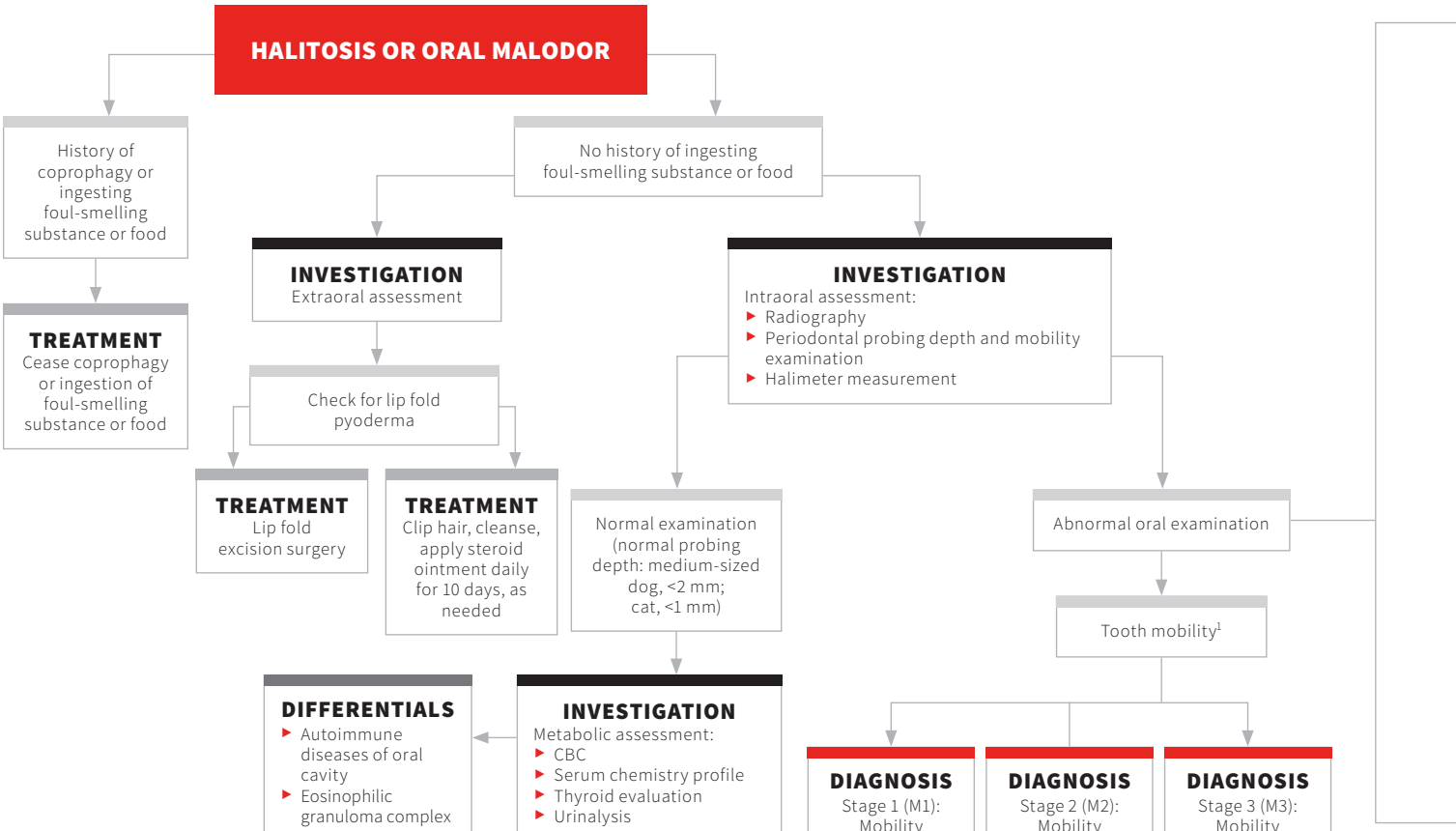
HALITOSIS

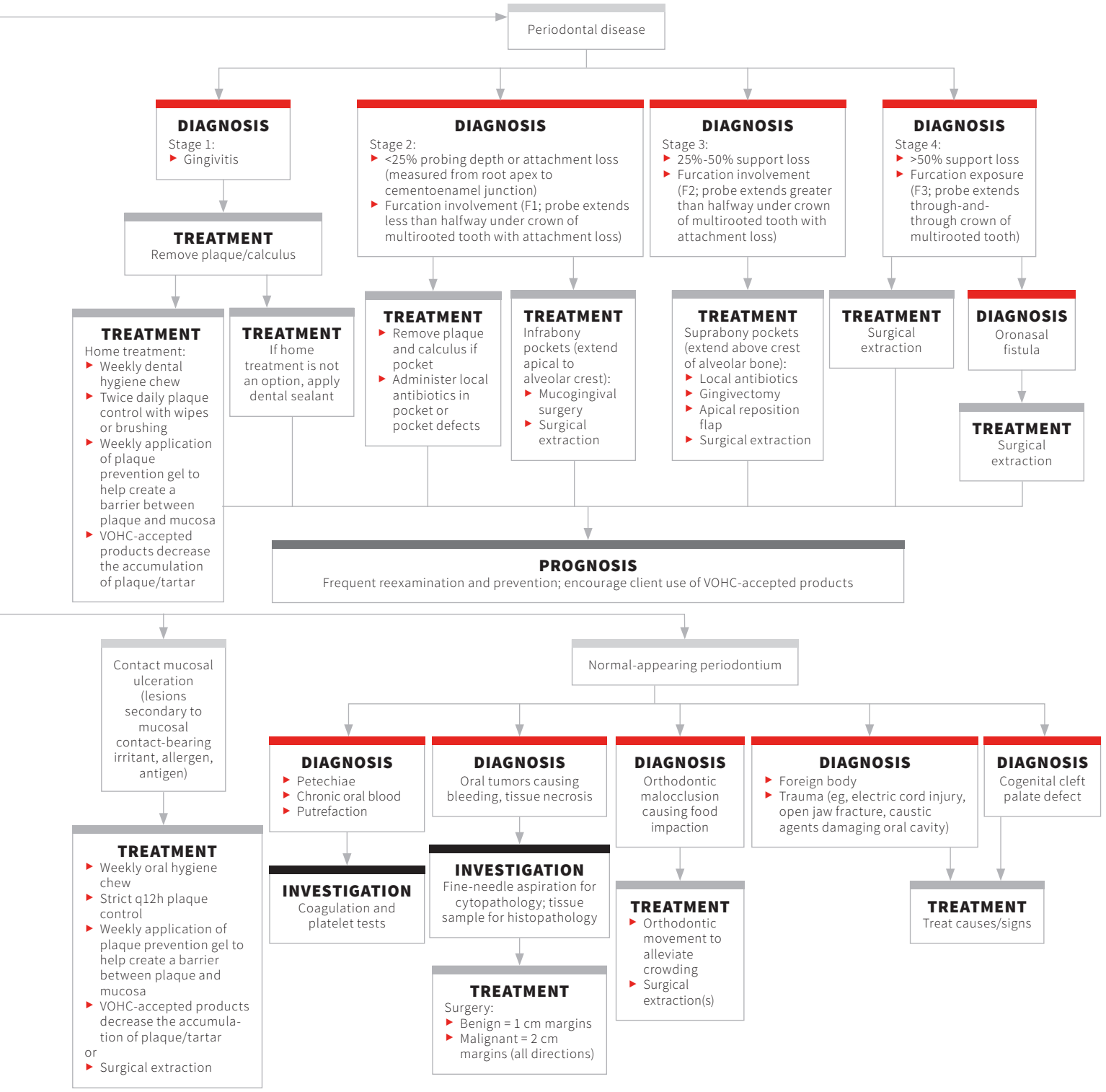
Jan Bellows, DVM, FAVD, DAVDC, DABVP
All Pets Dental
Weston, Florida



TYPES OF HALITOSIS CAUSED BY LOCAL AND METABOLIC CONDITIONS

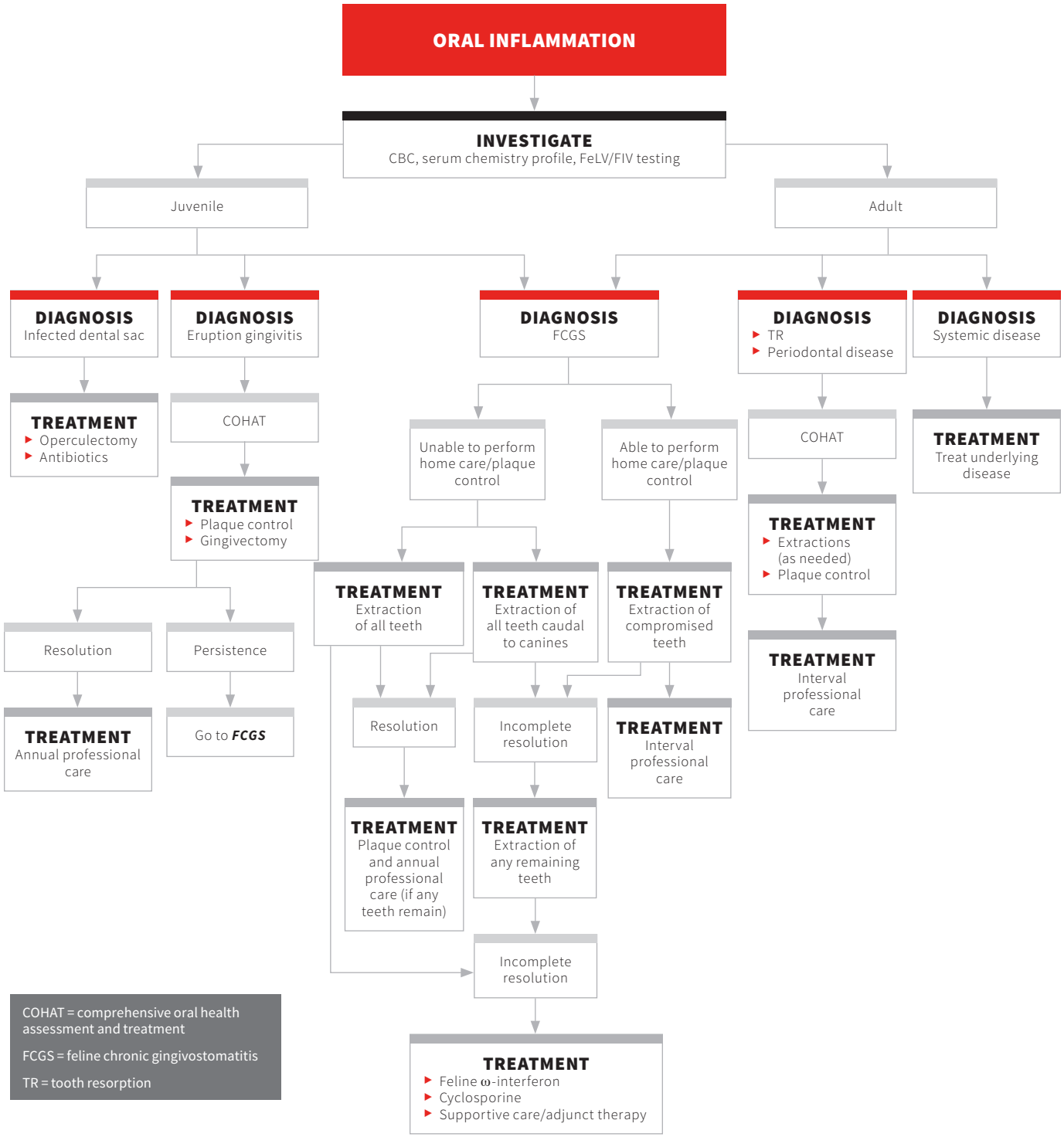
- Fetor oris/fetor ex ore: offensive odor arising from sources in the mouth
- Ozostomia: putrid smell detected in the mouth but derived from upper respiratory tract (eg, nasal passages, sinus cavities, pharynx, larynx)
- Stomatodysodia: foul breath originating from local areas in lower respiratory tract, particularly below the carina from the bronchi, bronchioles and alveoli, or other contiguous parts of the lung (eg, pleura)

VOHC = Veterinary Oral Health Council


Periodontal disease
DIAGNOSIS
Stage 1:
Gingivitis
TREATMENT
Remove plaque/calculus
TREATMENT
Home treatment:
Weekly dental hygiene chew
Twice daily plaque control with wipes or brushing
Weekly application of plaque prevention gel to help create a barrier between plaque and mucosa
VOHC-accepted products decrease the accumulation of plaque/tartar
TREATMENT
If home treatment is not an option, apply dental sealant
DIAGNOSIS
Stage 2:
<25% probing depth or attachment loss (measured from root apex to cementoenamel junction)
Furcation involvement (F1; probe extends less than halfway under crown of multirooted tooth with attachment loss)
TREATMENT
Remove plaque and calculus if pocket
Administer local antibiotics in pocket or pocket defects
TREATMENT
Infrabony pockets (extend apical to alveolar crest):
Mucogingival surgery
Surgical extraction
DIAGNOSIS
Stage 3:
25%-50% support loss
Furcation involvement (F2; probe extends greater than halfway under crown of multirooted tooth with attachment loss)
TREATMENT
Suprabony pockets (extend above crest of alveolar bone):
Local antibiotics
Gingivectomy
Apical reposition flap
Surgical extraction
DIAGNOSIS
Stage 4:
>50% support loss
Furcation exposure (F3; probe extends through-and-through crown of multirooted tooth)
TREATMENT
Surgical extraction
DIAGNOSIS
Oronasal fistula
TREATMENT
Surgical extraction
PROGNOSIS
Frequent reexamination and prevention; encourage client use of VOHC-accepted products
Contact mucosal ulceration (lesions secondary to mucosal contact-bearing irritant, allergen, antigen)
TREATMENT
Weekly oral hygiene chew
Strict q12h plaque control
Weekly application of plaque prevention gel to help create a barrier between plaque and mucosa
VOHC-accepted products decrease the accumulation of plaque/tartar
or
Surgical extraction
Normal-appearing periodontium
DIAGNOSIS
Petechiae
Chronic oral blood
Putrefaction
INVESTIGATION
Coagulation and platelet tests
DIAGNOSIS
Oral tumors causing bleeding, tissue necrosis
INVESTIGATION
Fine-needle aspiration for cytopathology; tissue sample for histopathology
TREATMENT
Surgery:
Benign = 1 cm margins
Malignant = 2 cm margins (all directions)
DIAGNOSIS
Orthodontic malocclusion causing food impaction
TREATMENT
Orthodontic movement to alleviate crowding
Surgical extraction(s)
DIAGNOSIS
Foreign body
Trauma (eg, electric cord injury, open jaw fracture, caustic agents damaging oral cavity)
DIAGNOSIS
Cogenital cleft palate defect
TREATMENT
Treat causes/signs

ORAL INFLAMMATION IN CATS

Judy Rochette, DVM, FAVD, DAVDC
West Coast Veterinary Dental Services
Vancouver, British Columbia



PERIODONTAL STAGING: ASSOCIATED TREATMENT OPTIONS

Mark M. Smith, VMD, DACVS, DAVDC*
Virginia Tech

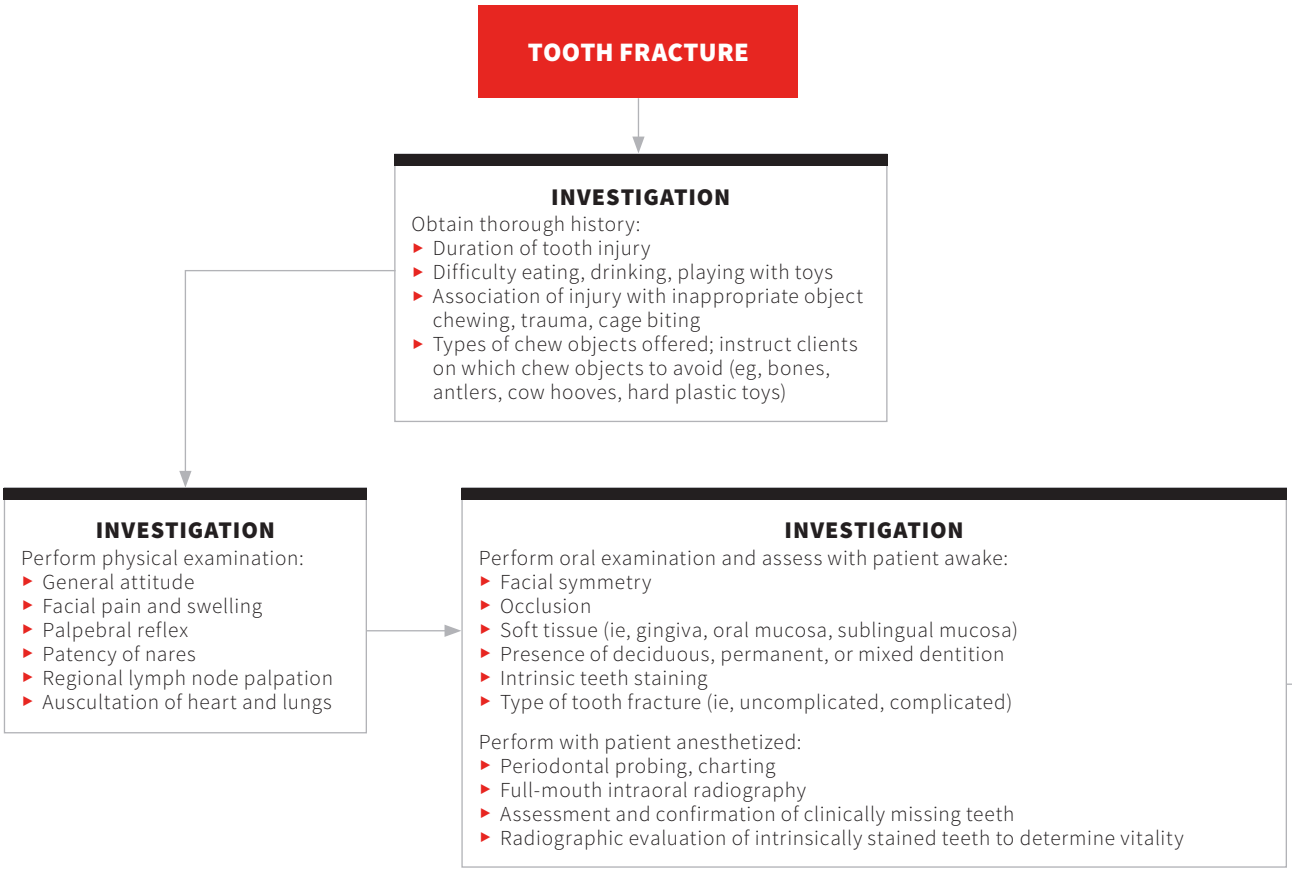
STAGE 0	▸ No attachment loss ▸ Typical plaque and calculus accumulation	**TREATMENT** ▸ Annual professional teeth cleaning • No prophylactic antimicrobial† ▸ No systemic antimicrobial ▸ Home care
STAGE 1	▸ No attachment loss ▸ Gingivitis, plaque, and calculus accumulation	**TREATMENT** ▸ Annual professional teeth cleaning • No prophylactic antimicrobial† ▸ No systemic antimicrobial ▸ Home care
STAGE 2	▸ Beginning (<25%) attachment loss ▸ Periodontal pockets 3-5 mm ▸ Gingivitis, plaque, and calculus accumulation	**TREATMENT** ▸ Annual professional teeth cleaning • No prophylactic antimicrobial† • Local doxycycline or clindamycin gel in periodontal pockets ≥4 mm ▸ Systemic antimicrobial for 14 days • Clindamycin • Amoxicillin–clavulanic acid ▸ Home care
STAGE 3	▸ Moderate (25%-50%) attachment loss ▸ Periodontal pockets 5-7 mm ▸ Gingivitis, plaque, and calculus accumulation	**TREATMENT** ▸ Semiannual professional teeth cleaning • No prophylactic antimicrobial† ▸ Systemic antimicrobial for 14 days • Clindamycin • Amoxicillin–clavulanic acid ▸ Local doxycycline or clindamycin gel in pockets ≥4 mm ▸ Home care beginning 14 days following treatment
STAGE 4	▸ Severe (>50%) attachment loss ▸ Periodontal pockets >7 mm ▸ Gingivitis, plaque, and calculus accumulation	**TREATMENT** ▸ Semiannual professional teeth cleaning • No prophylactic antimicrobial† ▸ Systemic antimicrobial for 14 days • Clindamycin • Amoxicillin–clavulanic acid ▸ Open surgical debridement in periodontal pockets ≥7 mm ▸ Local doxycycline or clindamycin gel in periodontal pockets ≥4 mm or ▸ Root planing and apically repositioned flap to eliminate pocket or ▸ Tooth extraction ▸ Home care beginning 14 days following treatment

*Byline reflects author information on original publication. On publication of this collection, the author's current affiliation is the Center for Veterinary Dentistry & Oral Surgery, Gaithersburg, Maryland.

†Preoperative IV antimicrobial therapy using a broad-spectrum bactericidal agent (eg, sodium ampicillin) is recommended for use in high risk patients with advanced periodontitis, immunosuppression secondary to metabolic disease, primary immunopathies, artificial implants, or cardiac disease.

APPROACH TO TOOTH FRACTURE*

Kendall Taney, DVM, DAVDC, FAVD
Center for Veterinary Dentistry & Oral Surgery
Gaithersburg, Maryland



Suggested Reading

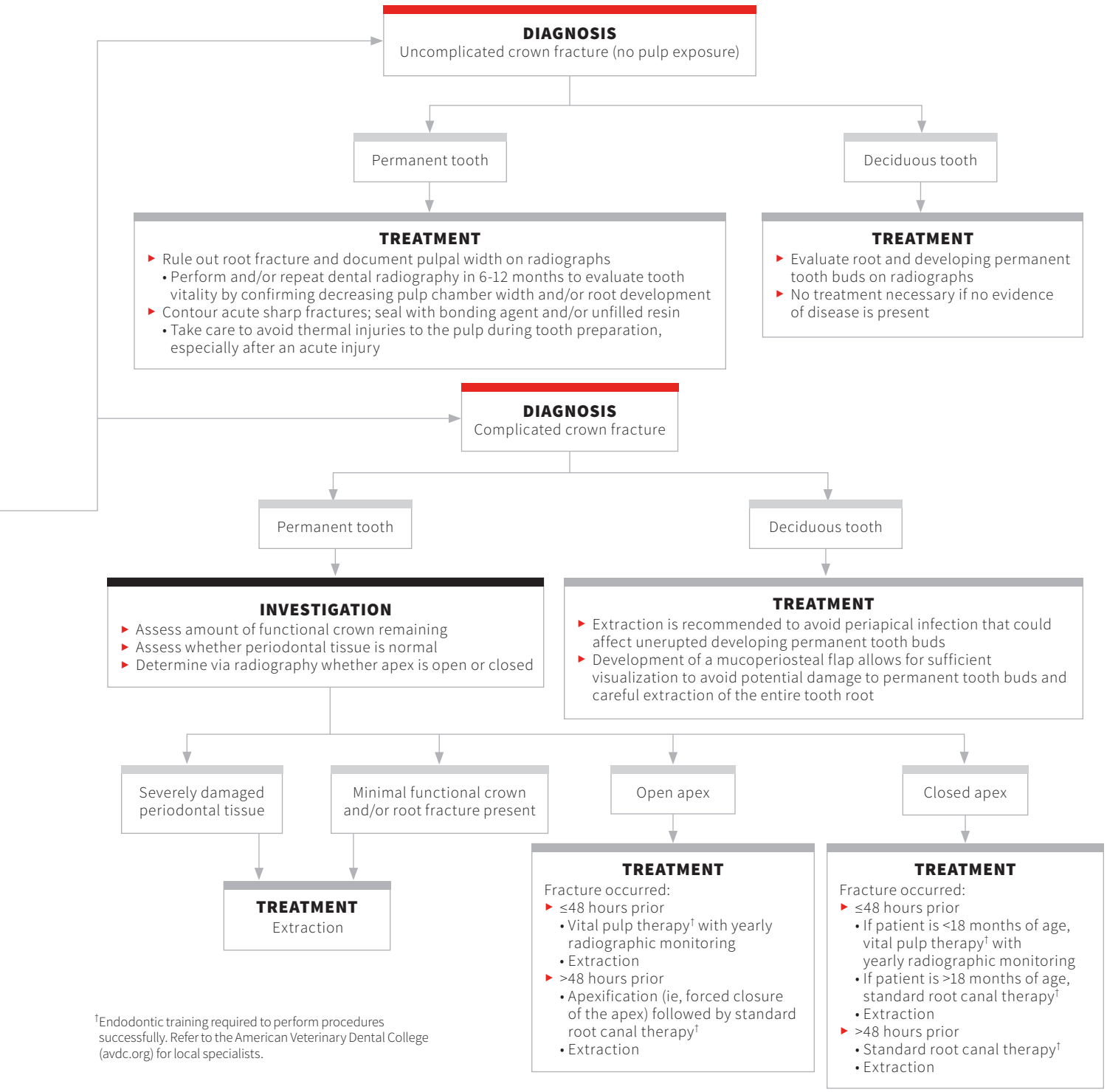
[Articles and step-by-step editions.] *J Vet Dent*. jov.sagepub.com.

AVDC Nomenclature Committee. Recommendations adopted by the AVDC board. American Veterinary Dental College website. http:// www.avdc.org/nomenclature3.html#toothfracture. Published October 2009. Accessed November 21, 2016.

Girard N, Southerden P, Hennet P. Root canal treatment in dogs and cats. *J Vet Dent*. 2006;23(3):148-160.

Luotenen N, Kuntsi-Vaattovaara H, Sarkiala-Kessel E, Junnila JJ, Laitinen-Vapaavuori O, Verstraete FJ. Vital pulp therapy in dogs: 190 cases (2001-2011). *J Am Vet Med Assoc*. 2014;244(4):449-459.

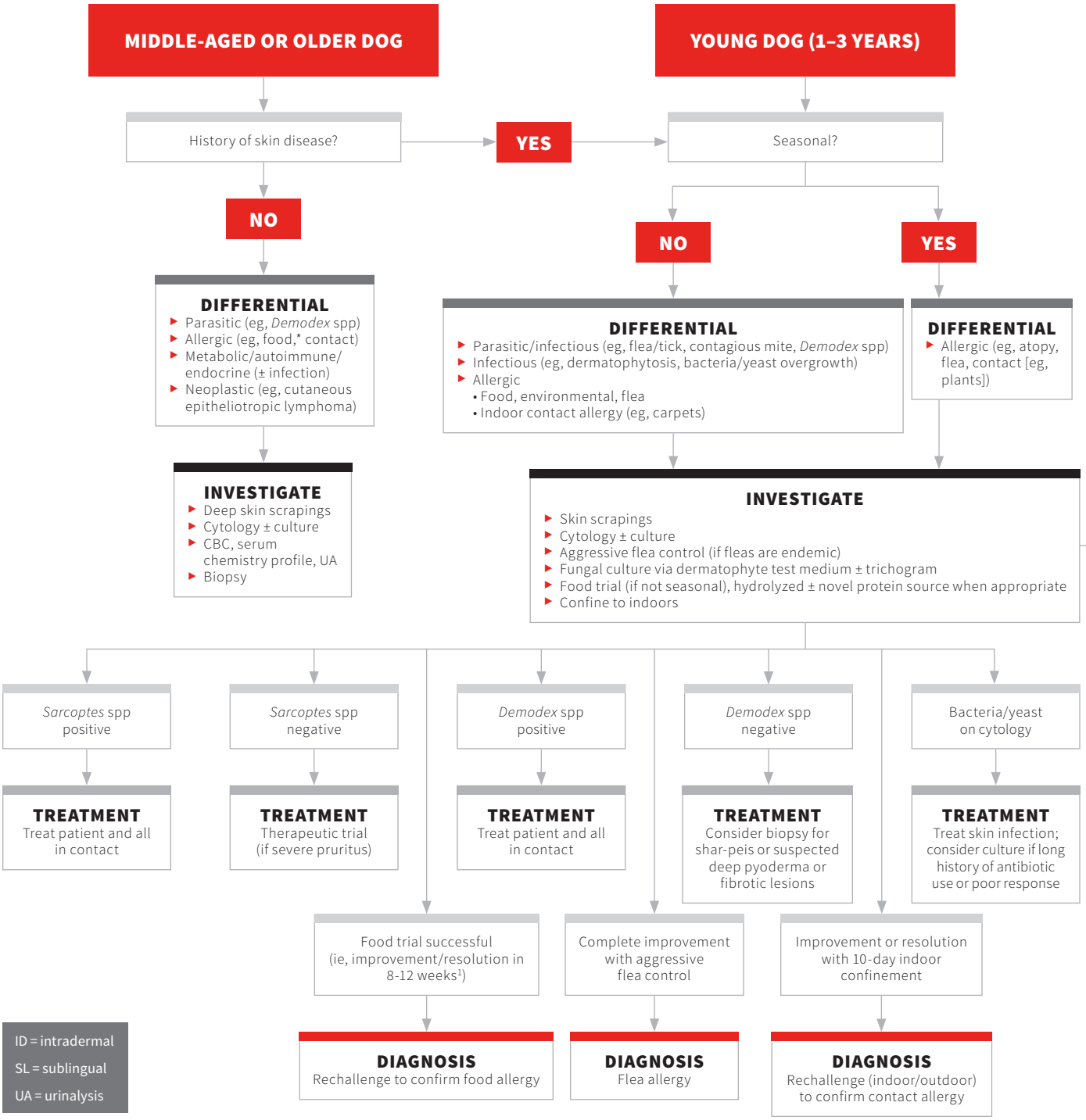
*Originally published as "Tooth Fracture in a Pediatric Patient"

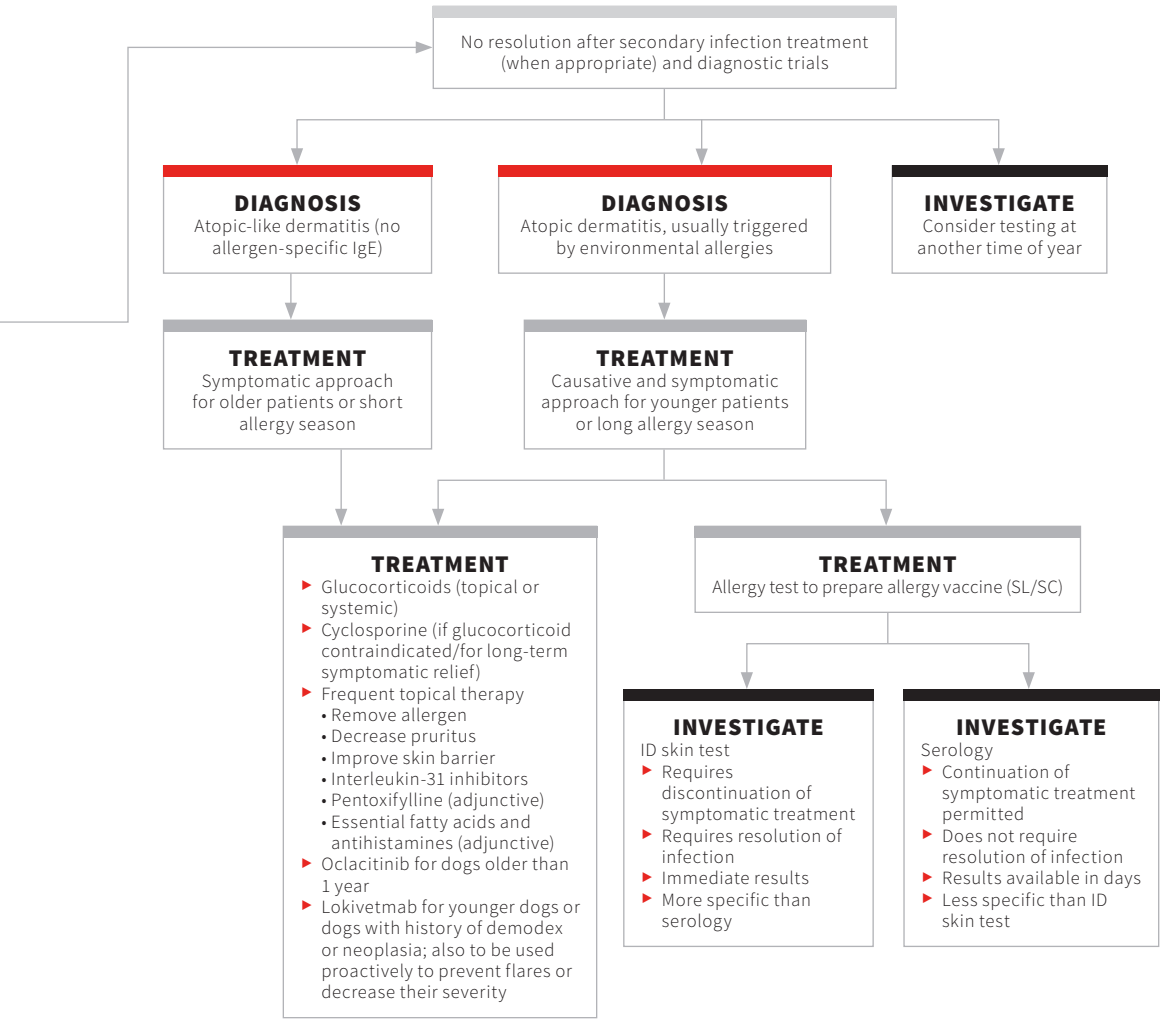


†Endodontic training required to perform procedures successfully. Refer to the American Veterinary Dental College (avdc.org) for local specialists.

CANINE PRURITIC DERMATITIS

Rosanna Marsella, DVM, DACVD
University of Florida





Reference

1. Rosser EJ Jr. Diagnosis of food allergy in dogs. *J Am Vet Med Assoc*. 1993;203(2):259-262.

Suggested Reading

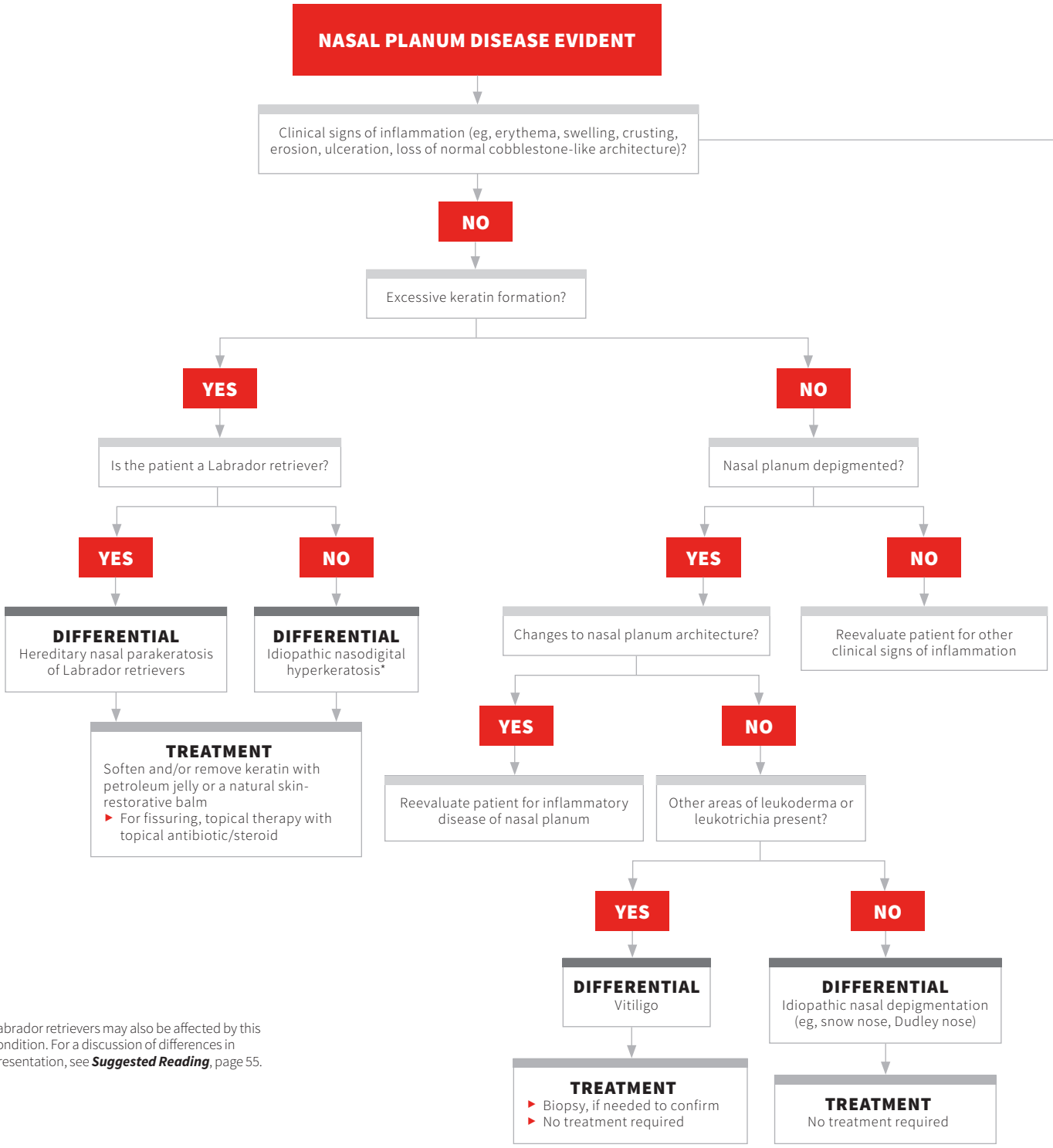
Jackson H, Marsella R, eds. *BSAVA Manual of Canine and Feline Dermatology*. 3rd ed. Quedgeley, Gloucester: BSAVA; 2012.

Marsella R, Sousa CA, Gonzales AJ, Fadok VA. Current understanding of the pathophysiologic mechanisms of canine atopic dermatitis. *J Am Vet Med Assoc*. 2012;241(2):194-207.

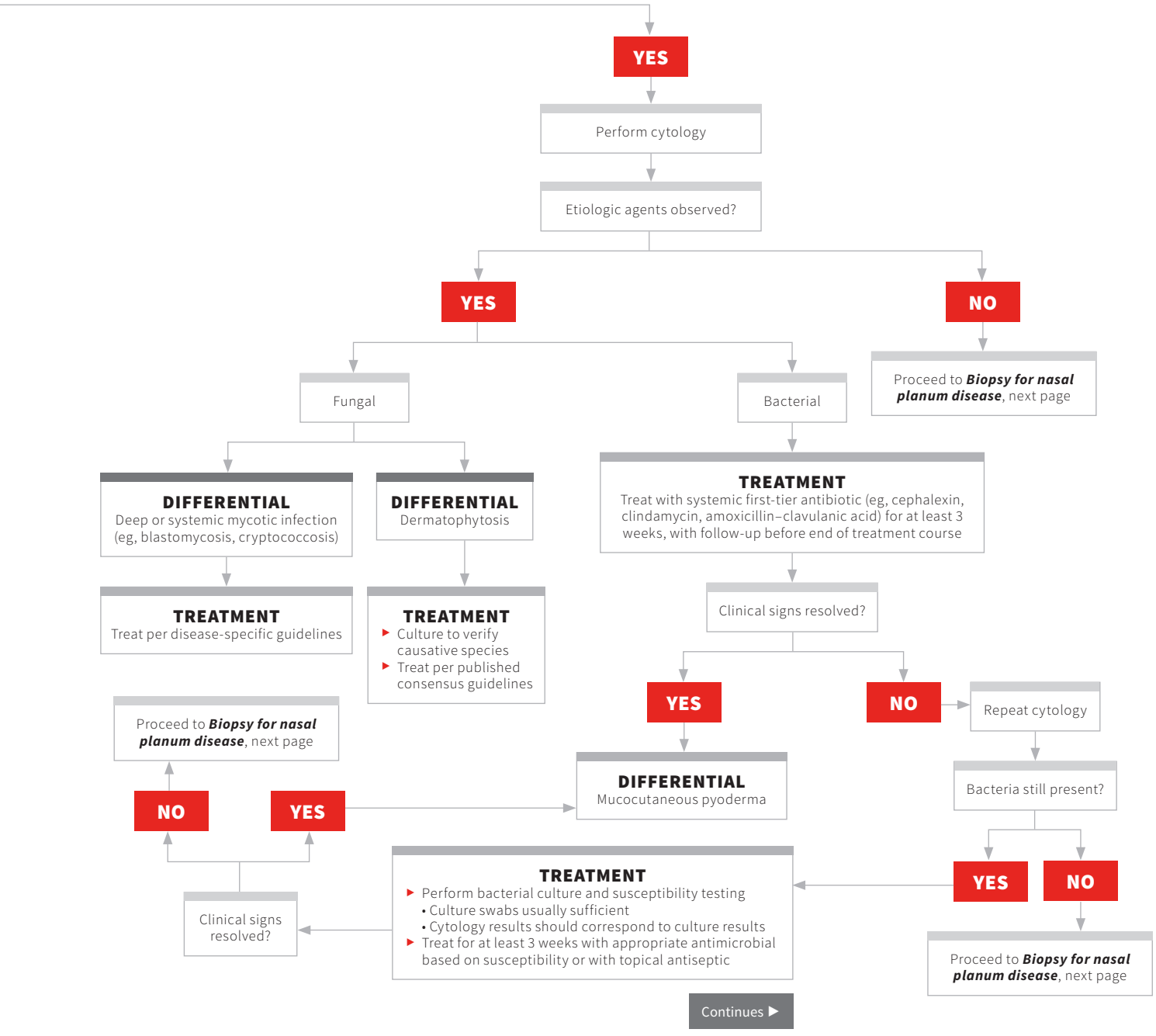
*Unlike food allergies, environmental and flea allergies do not commonly develop later in life; therefore, environmental and flea allergies are unlikely in middle-aged dogs unless the patient has a history of geographic movemement or environmental change.

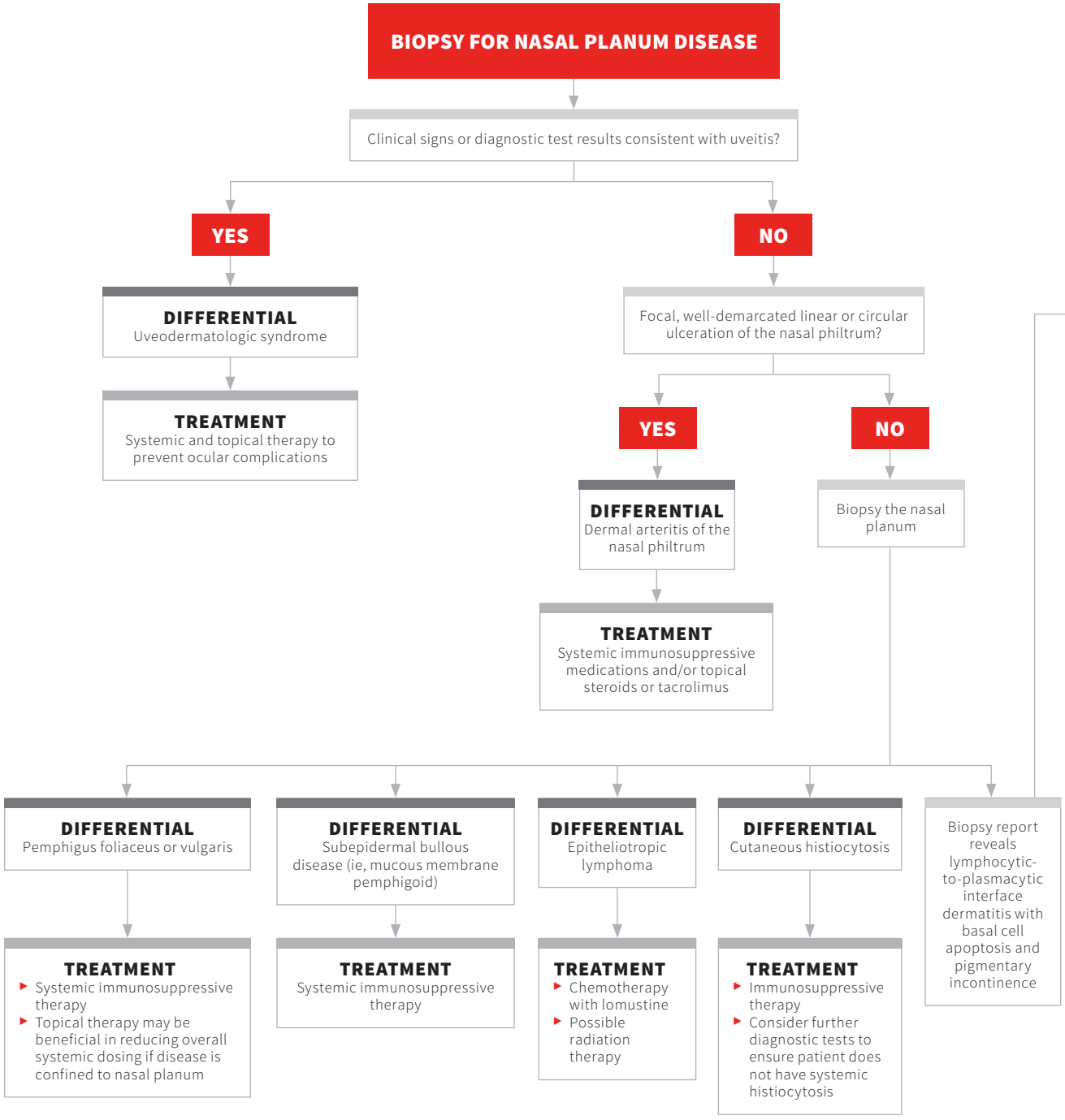
DIAGNOSING NASAL PLANUM DISEASE IN DOGS

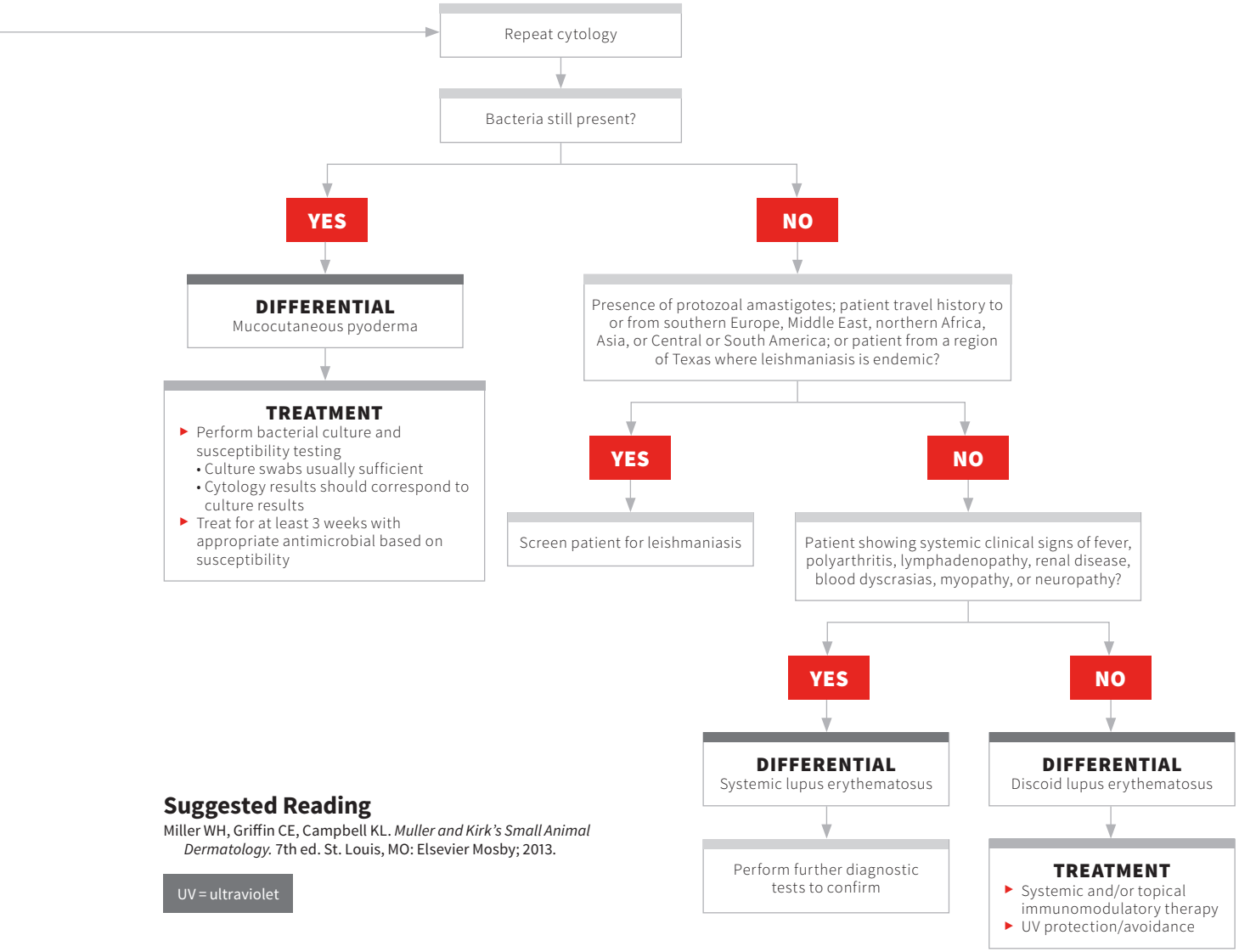
Darren Berger, DVM, DACVD
Iowa State University



*Labrador retrievers may also be affected by this condition. For a discussion of differences in presentation, see ***Suggested Reading***, page 55.


YES
Perform cytology
Etiologic agents observed?
YES
NO
Fungal
Bacterial
Proceed to ***Biopsy for nasal planum disease***, next page
DIFFERENTIAL
Deep or systemic mycotic infection (eg, blastomycosis, cryptococcosis)
DIFFERENTIAL
Dermatophytosis
TREATMENT
Treat with systemic first-tier antibiotic (eg, cephalexin, clindamycin, amoxicillin–clavulanic acid) for at least 3 weeks, with follow-up before end of treatment course
TREATMENT
Treat per disease-specific guidelines
TREATMENT
Culture to verify causative species
Treat per published consensus guidelines
Clinical signs resolved?
YES
NO
Repeat cytology
Proceed to ***Biopsy for nasal planum disease***, next page
DIFFERENTIAL
Mucocutaneous pyoderma
Bacteria still present?
NO
YES
YES
NO
TREATMENT
Perform bacterial culture and susceptibility testing
• Culture swabs usually sufficient
• Cytology results should correspond to culture results
Treat for at least 3 weeks with appropriate antimicrobial based on susceptibility or with topical antiseptic
Clinical signs resolved?
Proceed to ***Biopsy for nasal planum disease***, next page
Continues ▶


BIOPSY FOR NASAL PLANUM DISEASE
Clinical signs or diagnostic test results consistent with uveitis?
YES
NO
DIFFERENTIAL
Uveodermatologic syndrome
TREATMENT
Systemic and topical therapy to prevent ocular complications
Focal, well-demarcated linear or circular ulceration of the nasal philtrum?
YES
NO
DIFFERENTIAL
Dermal arteritis of the nasal philtrum
TREATMENT
Systemic immunosuppressive medications and/or topical steroids or tacrolimus
Biopsy the nasal planum
DIFFERENTIAL
Pemphigus foliaceus or vulgaris
TREATMENT
Systemic immunosuppressive therapy
Topical therapy may be beneficial in reducing overall systemic dosing if disease is confined to nasal planum
DIFFERENTIAL
Subepidermal bullous disease (ie, mucous membrane pemphigoid)
TREATMENT
Systemic immunosuppressive therapy
DIFFERENTIAL
Epitheliotropic lymphoma
TREATMENT
Chemotherapy with lomustine
Possible radiation therapy
DIFFERENTIAL
Cutaneous histiocytosis
TREATMENT
Immunosuppressive therapy
Consider further diagnostic tests to ensure patient does not have systemic histiocytosis
Biopsy report reveals lymphocytic-to-plasmacytic interface dermatitis with basal cell apoptosis and pigmentary incontinence



Suggested Reading

Miller WH, Griffin CE, Campbell KL. *Muller and Kirk's Small Animal Dermatology.* 7th ed. St. Louis, MO: Elsevier Mosby; 2013.

UV = ultraviolet

PERIORAL DERMATITIS IN DOGS

Jennifer Schissler Pendergraft, DVM, MS, DACVD*
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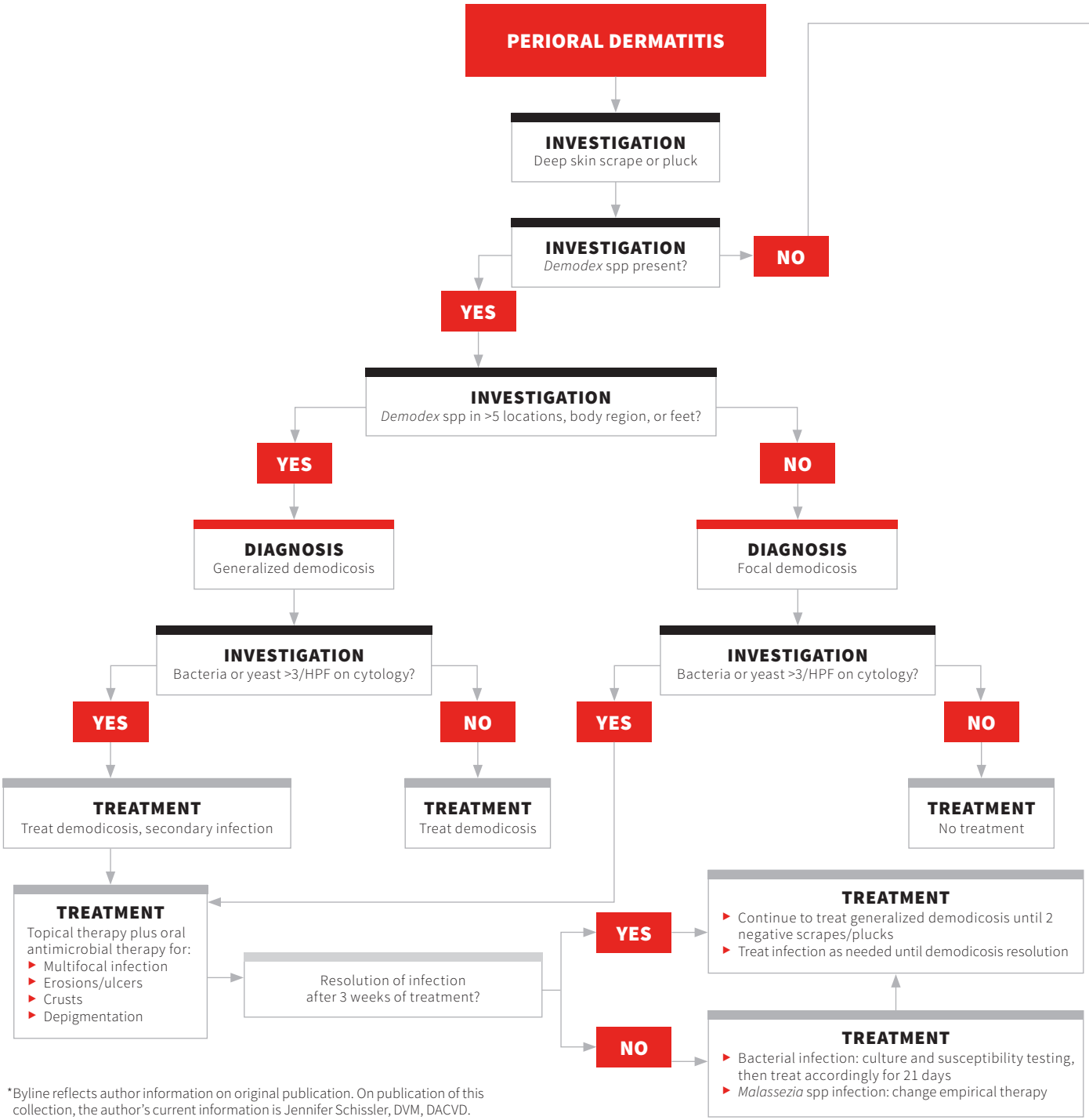
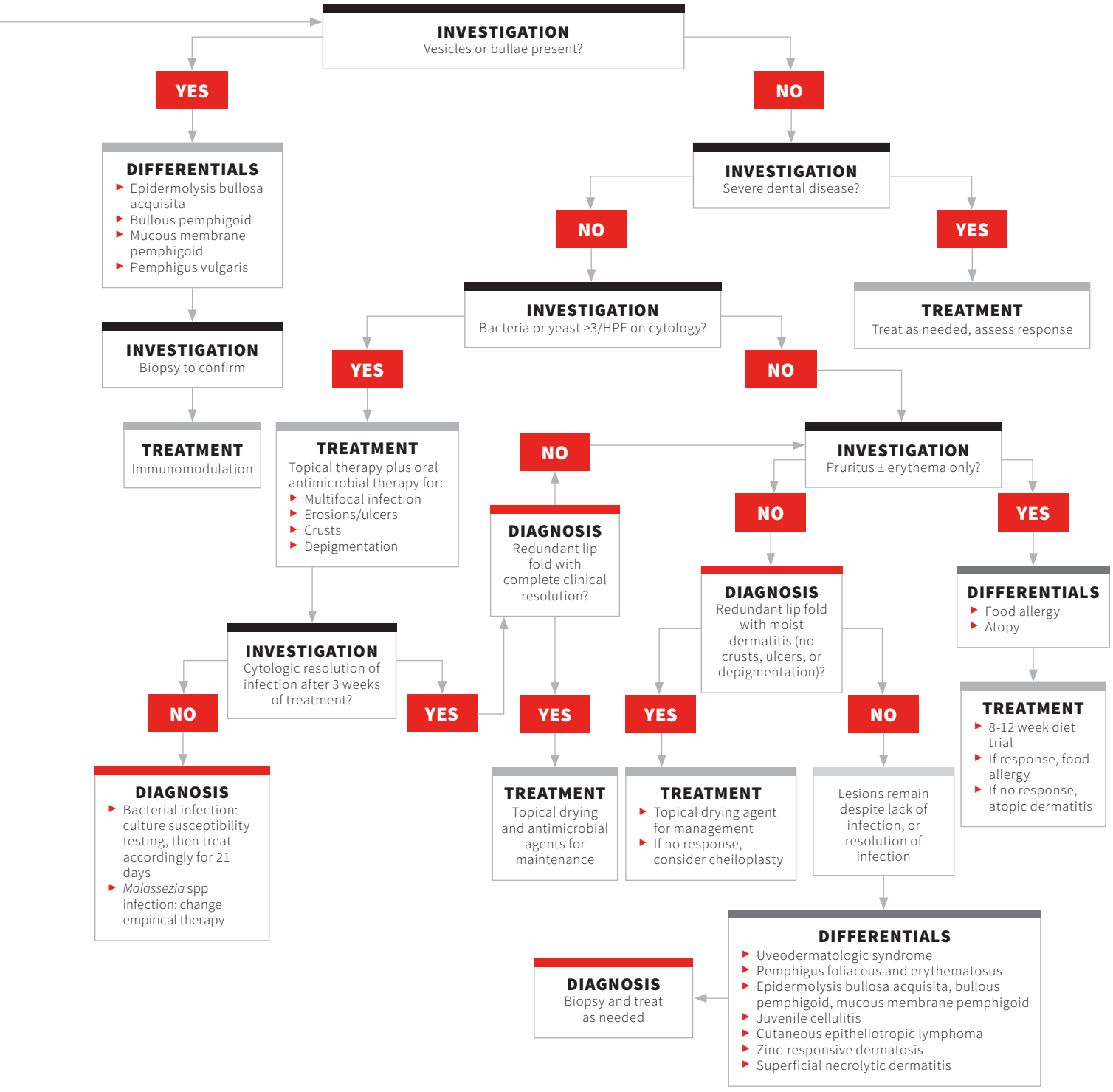
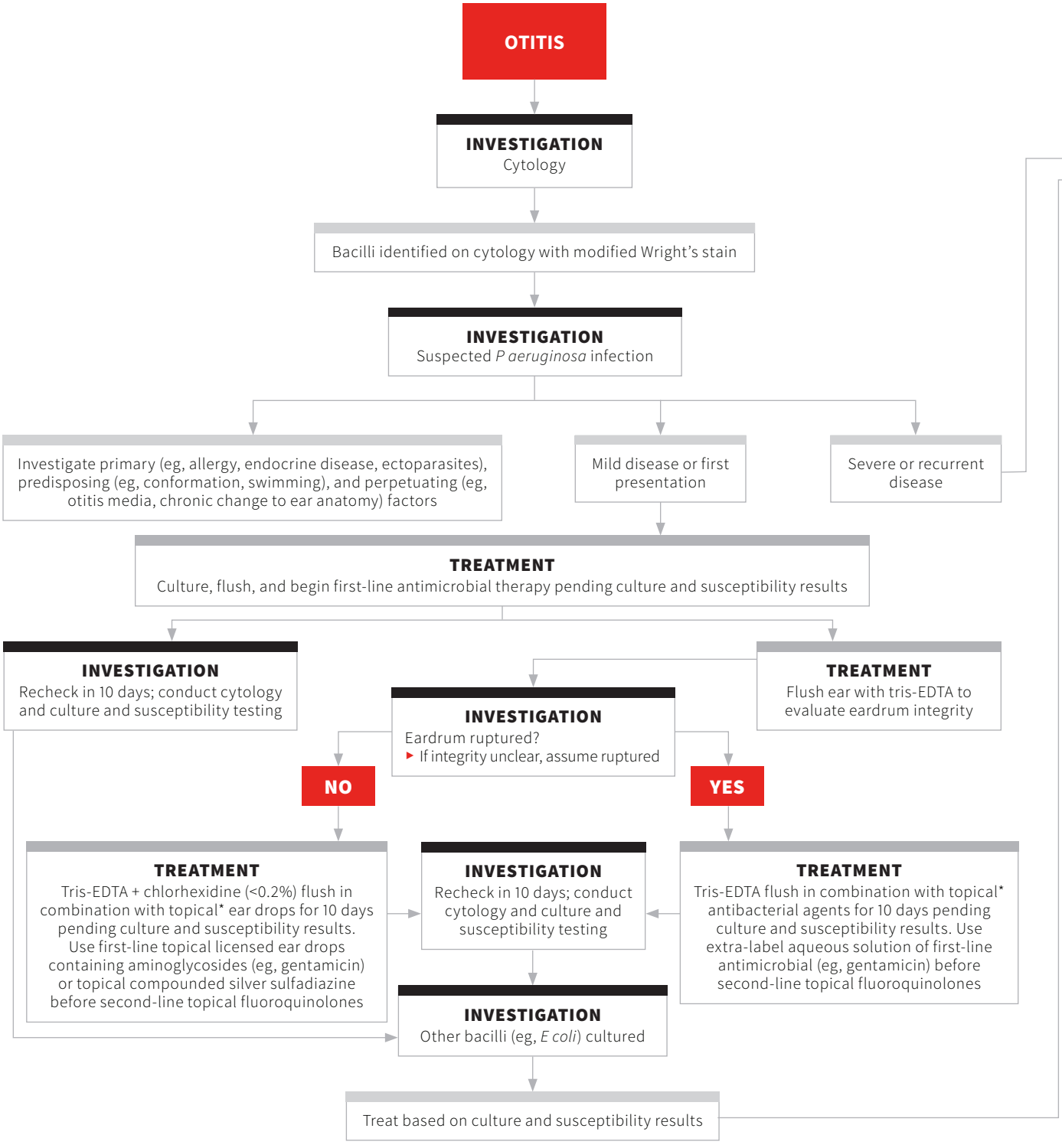


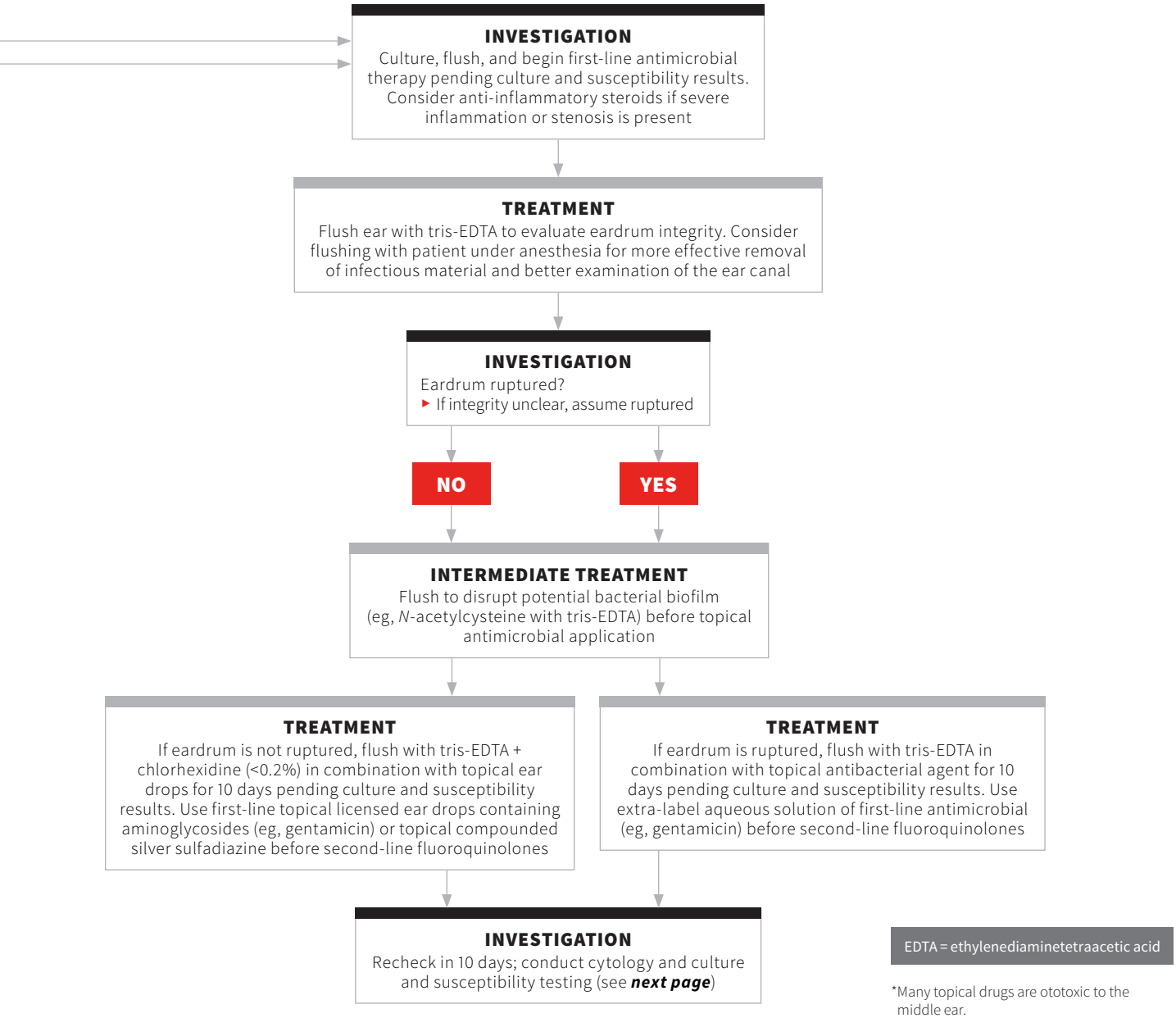
*Byline reflects author information on original publication. On publication of this collection, the author's current information is Jennifer Schissler, DVM, DACVD.


INVESTIGATION
Vesicles or bullae present?
YES
NO
DIFFERENTIALS
Epidermolysis bullosa acquisita
Bullous pemphigoid
Mucous membrane pemphigoid
Pemphigus vulgaris
INVESTIGATION
Biopsy to confirm
TREATMENT
Immunomodulation
INVESTIGATION
Severe dental disease?
NO
YES
TREATMENT
Treat as needed, assess response
INVESTIGATION
Bacteria or yeast >3/HPF on cytology?
YES
NO
TREATMENT
Topical therapy plus oral antimicrobial therapy for:
Multifocal infection
Erosions/ulcers
Crusts
Depigmentation
INVESTIGATION
Cytologic resolution of infection after 3 weeks of treatment?
NO
YES
DIAGNOSIS
Bacterial infection: culture susceptibility testing, then treat accordingly for 21 days
Malassezia spp infection: change empirical therapy
NO
DIAGNOSIS
Redundant lip fold with complete clinical resolution?
YES
TREATMENT
Topical drying and antimicrobial agents for maintenance
INVESTIGATION
Pruritus ± erythema only?
NO
YES
DIAGNOSIS
Redundant lip fold with moist dermatitis (no crusts, ulcers, or depigmentation)?
YES
TREATMENT
Topical drying agent for management
If no response, consider cheiloplasty
NO
Lesions remain despite lack of infection, or resolution of infection
DIFFERENTIALS
Food allergy
Atopy
TREATMENT
8-12 week diet trial
If response, food allergy
If no response, atopic dermatitis
DIFFERENTIALS
Uveodermatologic syndrome
Pemphigus foliaceus and erythematosus
Epidermolysis bullosa acquisita, bullous pemphigoid, mucous membrane pemphigoid
Juvenile cellulitis
Cutaneous epitheliotropic lymphoma
Zinc-responsive dermatosis
Superficial necrolytic dermatitis
DIAGNOSIS
Biopsy and treat as needed

PSEUDOMONAS AERUGINOSA OTITIS EXTERNA & MEDIA

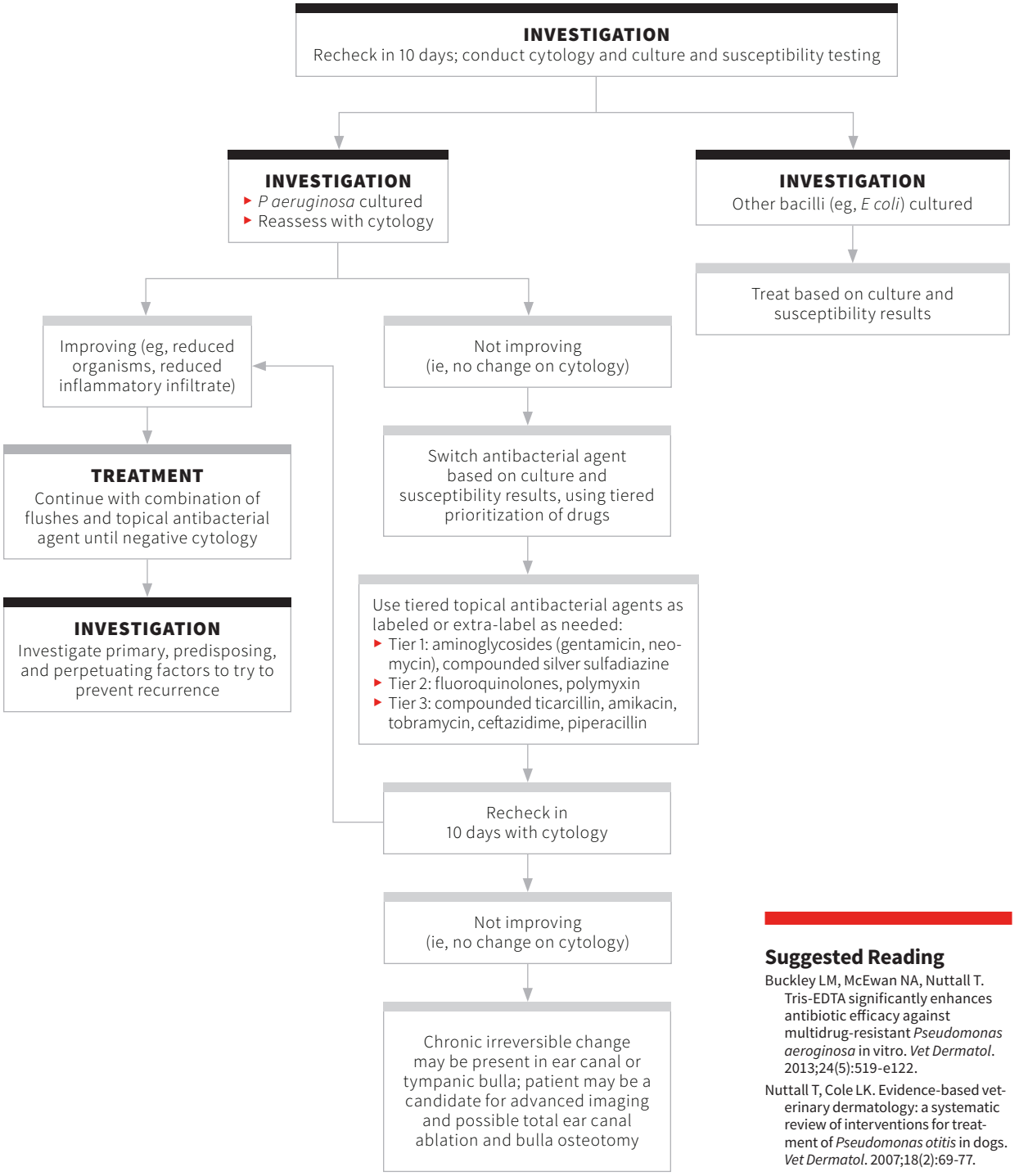
Susan Paterson, VetMB, MA, DVD, DECVD, MRCVS
Virtual Vet Derms
Kendal, United Kingdom





EDTA = ethylenediaminetetraacetic acid

*Many topical drugs are ototoxic to the middle ear.



Suggested Reading

Buckley LM, McEwan NA, Nuttall T. Tris-EDTA significantly enhances antibiotic efficacy against multidrug-resistant *Pseudomonas aeroginosa* in vitro. *Vet Dermatol*. 2013;24(5):519-e122.

Nuttall T, Cole LK. Evidence-based veterinary dermatology: a systematic review of interventions for treatment of *Pseudomonas otitis* in dogs. *Vet Dermatol*. 2007;18(2):69-77.

REFRACTORY DERMATOPHYTOSIS: 5 KEY EVALUATIONS WHEN TREATMENT FAILS

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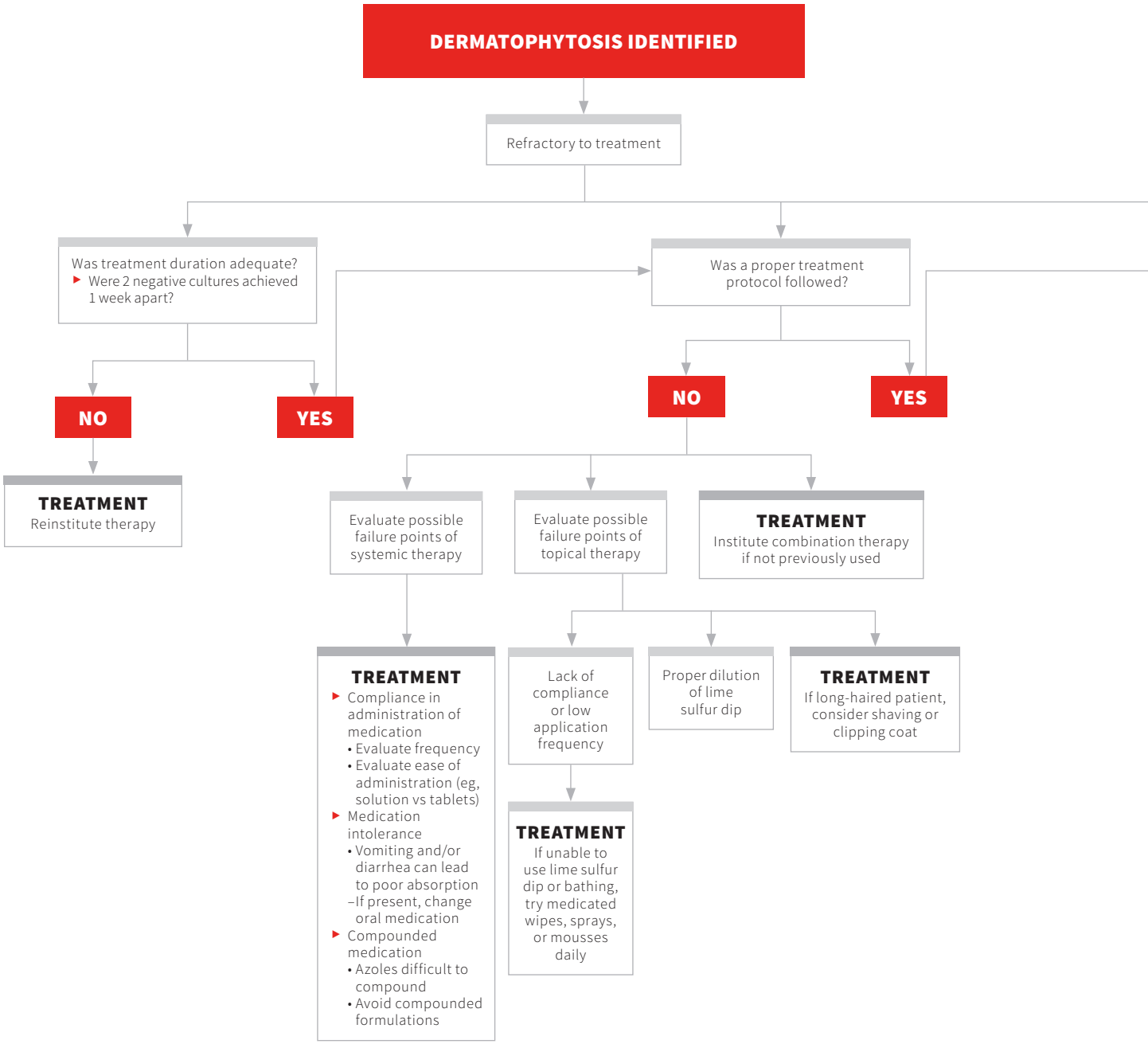

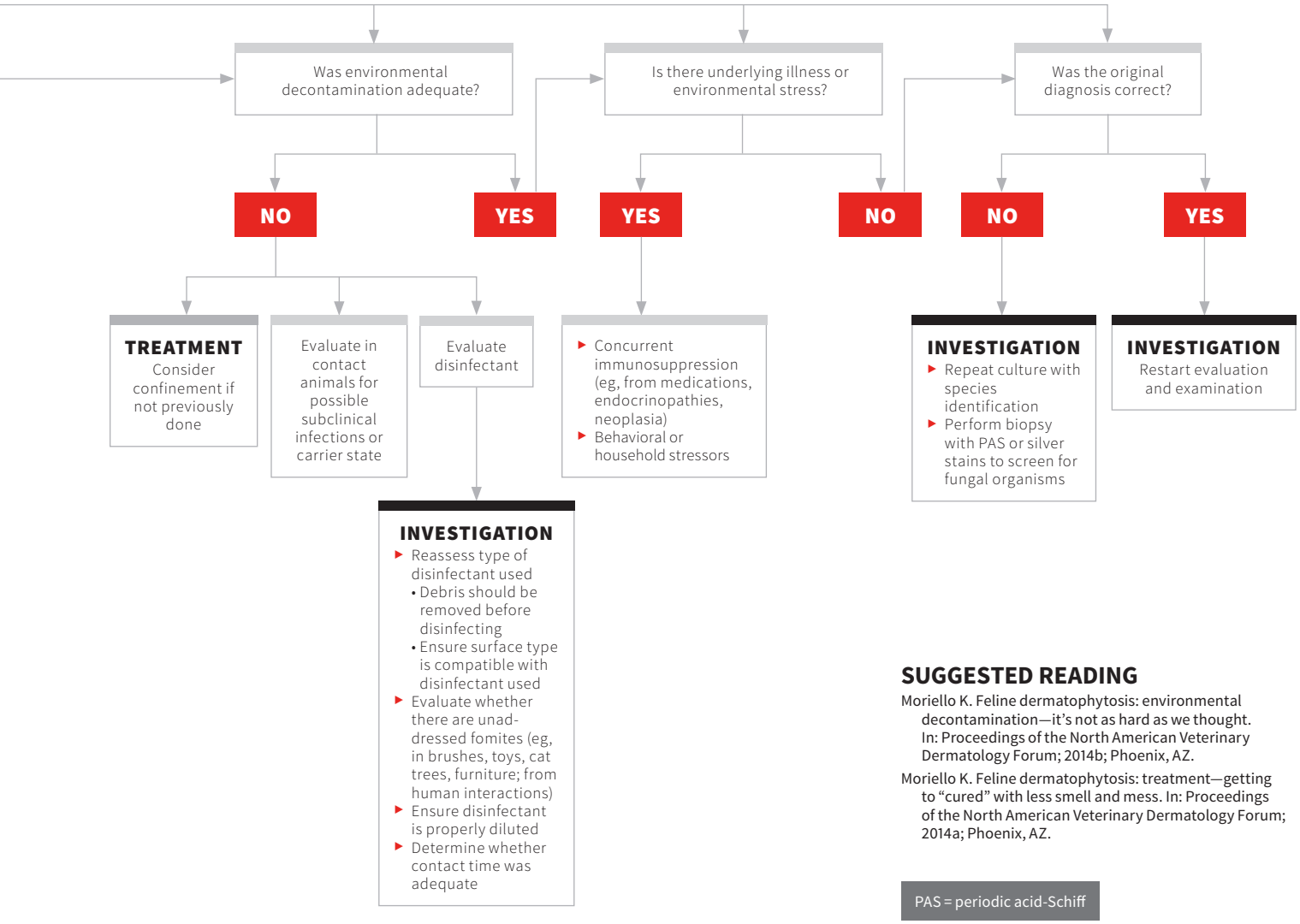

SUGGESTED READING

Moriello K. Feline dermatophytosis: environmental decontamination—it's not as hard as we thought. In: Proceedings of the North American Veterinary Dermatology Forum; 2014b; Phoenix, AZ.

Moriello K. Feline dermatophytosis: treatment—getting to "cured" with less smell and mess. In: Proceedings of the North American Veterinary Dermatology Forum; 2014a; Phoenix, AZ.

PAS = periodic acid-Schiff

SUSPECTED CUTANEOUS DRUG REACTION

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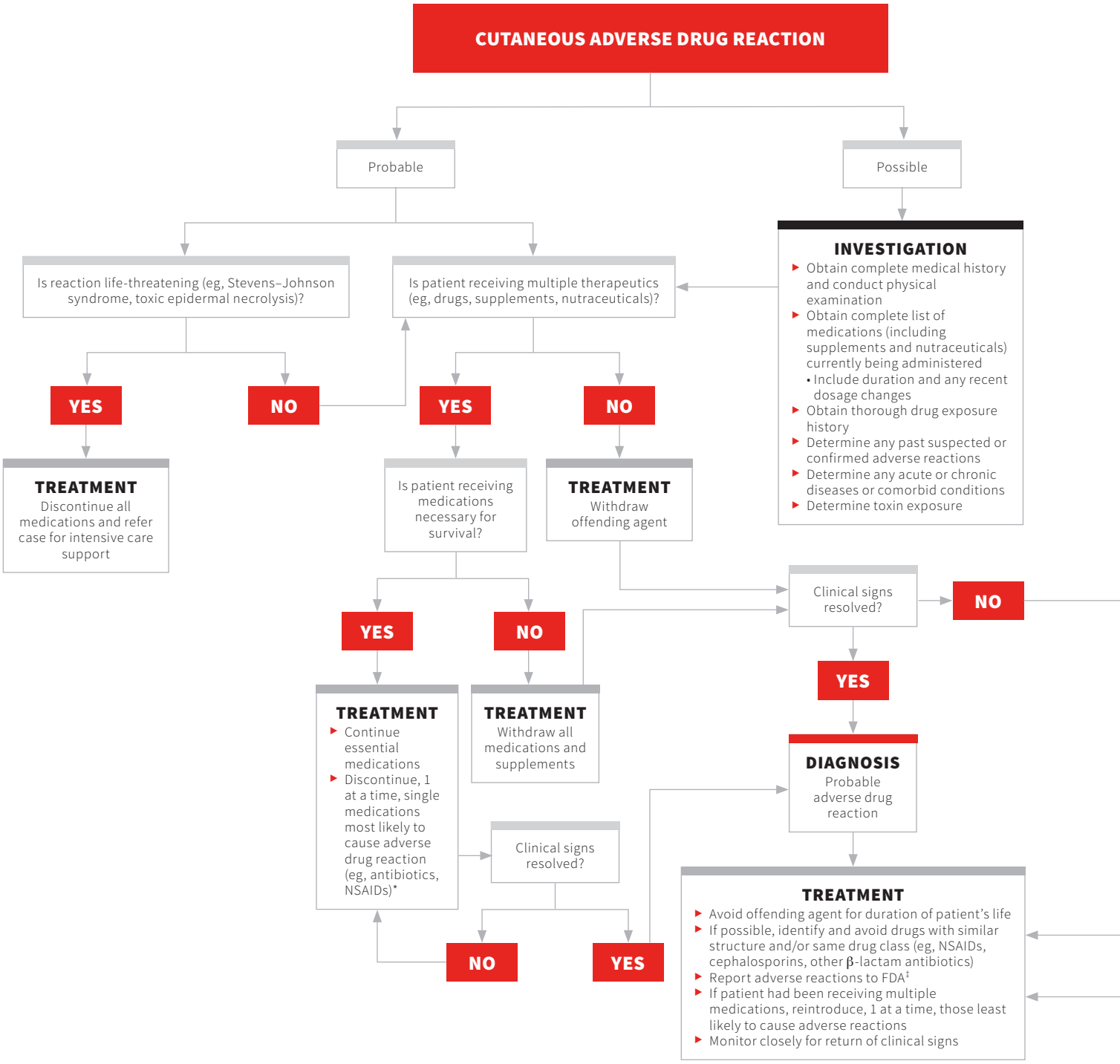
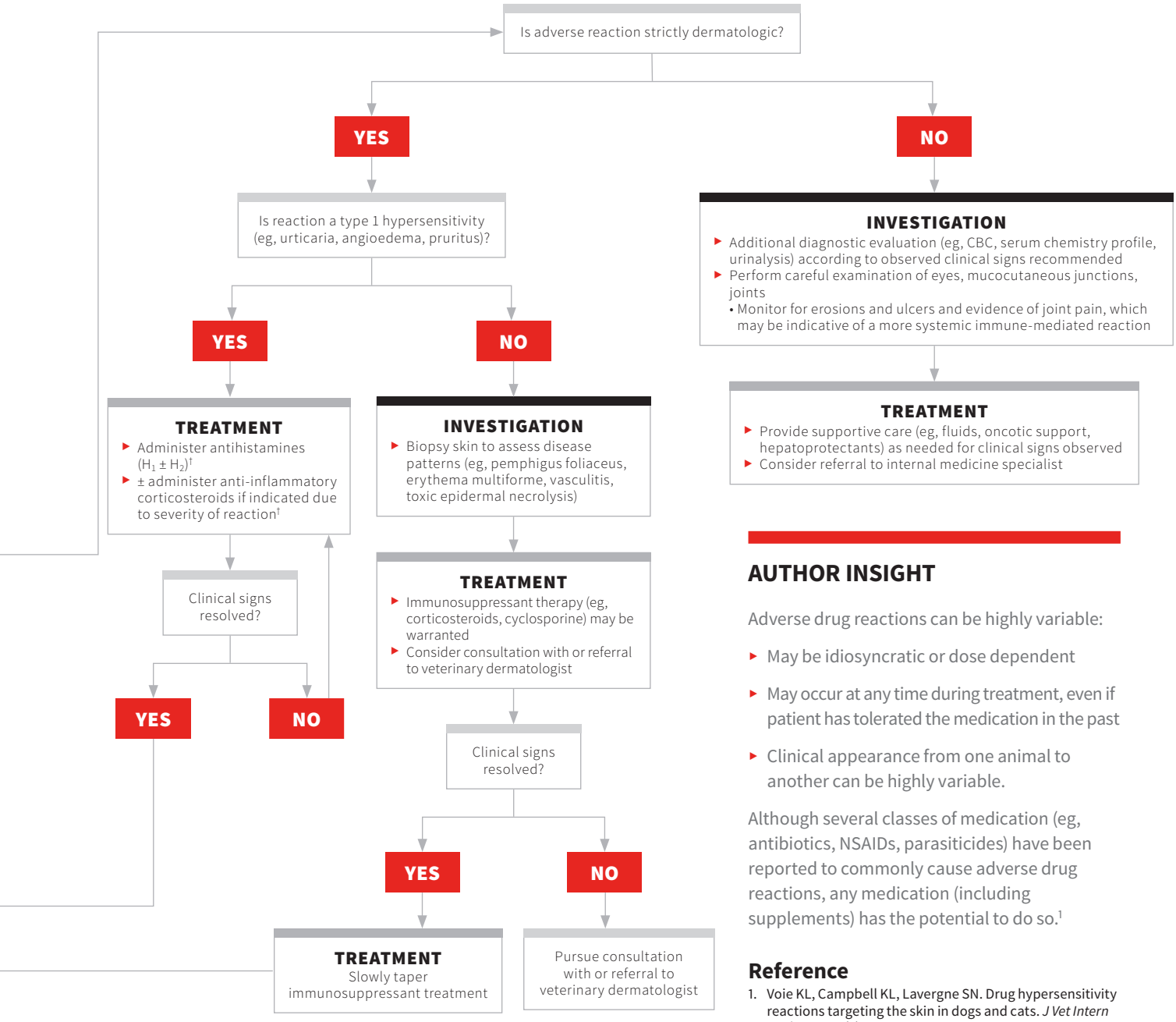




*If clinical signs of adverse drug reaction are life-threatening, withdraw all drugs.
†If initial treatment attempt was ineffective, use different medications than were previously used.
‡1-888-FDA-VETS

AUTHOR INSIGHT

Adverse drug reactions can be highly variable:

- May be idiosyncratic or dose dependent
- May occur at any time during treatment, even if patient has tolerated the medication in the past
- Clinical appearance from one animal to another can be highly variable.

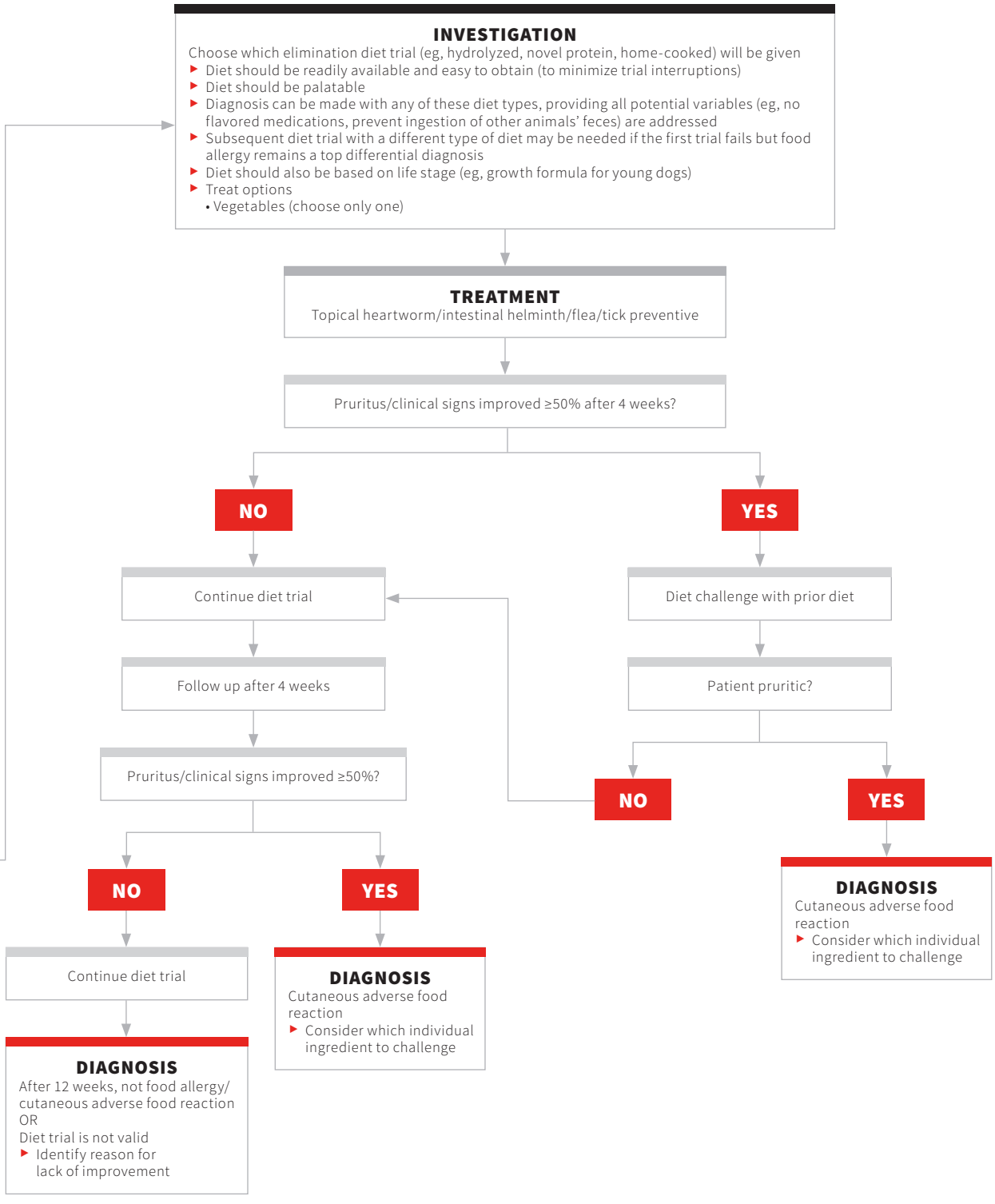
Although several classes of medication (eg, antibiotics, NSAIDs, parasiticides) have been reported to commonly cause adverse drug reactions, any medication (including supplements) has the potential to do so.[1]

Reference

1. Voie KL, Campbell KL, Lavergne SN. Drug hypersensitivity reactions targeting the skin in dogs and cats. *J Vet Intern Med*. 2012;26(4): 863-874.

SUSPECTED FOOD ALLERGY IN DOGS

Elizabeth R. Drake, DVM, DACVD
University of Tennessee


INVESTIGATION
Choose which elimination diet trial (eg, hydrolyzed, novel protein, home-cooked) will be given
Diet should be readily available and easy to obtain (to minimize trial interruptions)
Diet should be palatable
Diagnosis can be made with any of these diet types, providing all potential variables (eg, no flavored medications, prevent ingestion of other animals' feces) are addressed
Subsequent diet trial with a different type of diet may be needed if the first trial fails but food allergy remains a top differential diagnosis
Diet should also be based on life stage (eg, growth formula for young dogs)
Treat options
• Vegetables (choose only one)
TREATMENT
Topical heartworm/intestinal helminth/flea/tick preventive
Pruritus/clinical signs improved ≥50% after 4 weeks?
NO
YES
Continue diet trial
Follow up after 4 weeks
Pruritus/clinical signs improved ≥50%?
NO
YES
Continue diet trial
DIAGNOSIS
After 12 weeks, not food allergy/ cutaneous adverse food reaction
OR
Diet trial is not valid
Identify reason for lack of improvement
DIAGNOSIS
Cutaneous adverse food reaction
Consider which individual ingredient to challenge
Diet challenge with prior diet
Patient pruritic?
NO
YES
DIAGNOSIS
Cutaneous adverse food reaction
Consider which individual ingredient to challenge

VEHICULAR TRAUMA

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Adesola Odunayo, DVM, MS, DACVECC
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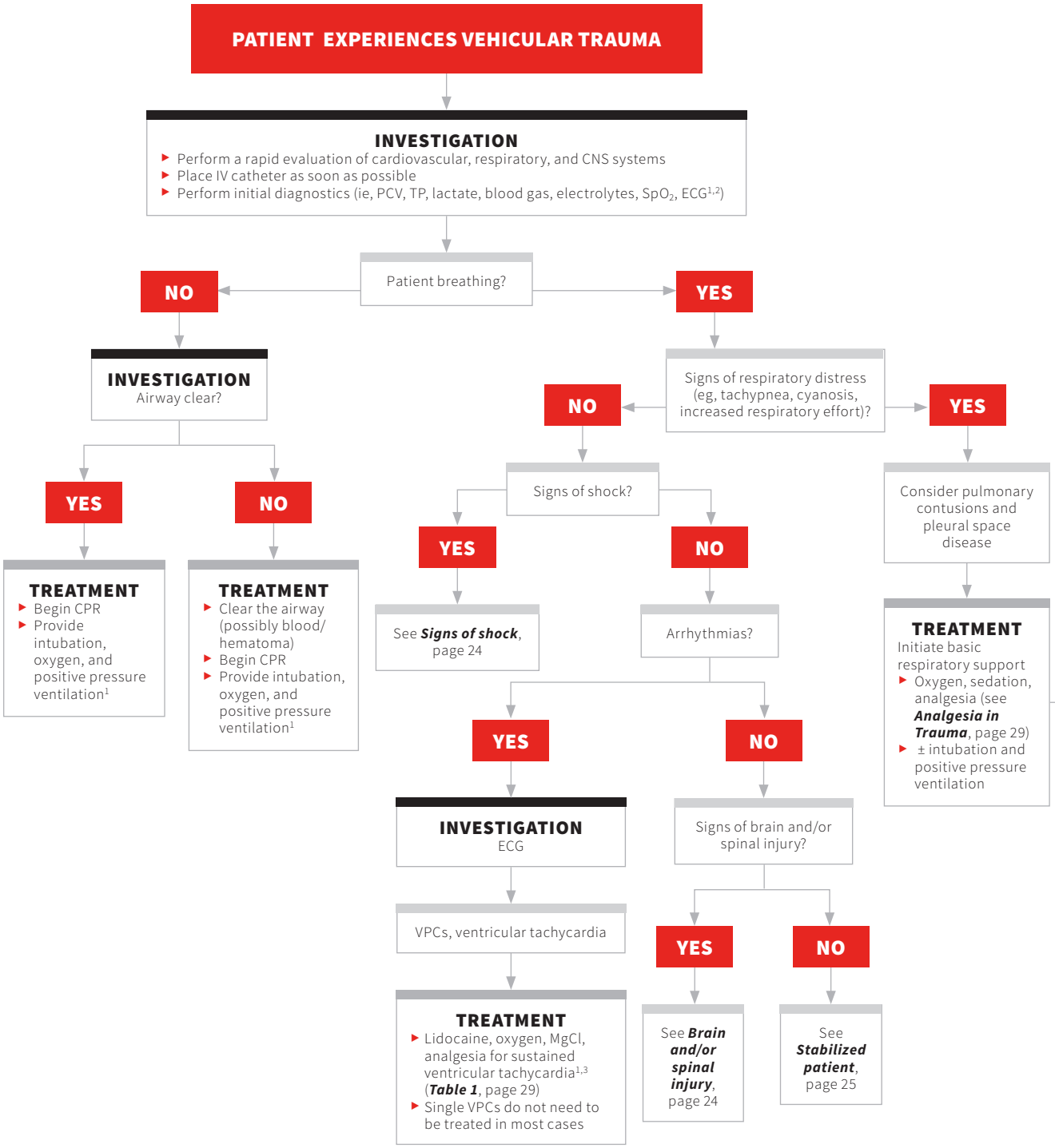

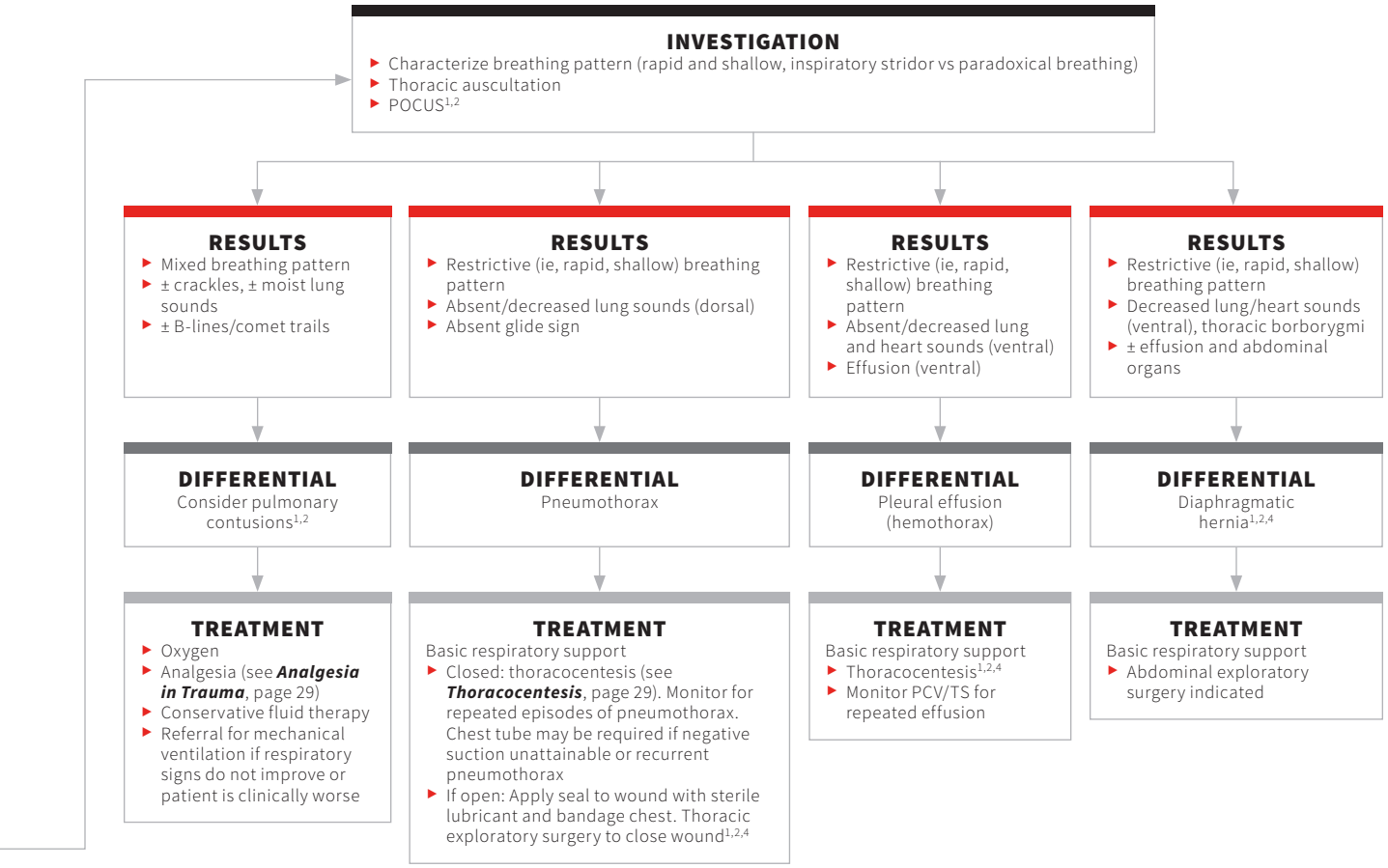

CPR = cardiopulmonary resuscitation
MgCl = magnesium chloride
PCV = packed cell volume
POCUS = point-of-care ultrasound
SpO_2 = oxygen saturation
TS = total solids
VPC = ventricular premature contraction

Continues ▶

Signs of shock

INVESTIGATION
MM, CRT, heart rate, BP, temperature, AFAST (see ***AFAST***, page 26), TFAST, mentation

Possible results
- Pale mucous membranes
- Prolonged CRT
- Tachycardia
- Bradycardia (cats)
- Hypotension
- Weak femoral pulses
- Hypothermia
- Lactate >22.5 mg/dL (>2.5 mmol/L)
- Altered mentation
- Cold extremities
- Free abdominal fluid
- Free thoracic fluid

DIFFERENTIALS
- Hypovolemic (secondary to hemorrhage) shock
- Distributive shock
- Septic shock possible if hollow viscus organ rupture present

TREATMENT
- Oxygen, analgesia (see ***Analgesia in Trauma***, page 29)
- Fluid therapy: Isotonic replacement fluid bolus, ± colloid, ± hypertonic saline, ± blood product transfusion[1] (***Table 1***, page 29)
- Investigate sources of hemorrhage and use external pressure/ligation as needed
- Active warming for hypothermic animals (especially in cats)[1,2,5,6]

Resuscitation endpoints (see ***Table 2***, page 30) met?

YES

TREATMENT
Decrease fluids to maintenance levels[1]

NO

TREATMENT
- Repeat fluid bolus
- Consider vasopressor support

Brain and/or spinal injury

INVESTIGATION
Cursory neurologic examination

RESULTS
- Nonambulatory; potentially altered pain perception

DIFFERENTIAL
- Spinal cord injury, pelvic limb fracture
- Assume unstable spinal cord injury

TREATMENT
- Analgesia (see ***Analgesia in Trauma***, page 29)
- Temporary immobilization until further diagnostics performed[7,8]

RESULTS
- Abnormal level of consciousness, cranial nerves, brain stem reflexes, motor responses

DIFFERENTIAL
Traumatic brain injury

TREATMENT
- Oxygen, hypertonic saline/mannitol (***Table 2***, page 30)
- Head elevation 15-30 degrees, analgesia (see ***Analgesia in Trauma***, page 29)
- Steroids contraindicated[9-11]

AFAST = abdominal focused assessment with sonography for trauma
BP = blood pressure
CK = creatine kinase
CRT = capillary refill time
Hct = hematocrit
LRS = lactated Ringer's solution
MAP = mean arterial pressure
MM = mucous membrane
MODS = multiple organ dysfunction
PE = pericardial effusion
POCUS = point of care ultrasound
PT = prothrombin time
PTT = partial thromboplastin time
RR = respiratory rate
SAP = serum alkaline phosphatase
SIRS = systemic inflammatory response syndrome
TFAST = thoracic focused assessment with sonography for trauma
TP = total protein

STABILIZED PATIENT

INVESTIGATION

Admit to hospital for at least 24 hours

- Closely monitor RR, respiratory effort, demeanor, and level of pain
- Perform serial PE, AFAST, TFAST (every 15 minutes to 12 hours as needed)
- CBC, serum chemistry profile, ± PT/PTT (repeat as necessary)
- Monitor urine output (at least 1-2 mL/kg/hour)[7]
- See ***Systemic Consequences of Trauma***, page 30

Therapeutic plan

- Fluid therapy with isotonic crystalloids (40-90 mL/kg/day)
- Continue analgesia with opioids. Consider NSAIDs (eg, robenacoxib, 2 mg/kg PO every 24 hours) if normotensive and ideally eating on own
- Provide oxygen if oxygen dependent
- Address any cutaneous wounds
- Antibiotic therapy as indicated for wounds, open fractures, or septic peritonitis
- Monitor Hct, BP, oxygenation, level of pain, mentation, and cranial nerve signs
- Recumbency care if not moving on own

Discharge criteria

- Eating and drinking well
- No longer oxygen dependent (based on primary clinical signs, respiratory rate, effort)
- Pain well-managed with oral analgesics
- Adequate plan for follow-up care depending on injuries sustained

Perform diagnostic evaluations

INVESTIGATION
Thoracic radiography

INVESTIGATION
See ***Orthopedic radiography***, page 26

INVESTIGATION
See ***Abdominal radiography***, page 26

INVESTIGATION
See ***POCUS (AFAST)***, page 26

RESULTS

- Incomplete diaphragmatic silhouette
- Obscured cardiac silhouette
- ± gas filled loops/stomach in thorax

DIFFERENTIAL
Diaphragmatic hernia

TREATMENT

Surgery

Ideally delayed 3-5 days to allow improvement of pulmonary contusions if present. Indications for emergency surgery:

- Herniated stomach
- Strangulated bowel/organ
- Inability to oxygenate properly despite appropriate treatment
- Ruptured viscera

RESULTS

- Silhouette effect
- Retraction of lung lobes
- Fissure lines
- Atelectasis

DIFFERENTIAL
Pleural effusion (hemothorax)

TREATMENT

- Typically resolves with supportive care. Thoracocentesis may be required if large volume of effusion. Oxygen support and blood transfusion may also be required.
- Assess coagulation times. Rarely requires surgical intervention, but emergent surgery may be required if large-volume bleed present[1,4,12]

RESULTS

- Widened radiolucent pleural space
- Atelectasis
- Elevation of heart from the sternum

DIFFERENTIAL
Pneumothorax

TREATMENT

- Thoracocentesis
- If thoracocentesis is needed twice within a very short period (ie, patient with recurrent pneumothorax) or negative suction cannot be obtained, consider thoracostomy tubes[1,4]

RESULTS

- Interstitial to alveolar pulmonary infiltrates

DIFFERENTIAL
Pulmonary contusions

TREATMENT

- Supportive care
- Rest, monitor for 24 hours
- May not be appreciable on radiographs for 24 hours[1,4]

RESULTS

- ***Fractures(s)*** (see ***Fracture[s]*** under ***Orthopedic radiography*** page 26)

Continues ▶

INVESTIGATION
Orthopedic radiography

RESULTS
- Fracture(s)

RESULTS
- Pelvic, appendicular, rib fractures

TREATMENT
- Pain management
- External coaptation when possible
- ± internal fixation
- Cage rest
- Flail chest: place affected side down[1,7,8]

RESULTS
Spinal fracture(s)
- Minimally displaced
- Minimal neurologic deficits
- Cervical trauma
- See ***Three Compartment Model***, page 30

TREATMENT
- Pain management
- External coaptation
- Cage rest
- Refer for CT ± MRI
- ± surgery[7,8]

RESULTS
Spinal fracture(s)
- Significant neurologic deficits
- Penetrating injury
- 2 of 3 spinal compartments affected indicates patient is unstable (see ***Three Compartment Model***, page 30)

TREATMENT
- Pain management
- Immobilize
- Refer for CT ± MRI
- ± surgery as soon as possible[7,8]

INVESTIGATION
Abdominal radiography

RESULTS
- Free gas

DIFFERENTIAL
Pneumoperitoneum

TREATMENT
Stabilization and emergency abdominal exploratory surgery

INVESTIGATION
POCUS (AFAST)

RESULTS
- Free abdominal fluid

INVESTIGATION
Abdominocentesis
- Compare PCV/TS, creatinine/potassium, bilirubin, glucose, and lactate of effusion to peripheral blood and cytology

RESULTS
- Peripheral blood to abdominal fluid glucose difference ≥20 mg/dL
- Effusion lactate ≥22.5 mg/dL (≥2.5 mmol/L) (dogs)
- Cytology: Neutrophils (often degenerate), presence of intracellular bacteria

DIFFERENTIAL
Septic abdomen

TREATMENT
Stabilize, initiate broad spectrum antibiotics and emergency abdominal exploratory surgery to evaluate and repair possible damage

RESULTS
- Effusion:serum bilirubin ratio >2
- Cytology: bile pigments

DIFFERENTIAL
Biliary rupture

TREATMENT
Emergency abdominal exploratory surgery to evaluate and repair possible damage as soon as patient is stable[1]

RESULTS
- PCV/TS of effusion ≈ PCV/TS of peripheral blood

DIFFERENTIAL
Traumatic hemoabdomen

TREATMENT
- Most respond to supportive care, fluid therapy, blood transfusion, and restricted activity. May consider abdominal compression wrap, but it must be done correctly to be valuable. Surgical intervention is rarely required
- Abdominal exploratory and surgical intervention required when hemorrhage is profound and ongoing and patient cannot be stablized[1]

RESULTS
- Effusion:serum creatinine ratio >2
- Effusion: serum potassium ratio >1.9 (cats) >1.4 (dogs

DIFFERENTIAL
Uroabdomen

TREATMENT
- Place urinary catheter. An abdominal drain may be needed if urinary catheter is not fully draining the abdomen.
- Consider contrast study to identify source of leakage (eg, urethra, urinary bladder, ureter).
- Surgery can be delayed for 6-24 hours if the abdominal cavity is properly drained and there is no evidence of septic peritonitis.
- Urethral tears usually heal on their own without surgical intervention[1]

Continues ▶

ANCILLARY MATERIAL TO VEHICULAR TRAUMA

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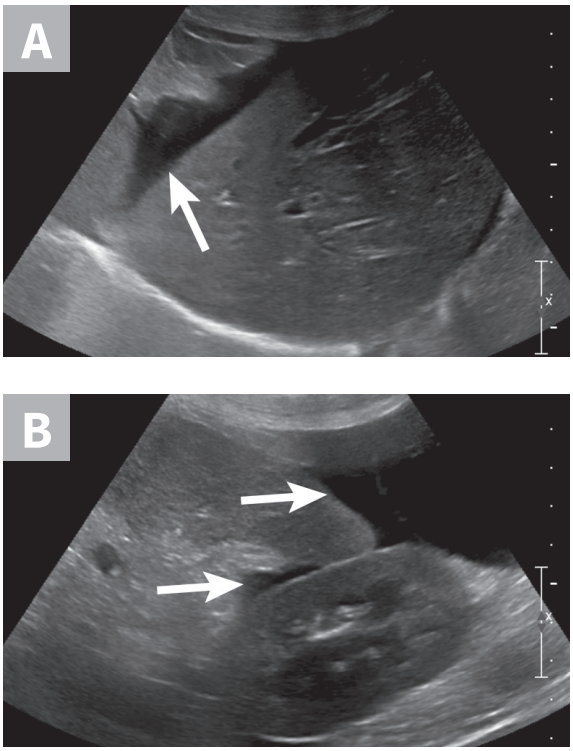
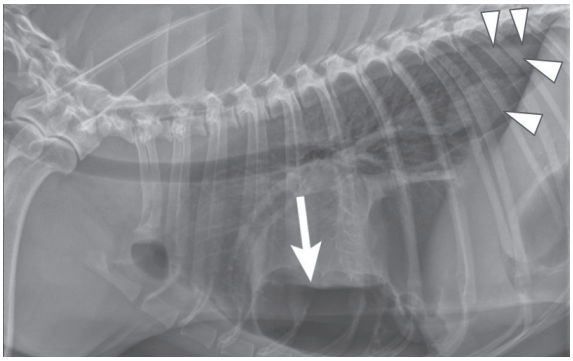
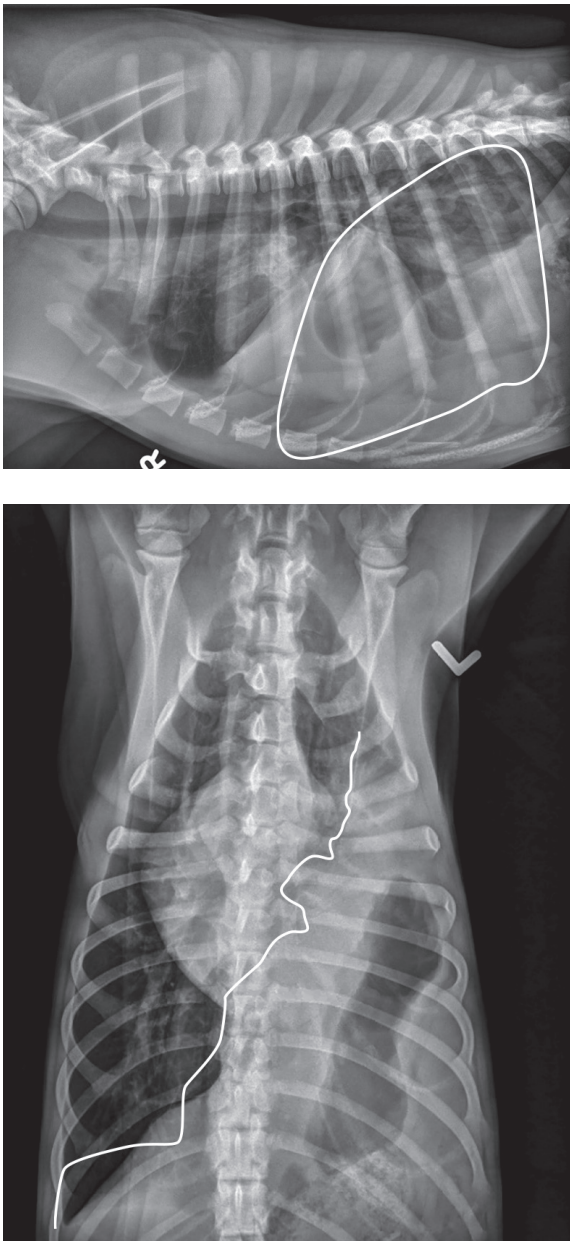


▲ **FIGURE 1** AFAST: Anechoic free abdominal fluid (***arrows***) at the diaphragmatico-hepatic view (***A***) and splenorenal site (***B***). *Images courtesy of Silke Hecht, DACVR, DECVDI*



▲ **FIGURE 2** Lateral thoracic radiograph of the pneumothorax in a dog. Increased gas opacity in the pleural space, retraction of the lung lobes from the thoracic wall and diaphragm (***arrowheads***), separation of the cardiac silhouette from the sternum (***arrow***), and diffusely increased opacity of the lungs due to atelectasis can be seen. *Image courtesy of Silke Hecht, DACVR, DECVDI*



▲ **FIGURE 3** Thoracic radiographs of diaphragmatic hernia in a dog. Cranial displacement of abdominal viscera (***circle***), loss of normal diaphragm outline (***line***), and displacement of thoracic structures can be seen. *Images courtesy of Silke Hecht, DACVR, DECVDI*

TABLE 1

GENERAL GUIDELINES FOR FLUID RESUSCITATION & BLOOD TRANSFUSION IN PATIENTS WITH TRAUMA

Perfusion Parameters	Normal Endpoints
Whole blood[5]	Dogs: 20-30 mL/kg given over 30 minutes to 4 hours, depending on how critical the patient is Cats: 50-60 mL/cat (NOT mL/kg) given over same time period as for dogs
Packed RBCs[5]	Dogs: 15 mL/kg given over same time frame as whole blood Cats: 30-40 mL/cat (NOT mL/kg) given over same time frame as for dogs
Synthetic colloid (controversial)[5]	1-5 mL/kg given over 15 minutes
Fresh frozen plasma[5]	15-30 mL/kg for patients with coagulopathy and active hemorrhage
Isotonic fluid shock bolus (LRS, Norm-R, 0.9% sodium chloride, Plasma-Lyte)[5,9]	10-25 mL/kg given over 15 minutes. End goals should be reassessed; may be repeated until entire shock dose administered. Dog shock dose: 90 mL/kg/hour; cat shock dose: 50-60 mL/kg/hour
Hypertonic saline[5,9]	4-6 mL/kg given over 15 minutes; may be repeated 2-3 times in 24 hours
Mannitol[9]	0.5-1.5 g/kg IV given over 15 minutes, may be repeated 2-3 times in 24 hours
Lidocaine[3]	2 mg/kg IV bolus, followed by 50-80 µg/kg/minute if rhythm converts

THORACOCENTESIS

Thoracocentesis is often a life-saving treatment that should be performed during initial stabilization, ideally prior to radiographic confirmation of pneumothorax or pleural effusion to prevent patient decompensation in radiology.[1,2,4]

ANALGESIA IN TRAUMA

Quick and effective analgesia is essential for patients with vehicular trauma. Opioids are the drug of choice because of their efficacy and limited adverse effects. NSAIDs should be avoided until the patient is hemodynamically stable. In addition, butorphanol has minimal analgesic effects and should not be used. IM or SC administration of pure µ-receptor agonists may cause vomiting; IV administration is strongly preferred.[1,13]

- Morphine (0.1-0.5 mg/kg IV every 4 hours)
- Hydromorphone (0.05-0.2 mg/kg IV every 4-6 hours)
- Methadone (0.1-0.5 mg/kg IV every 4-6 hours)
- Fentanyl (2-5 µg/kg bolus, then 2-6 µg/kg/hour IV CRI)
- Buprenorphine (0.01-0.03 mg/kg IV or IM every 6-8 hours)

Continues ▶

ANCILLARY MATERIAL TO VEHICULAR TRAUMA CONTINUED

THREE COMPARTMENT MODEL

- Dorsal column: laminae, spinous processes and their ligaments
- Middle column: dorsal longitudinal ligament, dorsal annulus, dorsal cortex of the vertebral bodies
- Ventral column: ventral longitudinal ligament, ventral annulus, ventral cortex of the vertebral bodies

SYSTEMIC CONSEQUENCES OF TRAUMA

- Common metabolic consequences[6,12]
  - –Activation of the coagulation cascade
  - –Hypothermia
  - –GI disturbance (eg, vomiting, diarrhea)
  - –Systemic inflammation (eg, SIRS, MODS)
- Common clinical pathologic abnormalities[2,6,12]
  - –Hyperglycemia
  - –Hyperlactatemia
  - –Metabolic acidosis
  - –Hypoalbuminemia
  - –Anemia
  - –Thrombocytopenia
  - –Increased ALT
  - –Increased CK
  - –Prolonged PT/PTT

TABLE 2

RESUSCITATION ENDPOINTS

Perfusion Parameters	Normal Endpoints
Heart rate	Dogs: 60-120 bpm Cats: 160-220 bpm
MM color	Pink
CRT	1-2 seconds
Temperature	99°F-102.5°F (37.2°C-39.2°C)
Mentation	Alert
SAP (systolic BP)	>90 mm Hg
MAP (mean BP)	>70 mm Hg
Urine output	1-2 mL/kg/hour
Lactate	<22.5 mg/dL (2.5 mmoL)

BP = blood pressure
CRT = capillary refill time
MAP = mean arterial pressure
MODS = multiple organ dysfunction
PT = prothrombin time
PTT = partial thromboplastin time
SIRS = systemic inflammatory response syndrome

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HEMOABDOMEN

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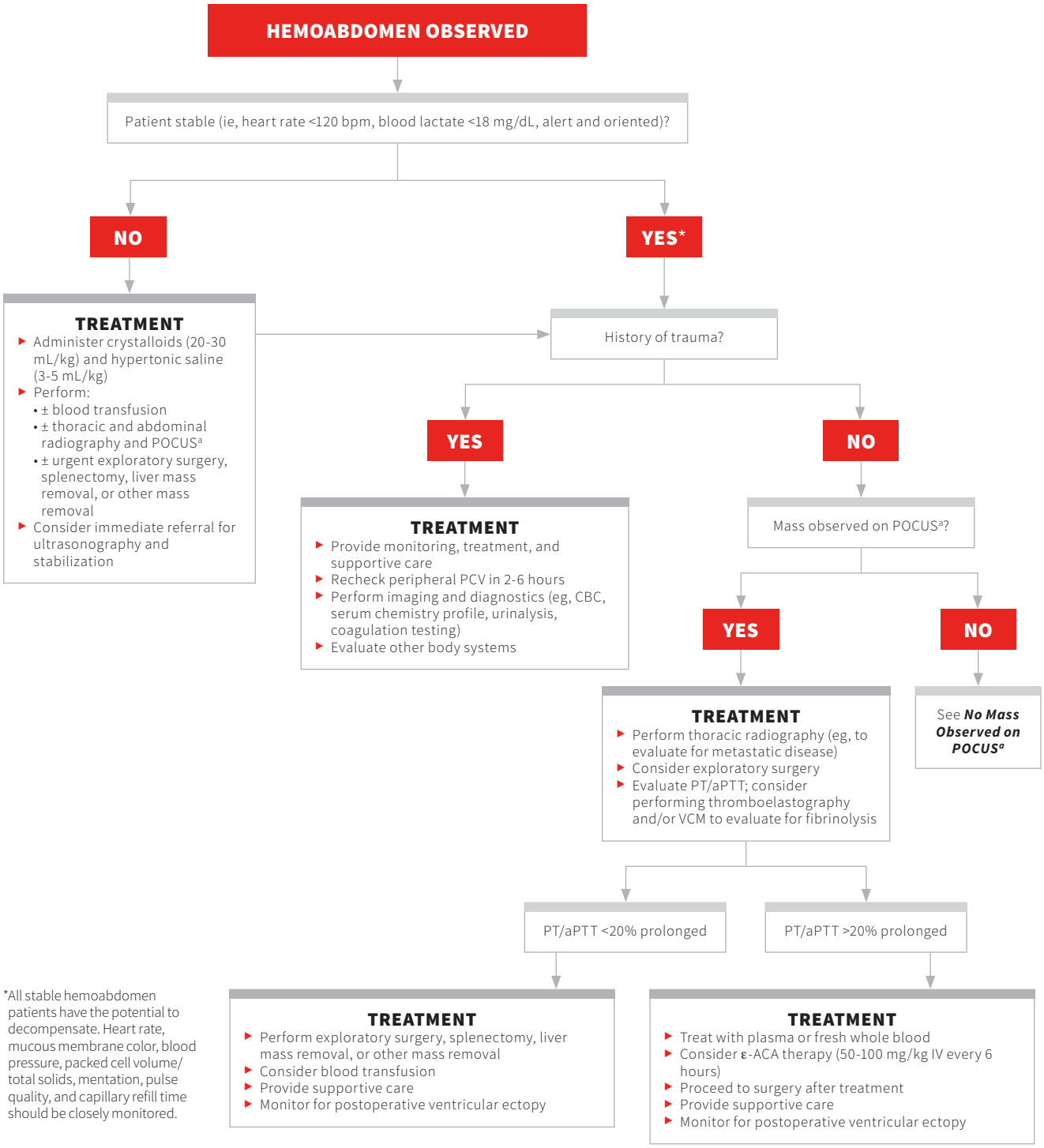
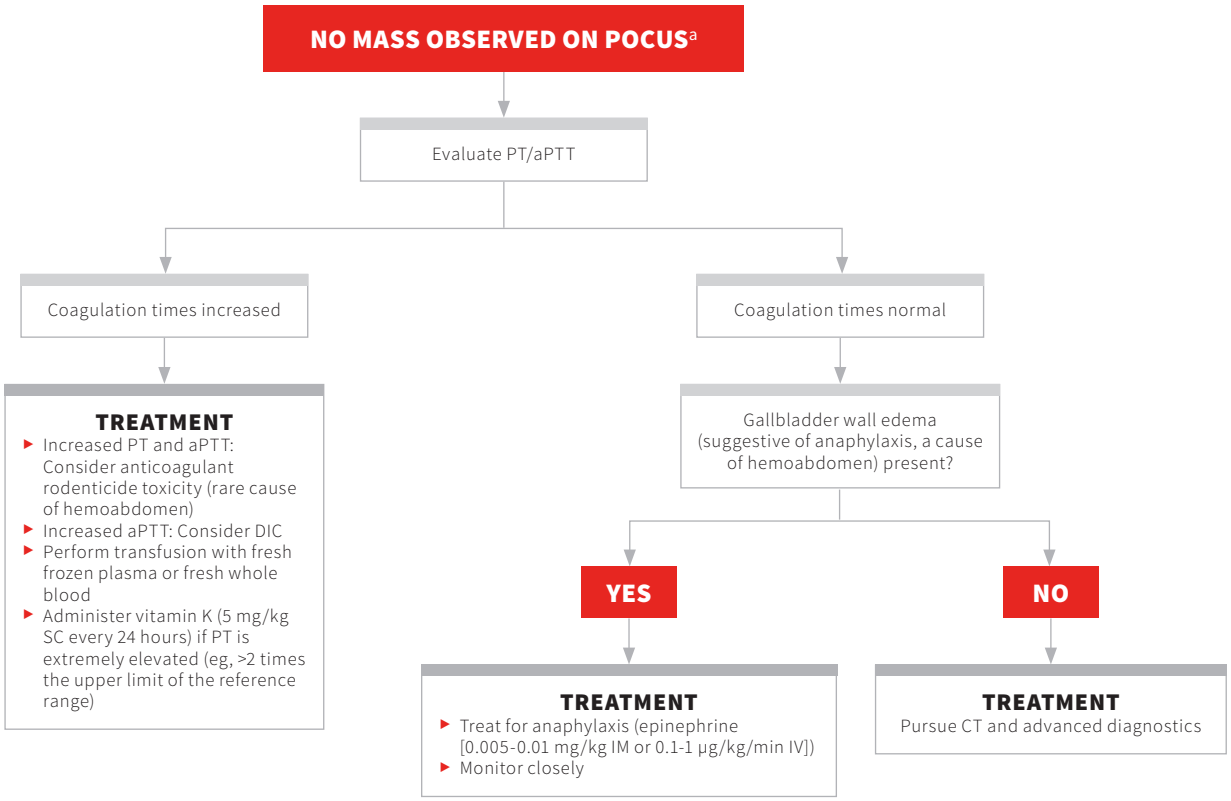


*All stable hemoabdomen patients have the potential to decompensate. Heart rate, mucous membrane color, blood pressure, packed cell volume/total solids, mentation, pulse quality, and capillary refill time should be closely monitored.



ACA = aminocaproic acid

aPTT = activated partial thromboplastin time

DIC = disseminated intravascular coagulation

PCV = packed cell volume

POCUS[a] = point-of-care ultrasonography (abdomen)

PT = prothrombin time

VCM = viscoelastic coagulation monitoring

SNAKE ENVENOMATION

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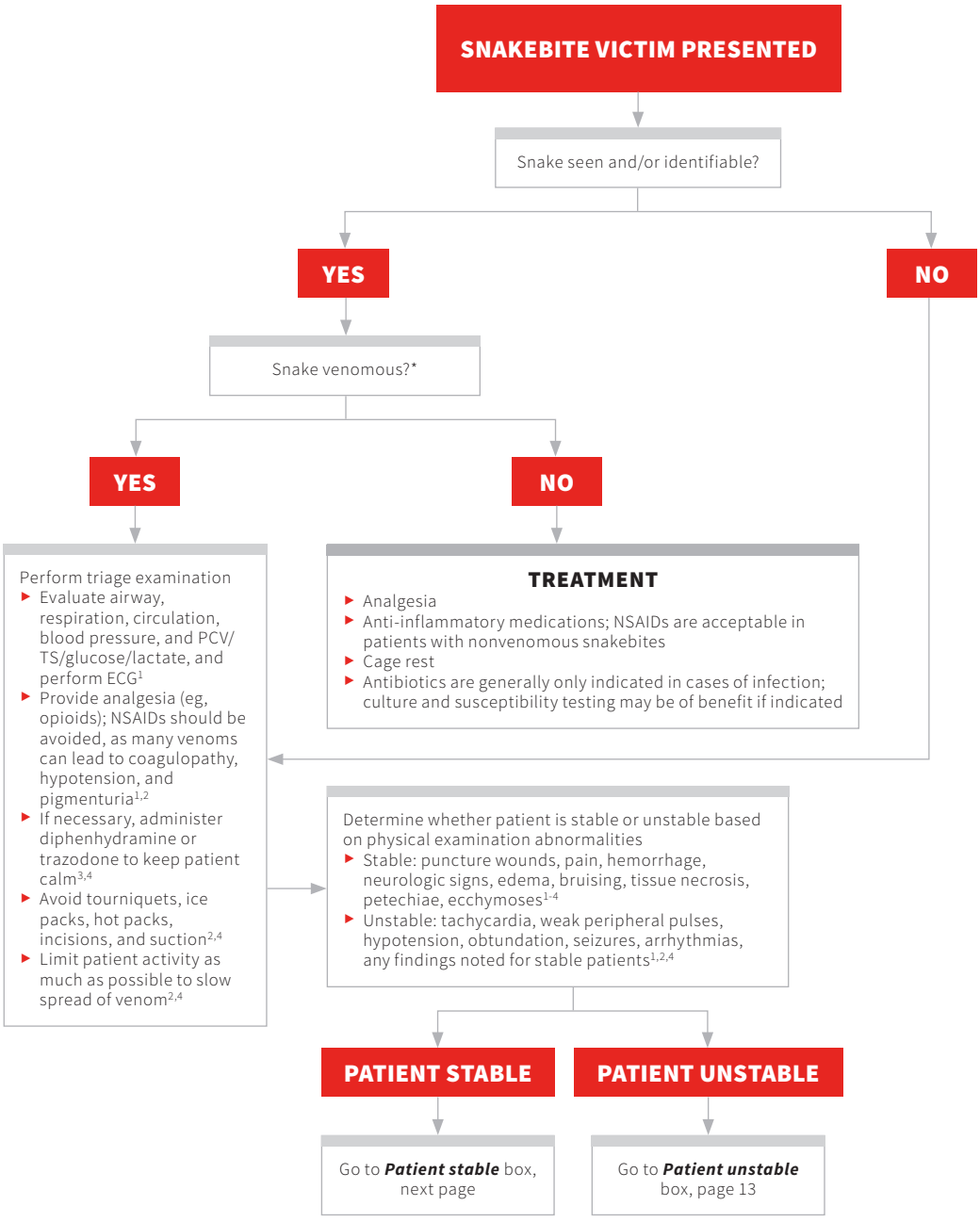

*Coral snakes and pit vipers (eg, rattlesnakes, copperheads, water moccasins, cottonmouths) are among the venomous snakes found in the United States.[2,4]

PCV = packed cell volume
TS = total solids

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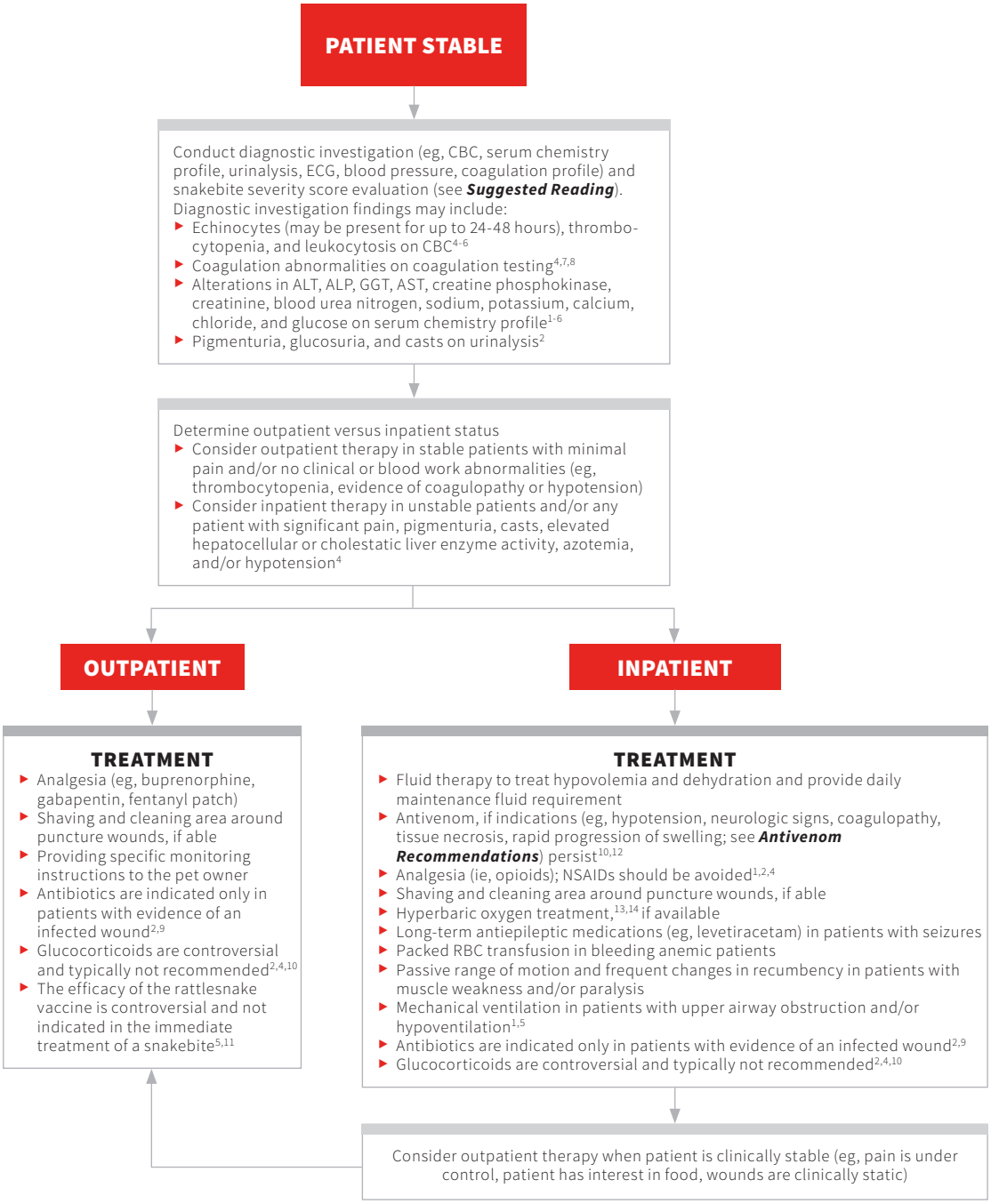
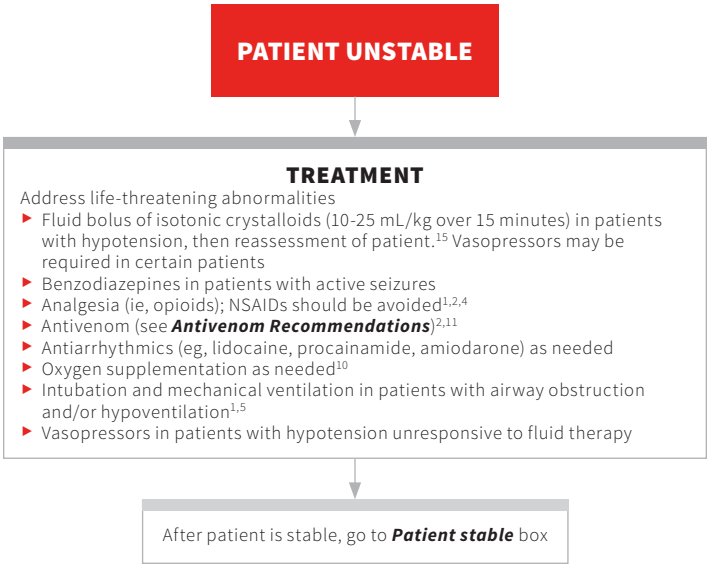


ALP = alkaline phosphatase
ALT = alanine transaminase
AST = aspartate aminotransferase
GGT = gamma-glutamyl transferase



ANTIVENOM RECOMMENDATIONS

- Clinicians should start with one vial of antivenom per patient; however, patients with a lower body weight may require more antivenom,[1-5] as smaller patients tend to receive a larger amount of venom per kg of body weight when bitten (eg, a Chihuahua vs a Great Dane injected with the same amount of venom).[5]
- If the antivenom is lyophilized, one vial should be reconstituted with crystalloid fluids (100-250 mL).[5]
- Antivenom should be administered intravenously over 1 to 2 hours.[2,3]
- Patients should be monitored for signs of anaphylactoid/anaphylactic reactions.
- Diphenhydramine may be considered if anaphylaxis or a mild anaphylactoid reaction to the antivenom is suspected, whereas epinephrine and intravenous fluids should be administered for severe anaphylaxis/anaphylactoid reactions.[2] Administration of antivenom should be stopped in both instances.[4] However, if the reaction is not severe, administration of antivenom should be slowly resumed after approximately 20 to 60 minutes.[4] Additional support in patients with hypotension (eg, vasopressors) and/or respiratory signs (eg, mechanical ventilation) may be required.[1]

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CANINE DIABETIC KETOACIDOSIS

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SUSPECTED CANINE DIABETIC KETOACIDOSIS

Physical examination (patient may have only 1 or more of the following signs):
- Polyuria
- Polydipsia
- Polyphagia
- Weight loss
- Vomiting
- Lethargy

Laboratory results:
- BG: hyperglycemia (>200 mg/dL)
- Blood gas (venous or arterial): metabolic acidosis
- Urine dipstick: glucosuria; ketonuria or ketonemia (Serum ketones can be measured if urine is unavailable.)

DIAGNOSIS
Diabetic ketoacidosis

TREATMENT

1. IV isotonic crystalloid therapy:
- Shock fluid therapy is warranted if hypovolemic or hypotensive shock are present: Start with 1/4 to 1/3 shock dose (90 mL/kg) and reassess until stable
- Correct dehydration, provide maintenance needs, and replace ongoing losses over 6-24 hours:
  - % dehydration × body weight (kg) × 1000 plus
  - 20 mL/kg/day (insensible losses) plus
  - 20 to 40 mL/kg/day (maintenance sensible losses) plus
- Account for vomiting, diarrhea, and polyuria (ongoing sensible losses)

TREATMENT

2. Electrolyte supplementation (see ***Table 1***, next page):
- Monitor serum potassium every 4-6 hours until within reference interval and stable; then every 12-24 hours
- Monitor serum phosphorus every 4-6 hours until >1.5; then every 6-24 hours
- When supplementing potassium and phosphorus concurrently, take into account the amount of potassium contained in the potassium phosphate
- Consider magnesium supplementation in instances of refractory hypokalemia

TREATMENT

3. Regular insulin:
- CRI protocol[1]:
  - Start with 2.2 U/kg of regular insulin to 250 mL of 0.9% saline and increase concentration, if necessary
  - Allow 50 mL of insulin solution to run through administration set because insulin adheres to plastic
  - Administer solution as shown in ***Table 2***, next page
- Intermittent low-dose IM protocol:
  - Administer 0.2 U/kg regular insulin IM initially; 1 hour later begin IM injections of 0.1 U/kg every hour
  - If BG <250 mg/dL, make 5% dextrose solution with hydration fluids and change dosage regimen to 0.1-0.4 U/kg SC insulin every 4-6 hours
- For either protocol:
  - Monitor BG every 1-2 hours
  - Goal for either protocol is to maintain BG between 200 and 300 mg/dL
  - Do not decrease BG faster than 70-100 mg/dL/h

*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Alice Huang, VMD, MS, DACVIM (SAIM), at Seattle Veterinary Specialists, Kirkland, Washington.

BG = blood glucose
cPLI = canine pancreatic lipase immunoreactivity
CRI = constant rate infusion
NPH = neutral protamine Hagedorn

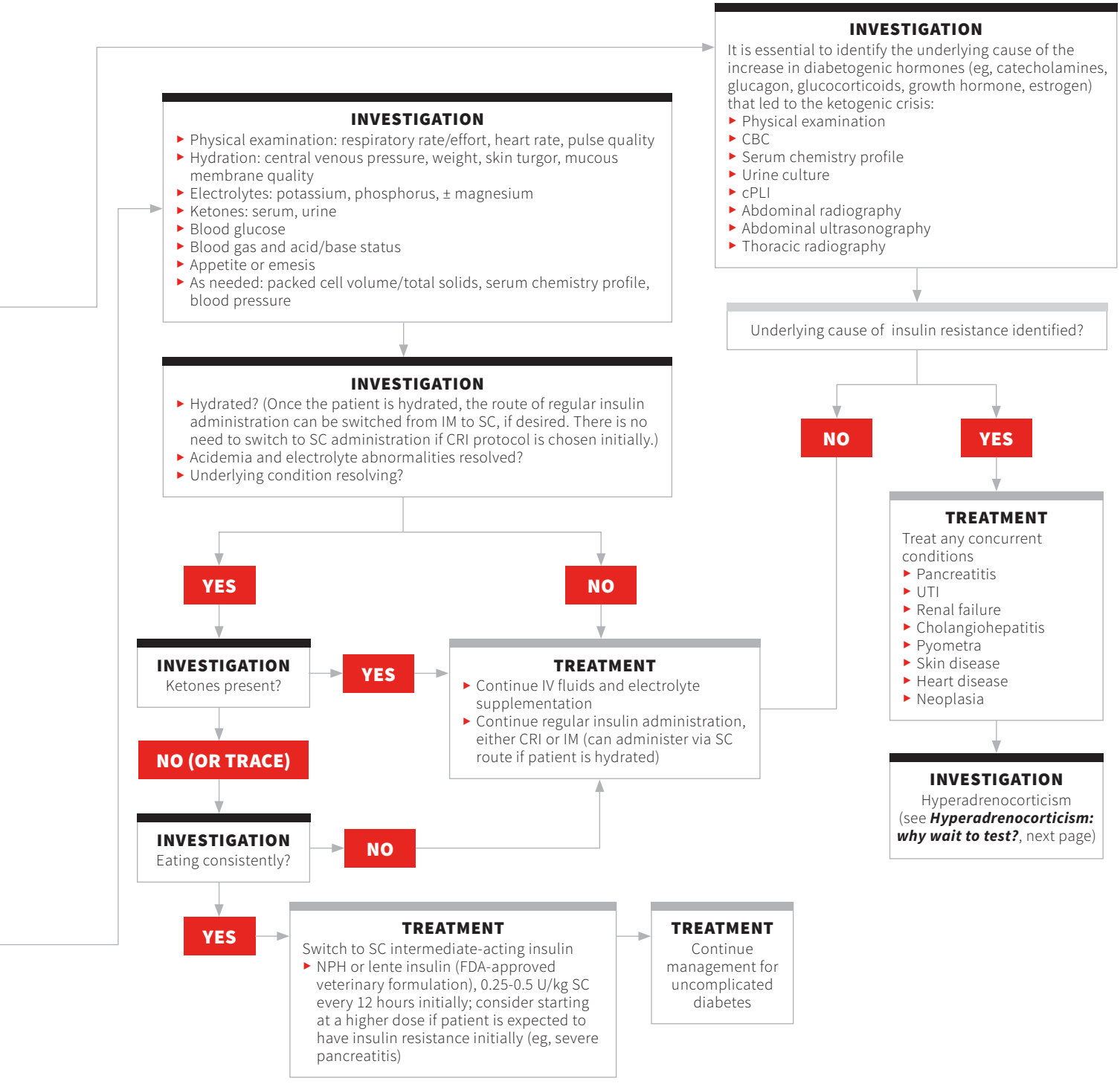
INVESTIGATION
It is essential to identify the underlying cause of the increase in diabetogenic hormones (eg, catecholamines, glucagon, glucocorticoids, growth hormone, estrogen) that led to the ketogenic crisis:
Physical examination
CBC
Serum chemistry profile
Urine culture
cPLI
Abdominal radiography
Abdominal ultrasonography
Thoracic radiography
Underlying cause of insulin resistance identified?
NO
YES
TREATMENT
Treat any concurrent conditions
Pancreatitis
UTI
Renal failure
Cholangiohepatitis
Pyometra
Skin disease
Heart disease
Neoplasia
INVESTIGATION
Hyperadrenocorticism
(see Hyperadrenocorticism: why wait to test?, next page)
INVESTIGATION
Physical examination: respiratory rate/effort, heart rate, pulse quality
Hydration: central venous pressure, weight, skin turgor, mucous membrane quality
Electrolytes: potassium, phosphorus, ± magnesium
Ketones: serum, urine
Blood glucose
Blood gas and acid/base status
Appetite or emesis
As needed: packed cell volume/total solids, serum chemistry profile, blood pressure
INVESTIGATION
Hydrated? (Once the patient is hydrated, the route of regular insulin administration can be switched from IM to SC, if desired. There is no need to switch to SC administration if CRI protocol is chosen initially.)
Acidemia and electrolyte abnormalities resolved?
Underlying condition resolving?
YES
NO
INVESTIGATION
Ketones present?
YES
TREATMENT
Continue IV fluids and electrolyte supplementation
Continue regular insulin administration, either CRI or IM (can administer via SC route if patient is hydrated)
NO (OR TRACE)
INVESTIGATION
Eating consistently?
NO
YES
TREATMENT
Switch to SC intermediate-acting insulin
NPH or lente insulin (FDA-approved veterinary formulation), 0.25-0.5 U/kg SC every 12 hours initially; consider starting at a higher dose if patient is expected to have insulin resistance initially (eg, severe pancreatitis)
TREATMENT
Continue management for uncomplicated diabetes

TABLE 1

ELECTROLYTE SUPPLEMENTATION

Serum Potassium Concentration (mEq/L)	Potasssium Chloride Dose
>3.5 (maintenance)	0.05-0.1 mEq/kg/h
3-3.5	0.1-0.2 mEq/kg/h
2.5-3	0.2-0.3 mEq/kg/h
2-2.5	0.3-0.4 mEq/kg/h
<2	0.4-0.5 mEq/kg/h
Serum Phosphorus Concentration (mg/dL)	**Potassium Phosphorus Dose**
2-2.5	0.03 mmol/kg/h
1.5-2	0.06 mmol/kg/h
1-1.5	0.09 mmol/kg/h
<1	0.12 mmol/kg/h

TABLE 2

CRI INFUSION OF INSULIN

BG Concentration (mg/dL)	IV Hydration Fluids	Rate of Insulin Solution (mL/h)
>250	0.9% saline	10
200-250	0.9% saline + 2.5% dextrose	7
150-200	0.9% saline + 2.5% dextrose	5
100-150	0.9% saline + 5% dextrose	5
<100	0.9% saline + 5% dextrose	Discontinue

HYPERADRENOCORTICISM: WHY WAIT TO TEST?

Although hyperadrenocorticism is one of the most frequent causes of insulin resistance, it is not appropriate to test for it during a diabetic ketoacidosis crisis because false positives would be expected. Diagnostic testing for hyperadrenocorticism should not be performed until the patient has been systemically healthy for at least 2 weeks. Appropriate regulation of diabetes mellitus may be difficult to achieve prior to diagnosis of concurrent hyperadrenocorticism.

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Suggested Reading

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DIABETES REMISSION IN CATS

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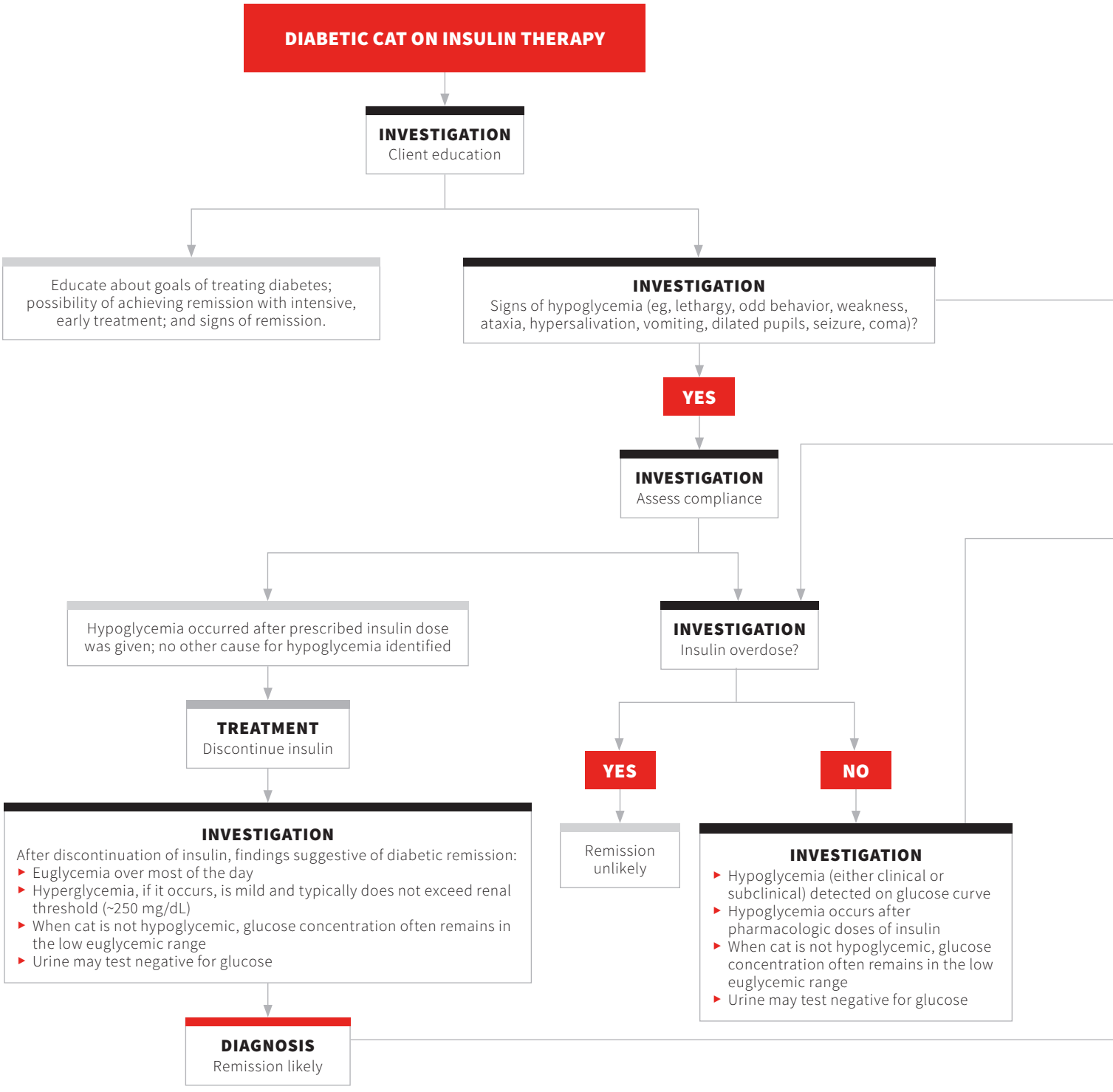
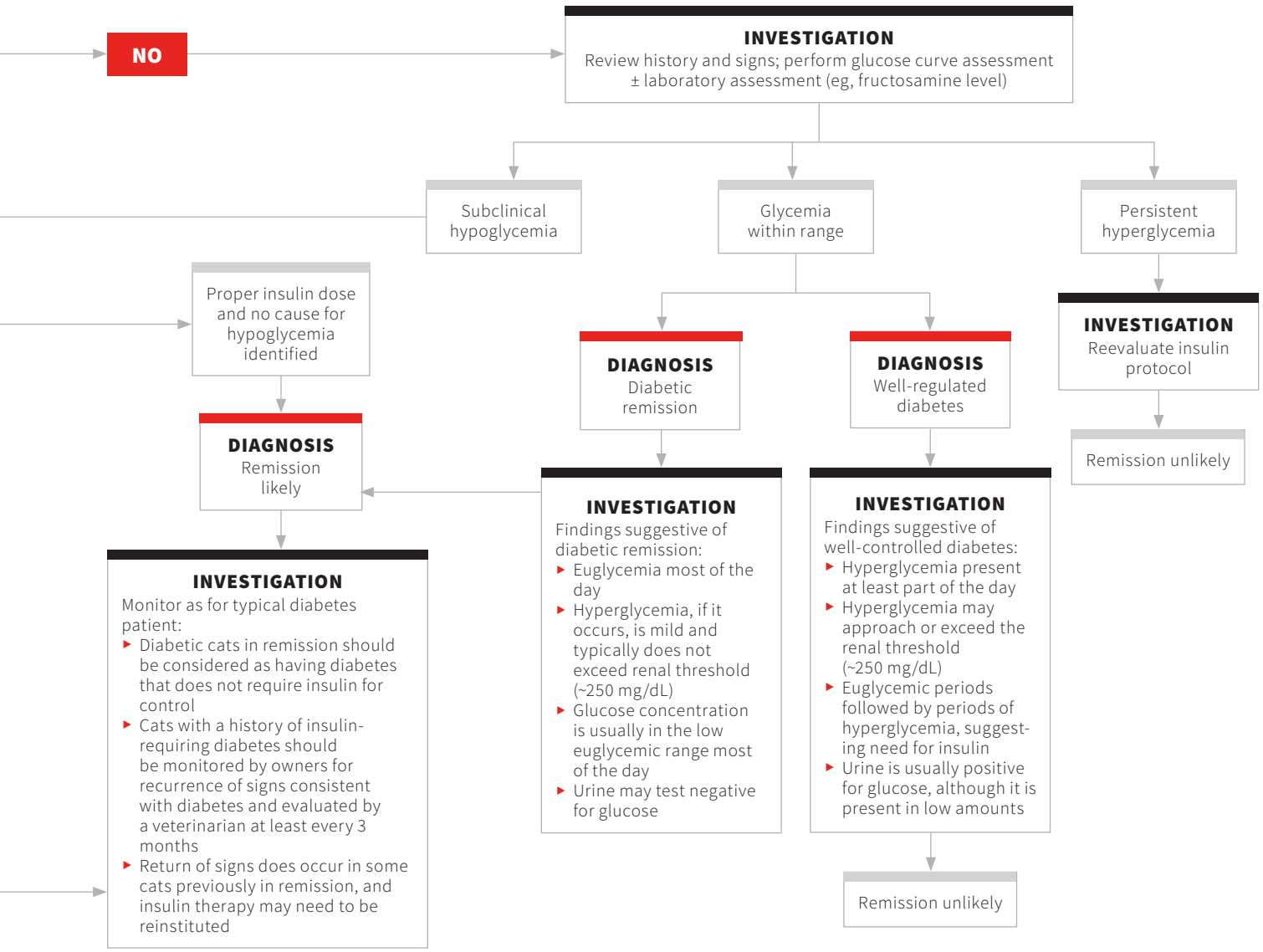


DIABETIC REMISSION AT A GLANCE

- Diabetic remission is achieved when insulin is no longer required to maintain normoglycemia.
- Other interventions (eg, diet) may still be used for blood glucose control.
- Likelihood of remission is highest in cats with newly diagnosed diabetes that receive intensive, early treatment.
- Some diabetic cats may alternate periods of overt diabetes with remission.
- Distinguishing diabetic remission from well-controlled diabetes may be difficult via history and clinical examination.
- Unless concurrent disease is present, well-controlled cats and cats in diabetic remission should not show signs typical of diabetes (eg, polydipsia, polyuria, weight loss).



INSULIN SELECTION IN DIABETIC DOGS & CATS

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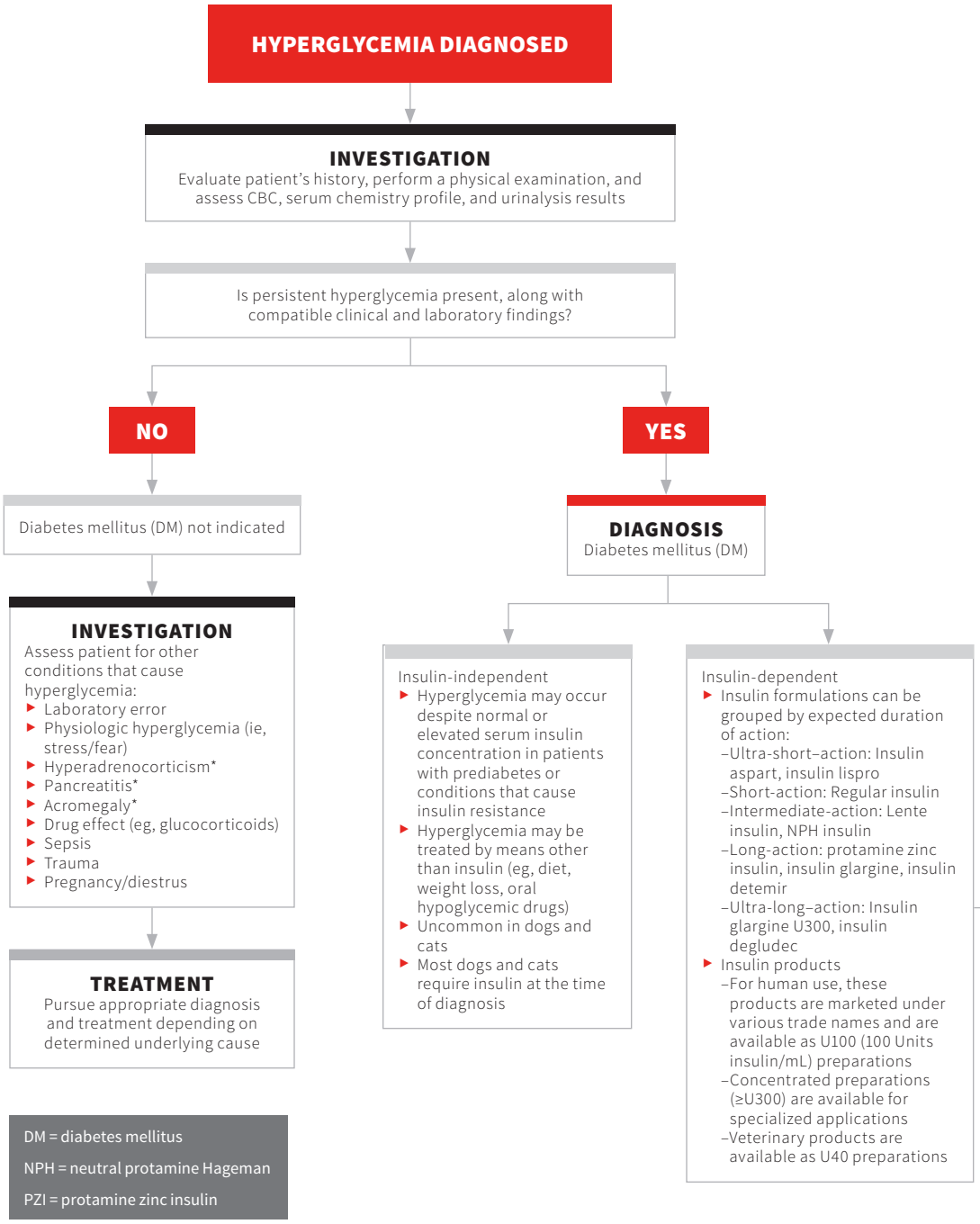
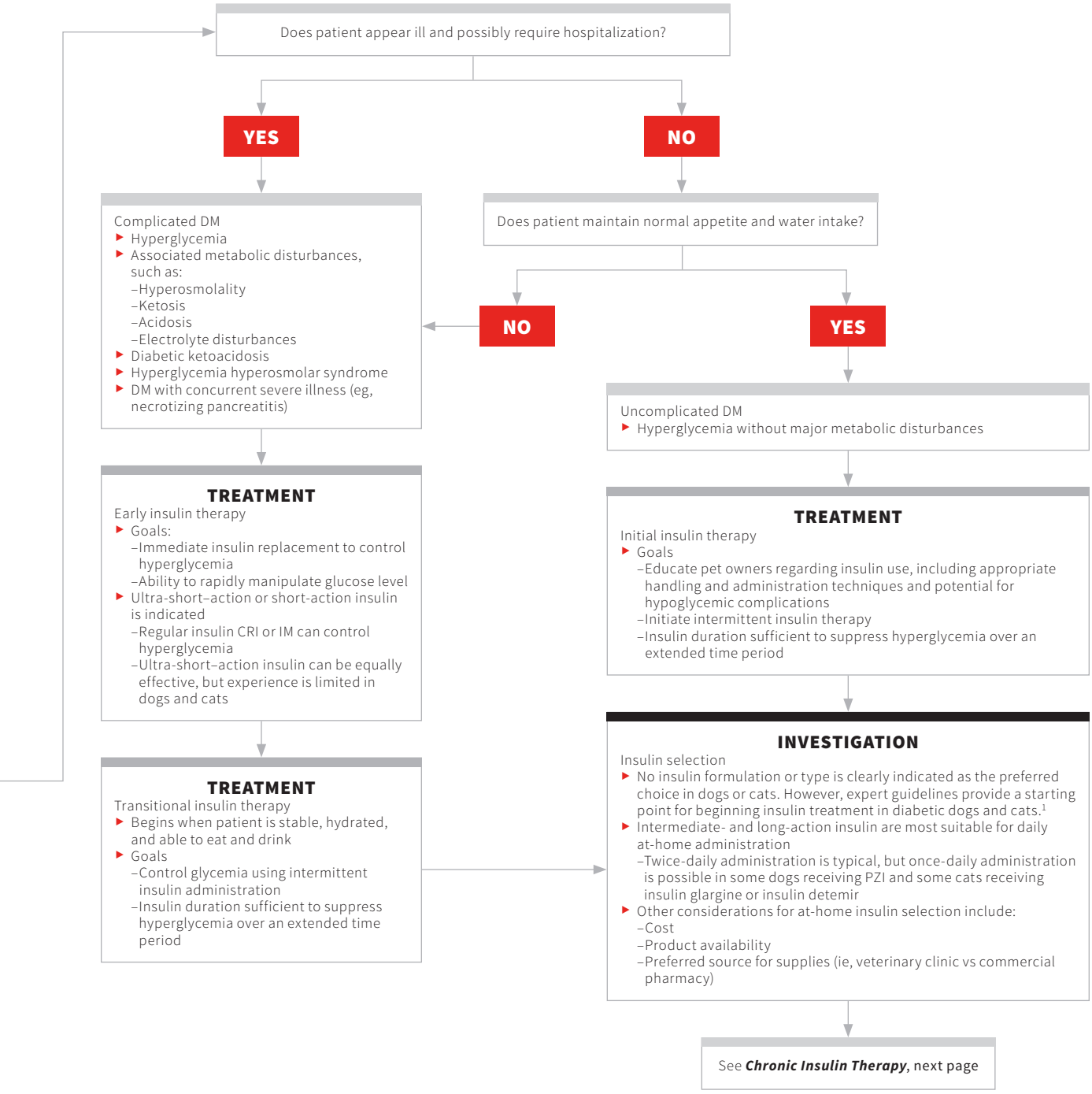


*Hyperglycemia may be partly caused by DM in these conditions, as DM may be present at the same time as or as a consequence of these disorders.



See ***Chronic Insulin Therapy***, next page

TREATMENT

Chronic insulin therapy

Long-term insulin therapy is guided by information obtained through periodic monitoring.

- Goals:
  - –Acceptable glycemic control and hypoglycemia avoidance
  - –Acceptable patient quality of life
  - –Treatment protocol balances patient needs and owner convenience
- Monitoring may include:
  - –Clinical assessment of DM signs, body weight, BCS, activity level, and general health
    - Periodic use of urine glucose and ketone monitoring is occasionally recommended. Large changes in urine glucose and/or the appearance of ketonuria may indicate deterioration of diabetes control in previously regulated dogs and cats. Absence of glucosuria can indicate hypoglycemia.
  - –Glycated protein assessment
    - Serum fructosamine and/or blood hemoglobin A1c levels provide information on blood glucose concentrations over preceding several weeks and months, respectively.
  - –Glycemic profile assessment
    - Can be assessed using standard 12- or 24-hour glucose curve(s) or continuous glucose monitoring (eg, interstitial glucose monitoring device) methods. These are the only routine methods that assess the pharmacodynamic profile of injected insulin in diabetic dogs and cats and are important to evaluate before making a change in insulin formulation (eg, intermediate- to long-action insulin) or type (eg, insulin glargine to insulin detemir).
    - Indications for performing a glucose curve include:
      - —Concern for hypoglycemia
      - —Requiring information not available through other assessments (eg, glycated protein measurement)
      - —Determining the time–action profile of insulin used
    - Curve results can be markedly influenced by the circumstances under which the curve is performed. Steps taken to minimize stress and anxiety (eg, performing measurements at home rather than in the clinic) may improve the reliability of information provided by the curve.

INVESTIGATION

- DM signs persist or signs of hypoglycemia are noted?
- Laboratory assessments indicate persistent hyperglycemia or frequent/intermittent hypoglycemia?
- Patient quality of life not optimal?

NO → No change needed

YES → Dose change may be needed

Dose adjustment guidelines:

- Reduce insulin dose by 25%-50% if hypoglycemia associated with clinical signs (eg, lethargy, weakness, seizure, among others) is noted or if there is clear evidence for biochemical hypoglycemia
  - –Discontinue insulin if there is evidence of persistent hypoglycemia
  - –Some cats receiving long-term insulin may develop persistent, often subclinical hypoglycemia, which may indicate diabetes remission
- Dose increase is typically done in 10%-20% increments in dogs
- Minimal dose change in cats is usually 0.5 Unit because of difficulty in accurately providing smaller volumes
- Switching to twice-daily administration is an option when patients on once-daily insulin administration require a dose increase due to persistent hyperglycemia. Dividing the daily dose may reduce the risk for hypoglycemia following a dose increase

Insulin (ie, formulation, type) change

- Switch insulin based on:
  - –Evidence for insufficient insulin duration
  - –Substantial peak effect that produces hypoglycemia
  - –Whether reduction in administration frequency is desired
- New insulin should have characteristics or features that address the identified deficiencies
  - –For example, if NPH has abbreviated duration of action in a dog, PZI may be selected as the new insulin because of its generally longer duration of action

Re-evaluate 1-2 weeks after any adjustment to the insulin protocol

Reference

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DM = diabetes mellitus

NPH = neutral protamine Hageman

PZI = protamine zinc insulin

HYPOGLYCEMIA

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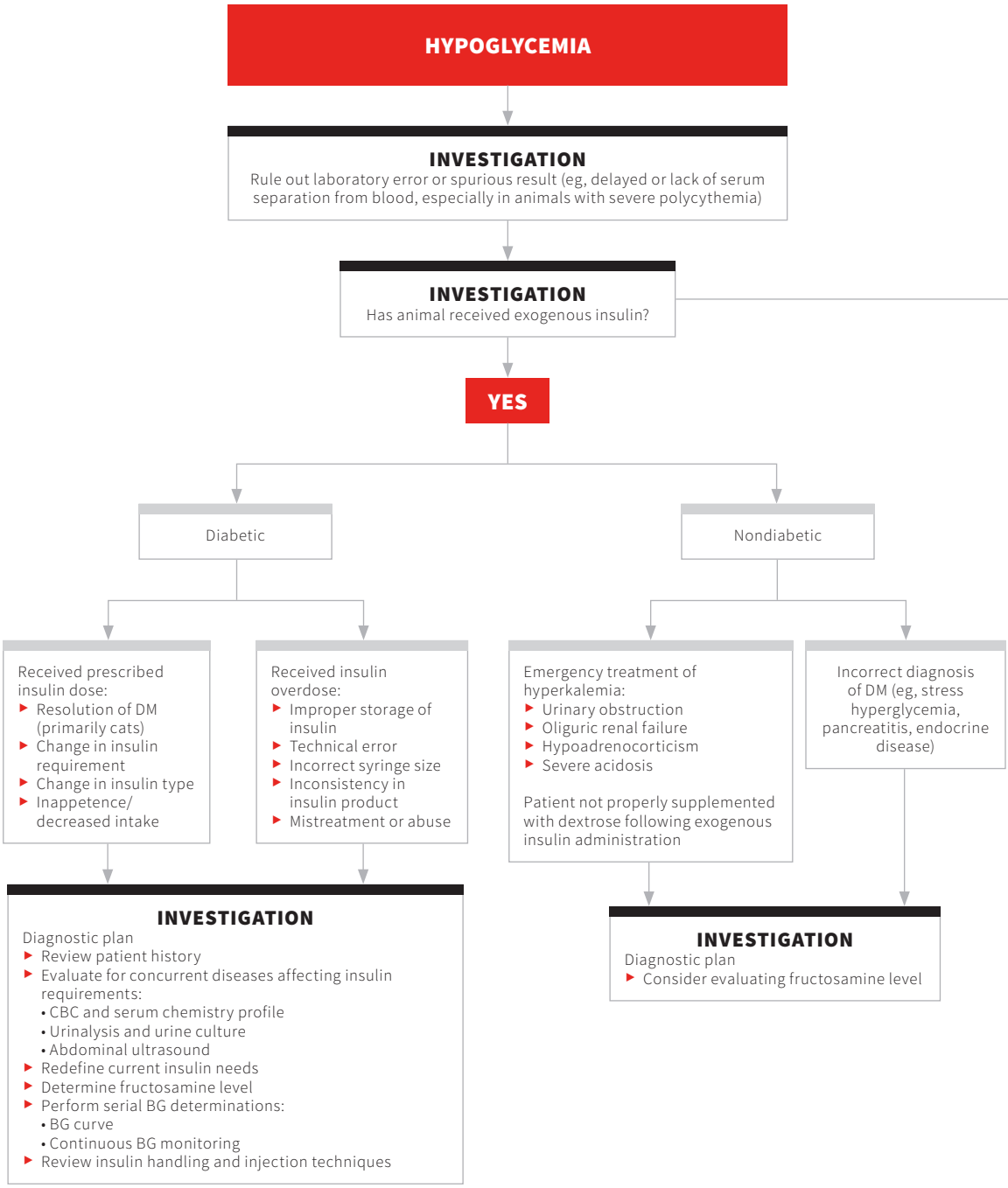

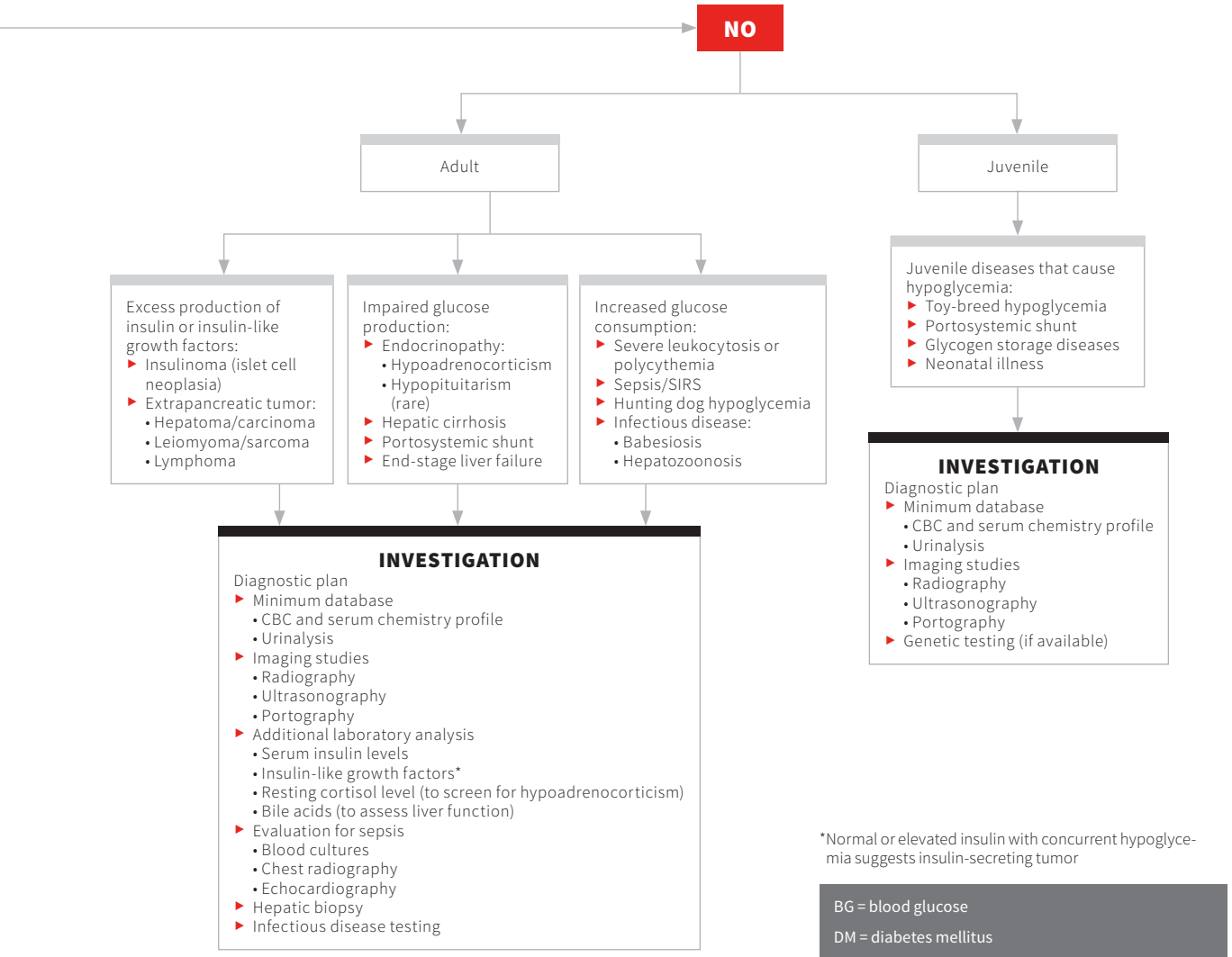

*Normal or elevated insulin with concurrent hypoglycemia suggests insulin-secreting tumor

BG = blood glucose

DM = diabetes mellitus

SIRS = systemic inflammatory response syndrome

LACK OF DIABETIC CONTROL IN CATS

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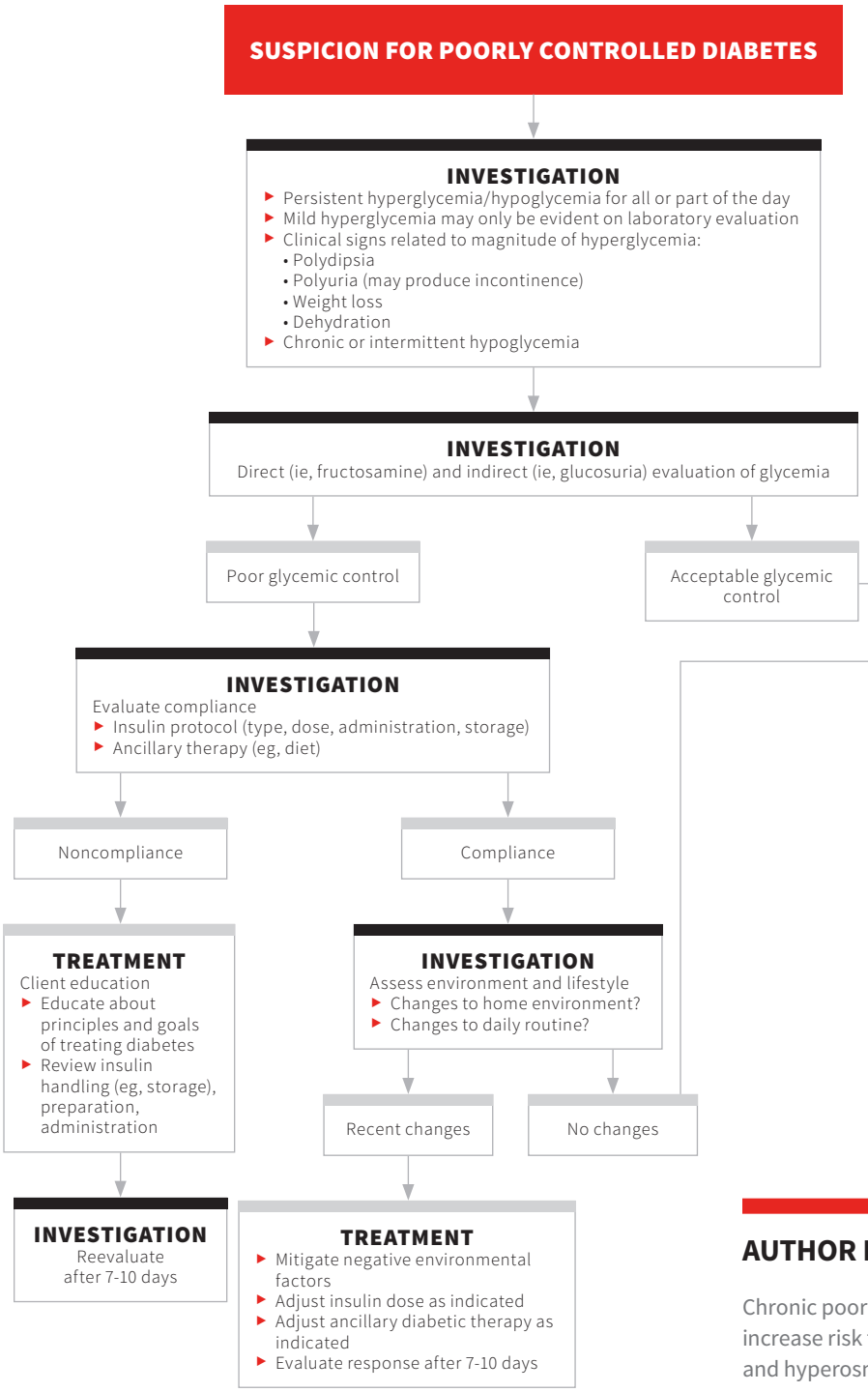
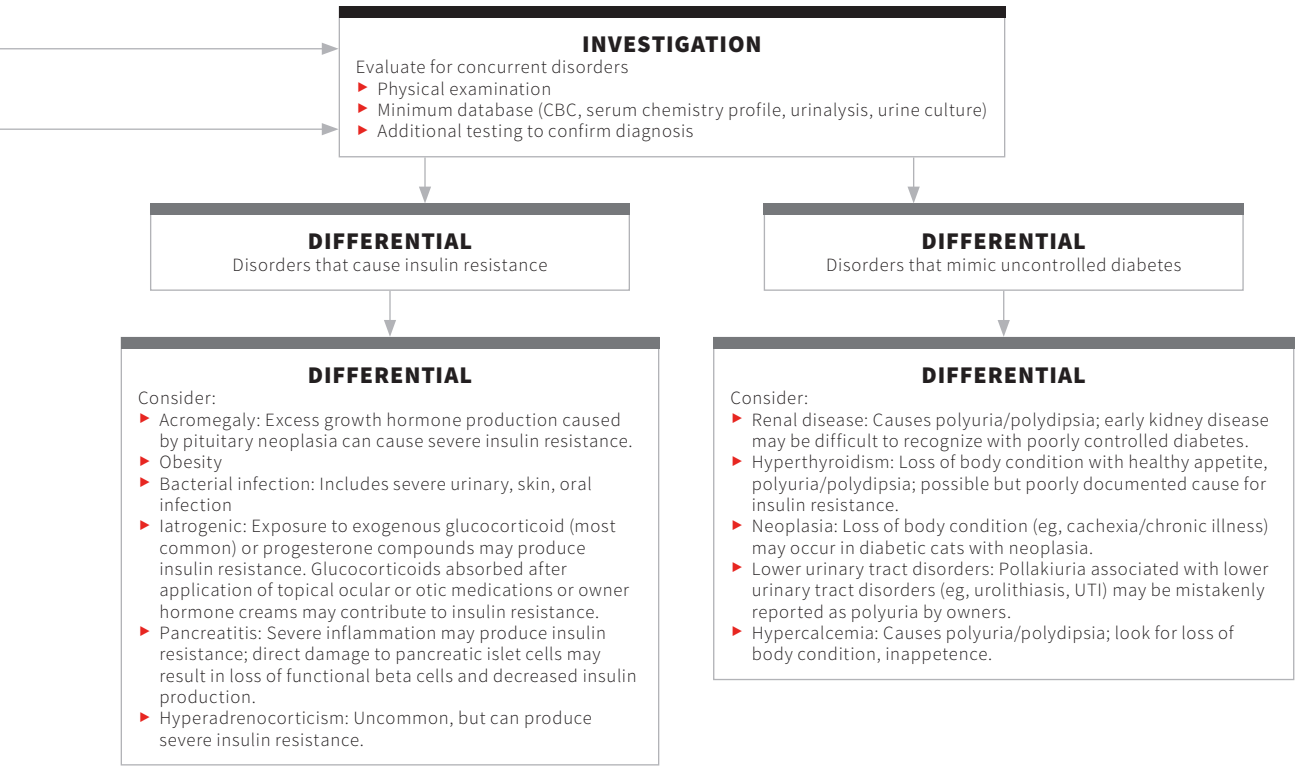


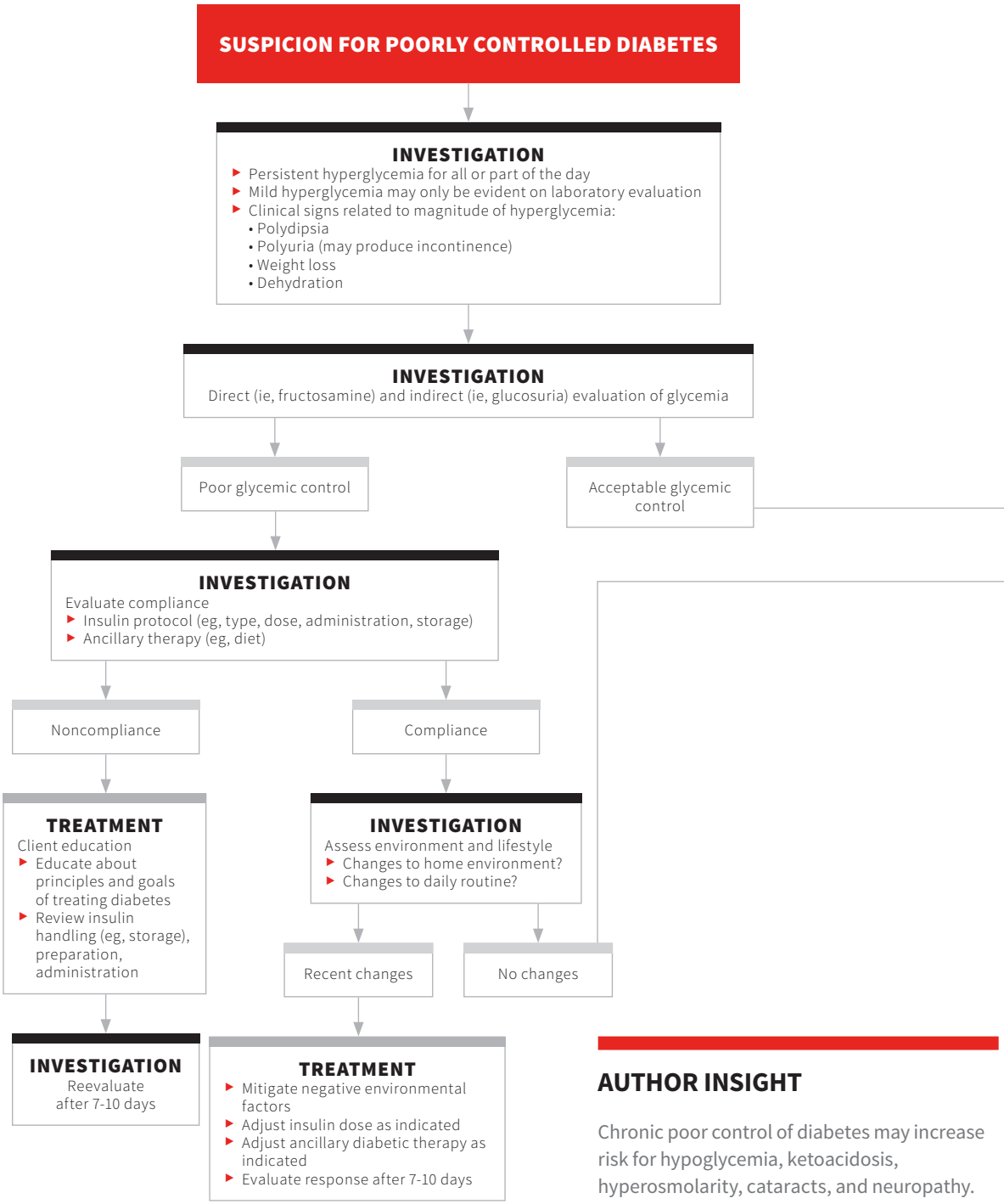
AUTHOR INSIGHT

Chronic poor control of diabetes may increase risk for hypoglycemia, ketoacidosis, and hyperosmolarity.


INVESTIGATION
Evaluate for concurrent disorders
Physical examination
Minimum database (CBC, serum chemistry profile, urinalysis, urine culture)
Additional testing to confirm diagnosis
DIFFERENTIAL
Disorders that cause insulin resistance
DIFFERENTIAL
Disorders that mimic uncontrolled diabetes
DIFFERENTIAL
Consider:
Acromegaly: Excess growth hormone production caused by pituitary neoplasia can cause severe insulin resistance.
Obesity
Bacterial infection: Includes severe urinary, skin, oral infection
Iatrogenic: Exposure to exogenous glucocorticoid (most common) or progesterone compounds may produce insulin resistance. Glucocorticoids absorbed after application of topical ocular or otic medications or owner hormone creams may contribute to insulin resistance.
Pancreatitis: Severe inflammation may produce insulin resistance; direct damage to pancreatic islet cells may result in loss of functional beta cells and decreased insulin production.
Hyperadrenocorticism: Uncommon, but can produce severe insulin resistance.
DIFFERENTIAL
Consider:
Renal disease: Causes polyuria/polydipsia; early kidney disease may be difficult to recognize with poorly controlled diabetes.
Hyperthyroidism: Loss of body condition with healthy appetite, polyuria/polydipsia; possible but poorly documented cause for insulin resistance.
Neoplasia: Loss of body condition (eg, cachexia/chronic illness) may occur in diabetic cats with neoplasia.
Lower urinary tract disorders: Pollakiuria associated with lower urinary tract disorders (eg, urolithiasis, UTI) may be mistakenly reported as polyuria by owners.
Hypercalcemia: Causes polyuria/polydipsia; look for loss of body condition, inappetence.

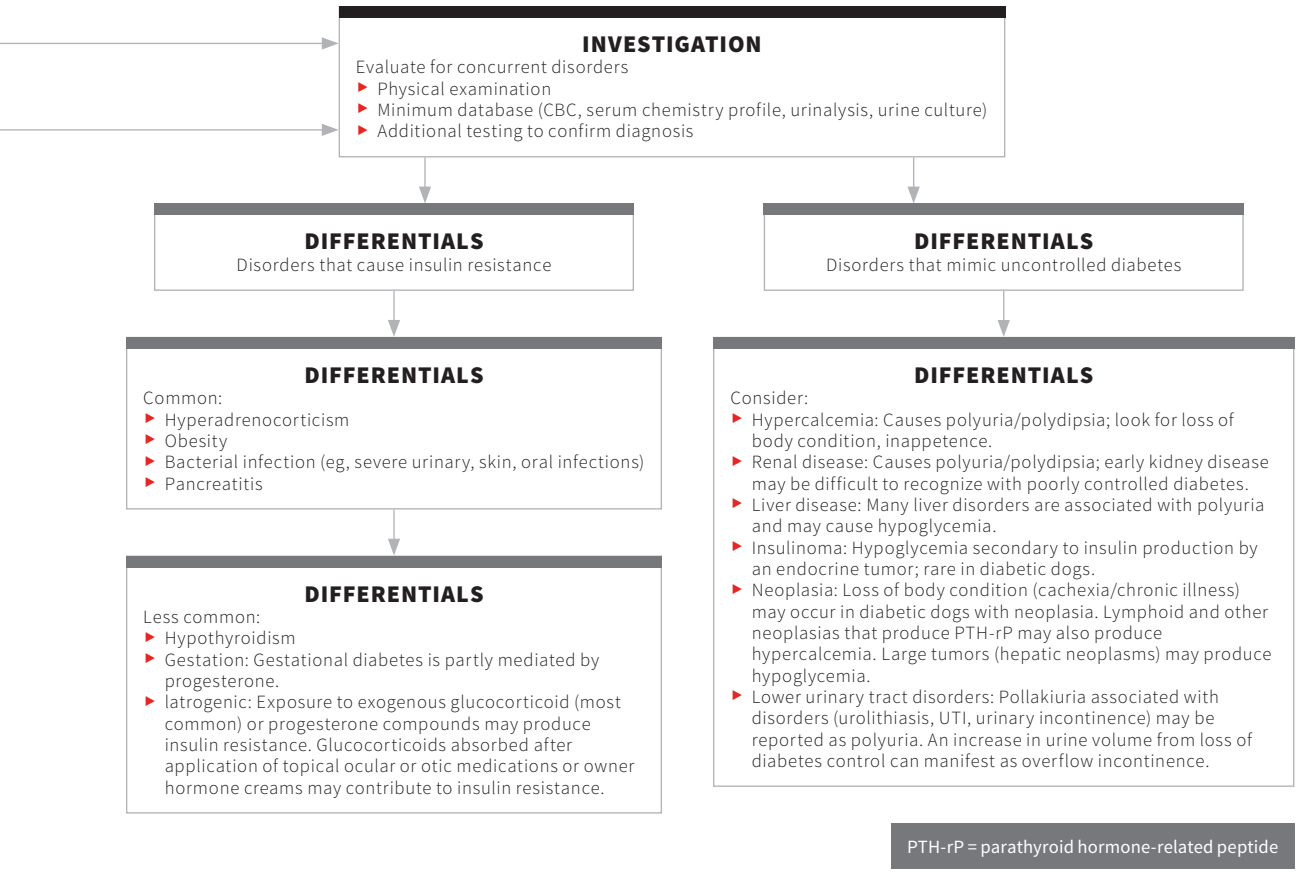
LACK OF DIABETIC CONTROL IN DOGS

Thomas Schermerhorn, VMD, DACVIM (SAIM)
Kansas State University



AUTHOR INSIGHT

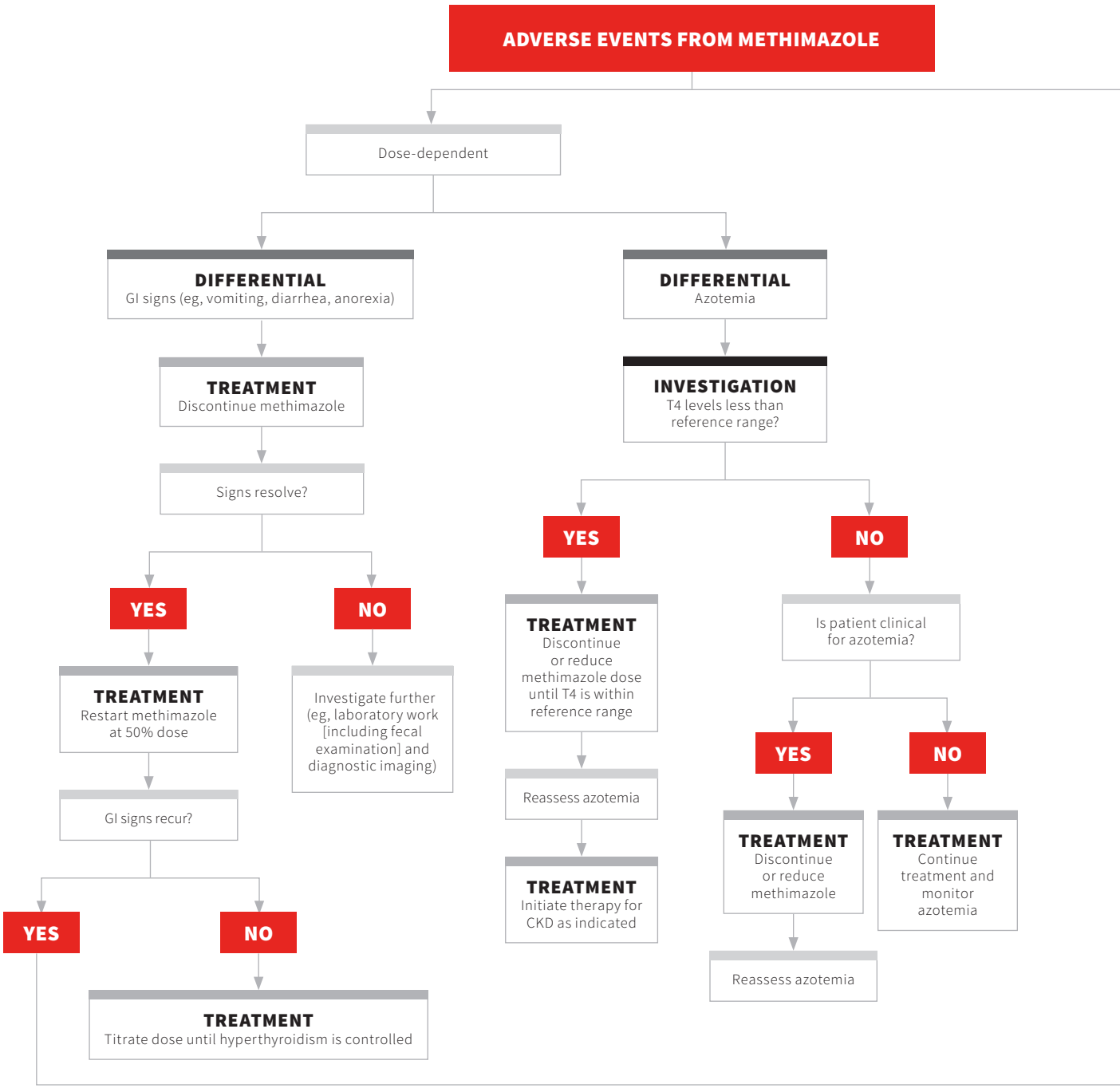
Chronic poor control of diabetes may increase risk for hypoglycemia, ketoacidosis, hyperosmolarity, cataracts, and neuropathy.

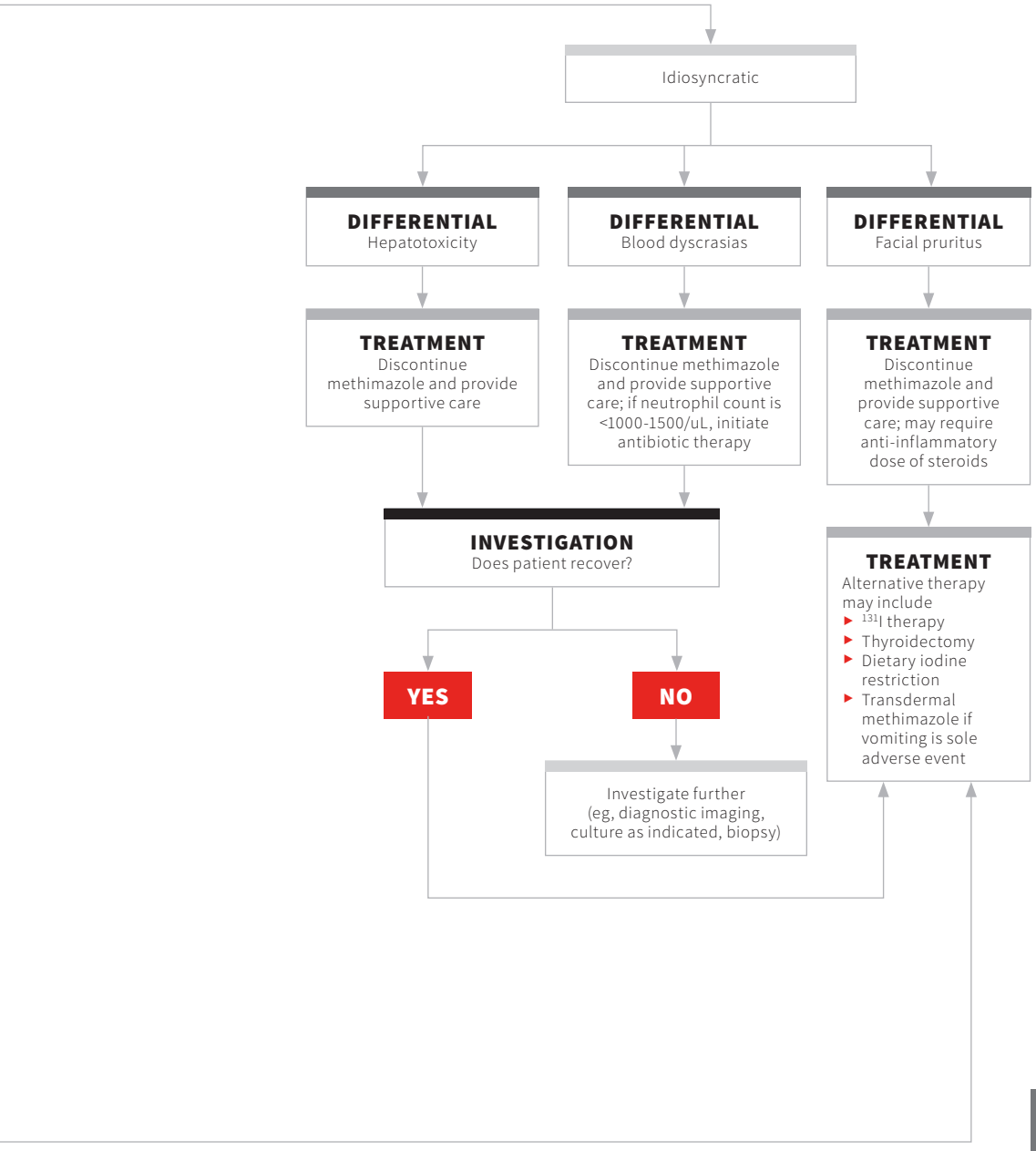


PTH-rP = parathyroid hormone-related peptide

METHIMAZOLE INTOLERANCE IN A HYPERTHYROID CAT

Alex Gallagher, DVM, MS, DACVIM
University of Florida





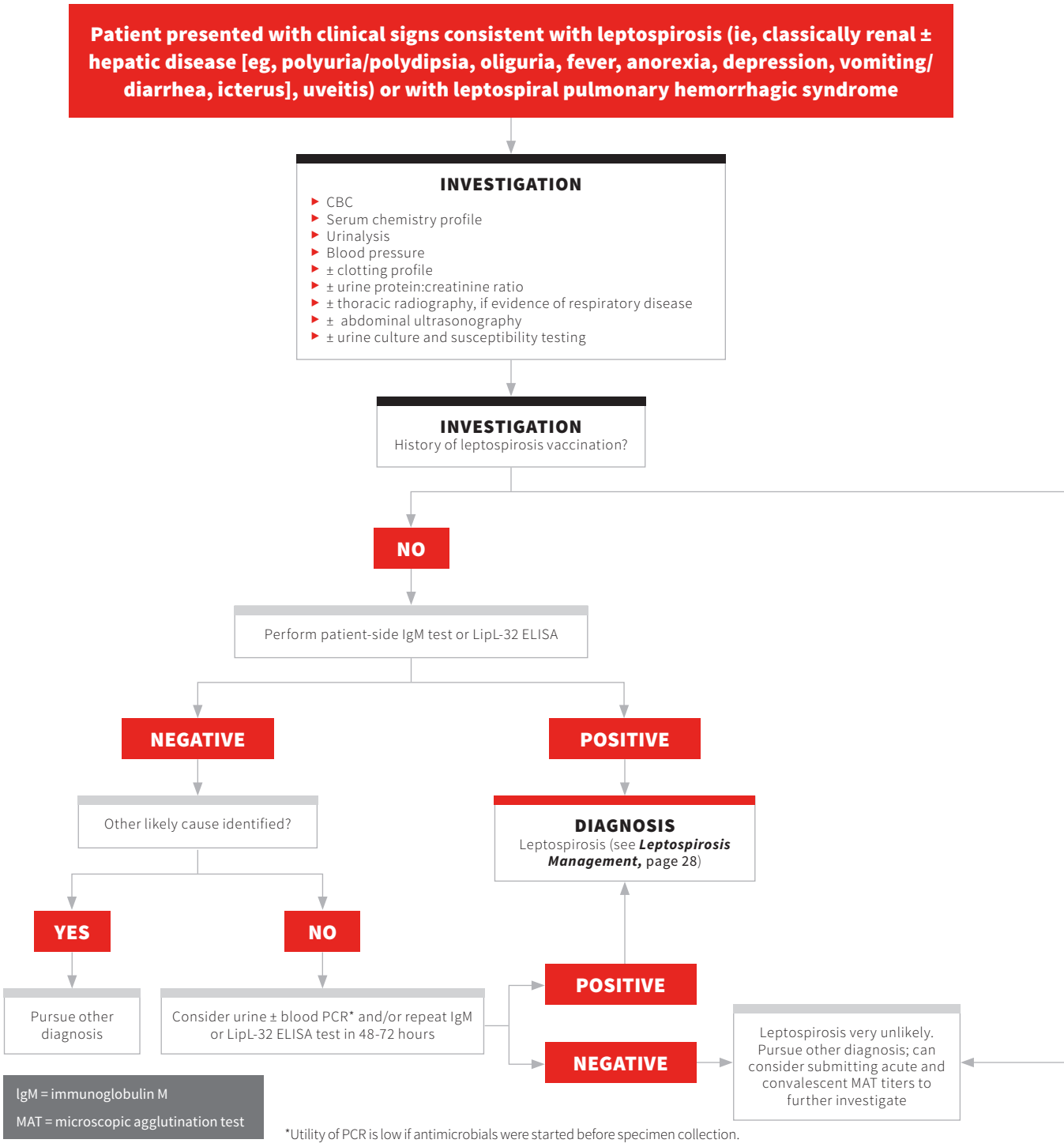
^{131}I = radioiodine

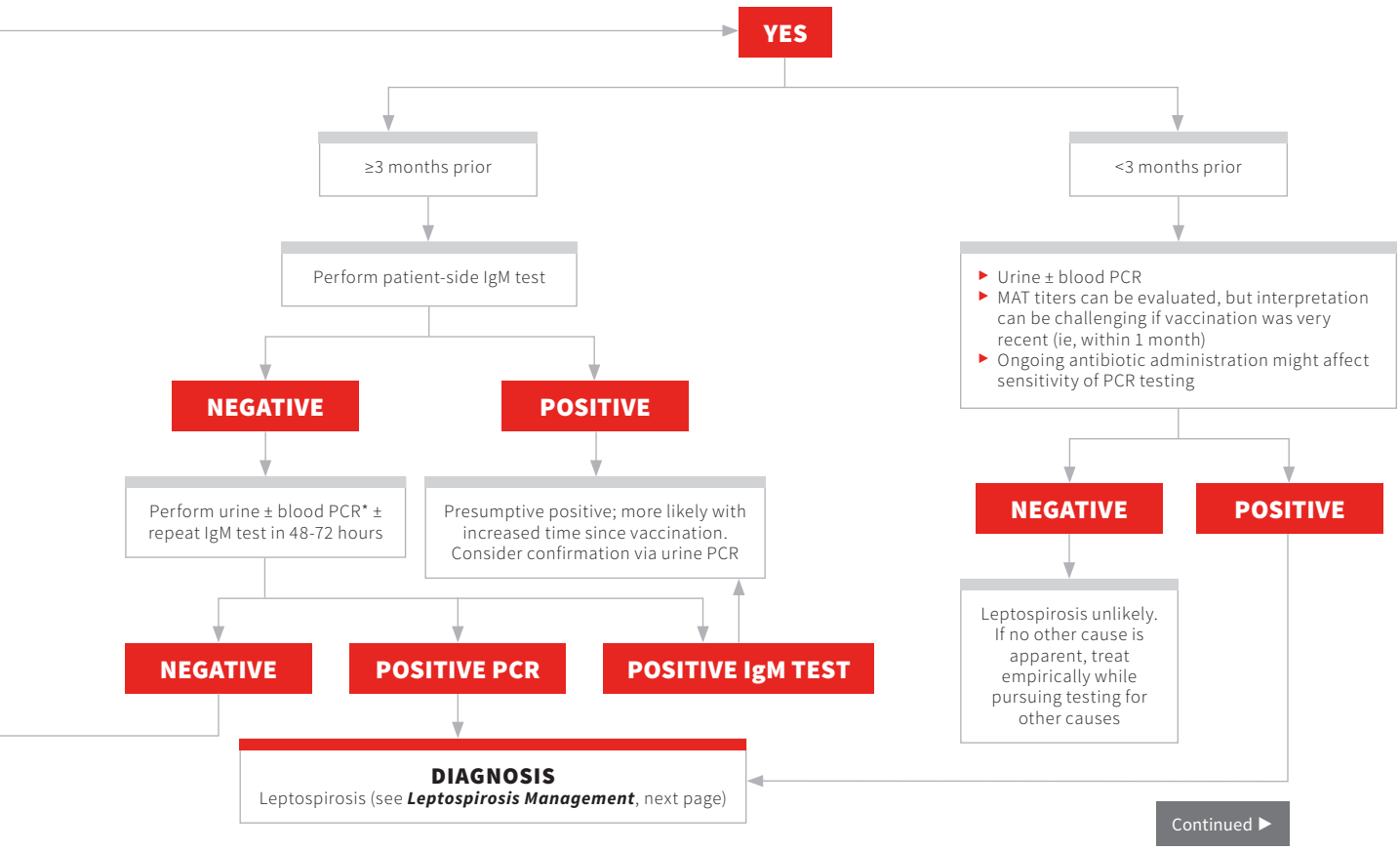
CKD = chronic kidney disease

T4 = total thyroxine

LEPTOSPIROSIS

J. Scott Weese, DVM, DVSc, DACVIM
Ontario Veterinary College
Ontario, Canada




YES
≥3 months prior
Perform patient-side IgM test
NEGATIVE
POSITIVE
Perform urine ± blood PCR* ± repeat IgM test in 48-72 hours
Presumptive positive; more likely with increased time since vaccination. Consider confirmation via urine PCR
NEGATIVE
POSITIVE PCR
POSITIVE IgM TEST
DIAGNOSIS
Leptospirosis (see ***Leptospirosis Management***, next page)
<3 months prior
Urine ± blood PCR
MAT titers can be evaluated, but interpretation can be challenging if vaccination was very recent (ie, within 1 month)
Ongoing antibiotic administration might affect sensitivity of PCR testing
NEGATIVE
POSITIVE
Leptospirosis unlikely. If no other cause is apparent, treat empirically while pursuing testing for other causes

Continued ▶

Leptospirosis Management

Once leptospirosis diagnosis is confirmed, patients should be treated with antimicrobials and supportive care as needed.

Antimicrobials

- If the patient can tolerate oral medication: Doxycycline (5 mg/kg PO every 12 hours) for 14 days[1])
- If the patient cannot tolerate oral medication: Ampicillin (20 mg/kg IV every 6 hours), then, if possible, de-escalated to oral doxycycline (5 mg/kg PO every 12 hours) for an additional 14 days[1]

Supportive Care

- IV fluids for replacement, diuresis, acid-base balance, and electrolyte maintenance
- Antiemetics
- Nutritional support for renal or hepatic injury
- Renal replacement therapy can be considered in oliguric dogs developing volume overload, severe hyperkalemia, or severe azotemia nonresponsive to medical management.[1]
- Other care as needed based on clinical syndrome and patient response to treatment

During hospitalization, hydration status should be carefully monitored (ie, measure "ins and outs," thoracic auscultation, blood pressure), as should BUN/creatinine, acid-base/electrolytes, ± hepatic enzymes (as often as every 24 hours initially). PCV should be rechecked as often as every 24 hours initially, and CBC should be repeated as often as every 48 hours if thrombocytopenia is present and/or in severe cases. Urine specific gravity should also be rechecked every few days once fluid therapy has been discontinued, and clotting factors should be rechecked if abnormal.

Approximately 1 week after the patient is discharged, serum chemistry profile should be repeated, as should CBC if abnormalities were present at the time of discharge. Serum chemistry profile should be rechecked again in 3 to 7 days if results are still abnormal. Urine specific gravity should be monitored regularly if abnormal.

TABLE

LEPTOSPIROSIS TESTS & CONSIDERATIONS

Test	Target	Sample type	Patient-side?	Impacted by vaccination?	Impacted by antimicrobial treatment?
MAT	Antibody (IgM and IgG)	Serum	No	Yes	No
Lepto rapid test	Antibody (IgM)	Serum	Yes	Yes	No
LipL-32 Leptospira	Antibody (IgG>IgM)	Serum	Yes	Yes	No
PCR	Antigen	Urine, whole blood	No	No	Potentially

Reference

1. Sykes JE, Hartmann K, Lunn KF, Moore GE, Stoddard RA, Goldstein RE. 2010 ACVIM small animal consensus statement on leptospirosis: diagnosis, epidemiology, treatment, and prevention. *J Vet Intern Med*. 2011;25(1):1-13.

Suggested Reading

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Curtis KM, Foster PC, Smith PS, et al. Performance of a recombinant LipL32 based rapid in-clinic ELISA (SNAP Lepto) for the detection of antibodies against Leptospira in dogs. *Intern J Appl Res Vet Med*. 2015;13(3):182-189.

Lizer J, Grahlmann M, Hapke H, Velineni S, Lin D, Kohn B. Evaluation of a rapid IgM detection test for diagnosis of acute leptospirosis in dogs. *Vet Rec*. 2017;180(21):517.

Lizer J, Velineni S, Weber A, Krecic M, Meeus P. Evaluation of 3 serological tests for early detection of Leptospira-specific antibodies in experimentally infected dogs. *J Vet Intern Med*. 2018;32(1):201-207.

Midence JN, Leutenegger CM, Chandler AM, Goldstein RE. Effects of recent Leptospira vaccination on whole blood real-time PCR testing in healthy client-owned dogs. *J Vet Intern Med*. 2012;26(1):149-152.

Schuller S, Francey T, Hartmann K, et al. European consensus statement on leptospirosis in dogs and cats. *J Small Anim Pract*. 2015;56(3):159-179.

IgG = immunoglobulin G

lgM = immunoglobulin M

MAT = microscopic agglutination test

FELINE INFECTIOUS PERITONITIS

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Ontario Veterinary College
The Cat Clinic
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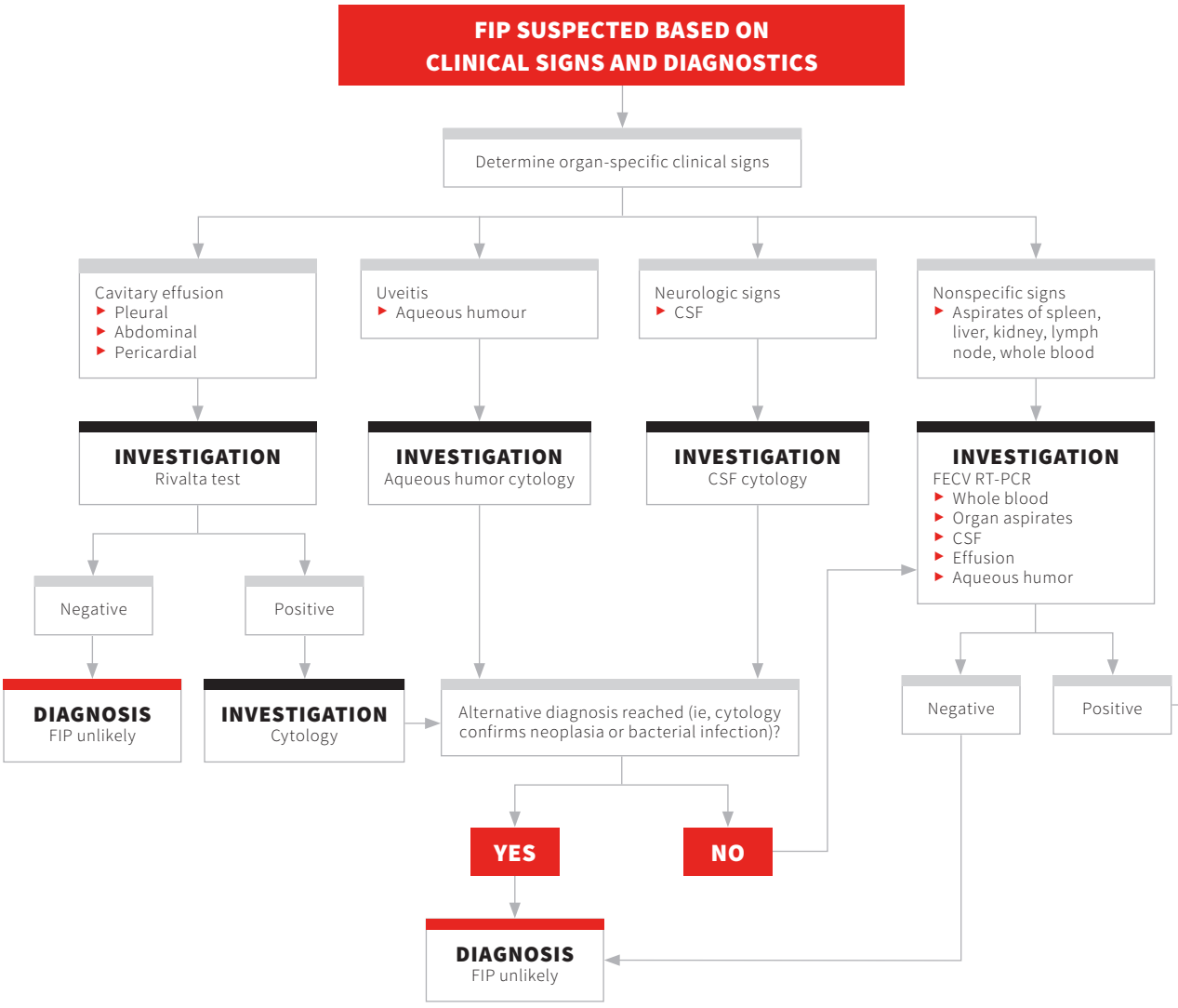
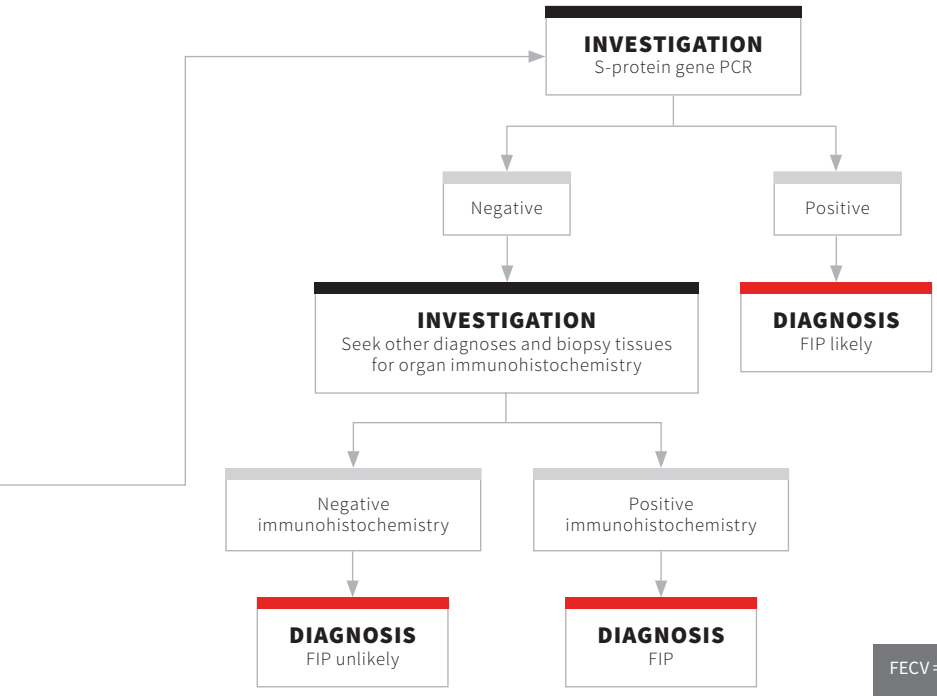
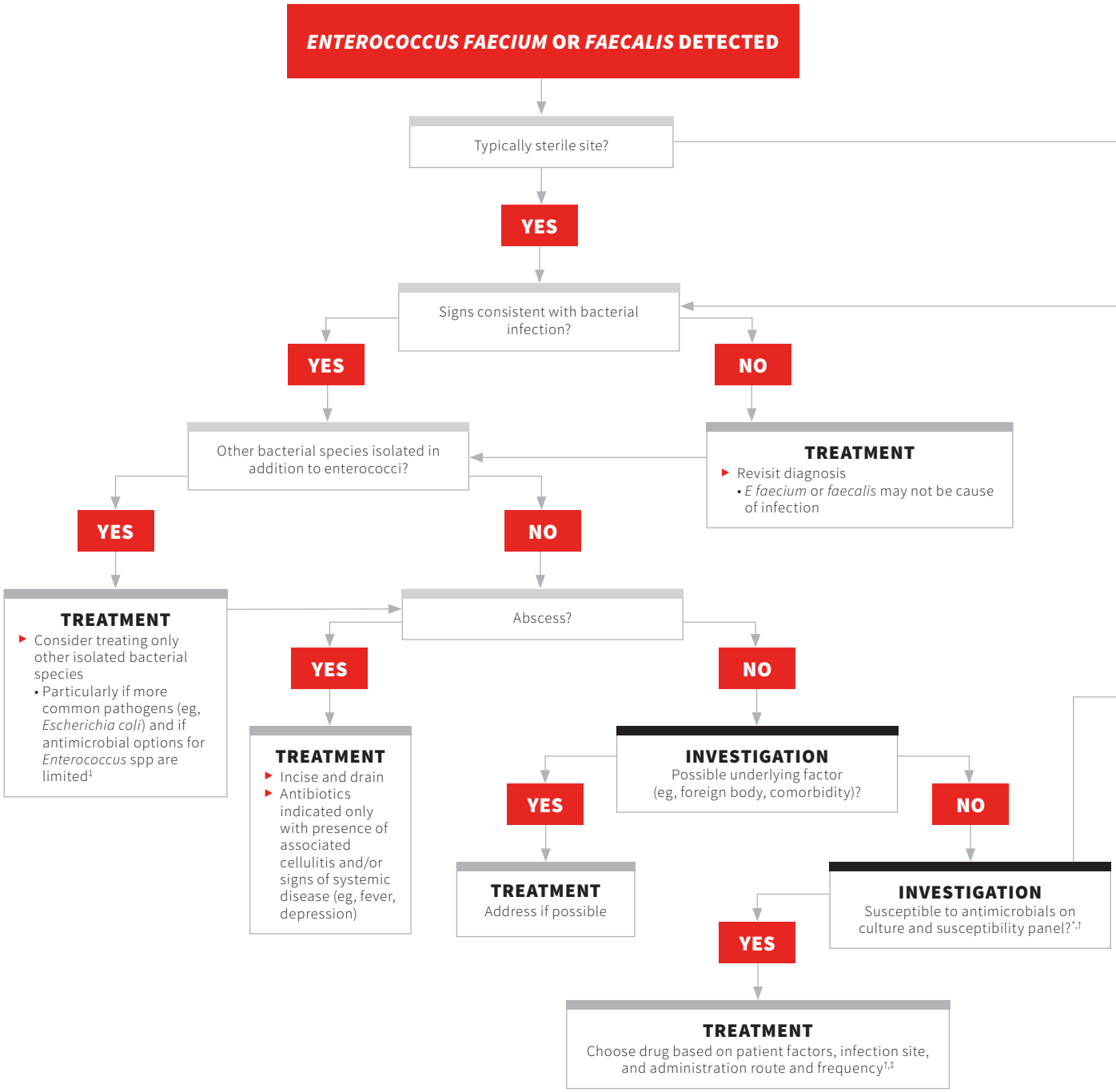


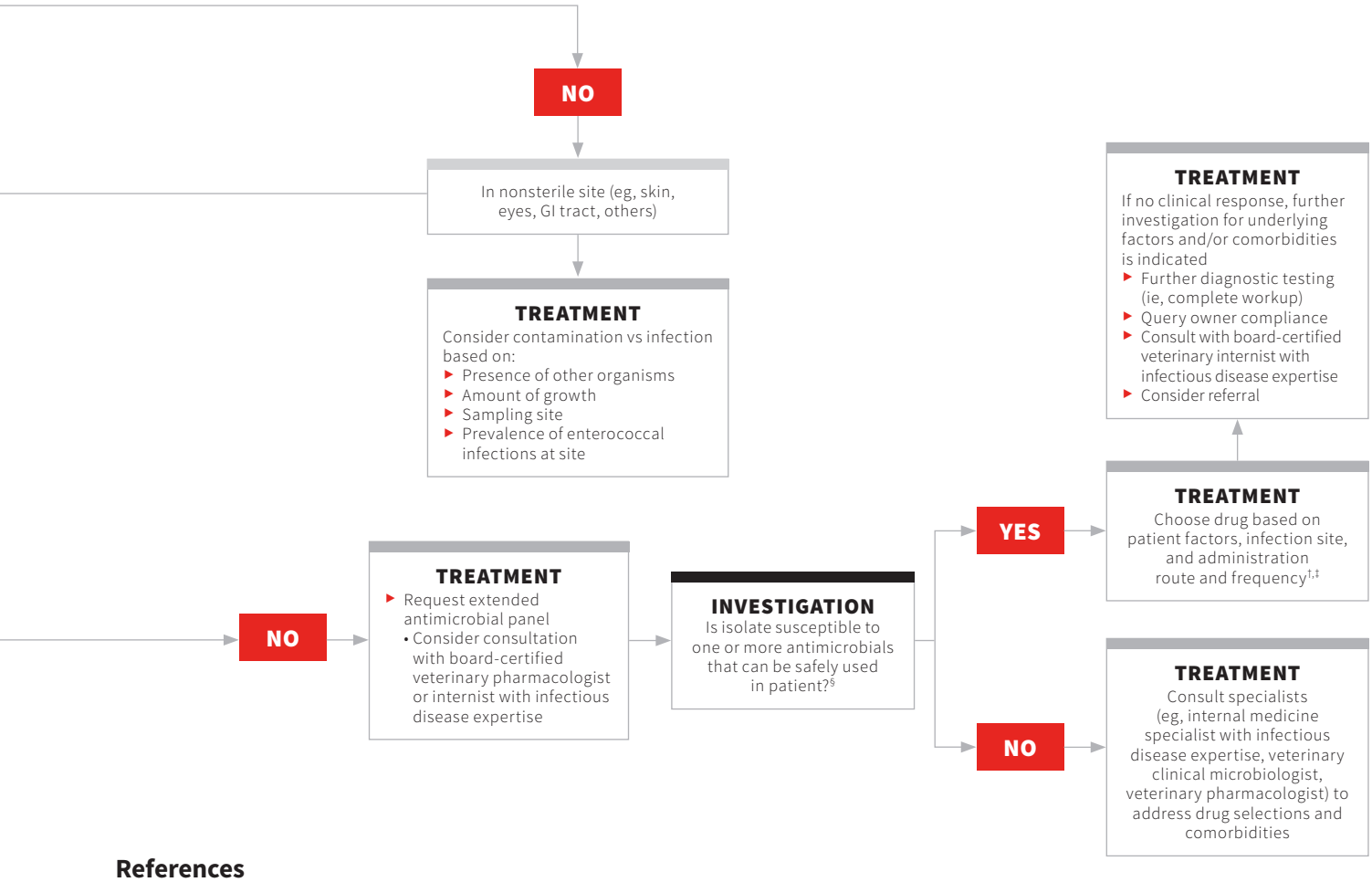


FECV = feline enteric coronavirus
RT = reverse transcriptase
S = spike

ISOLATION OF *ENTEROCOCCUS* SPP

J. Scott Weese, DVM, DVSc, DACVIM
University of Guelph





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1. Weese JS, Blondeau JM, Boothe D, et al. Antimicrobial use guidelines for treatment of urinary tract disease in dogs and cats: Antimicrobial guidelines working group of the International Society for Companion Animal Infectious Diseases. *Vet Med Int*. 2011;2011:263768.
2. Performance Standards for Antimicrobial Disk and Dilution Susceptibility Tests for Bacteria Isolated From Animals; Approved Standard, 4th ed—Wayne, PA: Clinical and Laboratory Standards Institute. CLSI Document VET01-A4. 33(7):1-72, 2013.

*Enterococci are inherently resistant to penicillin, trimethoprim-sulfa, clindamycin, and low levels of aminoglycosides. Cephalosporins have limited activity in vivo.[2] Most laboratories do not report results for these drugs.
†Consider ampicillin or gentamicin for isolates that do not have high-level gentamicin resistance.[2]
‡Amoxicillin and ampicillin are drugs of choice when the isolate is susceptible.
§Consult an infectious disease specialist and veterinary pharmacologist before considering drugs (eg, vancomycin, linezolid) to confirm that no as-effective, less costly, and/or safer alternatives are available.

ISOLATION OF METHICILLIN-RESISTANT *STAPHYLOCOCCUS*

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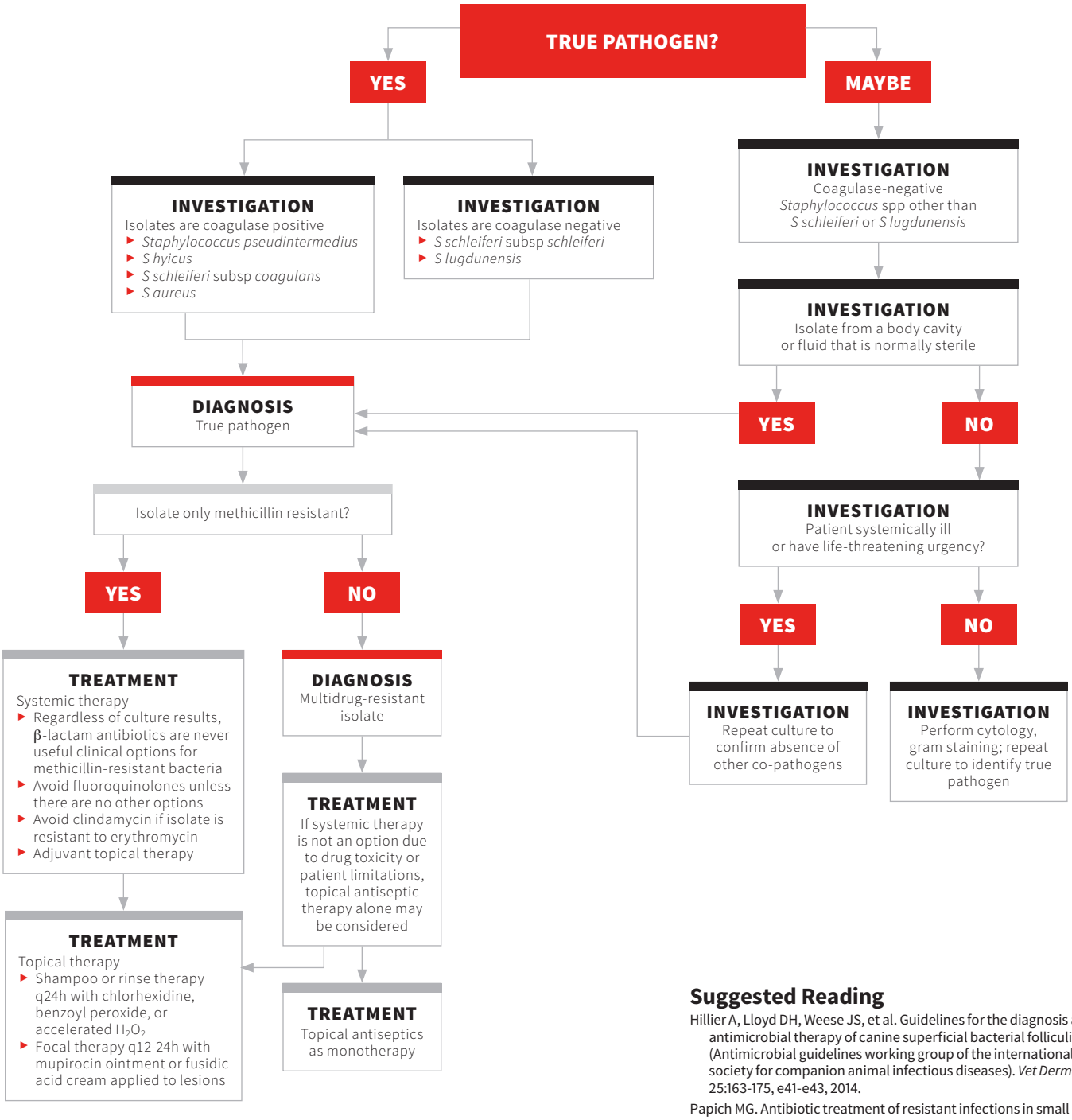
Karen A. Moriello, DVM, DACVD
University of Wisconsin–Madison



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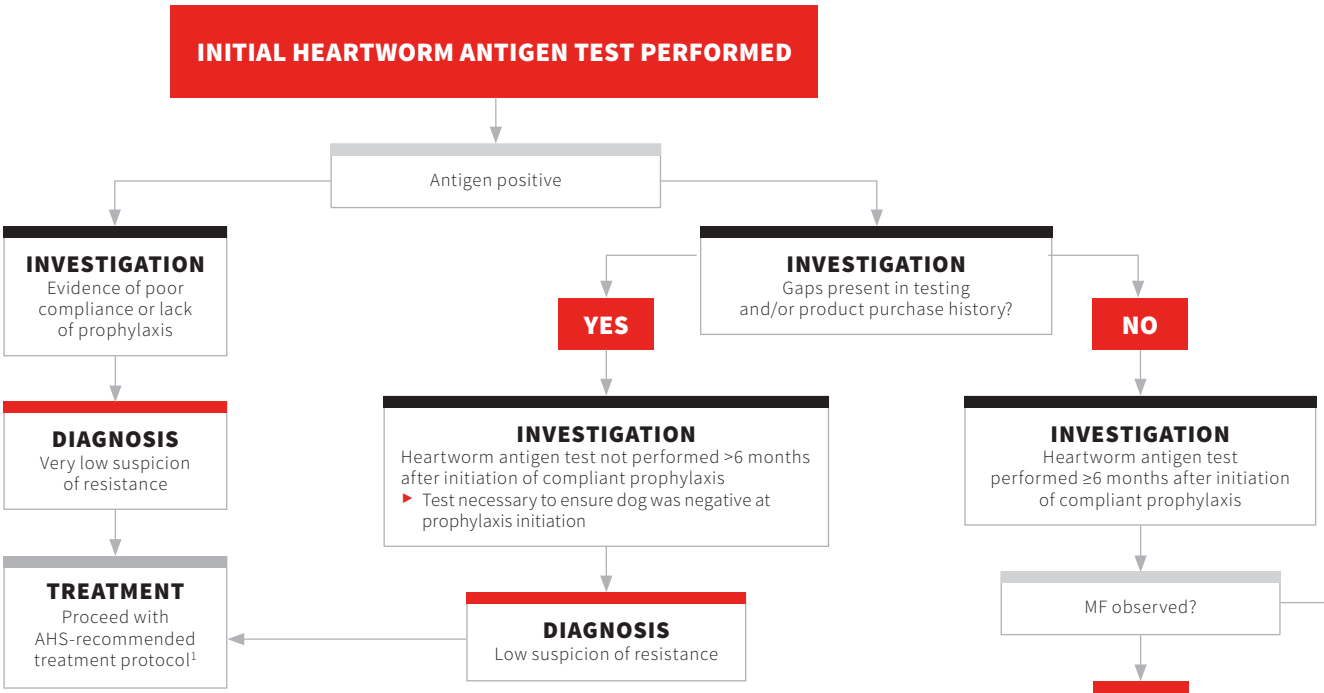
Papich MG. Antibiotic treatment of resistant infections in small animals. *Vet Clin North Am Small Anim Pract*. 43:1091-1107, 2013.

MACROCYCLIC LACTONE-RESISTANT HEARTWORM DISEASE

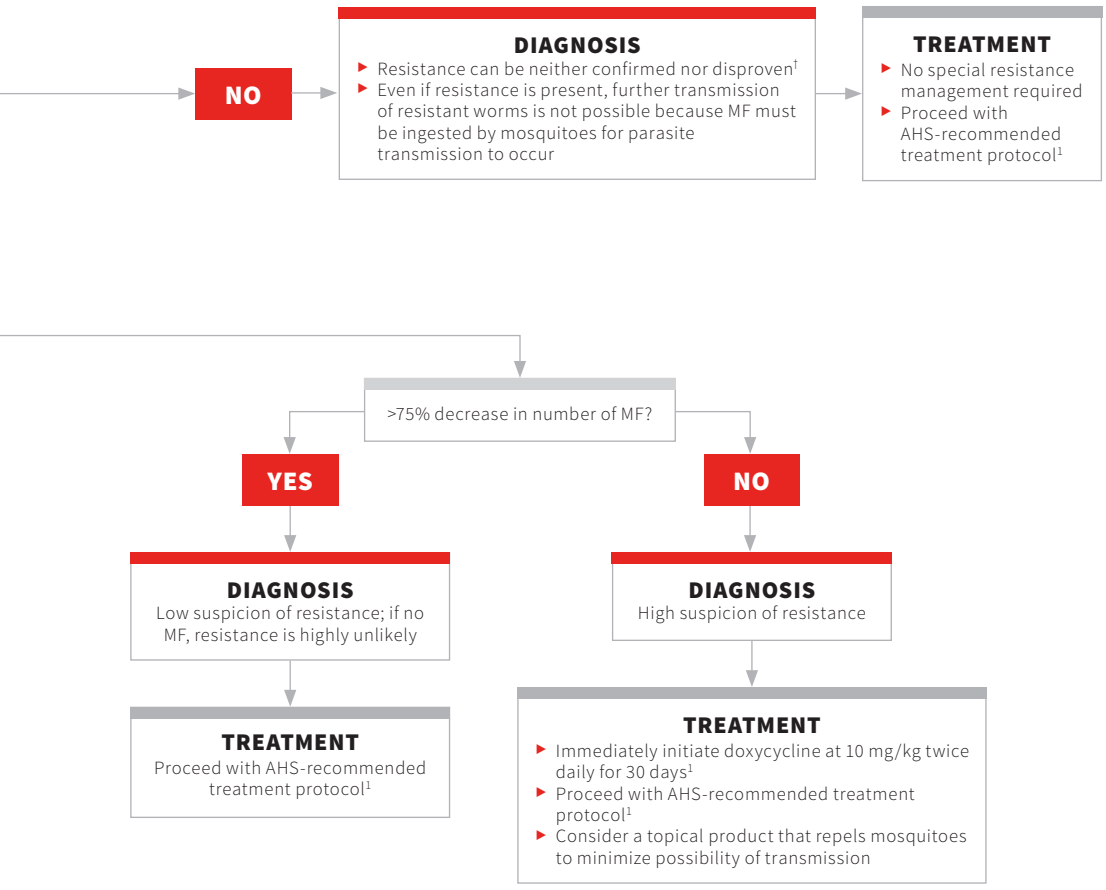
Andy Moorhead, DVM, MS, PhD
Ray M. Kaplan, DVM, PhD, DEVPC, DACVM (Parasitology)
University of Georgia



AUTHOR INSIGHT[1]

Resistance to ML preventives in canine heartworm cases has been proven, though there are few documented cases. To the authors' best knowledge, clinical patterns to date suggest that most proven cases of ML resistance in heartworms have been diagnosed in dogs residing in or translocated from the Mississippi Delta region and that cases of resistance are rare outside this region. However, it is possible that resistance is more widespread than currently recognized. No tests for resistance are available to determine the prevalence and distribution of resistance in heartworms,[5] making definitive diagnosis impossible.

AHS = American Heartworm Society
MF = microfilariae
ML = macrocyclic lactone



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2. Geary TG, Bourguinat C, Prichard RK. Evidence for macrocyclic lactone anthelmintic resistance in *Dirofilaria immitis*. *Top Companion Anim Med*. 2011;26(4):186-192.
3. Jackson RF, Seymour WG, Beckett RS. Routine use of 0.05 mg/kg of ivermectin as a microfilaricide. In: Otto GF, ed. Proceedings of the Heartworm Symposium, 1986; Washington, DC: 37-39.
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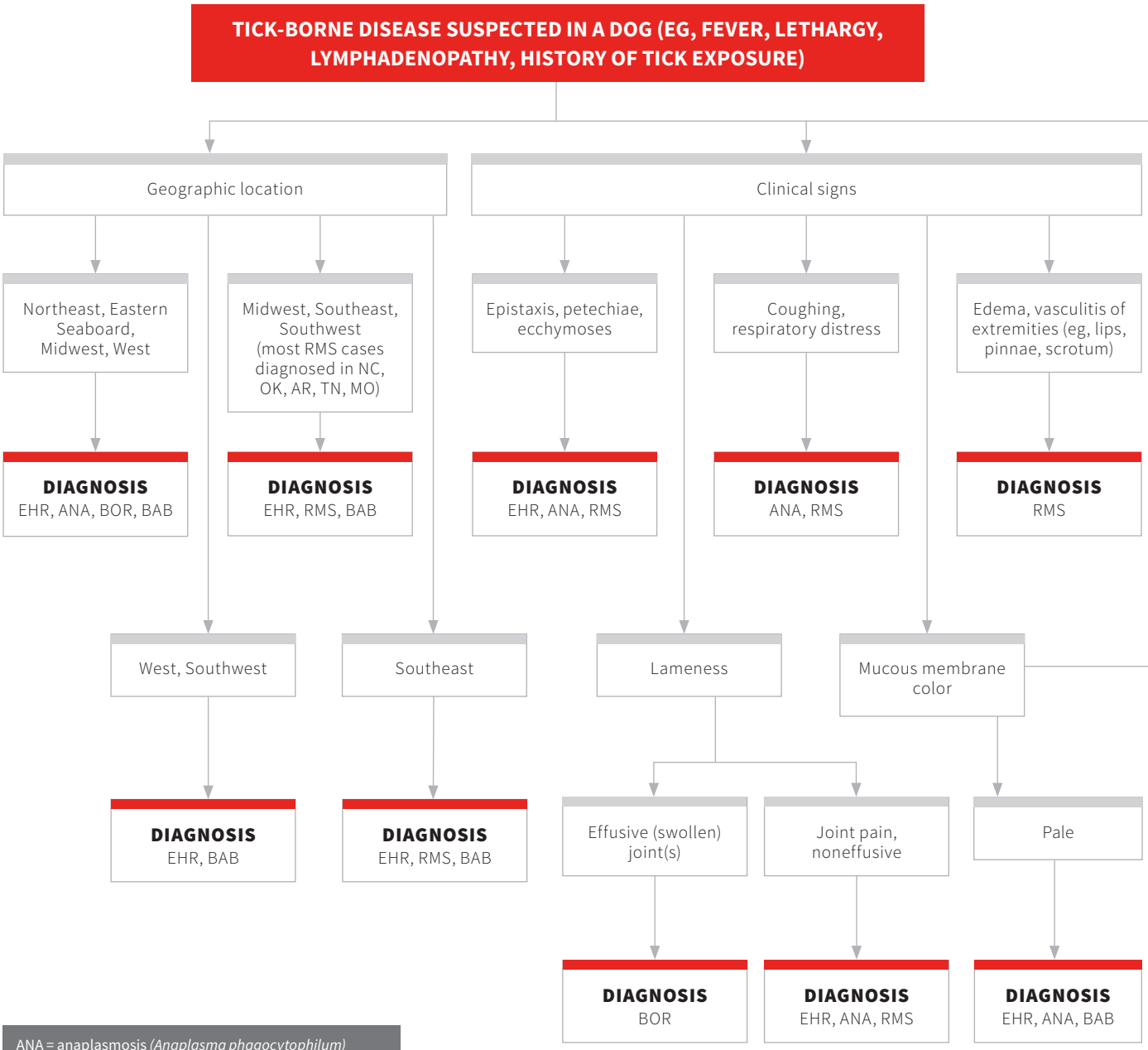
*Performance of the MF suppression test is recommended by the authors solely to identify resistant heartworm cases and is not a normal standard of care procedure for treating heartworm cases.

†To the authors' knowledge, all proven cases of resistance as of time of original publication (November 2015) have been microfilaremic. Lack of MF suggests that the case does not involve resistance, the dog has no female worms, or infections are still immature and have not yet become patent.

TICK-BORNE INFECTIOUS DISEASE IN U.S. DOGS

Craig Datz, DVM, MS, DABVP (Canine & Feline Practice)*
University of Missouri



ANA = anaplasmosis *(Anaplasma phagocytophilum)*
BAB = babesiosis (*Babesia canis, B gibsoni*)
BOR = borreliosis (Lyme disease; *Borrelia burgdorferi*)
EHR = ehrlichiosis *(Ehrlichia canis, E ewingii)*
RMS = Rocky Mountain spotted fever *(Rickettsia rickettsii)*

*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Craig Datz, DVM, DABVP (Canine & Feline Practice), DACVN, at Royal Canin USA, Columbia, Missouri.

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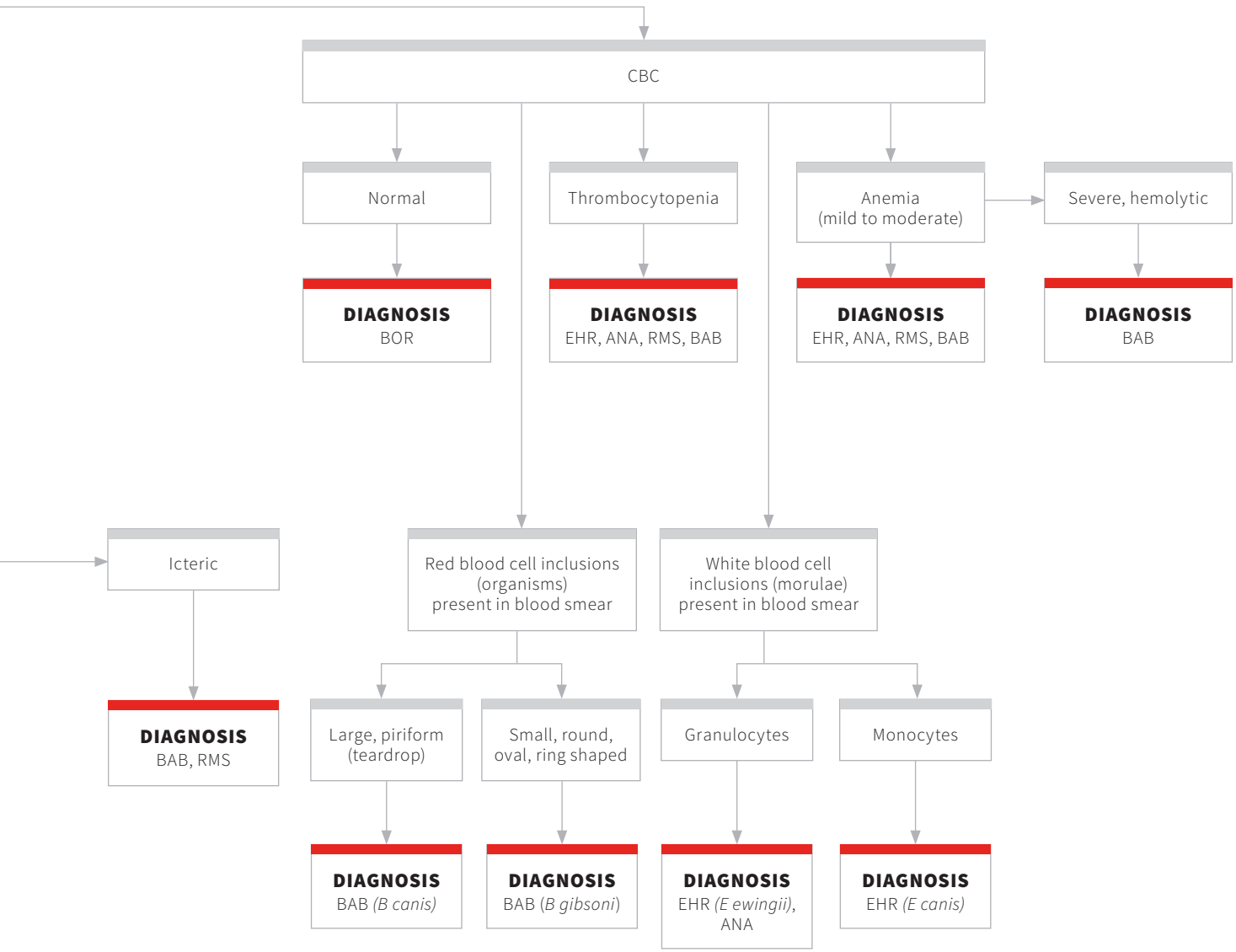
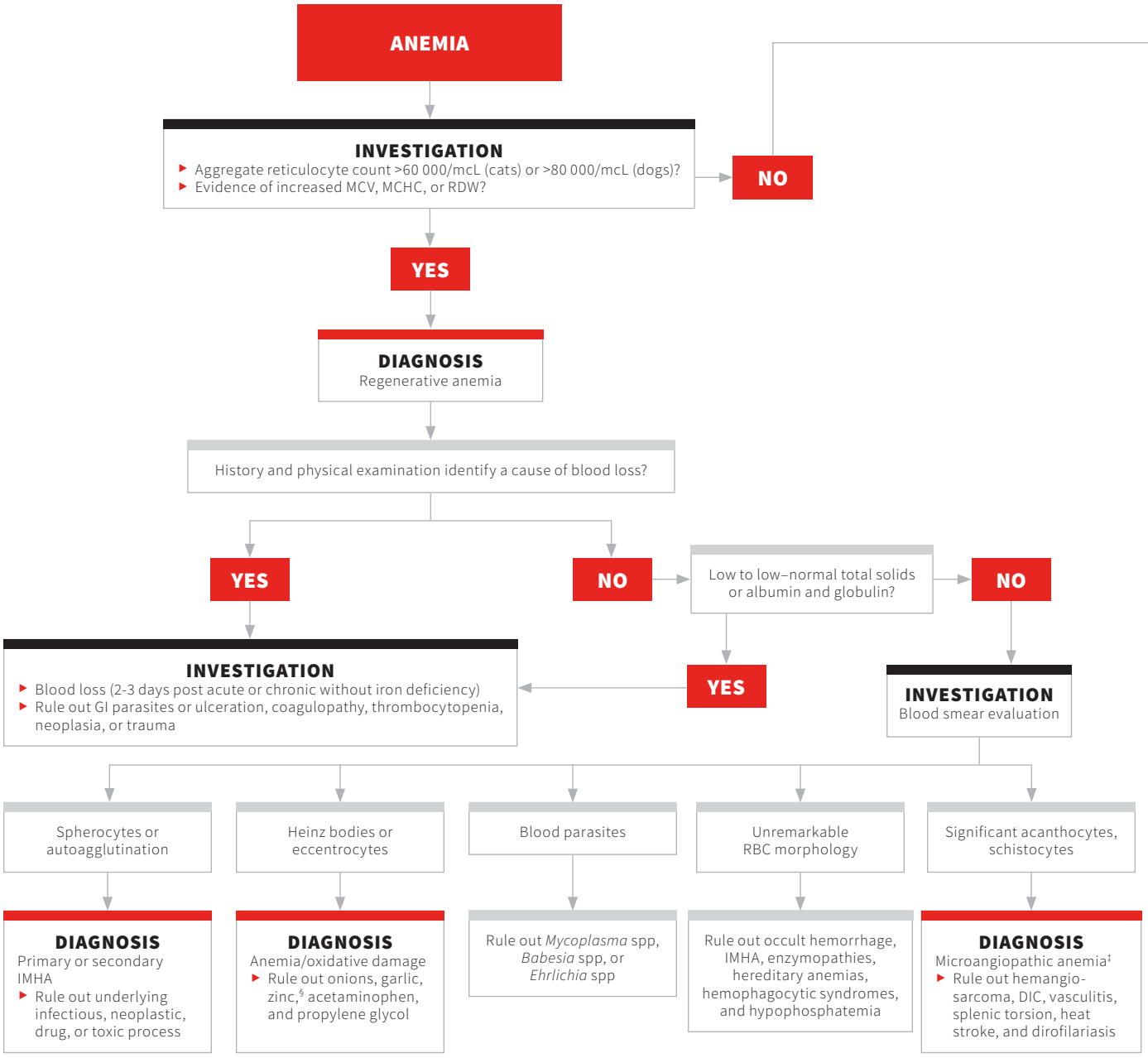


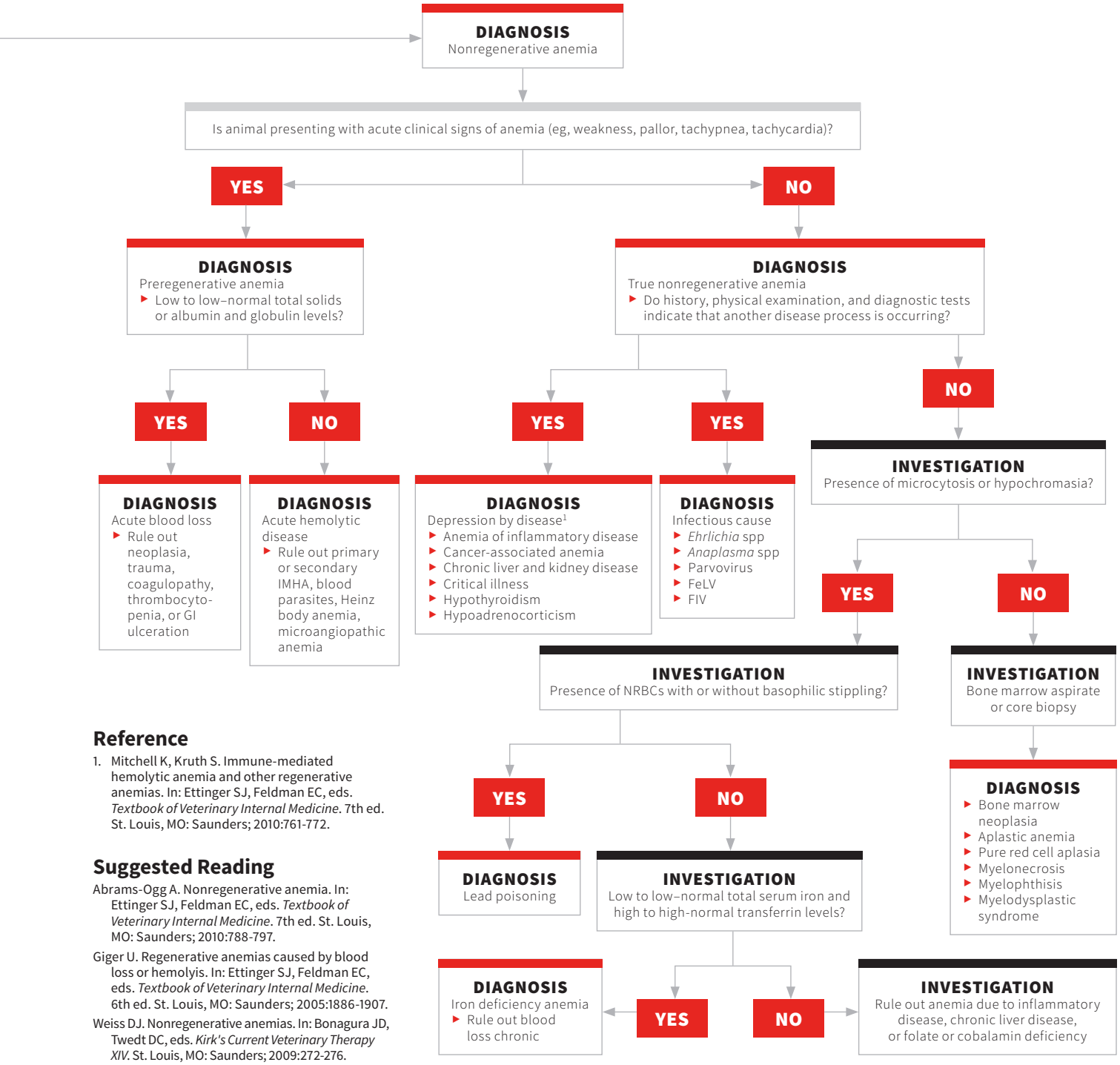
ANEMIA

Caroline M. Kiss, DVM*
Bess J. Pierce, DVM, DABVP, DACVIM†
Virginia-Maryland Regional College of Veterinary Medicine



DIC = disseminated intravascular coagulation
IMHA = immune-mediated hemolytic anemia
MCHC = mean corpuscular hemoglobin concentration
MCV = mean cell volume
NRBC = nucleated red blood cell
RDW = red cell distribution width

*Byline reflects author information on original publication. On publication of this collection, the author's current credentials are Caroline M. Kiss, DVM, DABVP, with no current affiliation.
†Byline reflects author information on original publication. On publication of this collection, the author's current affiliation is Lincoln Memorial University, Harrogate, Tennessee.
‡Microangiopathic anemia occasionally presents as nonregenerative anemia.
§Spherocyte-like RBC morphology may be noted in zinc toxicity; however, Heinz bodies are usually more prevalent.



Reference

1. Mitchell K, Kruth S. Immune-mediated hemolytic anemia and other regenerative anemias. In: Ettinger SJ, Feldman EC, eds. *Textbook of Veterinary Internal Medicine*. 7th ed. St. Louis, MO: Saunders; 2010:761-772.

Suggested Reading

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Weiss DJ. Nonregenerative anemias. In: Bonagura JD, Twedt DC, eds. *Kirk's Current Veterinary Therapy XIV*. St. Louis, MO: Saunders; 2009:272-276.

ANOREXIA IN THE RABBIT

Angela M. Lennox, DVM, DABVP (Avian)
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Indianapolis, Indiana

INVESTIGATION

Medical history
- Signalment
- Presenting clinical signs
- Progression, systems affected
- Date when last normal
- Medical and surgical history
- Response to any previous therapy?

Environmental history
- Complete dietary history, including recent changes
- Potential toxin exposure
- Exposure to other pets or illness in companions
- History of social structure, especially recent changes
- Changes in environment (eg, bedding, toys, visitors, disruptions)

INVESTIGATION

Complete physical examination

Abdominal
- Pain upon palpation
- Presence of masses, excessive fluid, food, and/or gas*

Cardiovascular
- Murmur, arrhythmia, rate

Dermatologic
- Hair loss, generalized or under eyes or chin, suggesting epiphora or ptyalism
- Evidence of ectoparasites
- Pododermatitis

Gastrointestinal
- Willingness to eat
- Amount and character of stool*

Ears/Ear Canal/Eyes
- Palpate base of ears
- Evaluate lens/anterior chamber
- Note evidence of exophthalmia or epiphora

Mentation
- General activity level
- Interest in environment*

Musculoskeletal
- Reluctance to move
- Lameness or discomfort
- Swelling

Neurologic
- Central and/or peripheral nerve deficit

Oral Cavity
- Examine incisors
- Perform cursory otoscopic examination of oral cavity
- Palpate jaw

Respiratory
- Increased rate of effort
- Auscultation
- Evidence of nasal discharge*

Urogenital
- Pain upon palpation of the bladder
- Character of urine

* Repeat these examinations every 3 to 4 hours while anorexia persists in the hospitalized patient

Evidence of disease in a specific body system or suggestion of environmental stress as a potential cause of anorexia?

YES

INVESTIGATION

Diagnostic tests specific to body system:
- Oral cavity: complete evaluation of oral cavity in anesthetized patient
- Abdominal: radiography
- Musculoskeletal: radiography of spine and limbs
- Eyes (exophthalmos): radiography

NO

INVESTIGATION

Initial diagnostic tests:
- CBC with blood smear
- Serum chemistry profile
- Abdominal radiography
- Complete evaluation of oral cavity in anesthetized patient
- Urinalysis

Evidence of specific disease?

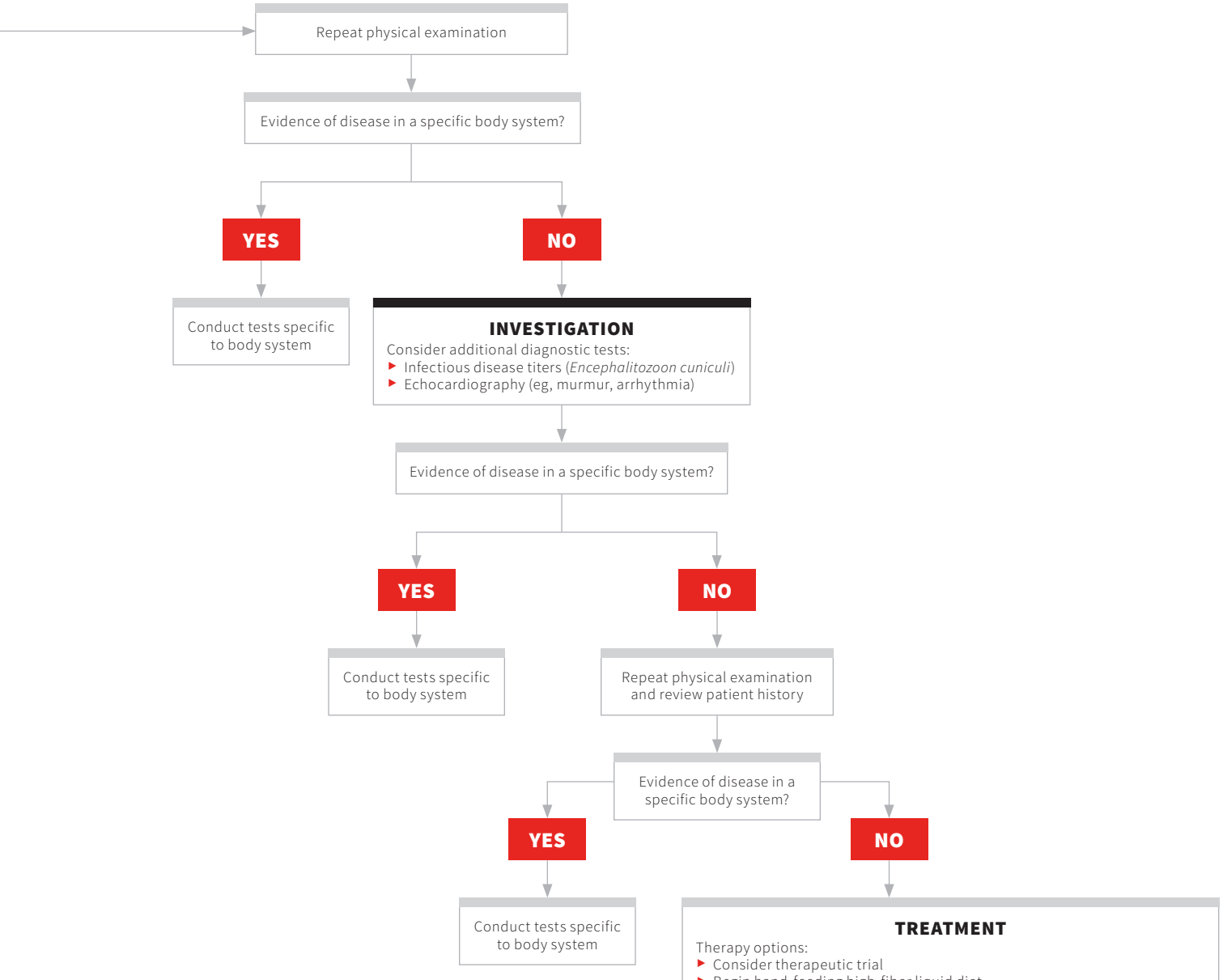
YES

- Initiate treatment for disease

or
- Conduct further diagnostic testing specific to body system

NO

If environmental stress is suspected, send patient home, have owner hand-feed, and allow 48 hours for adjustment

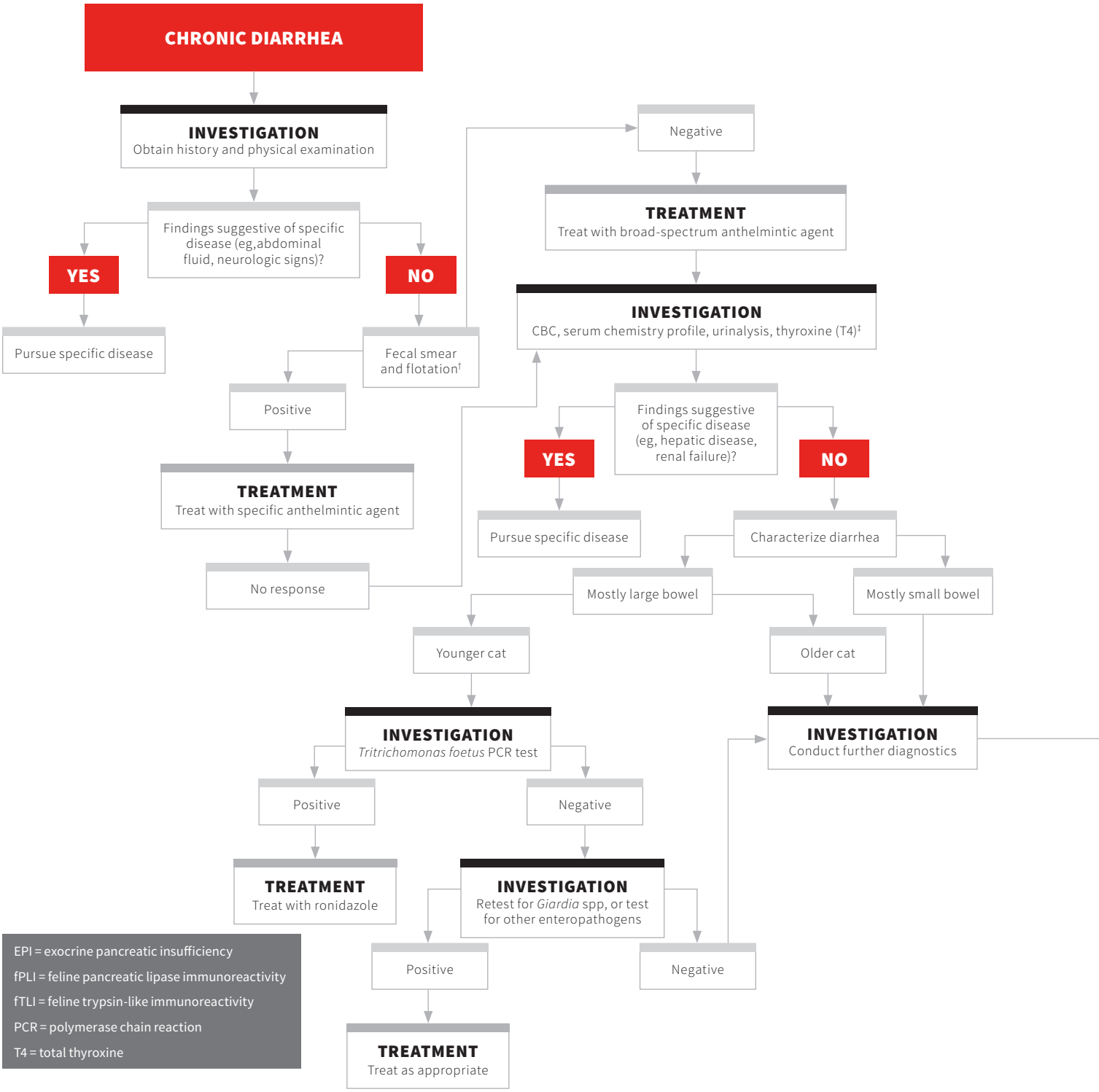


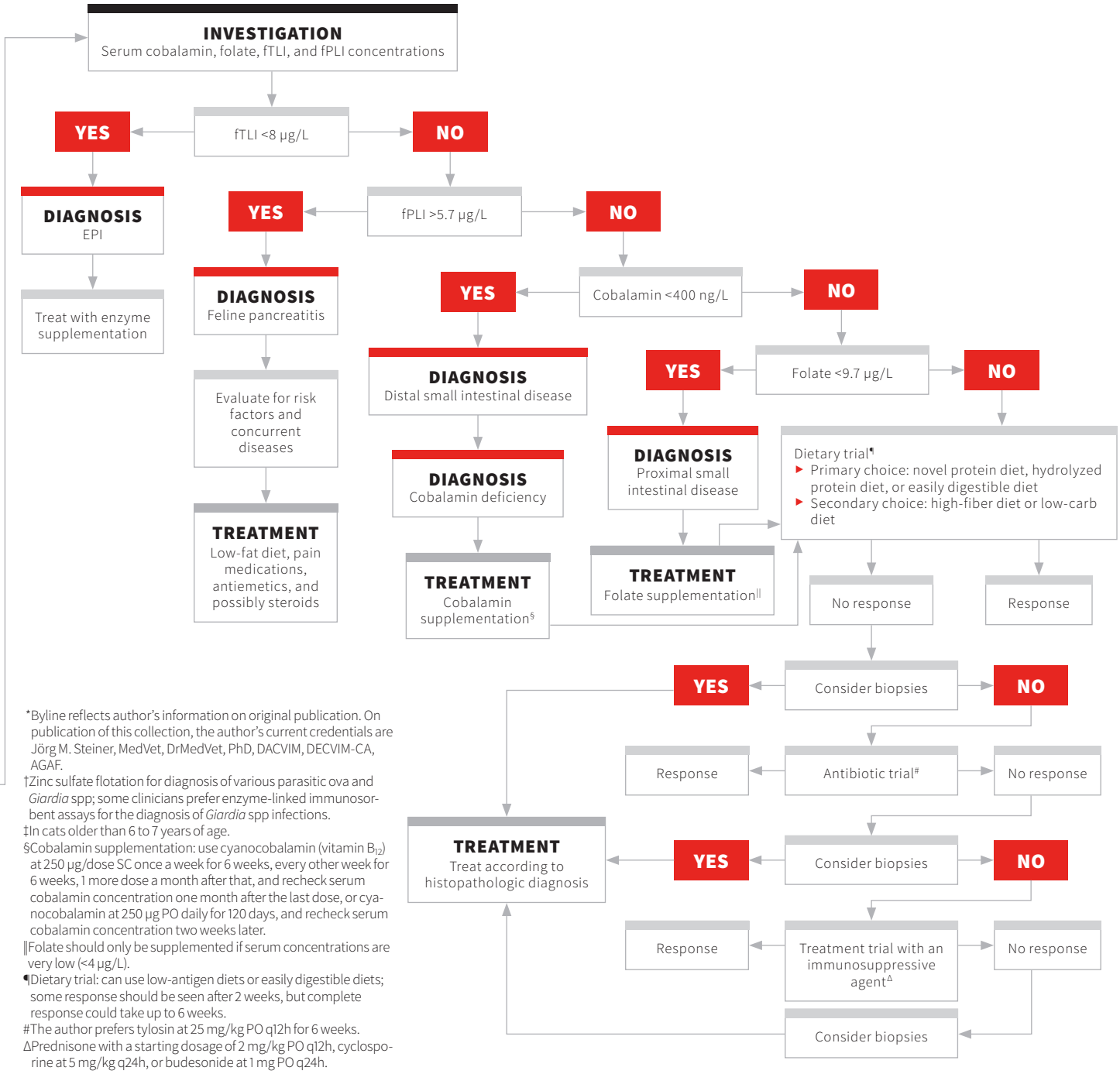
Suggested Reading

Lichtenberger M, Lennox AM. Updates and advanced therapies for gastrointestinal stasis in rabbits. *Vet Clin N Am Exotic Pet Prac*. 2010;13(3):525-543.

CHRONIC DIARRHEA IN CATS

Jörg M.Steiner, MedVet, DrMedVet, PhD, DACVIM, DECVIM-CA*
Texas A&M University





*Byline reflects author's information on original publication. On publication of this collection, the author's current credentials are Jörg M. Steiner, MedVet, DrMedVet, PhD, DACVIM, DECVIM-CA, AGAF.

†Zinc sulfate flotation for diagnosis of various parasitic ova and *Giardia* spp; some clinicians prefer enzyme-linked immunosorbent assays for the diagnosis of *Giardia* spp infections.

‡In cats older than 6 to 7 years of age.

§Cobalamin supplementation: use cyanocobalamin (vitamin B_{12}) at 250 µg/dose SC once a week for 6 weeks, every other week for 6 weeks, 1 more dose a month after that, and recheck serum cobalamin concentration one month after the last dose, or cyanocobalamin at 250 µg PO daily for 120 days, and recheck serum cobalamin concentration two weeks later.

‖Folate should only be supplemented if serum concentrations are very low (<4 µg/L).

¶Dietary trial: can use low-antigen diets or easily digestible diets; some response should be seen after 2 weeks, but complete response could take up to 6 weeks.

#The author prefers tylosin at 25 mg/kg PO q12h for 6 weeks.

ΔPrednisone with a starting dosage of 2 mg/kg PO q12h, cyclosporine at 5 mg/kg q24h, or budesonide at 1 mg PO q24h.

DIAGNOSING CONSTIPATION, OBSTIPATION, & MEGACOLON IN CATS

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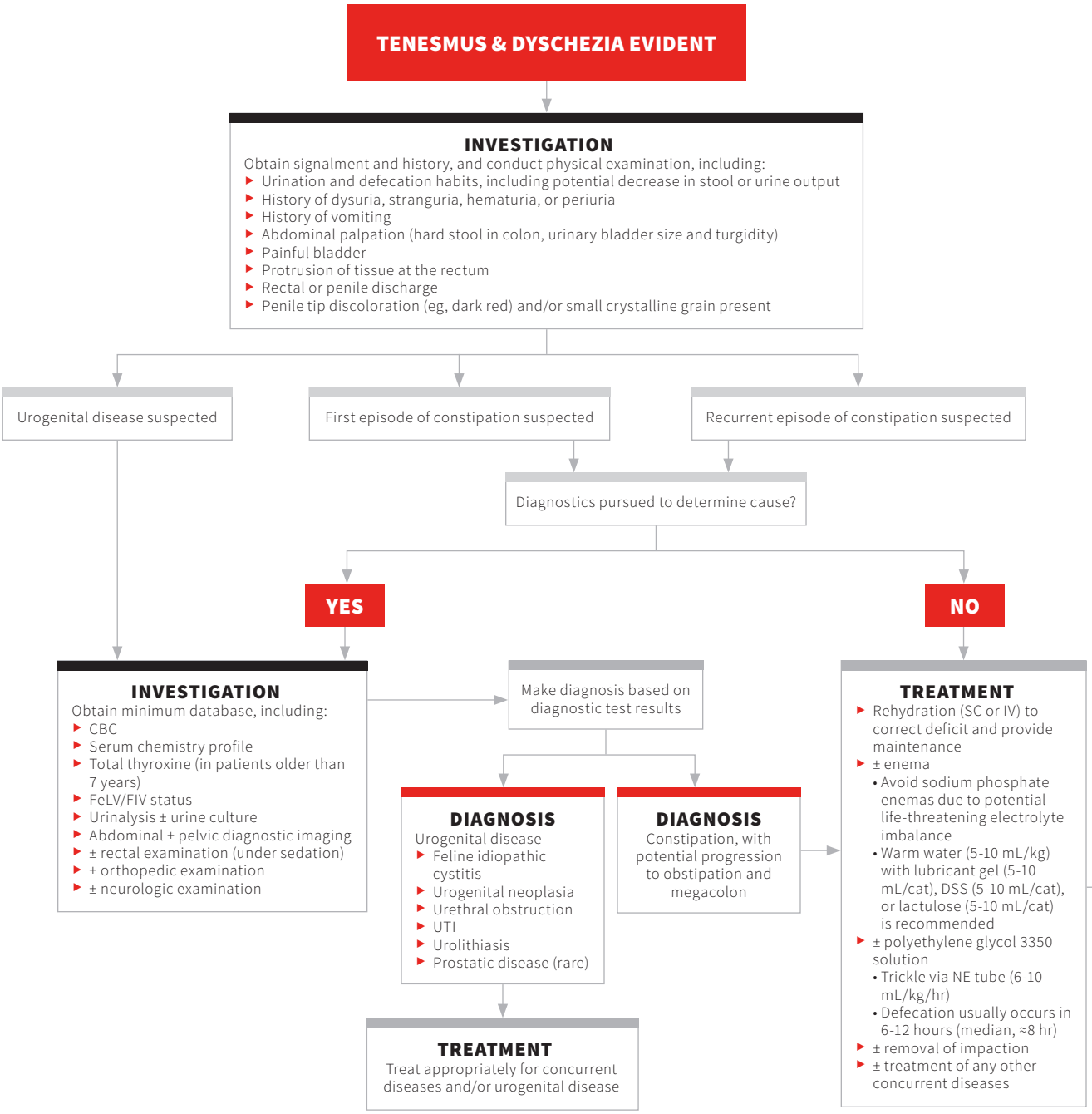
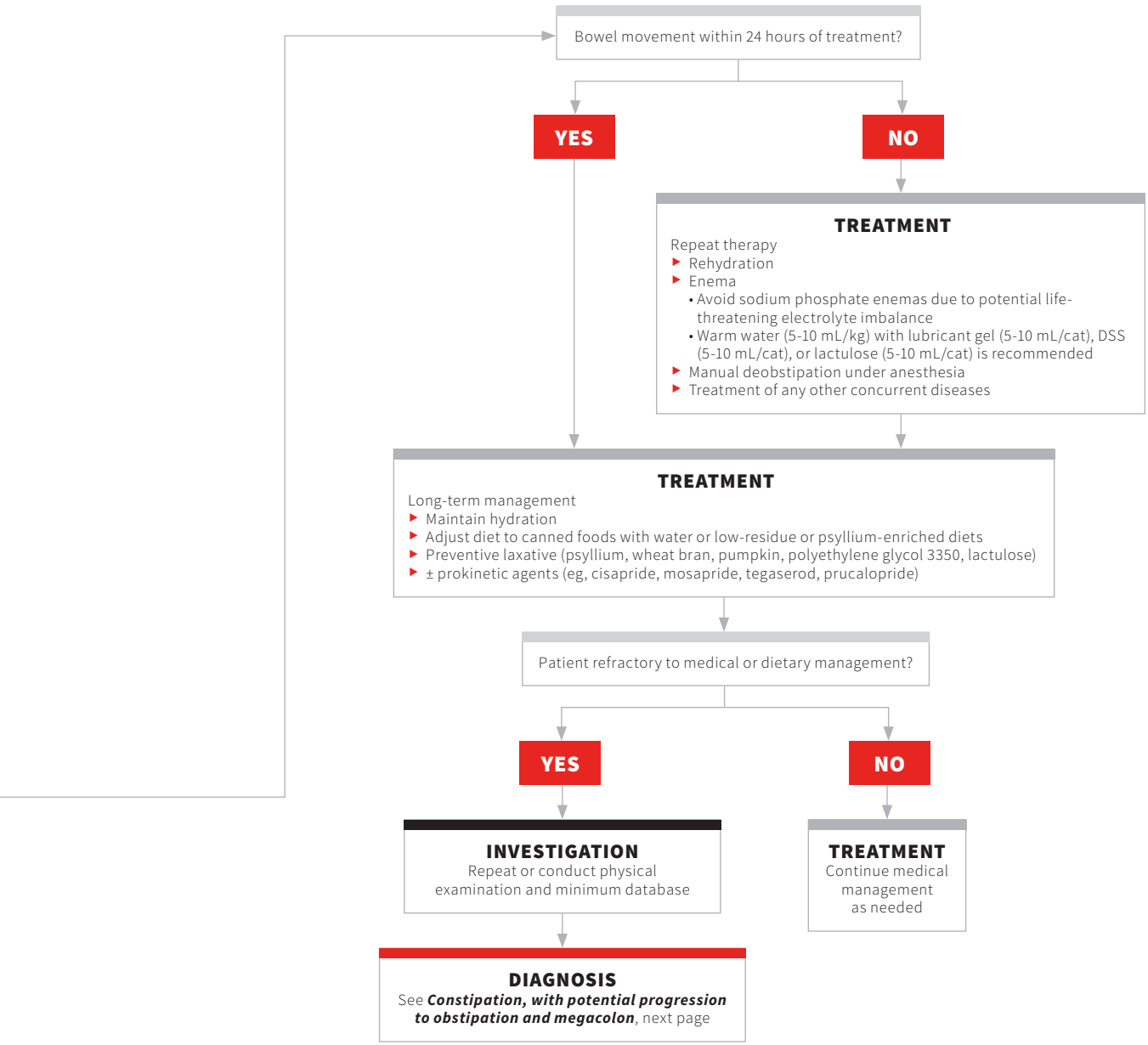
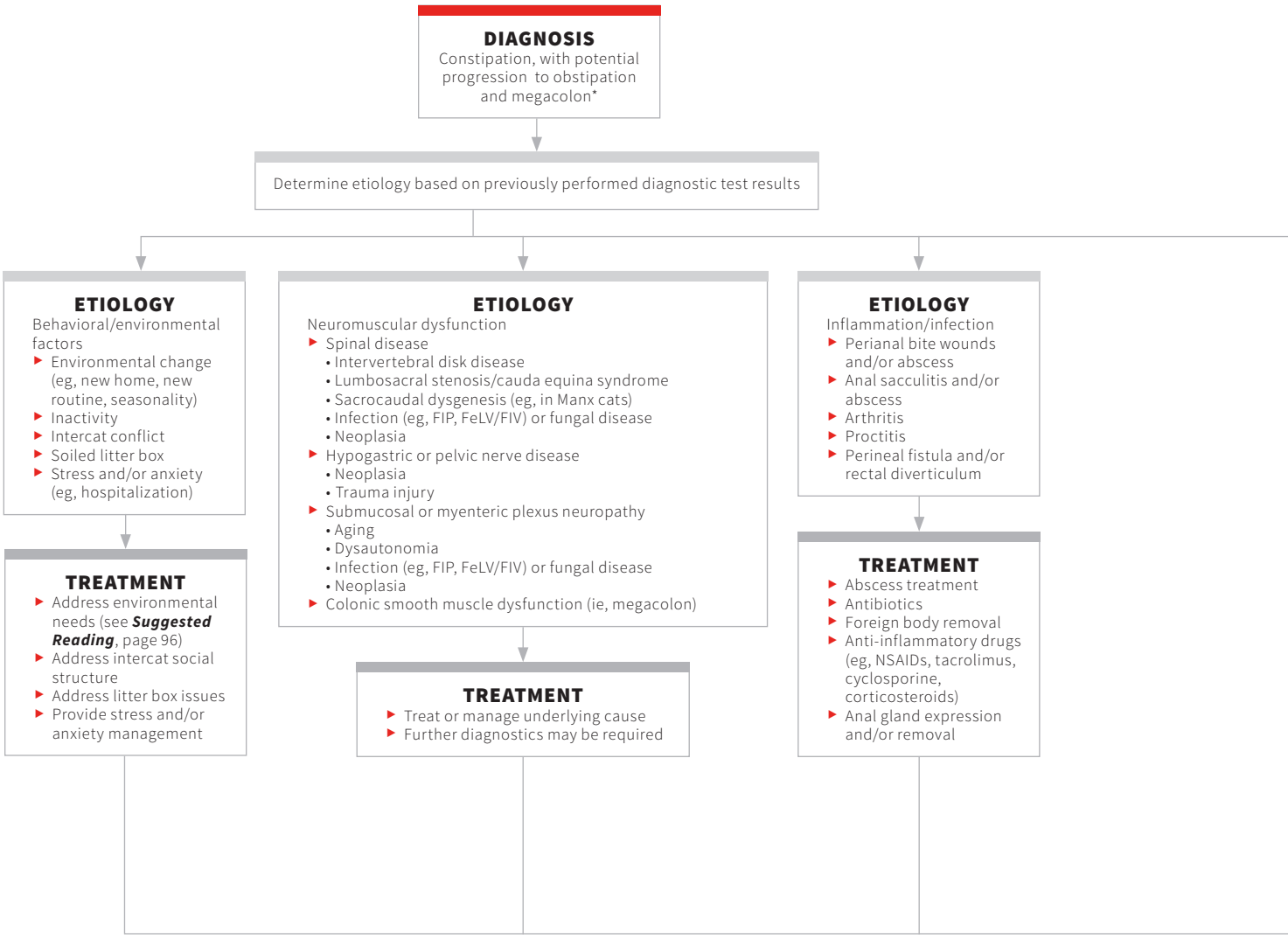


DSS = dioctyl sodium sulfosuccinate

NE = nasoesophageal

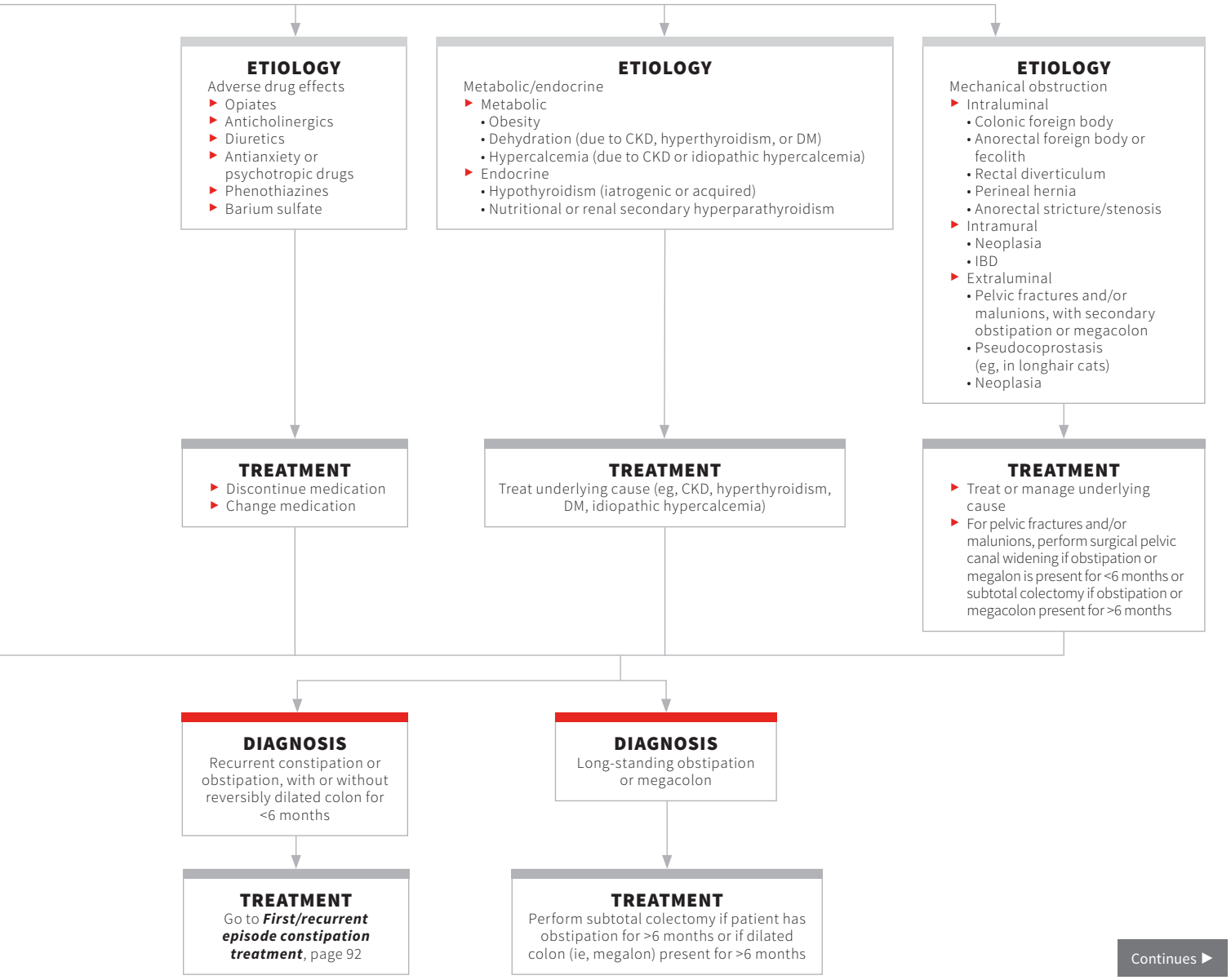

Bowel movement within 24 hours of treatment?
YES
NO
TREATMENT
Repeat therapy
Rehydration
Enema
• Avoid sodium phosphate enemas due to potential life-threatening electrolyte imbalance
• Warm water (5-10 mL/kg) with lubricant gel (5-10 mL/cat), DSS (5-10 mL/cat), or lactulose (5-10 mL/cat) is recommended
Manual deobstipation under anesthesia
Treatment of any other concurrent diseases
TREATMENT
Long-term management
Maintain hydration
Adjust diet to canned foods with water or low-residue or psyllium-enriched diets
Preventive laxative (psyllium, wheat bran, pumpkin, polyethylene glycol 3350, lactulose)
± prokinetic agents (eg, cisapride, mosapride, tegaserod, prucalopride)
Patient refractory to medical or dietary management?
YES
NO
INVESTIGATION
Repeat or conduct physical examination and minimum database
TREATMENT
Continue medical management as needed
DIAGNOSIS
See Constipation, with potential progression to obstipation and megacolon, next page

Continues ▶



*Megacolon is suggestive of neuromuscular dysfunction.

CKD = chronic kidney disease
DM = diabetes mellitus
IBD = inflammatory bowel disease


ETIOLOGY
Adverse drug effects
Opiates
Anticholinergics
Diuretics
Antianxiety or psychotropic drugs
Phenothiazines
Barium sulfate
ETIOLOGY
Metabolic/endocrine
Metabolic
• Obesity
• Dehydration (due to CKD, hyperthyroidism, or DM)
• Hypercalcemia (due to CKD or idiopathic hypercalcemia)
Endocrine
• Hypothyroidism (iatrogenic or acquired)
• Nutritional or renal secondary hyperparathyroidism
ETIOLOGY
Mechanical obstruction
Intraluminal
• Colonic foreign body
• Anorectal foreign body or fecolith
• Rectal diverticulum
• Perineal hernia
• Anorectal stricture/stenosis
Intramural
• Neoplasia
• IBD
Extraluminal
• Pelvic fractures and/or malunions, with secondary obstipation or megacolon
• Pseudocoprostasis (eg, in longhair cats)
• Neoplasia
TREATMENT
Discontinue medication
Change medication
TREATMENT
Treat underlying cause (eg, CKD, hyperthyroidism, DM, idiopathic hypercalcemia)
TREATMENT
Treat or manage underlying cause
For pelvic fractures and/or malunions, perform surgical pelvic canal widening if obstipation or megalon is present for <6 months or subtotal colectomy if obstipation or megacolon present for >6 months
DIAGNOSIS
Recurrent constipation or obstipation, with or without reversibly dilated colon for <6 months
DIAGNOSIS
Long-standing obstipation or megacolon
TREATMENT
Go to First/recurrent episode constipation treatment, page 92
TREATMENT
Perform subtotal colectomy if patient has obstipation for >6 months or if dilated colon (ie, megalon) present for >6 months

Continues ▶

FELCOM Cheapbooks13

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Suggested Reading

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COUGH IN CATS

Janice Dye, DVM, MS, PhD, DACVIM (SAIM)
Raleigh, North Carolina

SNEEZING OR NASAL DISCHARGE? NOISY (STERTOROUS) BREATHING? RETCHING OR GAGGING? ALTERED VOCALIZATION?

YES

Laryngopharyngeal cough

INVESTIGATION
Perform MDB, cranial and oral examination, skull or neck radiography or CT, rhinoscopy or laryngoscopy

DIFFERENTIALS
- Rhinitis/sinusitis
  - Infections
  - Inflammatory (idiopathic)
  - Atopy-related*
  - Neoplasia
- Laryngitis
  - Infectious
  - Inflammatory
  - Postnasal drip*
  - Injury (prior intubation)
  - Irritant exposure (eg, noxious fumes/gases, particles or dust, smoke inhalation, ETS, litter)
- Laryngeal or nasopharyngeal mass, polyp, stenosis, or foreign material (eg, hair, food, medication)
- Comorbid conditions
  - Megaesophagus
  - Laryngeal paralysis

NO

INVESTIGATION
Perform thoracic examination and auscultation, tracheal palpation, thoracic radiography, MDB

Adventitial lung sounds

Tracheobronchial cough

Radiographic evidence of bronchial, mixed bronchointerstitial, or bronchoalveolar pattern

INVESTIGATION
Bronchoscopy and/or airway sampling (cytology/culture), heartworm antigen and antibody

DIFFERENTIALS
- Tracheitis/bronchitis (rule-outs similar to laryngitis)
  - Infectious (viral)
  - Inflammatory (eg, feline asthma)
  - Atopy/allergy-related
  - HARD
  - Idiopathic
  - Irritant exposure
  - Foreign material
  - Postnasal drip*
- Airway obstruction
- Airway mucus, debris
- Airway fluid or hemorrhage
- Airway collapse
- Airway compression
- Comorbid conditions
  - Megaesophagus
  - Laryngeal paralysis

Normal or reduced bronchovesicular sounds

Nonspecific cough

INVESTIGATION
Serology, fecal examinations, BAL or fine-needle lung aspiration (cytology/culture), thoracic CT

DIFFERENTIALS
- Pneumonia/pneumonitis
  - Viral (eg, FHV-1, FCV)
  - Parasitic (eg, *Dirofilaria* spp, other)
  - Mycotic
  - Bacterial: primary (eg, *Bordetella* spp) or secondary (eg, dental disease)
  - Protozoal (*Toxoplasma* spp)
  - Atopy-related*
- Pleuritis or pleural space disease (eg, FIP, pleural effusions)
- Interstitial lung disease (eg, IPF, other subtypes)
- Neoplasia
- Pulmonary edema—if severe, CHF, fluid overload
- Extrapulmonary chronic inflammatory conditions (rhinitis, otitis, GERD)

AUTHOR INSIGHT

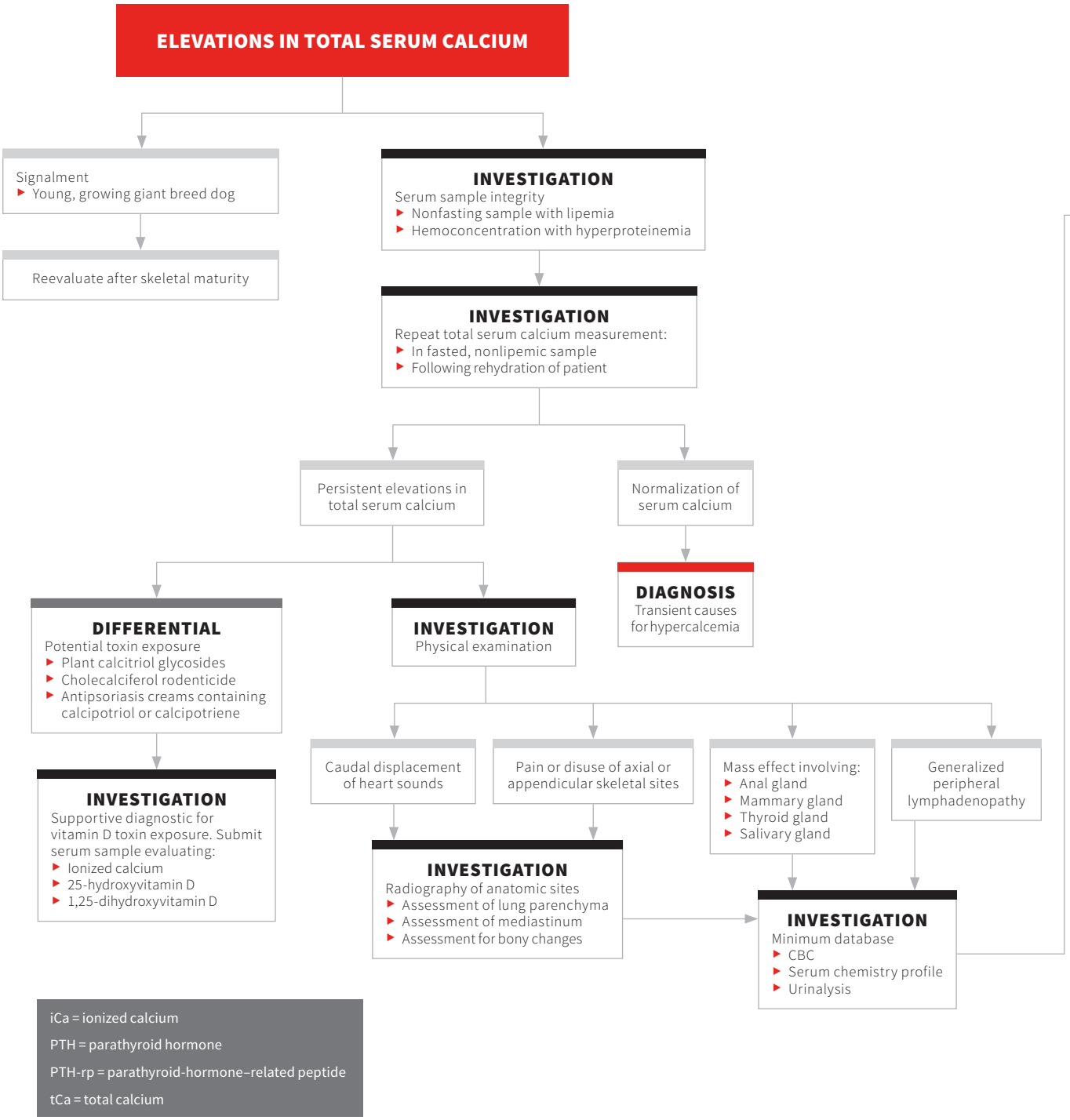
- Cats have a well-developed cough reflex. Coughing dispels mucus and inhaled substances from the lower airways.
- Coughing is not pathognomonic for a specific diagnosis. It often occurs intermittently, with or without wheezing or respiratory distress. Alternatively, it can occur with sneezing/nasal discharge, noisy or stertorous breathing, retching/gagging, or even vomiting.
- Chronic cough is frequently related to development of airway inflammation, airway obstruction, and mucous hypersecretory states.
- In cats, cough is commonly observed with bronchial or asthma-like disease; it is variable in parenchymal or pleural space disorders; and is uncommon in cardiac disease alone. However, concurrent pulmonary and cardiac disease may occur.
- Posttussive vomiting may be a result of air-induced distension of the stomach.

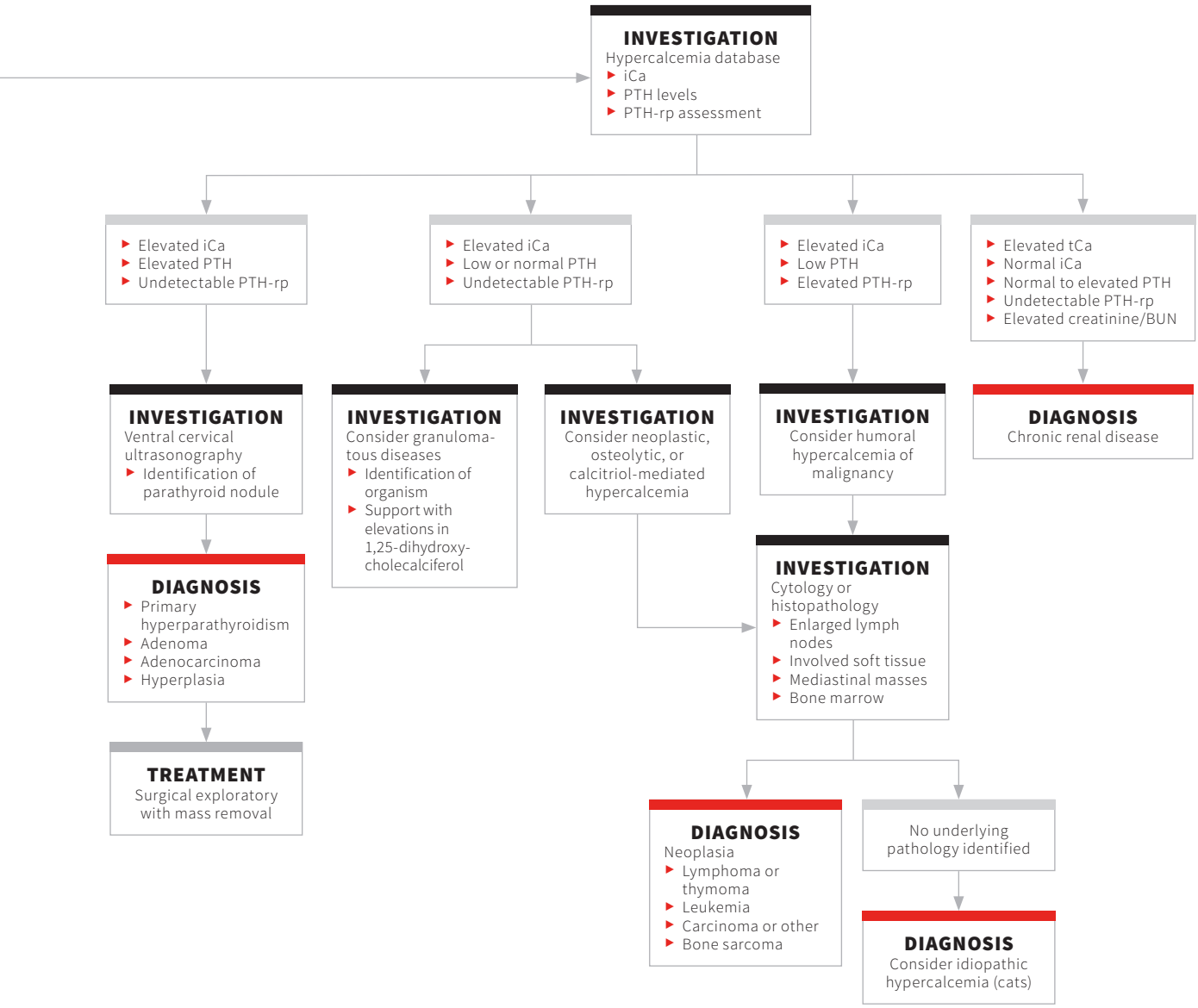
BAL = bronchoalveolar lavage
CHF = congestive heart failure
ETS = environmental tobacco smoke
FCV = feline coronavirus
FHV-1 = feline herpervirus type 1
GERD = gastrointestinal reflux disease
HARD = heartworm-associated respiratory disease
IPF = idiopathic pulmonary fibrosis
MDB = minimum database

*analogous to human condition

ELEVATIONS IN TOTAL SERUM CALCIUM

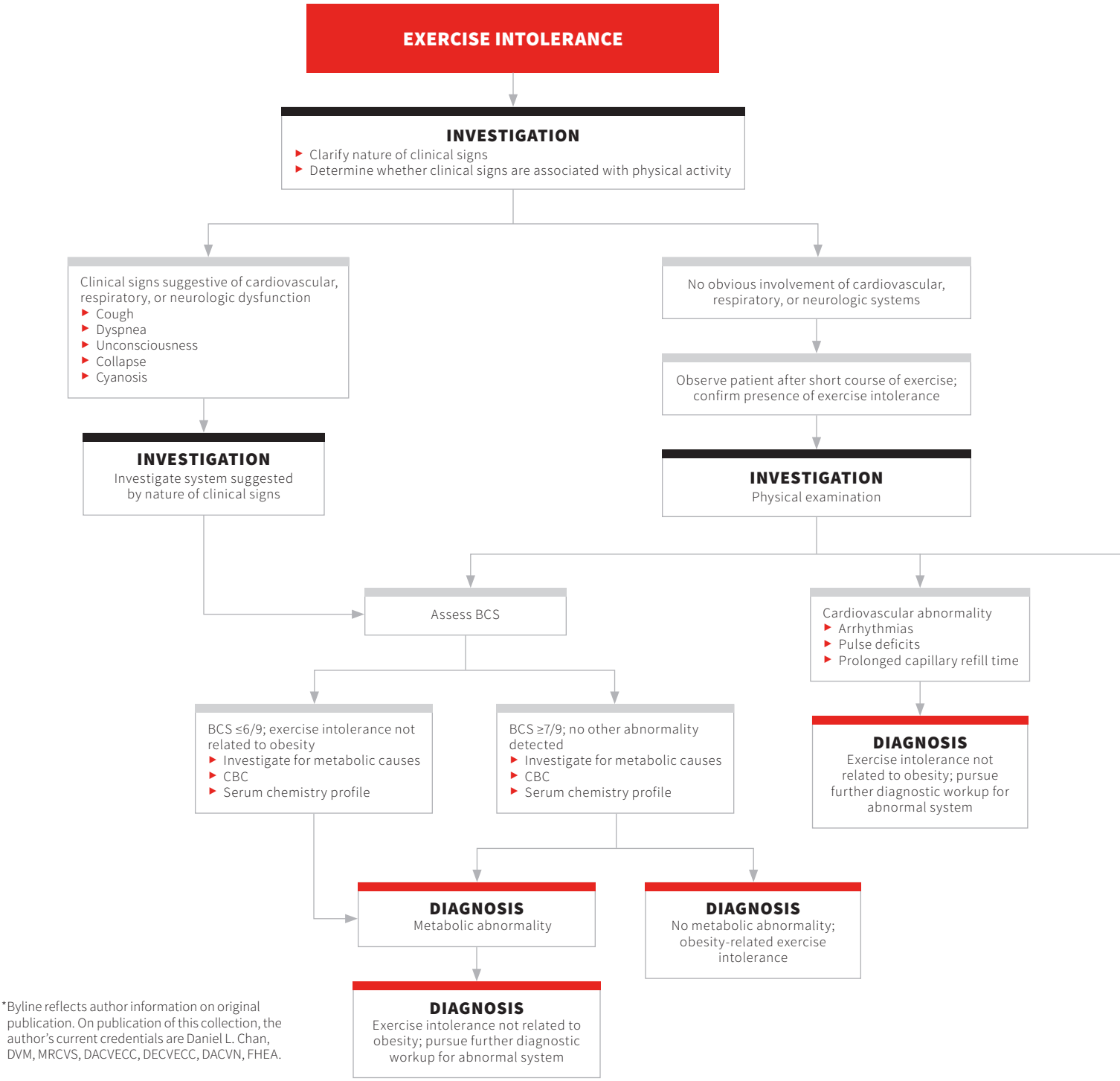
Timothy M. Fan, DVM, PhD, DACVIM (SAIM, Oncology)
University of Illinois



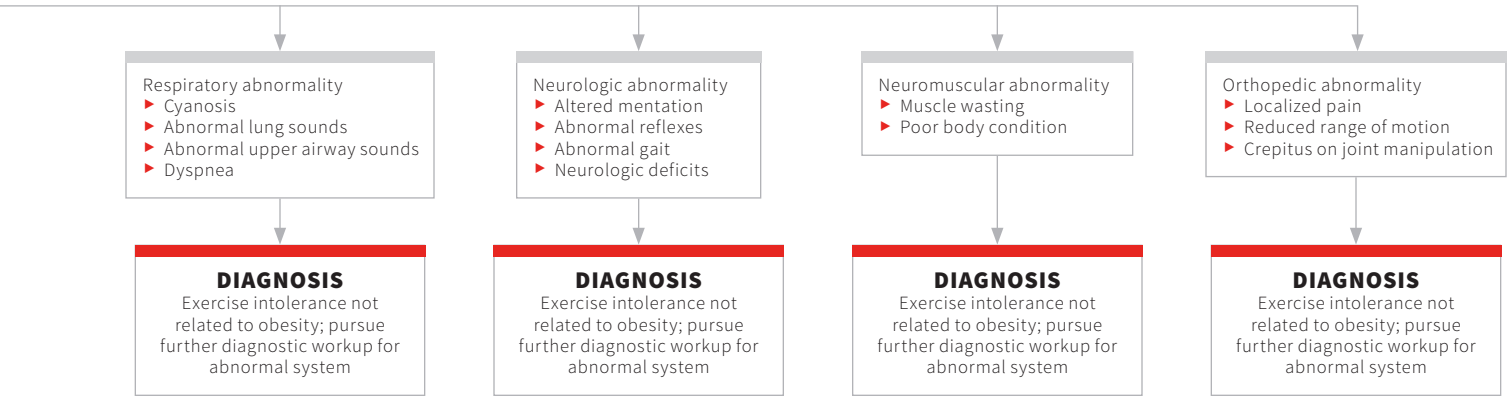

INVESTIGATION
Hypercalcemia database
iCa
PTH levels
PTH-rp assessment
Elevated iCa
Elevated PTH
Undetectable PTH-rp
Elevated iCa
Low or normal PTH
Undetectable PTH-rp
Elevated iCa
Low PTH
Elevated PTH-rp
Elevated tCa
Normal iCa
Normal to elevated PTH
Undetectable PTH-rp
Elevated creatinine/BUN
INVESTIGATION
Ventral cervical ultrasonography
Identification of parathyroid nodule
INVESTIGATION
Consider granulomatous diseases
Identification of organism
Support with elevations in 1,25-dihydroxycholecalciferol
INVESTIGATION
Consider neoplastic, osteolytic, or calcitriol-mediated hypercalcemia
INVESTIGATION
Consider humoral hypercalcemia of malignancy
DIAGNOSIS
Chronic renal disease
DIAGNOSIS
Primary hyperparathyroidism
Adenoma
Adenocarcinoma
Hyperplasia
INVESTIGATION
Cytology or histopathology
Enlarged lymph nodes
Involved soft tissue
Mediastinal masses
Bone marrow
TREATMENT
Surgical exploratory with mass removal
DIAGNOSIS
Neoplasia
Lymphoma or thymoma
Leukemia
Carcinoma or other
Bone sarcoma
No underlying pathology identified
DIAGNOSIS
Consider idiopathic hypercalcemia (cats)

EXERCISE INTOLERANCE

Daniel L. Chan, DVM, MRCVS, DACVECC, DACVN*
Royal Veterinary College
University of London



*Byline reflects author information on original publication. On publication of this collection, the author's current credentials are Daniel L. Chan, DVM, MRCVS, DACVECC, DECVECC, DACVN, FHEA.


Respiratory abnormality
Cyanosis
Abnormal lung sounds
Abnormal upper airway sounds
Dyspnea
DIAGNOSIS
Exercise intolerance not related to obesity; pursue further diagnostic workup for abnormal system
Neurologic abnormality
Altered mentation
Abnormal reflexes
Abnormal gait
Neurologic deficits
DIAGNOSIS
Exercise intolerance not related to obesity; pursue further diagnostic workup for abnormal system
Neuromuscular abnormality
Muscle wasting
Poor body condition
DIAGNOSIS
Exercise intolerance not related to obesity; pursue further diagnostic workup for abnormal system
Orthopedic abnormality
Localized pain
Reduced range of motion
Crepitus on joint manipulation
DIAGNOSIS
Exercise intolerance not related to obesity; pursue further diagnostic workup for abnormal system

FELINE WEIGHT LOSS

Margie Scherk, DVM, DABVP (Feline Practice)
catsINK
Vancouver, British Columbia

WEIGHT LOSS IN CATS

INVESTIGATION

Obtain complete history:

- Duration of weight loss
- Other clinical signs (eg, vomiting or regurgitation, diarrhea)
- Time when patient last appeared or acted normal
- Changes in diet or eating behavior
- Changes in environment (eg, change of humans or animals present in household, routines, placement of food bowls)
- Dropping of food or difficulty in prehending or chewing
- Evidence of orofacial pain
- Travel history
- Changes in water intake, urine volume or frequency
- Changes in attitude, energy, activity, or mobility
- Changes in litter box use (eg, volume, frequency, character, color of feces)

INVESTIGATION

Perform physical examination:

- Body weight
- % weight change* and BCS
- Muscle condition (eg, normal or wasted), see ***Suggested Reading***
- Blood pressure evaluation
- Coat condition
- Funduscopic examination
- Oral examination
- Abdominal palpation
- Pulse quality
- Thyroid palpation†
- Thoracic auscultation
- Lymph node palpation

Good appetite

Unable to eat

Poor appetite

DIFFERENTIALS

- Inadequate intake
  - Poor quality or insufficient quantity of food
- Maldigestion or malabsorption
  - IBD
  - Lymphoma
  - EPI
  - Systemic fungal infection (if regionally likely or if travel history indicates risk)
  - Age-related sarcopenia
- Excessive nutrient loss
  - Diabetes mellitus
  - Hyperthyroidism
  - Parasitism
  - Cachexia (eg, cardiac or neoplastic)
  - Protein-losing nephropathy
  - Protein-losing enteropathy
- Overutilization
  - Pregnancy or lactation
  - Strenuous workload
  - Temperature extremes
- Inability to access food (stress, environment, decreased mobility)

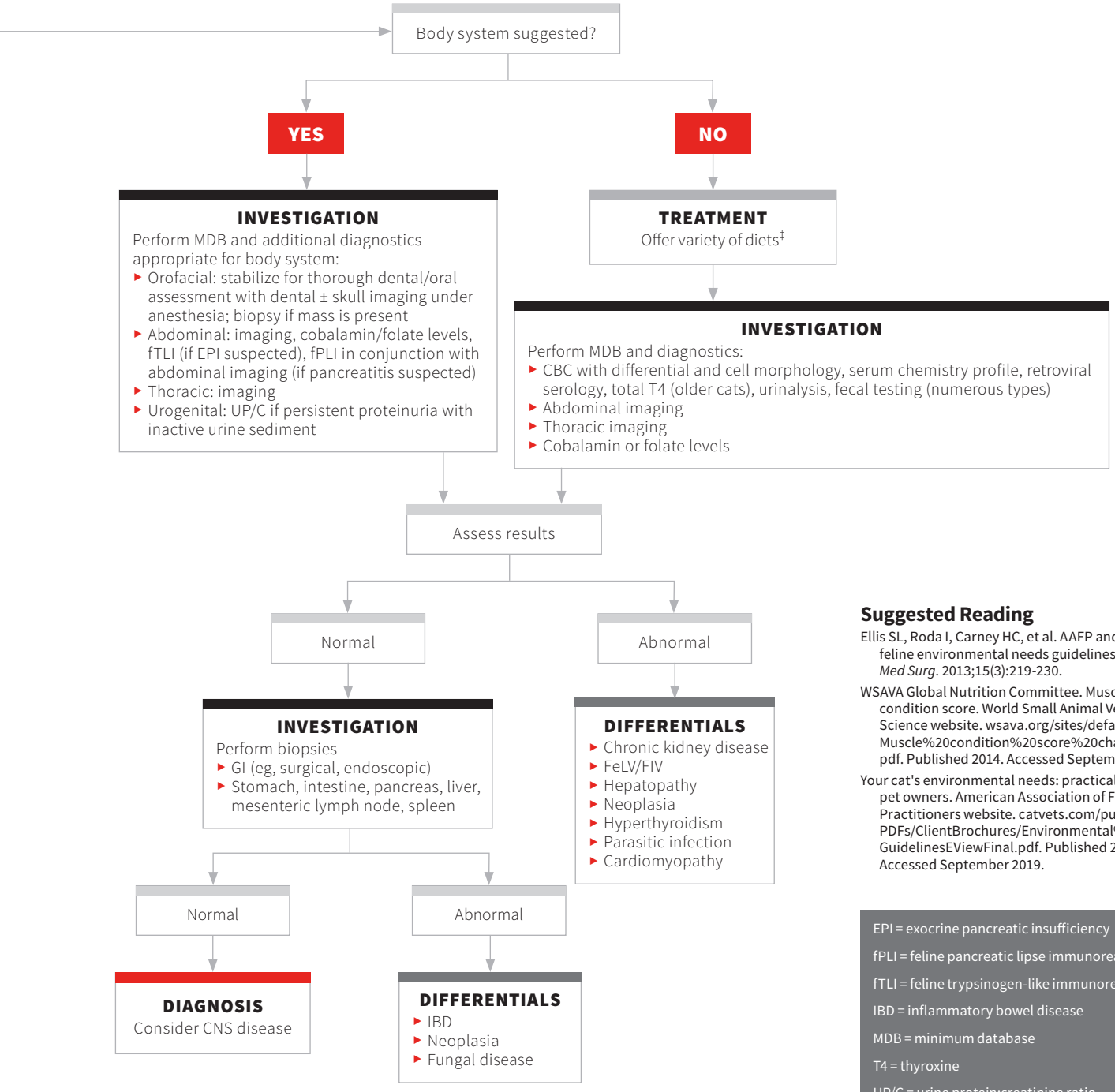
DIFFERENTIALS

- Oral neoplasia
- Orofacial or dental fracture
- Dental disease
- Dental pain
- Neuropathy
- Retrobulbar neoplasia or abscess
- Oral, pharyngeal, esophageal disease

DIFFERENTIALS

- Drug therapy (eg, antibiotics, chemotherapeutic agents)
- FeLV/FIV
- Chronic kidney disease
- Acute kidney injury
- Cholangitis or hepatitis, hepatic lipidosis
- Pancreatitis
- Biliary tree disease
- Neoplasia
- IBD
- Systemic fungal infection
- CNS disease
- Bored with diet‡
- Distress (eg, environmental, social)§
- Nasal disease
- Foreign body
- Abscess
- Fever
- Pain (arthritis, other)

*Percentage of weight change = (previous weight – current weight)/previous weight × 100
†Presence or absence of mass does not rule in or out hyperthyroidism
‡Offer a variety of diets; recheck weight in 10-14 days: if no weight gain, perform MDB; if weight gain, continue with new diet and recheck weight in 14 days
§If history reveals stress, enrich environment with opportunities to hide and perch, move feeding station to quiet and safe place, add feeding stations (easy access). See ***Suggested Reading*** for environmental needs resources.



Suggested Reading

Ellis SL, Roda I, Carney HC, et al. AAFP and ISFM feline environmental needs guidelines. *J Feline Med Surg*. 2013;15(3):219-230.

WSAVA Global Nutrition Committee. Muscle condition score. World Small Animal Veterinary Science website. wsava.org/sites/default/files/Muscle%20condition%20score%20chart-Cats.pdf. Published 2014. Accessed September 2019.

Your cat's environmental needs: practical tips for pet owners. American Association of Feline Practitioners website. catvets.com/public/PDFs/ClientBrochures/Environmental%20GuidelinesEViewFinal.pdf. Published 2013. Accessed September 2019.

EPI = exocrine pancreatic insufficiency

fPLI = feline pancreatic lipse immunoreactivity

fTLI = feline trypsinogen-like immunoreactivity

IBD = inflammatory bowel disease

MDB = minimum database

T4 = thyroxine

UP/C = urine protein:creatinine ratio

HAIRBALLS

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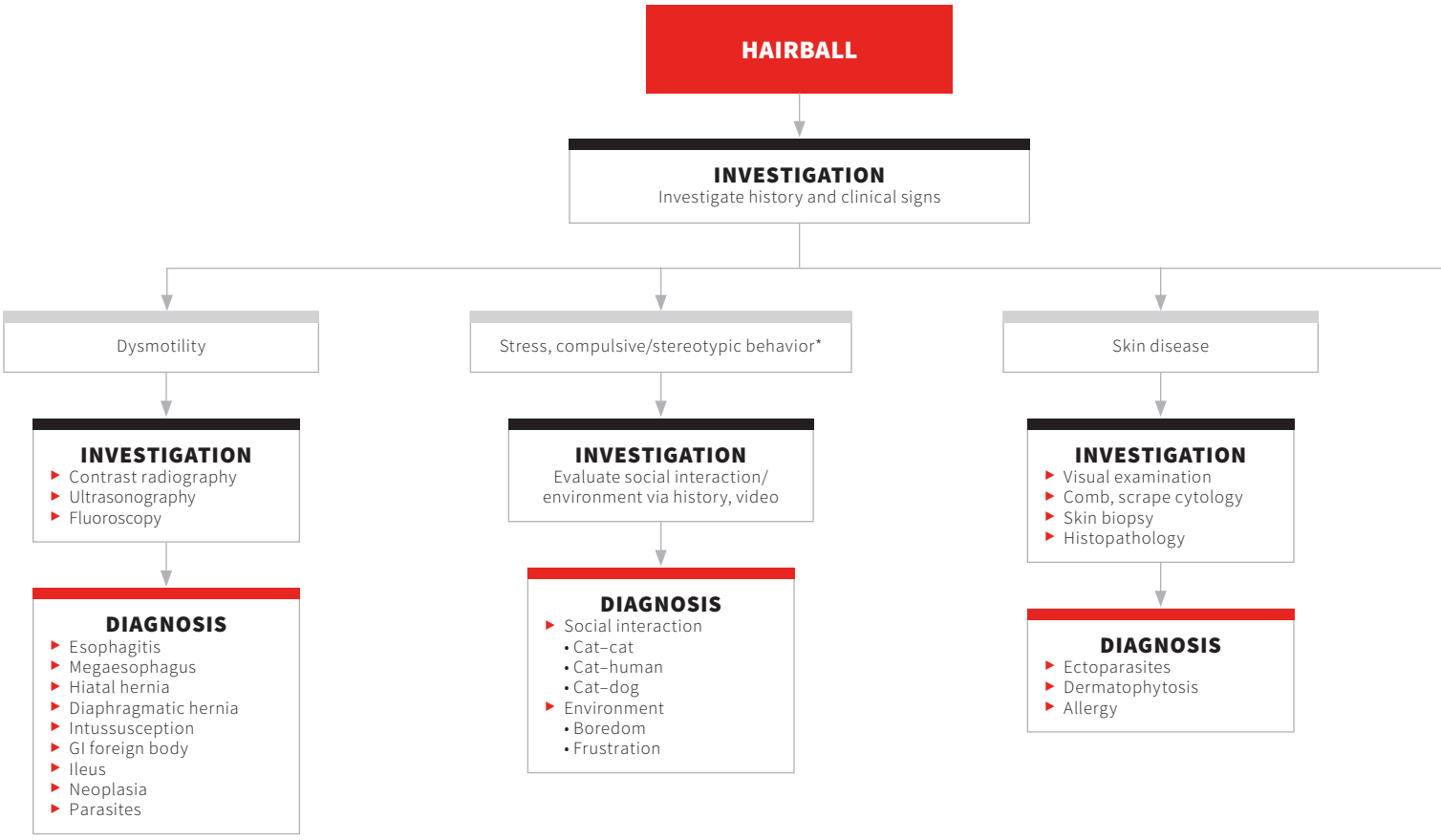
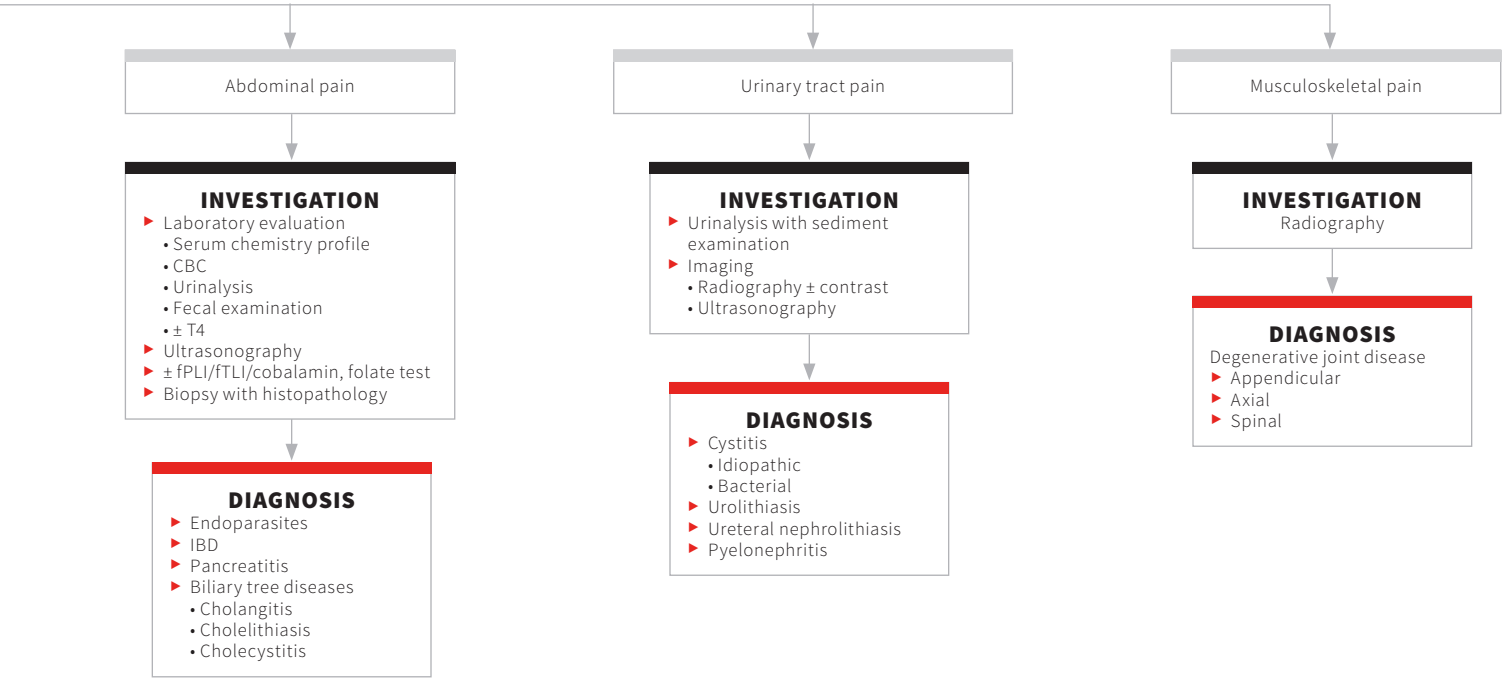


AUTHOR INSIGHT

- Clients may use the term *hairballs* (ie, tubular wads of ingested hair) to describe loose strands of hair in vomitus or regurgitated/vomited food; it is important to clarify what they are defining before determining a diagnostic and therapeutic approach.
- The extreme hairball is a trichobezoar—a hard concretion of hair lodged in the stomach that is too large to vomit or pass through the pylorus and intestines.
- Cats normally ingest small amounts of hair that pass in feces; increased hair ingestion (eg, skin/coat problems) or abnormal passage of hair (ie, changes in GI motility) may result in hairballs.
- Hairballs signify disease and, although common, are not normal.

*Stress can result in overgrooming, especially when a cat has compulsive tendencies and is unable to stop the behavior once initiated.[1]



Reference

1. Mege C. Skin conditions associated with behavioural disorders. In: Guaguere E, Prelaud P, eds. *A Practical Guide to Feline Dermatology*. Iselin, NJ: Merial; 1999:17.1-17.11.

fPLI = feline pancreatic lipase immunoreactivity

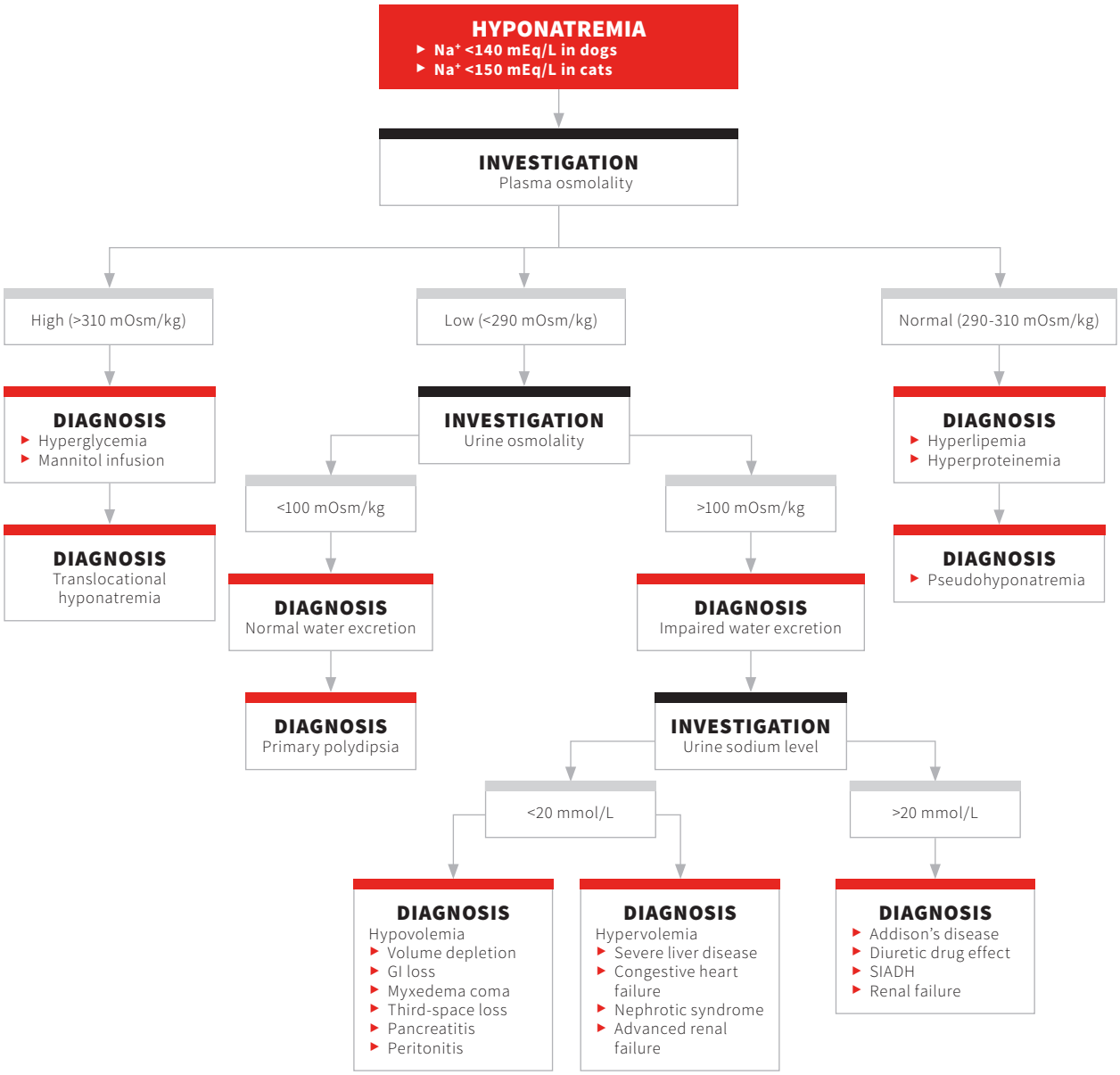
fTLI = feline trypsin-like immunoreactivity

IBD = inflammatory bowel disease

T4 = thyroxine

HYPONATREMIA

Cristina Perez Vera, DVM*
Sally Bissett, BVSc, MVSc, DACVIM†
North Carolina State University



Na+ = sodium

SIADH = syndrome of inappropriate secretion of antidiuretic hormone

*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Cristina Perez Vera, DVM, PhD, DACVIM, DECVIM, at Tierklinik Aarau West, Oberentfelden, Switzerland.
†Byline reflects author information on original publication. On publication of this collection, Sally Bissett's current affiliation is Regional Veterinary Emergency and Specialty Center, Turnersville, New Jersey.

ICTERUS IN DOGS

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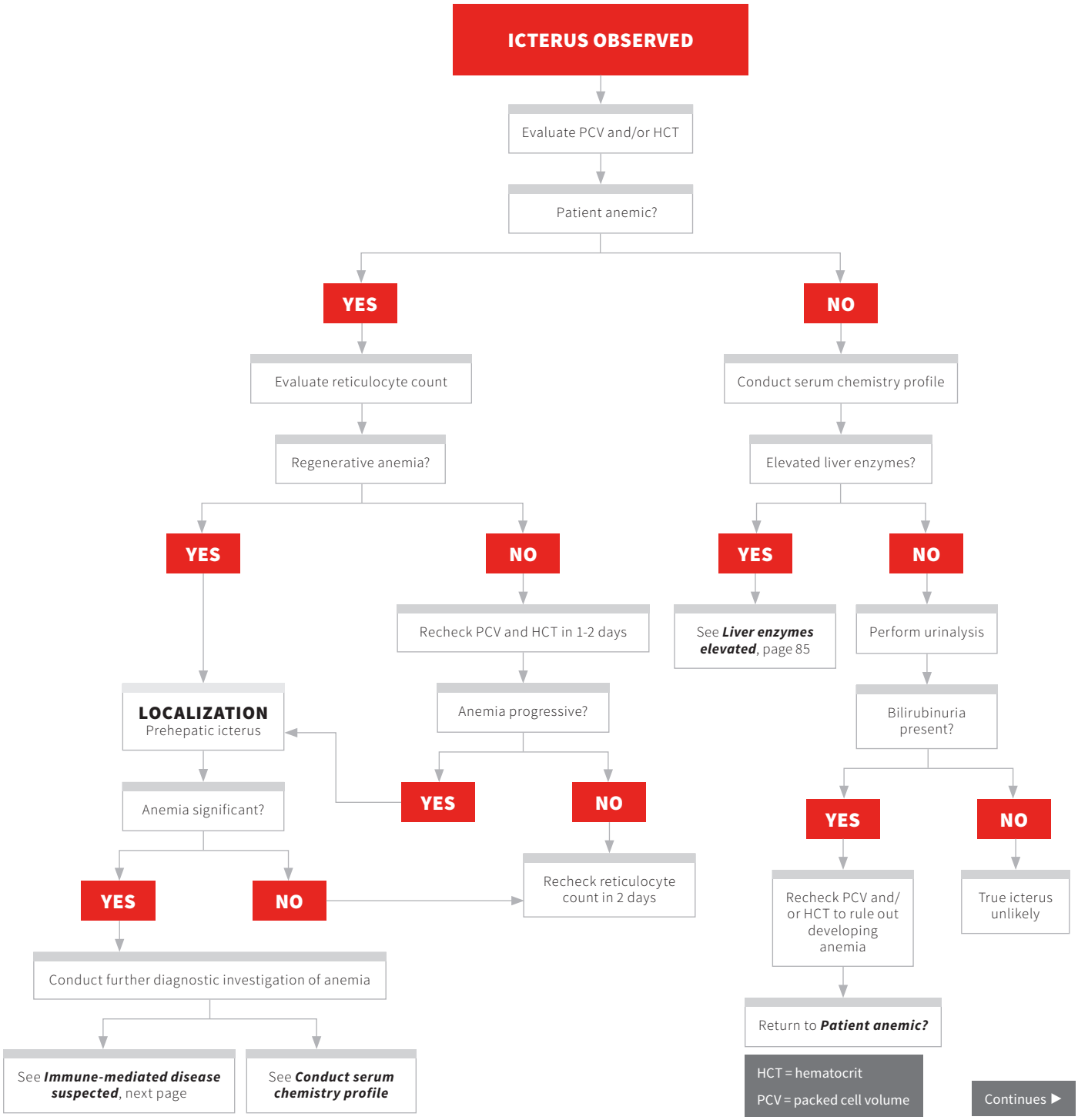
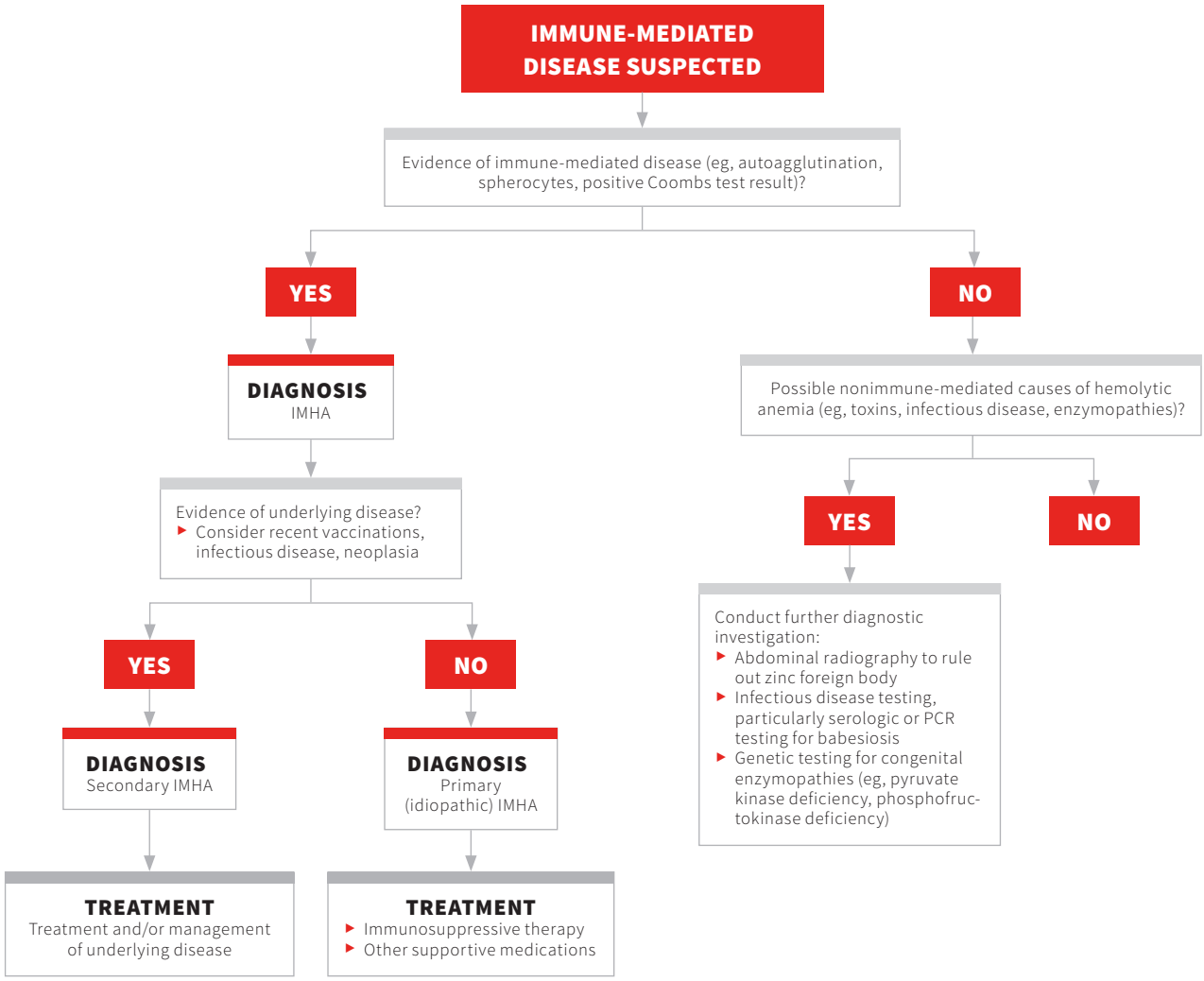
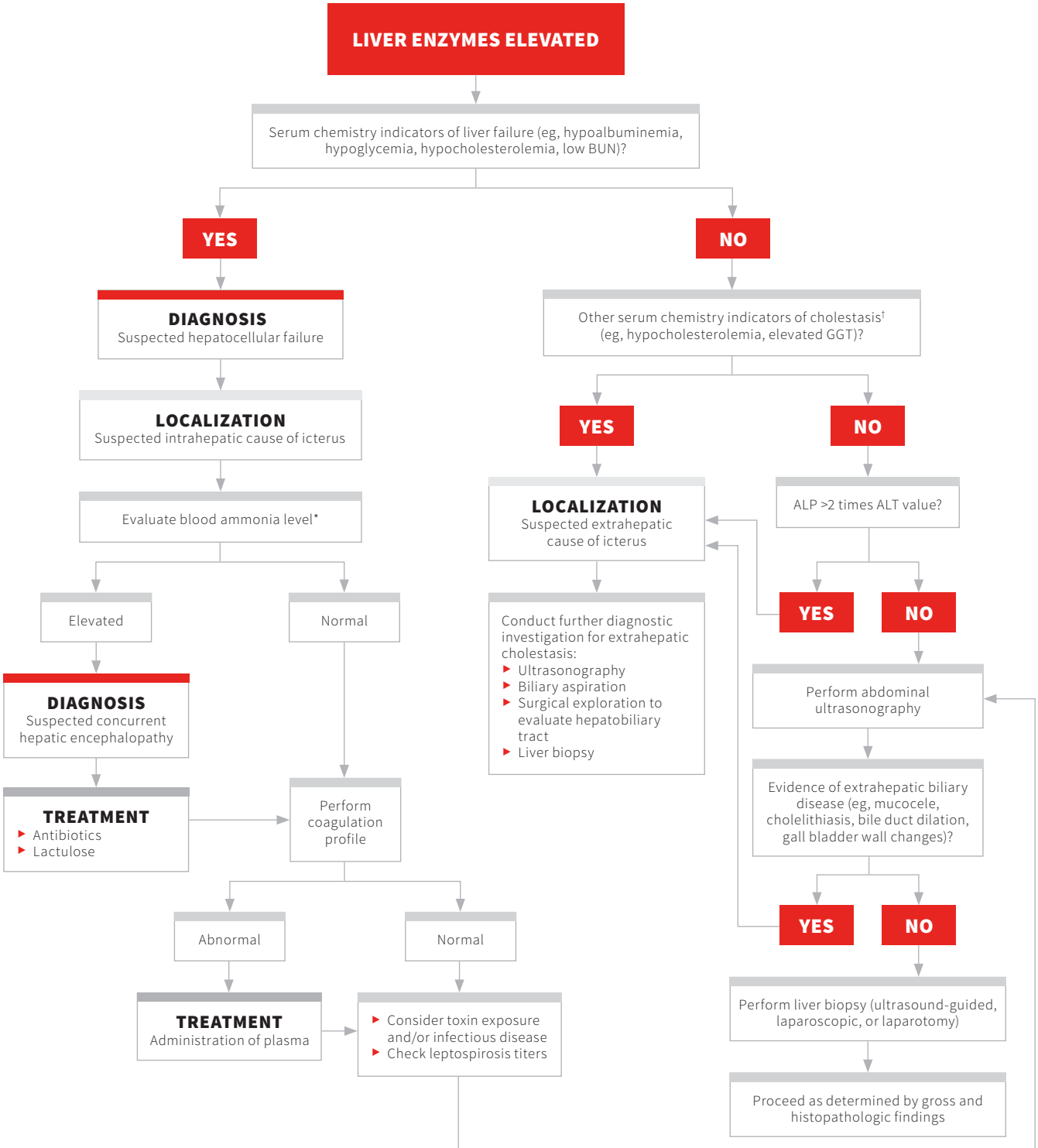




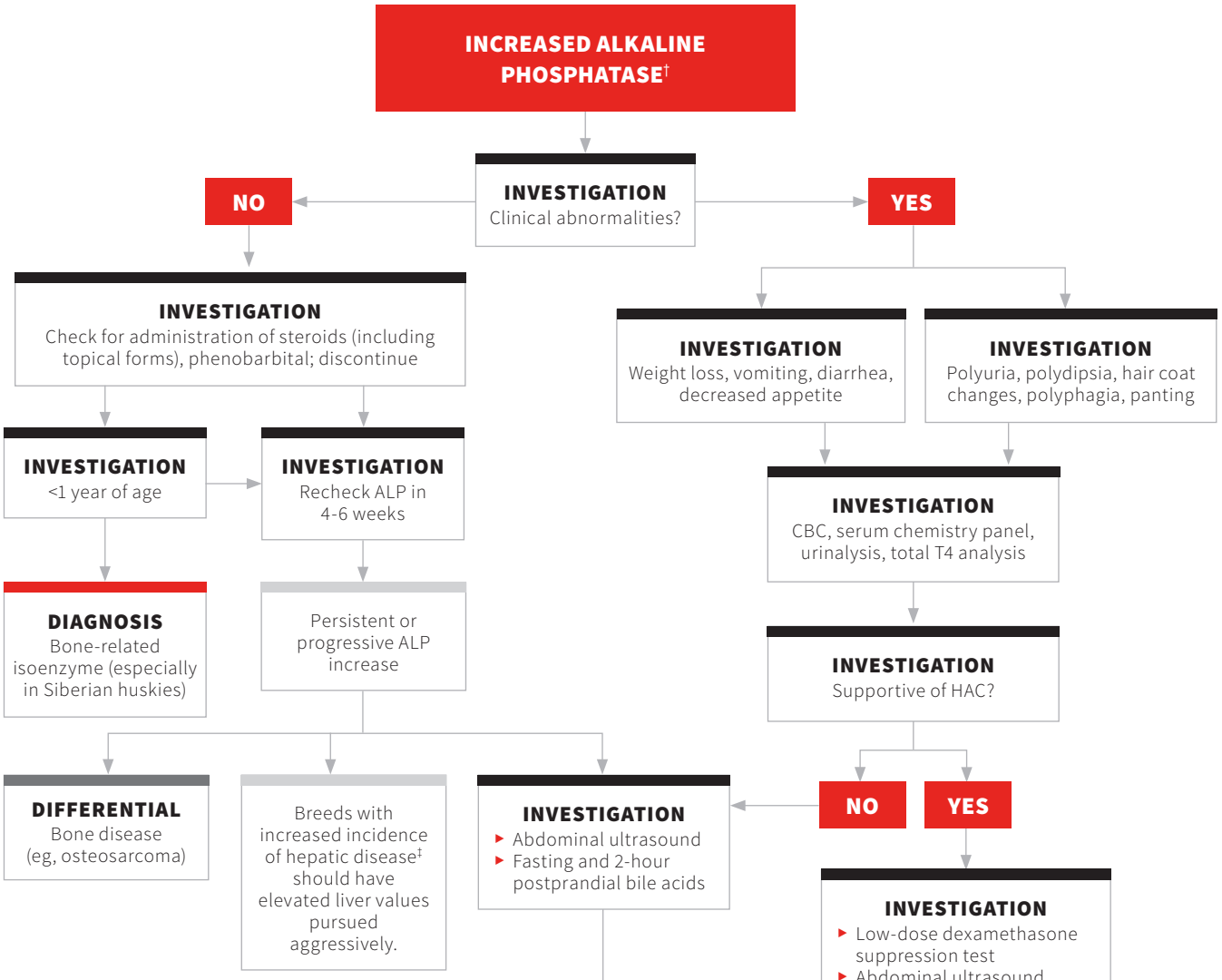
ALP = alkaline phosphatase
ALT = alanine aminotransferase
GGT = gamma-glutamyl transpeptidase
IMHA = immune-mediated hemolytic anemia



*Clinicians should reference their laboratory's reference range when determining whether a value may be elevated, as reference ranges can vary between laboratories.
†Hypercholesterolemia should only be considered significant if the patient has been fasted for 8 hours prior to sample collection.

INCREASED LIVER ENZYMES IN DOGS: ALP

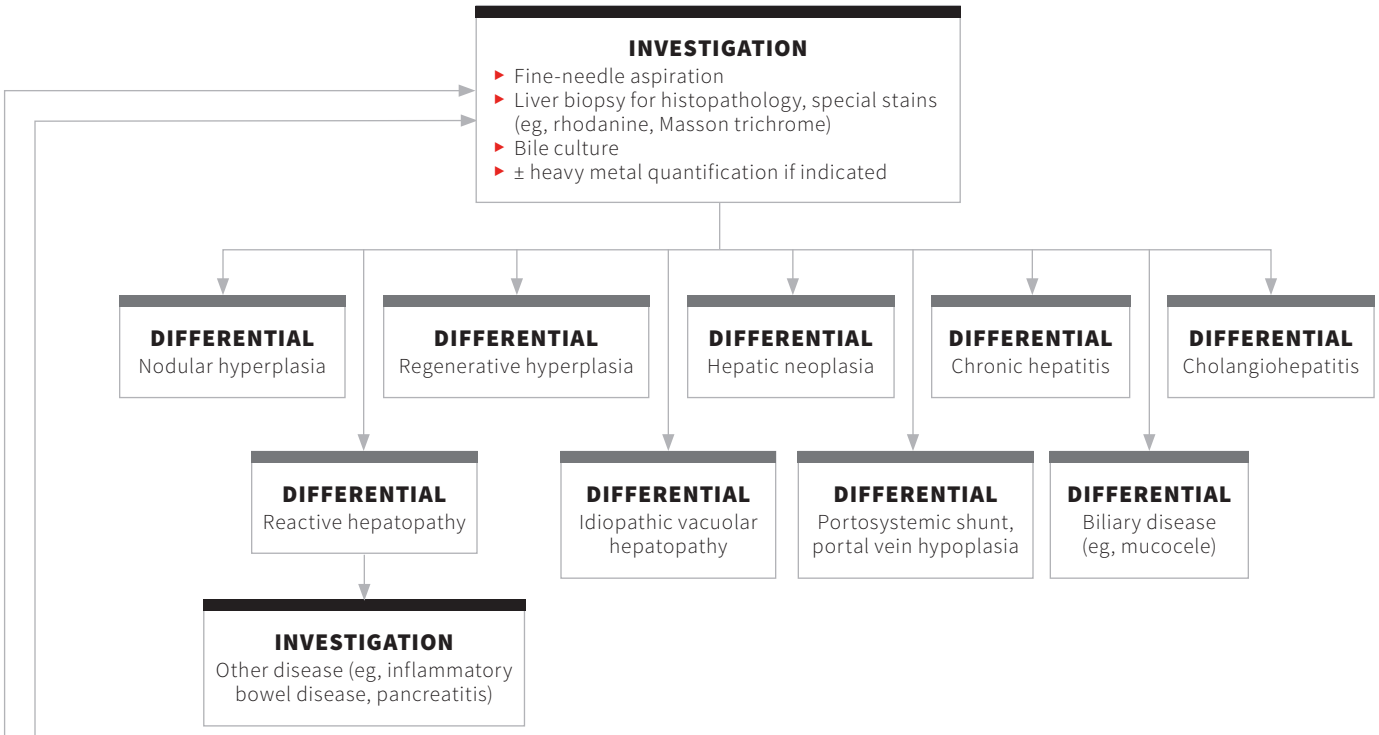
Julie Allen, BVMS, MS, MRCVS, DACVIM (SAIM)*
North Carolina State University



*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Julie Allen, BVMS, MS, MRCVS, DACVIM (SAIM), DACVP, at Veterinary Information Network.

†Any increased ALP should be rechecked at least once to rule out a spurious result or laboratory error, particularly in a nonclinical patient.

‡More aggressive evaluation should be considered in breeds predisposed to hepatic disease: Bedlington terriers, West Highland white terriers, Labrador retrievers, standard poodles, cocker spaniels, English springer spaniels, Doberman pinschers, dalmatians, Scottish terriers, and Skye terriers. Early diagnosis may improve treatment success and prognosis.



Suggested Reading

Bain PJ. Liver. In: Latimer KS, ed. *Duncan & Prasse's Veterinary Laboratory Medicine: Clinical Pathology*. 5th ed. Ames, IA: Wiley Blackwell; 2011:211-230.

Brovida C, Rothuizen J. Liver and pancreatic diseases. In: Ettinger SJ, Feldman EC, eds. *Textbook of Veterinary Internal Medicine*. 7th ed. St. Louis, MO: Saunders Elsevier; 2010:1609-1994.

Twedt DC. Abnormal liver enzymes. A clinical approach. In: American Board of Veterinary Practitioners Proceedings, 2014, Nashville, TN.

ACTH = adrenocorticotropic hormone

ALP = alkaline phosphatase

HAC = hyperadrenocorticism

INCREASED LIVER ENZYMES IN DOGS: ALT

Julie Allen, BVMS, MS, MRCVS, DACVIM (SAIM)*
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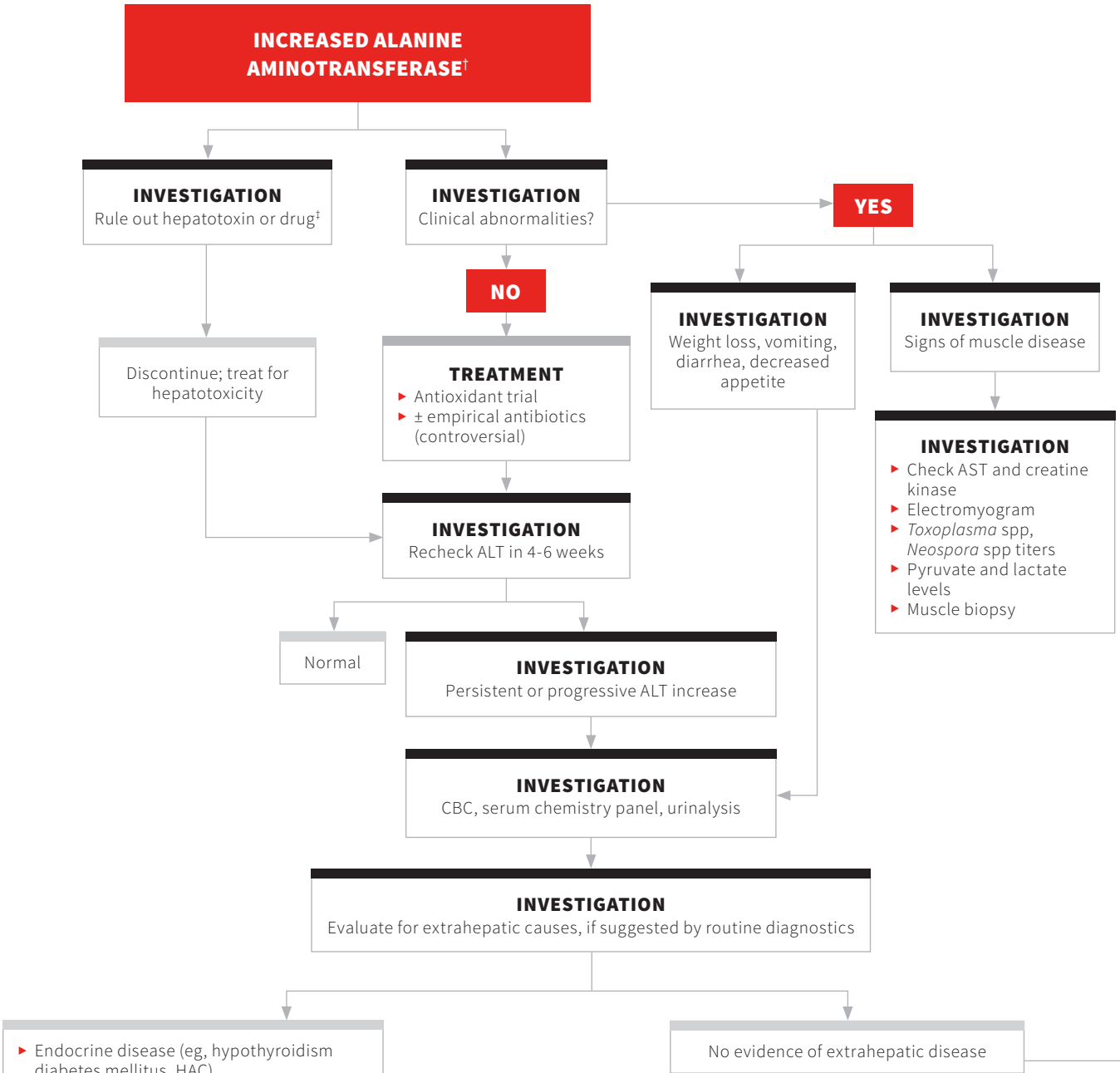
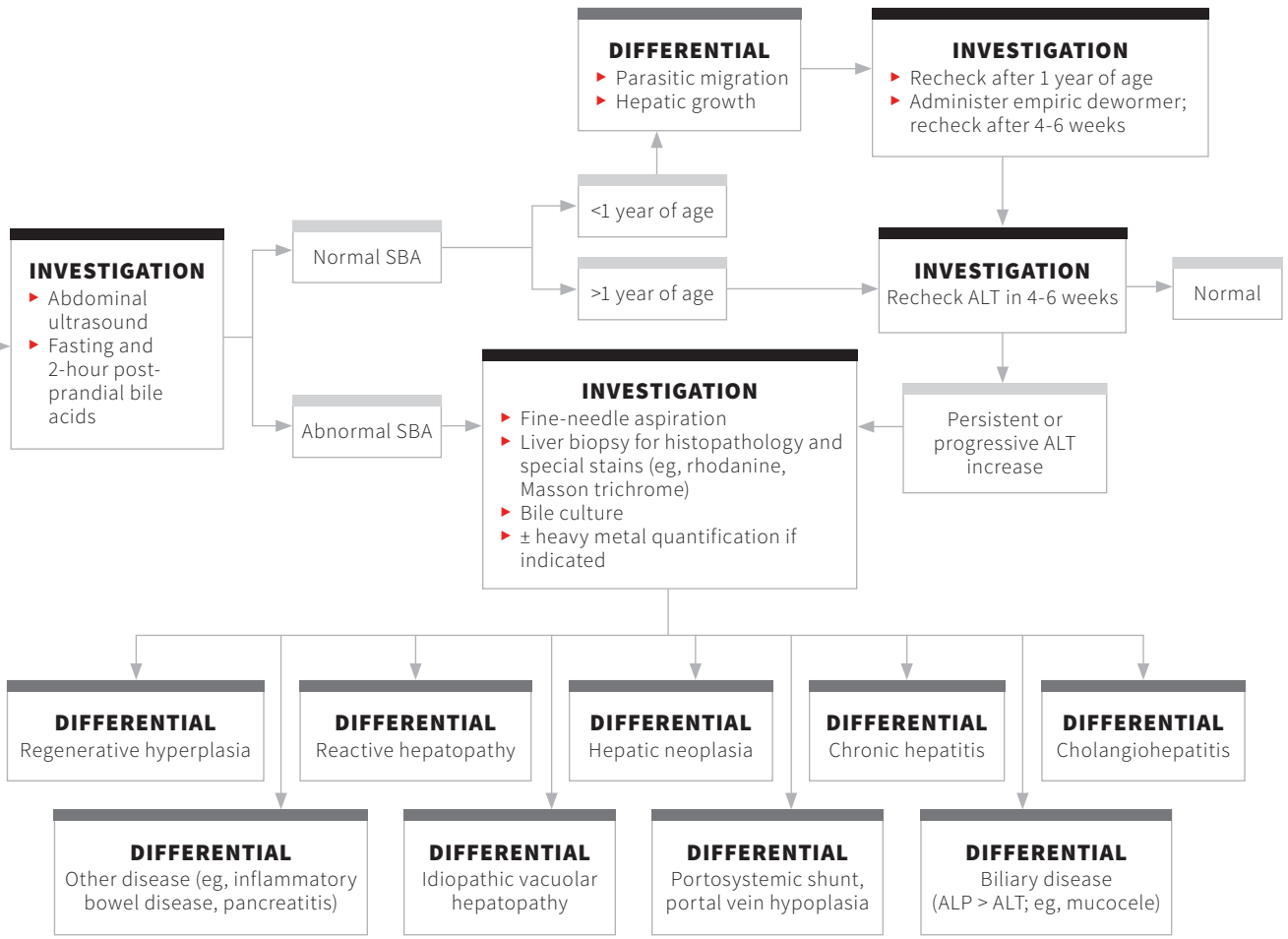


*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Julie Allen, BVMS, MS, MRCVS, DACVIM (SAIM), DACVP, at Veterinary Information Network.

†Any increase in ALT should be rechecked at least once to rule out a spurious result or laboratory error, particularly if the patient is subclinical. More aggressive evaluation should be considered in breeds predisposed to hepatic disease: Bedlington terriers, West Highland white terriers, Labrador retrievers, standard poodles, cocker spaniels, English springer spaniels, Doberman pinschers, dalmatians, and Skye terriers.

‡Acetaminophen, azathioprine, hepatotoxic mushrooms, lomustine, blue-green algae, xylitol, cycad palm, and aflatoxin.



Suggested Reading

Bain PJ. Liver. In: Latimer KS, ed. *Duncan & Prasse's Veterinary Laboratory Medicine: Clinical Pathology*. 5th ed. Ames, IA: Wiley Blackwell; 2011:211-230.

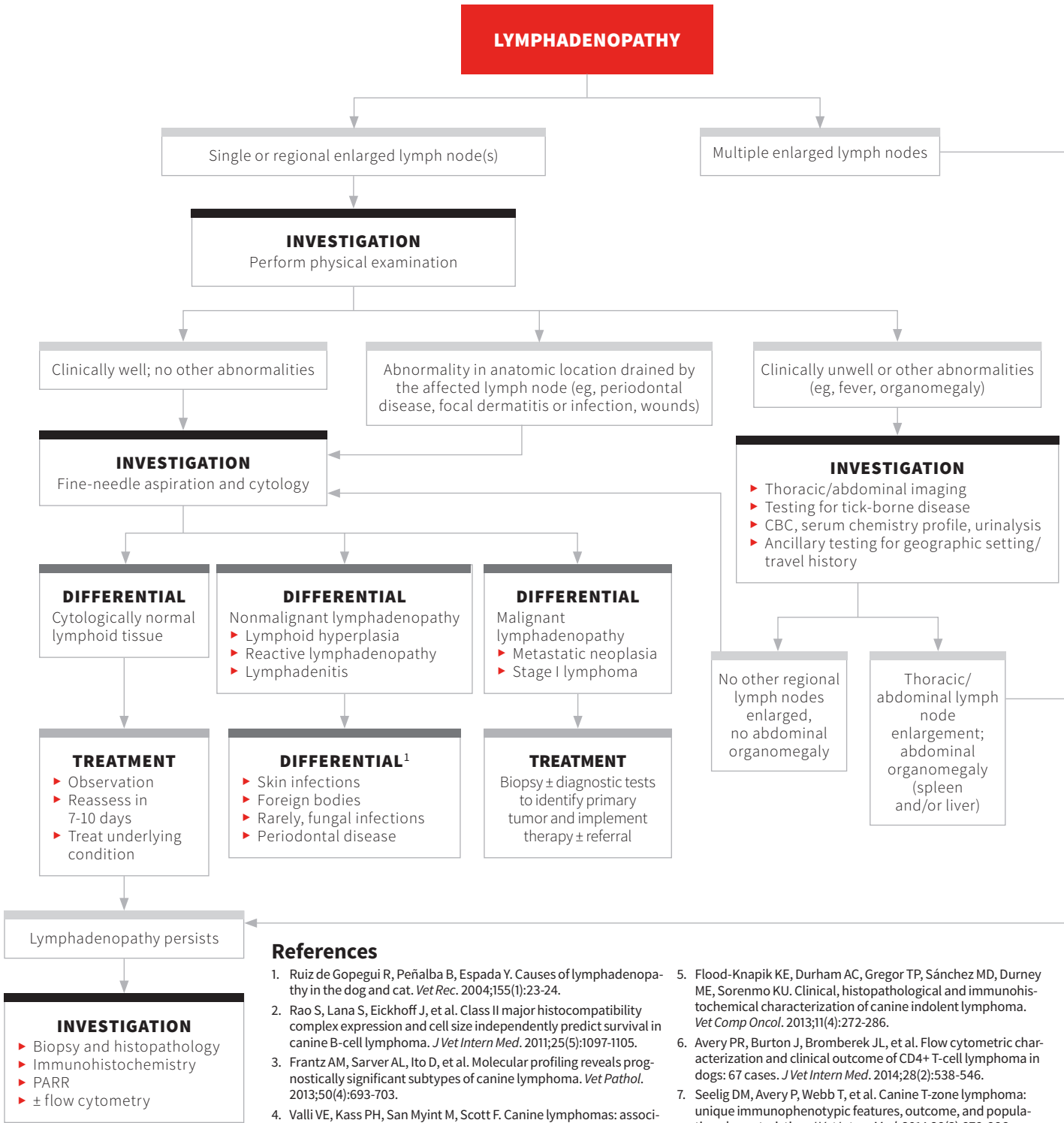
Brovida C, Rothuizen J. Liver and pancreatic diseases. In: Ettinger SJ, Feldman EC, eds. *Textbook of Veterinary Internal Medicine*. 7th ed. St. Louis, MO: Saunders Elsevier; 2010:1609-1994.

Twedt DC. Abnormal liver enzymes. A clinical approach. In: American Board of Veterinary Practitioners Proceedings, 2014, Nashville, TN.

ALP = alkaline phosphatase
ALT = alanine aminotransferase
AST = aspartate transaminase
HAC = hyperadrenocorticism
SBA = serum bile acids

LYMPHADENOPATHY IN DOGS

Michelle G. Ritt, DVM, DACVIM
Claire Cannon, BVSc (Hons), DACVIM (Oncology)
Jaime F. Modiano, VMD, PhD
University of Minnesota



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2. Rao S, Lana S, Eickhoff J, et al. Class II major histocompatibility complex expression and cell size independently predict survival in canine B-cell lymphoma. *J Vet Intern Med*. 2011;25(5):1097-1105.
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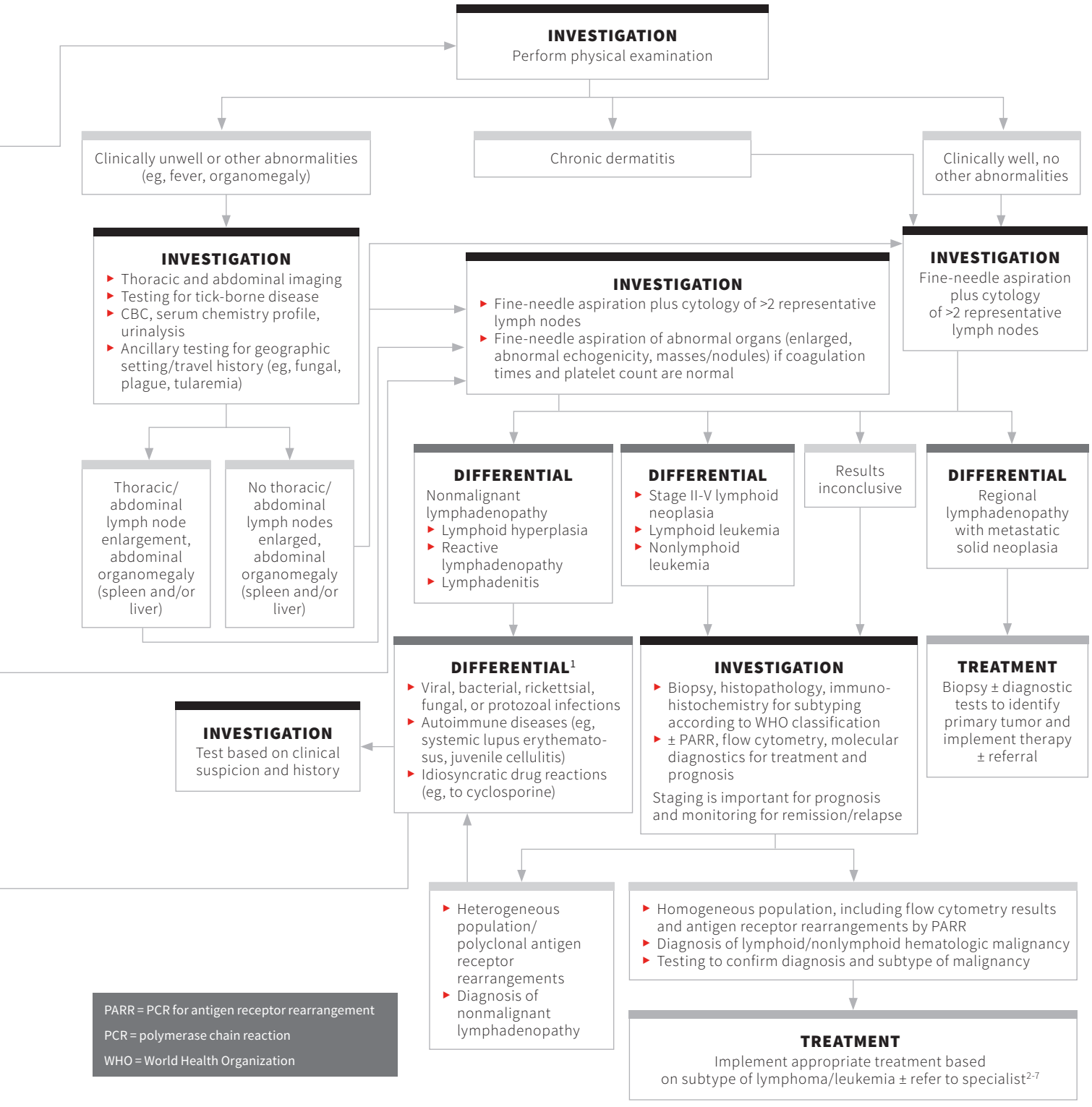
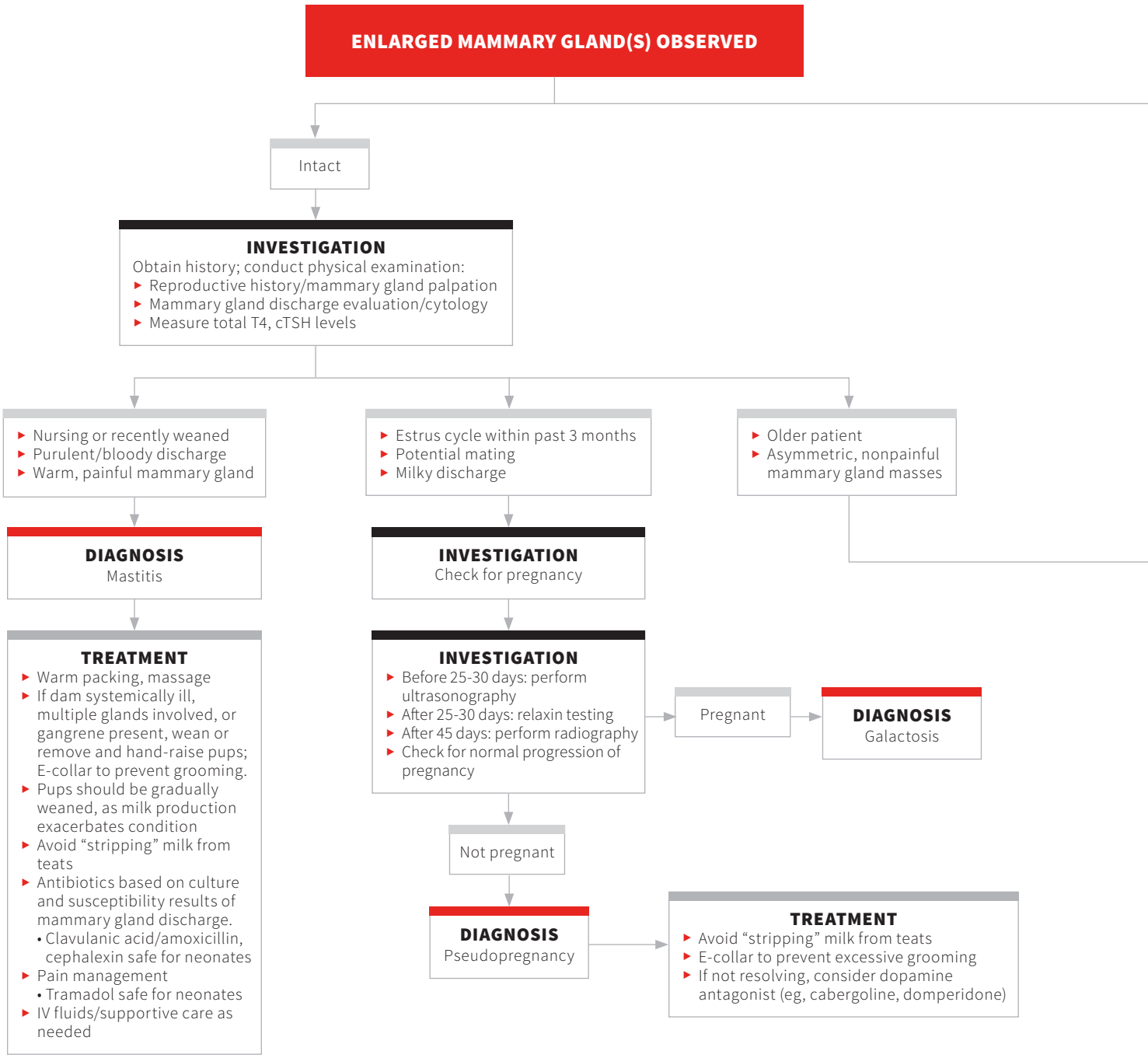
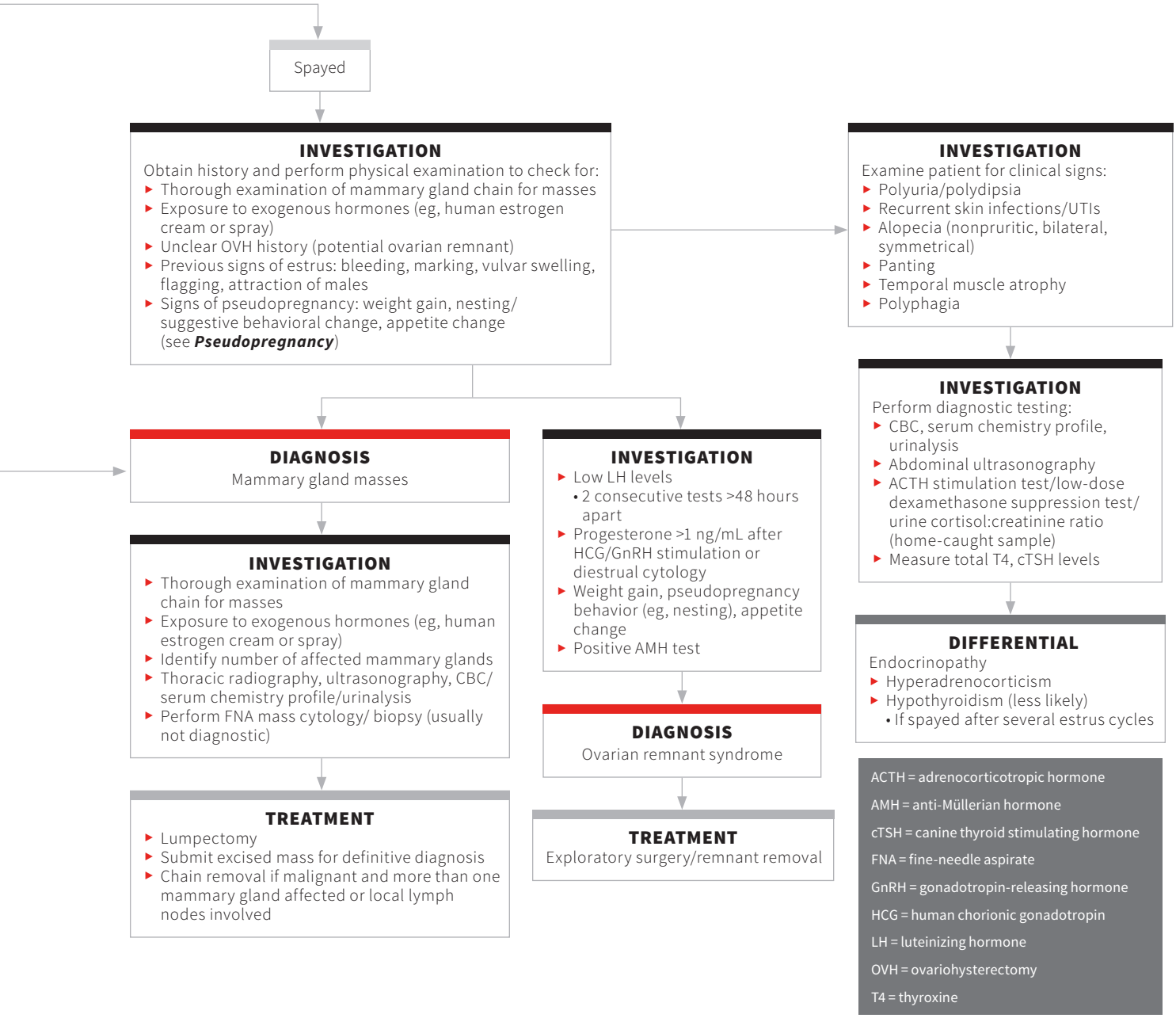
7. Seelig DM, Avery P, Webb T, et al. Canine T-zone lymphoma: unique immunophenotypic features, outcome, and population characteristics. *J Vet Intern Med*. 2014;28(3):878-886.


INVESTIGATION
Perform physical examination
Clinically unwell or other abnormalities (eg, fever, organomegaly)
Chronic dermatitis
Clinically well, no other abnormalities
INVESTIGATION
Thoracic and abdominal imaging
Testing for tick-borne disease
CBC, serum chemistry profile, urinalysis
Ancillary testing for geographic setting/travel history (eg, fungal, plague, tularemia)
INVESTIGATION
Fine-needle aspiration plus cytology of >2 representative lymph nodes
Fine-needle aspiration of abnormal organs (enlarged, abnormal echogenicity, masses/nodules) if coagulation times and platelet count are normal
INVESTIGATION
Fine-needle aspiration plus cytology of >2 representative lymph nodes
Thoracic/ abdominal lymph node enlargement, abdominal organomegaly (spleen and/or liver)
No thoracic/ abdominal lymph nodes enlarged, abdominal organomegaly (spleen and/or liver)
DIFFERENTIAL
Nonmalignant lymphadenopathy
Lymphoid hyperplasia
Reactive lymphadenopathy
Lymphadenitis
DIFFERENTIAL
Stage II-V lymphoid neoplasia
Lymphoid leukemia
Nonlymphoid leukemia
Results inconclusive
DIFFERENTIAL
Regional lymphadenopathy with metastatic solid neoplasia
INVESTIGATION
Test based on clinical suspicion and history
DIFFERENTIAL[1]
Viral, bacterial, rickettsial, fungal, or protozoal infections
Autoimmune diseases (eg, systemic lupus erythematosus, juvenile cellulitis)
Idiosyncratic drug reactions (eg, to cyclosporine)
INVESTIGATION
Biopsy, histopathology, immunohistochemistry for subtyping according to WHO classification
± PARR, flow cytometry, molecular diagnostics for treatment and prognosis
Staging is important for prognosis and monitoring for remission/relapse
TREATMENT
Biopsy ± diagnostic tests to identify primary tumor and implement therapy ± referral
Heterogeneous population/ polyclonal antigen receptor rearrangements
Diagnosis of nonmalignant lymphadenopathy
Homogeneous population, including flow cytometry results and antigen receptor rearrangements by PARR
Diagnosis of lymphoid/nonlymphoid hematologic malignancy
Testing to confirm diagnosis and subtype of malignancy
TREATMENT
Implement appropriate treatment based on subtype of lymphoma/leukemia ± refer to specialist[2-7]
PARR = PCR for antigen receptor rearrangement
PCR = polymerase chain reaction
WHO = World Health Organization

MAMMARY GLAND ENLARGEMENT IN FEMALE DOGS

Valeria M. Tanco, DVM, MSc, PhD, DACT
Charleston Veterinary Referral Center
Charleston, South Carolina

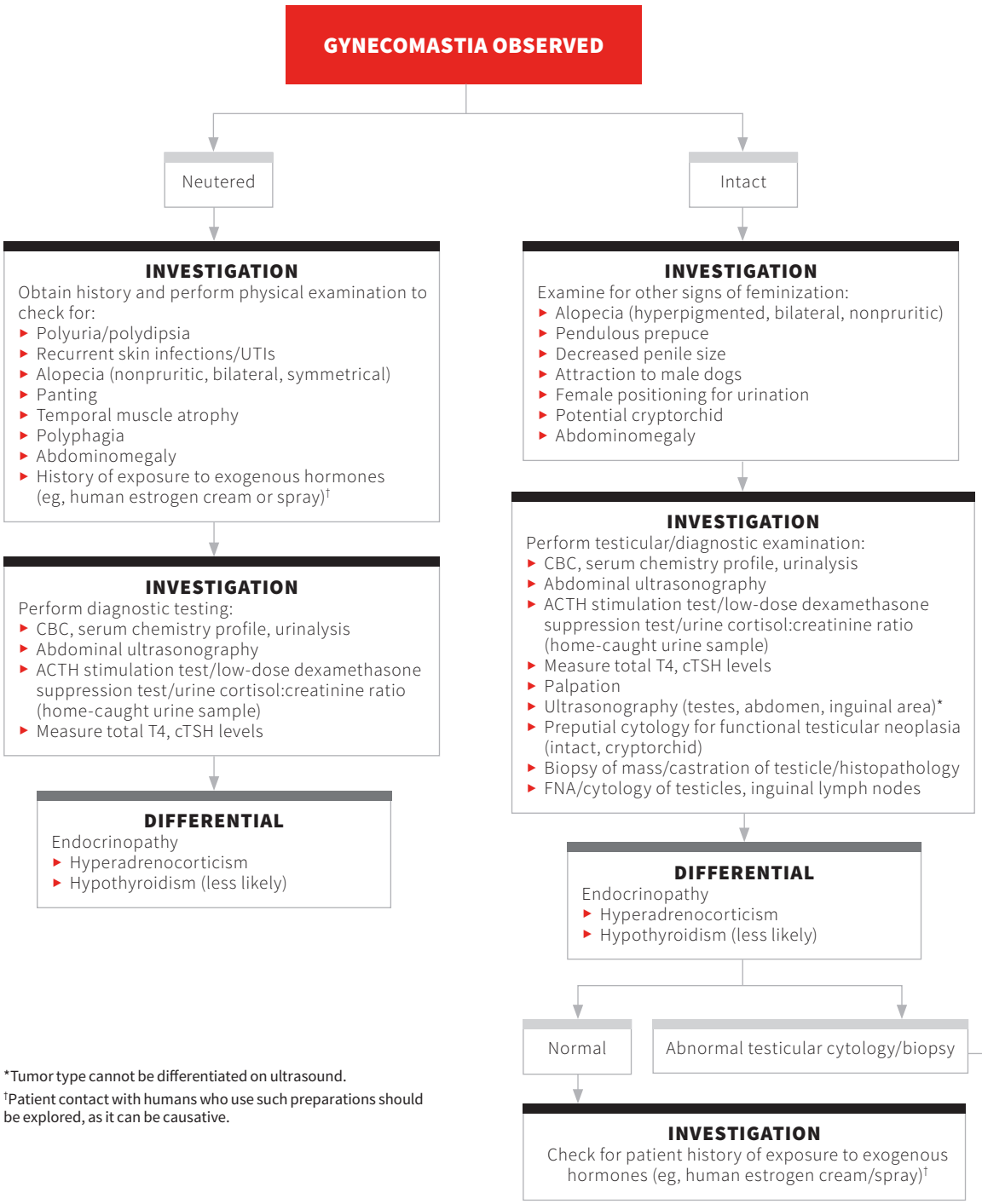




ACTH = adrenocorticotropic hormone
AMH = anti-Müllerian hormone
cTSH = canine thyroid stimulating hormone
FNA = fine-needle aspirate
GnRH = gonadotropin-releasing hormone
HCG = human chorionic gonadotropin
LH = luteinizing hormone
OVH = ovariohysterectomy
T4 = thyroxine

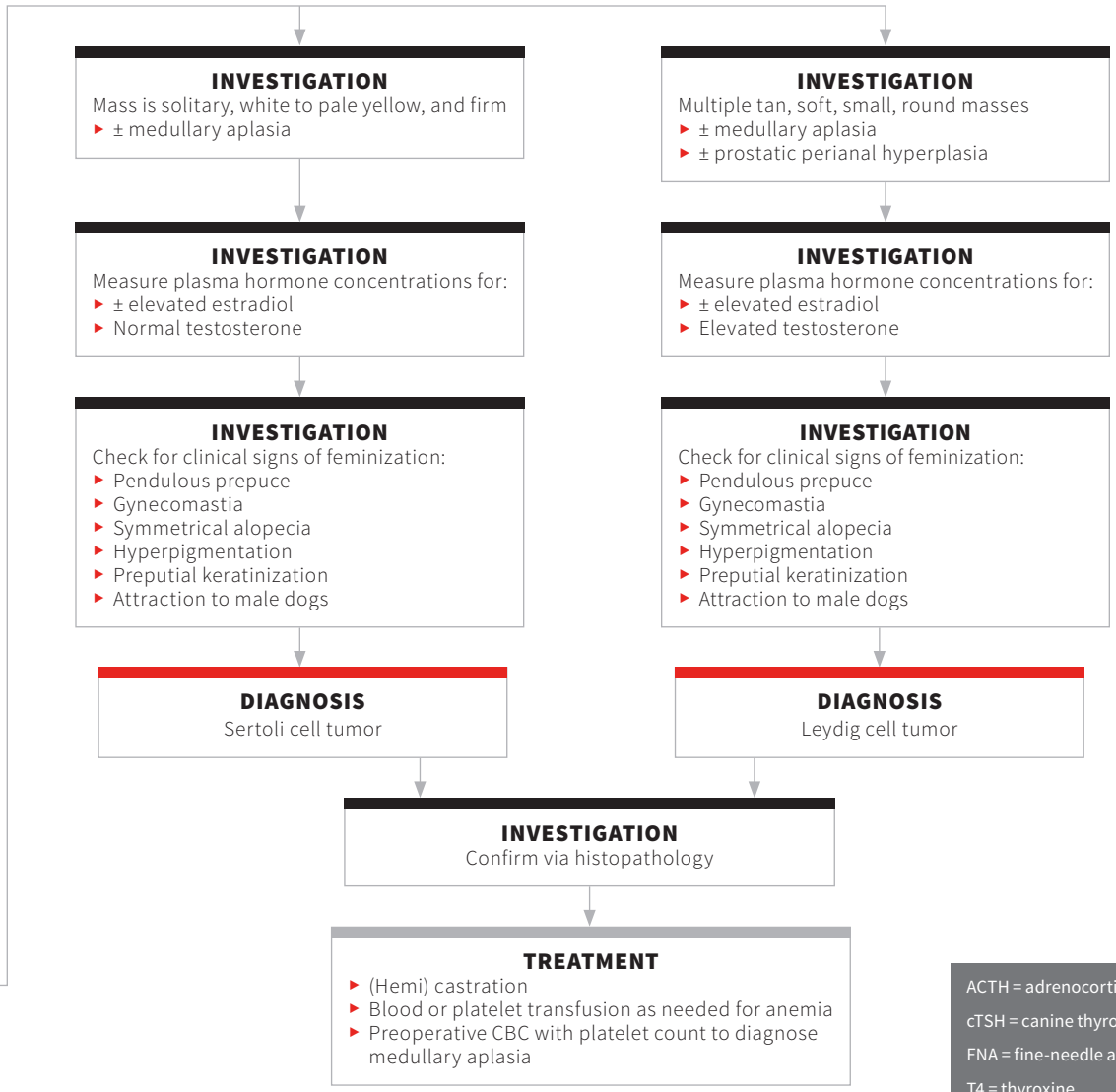
MAMMARY GLAND ENLARGEMENT IN MALE DOGS

Valeria M. Tanco, DVM, MSc, PhD, DACT
Charleston Veterinary Referral Center
Charleston, South Carolina



*Tumor type cannot be differentiated on ultrasound.

†Patient contact with humans who use such preparations should be explored, as it can be causative.



ACTH = adrenocorticotropic hormone

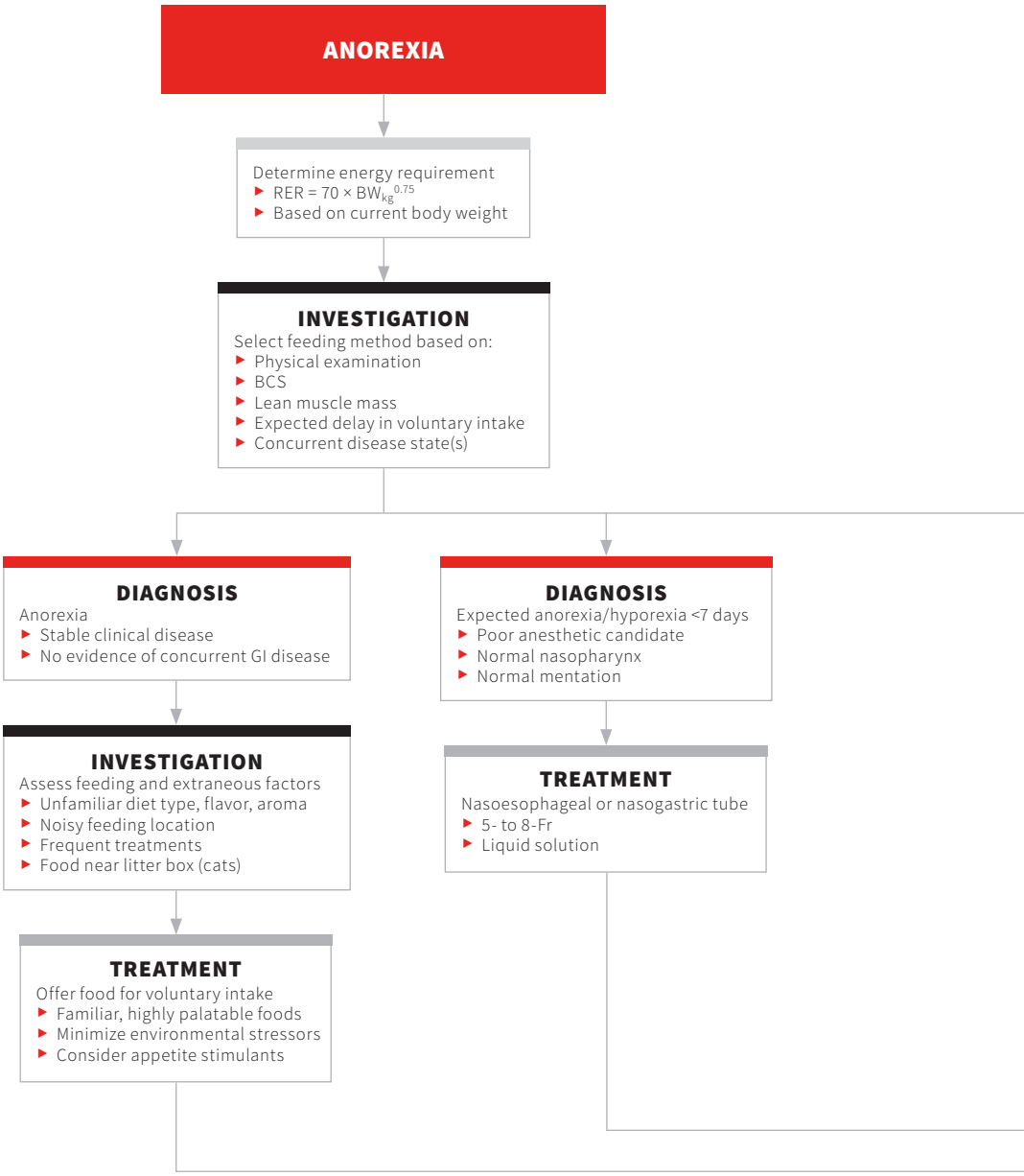
cTSH = canine thyroid stimulating hormone

FNA = fine-needle aspiration

T4 = thyroxine

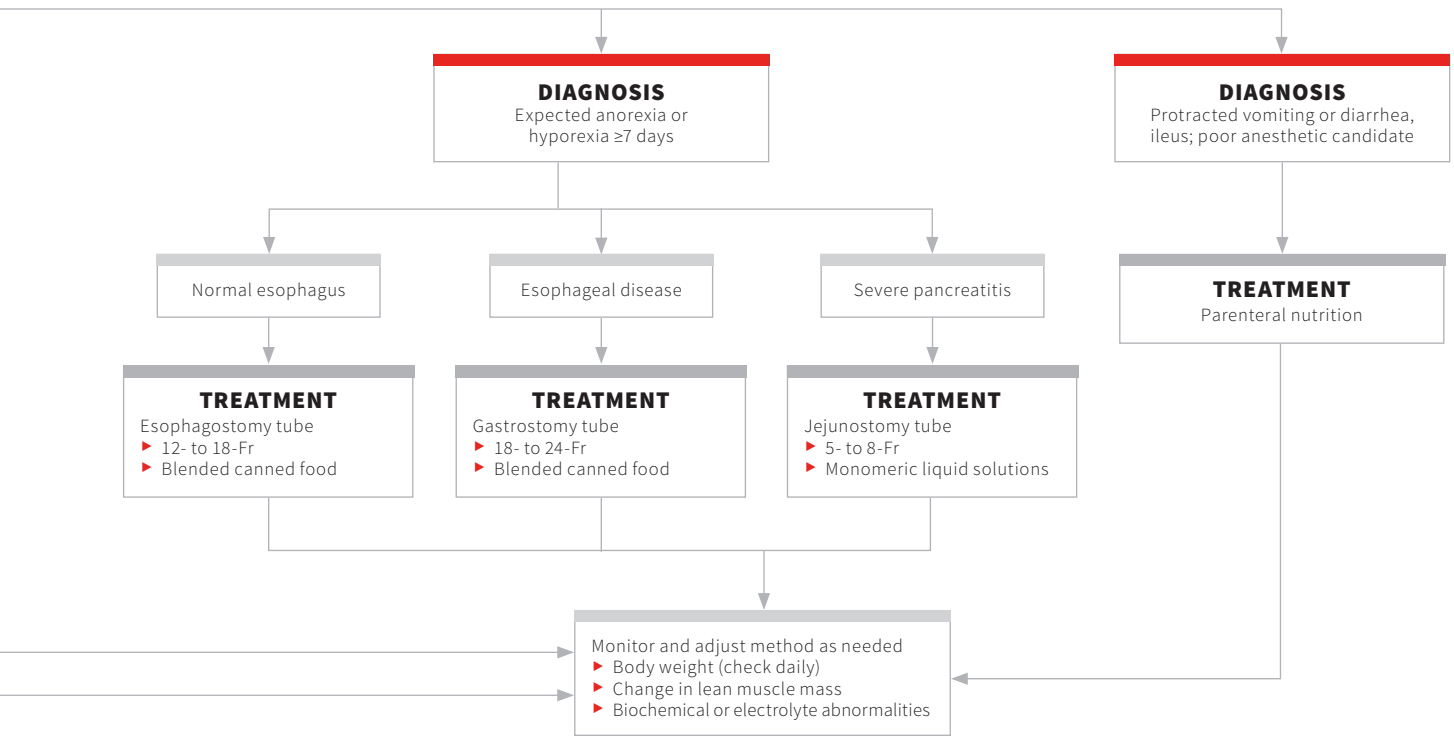
MANAGING ANOREXIA

Lisa P. Weeth, DVM, DACVN*
Red Bank Veterinary Hospital
Tinton Falls, New Jersey



BW = body weight
RER = resting energy requirement

*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Lisa P. Weeth, DVM, MRCVS, DACVN, at Weeth Nutrition Services, Los Angeles, California.


DIAGNOSIS
Expected anorexia or hyporexia ≥7 days
DIAGNOSIS
Protracted vomiting or diarrhea, ileus; poor anesthetic candidate
Normal esophagus
Esophageal disease
Severe pancreatitis
TREATMENT
Parenteral nutrition
TREATMENT
Esophagostomy tube
12- to 18-Fr
Blended canned food
TREATMENT
Gastrostomy tube
18- to 24-Fr
Blended canned food
TREATMENT
Jejunostomy tube
5- to 8-Fr
Monomeric liquid solutions
Monitor and adjust method as needed
Body weight (check daily)
Change in lean muscle mass
Biochemical or electrolyte abnormalities

OBESITY IN CATS

Deborah E. Linder, DVM, MS, DACVN
Cummings School of Veterinary Medicine at Tufts University

OBESITY DIAGNOSED*

Conduct nutrition assessment, including diet and environment[1-3]

Obtain dietary history[1-3]:
- Current diet (ie, brand, amount, frequency, flavor, dry vs wet)
- Treats, chews
- Table scraps
- Access to wildlife (eg, birds, mice)
- Medication administration
- Supplements

Conduct environmental assessment[1-3]:
- Owner description of meal time
- Number of family members
- Number of pets
- Possible access to other pets' food
- Amount of physical activity
- Enrichment (indoor, outdoor)[4]

Evaluate patient's relationship with owner[5] and owner's willingness and readiness to change[6]:
- No interest
- Contemplation
- Preparation
- Action
- Maintenance

Conduct physical assessment[1-3]:
- BCS[7]
  - Overweight score: 6/9-7/9
  - Obese score: 8/9-9/9
- MCS[8]
- Complete physical examination
- Diagnostics (ie, CBC, serum chemistry profile, urinalysis, diagnostic imaging) to determine comorbidities
- Quality-of-life assessment[9]

Go to ***Treatment***, next page

MCS = muscle condition score

*The AAHA Weight Management Guidelines define obesity as a BCS 8/9 or above,[1] although the guidelines, as well as this algorithm, may be helpful for any pet that is above ideal body weight.

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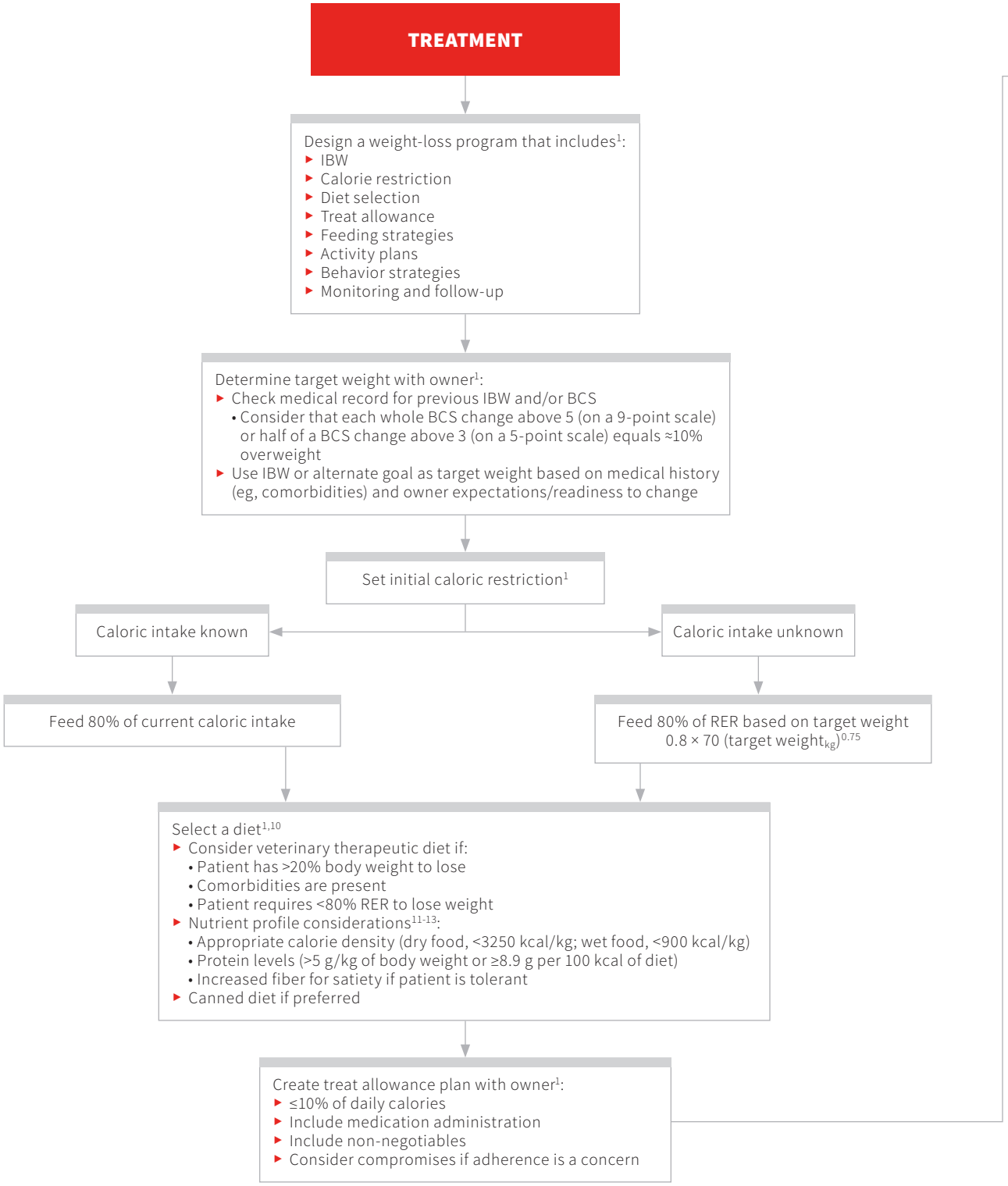
TREATMENT
Design a weight-loss program that includes[1]:
IBW
Calorie restriction
Diet selection
Treat allowance
Feeding strategies
Activity plans
Behavior strategies
Monitoring and follow-up
Determine target weight with owner[1]:
Check medical record for previous IBW and/or BCS
Consider that each whole BCS change above 5 (on a 9-point scale) or half of a BCS change above 3 (on a 5-point scale) equals ≈10% overweight
Use IBW or alternate goal as target weight based on medical history (eg, comorbidities) and owner expectations/readiness to change
Set initial caloric restriction[1]
Caloric intake known
Caloric intake unknown
Feed 80% of current caloric intake
Feed 80% of RER based on target weight
0.8 × 70 (target weight$_{kg}$)$^{0.75}$
Select a diet[1,10]
Consider veterinary therapeutic diet if:
Patient has >20% body weight to lose
Comorbidities are present
Patient requires <80% RER to lose weight
Nutrient profile considerations[11-13]:
Appropriate calorie density (dry food, <3250 kcal/kg; wet food, <900 kcal/kg)
Protein levels (>5 g/kg of body weight or ≥8.9 g per 100 kcal of diet)
Increased fiber for satiety if patient is tolerant
Canned diet if preferred
Create treat allowance plan with owner[1]:
≤10% of daily calories
Include medication administration
Include non-negotiables
Consider compromises if adherence is a concern

Encourage active lifestyle[1,4]:
- Kibble hidden around the house
- Food-dispensing toys
- Laser pointers, strings
- Kibble tossed around room
- Motorized mice/toys

FOLLOW-UP

Develop monitoring plan with owner[1]:
- Weigh-in every 2-4 weeks
- Determine adherence
  - Revisit diet history
- Record body weight, BCS, MCS
- Calculate rate of weight loss
  - Aim for 0.5%-2% of body weight per week
- Slow rate of weight loss if:
  - Rate >2% per week
  - Comorbid condition
  - MCS decreases at all (ie, there should be no palpable muscle wasting)
- Reduce caloric intake by 10% if:
  - Owner is adherent and rate is <0.5% per week

Troubleshoot with owner in cases of[1,5]:
- Begging behavior
  - Try autofeeders
  - Try increased fiber for satiety
- Multipet households
  - Separate pets with baby gate or by room
  - Try microchip reader feeders (ie, devices that only allow access to pets by scanning microchip)
- Weight-loss plateau
  - Increase physical activity
  - Consider food with lower calorie density
- Owner guilt or emotional concerns[5]
  - Discuss concerns with open-ended questions
  - Brainstorm treatment strategies with owner
  - Consider all members of household
  - Discuss alternatives to *food is love* method of care (eg, walking, grooming, interactive play)

References

1. Brooks D, Churchill J, Fein K, et al. 2014 AAHA weight management guidelines for dogs and cats. *J Am Anim Hosp Assoc*. 2014;50(1):1-11.
2. Baldwin K, Bartges J, Buffington T, et al. AAHA nutritional assessment guidelines for dogs and cats. *J Am Anim Hosp Assoc*. 2010;46(4):285-296.
3. Freeman L, Becvarova I, Cave N, et al. WSAVA nutritional assessment guidelines. *Compend Contin Educ Vet*. 2011;33(8):E1-E9.
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5. Linder D, Mueller M. Pet obesity management: beyond nutrition. *Vet Clin North Am Small Anim Pract*. 2014;44(4):789-806.
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9. German AJ, Holden SL, Wiseman-Orr ML, et al. Quality of life is reduced in obese dogs but improves after successful weight loss. *Vet J*. 2012;192(3):428-434.
10. Linder DE, Parker VJ. Dietary aspects of weight management in cats and dogs. *Vet Clin North Am Small Anim Pract*. 2016;46(5):869-882.
11. Association of American Feed Control Officials. Model regulations for pet food and specialty pet food under the model bill. In: Association of American Feed Control Officials, ed. *AAFCO 2017 Official Publication*. 2017:136-149. http://www.aafco.org/publications. Accessed March 1, 2017.
12. Linder DE, Freeman LM, Morris P, et al. Theoretical evaluation of risk for nutritional deficiency with caloric restriction in dogs. *Vet Q*. 2012;32(3-4):123-129.
13. Linder DE, Freeman LM, Holden SL, Biourge V, German AJ. Status of selected nutrients in obese dogs undergoing caloric restriction. *BMC Vet Res*. 2013;9:219.

IBW = ideal body weight

MCS = muscle condition score

RER = resting energy requirement

OBESITY IN DOGS

Deborah E. Linder, DVM, MS, DACVN
Cummings School of Veterinary Medicine at Tufts University

OBESITY DIAGNOSED*

Conduct nutrition assessment, including diet and environment[1-3]

Obtain dietary history[1-3]:
- Current diet (ie, brand, amount, frequency, flavor, dry vs wet)
- Treats, chews
- Table scraps
- Bones, rawhides
- Medication administration
- Supplements

Conduct environmental assessment[1-3]:
- Owner description of meal time
- Number of family members
- Number of pets
- Possible access to other pets' food
- Amount of physical activity[4]

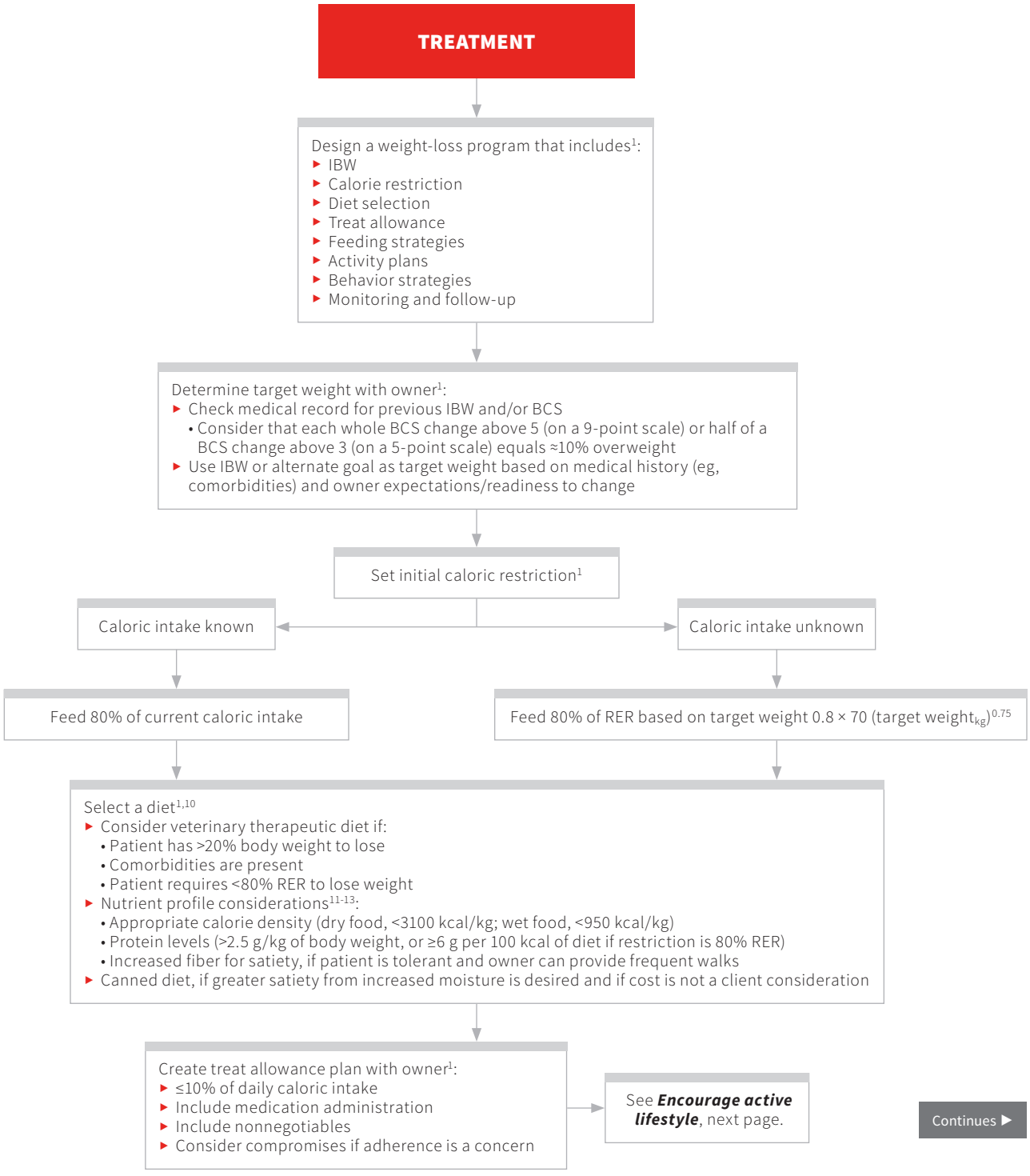
Evaluate patient's relationship with owner[5] and owner's willingness and readiness to change[6]:
- No interest
- Contemplation
- Preparation
- Action
- Maintenance

Conduct physical assessment[1-3]:
- BCS[7]
  - Overweight score: 6/9-7/9
  - Obese score: 8/9-9/9
- MCS[8]
- Complete physical examination, including orthopedic examination
- Diagnostics (ie, CBC, serum chemistry profile, urinalysis, diagnostic imaging) to determine comorbidities
- Quality-of-life assessment[9]

Go to ***Treatment***, next page

*The AAHA Weight Management Guidelines define obesity as having a BCS 8/9 or above,[1] although the guidelines, as well as this algorithm, may be helpful for any pet that is above ideal body weight.

IBW = ideal body weight
MCS = muscle condition score
RER = resting energy requirement


TREATMENT
Design a weight-loss program that includes[1]:
IBW
Calorie restriction
Diet selection
Treat allowance
Feeding strategies
Activity plans
Behavior strategies
Monitoring and follow-up
Determine target weight with owner[1]:
Check medical record for previous IBW and/or BCS
Consider that each whole BCS change above 5 (on a 9-point scale) or half of a BCS change above 3 (on a 5-point scale) equals ≈10% overweight
Use IBW or alternate goal as target weight based on medical history (eg, comorbidities) and owner expectations/readiness to change
Set initial caloric restriction[1]
Caloric intake known
Caloric intake unknown
Feed 80% of current caloric intake
Feed 80% of RER based on target weight 0.8 × 70 (target weight$_{kg}$)$^{0.75}$
Select a diet[1,10]
Consider veterinary therapeutic diet if:
Patient has >20% body weight to lose
Comorbidities are present
Patient requires <80% RER to lose weight
Nutrient profile considerations[11-13]:
Appropriate calorie density (dry food, <3100 kcal/kg; wet food, <950 kcal/kg)
Protein levels (>2.5 g/kg of body weight, or ≥6 g per 100 kcal of diet if restriction is 80% RER)
Increased fiber for satiety, if patient is tolerant and owner can provide frequent walks
Canned diet, if greater satiety from increased moisture is desired and if cost is not a client consideration
Create treat allowance plan with owner[1]:
≤10% of daily caloric intake
Include medication administration
Include nonnegotiables
Consider compromises if adherence is a concern
See Encourage active lifestyle, next page.

Continues ▶

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Encourage active lifestyle[1,4]:
- Walks
- Food-dispensing toys
- Chase balls or treats
- Underwater treadmill
- Agility course
- Active play or walking
- Obedience classes

FOLLOW-UP

Develop monitoring plan with owner[1]:
- Weigh-in every 2-4 weeks
- Determine adherence
  - Revisit diet history
- Record body weight, BCS, MCS
- Calculate rate of weight loss
  - Aim for 1%-2% of body weight per week
- Slow rate of weight loss if:
  - Rate >2% per week
  - Comorbid condition
  - MCS decreases at all (ie, there should be no palpable muscle wasting)
- Reduce caloric intake by 10% if:
  - Owner is adherent and rate is <1% per week

Troubleshoot with owner in cases of[1,5]:
- Begging behavior
  - Try autofeeders
  - Try increased fiber for satiety
- Multipet households
  - Separate pets with baby gate or by room
  - Try microchip reader feeders (ie, devices that only allow access to pets by scanning microchip)
- Weight-loss plateau
  - Increase physical activity
  - Consider food with lower calorie density
- Owner guilt or emotional concerns[5]
  - Discuss concerns with open-ended questions
  - Brainstorm treatment strategies with owner
  - Consider all members of household
  - Discuss alternatives to *food is love* method of care (eg, walking, grooming, interactive play)

References

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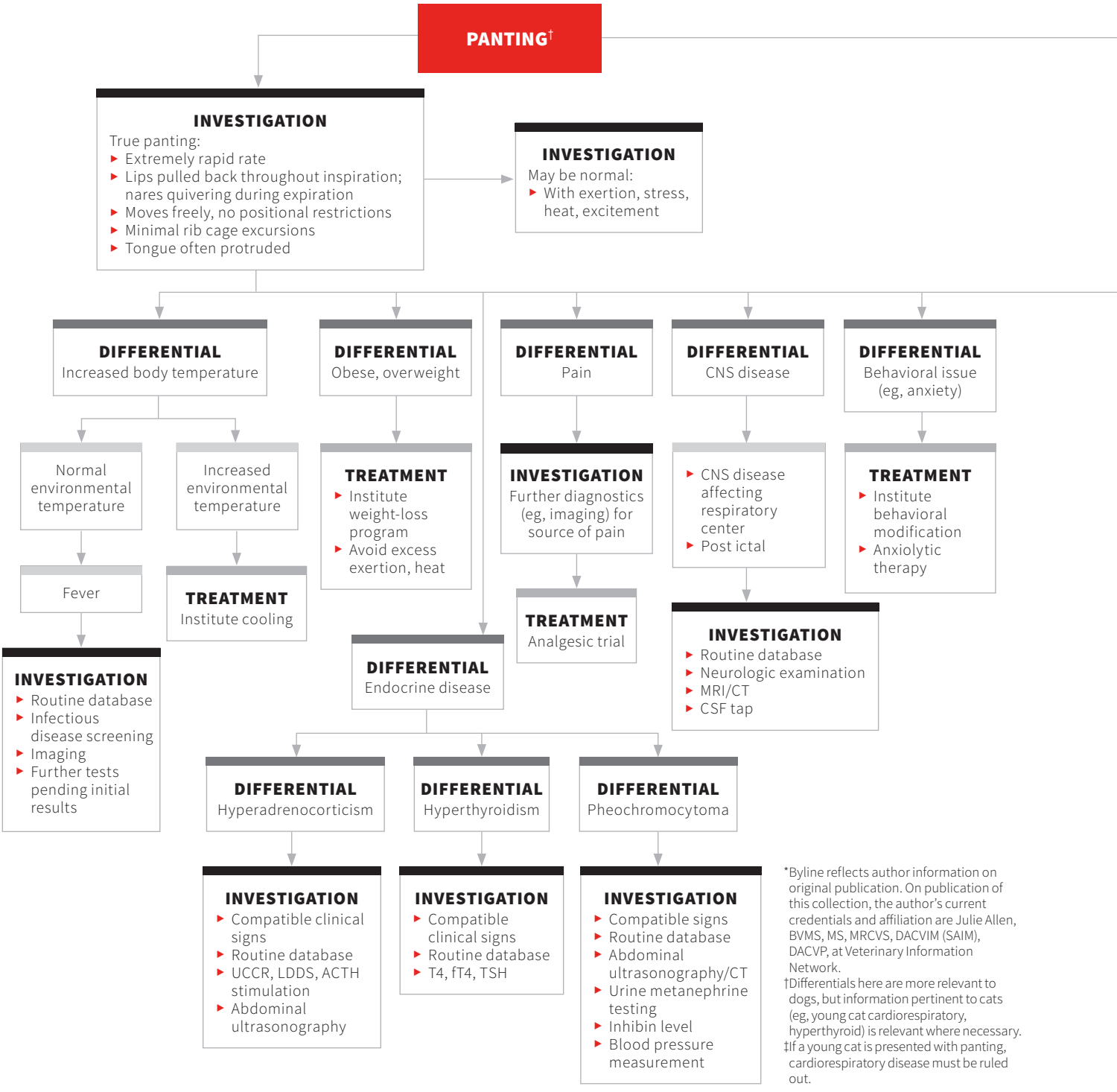
13. Linder DE, Freeman LM, Holden SL, Biourge V, German AJ. Status of selected nutrients in obese dogs undergoing caloric restriction. *BMC Vet Res.* 2013;9:219.

MCS = muscle condition score

PANTING

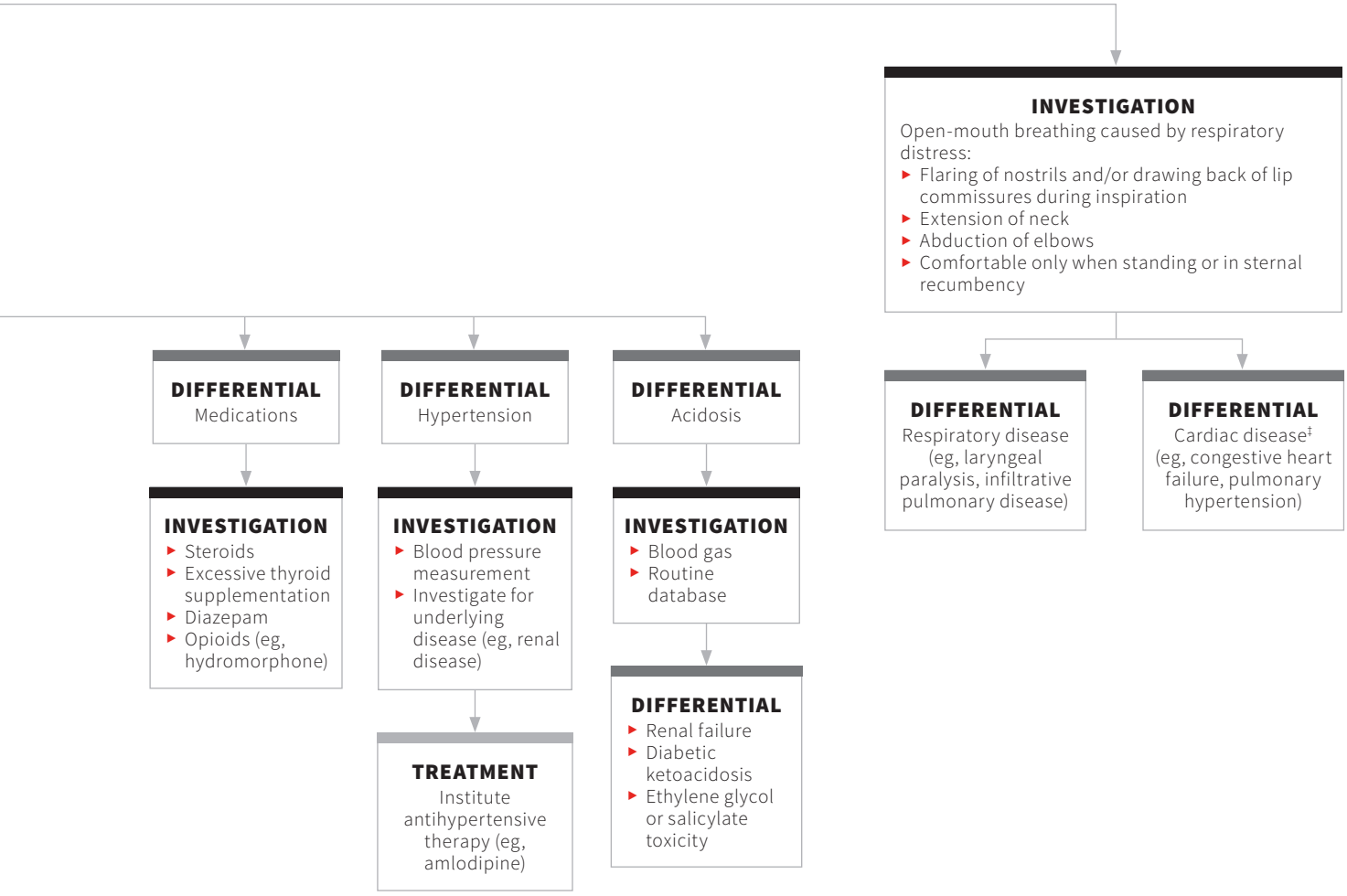
Julie Allen, BVMS, MS, MRCVS, DACVIM (SAIM)*
North Carolina State University



*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Julie Allen, BVMS, MS, MRCVS, DACVIM (SAIM), DACVP, at Veterinary Information Network.

†Differentials here are more relevant to dogs, but information pertinent to cats (eg, young cat cardiorespiratory, hyperthyroid) is relevant where necessary.

‡If a young cat is presented with panting, cardiorespiratory disease must be ruled out.



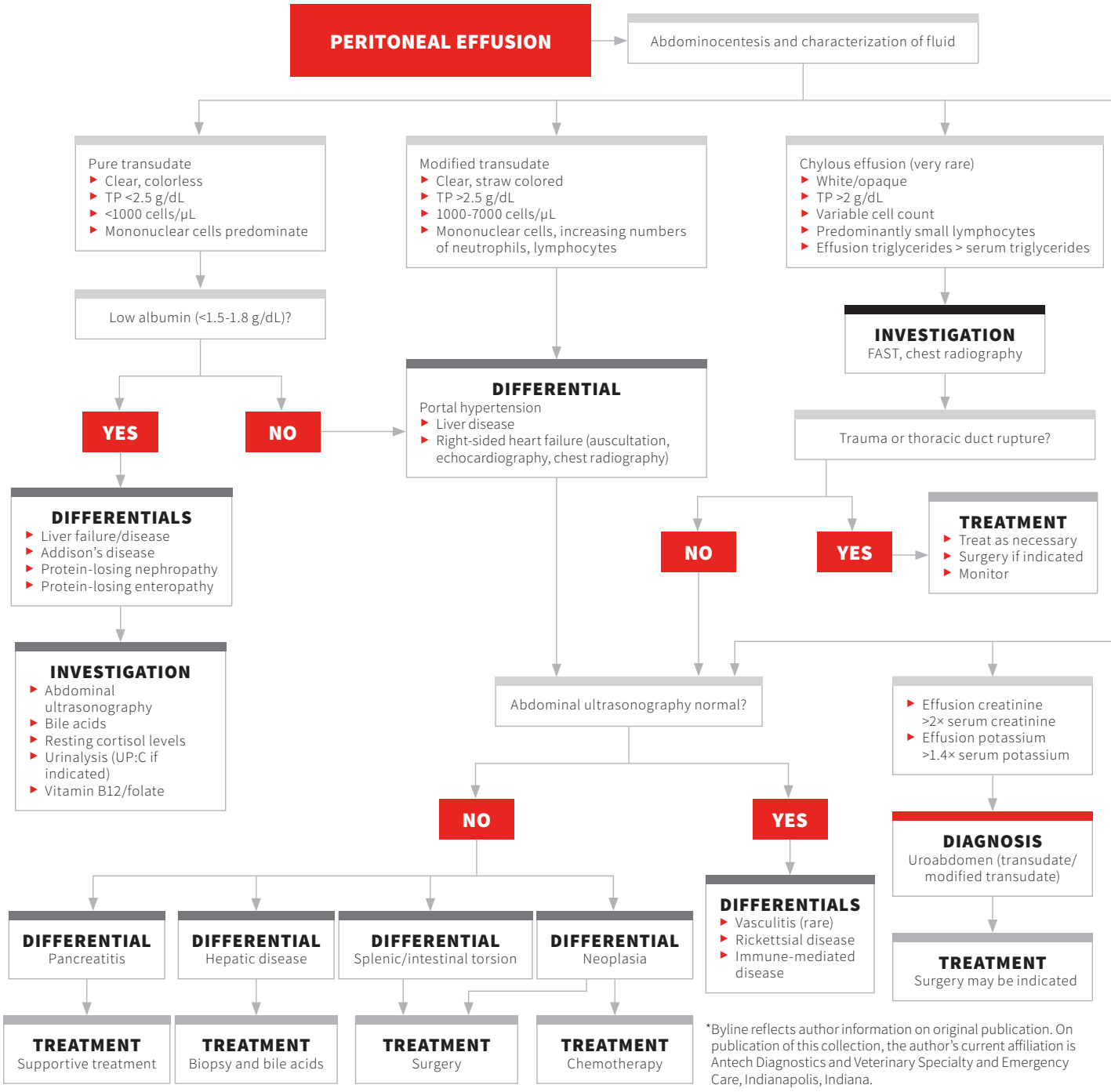
ACTH = adrenocorticotropic hormone
fT4 = free thyroxine
LDDS = low-dose dexamethasone suppression
T4 = total thyroxine
TSH = thyroid stimulating hormone
UCCR = urine cortisol: creatinine ratio

Reference

1. Kittleson M, Rishniw M. Panting and tachypnea. Veterinary Information Network. http://www.vin.com.

PERITONEAL EFFUSION

Gretchen Statz, DVM, DACVECC*
Internal Medicine Consultant
Antech Diagnostics



*Byline reflects author information on original publication. On publication of this collection, the author's current affiliation is Antech Diagnostics and Veterinary Specialty and Emergency Care, Indianapolis, Indiana.

Suggested Reading

Aronsohn MG, Dubiel B, Roberts B, Powers BE. Prognosis for acute nontraumatic hemoperitoneum in the dog: a retreospective analysis of 60 cases (2003-2006). *J Am Anim Hosp Assoc*. 2009;45:72-77.

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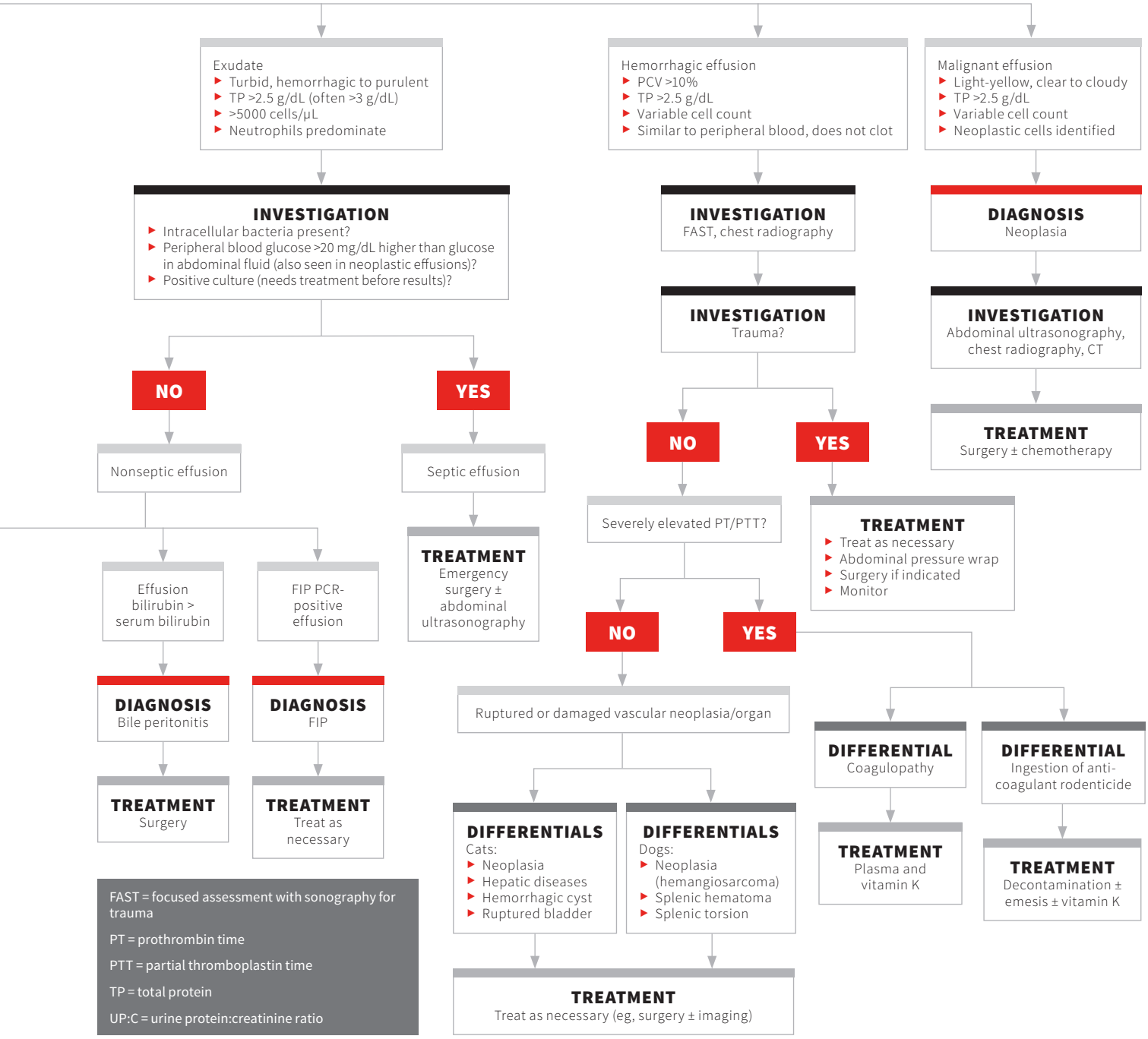
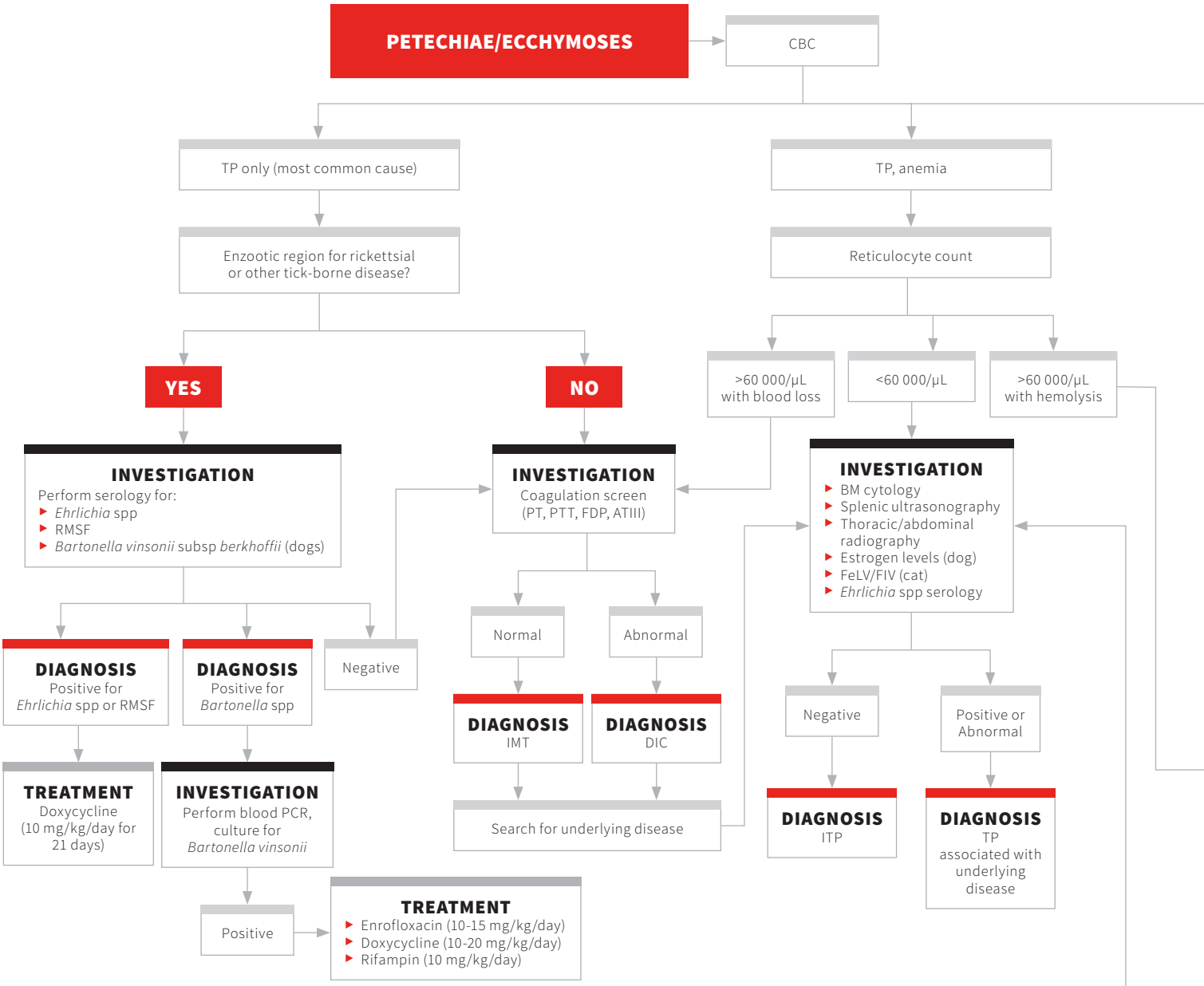
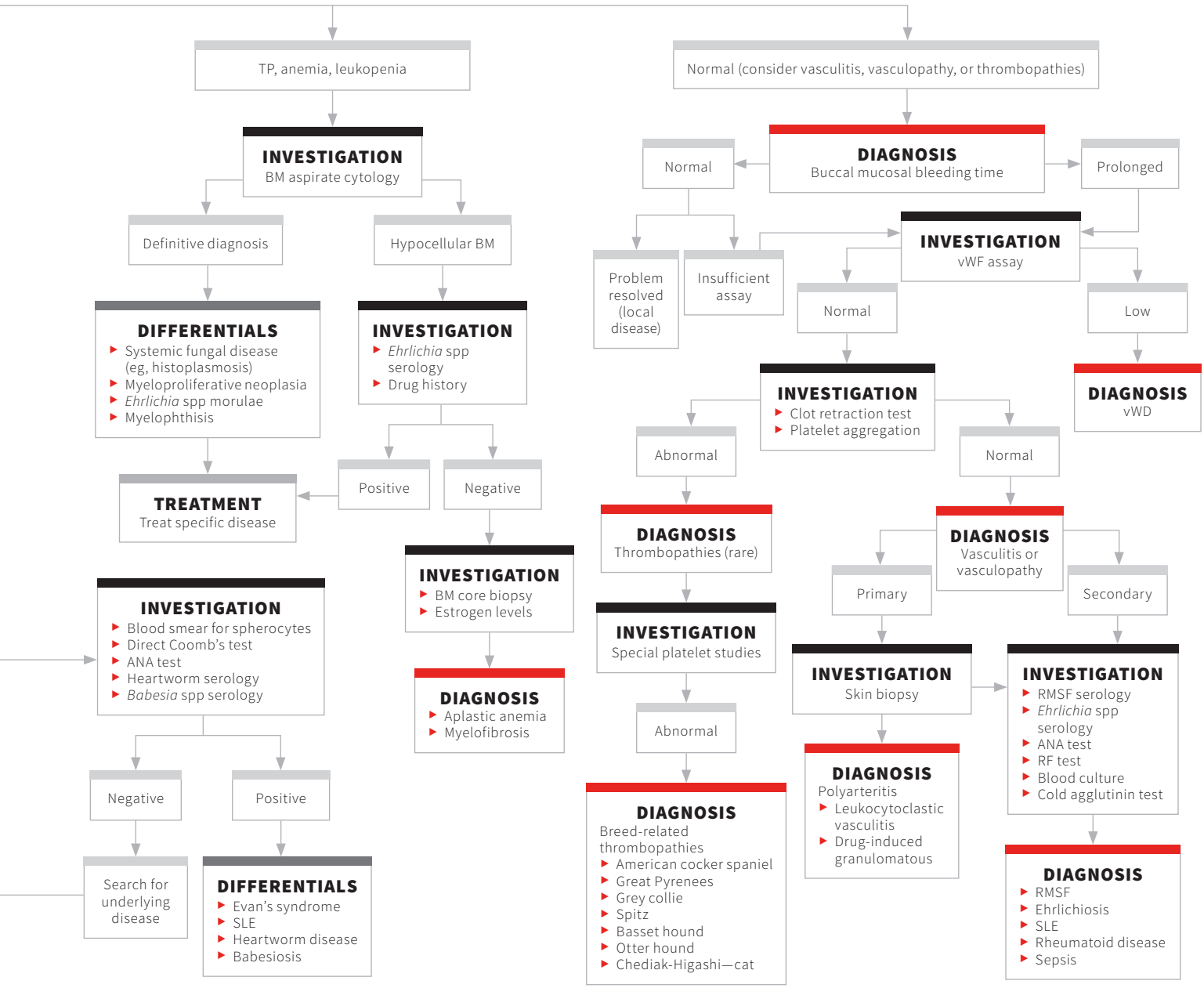


PETECHIAE & ECCHYMOSES*

T. Mark Neer, DVM, DACVIM†



ANA = antinuclear antibodies
ATIII = antithrombin III
BM = bone marrow
DIC = disseminated intravascular coagulation
FDP = fibrin degradation product
IMT = immune-mediated thrombocytopenia
ITP = idiopathic thrombocytopenia
PT = prothrombin time
PTT = partial thromboplastin time
RF = rheumatoid factor
RMSF = Rocky Mountain spotted fever
SLE = systemic lupus erythematosus
TP = thrombocytopenia
vWD = von Willebrand's disease
vWF = von Willebrand's factor



*Article originally published as "Petechiation/Ecchymoses"
†Byline reflects author information on original publication. On publication of this collection, T. Mark Neer is retired after a successful tenure at Oklahoma State University.

PTYALISM & PSEUDOPTYALISM

Julie Allen, BVMS, MS, DACVIM (SAIM)*
Antech Diagnostics

DROOLING†

- Ptyalism‡ (ie, excessive saliva production)
- Pseudoptyalism‡ (ie, inability to swallow normal saliva production; drooling)

INVESTIGATION

History and examination:
- Complete physical examination, evaluating salivary gland size and symmetry
- Full neurologic examination
- Breed: giant breeds (eg, St. Bernard) or Yorkshire and Maltese terriers (increased PSS incidence)
- Age: young animals likely to ingest toxins/FBs or have congenital issues; neoplasia likely in older animals
- History of vaccination (eg, rabies, calicivirus) or trauma (eg, electric cord injury)
- Exposure to toxins, medications, topical products
- Duration: acute (eg, FB) vs chronic (eg, neoplasm)
- Discoloration of saliva (eg, blood, purulent discharge) suggestive of oral problem
- Halitosis may indicate oral, esophageal, or gastric disease
- Pawing at face or mouth may indicate orofacial pain, hypocalcemia
- Change in eating behavior: dropping food, chewing on one side of the mouth, pseudoanorexia or hyporexia
- Diet (eg, high-protein) can cause drooling because of precipitation of HE in patients with liver dysfunction
- Other GI (ie, retching, regurgitation, vomiting, weight loss) or neurologic (ie, seizures, gagging, dysphagia) signs

- Known toxin
- Known medication exposure
- Oral cavity abnormal

DIFFERENTIAL

Toxicity
- Household cleaners
- Plants or trees (eg, Kentucky coffee tree, poinsettia)
- Insecticide or pesticide (eg, boric acid, aldicarb)
- Rodenticide (eg, zinc phosphide)
- Illicit drugs (eg, cocaine, amphetamines)
- Animal venoms (eg, black widow spider, scorpion, toads [*Bufo* spp], coral snake, sea hare [*Aplysia* spp])
- Human sleep aids (eg, zolpidem)
- Mushrooms (eg, *Amanita muscaria*)
- Metaldehyde
- Human tricyclic antidepressants (eg, clozapine)
- 5-hydroxytryptophan (ie, Griffonia seed extract)

TREATMENT

- Depends on toxin exposure
- Dilution if caustic agent
- Decontamination (eg, activated charcoal)
- Supportive care
- Specific antidote (if available)

DIFFERENTIAL

Medication reaction
- Medications or topical products given topically or PO (eg, selamectin, moxidectin–imidacloprid)
- Cholinergic drugs (eg, bethanechol), anticholinesterase drugs (eg, pyridostigmine), cholinesterase inhibitors (eg, OP)
- Pyrethrins or pyrethroids
- Ivermectin
- Bitter drugs

TREATMENT

- Discontinue medication
- Supportive care

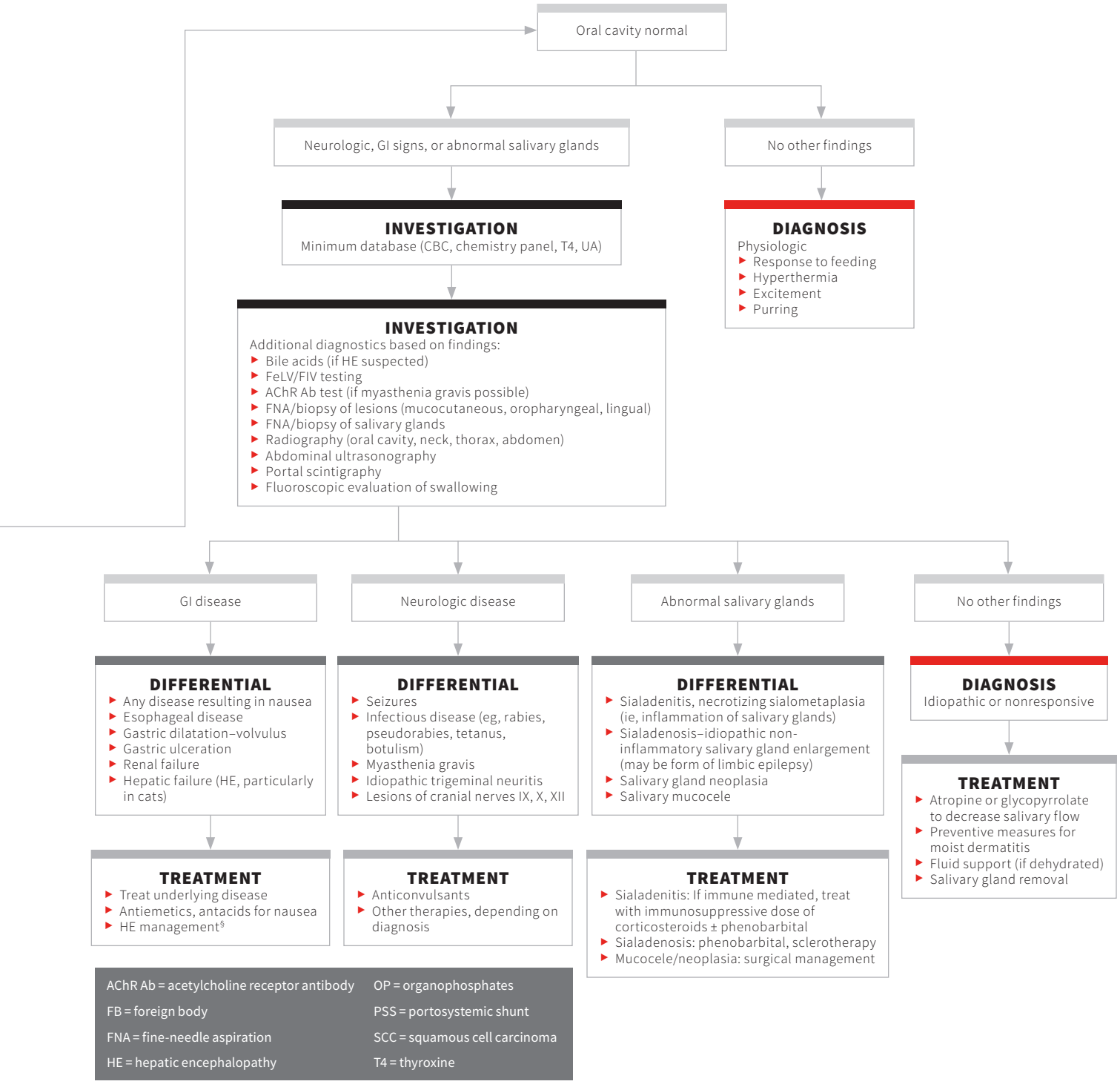
DIFFERENTIAL

- Periodontal disease
- Stomatitis (eg, calicivirus, FeLV/FIV, caustic agent)
- Immune-mediated disease (eg, pemphigus)
- Tongue lesion (eg, linear FB), glossitis (eg, uremia, caustic agent), tumor
- Oropharyngeal disease (eg, tonsillar SCC)
- Lip fold abnormalities
- Faucitis

TREATMENT

- Depends on underlying disease
- Dental cleaning or extractions
- Anti-inflammatory or immunomodulatory therapy
- Surgical resection or correction

*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Julie Allen, BVMS, MS, MRCVS, DACVIM (SAIM), DACVP, at Veterinary Information Network.
†Rabies should always be considered in patients presenting with drooling.
‡Distinction between ptyalism and pseudoptyalism is not absolute; oropharyngeal and CNS diseases can result in increased salivary production and inability to swallow.
§HE management includes low-protein diet, enemas, oral antibiotics, lactulose, zinc supplementation, and supportive care.



AChR Ab = acetylcholine receptor antibody
FB = foreign body
FNA = fine-needle aspiration
HE = hepatic encephalopathy
OP = organophosphates
PSS = portosystemic shunt
SCC = squamous cell carcinoma
T4 = thyroxine

REFRACTORY CANINE IMMUNE-MEDIATED HEMOLYTIC ANEMIA

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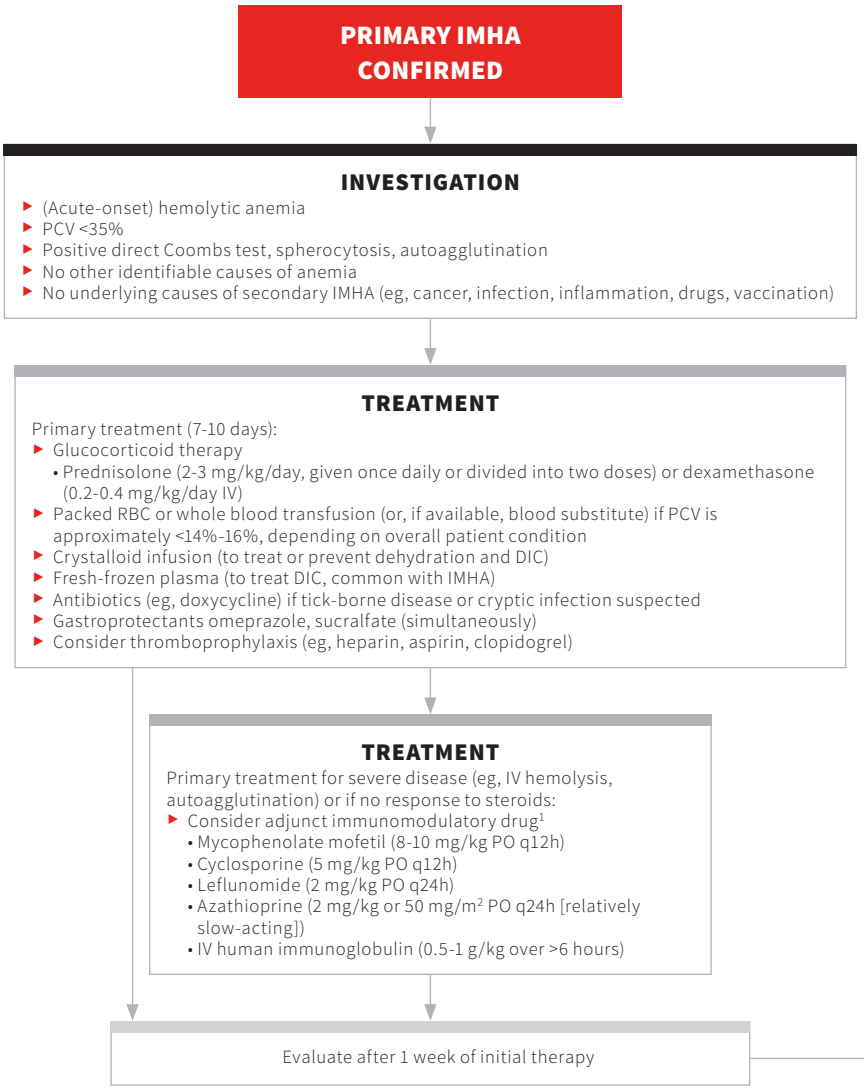
Barbara Kohn, Dr med vet, PD, Prof, DECVIM
Freie Universität Berlin



DIC = disseminated intravascular coagulation
IMHA = immune-mediated hemolytic anemia
PCV = packed cell volume

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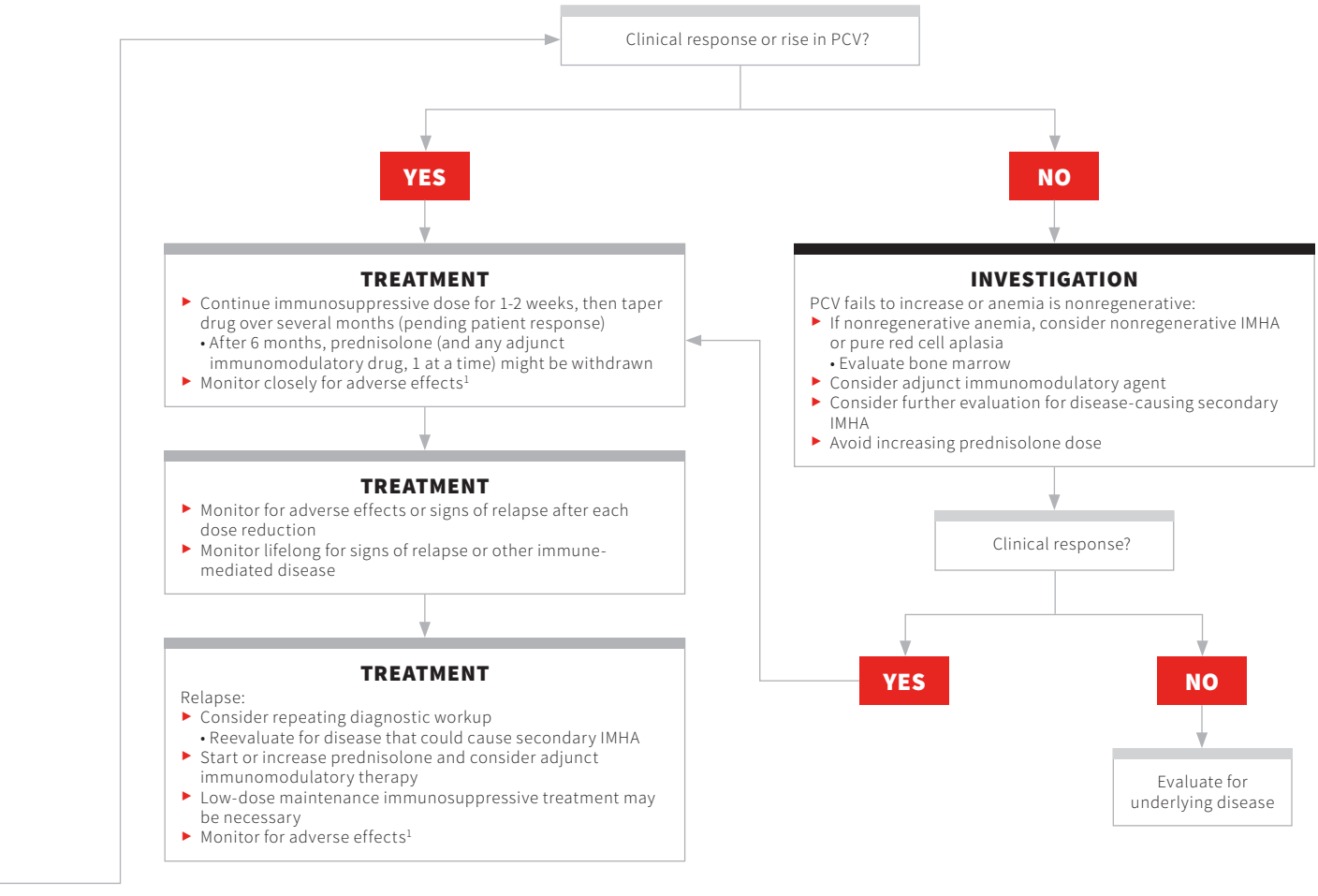

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Suggested Reading

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HORNER SYNDROME

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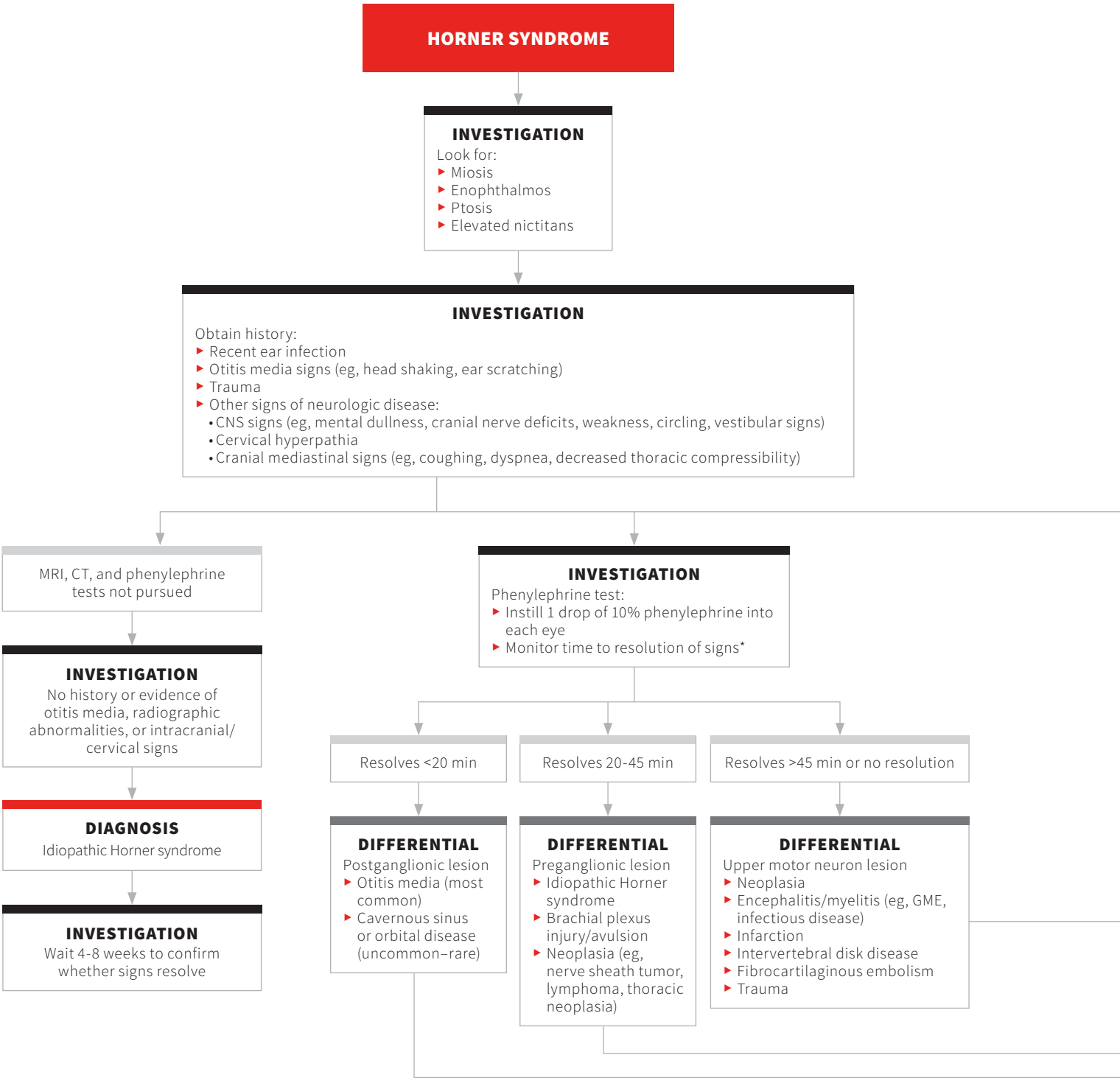
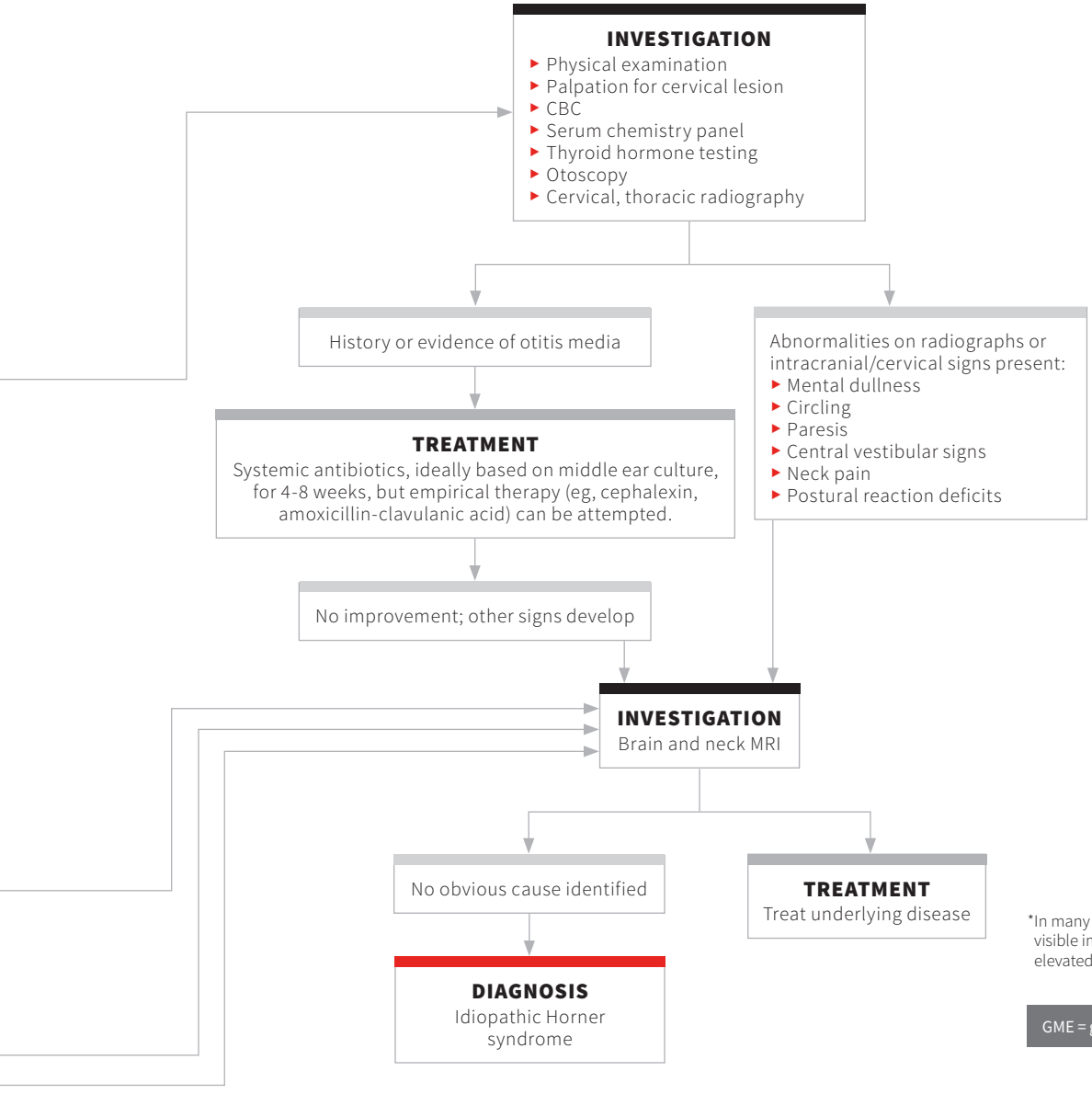


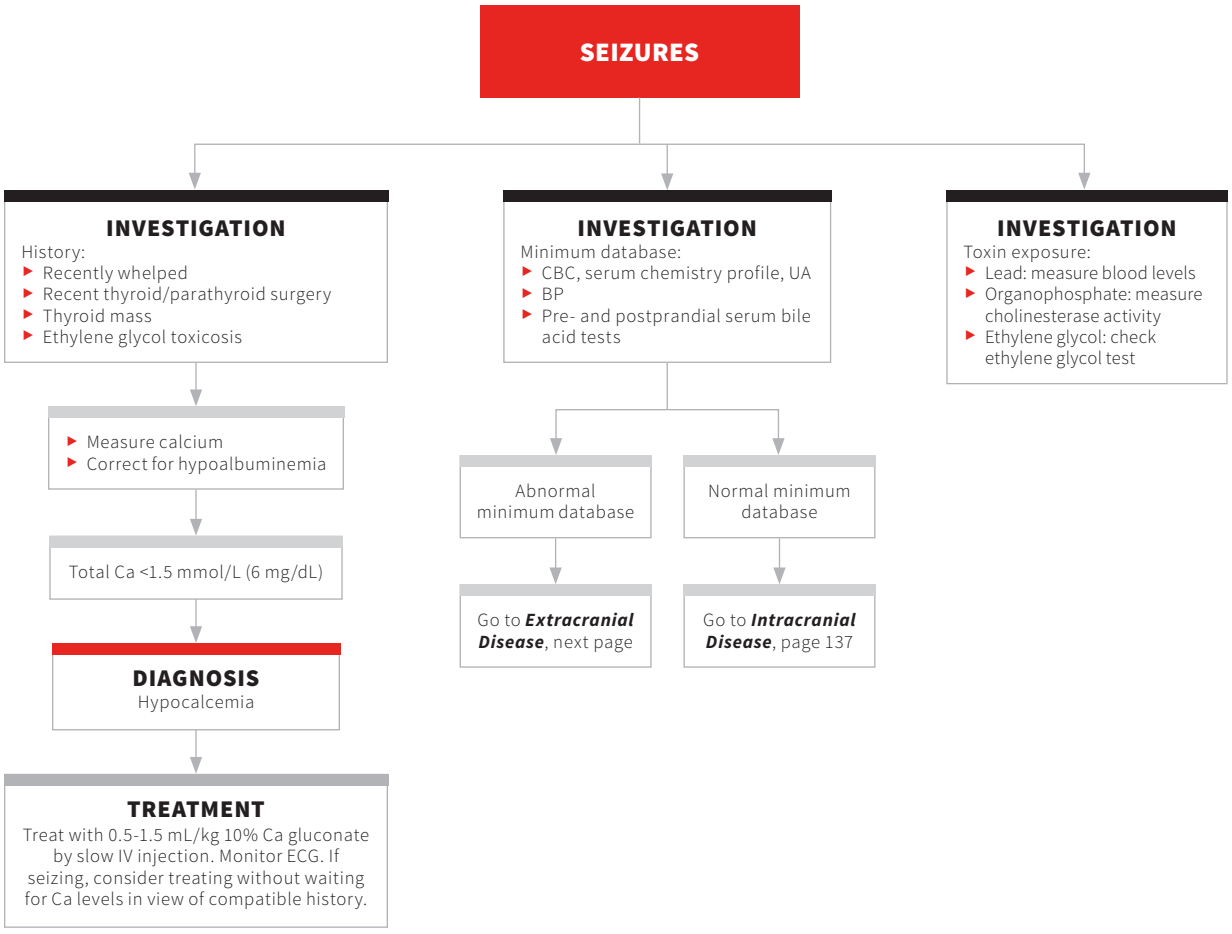


*In many cases, time to resolution is determined by visible improvement of enophthalmos, ptosis, and elevated nictitans. Miosis may not resolve.

GME = granulomatous meningoencephalitis

INTRACRANIAL VS EXTRACRANIAL SEIZURES

Natasha Olby, VetMB, PhD, DACVIM (Neurology)
North Carolina State University



BP = blood pressure
Ca = calcium
UA = urinalysis

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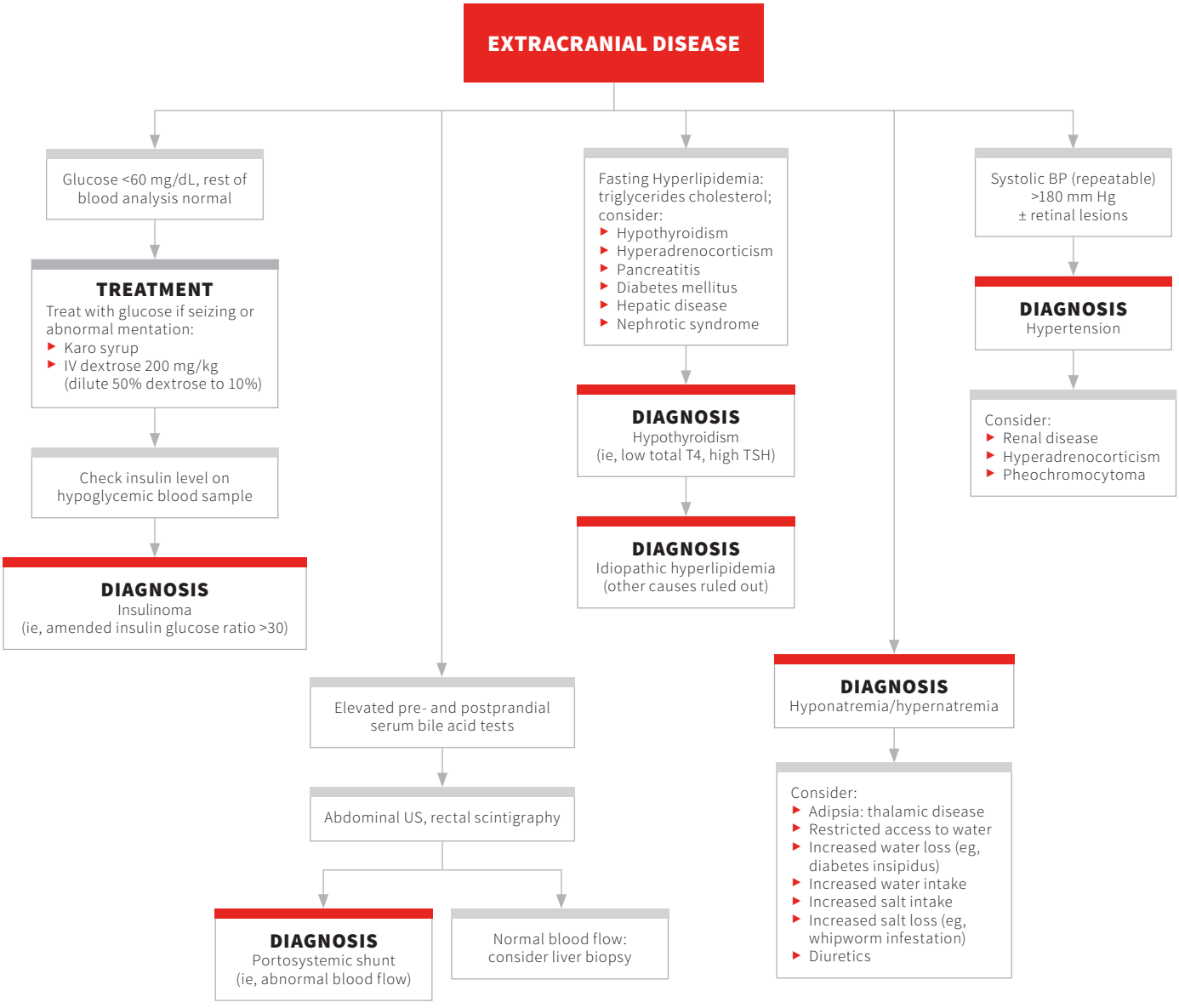
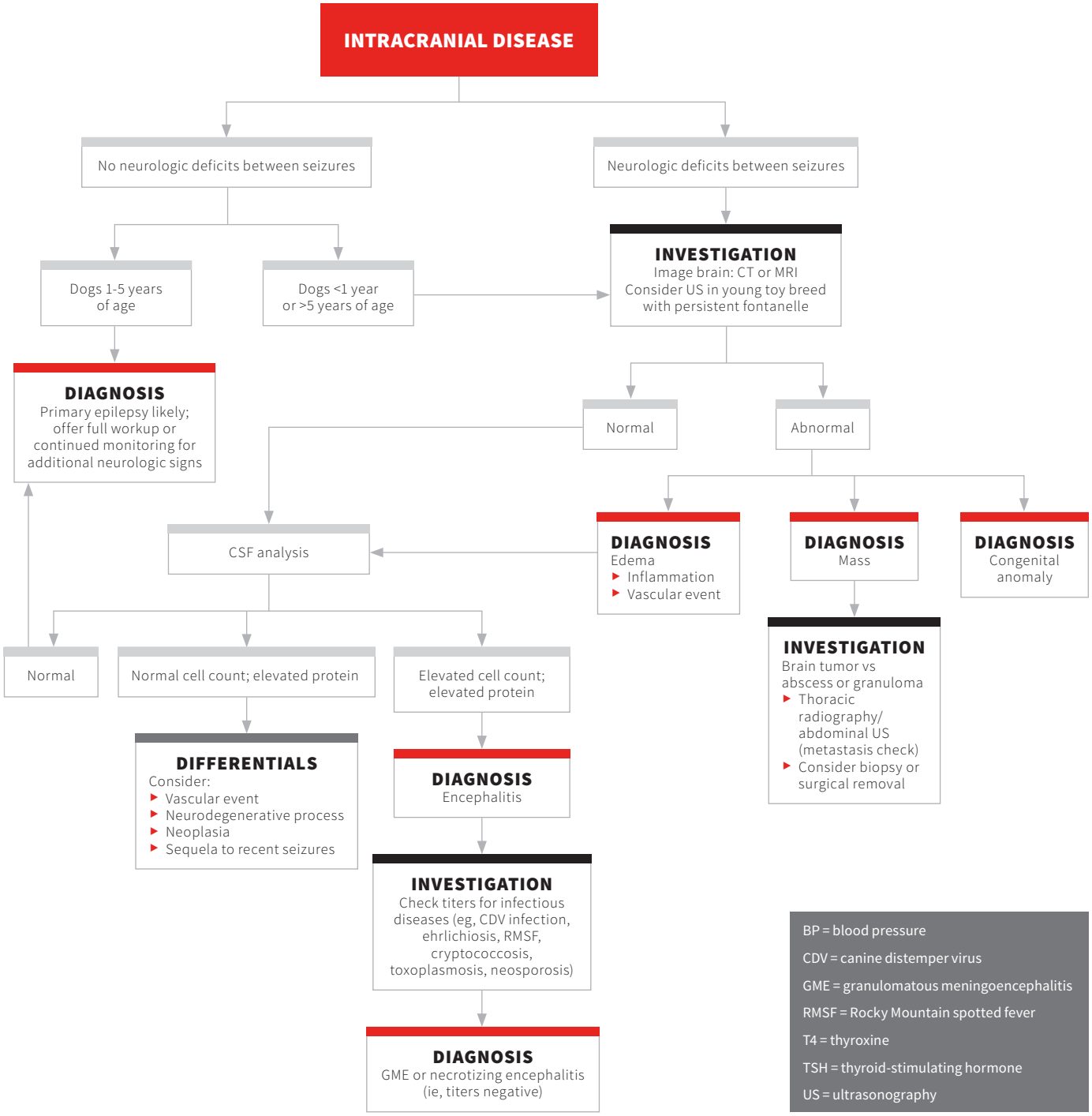
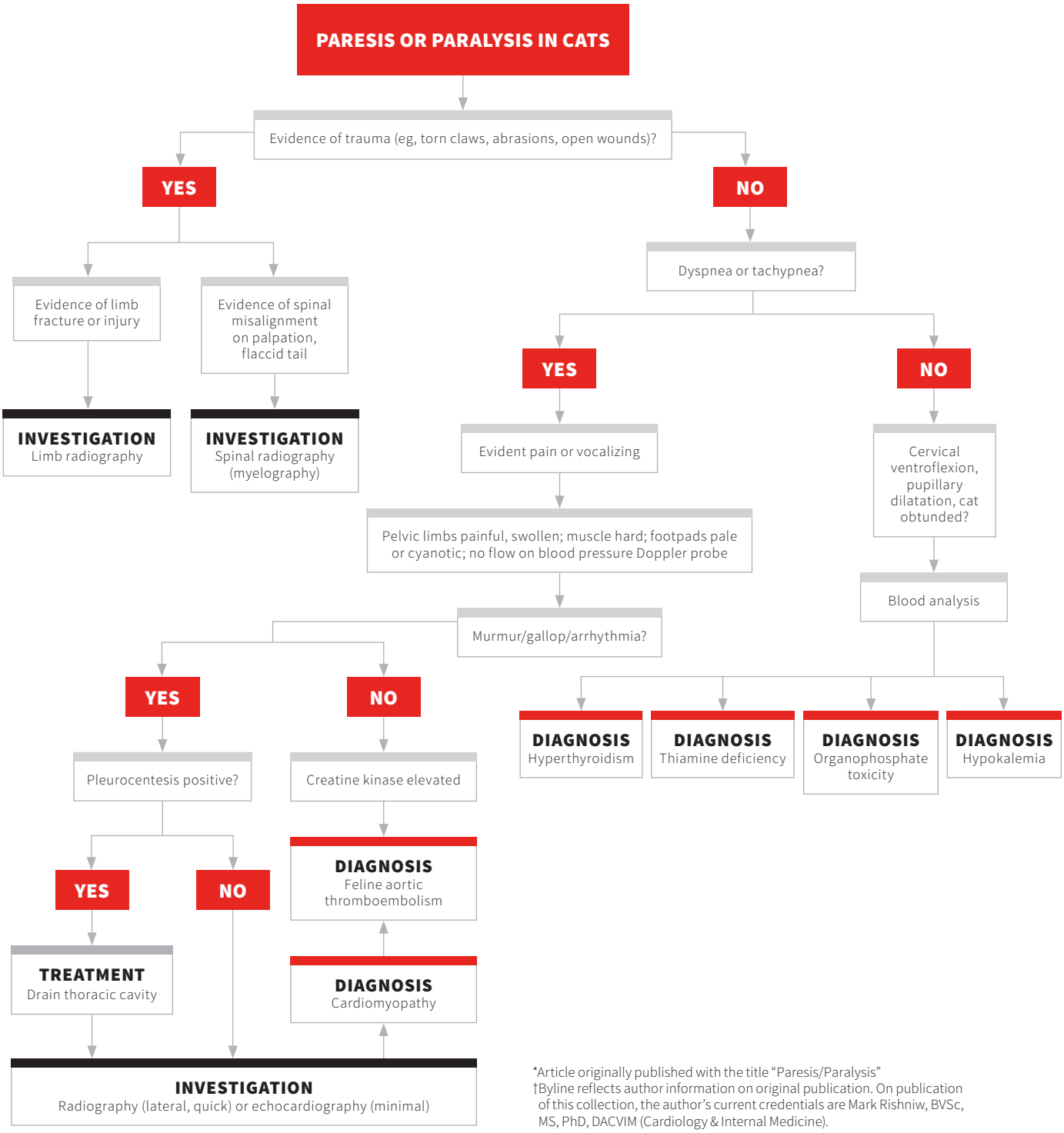

EXTRACRANIAL DISEASE
Glucose <60 mg/dL, rest of blood analysis normal
TREATMENT
Treat with glucose if seizing or abnormal mentation:
Karo syrup
IV dextrose 200 mg/kg (dilute 50% dextrose to 10%)
Check insulin level on hypoglycemic blood sample
DIAGNOSIS
Insulinoma
(ie, amended insulin glucose ratio >30)
Elevated pre- and postprandial serum bile acid tests
Abdominal US, rectal scintigraphy
DIAGNOSIS
Portosystemic shunt
(ie, abnormal blood flow)
Normal blood flow: consider liver biopsy
Fasting Hyperlipidemia: triglycerides cholesterol; consider:
Hypothyroidism
Hyperadrenocorticism
Pancreatitis
Diabetes mellitus
Hepatic disease
Nephrotic syndrome
DIAGNOSIS
Hypothyroidism
(ie, low total T4, high TSH)
DIAGNOSIS
Idiopathic hyperlipidemia
(other causes ruled out)
DIAGNOSIS
Hyponatremia/hypernatremia
Consider:
Adipsia: thalamic disease
Restricted access to water
Increased water loss (eg, diabetes insipidus)
Increased water intake
Increased salt intake
Increased salt loss (eg, whipworm infestation)
Diuretics
Systolic BP (repeatable) >180 mm Hg ± retinal lesions
DIAGNOSIS
Hypertension
Consider:
Renal disease
Hyperadrenocorticism
Pheochromocytoma



BP = blood pressure
CDV = canine distemper virus
GME = granulomatous meningoencephalitis
RMSF = Rocky Mountain spotted fever
T4 = thyroxine
TSH = thyroid-stimulating hormone
US = ultrasonography

PARESIS OR PARALYSIS IN CATS*

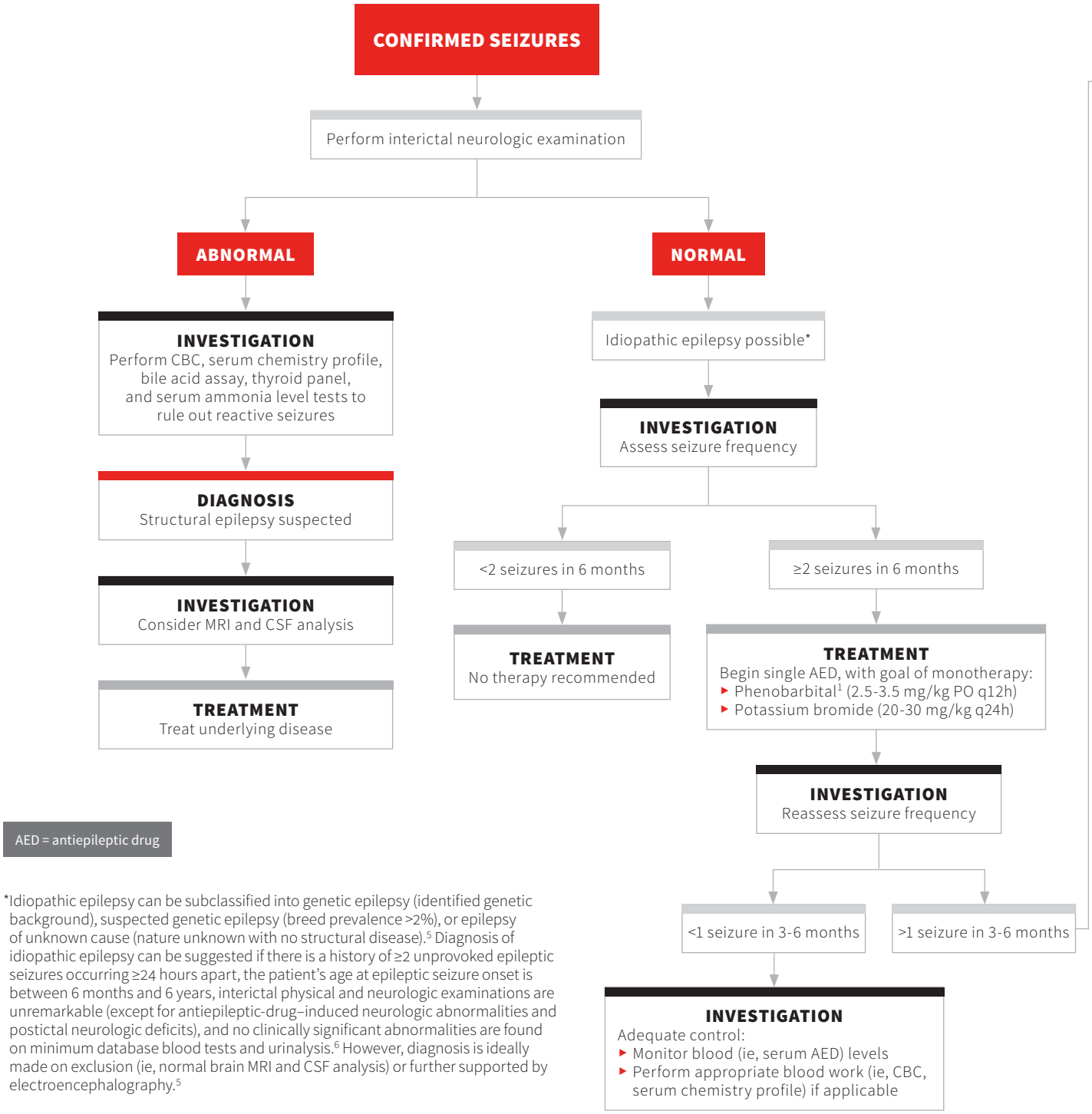
Mark Rishniw, BVSc, MS, DACVIM (Cardiology & Internal Medicine)†
Cornell University



*Article originally published with the title "Paresis/Paralysis"
†Byline reflects author information on original publication. On publication of this collection, the author's current credentials are Mark Rishniw, BVSc, MS, PhD, DACVIM (Cardiology & Internal Medicine).

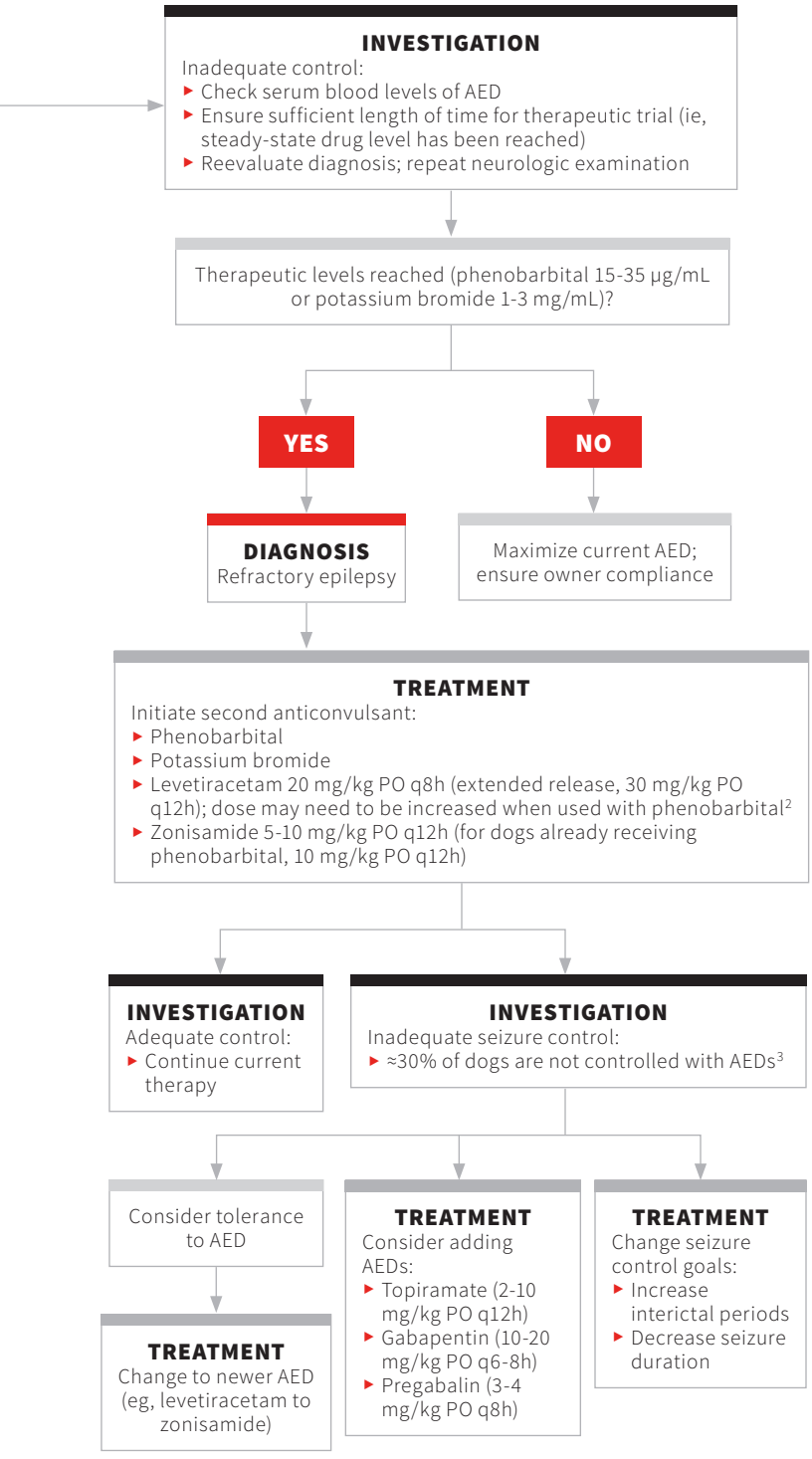
REFRACTORY SEIZURES

Hillary Haydon Greatting, DVM, MS, DACVIM
Tom Jukier, DVM
Washington State University



AED = antiepileptic drug

*Idiopathic epilepsy can be subclassified into genetic epilepsy (identified genetic background), suspected genetic epilepsy (breed prevalence >2%), or epilepsy of unknown cause (nature unknown with no structural disease).[5] Diagnosis of idiopathic epilepsy can be suggested if there is a history of ≥2 unprovoked epileptic seizures occurring ≥24 hours apart, the patient's age at epileptic seizure onset is between 6 months and 6 years, interictal physical and neurologic examinations are unremarkable (except for antiepileptic-drug–induced neurologic abnormalities and postictal neurologic deficits), and no clinically significant abnormalities are found on minimum database blood tests and urinalysis.[6] However, diagnosis is ideally made on exclusion (ie, normal brain MRI and CSF analysis) or further supported by electroencephalography.[5]



AUTHOR INSIGHT

- A definition of refractory epilepsy is not established for dogs, but it is generally agreed that an animal with frequent or severe seizures or intolerable side effects despite appropriate antiepileptic drug (AED) therapy is considered refractory to treatment.[3]
- Levetiracetam and zonisamide are increasingly the drugs of choice for monotherapy by some neurologists. A recent study found no reduction in monthly seizure frequency when levetiracetam was used as a sole agent.[4] However, little additional information is available in the veterinary literature on the efficacy of these medications as sole agents.
- Some AEDs (eg, levetiracetam, zonisamide) have a reported "honeymoon effect," with dogs developing tolerance over time.
- Gabapentin and pregabalin are not known efficacious AEDs but anecdotally may help with seizure control in patients tolerant to other medications.

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SPINAL PAIN

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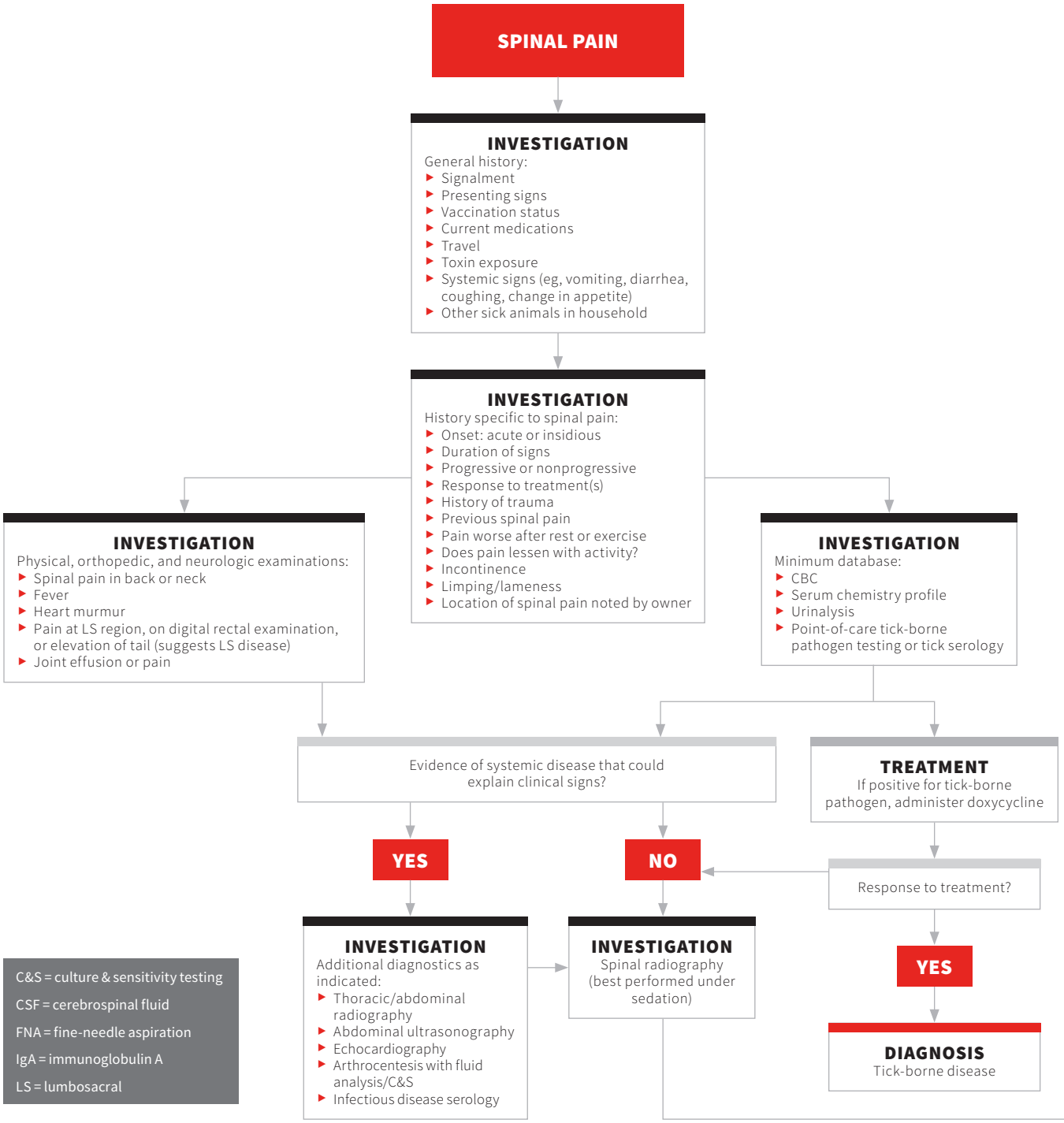
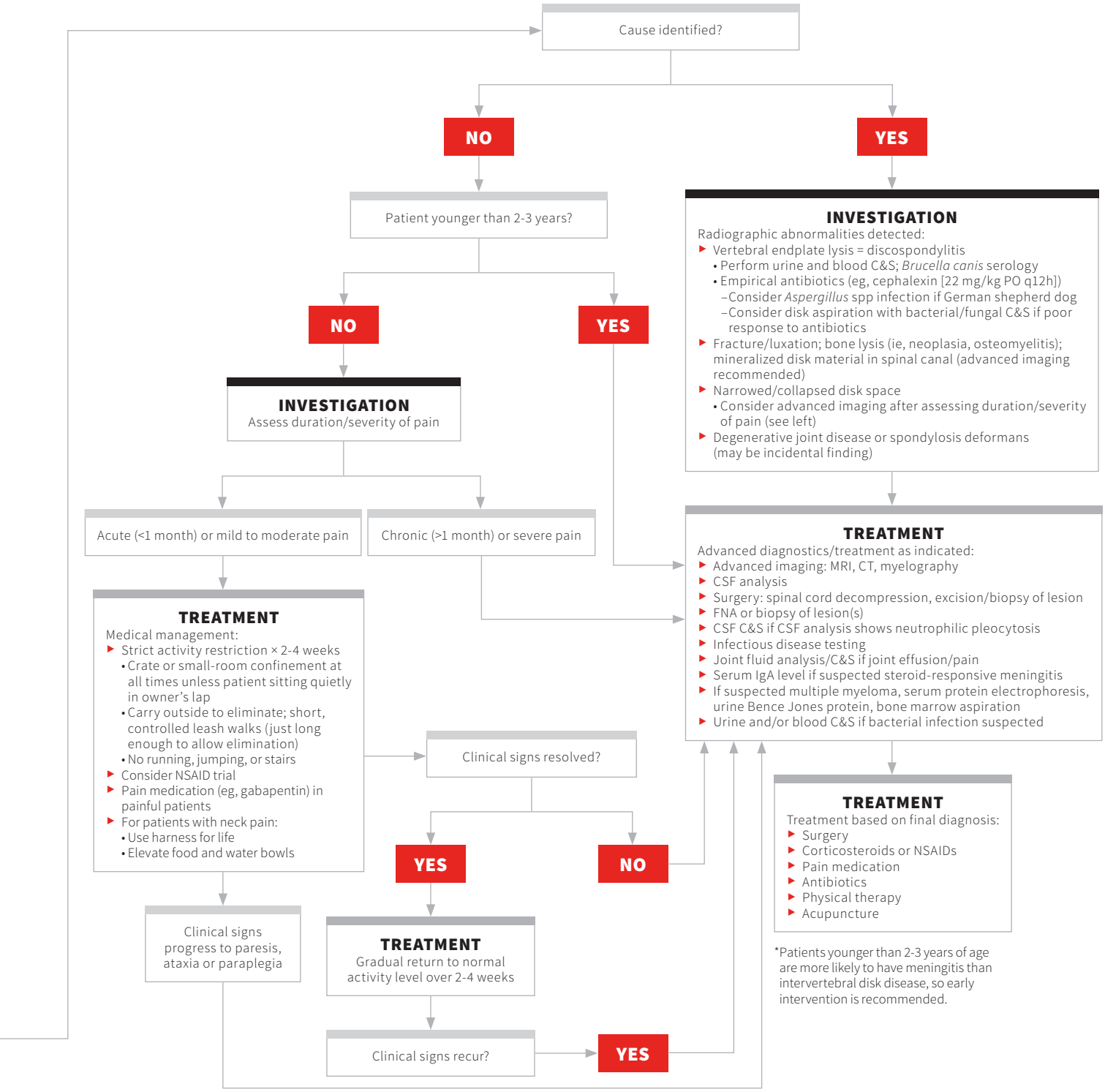
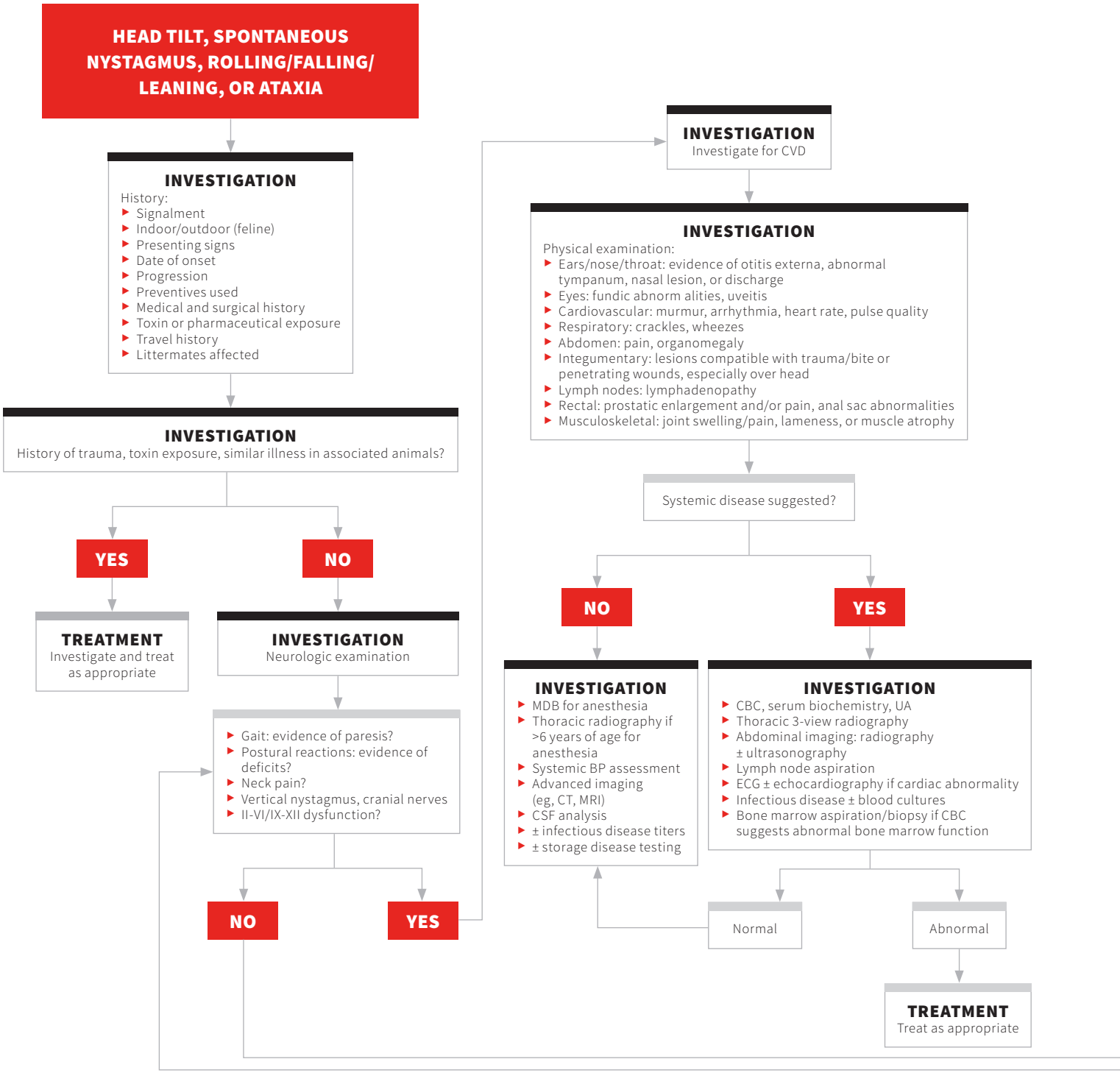


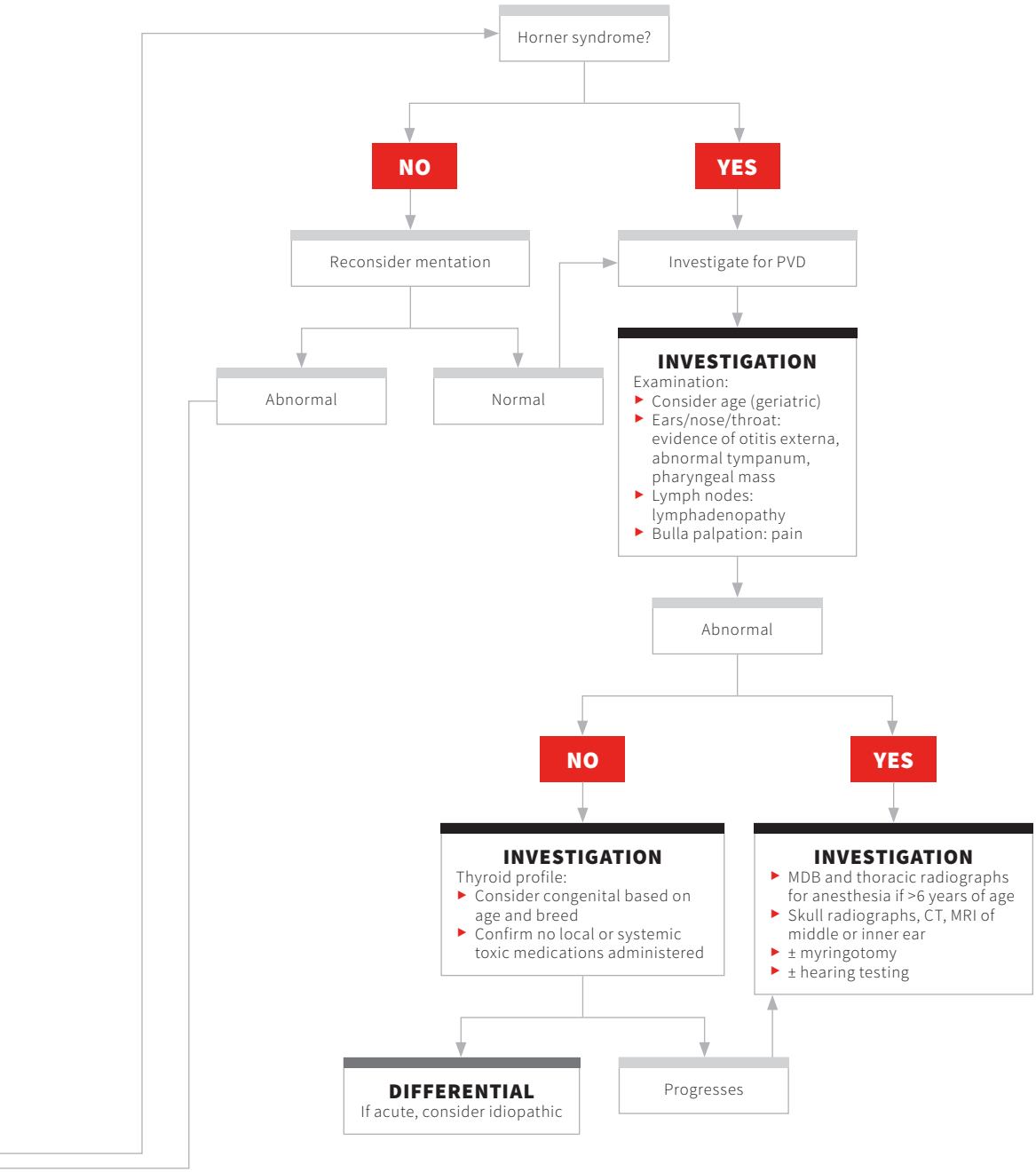


*Patients younger than 2-3 years of age are more likely to have meningitis than intervertebral disk disease, so early intervention is recommended.

VESTIBULAR DISEASE

Simon R. Platt, BVMS, MRCVS, DACVIM (Neurology), DECVN*
University of Georgia



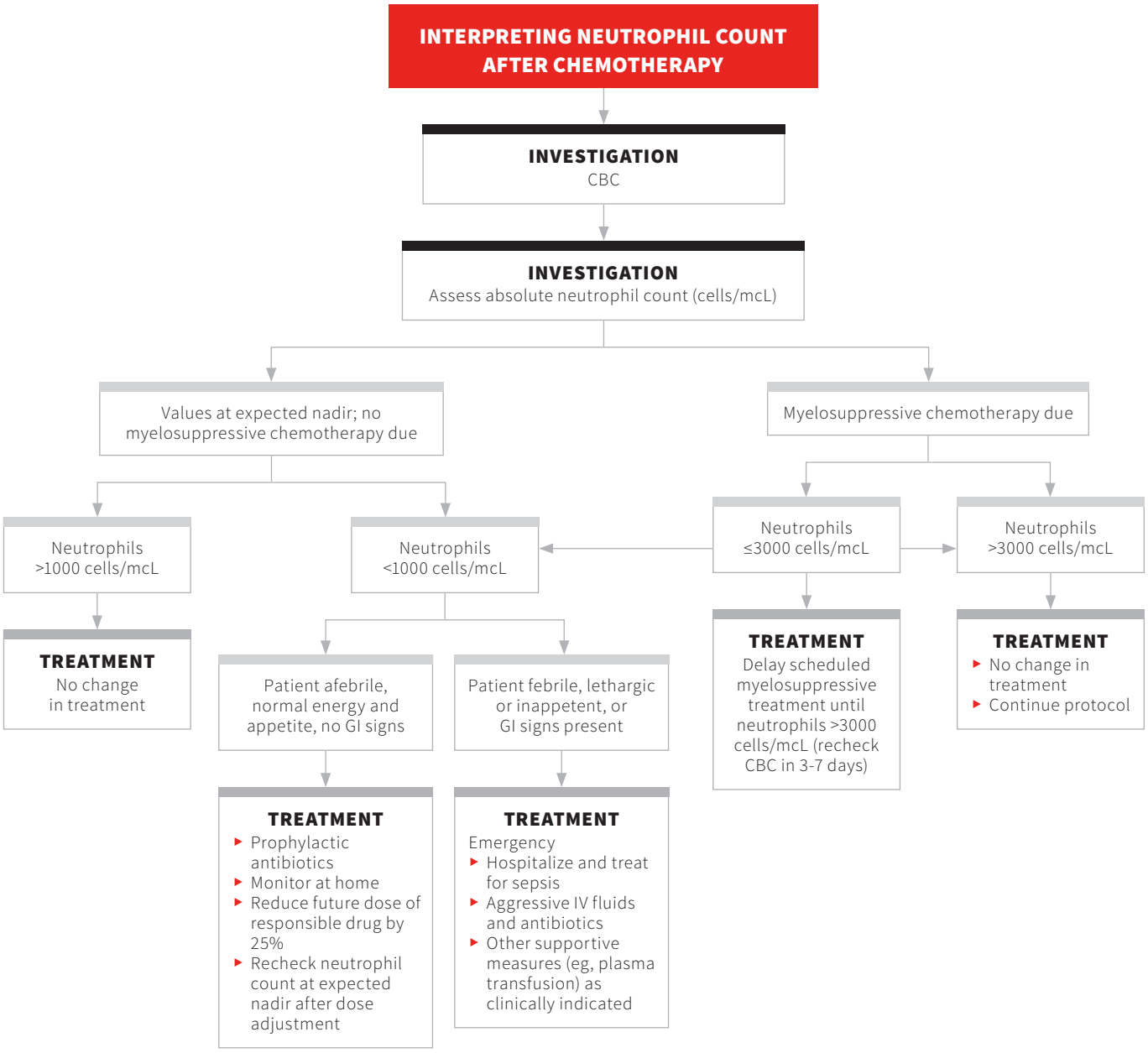


*Byline reflects author information on original publication. On publication of this collection, the author's current credentials are Simon R. Platt, BVMS, MRCVS, DACVIM (Neurology), DECVN, FRCVS.

BP = blood pressure
CVD = central vestibular disease
MDB = minimum database
PVD = peripheral vestibular disease
UA = urinalysis

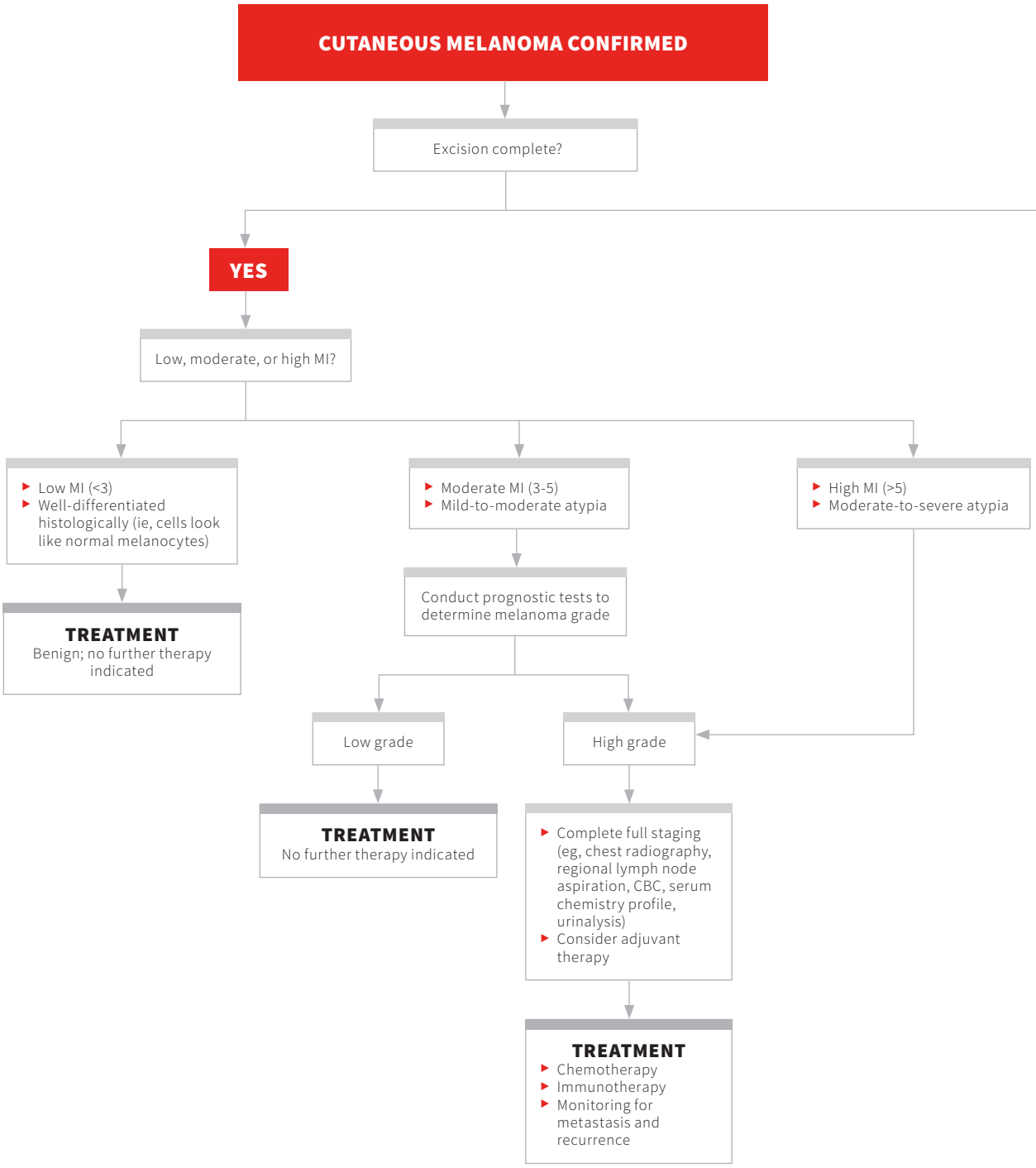
INTERPRETING NEUTROPHIL COUNT AFTER CHEMOTHERAPY

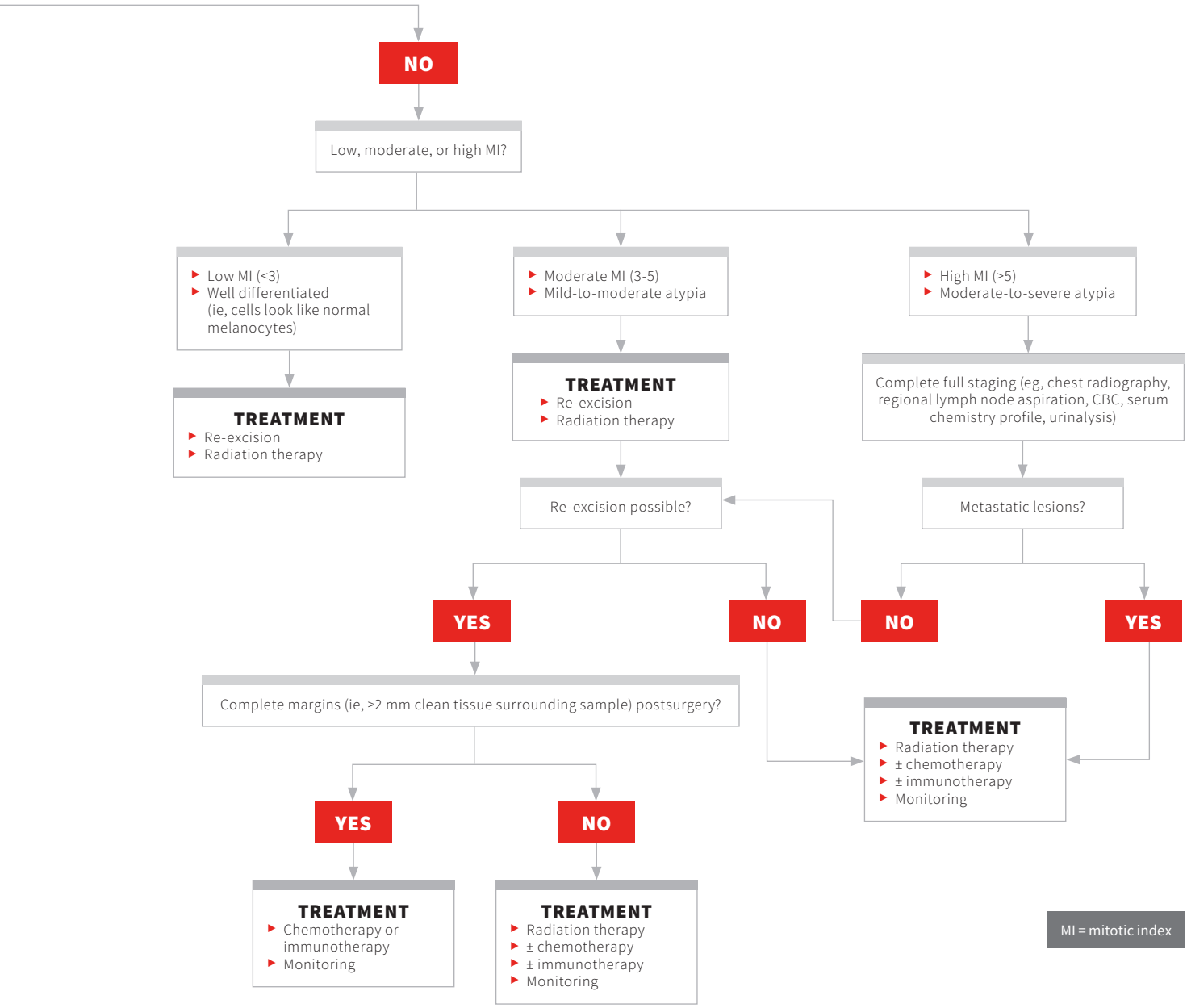
Antony S. Moore, BVSc, MVSc, DACVIM (Oncology)
Angela E. Frimberger, VMD, DACVIM (Oncology)
Veterinary Oncology Consultants
Wauchope, New South Wales, Australia



MANAGEMENT OF CUTANEOUS MELANOMA

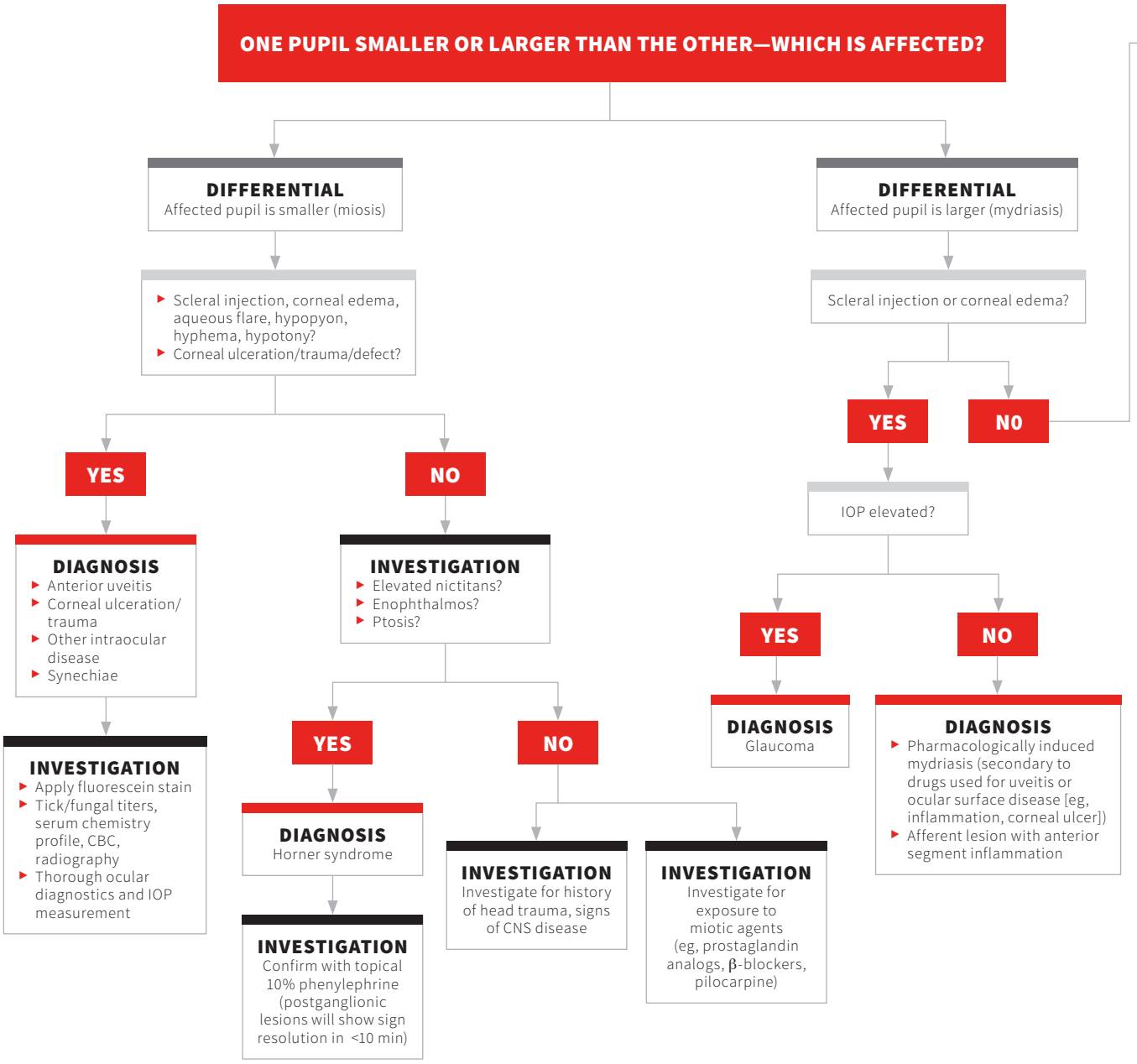
Heather M. Wilson-Robles, DVM, DACVIM (Oncology)
Texas A&M University



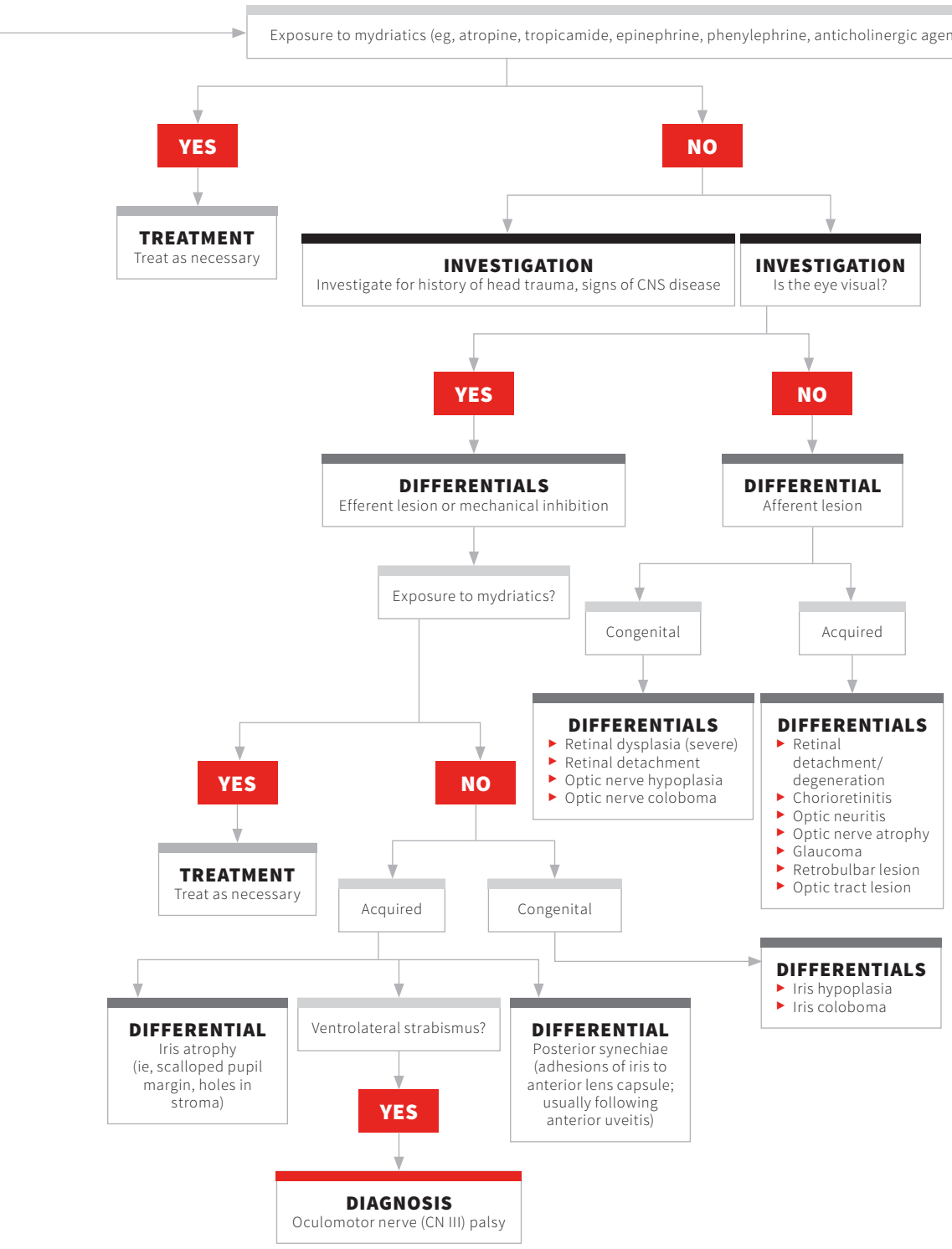

NO
Low, moderate, or high MI?
Low MI (<3)
Well differentiated (ie, cells look like normal melanocytes)
TREATMENT
Re-excision
Radiation therapy
Moderate MI (3-5)
Mild-to-moderate atypia
TREATMENT
Re-excision
Radiation therapy
Re-excision possible?
YES
NO
Complete margins (ie, >2 mm clean tissue surrounding sample) postsurgery?
YES
NO
TREATMENT
Chemotherapy or immunotherapy
Monitoring
TREATMENT
Radiation therapy
± chemotherapy
± immunotherapy
Monitoring
High MI (>5)
Moderate-to-severe atypia
Complete full staging (eg, chest radiography, regional lymph node aspiration, CBC, serum chemistry profile, urinalysis)
Metastatic lesions?
NO
YES
TREATMENT
Radiation therapy
± chemotherapy
± immunotherapy
Monitoring
MI = mitotic index

ANISOCORIA

Caryn E. Plummer, DVM, DACVO
University of Florida



CN III = third cranial nerve (ie, oculomotor nerve)
IOP = intraocular pressure



PUPILLARY TESTING: DETERMINING WHICH PUPIL IS AFFECTED

Anisocoria (ie, asymmetric pupils at rest, potentially caused by ocular or neurologic disorders or pharmacologic intervention) may not be apparent at outset. Pupil response to light and dark stimulation should be checked:

- In ambient lighting, use a dim light source held at least arm's distance from the patient to visualize the tapetal reflection, which will delineate pupil size.
- Darken the room and using the light source to visualize the tapetal reflection, evaluate the degree of dilation in each pupil: both pupils should dilate in darkened conditions; one pupil failing to dilate indicates the affected eye.
- Stimulate each eye separately with a bright light: both pupils should constrict in response to bright light; one pupil failing to constrict fully indicates the affected eye.

Suggested Reading

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ANTERIOR UVEITIS

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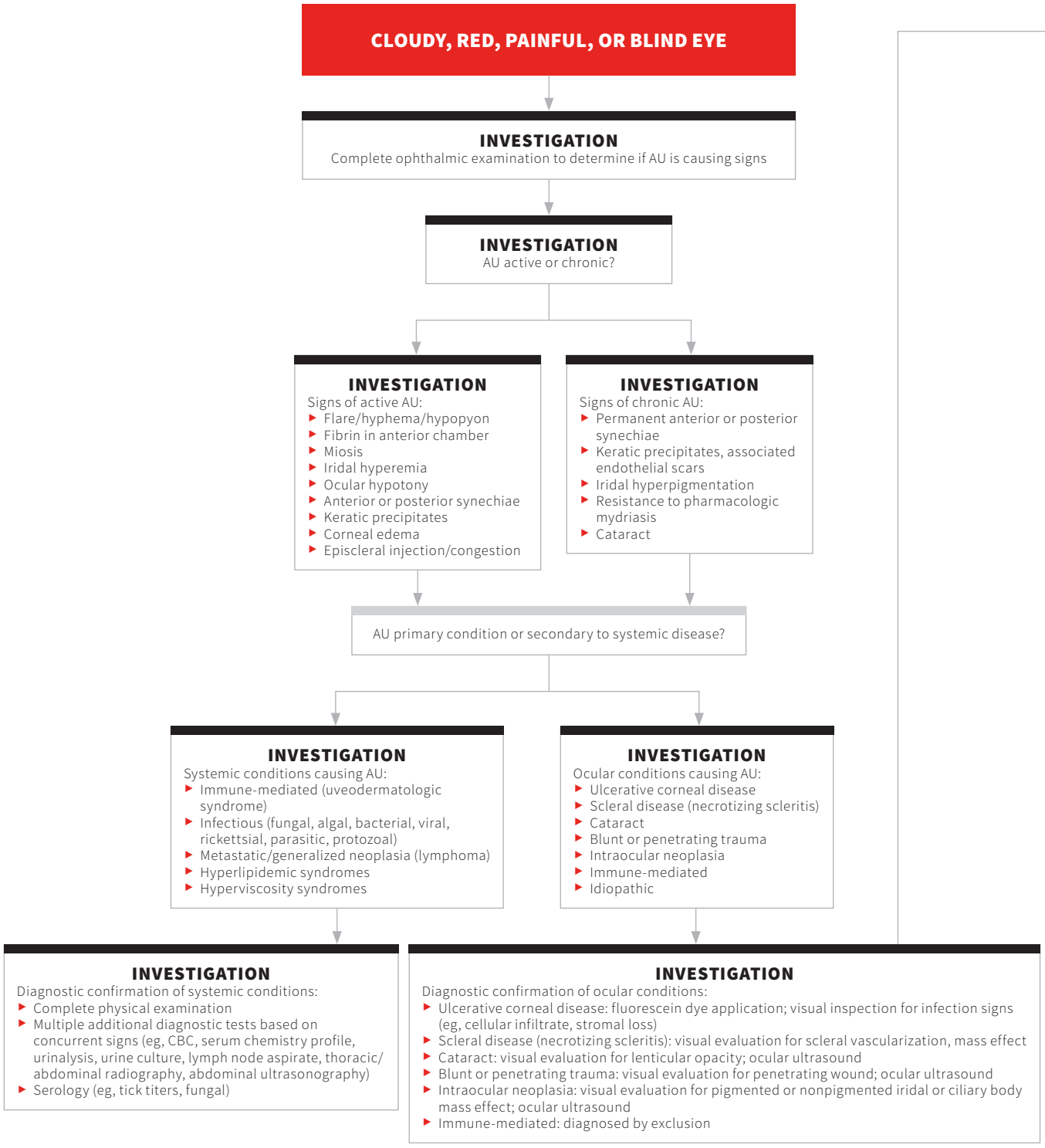
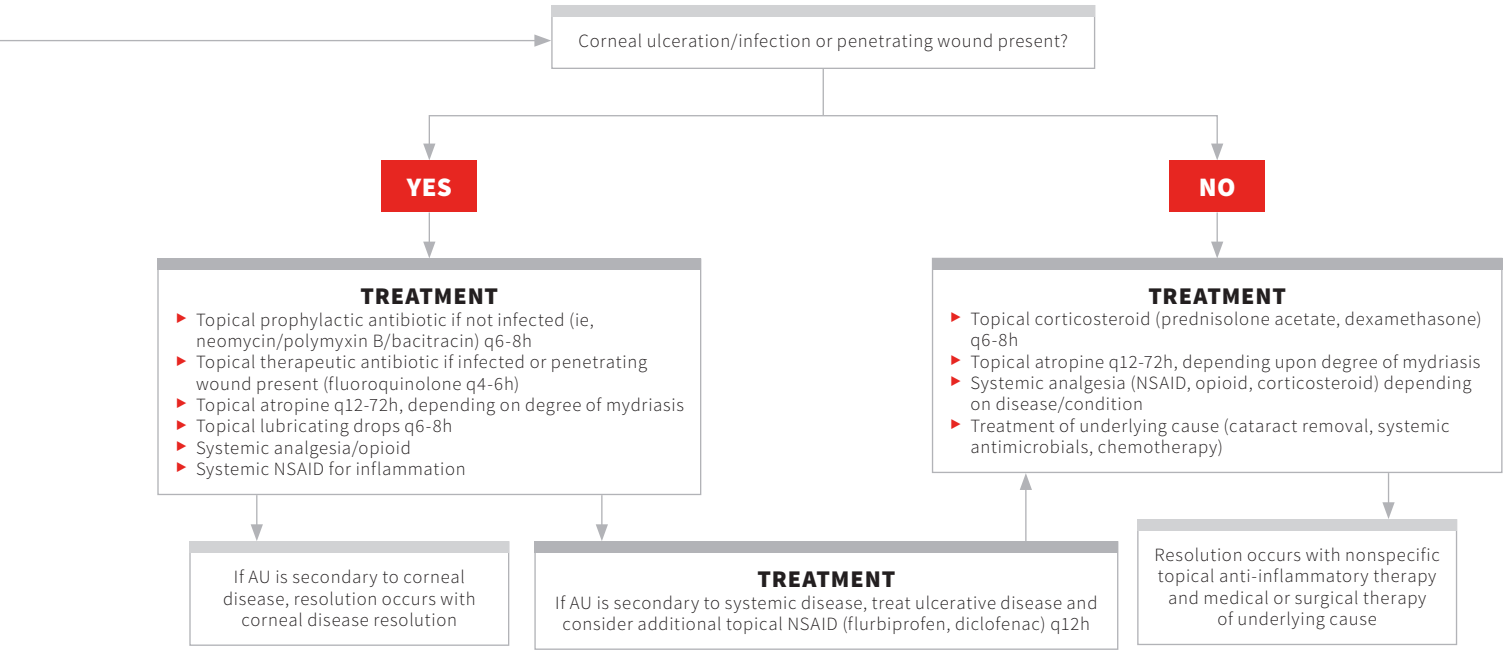
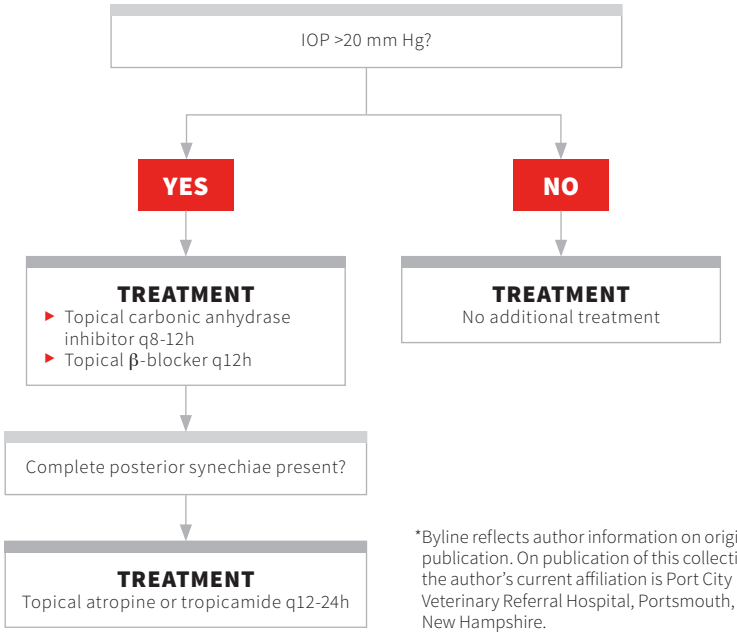
North Carolina State University





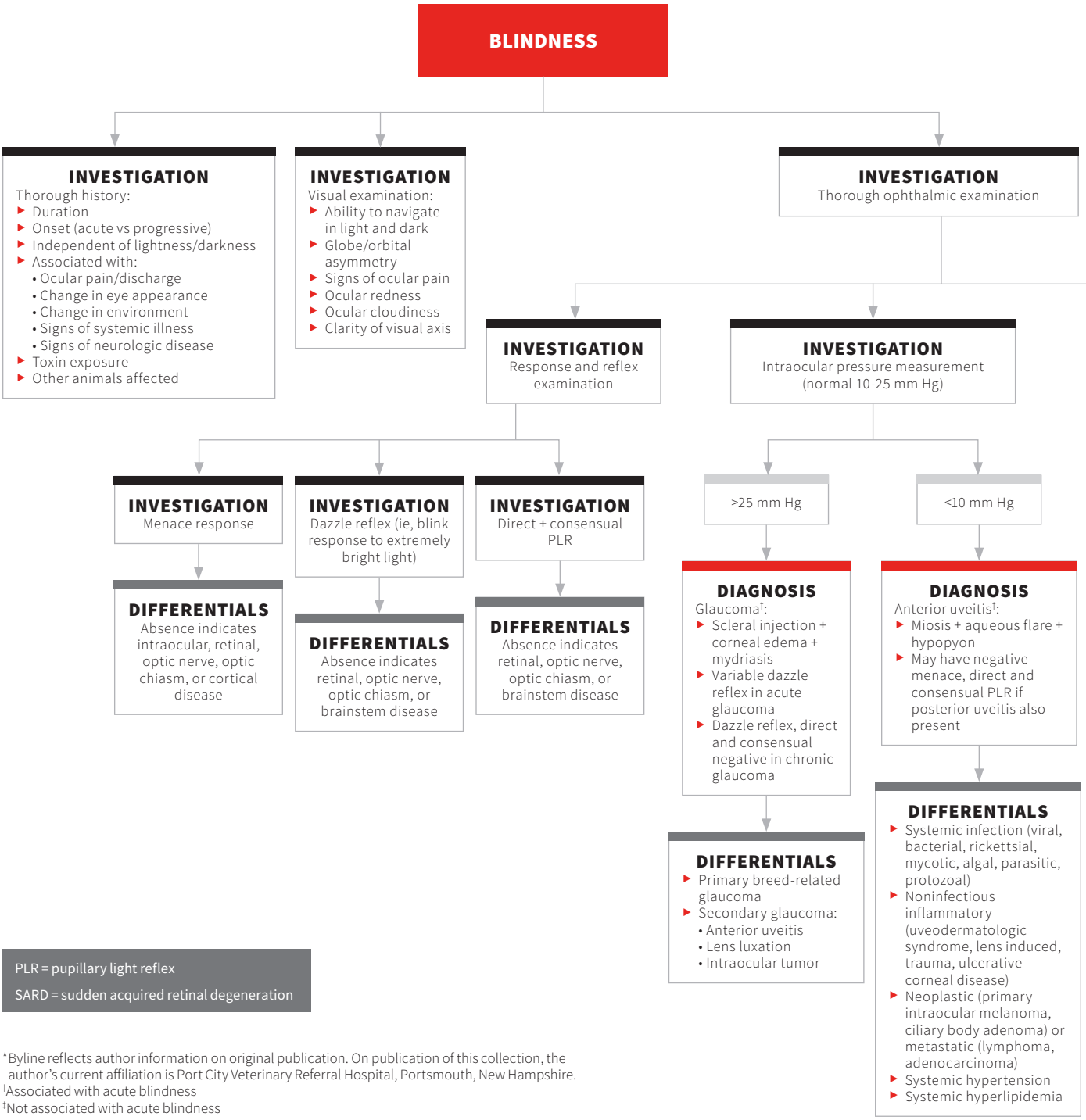


*Byline reflects author information on original publication. On publication of this collection, the author's current affiliation is Port City Veterinary Referral Hospital, Portsmouth, New Hampshire.

AU = anterior uveitis
IOP = intraocular pressure

BLINDNESS

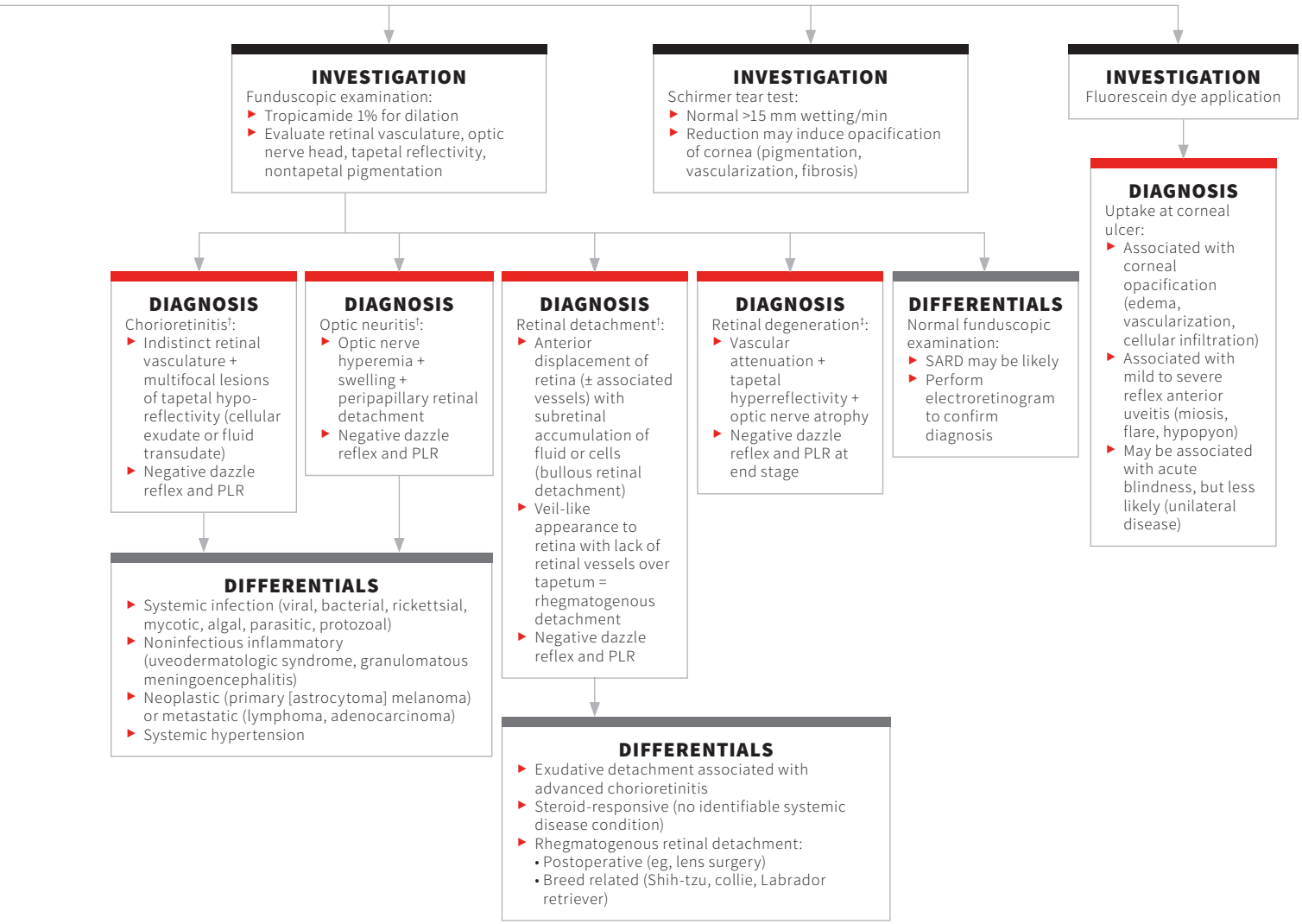
Alison Clode, DVM, DACVO*
North Carolina State University



PLR = pupillary light reflex

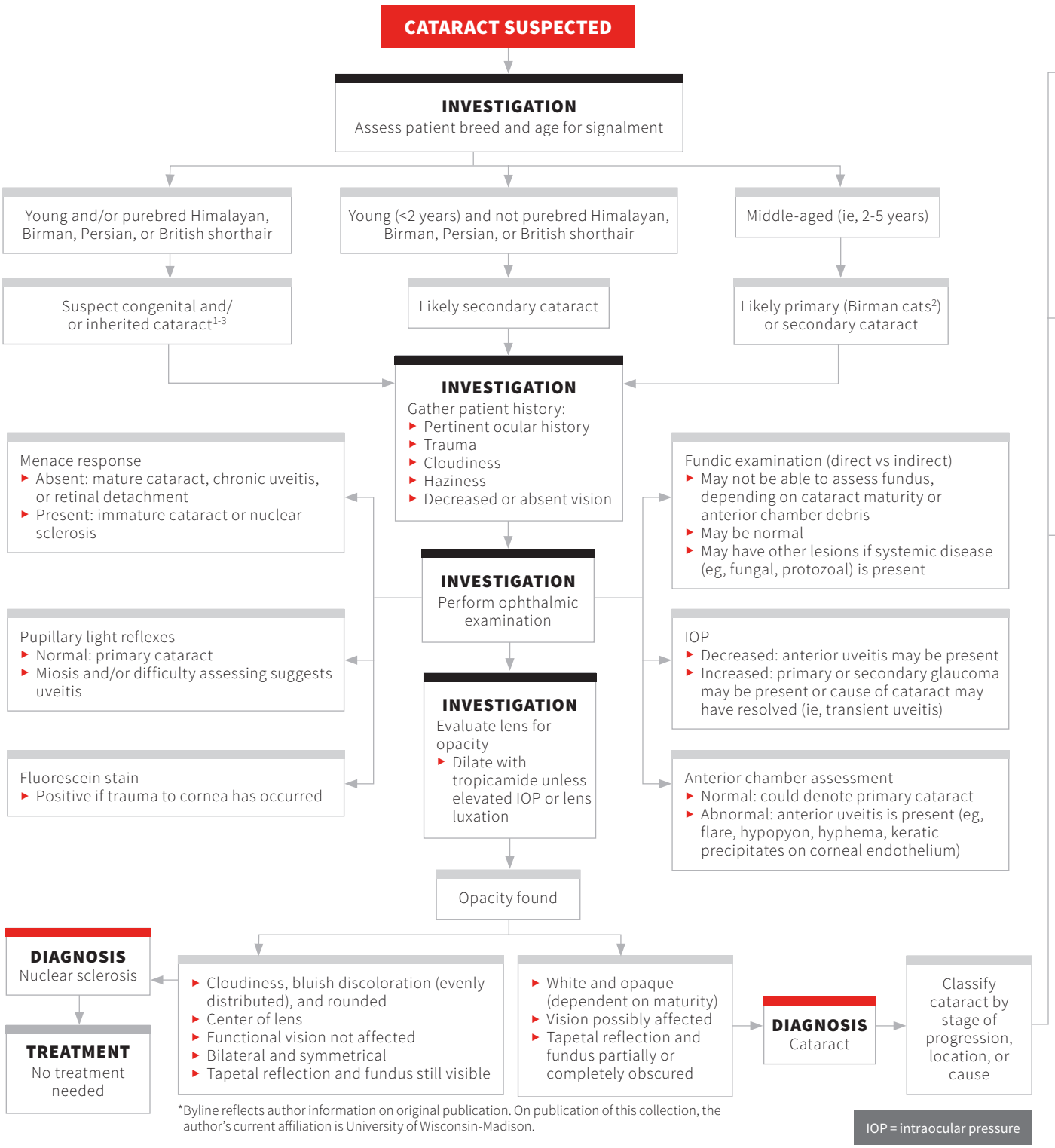
SARD = sudden acquired retinal degeneration

*Byline reflects author information on original publication. On publication of this collection, the author's current affiliation is Port City Veterinary Referral Hospital, Portsmouth, New Hampshire.
†Associated with acute blindness
‡Not associated with acute blindness


INVESTIGATION
Funduscopic examination:
Tropicamide 1% for dilation
Evaluate retinal vasculature, optic nerve head, tapetal reflectivity, nontapetal pigmentation
INVESTIGATION
Schirmer tear test:
Normal >15 mm wetting/min
Reduction may induce opacification of cornea (pigmentation, vascularization, fibrosis)
INVESTIGATION
Fluorescein dye application
DIAGNOSIS
Chorioretinitis†:
Indistinct retinal vasculature + multifocal lesions of tapetal hypo-reflectivity (cellular exudate or fluid transudate)
Negative dazzle reflex and PLR
DIAGNOSIS
Optic neuritis†:
Optic nerve hyperemia + swelling + peripapillary retinal detachment
Negative dazzle reflex and PLR
DIAGNOSIS
Retinal detachment†:
Anterior displacement of retina (± associated vessels) with subretinal accumulation of fluid or cells (bullous retinal detachment)
Veil-like appearance to retina with lack of retinal vessels over tapetum = rhegmatogenous detachment
Negative dazzle reflex and PLR
DIAGNOSIS
Retinal degeneration†:
Vascular attenuation + tapetal hyperreflectivity + optic nerve atrophy
Negative dazzle reflex and PLR at end stage
DIFFERENTIALS
Normal funduscopic examination:
SARD may be likely
Perform electroretinogram to confirm diagnosis
DIAGNOSIS
Uptake at corneal ulcer:
Associated with corneal opacification (edema, vascularization, cellular infiltration)
Associated with mild to severe reflex anterior uveitis (miosis, flare, hypopyon)
May be associated with acute blindness, but less likely (unilateral disease)
DIFFERENTIALS
Systemic infection (viral, bacterial, rickettsial, mycotic, algal, parasitic, protozoal)
Noninfectious inflammatory (uveodermatologic syndrome, granulomatous meningoencephalitis)
Neoplastic (primary [astrocytoma] melanoma) or metastatic (lymphoma, adenocarcinoma)
Systemic hypertension
DIFFERENTIALS
Exudative detachment associated with advanced chorioretinitis
Steroid-responsive (no identifiable systemic disease condition)
Rhegmatogenous retinal detachment:
• Postoperative (eg, lens surgery)
• Breed related (Shih-tzu, collie, Labrador retriever)

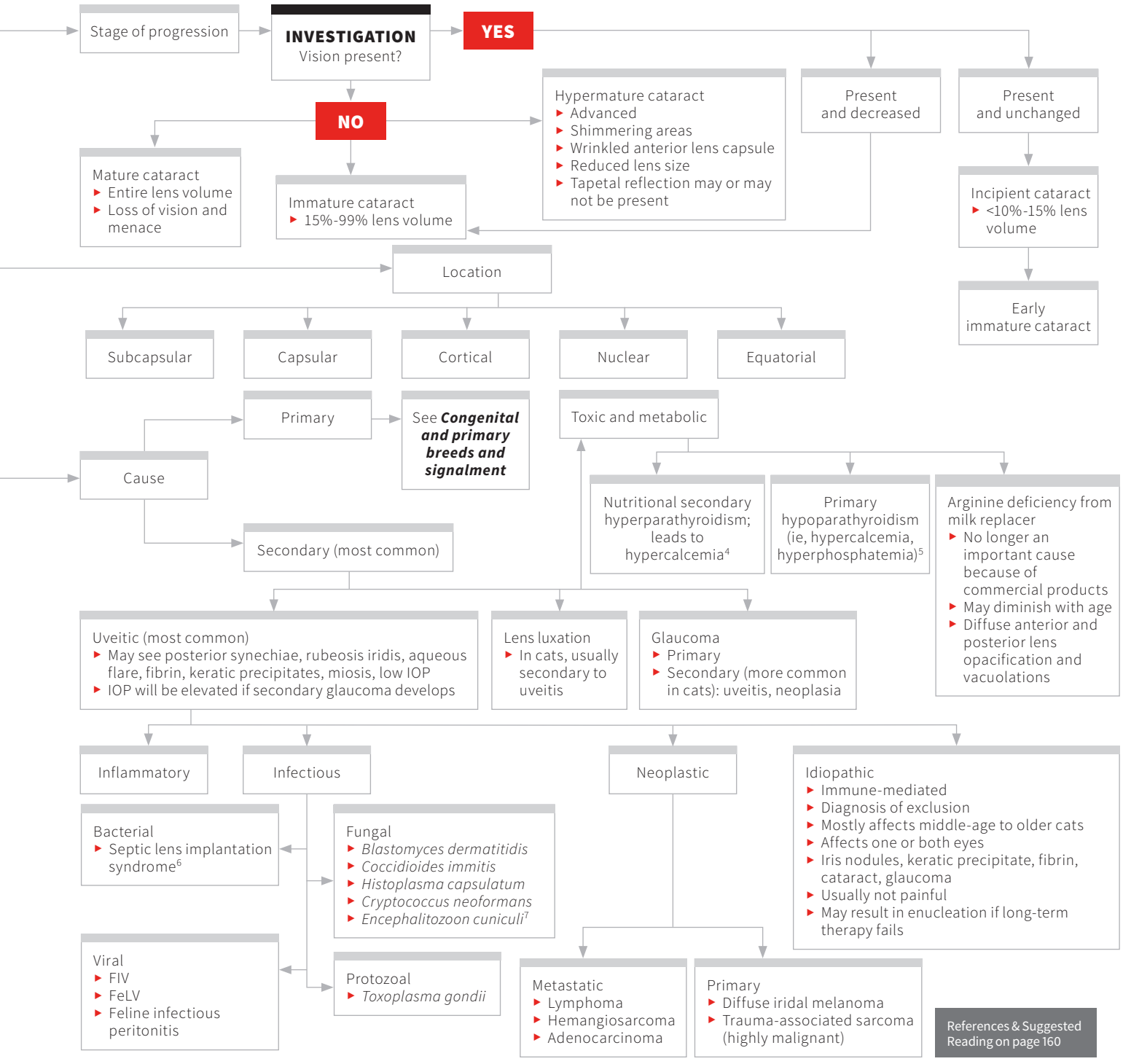
CATARACTS IN CATS: DIAGNOSIS

Mary Rebecca Telle, DVM*
Diane Hendrix, DVM, DACVO
University of Tennessee



*Byline reflects author information on original publication. On publication of this collection, the author's current affiliation is University of Wisconsin-Madison.

IOP = intraocular pressure



References & Suggested Reading on page 160

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CATARACTS IN CATS: DIAGNOSIS
REFERENCES & SUGGESTED READING

References

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5. Stiles J. Cataracts in a kitten with nutritional secondary hyperparathyroidism. *Prog Vet Comp Ophthalmol*. 1991;4:296-298.
6. Bell CM, Pot SA, Dubielzig RR. Septic implantation syndrome in dogs and cats: a distinct pattern of endophthalmitis with lenticular abscess. *Vet Ophthalmol*. 2013;16(3):180-185.
7. Benz P, Maass G, Csokai J, et al. Detection of Encephalitozoon cuniculi in the feline cataractous lens. *Vet Ophthalmol*. 2011;14(Suppl 1):37-47.

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Peiffer RL, Gelatt KN. Cataracts in the cat. *Feline Pract*. 1974;4:34-38.

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CATARACTS IN CATS: TREATMENT

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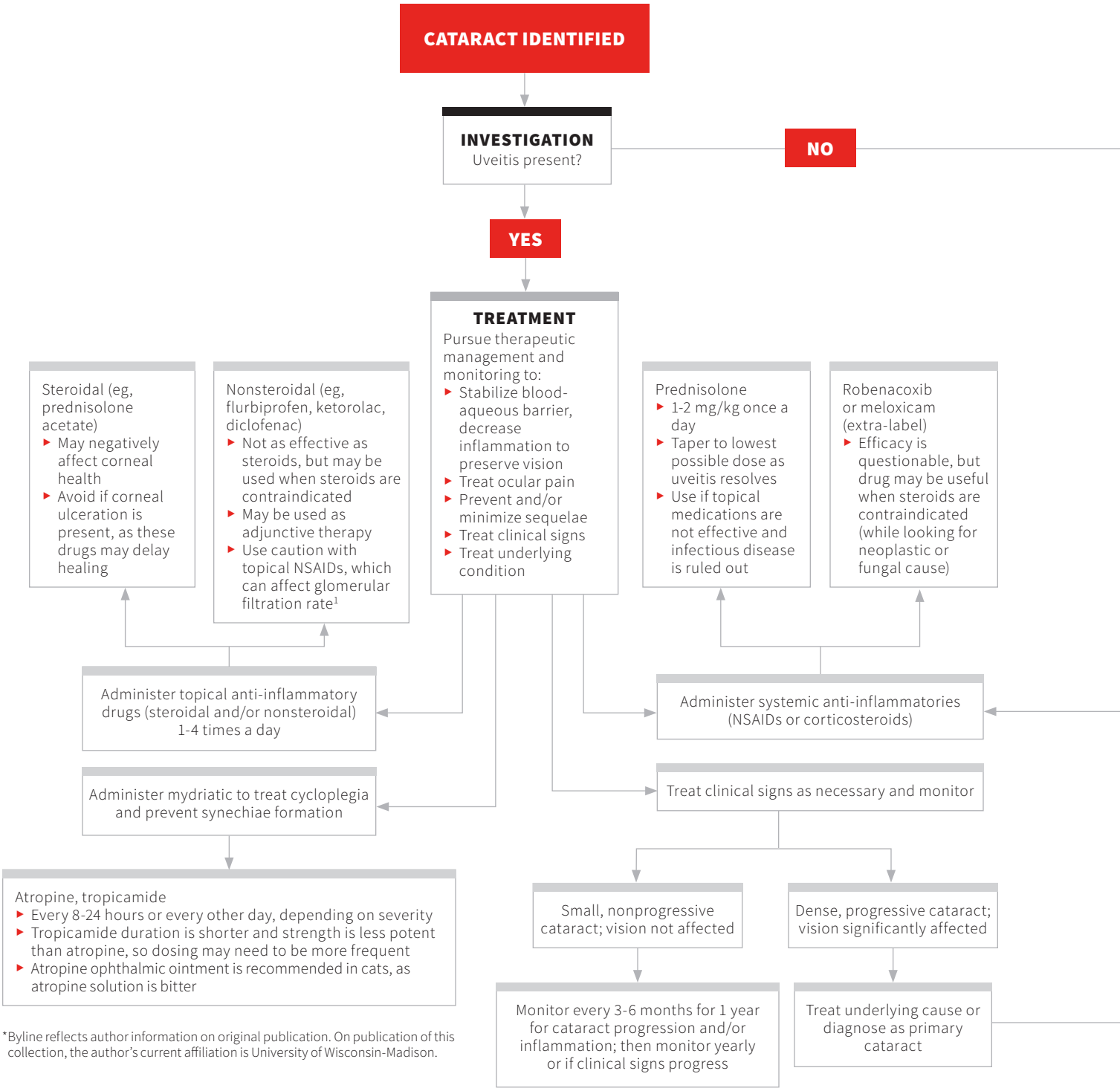
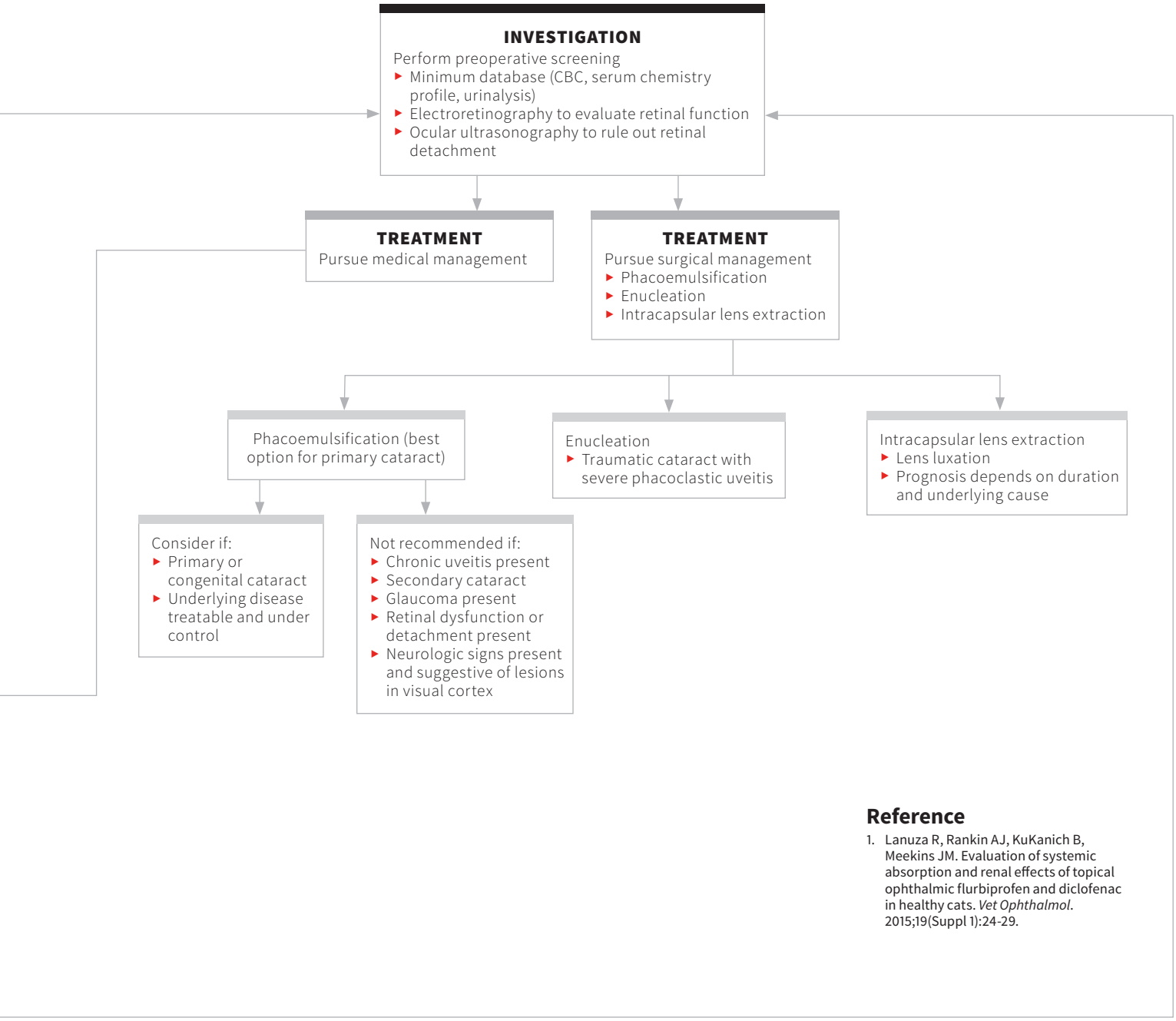
Diane Hendrix, DVM, DACVO
University of Tennessee



*Byline reflects author information on original publication. On publication of this collection, the author's current affiliation is University of Wisconsin-Madison.



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DECREASED TEAR PRODUCTION IN DOGS

Ian Herring, DVM, MS, DACVO
Virginia–Maryland College of Veterinary Medicine

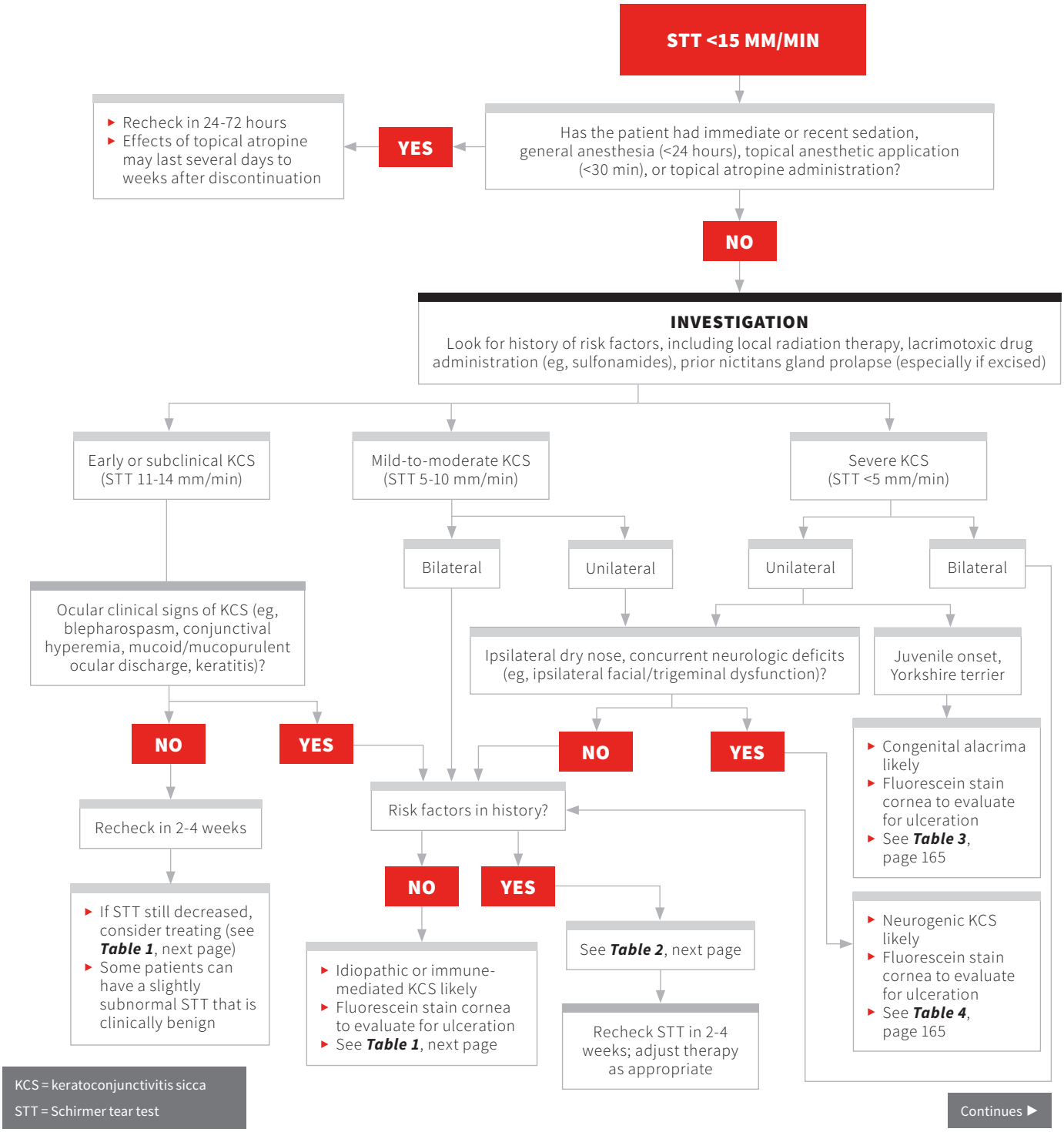

TABLE 1

TREATMENT FOR IDIOPATHIC/IMMUNE-MEDIATED KCS

Treatment	Application
Topical ophthalmic cyclosporine (1%-2% solution, 0.2% ointment), tacrolimus (0.03%)	Apply to affected eye(s) q12h; lifelong therapy required
Topical lacrimomimetic PRN (ie, artificial tears; products containing sodium hyaluronate may be particularly beneficial)	Apply until signs resolve (usually unnecessary when STT >8 mm/min)
Topical ophthalmic broad-spectrum antibiotic (eg, neomycin, polymyxin B, gramicidin solution)	Apply to affected eye(s) q8h for 10-14 days or until corneal ulceration (if present) is healed
Topical mucolytic medication (2.5%-5% *N*-acetylcysteine)	Apply q6-8h if copious mucopurulent discharge is present
Warm compress to eyelids, periocular hair trimming, facial cleaning	Apply as warranted

TABLE 2

TREATMENT FOR KCS SECONDARY TO RISK FACTORS

Treatment	Application
Discontinue lacrimotoxic medication if suspected in KCS pathogenesis	N/A
Topical ophthalmic cyclosporine or tacrolimus can be attempted (see ***Table 1***) but is generally ineffective in these cases	Apply to affected eye(s) q12h
Topical lacrimomimetic PRN (ie, artificial tears; products containing sodium hyaluronate may be particularly beneficial)	Apply until signs resolve (usually unnecessary when STT >8 mm/min)
Topical ophthalmic broad-spectrum antibiotic (eg, neomycin, polymyxin B, gramicidin solution)	Apply to affected eye(s) q8h for 10-14 days or until corneal ulceration (if present) is healed
Topical mucolytic medication (2.5%-5% *N*-acetylcysteine)	Apply q6-8h if copious mucopurulent discharge is present
Warm compress to eyelids, periocular hair trimming, facial cleaning	Apply as warranted

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TABLE 3

TREATMENT FOR CONGENITAL ALACRIMA

Treatment	Application
Topical lacrimomimetic PRN (ie, artificial tears; products containing sodium hyaluronate may be particularly beneficial)	Apply until signs resolve (usually necessary when STT >8 mm/min)
Topical ophthalmic broad-spectrum antibiotic (eg, neomycin, polymyxin B, gramicidin solution)	Apply to affected eye(s) q8h for 10-14 days or until corneal ulceration (if present) is healed
Topical mucolytic medication (2.5%-5% *N*-acetylcysteine)	Apply q6-8h if copious mucopurulent discharge is present
Warm compress to eyelids, periocular hair trimming, facial cleaning	Apply as warranted

TABLE 4

TREATMENT FOR NEUROGENIC KCS

Treatment	Application
Topical ophthalmic 0.2% pilocarpine q6-8h or 2% oral pilocarpine	Initial oral dose, 2 drops/20 pounds (10 kg) body weight q12h; increase 1 drop at a time to effect or until systemic toxicity (eg, salivation, vomiting, diarrhea) is evident
Topical lacrimomimetic PRN (ie, artificial tears; products containing sodium hyaluronate may be particularly beneficial)	Apply until signs resolve (usually unnecessary when STT >8 mm/min)
Topical ophthalmic broad-spectrum antibiotic (eg, neomycin, polymyxin B, gramicidin solution)	Apply to affected eye(s) q8h for 10-14 days or until corneal ulceration (if present) is healed
Topical mucolytic medication (2.5%-5% *N*-acetylcysteine)	Apply q6-8h if copious mucopurulent discharge is present
Monitor for blepharospasm	N/A

*Spontaneous resolution may occur; therapy can be discontinued after resolution.

Suggested Reading

Guiliano EA. Diseases and surgery of the canine lacrimal secretory system. In: Gelatt KN, Gilger BC, Kern TJ, eds. *Veterinary Ophthalmology*. 5th ed. Oxford, UK: John Wiley & Sons; 2013:912-944.

Matheis FL, Walser-Reinhardt L, Spiess BM. Canine neurogenic keratoconjuctivitis sicca: 11 cases (2006-2010). *Vet Ophthalmol*. 2012;15(4):288-290.

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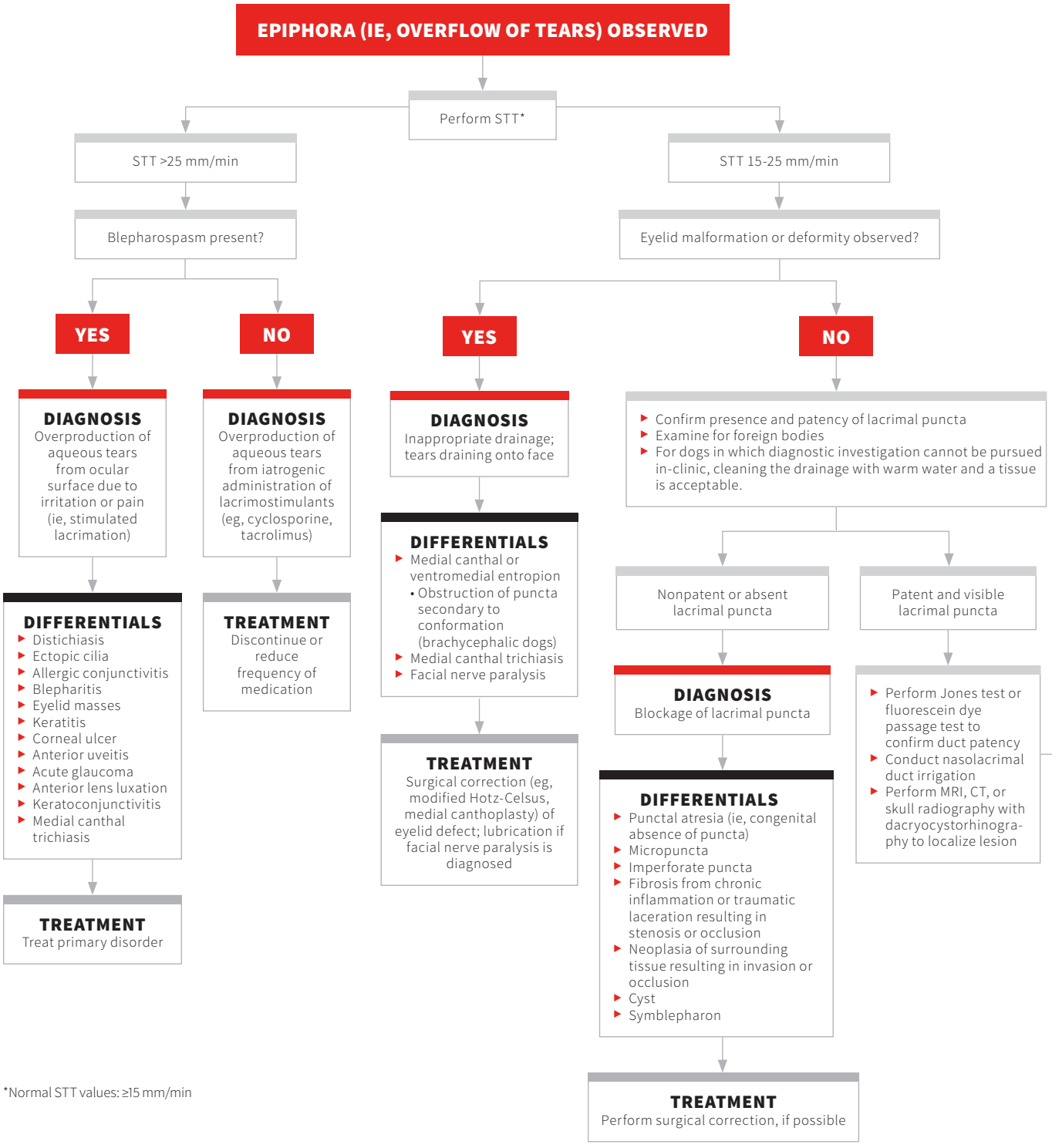
Westermeyer HD, Ward DA, Abrams K. Breed predisposition to congenital alacrima in dogs. *Vet Ophthalmol*. 2009;12(1):1-5.

KCS = keratoconjunctivitis sicca

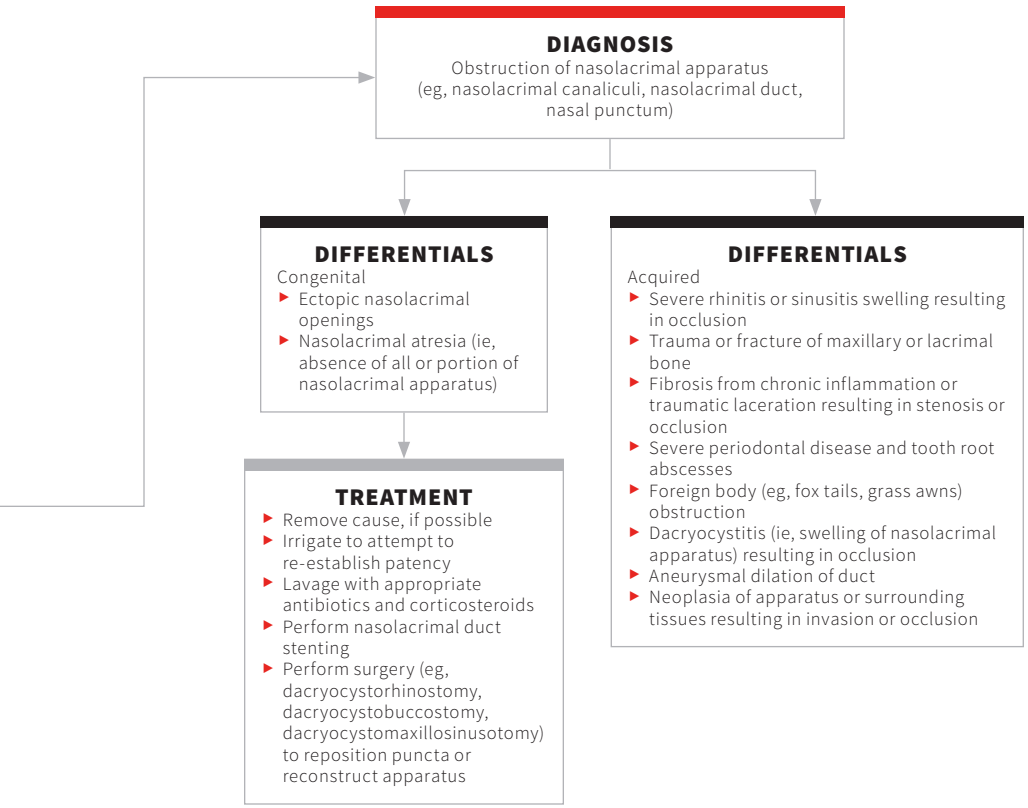
STT = Schirmer tear test

EPIPHORA IN DOGS

Christina Korb, DVM
DJ Haeussler Jr, DVM, MS, DACVO
The Animal Eye Institute
Cincinnati, Ohio



*Normal STT values: ≥15 mm/min



Suggested Reading

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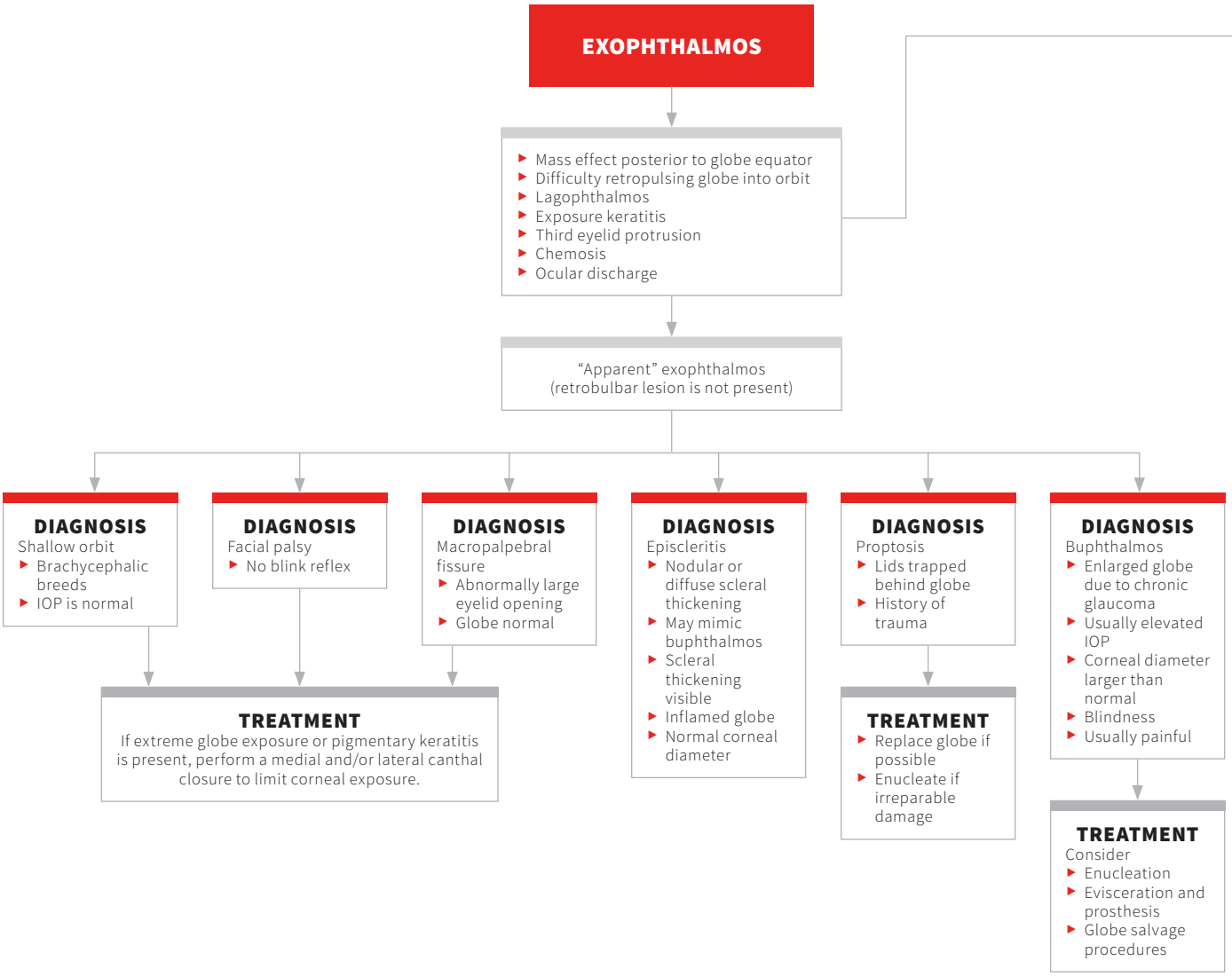
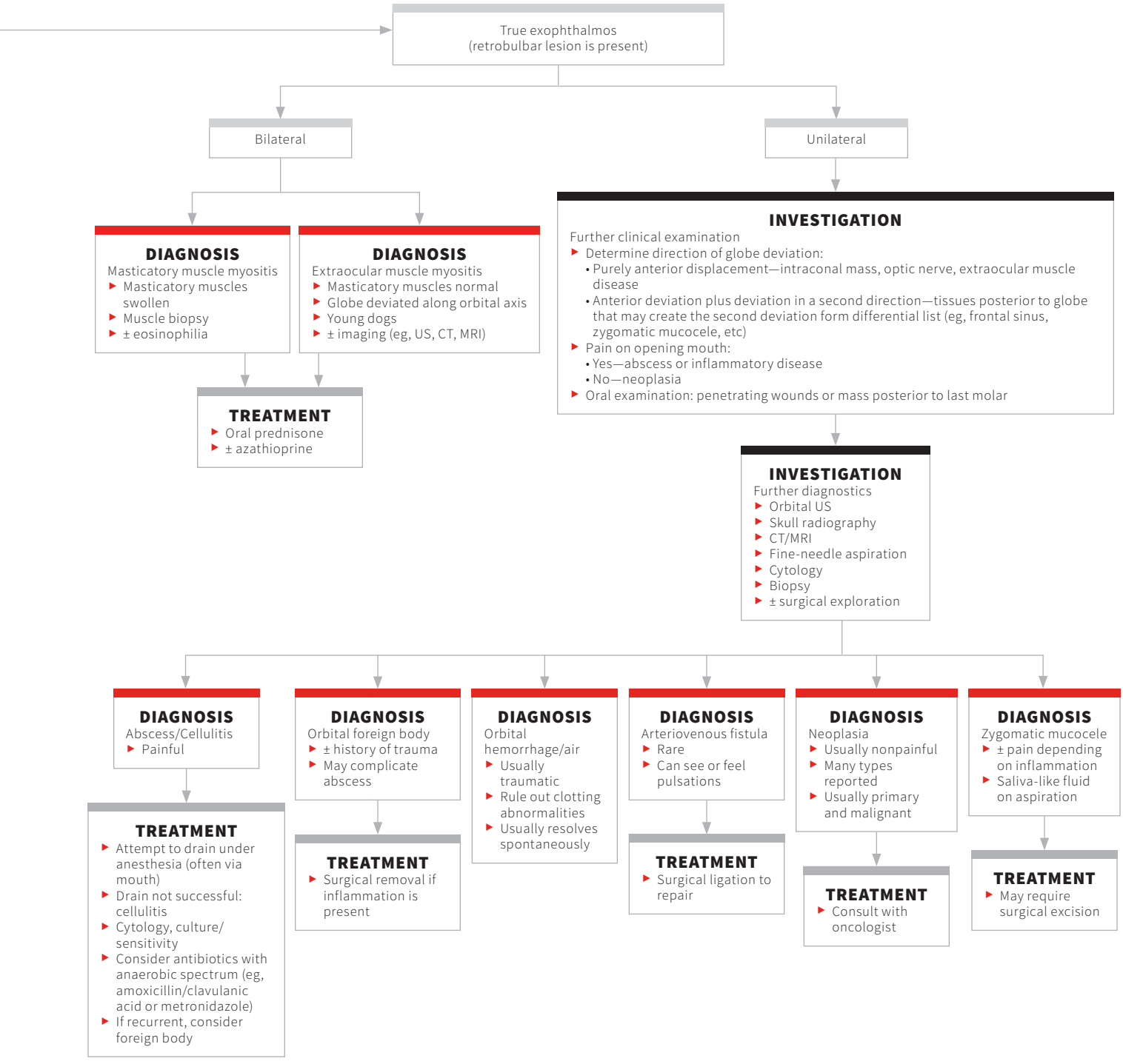
EXOPHTHALMOS

Paul E. Miller, DVM, DACVO
University of Wisconsin–Madison

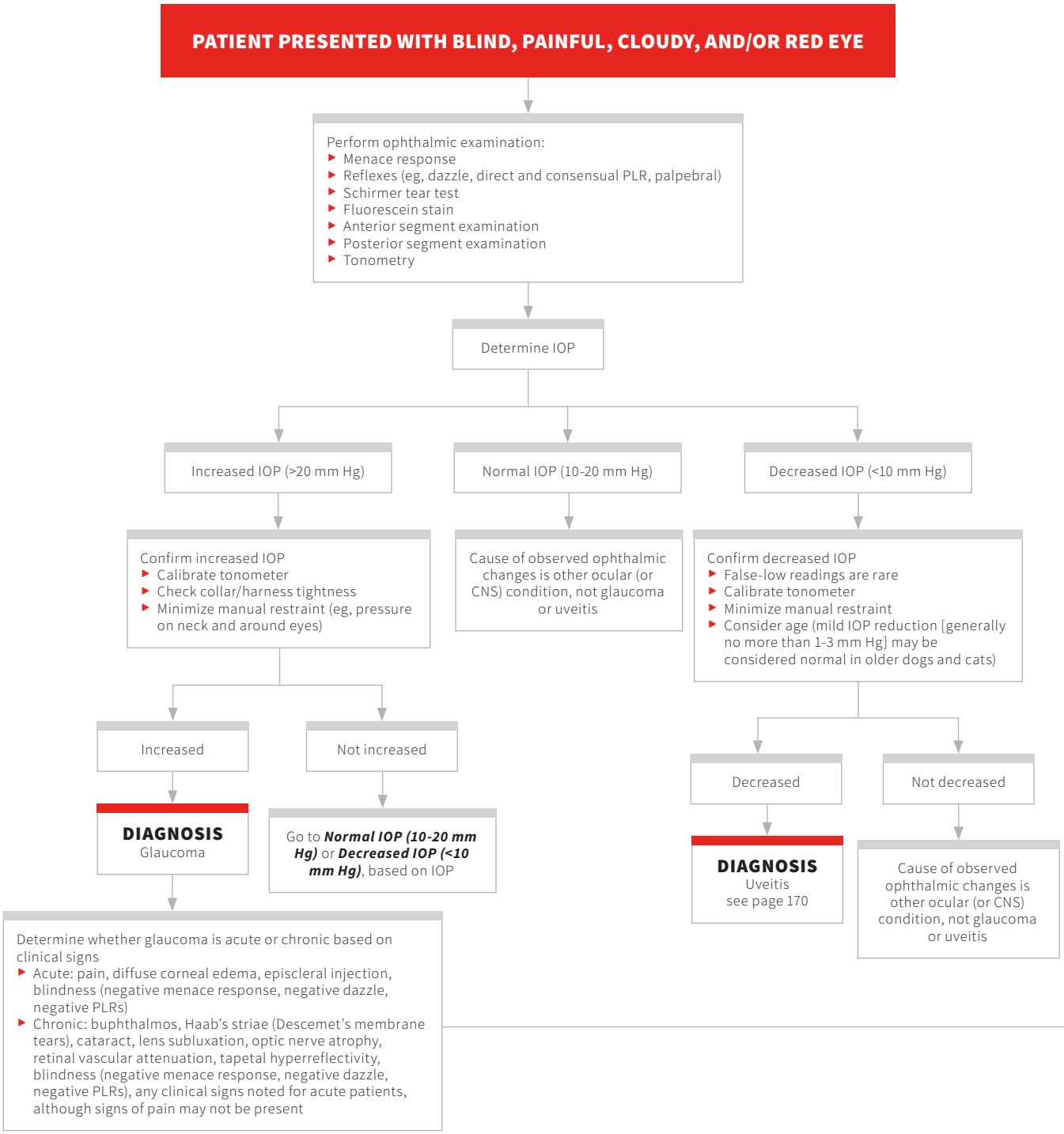


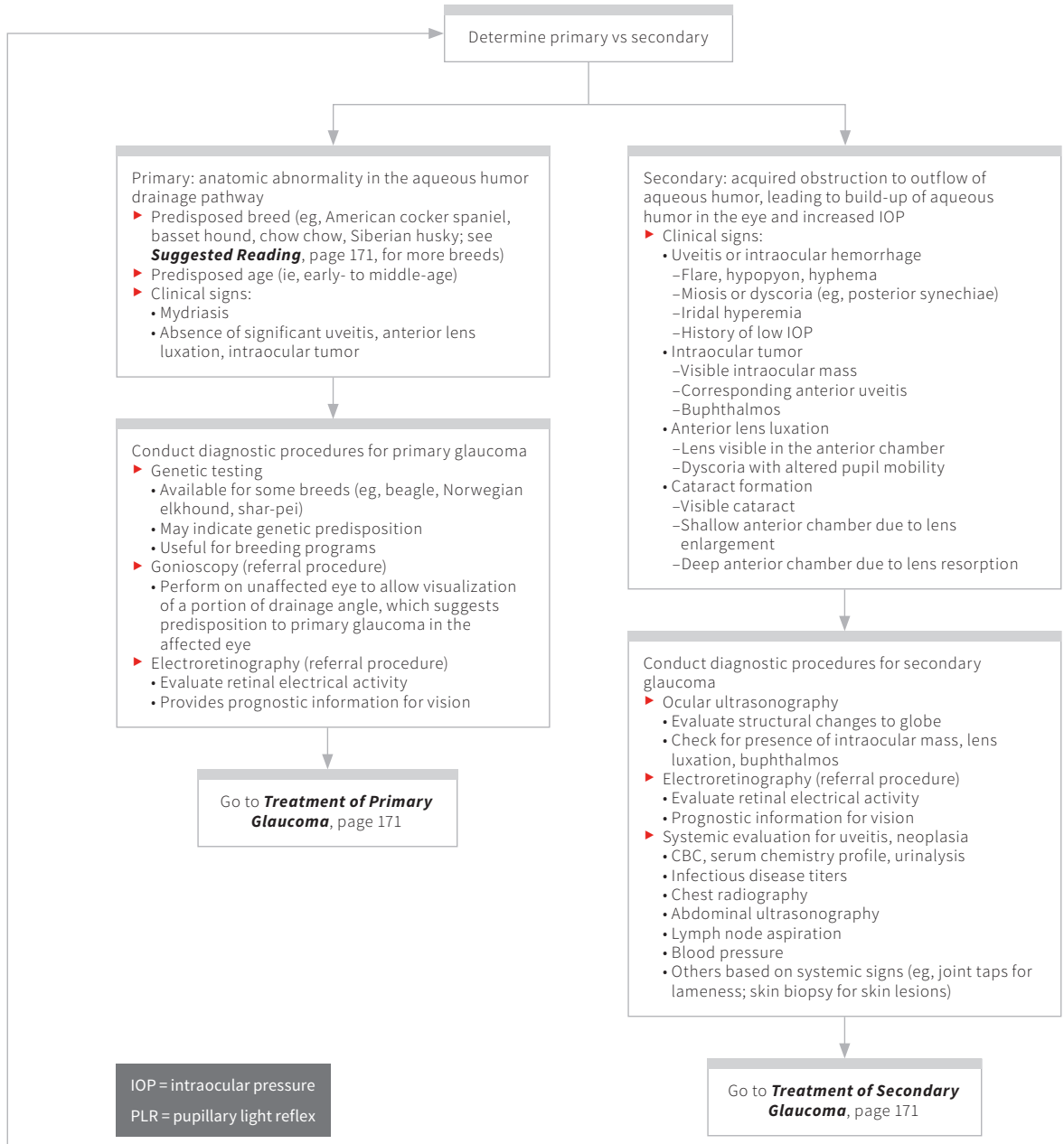
IOP = intraocular pressure
US = ultrasound


True exophthalmos
(retrobulbar lesion is present)
Bilateral
Unilateral
DIAGNOSIS
Masticatory muscle myositis
Masticatory muscles swollen
Muscle biopsy
± eosinophilia
DIAGNOSIS
Extraocular muscle myositis
Masticatory muscles normal
Globe deviated along orbital axis
Young dogs
± imaging (eg, US, CT, MRI)
TREATMENT
Oral prednisone
± azathioprine
INVESTIGATION
Further clinical examination
Determine direction of globe deviation:
Purely anterior displacement—intraconal mass, optic nerve, extraocular muscle disease
Anterior deviation plus deviation in a second direction—tissues posterior to globe that may create the second deviation form differential list (eg, frontal sinus, zygomatic mucocele, etc)
Pain on opening mouth:
Yes—abscess or inflammatory disease
No—neoplasia
Oral examination: penetrating wounds or mass posterior to last molar
INVESTIGATION
Further diagnostics
Orbital US
Skull radiography
CT/MRI
Fine-needle aspiration
Cytology
Biopsy
± surgical exploration
DIAGNOSIS
Abscess/Cellulitis
Painful
TREATMENT
Attempt to drain under anesthesia (often via mouth)
Drain not successful: cellulitis
Cytology, culture/sensitivity
Consider antibiotics with anaerobic spectrum (eg, amoxicillin/clavulanic acid or metronidazole)
If recurrent, consider foreign body
DIAGNOSIS
Orbital foreign body
± history of trauma
May complicate abscess
TREATMENT
Surgical removal if inflammation is present
DIAGNOSIS
Orbital hemorrhage/air
Usually traumatic
Rule out clotting abnormalities
Usually resolves spontaneously
DIAGNOSIS
Arteriovenous fistula
Rare
Can see or feel pulsations
TREATMENT
Surgical ligation to repair
DIAGNOSIS
Neoplasia
Usually nonpainful
Many types reported
Usually primary and malignant
TREATMENT
Consult with oncologist
DIAGNOSIS
Zygomatic mucocele
± pain depending on inflammation
Saliva-like fluid on aspiration
TREATMENT
May require surgical excision

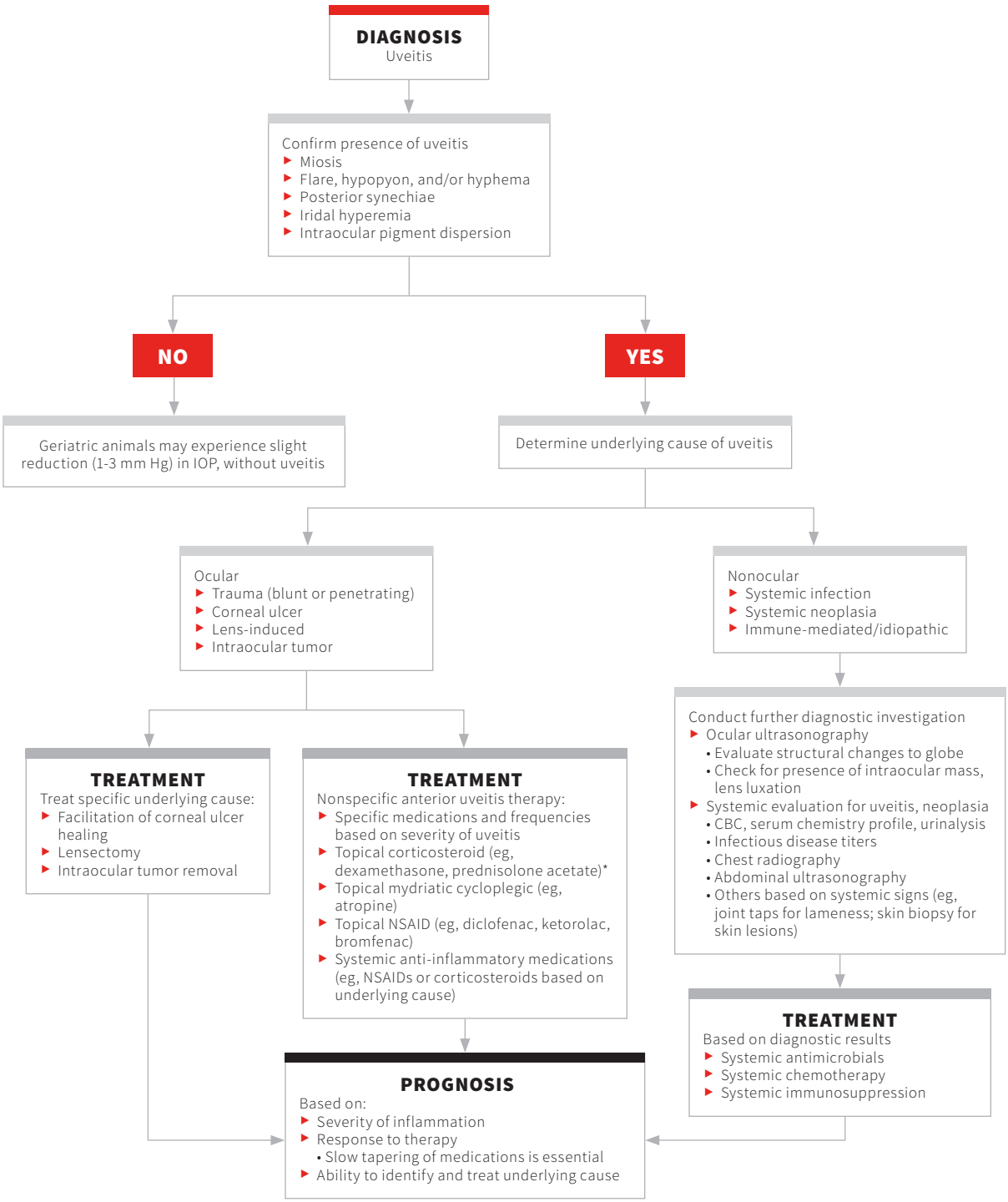
INCREASED & DECREASED INTRAOCULAR PRESSURE

Alison Clode, DVM, DACVO
Port City Veterinary Referral Hospital
Portsmouth, New Hampshire




Determine primary vs secondary
Primary: anatomic abnormality in the aqueous humor drainage pathway
▸ Predisposed breed (eg, American cocker spaniel, basset hound, chow chow, Siberian husky; see ***Suggested Reading***, page 171, for more breeds)
▸ Predisposed age (ie, early- to middle-age)
▸ Clinical signs:
• Mydriasis
• Absence of significant uveitis, anterior lens luxation, intraocular tumor
Conduct diagnostic procedures for primary glaucoma
▸ Genetic testing
• Available for some breeds (eg, beagle, Norwegian elkhound, shar-pei)
• May indicate genetic predisposition
• Useful for breeding programs
▸ Gonioscopy (referral procedure)
• Perform on unaffected eye to allow visualization of a portion of drainage angle, which suggests predisposition to primary glaucoma in the affected eye
▸ Electroretinography (referral procedure)
• Evaluate retinal electrical activity
• Provides prognostic information for vision
Go to ***Treatment of Primary Glaucoma***, page 171
Secondary: acquired obstruction to outflow of aqueous humor, leading to build-up of aqueous humor in the eye and increased IOP
▸ Clinical signs:
• Uveitis or intraocular hemorrhage
–Flare, hypopyon, hyphema
–Miosis or dyscoria (eg, posterior synechiae)
–Iridal hyperemia
–History of low IOP
• Intraocular tumor
–Visible intraocular mass
–Corresponding anterior uveitis
–Buphthalmos
• Anterior lens luxation
–Lens visible in the anterior chamber
–Dyscoria with altered pupil mobility
• Cataract formation
–Visible cataract
–Shallow anterior chamber due to lens enlargement
–Deep anterior chamber due to lens resorption
Conduct diagnostic procedures for secondary glaucoma
▸ Ocular ultrasonography
• Evaluate structural changes to globe
• Check for presence of intraocular mass, lens luxation, buphthalmos
▸ Electroretinography (referral procedure)
• Evaluate retinal electrical activity
• Prognostic information for vision
▸ Systemic evaluation for uveitis, neoplasia
• CBC, serum chemistry profile, urinalysis
• Infectious disease titers
• Chest radiography
• Abdominal ultrasonography
• Lymph node aspiration
• Blood pressure
• Others based on systemic signs (eg, joint taps for lameness; skin biopsy for skin lesions)
Go to ***Treatment of Secondary Glaucoma***, page 171
IOP = intraocular pressure
PLR = pupillary light reflex

Continues ▶


DIAGNOSIS
Uveitis
Confirm presence of uveitis
Miosis
Flare, hypopyon, and/or hyphema
Posterior synechiae
Iridal hyperemia
Intraocular pigment dispersion
NO
YES
Geriatric animals may experience slight reduction (1-3 mm Hg) in IOP, without uveitis
Determine underlying cause of uveitis
Ocular
Trauma (blunt or penetrating)
Corneal ulcer
Lens-induced
Intraocular tumor
Nonocular
Systemic infection
Systemic neoplasia
Immune-mediated/idiopathic
TREATMENT
Treat specific underlying cause:
Facilitation of corneal ulcer healing
Lensectomy
Intraocular tumor removal
TREATMENT
Nonspecific anterior uveitis therapy:
Specific medications and frequencies based on severity of uveitis
Topical corticosteroid (eg, dexamethasone, prednisolone acetate)*
Topical mydriatic cycloplegic (eg, atropine)
Topical NSAID (eg, diclofenac, ketorolac, bromfenac)
Systemic anti-inflammatory medications (eg, NSAIDs or corticosteroids based on underlying cause)
Conduct further diagnostic investigation
Ocular ultrasonography
• Evaluate structural changes to globe
• Check for presence of intraocular mass, lens luxation
Systemic evaluation for uveitis, neoplasia
• CBC, serum chemistry profile, urinalysis
• Infectious disease titers
• Chest radiography
• Abdominal ultrasonography
• Others based on systemic signs (eg, joint taps for lameness; skin biopsy for skin lesions)
TREATMENT
Based on diagnostic results
Systemic antimicrobials
Systemic chemotherapy
Systemic immunosuppression
PROGNOSIS
Based on:
Severity of inflammation
Response to therapy
• Slow tapering of medications is essential
Ability to identify and treat underlying cause

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TREATMENT OF PRIMARY GLAUCOMA

- IOP-lowering medication
  - Specific medications and frequencies vary with acute vs chronic and primary vs secondary glaucoma
  - Carbonic anhydrase inhibitors (eg, dorzolamide, brinzolamide)
  - Prostaglandin analogues (eg, latanoprost, travoprost)
  - β blockers (eg, timolol)
  - Parasympathomimetic medications (eg, demecarium bromide, pilocarpine)
- Glaucoma-specific surgical intervention
  - Laser cycloablation (referral procedure)
  - Gonioimplantation (referral procedure)

TREATMENT OF SECONDARY GLAUCOMA

- IOP-lowering medication
  - Specific medications and frequencies vary with acute vs chronic and primary vs secondary glaucoma
  - Carbonic anhydrase inhibitors (eg, dorzolamide, brinzolamide)
  - β blockers (eg, timolol)
- Treatment of underlying cause
  - Topical anti-inflammatory medications
  - Systemic anti-inflammatory medications
  - Other medications, as indicated for cause of uveitis†
  - Lensectomy (referral procedure)
  - Intraocular tumor removal (referral procedure)
- Glaucoma-specific surgical intervention
  - Laser cycloablation (referral procedure)
  - Gonioimplantation (referral procedure)

PROGNOSIS

Prognosis depends on underlying cause and response to treatment. If signs of pain continue, definitive treatment may require:

- Enucleation
- Evisceration
- Ciliary body chemical ablation (eg, intravitreal gentamicin injection)

IOP = intraocular pressure

*Provided no corneal ulcer is present

†In patients with secondary glaucoma due to posterior synechiae and iris bombe, atropine may be indicated to break open the pupil and restore aqueous humor flow.

Suggested Reading

Bergstrom BE, Stiles J, Townsend WM. Canine panuveitis: a retrospective evaluation of 55 cases (2000- 2015). *Vet Ophthalmol*. 2017;20(5):390-397.

Plummer CE, Regnier A, Gelatt KN. The canine glaucomas. In: Gelatt KN, Gilger BC, Kern TJ, eds. *Veterinary Ophthalmology*. 5th ed. Ames, IA: Wiley-Blackwell; 2013:1050-1145.

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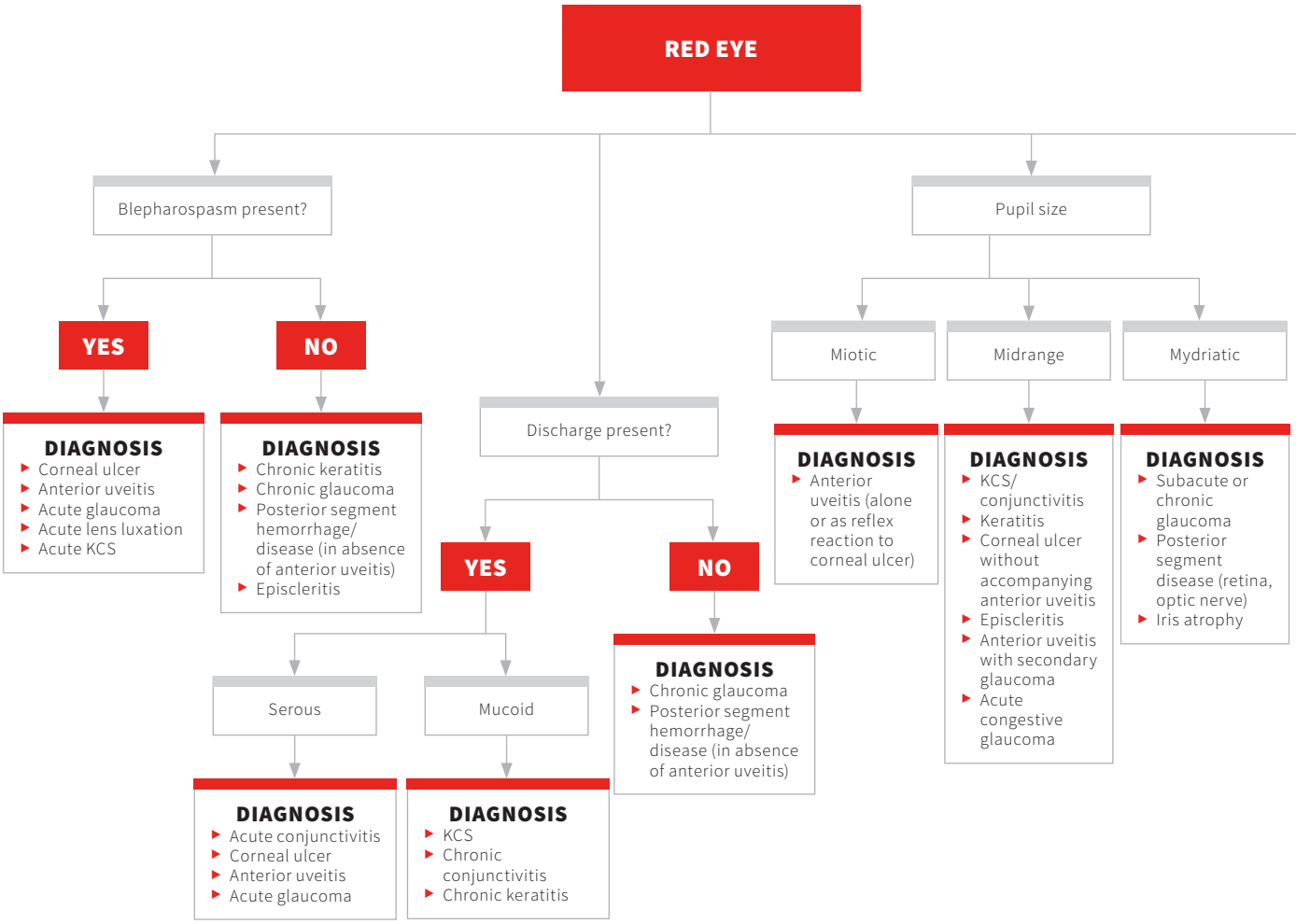
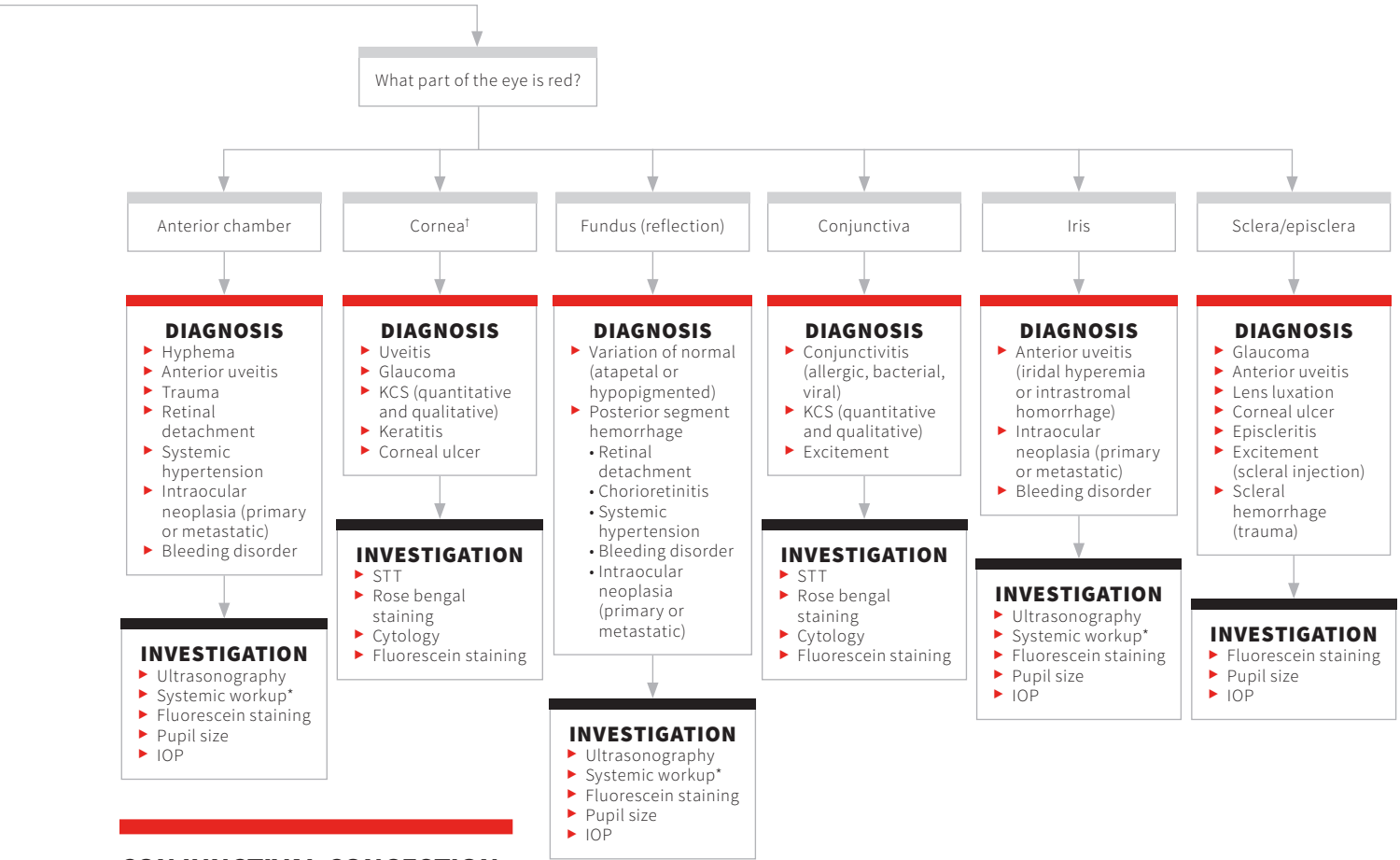
RED EYE

Caryn E. Plummer, DVM, DACVO
University of Florida



CHECKLIST: EXAMINING THE RED EYE

- ☐ Observe eye to determine what part(s) are affected, what appears normal or abnormal, whether eye is painful, and presence of discharge.
- ☐ Develop list of diagnostic differentials based on ocular changes and conduct diagnostic testing.
- ☐ Determine whether eye is visual.
- ☐ Assess pupil size and light reflexes.
- ☐ Perform STT for quantitative assessment of tear production.
- ☐ Perform ocular surface staining:
  - Fluorescein staining to assess corneal epithelium defects, NLD patency, and tear film breakup time and stability
  - Seidel test to identify leakage of aqueous humor through cornea
  - Rose bengal staining to measure precorneal tear film quality and integrity
- ☐ Perform tonometry to estimate IOP: elevation consistent with glaucoma, decrease consistent with intraocular inflammation.
- ☐ Complete examination and systemic diagnostic testing as indicated.



CONJUNCTIVAL CONGESTION OR EPISCLERAL INJECTION?

- Conjunctival vessels are movable over sclera.
- Episcleral vessels are fixed and typically larger.
- Conjunctival vessels will blanch more rapidly following application of a vaso-constricting agent (ie, phenylephrine, epinephrine).

*Systemic workup = minimum database, systemic blood pressure, coagulation parameters, infectious disease titers, thoracic or abdominal imaging

†Cornea is rarely red unless vascularization is present; blood may also be present within the stroma or behind the cornea, within the anterior chamber, giving the impression of a red cornea

IOP = intraocular pressure
KCS = keratoconjunctivitis sicca
NLD = nasolacrimal duct
STT = Schirmer tear test

CLINICAL SIGNS OF SELECTED OPHTHALMIC DISEASES

Kenneth L. Abrams, DVM, DACVO*
Veterinary Ophthalmology Services, Inc
Warwick, Rhode Island

ADNEXAL DISEASES

	Ocular Discharge				Corneal Opacity			Intraocular Pressure				Specific Findings & Comments
	Mucous	Serous	Squinting	Conjunctival redness	Vessels	Pigment	Edema	Increased	Decreased	Aqueous flare/cells	Decreased vision	
Distichiasis	–	1–2	±	±	–	–	–	–	–	–	–	2nd row of cilia
Ectopic cilia	–	3–4	4	3	–	–	±	–	–	–	–	• Cilium protrudes through palpebral conjunctiva • Can cause corneal ulcers
Entropion	±	2–4	2–4	1–3	–	–	–	–	–	–	–	• Rolled-in eyelid • Can cause corneal ulcers
Prolapsed nictitans (cherry eye)	±	±	–	±	–	–	–	–	–	–	–	Observation of prolapsed gland
Everted cartilage	–	–	–	±	–	–	–	–	–	–	–	Scrolled cartilage
Blocked nasolacrimal duct	±	2–3	±	–	–	–	–	–	–	–	–	Negative fluorescein passage test
Follicular hyperplasia	–	1–2	–	±	–	–	–	–	–	–	–	Prominent third eyelid follicles

— = not present; ± = variable; 1 = mild; 2-3 = moderate; 4 = severe; ERG = electroretinogram

*Byline reflects author information on original publication. On publication of this collection, the author is retired after a successful tenure with Veterinary Ophthalmology Services, Inc.

Continues ▶

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CORNEAL DISEASES

	Ocular Discharge				Corneal Opacity			Intraocular Pressure				Specific Findings & Comments
	Mucous	Serous	Squinting	Conjunctival redness	Vessels	Pigment	Edema	Increased	Decreased	Aqueous flare/cells	Decreased vision	
Corneal ulcer, uninfected	±	2–4	2–4	1–4	1–4	–	1–3	–	–	–	–	Fluorescein stain + can cause uveitis
Kerato-conjunctivitis sicca	2–4	–	1–3	2–4	1–4	1–4	±	–	–	–	–	Schirmer tear test <15 mm/min
Pannus	–	0–2	–	1–3	2–4	2–4	±	–	–	–	1–4	Usually starts as infero-temporal disease
Pigmentary keratitis	±	±	–	1–2	1–2	3–4	–	–	–	–	1–4	Usually nasal areas first
Endothelial dystrophy	–	–	–	1–3	–	–	1–4	–	–	–	±	Generalized edema—older patient

— = not present; ± = variable; 1 = mild; 2-3 = moderate; 4 = severe; ERG = electroretinogram

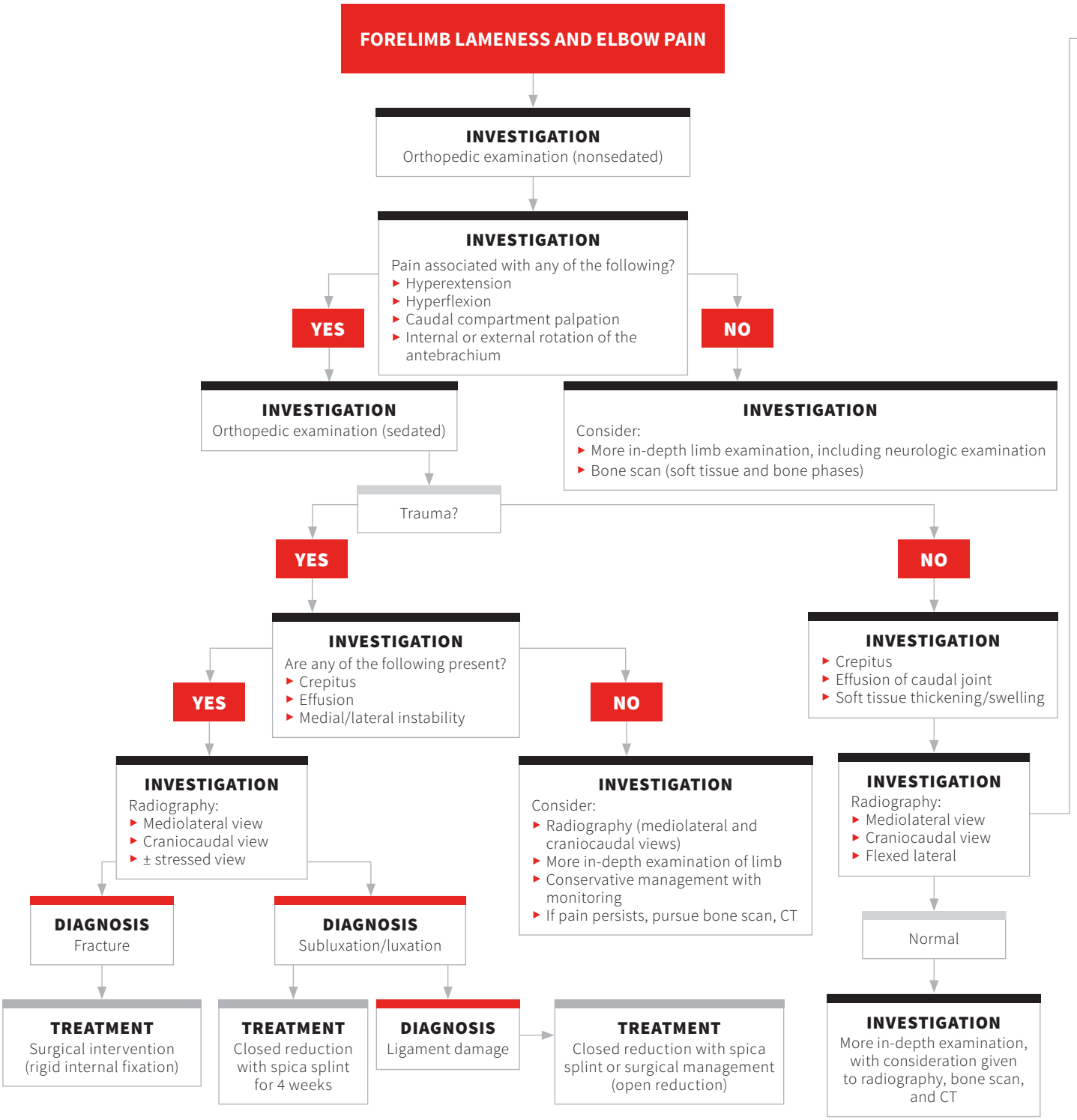
INTRAOCULAR DISEASES

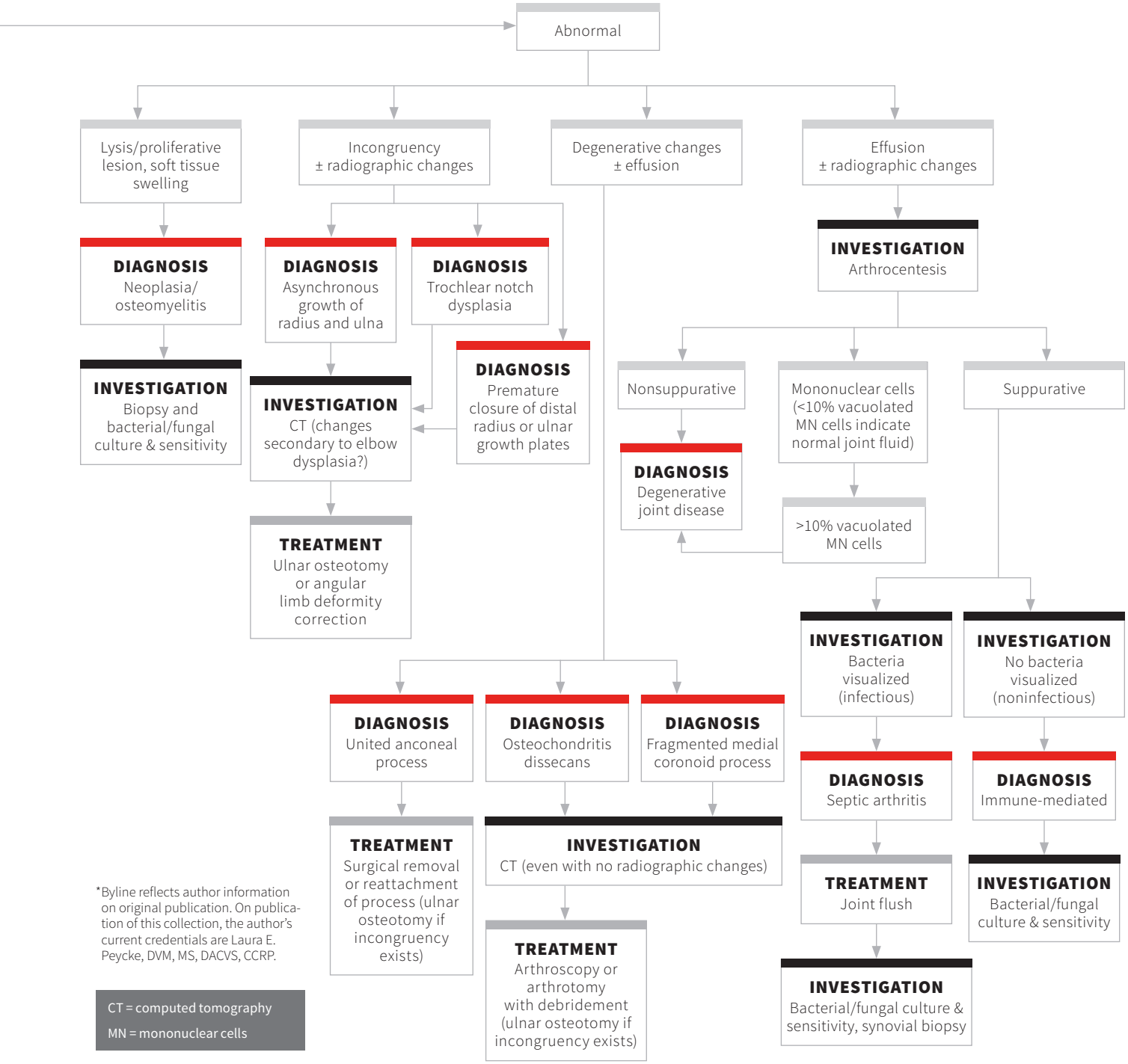
	Ocular Discharge				Corneal Opacity			Intraocular Pressure				Specific Findings & Comments
	Mucous	Serous	Squinting	Conjunctival redness	Vessels	Pigment	Edema	Increased	Decreased	Aqueous flare/cells	Decreased vision	
Cataract	–	–	–	–	–	–	–	–	–	–	1–4	Lens opacification
Acute glaucoma	±	±	±	2–4	–	–	–	4	–	±	4	Lethargy is prominent
Uveitis	±	±	1–4	1–3	–	–	–	–	3–4	2–4	1–4	Light sensitive
Anterior lens luxation	±	±	3–4	1–4	–	–	±	1–4	–	–	2–4	Observation of lens in anterior chamber
Retinal detachment	–	–	–	–	–	–	–	–	–	–	2–4	Various focal points of fundic structures
Retinal degeneration	–	–	–	–	–	–	–	–	–	–	3–4	Fundus: vascular attenuation, tapetal hyperreflectivity; ERG diminished
Optic neuritis	–	–	–	–	–	–	–	–	–	–	4	Papilledema? Pupil reflexes absent; ERG present

— = not present; ± = variable; 1 = mild; 2-3 = moderate; 4 = severe; ERG = electroretinogram

FORELIMB LAMENESS & ELBOW PAIN

Laura E. Peycke, DVM, MS, DACVS*
Texas A&M University



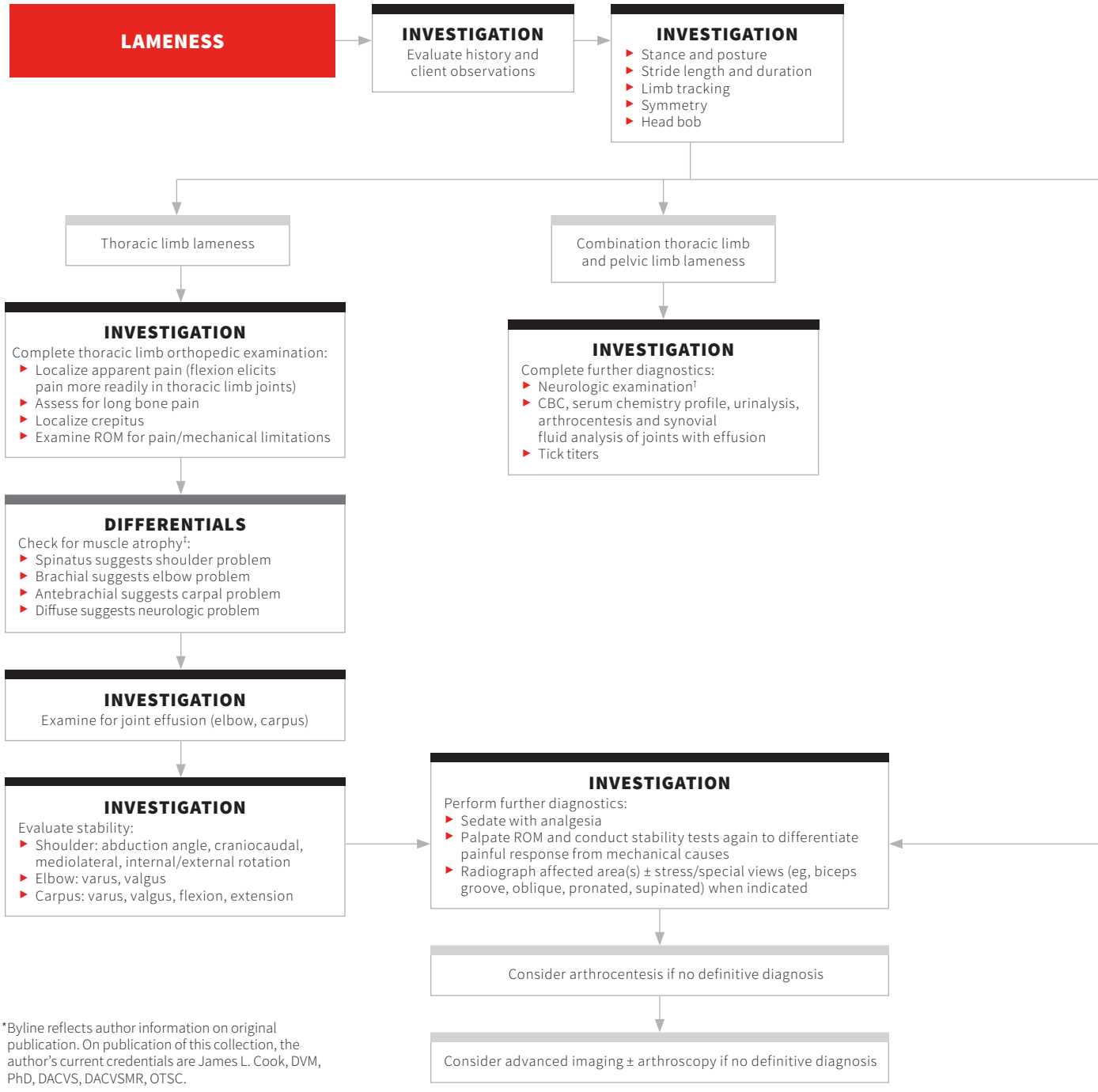


*Byline reflects author information on original publication. On publication of this collection, the author's current credentials are Laura E. Peycke, DVM, MS, DACVS, CCRP.

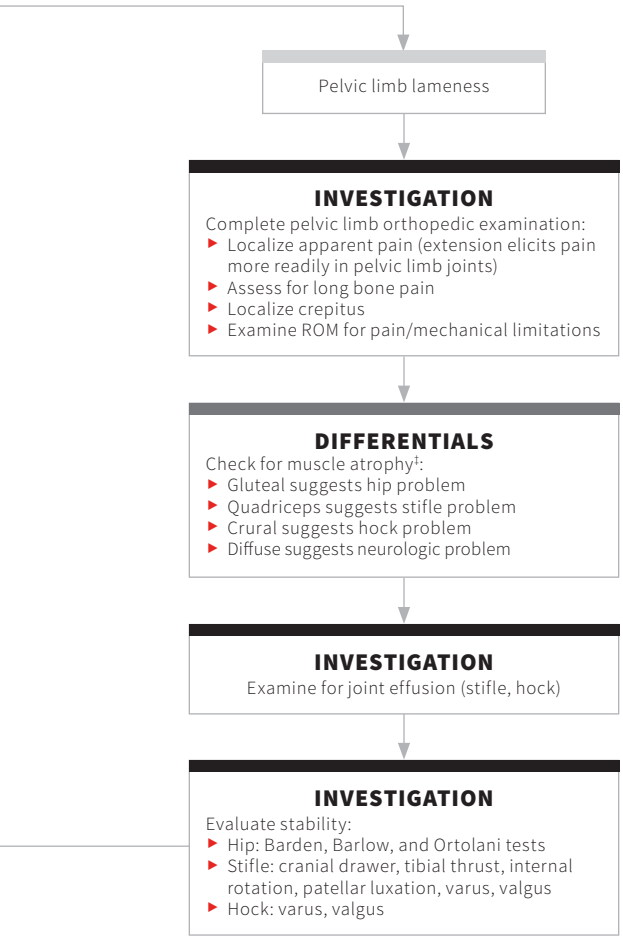
CT = computed tomography
MN = mononuclear cells

LAMENESS IN DOGS

James L. Cook, DVM, PhD, DACVS, DACVSMR*
University of Missouri



*Byline reflects author information on original publication. On publication of this collection, the author's current credentials are James L. Cook, DVM, PhD, DACVS, DACVSMR, OTSC.



ROM = range of motion

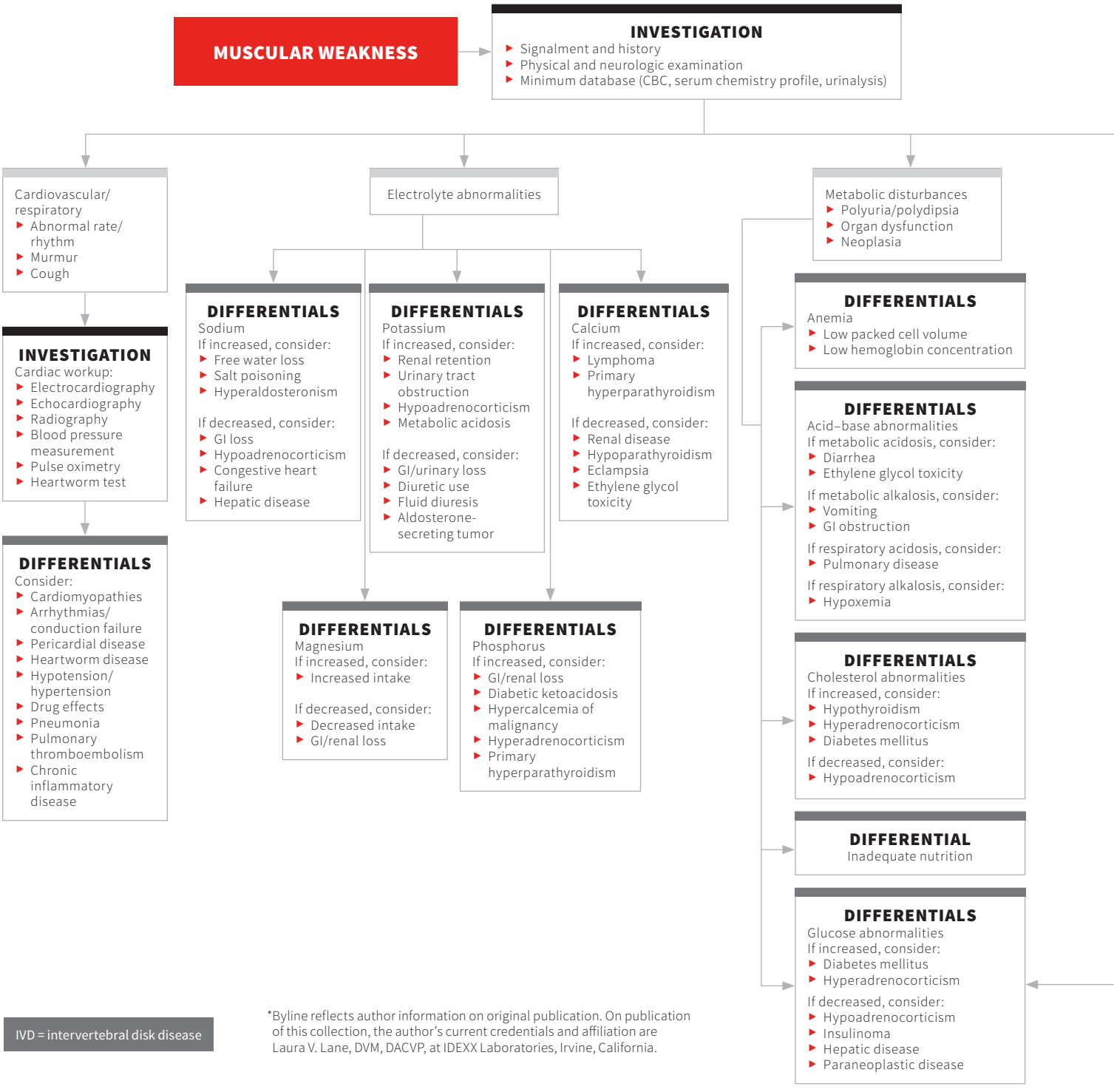
ORTHOPEDIC EXAMINATION: TECHNIQUES & SIGNS

- **Stance & posture**: Look for externally rotated limb, shifting weight off ≥1 limbs, sitting with ≥1 pelvic limbs extended (ie, sit test), elbows abducted, and pelvic limb adducted.
- **Stride length & duration**: Evaluate walk and trot for stride length symmetry and duration of stance; shortened stride, decreased stance time, and stilted gait indicate lameness and can help localize if asymmetric.
- **Limb tracking**: Look for limb crossing the center line during straight line walk or trot, circumduction of limb, wide-based in thoracic and pelvic limbs, and asymmetry of limb tracking.
- **Symmetry**: Examine for above signs plus loading (ie, pad flare), joint angles, swing phase duration and distance, pelvic excursion (ie, hip hike), turning (eg, circles, figure-8 patterns), and ascents and descents.
- **Head bob**: Check whether head is lifted when affected thoracic limb is in stance phase (ie, up on lame, down on sound).

†Nerve root signature is a common cause of lameness.
‡When localizing a problem, evaluating apparent pain may be less effective than identifying muscle atrophy, as it is difficult to flex one joint without affecting others, either by movement or engagement of muscles that cross multiple muscles (eg, biceps during elbow extension).

MUSCULAR WEAKNESS

Laura V. Lane, DVM*
Theresa E. Rizzi, DVM, DACVP
Oklahoma State University



IVD = intervertebral disk disease

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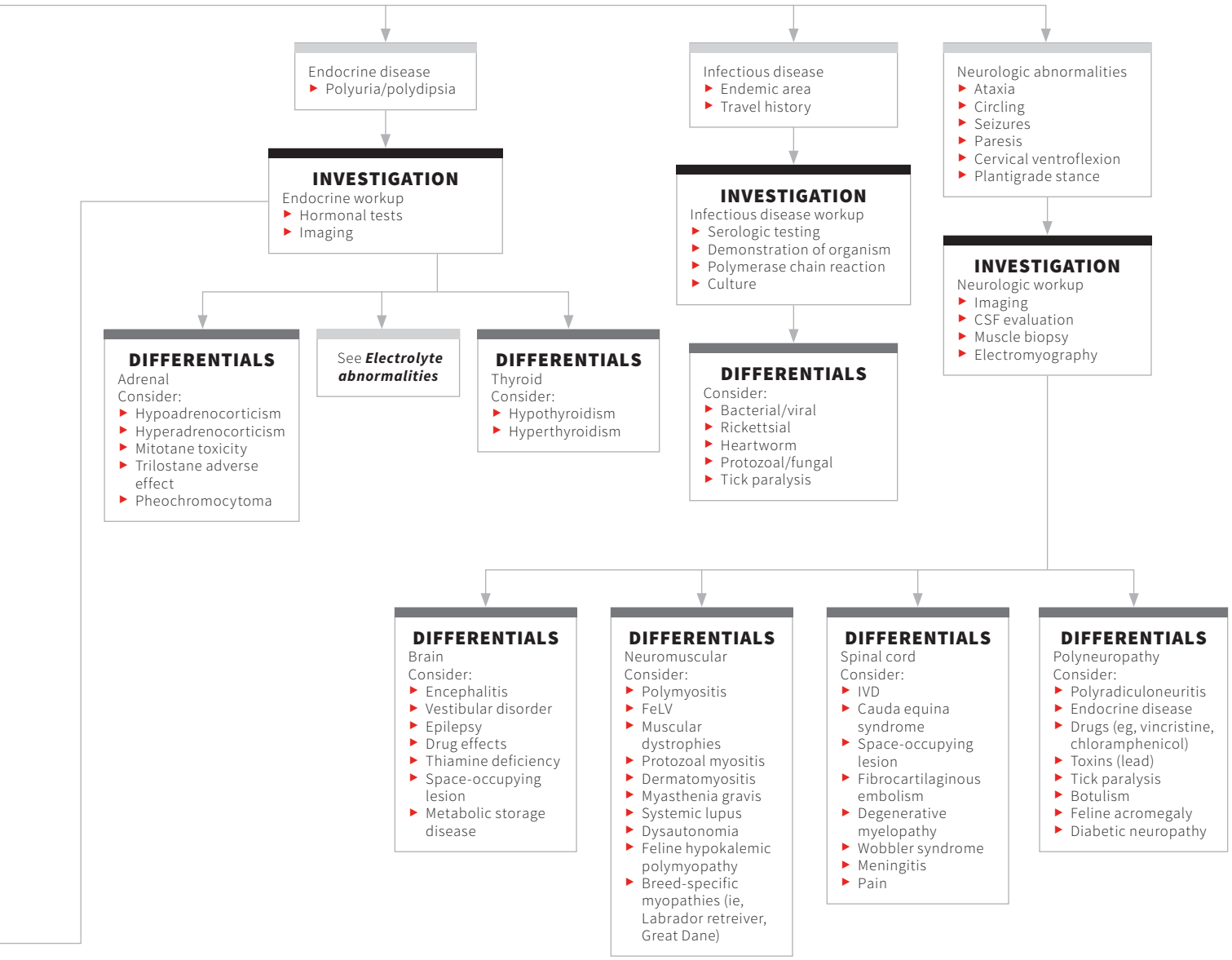
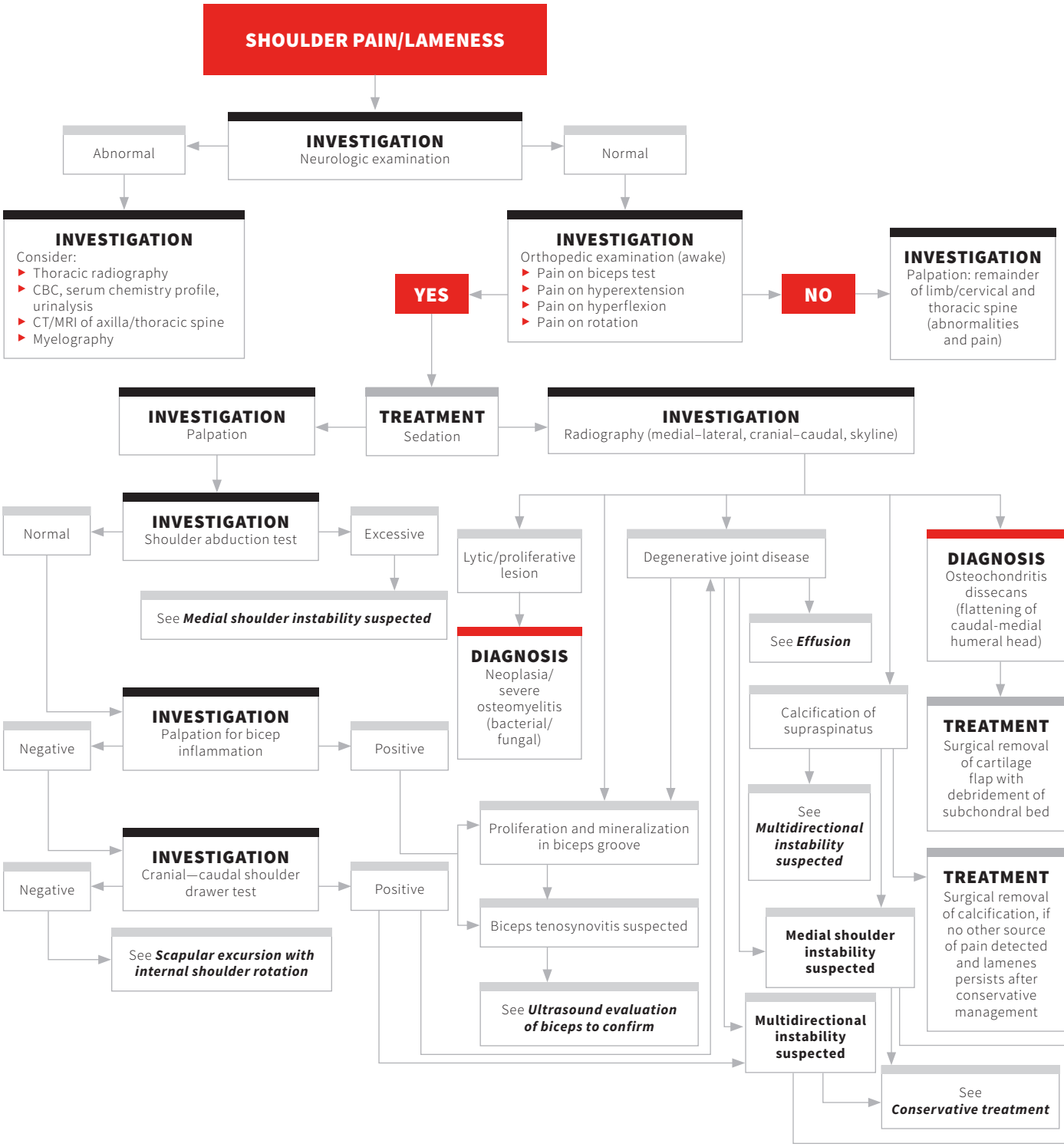
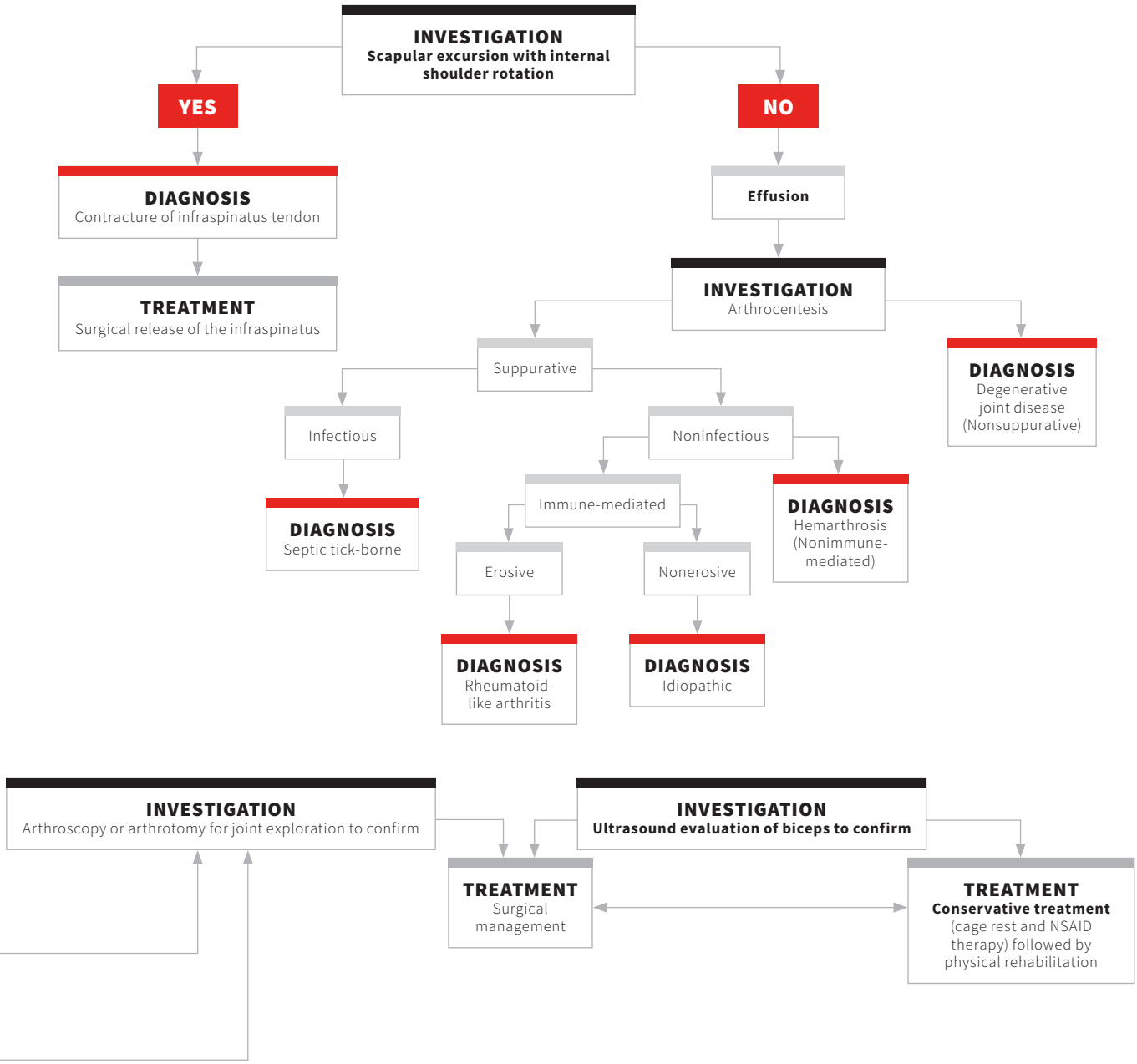

Endocrine disease
Polyuria/polydipsia
INVESTIGATION
Endocrine workup
Hormonal tests
Imaging
DIFFERENTIALS
Adrenal
Consider:
Hypoadrenocorticism
Hyperadrenocorticism
Mitotane toxicity
Trilostane adverse effect
Pheochromocytoma
See Electrolyte abnormalities
DIFFERENTIALS
Thyroid
Consider:
Hypothyroidism
Hyperthyroidism
Infectious disease
Endemic area
Travel history
INVESTIGATION
Infectious disease workup
Serologic testing
Demonstration of organism
Polymerase chain reaction
Culture
DIFFERENTIALS
Consider:
Bacterial/viral
Rickettsial
Heartworm
Protozoal/fungal
Tick paralysis
Neurologic abnormalities
Ataxia
Circling
Seizures
Paresis
Cervical ventroflexion
Plantigrade stance
INVESTIGATION
Neurologic workup
Imaging
CSF evaluation
Muscle biopsy
Electromyography
DIFFERENTIALS
Brain
Consider:
Encephalitis
Vestibular disorder
Epilepsy
Drug effects
Thiamine deficiency
Space-occupying lesion
Metabolic storage disease
DIFFERENTIALS
Neuromuscular
Consider:
Polymyositis
FeLV
Muscular dystrophies
Protozoal myositis
Dermatomyositis
Myasthenia gravis
Systemic lupus
Dysautonomia
Feline hypokalemic polymyopathy
Breed-specific myopathies (ie, Labrador retreiver, Great Dane)
DIFFERENTIALS
Spinal cord
Consider:
IVD
Cauda equina syndrome
Space-occupying lesion
Fibrocartilaginous embolism
Degenerative myelopathy
Wobbler syndrome
Meningitis
Pain
DIFFERENTIALS
Polyneuropathy
Consider:
Polyradiculoneuritis
Endocrine disease
Drugs (eg, vincristine, chloramphenicol)
Toxins (lead)
Tick paralysis
Botulism
Feline acromegaly
Diabetic neuropathy

SHOULDER PAIN/LAMENESS

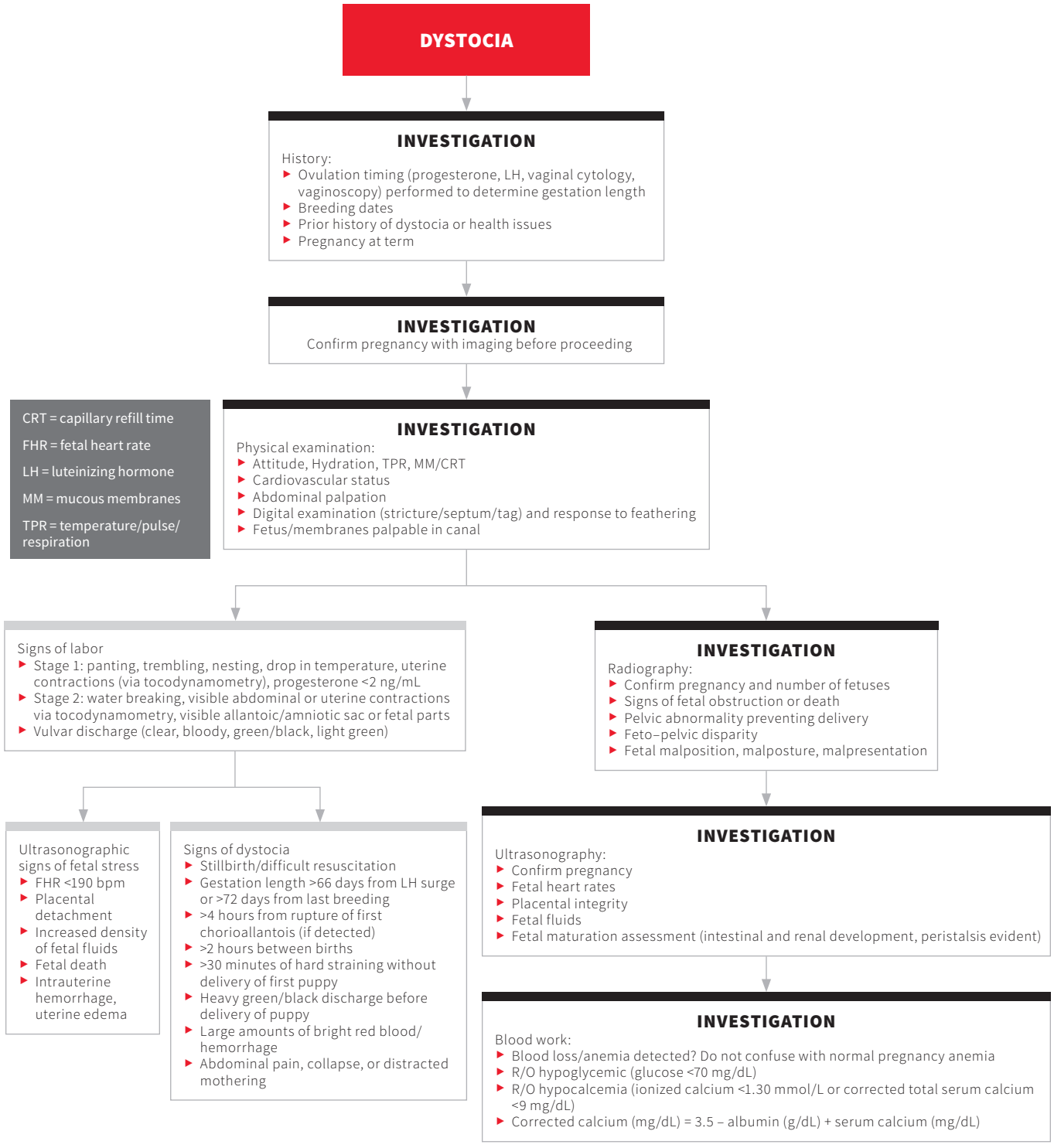
Derek B. Fox, DVM, PhD, DACVS
University of Missouri-Columbia

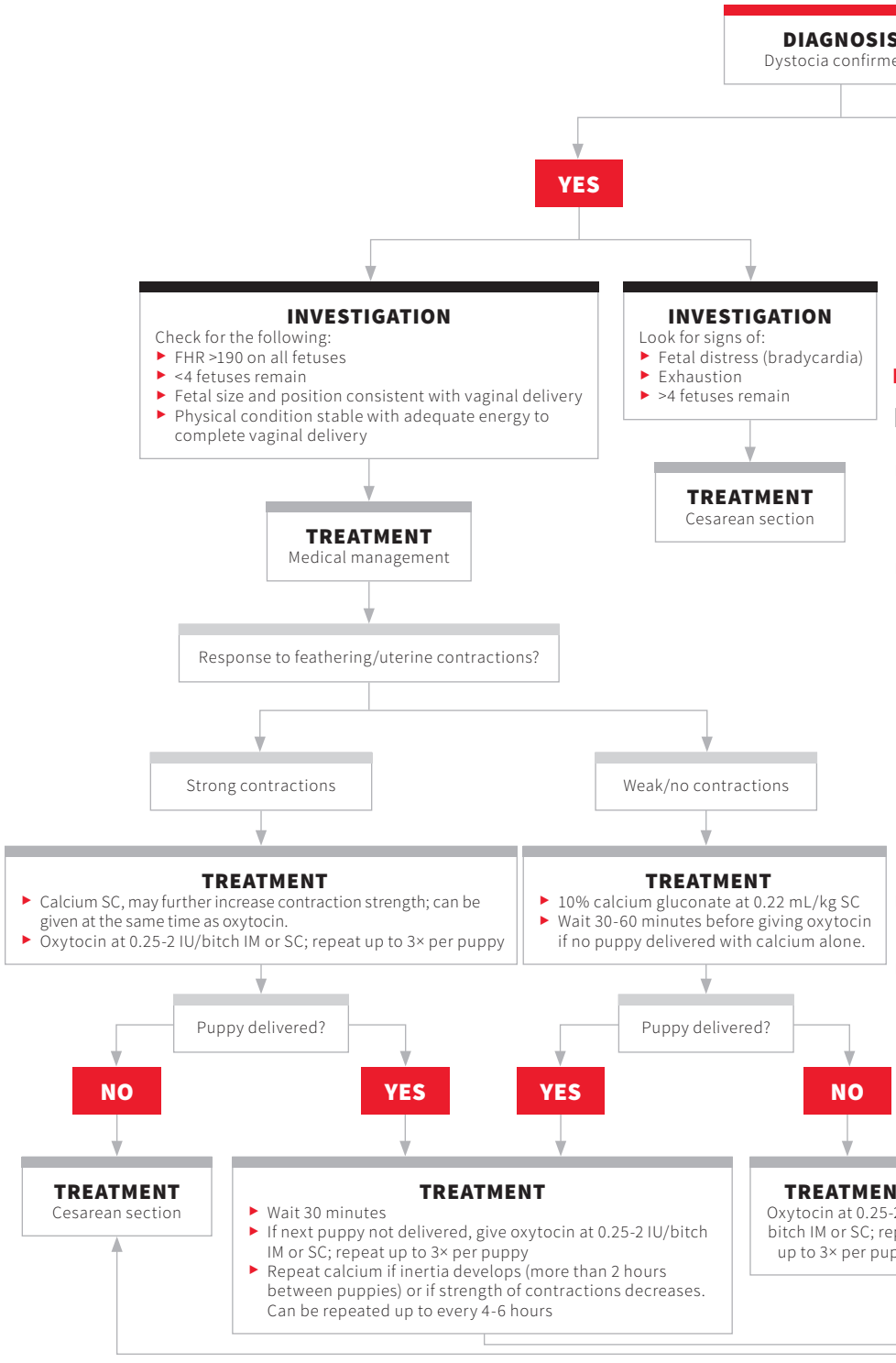



INVESTIGATION
Scapular excursion with internal shoulder rotation
YES
NO
DIAGNOSIS
Contracture of infraspinatus tendon
TREATMENT
Surgical release of the infraspinatus
Effusion
INVESTIGATION
Arthrocentesis
Suppurative
DIAGNOSIS
Degenerative joint disease (Nonsuppurative)
Infectious
Noninfectious
DIAGNOSIS
Septic tick-borne
Immune-mediated
DIAGNOSIS
Hemarthrosis (Nonimmune-mediated)
Erosive
Nonerosive
DIAGNOSIS
Rheumatoid-like arthritis
DIAGNOSIS
Idiopathic
INVESTIGATION
Arthroscopy or arthrotomy for joint exploration to confirm
INVESTIGATION
Ultrasound evaluation of biceps to confirm
TREATMENT
Surgical management
TREATMENT
Conservative treatment (cage rest and NSAID therapy) followed by physical rehabilitation

DYSTOCIA IN THE BITCH

Cheryl Lopate, DVM, MS, DACT
Reproductive Revolutions
Aurora, Oregon
Wilsonville Veterinary Clinic
Wilsonville, Oregon



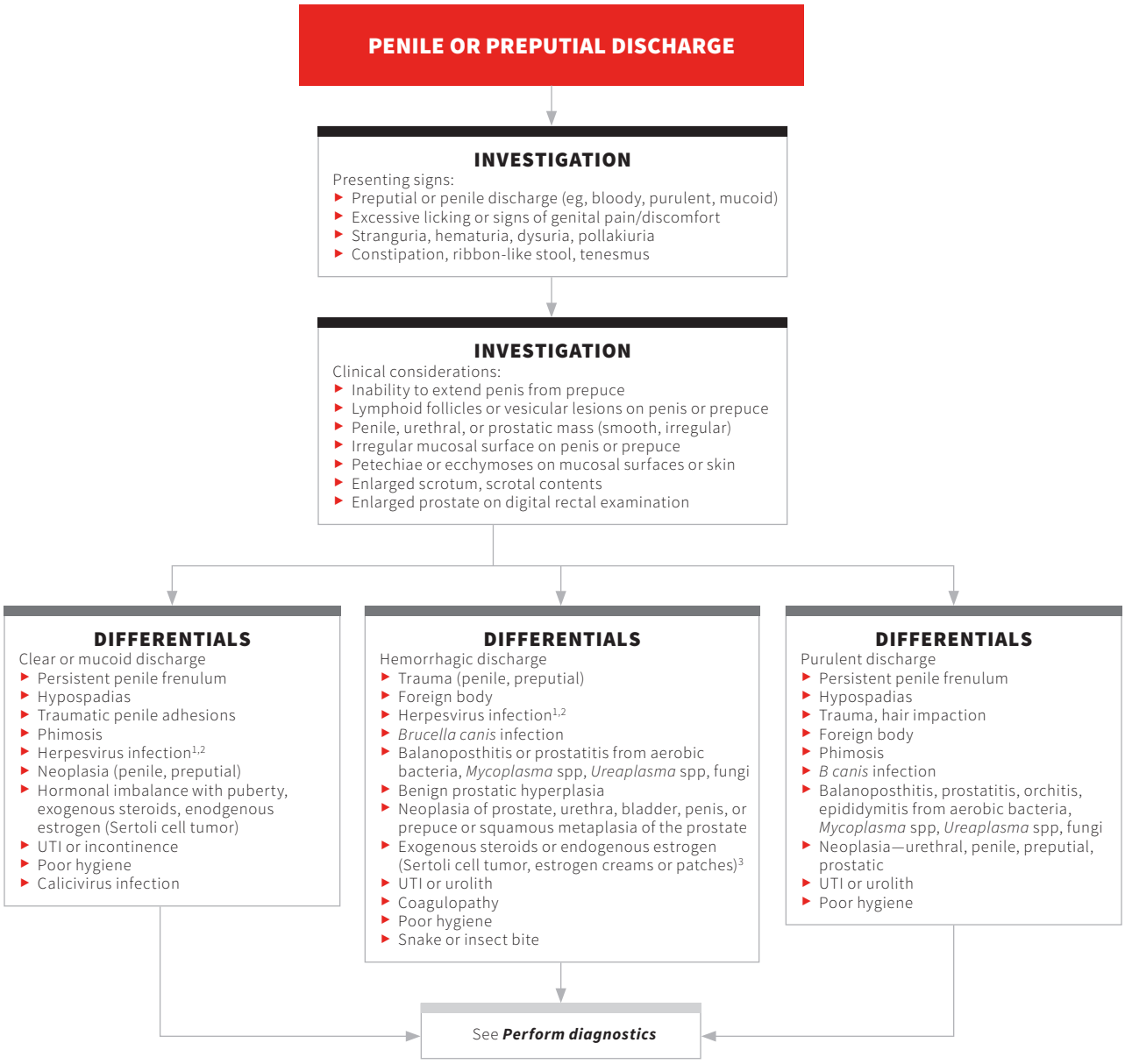


FAST FACTS

- Feathering = repeated strokes with firm pressure on dorsal vestibulovaginal wall; should elicit strong abdominal contraction.
- Calcium may increase the strength of contractions. If given first, then oxytocin may not be necessary.
  - Calcium gluconate 10% does not need to be diluted and can be dosed at 0.5 mL/10 lb (4.5 kg) SC.
  - Calcium gluconate 23% should be diluted at least 1:3 to prevent severe tissue reaction or irritation; anecdotal evidence has shown a 1:4 dilution with sterile water may be dosed at 1 mL/10 lb (4.5 kg) SC.
- Calcium may be repeated q4-6h to maintain stronger uterine contractions.
- Oxytocin can result in decreased placental blood flow or premature placental separation, leading to fetal hypoxia, bradycardia, or death. Repeated doses against fetal obstruction can result in uterine rupture. Calcium is safer if there is concern about obstruction.

PENILE DISCHARGE IN DOGS

Cheryl Lopate, DVM, MS, DACT*
Reproductive Revolutions
Aurora, Oregon

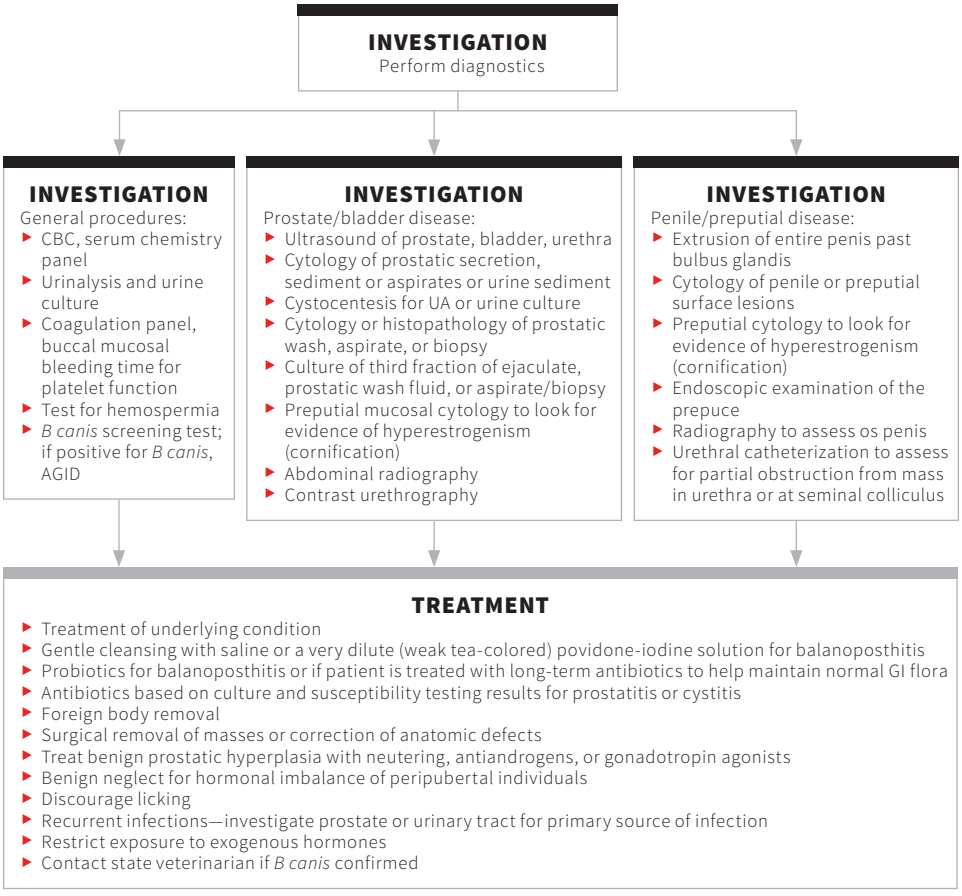


References

1. Hill H, Maré CJ. Genital disease in dogs caused by canine herpesvirus. *Am J Vet Res*. 1974;35:669-672.
2. Anvik JO. Clinical considerations of canine herpesvirus infection. *Vet Med*. 1974;86:394-403.
3. Doig PA, Ruhnke HL, Bosu WT. The genital Mycoplasma and Ureaplasma flora of healthy and diseased dogs. *Can J Comp Med*. 1981;45:233-238.
4. Rosendal S. Canine mycoplasmas: Their ecologic niche and role in disease. *J Am Vet Med Assoc*. 1982;180:1212-1214.

AGID = agar gel immunodiffusion
UA = urinalysis

*Byline reflects author information on original publication. On publication of this collection, the author is also at Wilsonville Veterinary Clinic, Wilsonville, Oregon.



HOW TO PERFORM PROSTATIC WASH

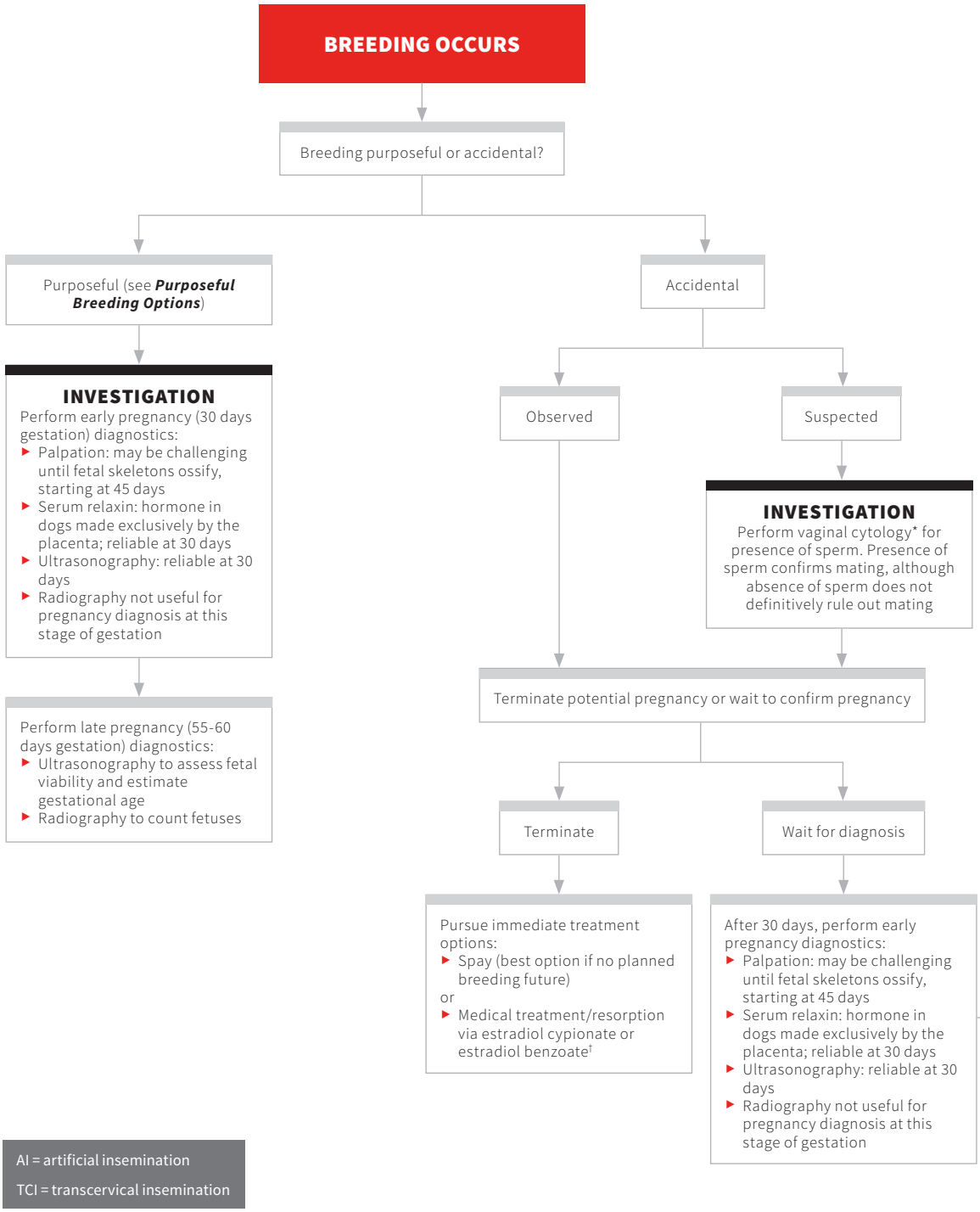
- Allow the patient to urinate
- Sedate the patient
- Empty the dog's bladder via catheter and flush with 5-10 mL of saline, save sample for urine cytology and culture
- Pass a polypropylene or red rubber catheter over the pelvis rim, with a digit in the rectum, position the tip of the catheter just caudal to the prostate
- Vigorously massage the prostate per rectum with the inserted digit
- Occlude the urethral opening and inject 5-10 mL sterile saline
- Advance the catheter forward a few centimeters while aspirating as much sample as possible
- Perform cytology and culture on the recovered sample and compare to urine sample

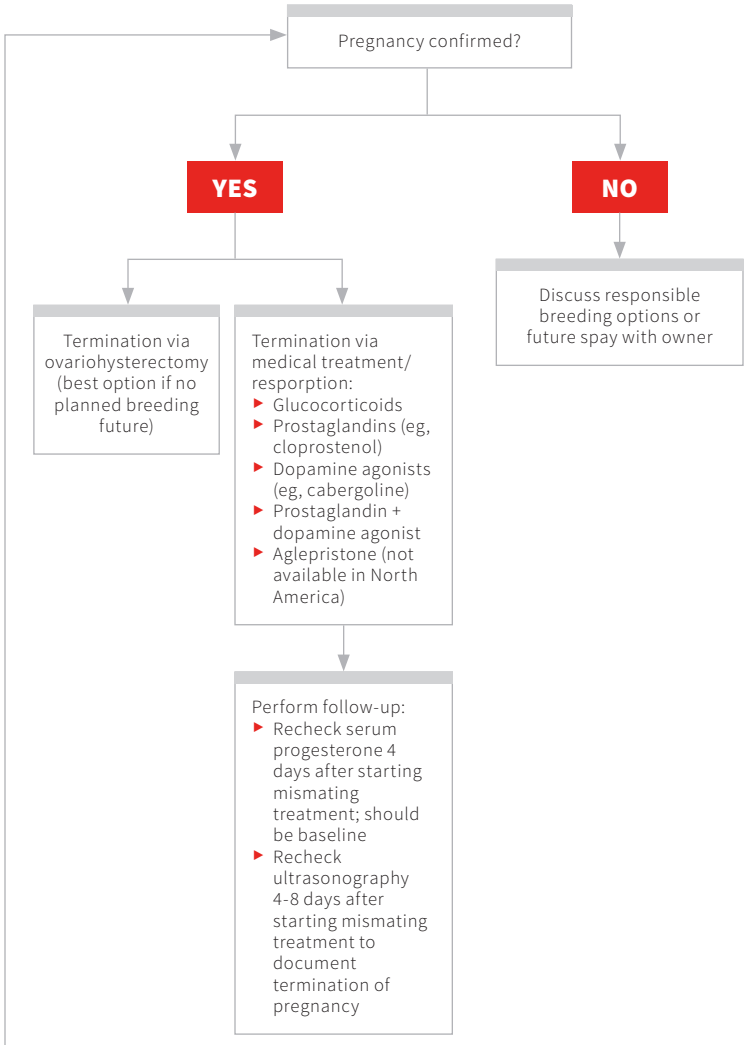
AUTHOR INSIGHT

- To differentiate UTI from prostatic infection, compare culture and cytology of a cystocentesis or catheter-obtained sample with a prostatic fluid wash or prostatic aspirate sample.
- All bacteria that can cause pathology can also be normal flora. High numbers of a single organism along with signs of infection or inflammation suggest the organism is pathologic. Mixed populations indicate normal flora; antibiotics are not required. *Mycoplasma* spp is a common co-isolate when infection is present, but it may not indicate pathologic infection.[4] Common normal floras include *Escherichia coli*, *Pseudomonas* spp, *Staphylococcus* spp, *Streptococcus* spp, *Pasteurella* spp, *Klebsiella* spp, and *Mycoplasma* spp.
- Some yellowish-white to slight light-greenish-tinged preputial discharge is normal. Intact and brachycephalic dogs tend to have increased normal discharge. The amount of normal discharge tends to increase with age, as self-grooming diminishes with aging.
- Infection may be secondary to inappropriate or prolonged antibiotic therapy and bacterial overgrowth of pathologic organisms.

PREGNANCY IN DOGS

Bruce W. Christensen, DVM, MS, DACT
Kokopelli Assisted Reproductive Services
Woodland, California





PURPOSEFUL BREEDING OPTIONS

Breeding should be timed and managed using vaginal cytology, vaginoscopy, and progesterone to determine luteinizing hormone surge, ovulation date, fertile period, and estimated whelping date. Insemination options include:

- Fresh semen (natural mating, vaginal AI, TCI)
- Chilled/shipped semen (vaginal AI, TCI)
- Frozen semen (TCI, surgical AI)

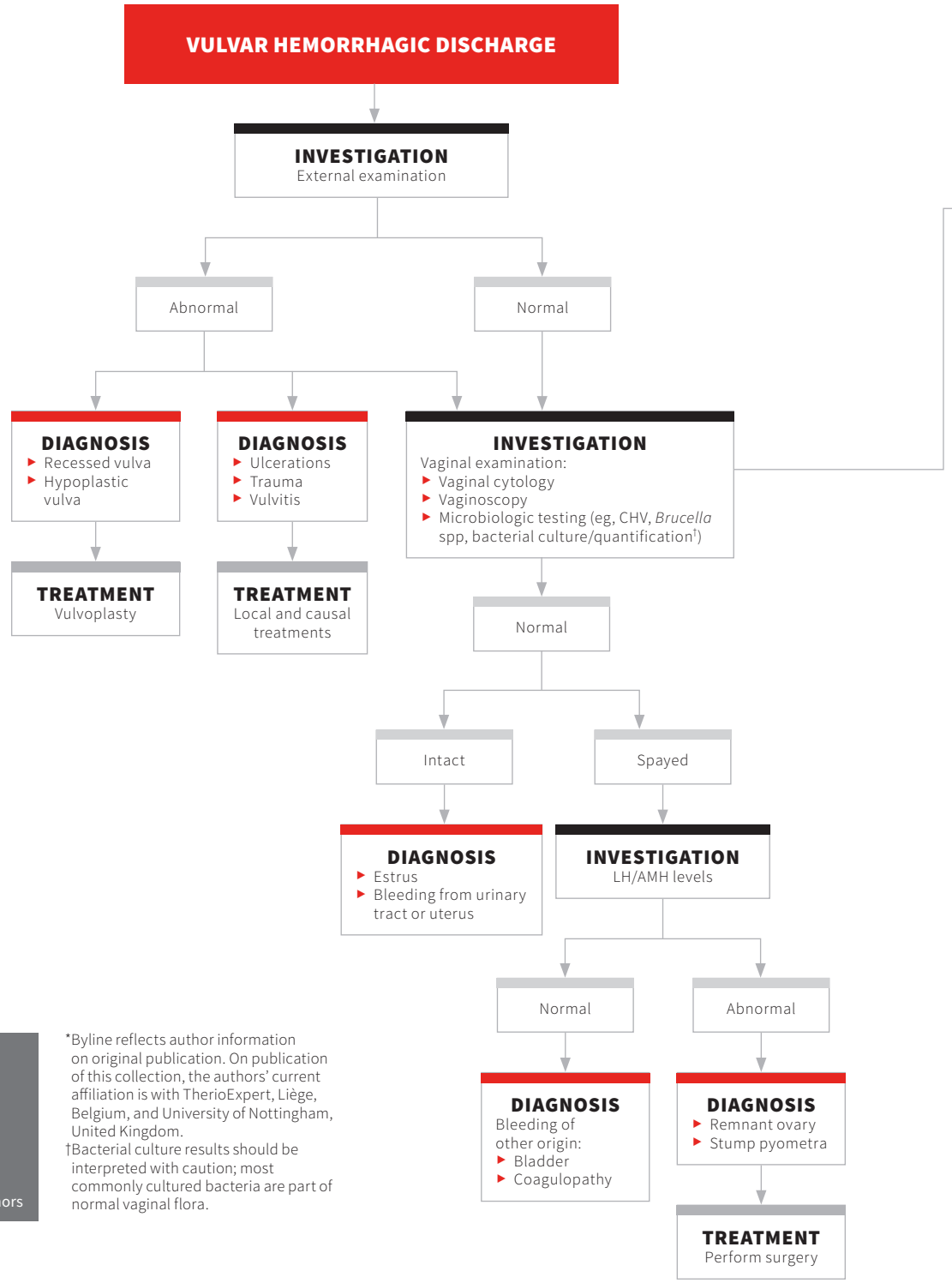
Reference

1. Whitacre MD, Yates DJ, VanCamp SD, Meuten DJ. Detection of intravaginal spermatozoa after natural mating in the bitch. *Vet Clin Pathol*. 1992;21(3):85-87.

*Although negative predictive value of vaginal cytology within the first 24 hours of mating has been reported to be as great as the positive predictive value,[1] the possibility of a false negative still warrants confirming pregnancy later. Therefore, the value of performing vaginal cytology is questionable.
†Must administer within 1-2 days of mating. Use caution with timing and dose. Adverse effects include aplastic anemia and pyometra.

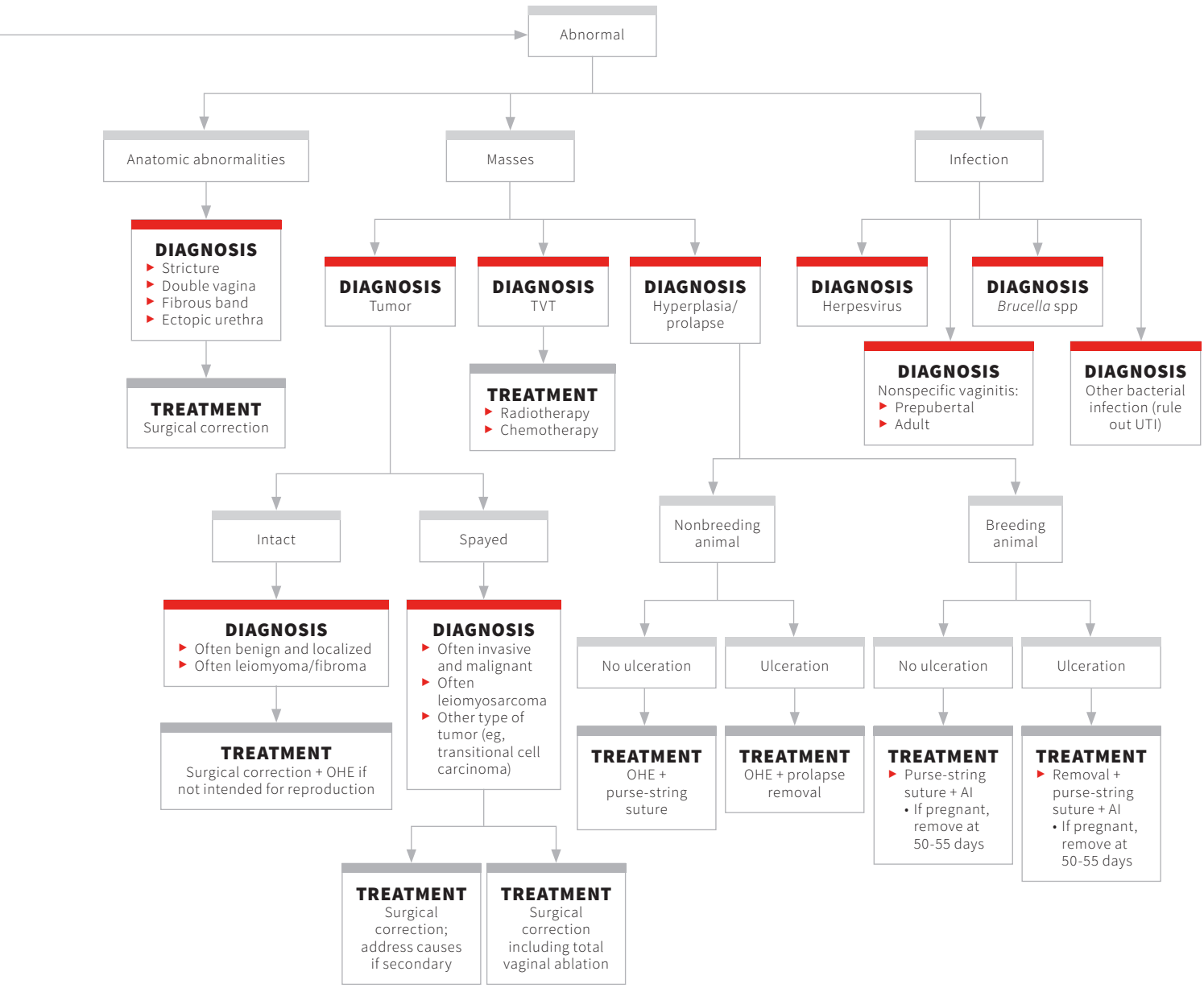
VULVAR HEMORRHAGIC DISCHARGE

John P. Verstegen III, DVM, MSc, PhD, DECAR*
Karine J. Onclin, DVM, PhD, DECAR*
University of Florida



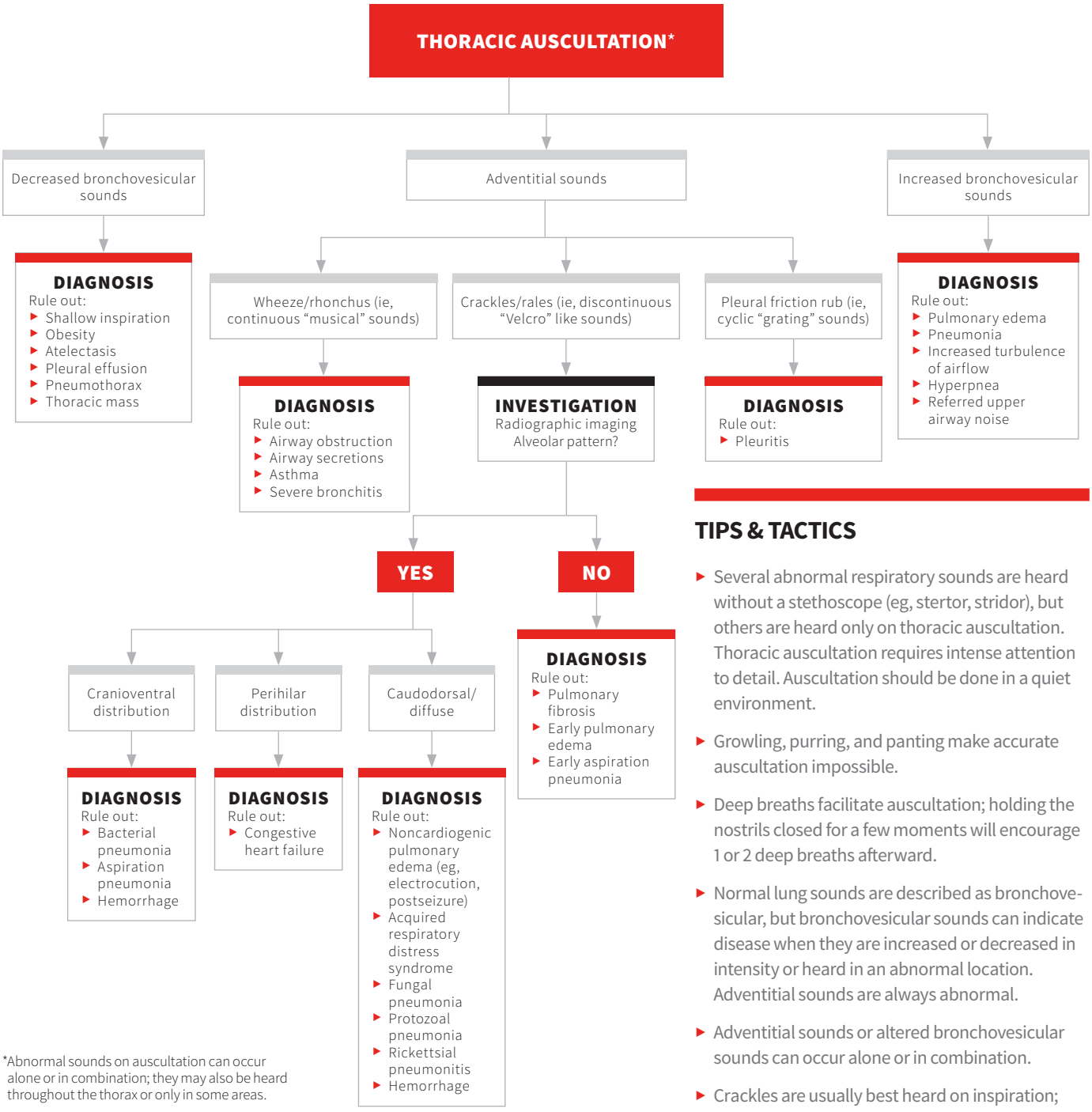
AI = artificial insemination
AMH = anti-Müllerian hormone
CHV = canine herpesvirus
LH = luteinizing hormone
OHE = ovariohysterectomy
TVT = transmissible venereal tumors

*Byline reflects author information on original publication. On publication of this collection, the authors' current affiliation is with TherioExpert, Liège, Belgium, and University of Nottingham, United Kingdom.
†Bacterial culture results should be interpreted with caution; most commonly cultured bacteria are part of normal vaginal flora.


Abnormal
Anatomic abnormalities
Masses
Infection
DIAGNOSIS
Stricture
Double vagina
Fibrous band
Ectopic urethra
TREATMENT
Surgical correction
DIAGNOSIS
Tumor
DIAGNOSIS
TVT
DIAGNOSIS
Hyperplasia/ prolapse
TREATMENT
Radiotherapy
Chemotherapy
DIAGNOSIS
Herpesvirus
DIAGNOSIS
Brucella spp
DIAGNOSIS
Nonspecific vaginitis:
Prepubertal
Adult
DIAGNOSIS
Other bacterial infection (rule out UTI)
Intact
Spayed
Nonbreeding animal
Breeding animal
DIAGNOSIS
Often benign and localized
Often leiomyoma/fibroma
TREATMENT
Surgical correction + OHE if not intended for reproduction
DIAGNOSIS
Often invasive and malignant
Often leiomyosarcoma
Other type of tumor (eg, transitional cell carcinoma)
No ulceration
Ulceration
No ulceration
Ulceration
TREATMENT
OHE + purse-string suture
TREATMENT
OHE + prolapse removal
TREATMENT
Purse-string suture + AI
• If pregnant, remove at 50-55 days
TREATMENT
Removal + purse-string suture + AI
• If pregnant, remove at 50-55 days
TREATMENT
Surgical correction; address causes if secondary
TREATMENT
Surgical correction including total vaginal ablation

ABNORMAL RESPIRATORY SOUNDS

Leah A. Cohn, DVM, PhD, DACVIM (SAIM)
University of Missouri



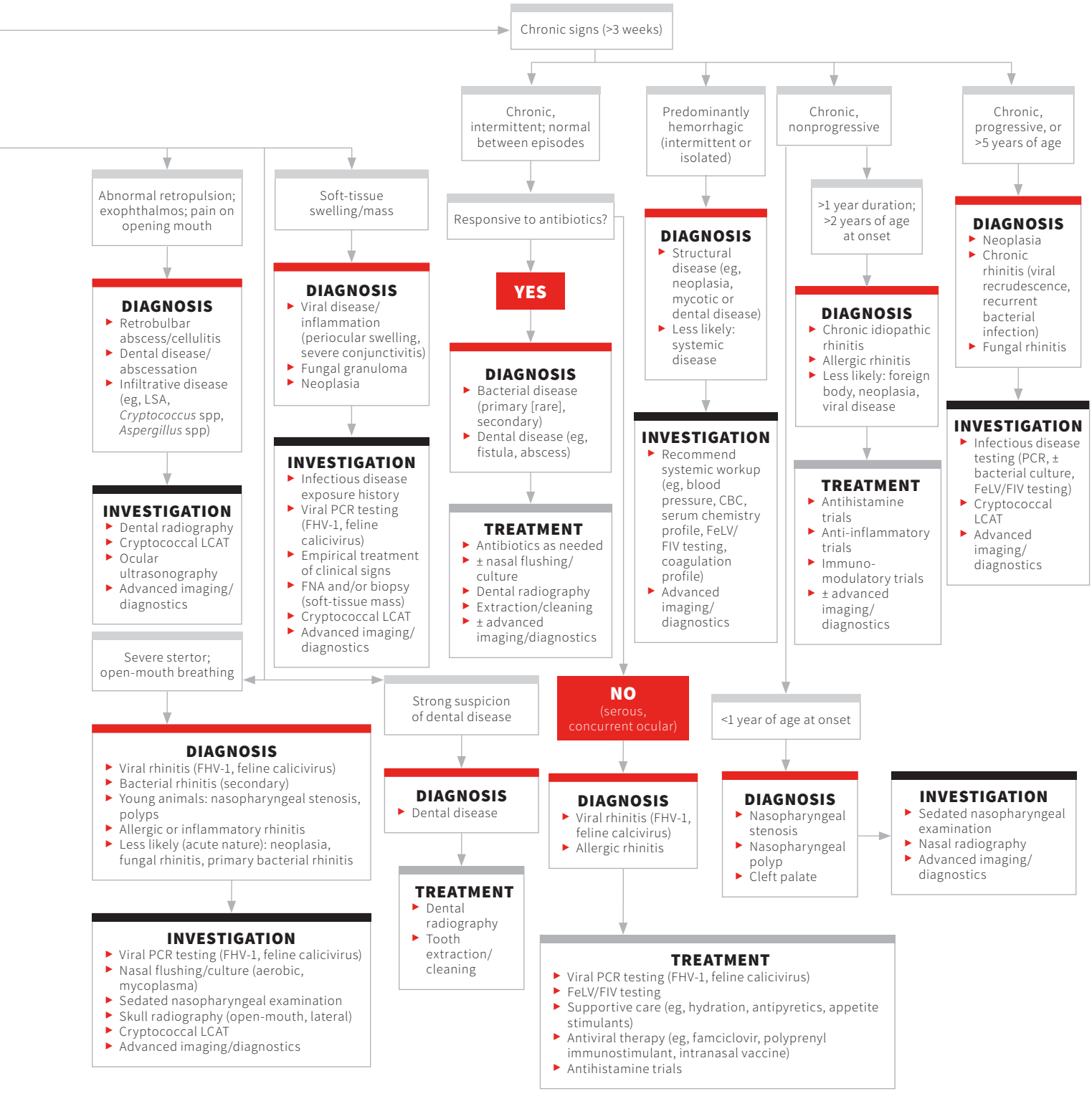
*Abnormal sounds on auscultation can occur alone or in combination; they may also be heard throughout the thorax or only in some areas.

TIPS & TACTICS

- Several abnormal respiratory sounds are heard without a stethoscope (eg, stertor, stridor), but others are heard only on thoracic auscultation. Thoracic auscultation requires intense attention to detail. Auscultation should be done in a quiet environment.
- Growling, purring, and panting make accurate auscultation impossible.
- Deep breaths facilitate auscultation; holding the nostrils closed for a few moments will encourage 1 or 2 deep breaths afterward.
- Normal lung sounds are described as bronchovesicular, but bronchovesicular sounds can indicate disease when they are increased or decreased in intensity or heard in an abnormal location. Adventitial sounds are always abnormal.
- Adventitial sounds or altered bronchovesicular sounds can occur alone or in combination.
- Crackles are usually best heard on inspiration; inducing a cough may facilitate auscultation when the cough subsides.

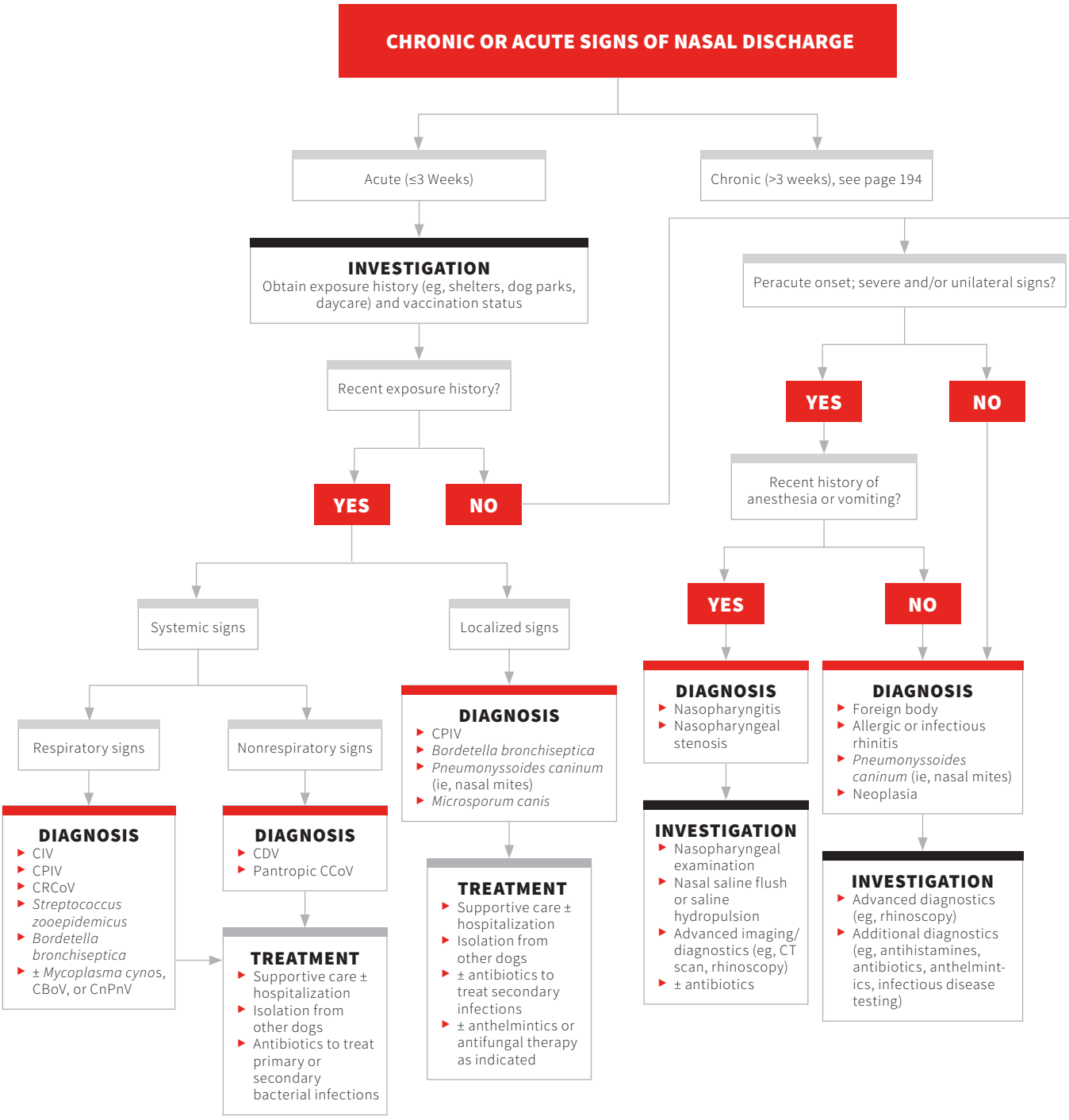
NASAL DISCHARGE IN CATS

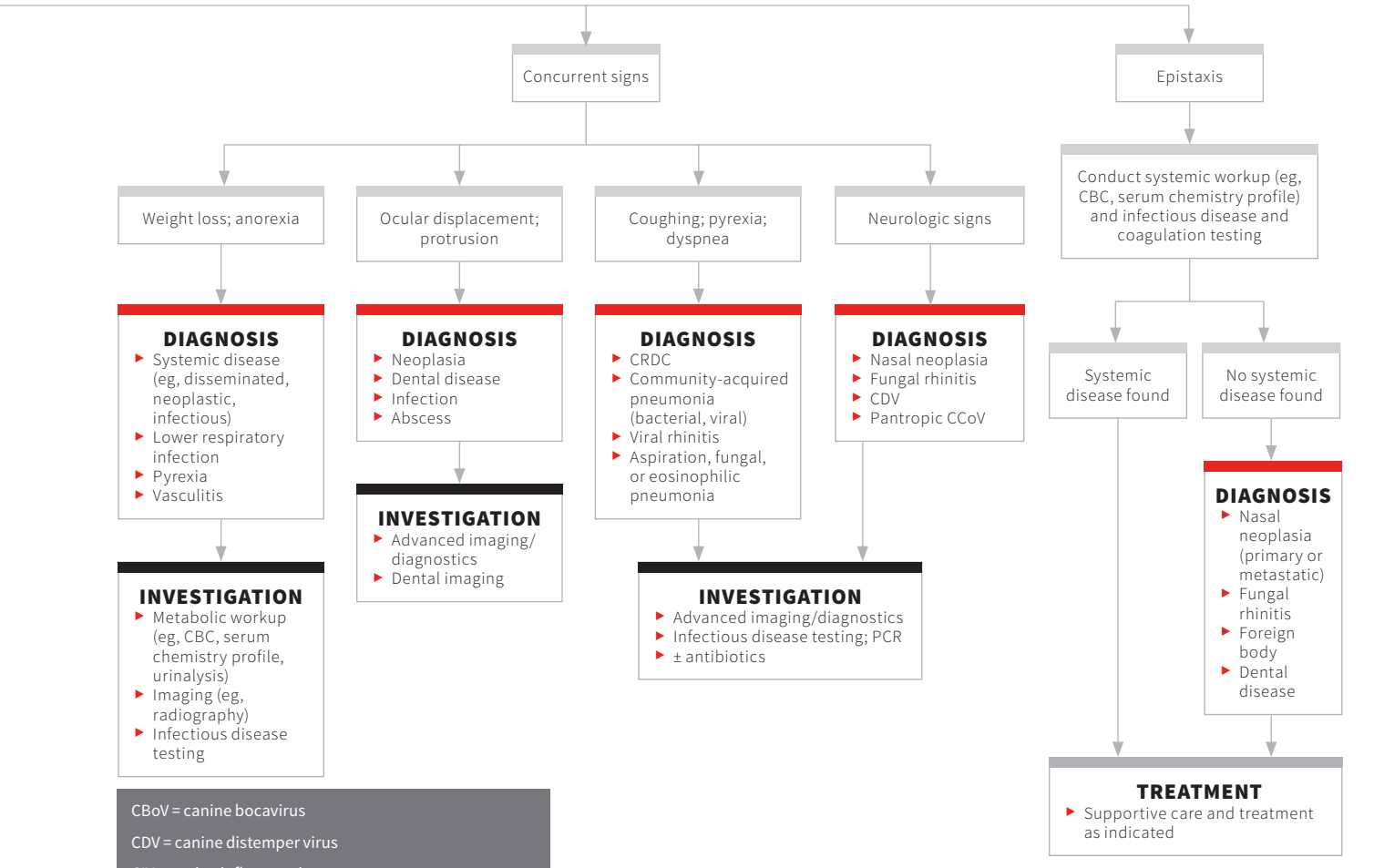
Douglas Palma, DVM, DACVIM (SAIM)
The Animal Medical Center
New York, New York


Chronic signs (>3 weeks)
Chronic, intermittent; normal between episodes
Predominantly hemorrhagic (intermittent or isolated)
Chronic, nonprogressive
Chronic, progressive, or >5 years of age
Abnormal retropulsion; exophthalmos; pain on opening mouth
Soft-tissue swelling/mass
Responsive to antibiotics?
YES
NO (serous, concurrent ocular)
>1 year duration; >2 years of age at onset
<1 year of age at onset
Severe stertor; open-mouth breathing
Strong suspicion of dental disease
DIAGNOSIS
Retrobulbar abscess/cellulitis
Dental disease/ abscessation
Infiltrative disease (eg, LSA, Cryptococcus spp, Aspergillus spp)
Viral disease/ inflammation (periocular swelling, severe conjunctivitis)
Fungal granuloma
Neoplasia
Bacterial disease (primary [rare], secondary)
Dental disease (eg, fistula, abscess)
Structural disease (eg, neoplasia, mycotic or dental disease)
Less likely: systemic disease
Chronic idiopathic rhinitis
Allergic rhinitis
Less likely: foreign body, neoplasia, viral disease
Neoplasia
Chronic rhinitis (viral recrudescence, recurrent bacterial infection)
Fungal rhinitis
Viral rhinitis (FHV-1, feline calicivirus)
Bacterial rhinitis (secondary)
Young animals: nasopharyngeal stenosis, polyps
Allergic or inflammatory rhinitis
Less likely (acute nature): neoplasia, fungal rhinitis, primary bacterial rhinitis
Dental disease
Viral rhinitis (FHV-1, feline calicivirus)
Allergic rhinitis
Nasopharyngeal stenosis
Nasopharyngeal polyp
Cleft palate
INVESTIGATION
Dental radiography
Cryptococcal LCAT
Ocular ultrasonography
Advanced imaging/ diagnostics
Infectious disease exposure history
Viral PCR testing (FHV-1, feline calicivirus)
Empirical treatment of clinical signs
FNA and/or biopsy (soft-tissue mass)
Cryptococcal LCAT
Advanced imaging/ diagnostics
Recommend systemic workup (eg, blood pressure, CBC, serum chemistry profile, FeLV/ FIV testing, coagulation profile)
Advanced imaging/ diagnostics
Infectious disease testing (PCR, ± bacterial culture, FeLV/FIV testing)
Cryptococcal LCAT
Advanced imaging/ diagnostics
Viral PCR testing (FHV-1, feline calicivirus)
Nasal flushing/culture (aerobic, mycoplasma)
Sedated nasopharyngeal examination
Skull radiography (open-mouth, lateral)
Cryptococcal LCAT
Advanced imaging/diagnostics
Sedated nasopharyngeal examination
Nasal radiography
Advanced imaging/ diagnostics
TREATMENT
Antibiotics as needed
± nasal flushing/ culture
Dental radiography
Extraction/cleaning
± advanced imaging/diagnostics
Antihistamine trials
Anti-inflammatory trials
Immuno-modulatory trials
± advanced imaging/ diagnostics
Dental radiography
Tooth extraction/ cleaning
Viral PCR testing (FHV-1, feline calicivirus)
FeLV/FIV testing
Supportive care (eg, hydration, antipyretics, appetite stimulants)
Antiviral therapy (eg, famciclovir, polyprenyl immunostimulant, intranasal vaccine)
Antihistamine trials

NASAL DISCHARGE IN DOGS

Douglas Palma, DVM, DACVIM (SAIM)
The Animal Medical Center
New York City, New York





CBoV = canine bocavirus
CDV = canine distemper virus
CIV = canine influenza virus
CnPnV = canine pneumovirus
CPIV = canine parainfluenza virus
CRCoV = canine respiratory coronavirus
CRDC = canine respiratory disease complex
Pantropic CCoV = pantropic variant of canine coronavirus

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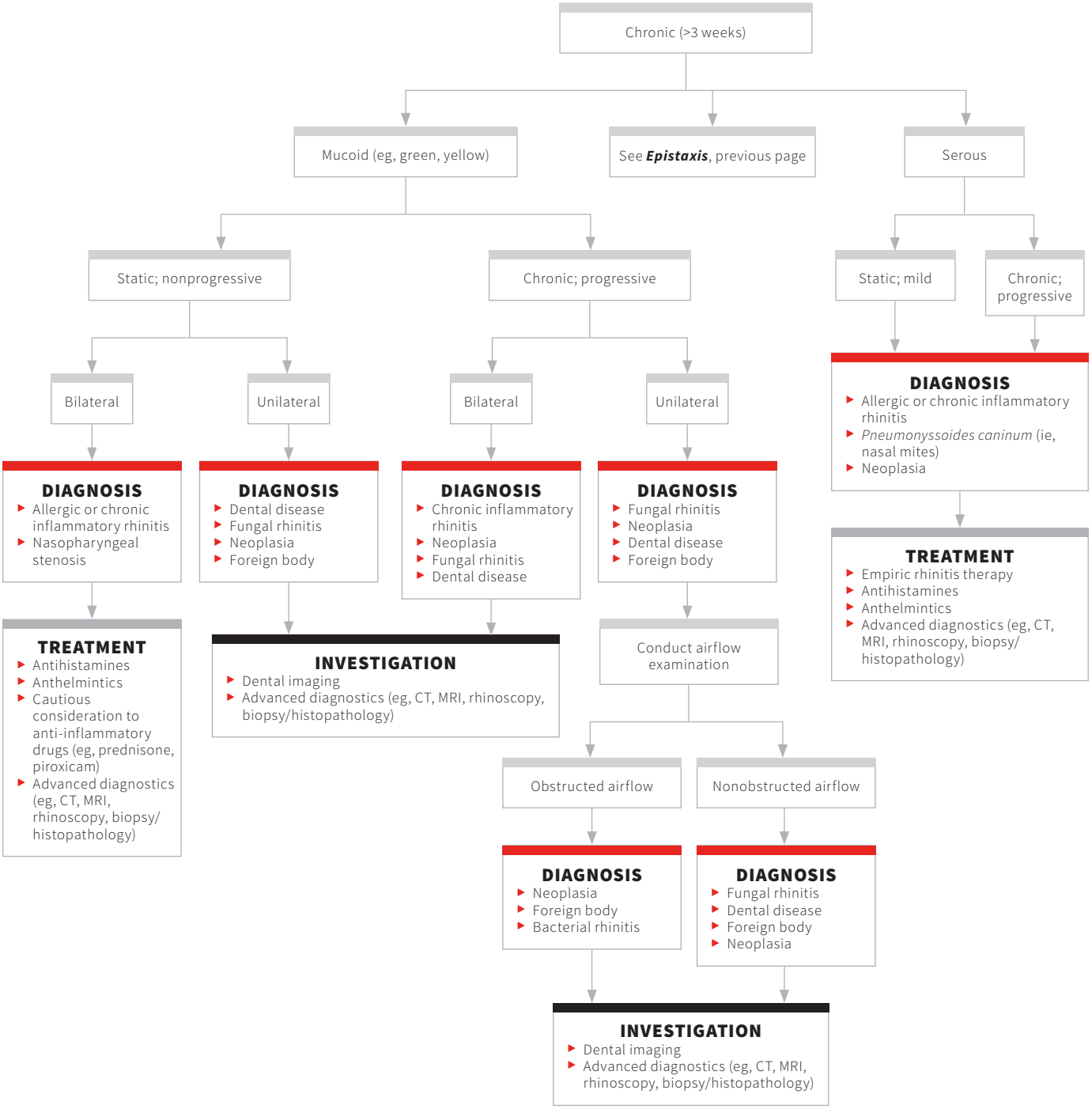
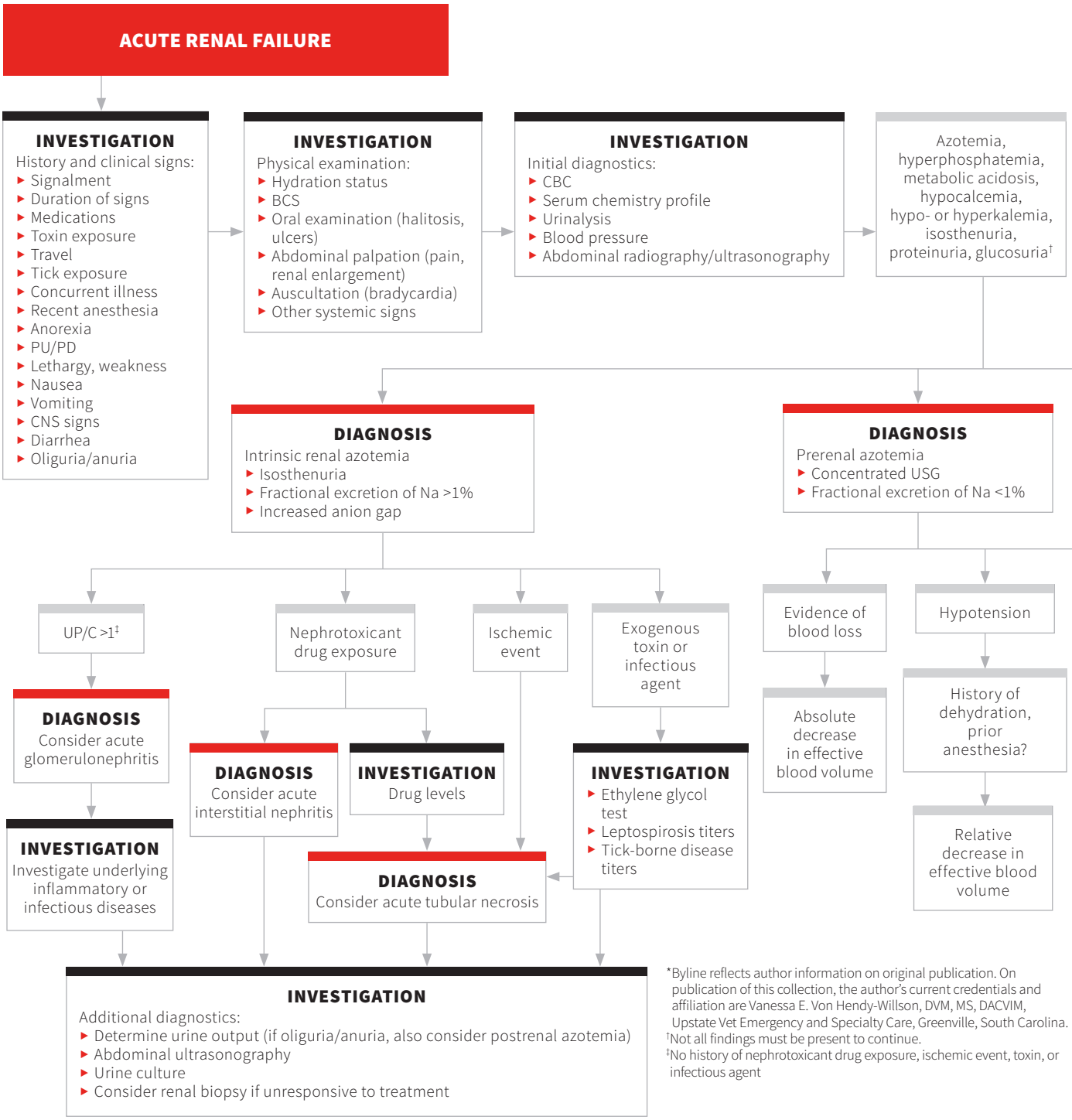
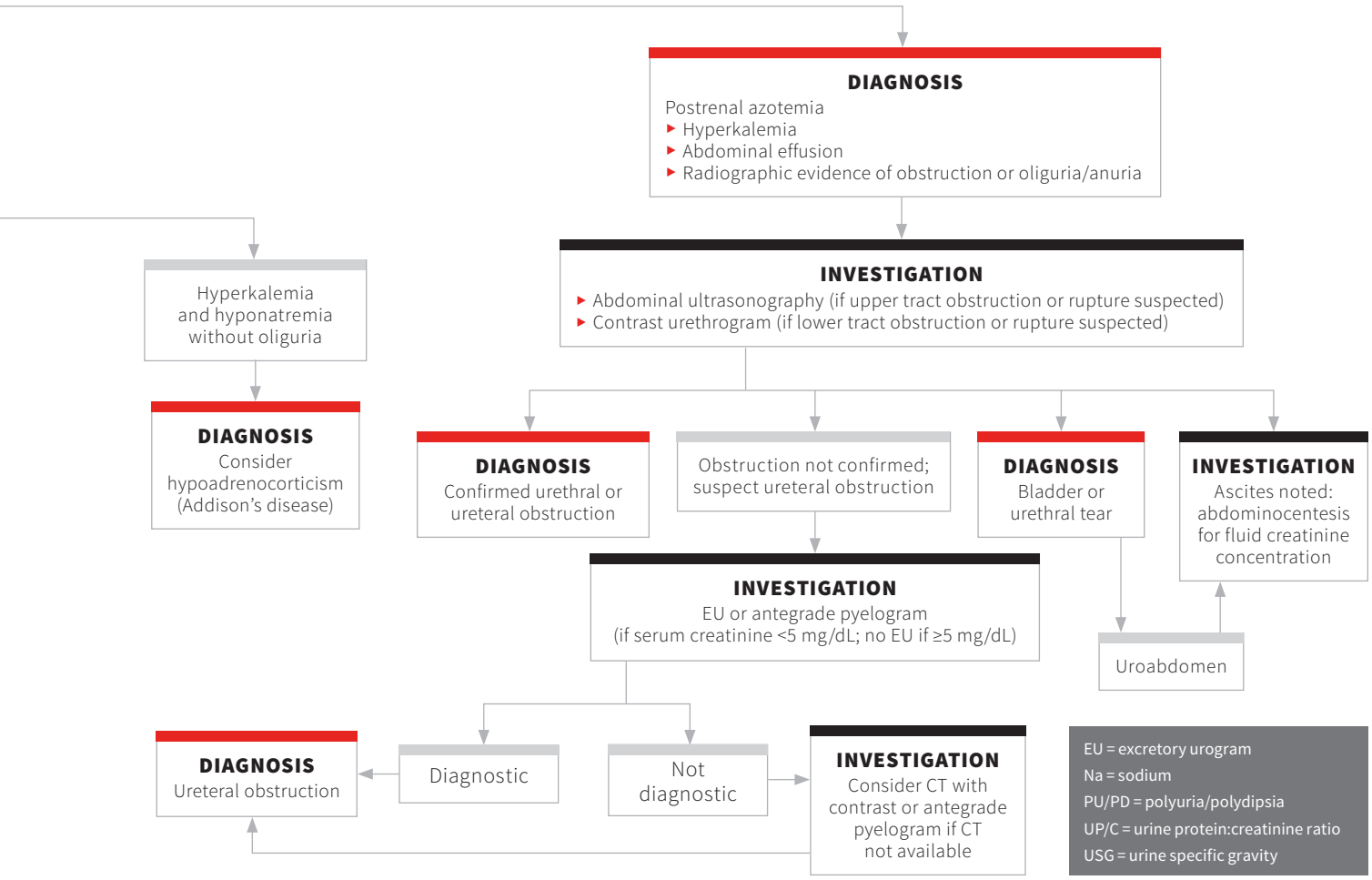

Chronic (>3 weeks)
Mucoid (eg, green, yellow)
See Epistaxis, previous page
Serous
Static; nonprogressive
Chronic; progressive
Static; mild
Chronic; progressive
Bilateral
Unilateral
Bilateral
Unilateral
DIAGNOSIS
Allergic or chronic inflammatory rhinitis
Nasopharyngeal stenosis
DIAGNOSIS
Dental disease
Fungal rhinitis
Neoplasia
Foreign body
DIAGNOSIS
Chronic inflammatory rhinitis
Neoplasia
Fungal rhinitis
Dental disease
DIAGNOSIS
Fungal rhinitis
Neoplasia
Dental disease
Foreign body
DIAGNOSIS
Allergic or chronic inflammatory rhinitis
Pneumonyssoides caninum (ie, nasal mites)
Neoplasia
TREATMENT
Empiric rhinitis therapy
Antihistamines
Anthelmintics
Advanced diagnostics (eg, CT, MRI, rhinoscopy, biopsy/histopathology)
TREATMENT
Antihistamines
Anthelmintics
Cautious consideration to anti-inflammatory drugs (eg, prednisone, piroxicam)
Advanced diagnostics (eg, CT, MRI, rhinoscopy, biopsy/histopathology)
INVESTIGATION
Dental imaging
Advanced diagnostics (eg, CT, MRI, rhinoscopy, biopsy/histopathology)
Conduct airflow examination
Obstructed airflow
Nonobstructed airflow
DIAGNOSIS
Neoplasia
Foreign body
Bacterial rhinitis
DIAGNOSIS
Fungal rhinitis
Dental disease
Foreign body
Neoplasia
INVESTIGATION
Dental imaging
Advanced diagnostics (eg, CT, MRI, rhinoscopy, biopsy/histopathology)

ACUTE RENAL FAILURE

Vanessa E. Von Hendy-Willson, DVM*
Larry G. Adams, DVM, PhD, DACVIM (SAIM)
Purdue University

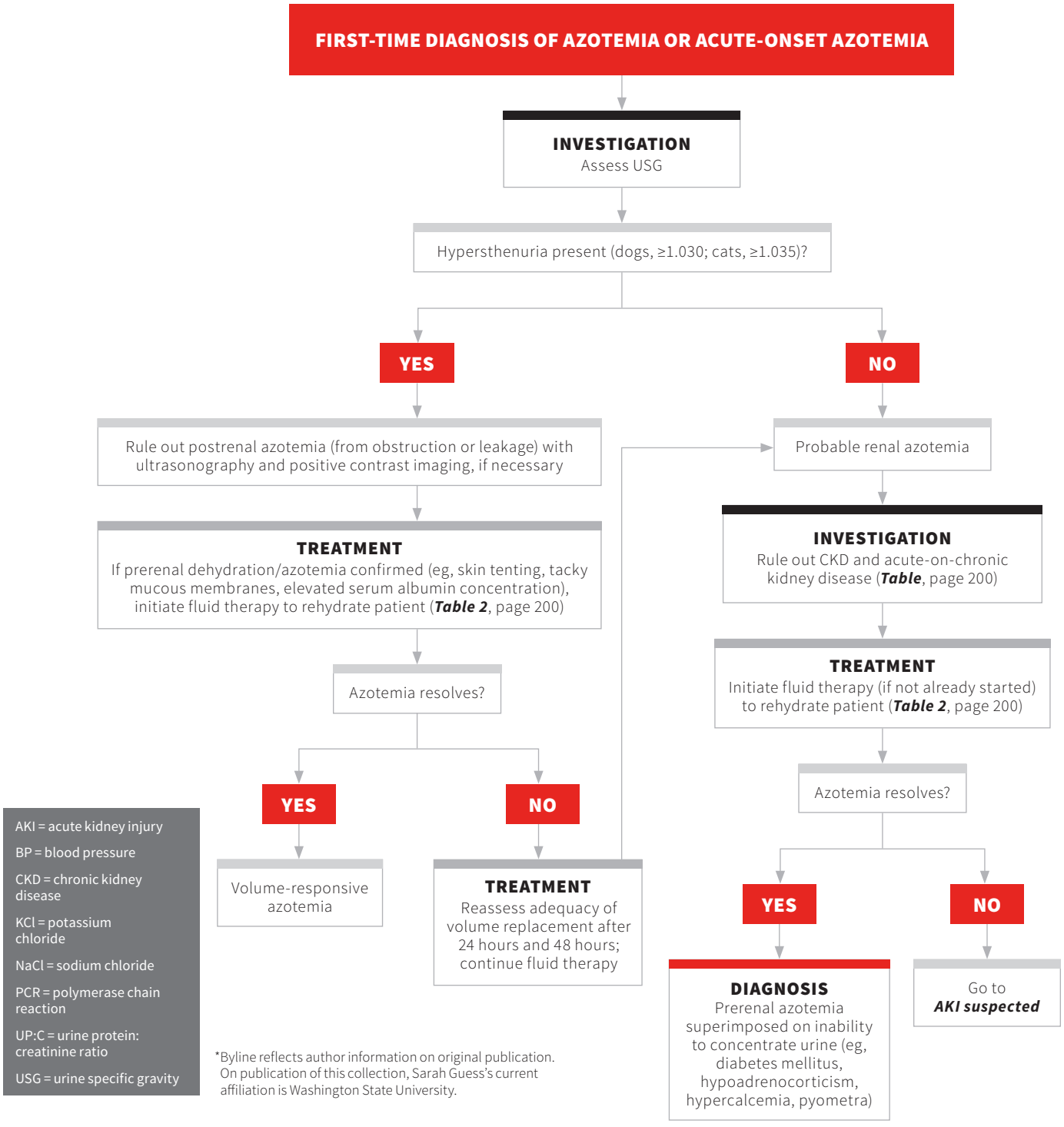


*Byline reflects author information on original publication. On publication of this collection, the author's current credentials and affiliation are Vanessa E. Von Hendy-Willson, DVM, MS, DACVIM, Upstate Vet Emergency and Specialty Care, Greenville, South Carolina.
†Not all findings must be present to continue.
‡No history of nephrotoxicant drug exposure, ischemic event, toxin, or infectious agent


DIAGNOSIS
Postrenal azotemia
▸ Hyperkalemia
▸ Abdominal effusion
▸ Radiographic evidence of obstruction or oliguria/anuria
INVESTIGATION
▸ Abdominal ultrasonography (if upper tract obstruction or rupture suspected)
▸ Contrast urethrogram (if lower tract obstruction or rupture suspected)
Hyperkalemia and hyponatremia without oliguria
DIAGNOSIS
Consider hypoadrenocorticism (Addison's disease)
DIAGNOSIS
Confirmed urethral or ureteral obstruction
Obstruction not confirmed; suspect ureteral obstruction
DIAGNOSIS
Bladder or urethral tear
INVESTIGATION
Ascites noted: abdominocentesis for fluid creatinine concentration
INVESTIGATION
EU or antegrade pyelogram
(if serum creatinine <5 mg/dL; no EU if ≥5 mg/dL)
Uroabdomen
DIAGNOSIS
Ureteral obstruction
Diagnostic
Not diagnostic
INVESTIGATION
Consider CT with contrast or antegrade pyelogram if CT not available
EU = excretory urogram
Na = sodium
PU/PD = polyuria/polydipsia
UP/C = urine protein:creatinine ratio
USG = urine specific gravity

AZOTEMIA & ACUTE KIDNEY INJURY

Gregory F. Grauer, DVM, MS, DACVIM (SAIM)
Sarah Guess, DVM, MS*
Kansas State University



AKI = acute kidney injury
BP = blood pressure
CKD = chronic kidney disease
KCl = potassium chloride
NaCl = sodium chloride
PCR = polymerase chain reaction
UP:C = urine protein: creatinine ratio
USG = urine specific gravity

*Byline reflects author information on original publication. On publication of this collection, Sarah Guess's current affiliation is Washington State University.

AKI SUSPECTED

INVESTIGATION
Perform complete minimum database (ie, CBC, serum chemistry profile with electrolytes, urinalysis with sediment examination, BP)

DIAGNOSTICS
- Leptospirosis serum titers and urine leptospirosis PCR
- Urine culture, UP:C
- Abdominal ultrasonography to find obstruction caused by nephrolithiasis, evidence of pyelonephritis, or abdominal effusion

INVESTIGATION
Rule out CKD via imaging, history, and physical examination (***Table 1***, next page)

AKI confirmed
- Acutely elevated serum creatinine with isosthenuria (USG 1.008-1.012) or minimally concentrated urine (USG 1.013-1.030/1.035)

Cause known?

NO

YES

TREATMENT
Treat underlying condition (eg, toxicity, thromboembolism)

TREATMENT
Initiate fluid therapy and diagnostics for nonspecific AKI. Other therapy may be considered:
- Antihypertensive therapy (eg, amlodipine)
- Antibiotics if pyelonephritis or leptospirosis is suspected; avoid aminoglycosides and other nephrotoxic antibiotics
- Pain medications; avoid NSAIDs
- Nutritional therapy (eg, nasoesophageal tube, esophagostomy tube, total parenteral nutrition) if patient not vomiting
- Treatment for uremic ulcers, if present
- Antiemetics (eg, ondansetron, dolasetron)

TREATMENT
Fluid therapy

STEP 1
Choose replacement fluids (eg, lactated Ringer's solution, 0.9% NaCl [if hyperkalemic])

STEP 2
Estimate dehydration % (eg, skin tenting, capillary refill time, central venous pressure); determine timeline for correction (eg, 4-6 hours; ***Table 2***, next page)

STEP 3
Determine if electrolyte supplementation is needed (eg, KCl ≤0.5 mEq/kg/hr)

STEP 4
Initiate fluid therapy
- Measure body weight every 4-6 hours
- Quantify urine output if possible (***Table 3***, next page)

STEP 5
Reassess
- If weight loss occurs, calculate kg lost × 1000 and add to current fluid rate; recheck weight in 4-6 hours
- Base fluid therapy volume on urine output

STEP 6
- Monitor for signs of over-hydration (eg, increased bronchovesicular sounds, tachycardia, serous nasal discharge, chemosis, weight gain, increased central venous pressure)
- Recheck creatinine and electrolytes every 6-24 hours

Anuria
- Urine output 0 mL/kg/hr, despite adequate rehydration
- Cautious use of volume expansion and/or diuretics because of no proven benefit
- Reassess
- Prognosis is poor, especially if no response to therapy and worsening azotemia

Oliguria
- Urine output <0.5 mL/kg/hr after rehydration
- Cautious use of volume expansion and/or diuretics because of no proven benefit
- Reassess
- Prognosis is guarded, unless converted to polyuria, for which prognosis improves with response to rehydration

Polyuria
- Urine output >1 mL/kg/hr
- Base fluid rate on output and body weight gain or loss
- Prognosis is fair-to-good, especially with resolving azotemia

Continues ▶

TABLE 1

HISTORY, EXAMINATION, & LABORATORY FINDINGS OF CKD & AKI

Findings	CKD	AKI
Weight loss, poor coat condition, poor body condition	+	-
Small, irregular kidneys (on radiography or palpation)	+	-
Nonregenerative anemia	+	-
Acidosis/hyperkalemia	-	+
Oliguria/anuria	-	+
Small, irregular kidneys with hyperechoic cortices with or without loss of corticomedullary junction (on ultrasonography)	+	-
Chronic history of polyuria/polydipsia or stage 1 CKD	+	-
Urine sediment changes compatible with tubular cell damage (eg, granular casts, renal tubular epithelial cells)	-	+
Relatively severe signs for magnitude of azotemia	-	+

+ = presence more likely; - = presence less likely

TABLE 2

EXAMPLE OF FLUID VOLUME REQUIREMENTS FOR A 20-KG DOG WITH 8% DEHYDRATION & AKI

Deficit needs (8% × 20 kg) over 6 hours	= 1600 mL (replace over 4-6 hours)
Maintenance (60 mL/kg/day)	= 1200 mL/day
Continuing losses (dog vomits 4 times at 100 mL/episode)	= 400 mL/day
Total	= 3200 mL/day (deficit replaced over 4-6 hours if possible)

TABLE 3

HYPOTHETICAL COMPARISON OF TOTAL FLUID NEEDS IN NORMAL, OLIGURIC, & POLYURIC DOGS

	Normal	Oliguric	Polyuric
Insensible fluid needs	20 mL/kg/day	20 mL/kg/day	20 mL/kg/day
Sensible fluid needs (urine output)	40 mL/kg/day	6 mL/kg/day	165 mL/kg/day
Total	60 mL/kg/day	26 mL/kg/day	185 mL/kg/day

Suggested Reading

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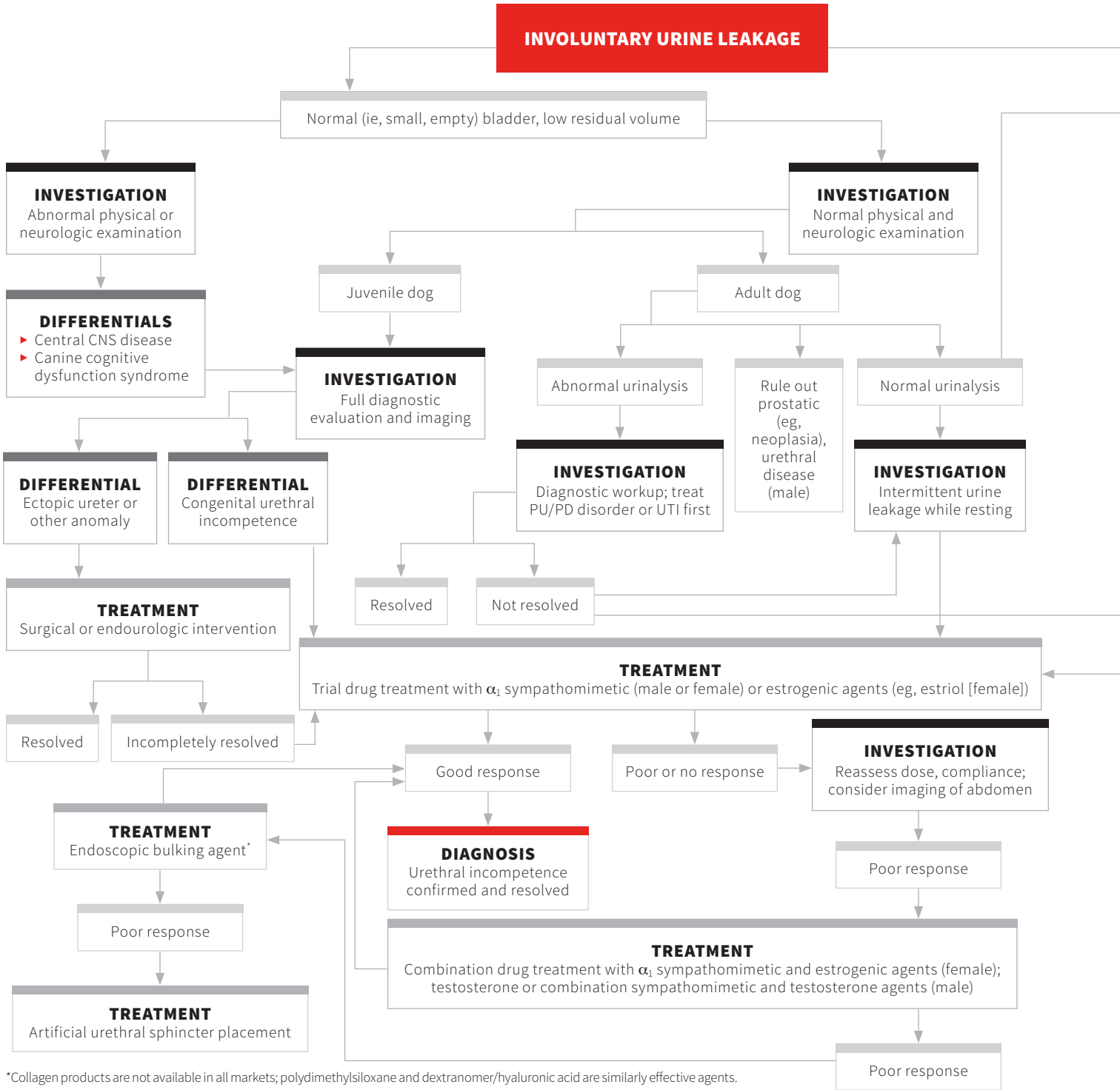
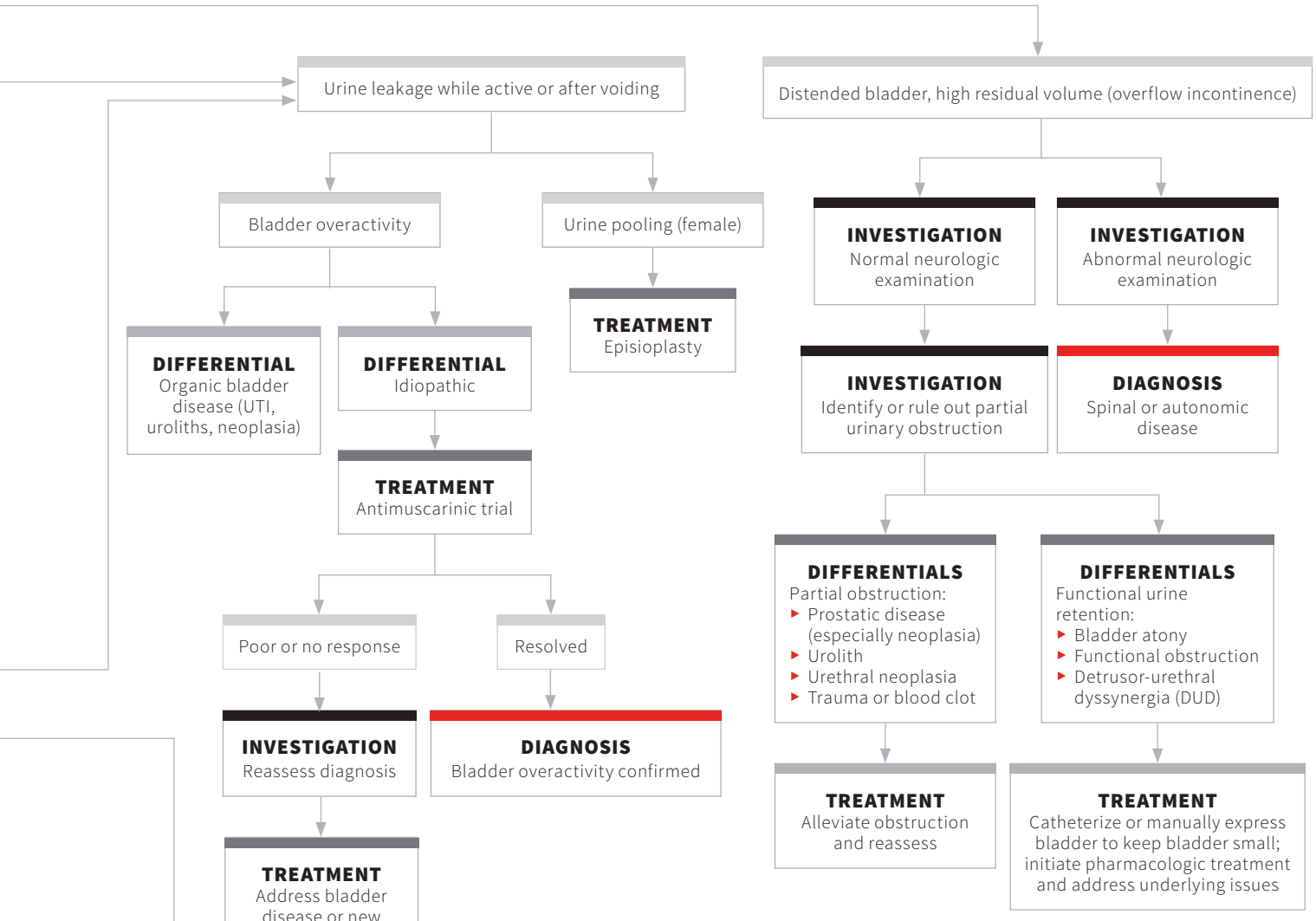
Ross L. Acute renal failure. In: Bonagura JD, Twedt DC, eds. *Kirk's Current Veterinary Therapy XIV*. St. Louis, MO: Saunders Elsevier; 2009:879-882.

CANINE URINARY INCONTINENCE

India F. Lane, DVM, MS, EdD, DACVIM
University of Tennessee



*Collagen products are not available in all markets; polydimethylsiloxane and dextranomer/hyaluronic acid are similarly effective agents.



DUD = detrusor-urethral dyssynergia

PU/PD = polyuria/polydipsia

DIAGNOSTIC PLAN

- Signalment
- History and/or observation
- Pattern of incontinence
- Physical and neurologic assessment
- Bladder palpation
- Urinalysis and culture
- Confirmation of diagnosis
- Management of refractory cases
  - Advanced diagnostics for juvenile or refractory cases (eg, imaging, cystoscopy)
- Trial treatment
- Assessment of response
- Management of concurrent disorders

FELINE CALCIUM OXALATE UROLITHIASIS

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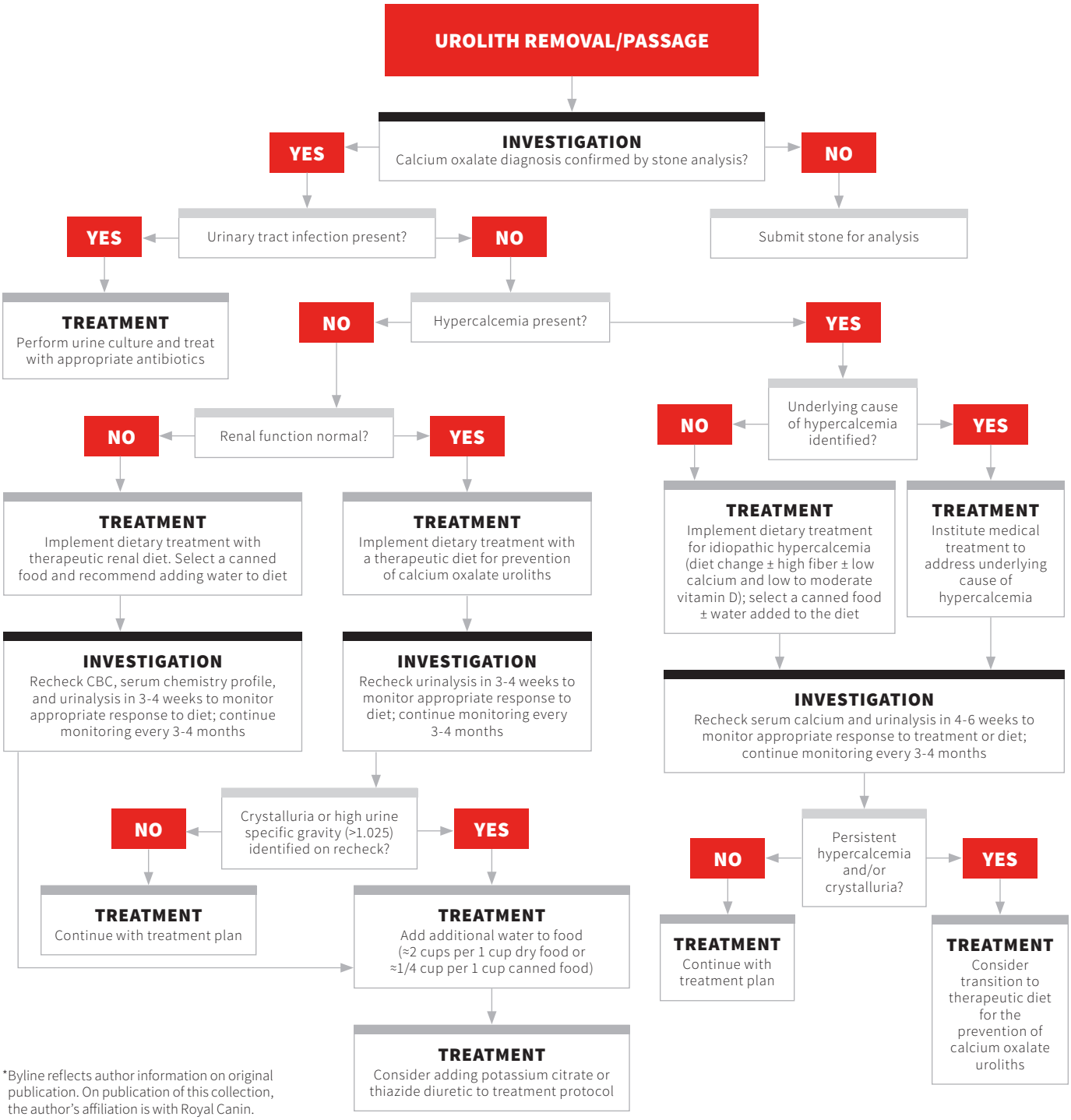

*Byline reflects author information on original publication. On publication of this collection, the author's affiliation is with Royal Canin.

FLUOROQUINOLONE-RESISTANT *ESCHERICHIA COLI*

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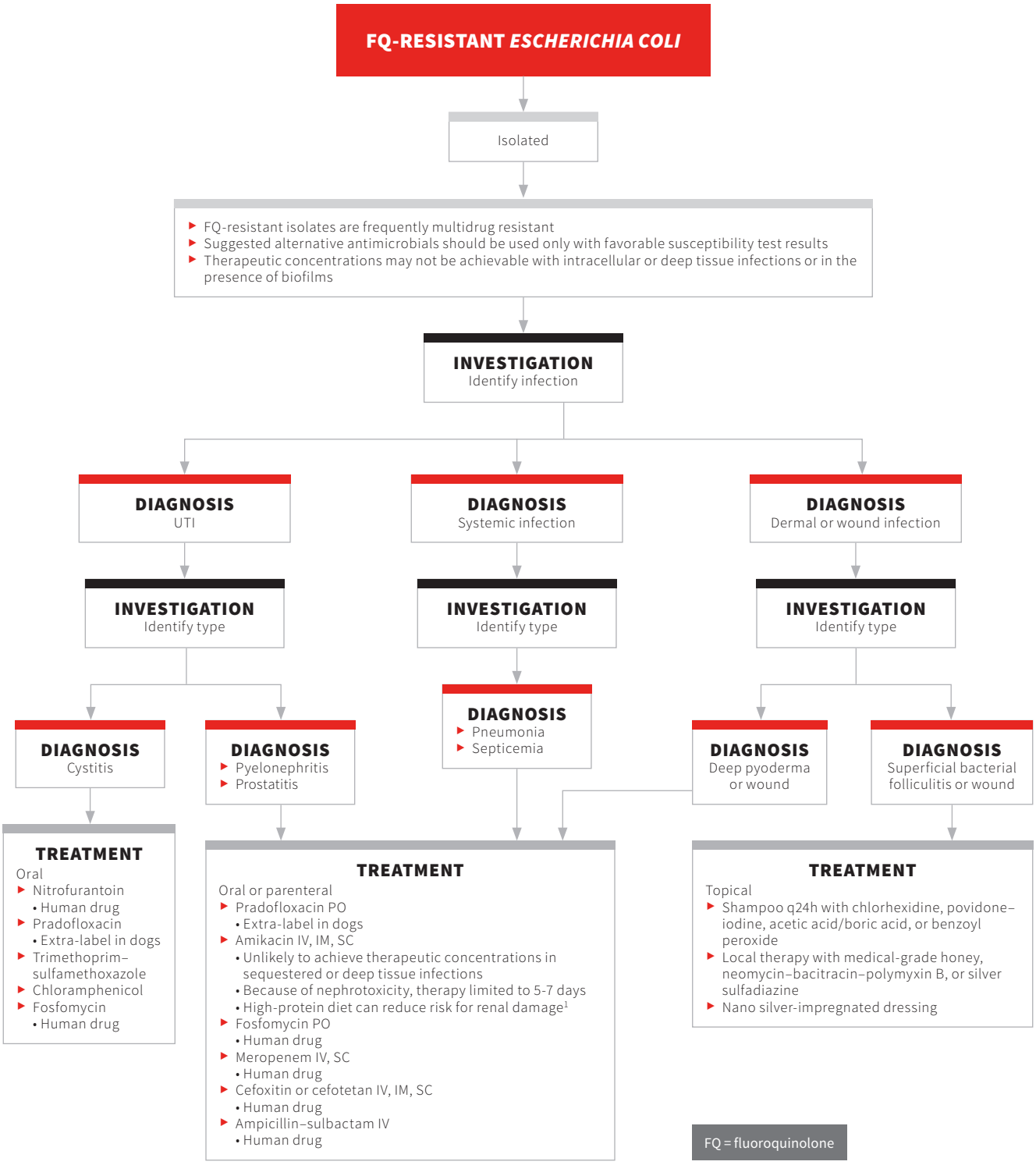
University of Saskatchewan



AUTHOR INSIGHT

- Some FQ-resistant isolates are susceptible to third-generation FQ pradofloxacin.
- Most are susceptible to nitrofurantoin, amikacin, fosfomycin, and meropenem.

Reference

1. Grauer GF, Greco DS, Behrend EN, et al. Effects of dietary protein conditioning on gentamicin-induced nephrotoxicosis in healthy male dogs. *Am J Vet Res*. 1994;55(1):90-97.

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Maaland M, Guardabassi L. In vitro antimicrobial activity of nitrofurantoin against *Escherichia coli* and *Staphylococcus pseudintermedius* isolated from dogs and cats. *Vet Microbiol*. 2011;151(3-4):396-399.

Shaheen BW, Boothe DM, Oyarzabal OA, Smaha T. Antimicrobial resistance profiles and clonal relatedness of canine and feline *Escherichia coli* pathogens expressing multidrug resistance in the United States. *J Vet Intern Med*. 2010;24(2):323-330.

HEMATURIA IN DOGS

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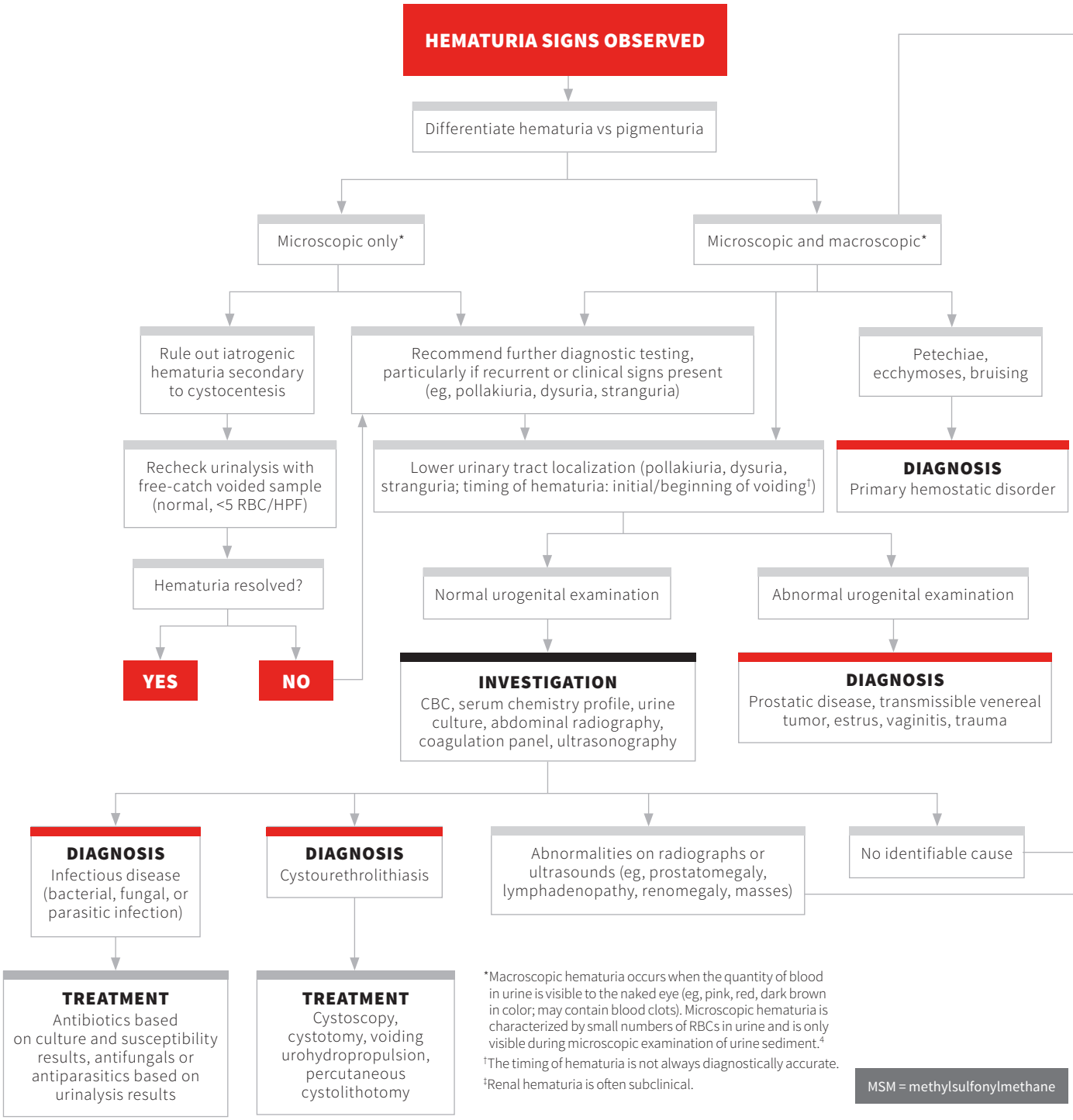
Friendship Hospital for Animals
Washington, DC

Mary Anna Labato, DVM, DACVIM (SAIM)
Cummings School of Veterinary Medicine at Tufts University

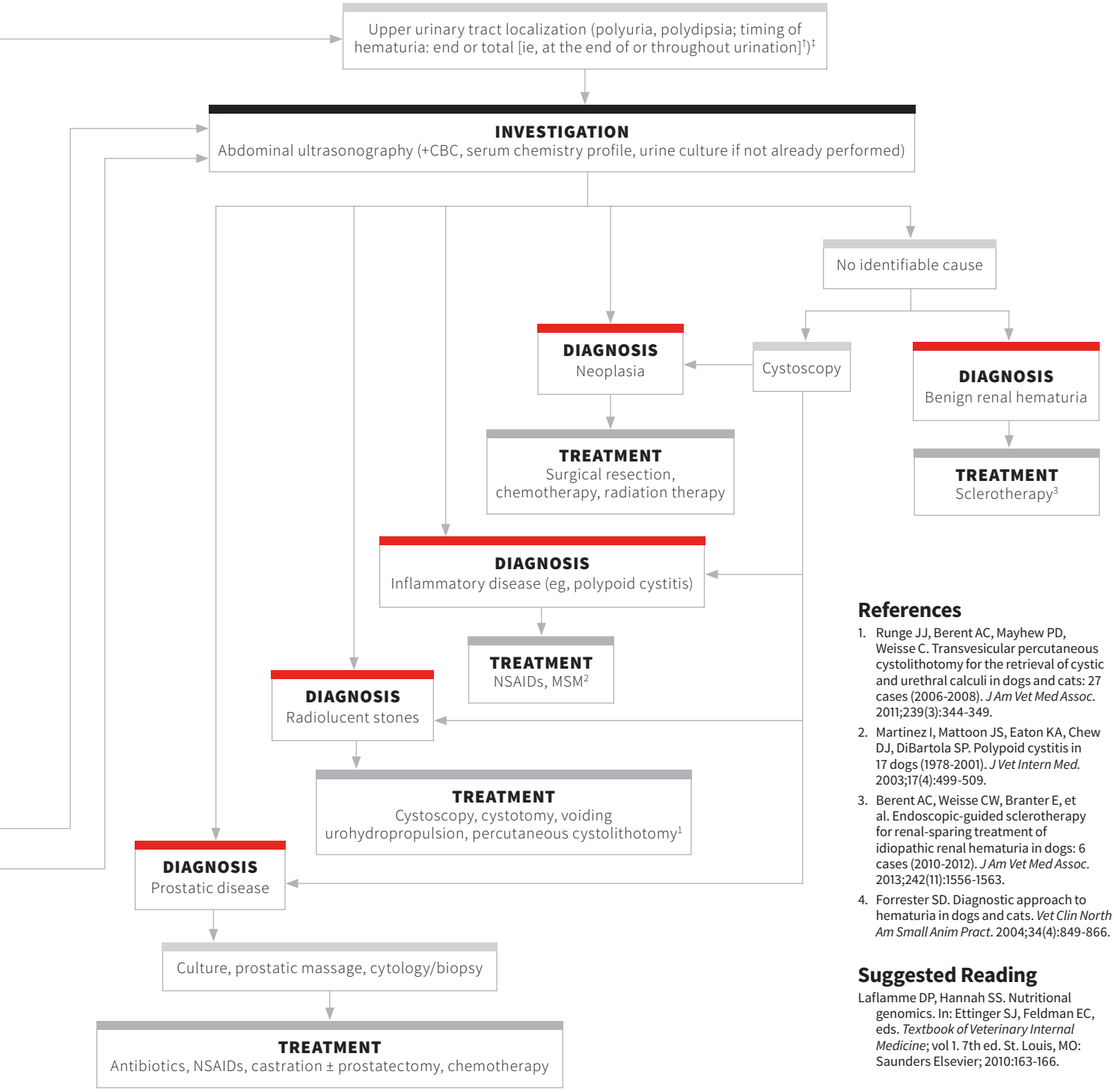


*Macroscopic hematuria occurs when the quantity of blood in urine is visible to the naked eye (eg, pink, red, dark brown in color; may contain blood clots). Microscopic hematuria is characterized by small numbers of RBCs in urine and is only visible during microscopic examination of urine sediment.[4]

†The timing of hematuria is not always diagnostically accurate.

‡Renal hematuria is often subclinical.

MSM = methylsulfonylmethane



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1. Runge JJ, Berent AC, Mayhew PD, Weisse C. Transvesicular percutaneous cystolithotomy for the retrieval of cystic and urethral calculi in dogs and cats: 27 cases (2006-2008). *J Am Vet Med Assoc*. 2011;239(3):344-349.
2. Martinez I, Mattoon JS, Eaton KA, Chew DJ, DiBartola SP. Polypoid cystitis in 17 dogs (1978-2001). *J Vet Intern Med*. 2003;17(4):499-509.
3. Berent AC, Weisse CW, Branter E, et al. Endoscopic-guided sclerotherapy for renal-sparing treatment of idiopathic renal hematuria in dogs: 6 cases (2010-2012). *J Am Vet Med Assoc*. 2013;242(11):1556-1563.
4. Forrester SD. Diagnostic approach to hematuria in dogs and cats. *Vet Clin North Am Small Anim Pract*. 2004;34(4):849-866.

Suggested Reading

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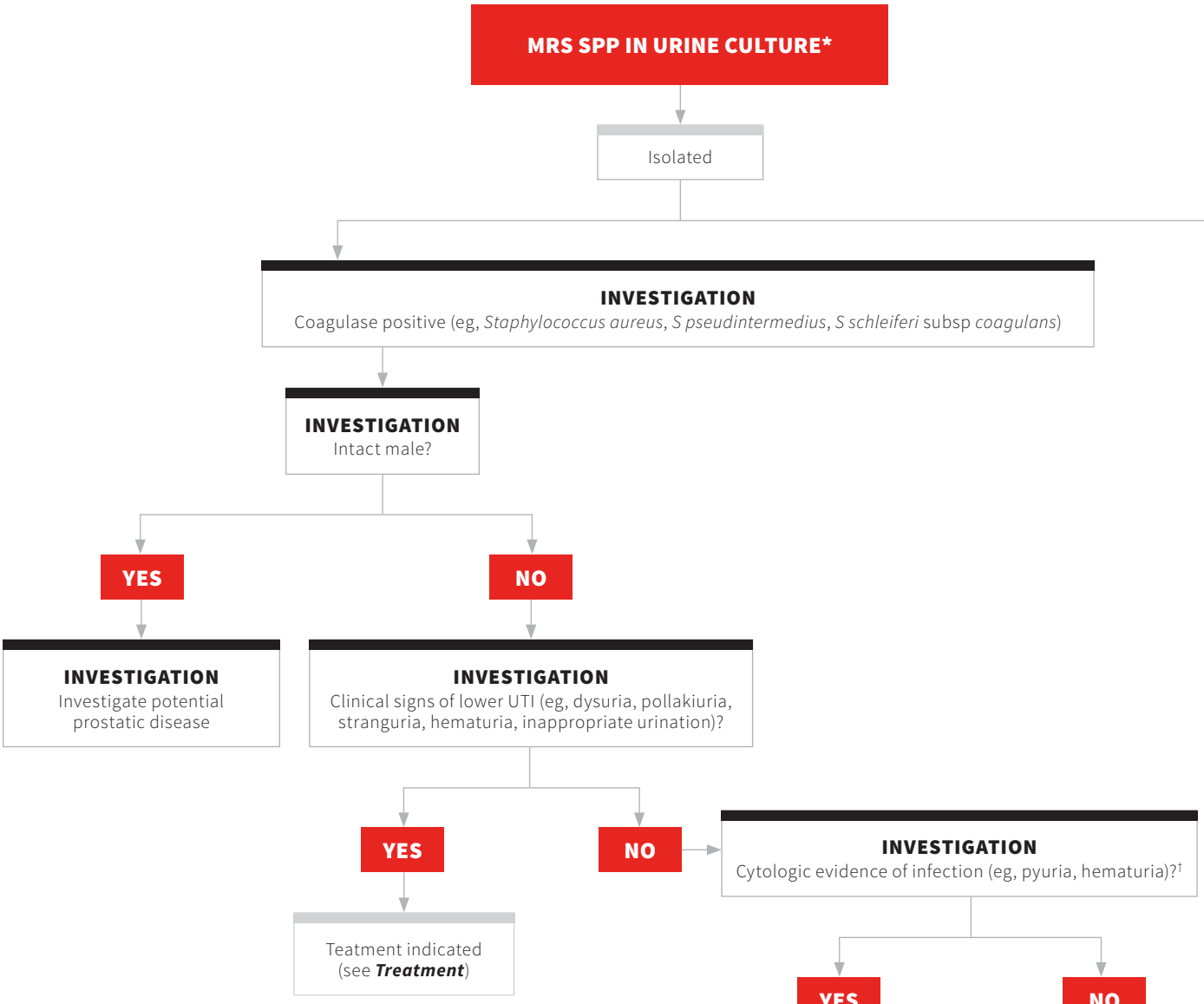
ISOLATION OF METHICILLIN-RESISTANT *STAPHYLOCOCCUS* SPP IN CANINE URINE CULTURE

J. Scott Weese, DVM, DVSc, DACVIM
University of Guelph



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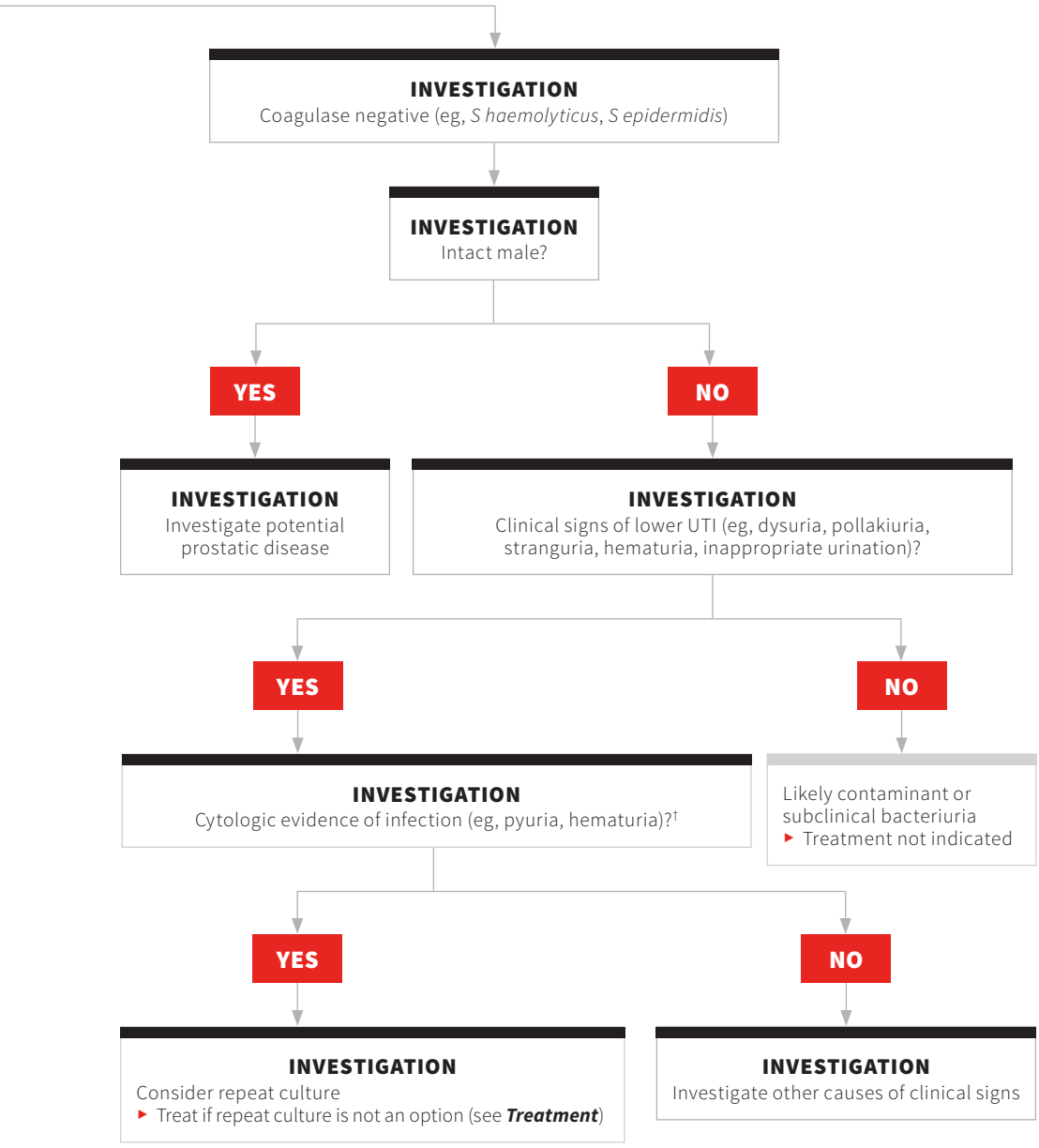
Nicolle LE, Bradley S, Colgan R, Rice JC, Schaeffer A, Hooton TM, Infectious Diseases Society of America, American Society of Nephrology, American Geriatric Society. Infectious Diseases Society of America guidelines for the diagnosis and treatment of asymptomatic bacteriuria in adults. *Clin Infect Dis*. 2005;40(5):643-654.

Weese JS, Blondeau J, Boothe D, et al. Antimicrobial use guidelines for treatment of urinary tract disease in dogs and cats: antimicrobial guidelines working group of the International Society for Companion Animal Infectious Diseases. *Vet Med Int*. 2011. doi:10.4061/2011/263768.

Weese JS, van Duijkeren E. Methicillin-resistant *Staphylococcus aureus* and *Staphylococcus pseudintermedius* in veterinary medicine. *Vet Microbiol*. 2010;140(3-4):418-429.



TREATMENT

MRS spp are inherently resistant to all β-lactams (eg, penicillins, cephalosporins, carbapenems); response of MRS spp to fluoroquinolones is unpredictable.

- Possible treatment options include nitrofurantoin, fosfomycin (dogs only), doxycycline, minocycline, trimethoprim-sulfonamide, aminoglycosides.
- Consider and address potential underlying causes (eg, uroliths, endocrinopathy, anatomical defects), especially if recurrent disease.

Acute, Sporadic UTI

- Treat for 3-7 days.

Recurrent or Relapsing UTI

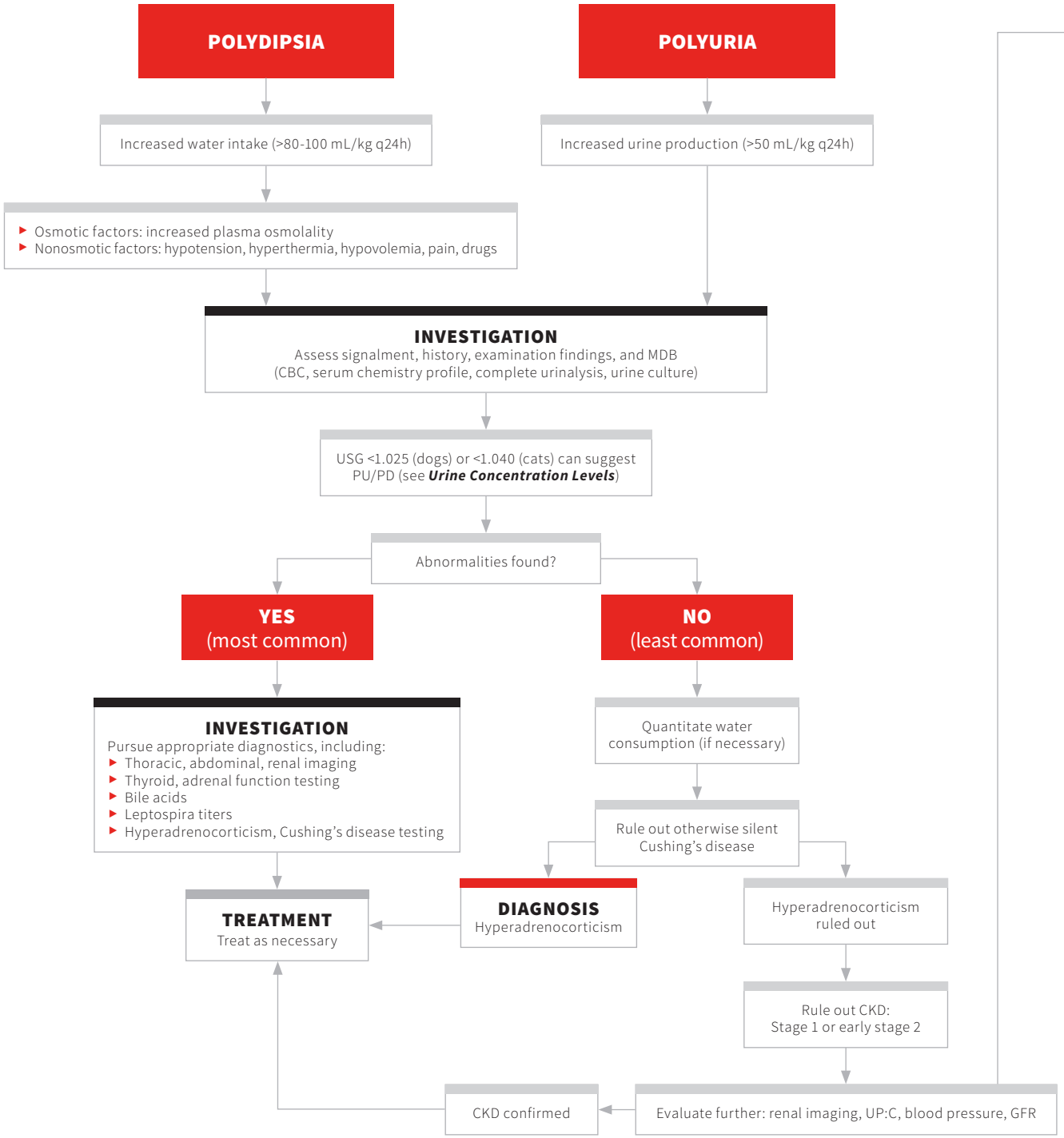
- Treat for 3-28 days.
  - Shorter durations (eg, 3-5 days) may be appropriate for acute, recurring infections.
  - Longer durations (eg, 14-28 days) may be indicated for cases in which elimination of the bacteria may be difficult (eg, chronic infection with significant bladder wall invasion, bladder mass, debris in bladder).

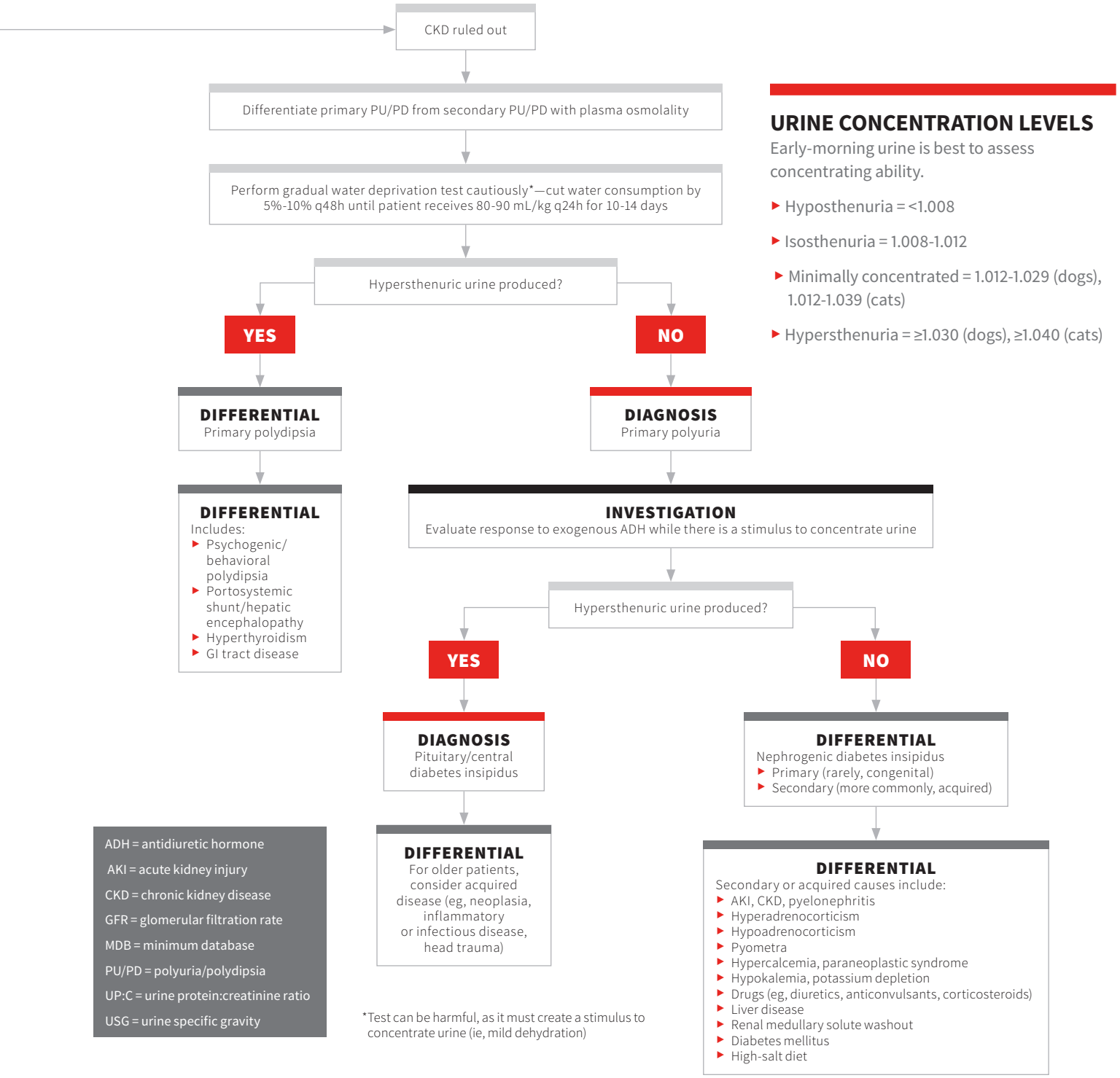
*Cystocentesis should be used when possible. If culture is from a free-catch sample, repeat with cystocentesis to rule out contamination.
†This can also occur with noninfectious etiologies.

MRS = methicillin-resistant *Staphylococcus*

POLYURIA & POLYDIPSIA IN DOGS & CATS

Gregory F. Grauer, DVM, MS, DACVIM (SAIM)
Kansas State University





URINE CONCENTRATION LEVELS

Early-morning urine is best to assess concentrating ability.

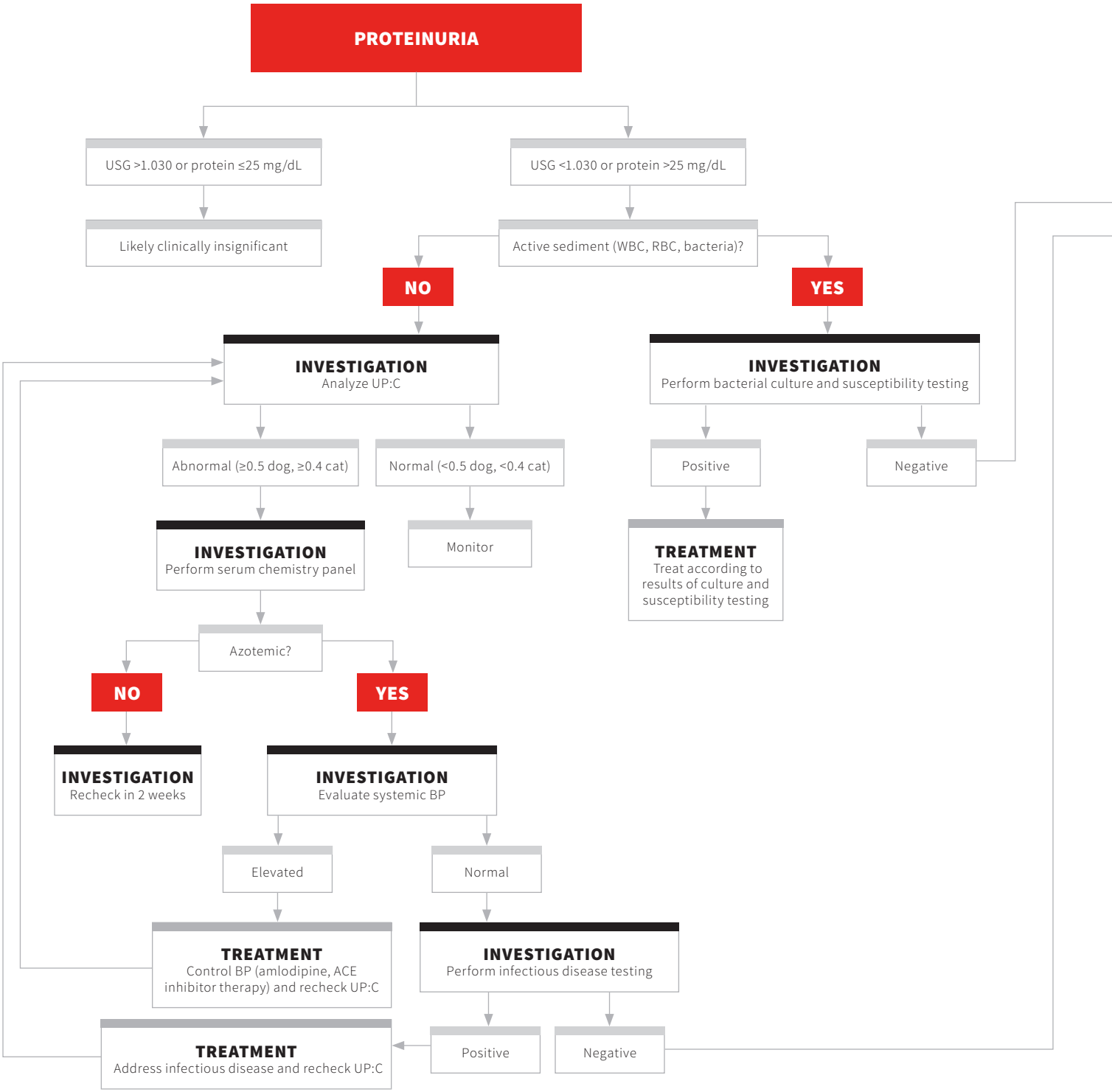
- Hyposthenuria = <1.008
- Isosthenuria = 1.008-1.012
- Minimally concentrated = 1.012-1.029 (dogs), 1.012-1.039 (cats)
- Hypersthenuria = ≥1.030 (dogs), ≥1.040 (cats)

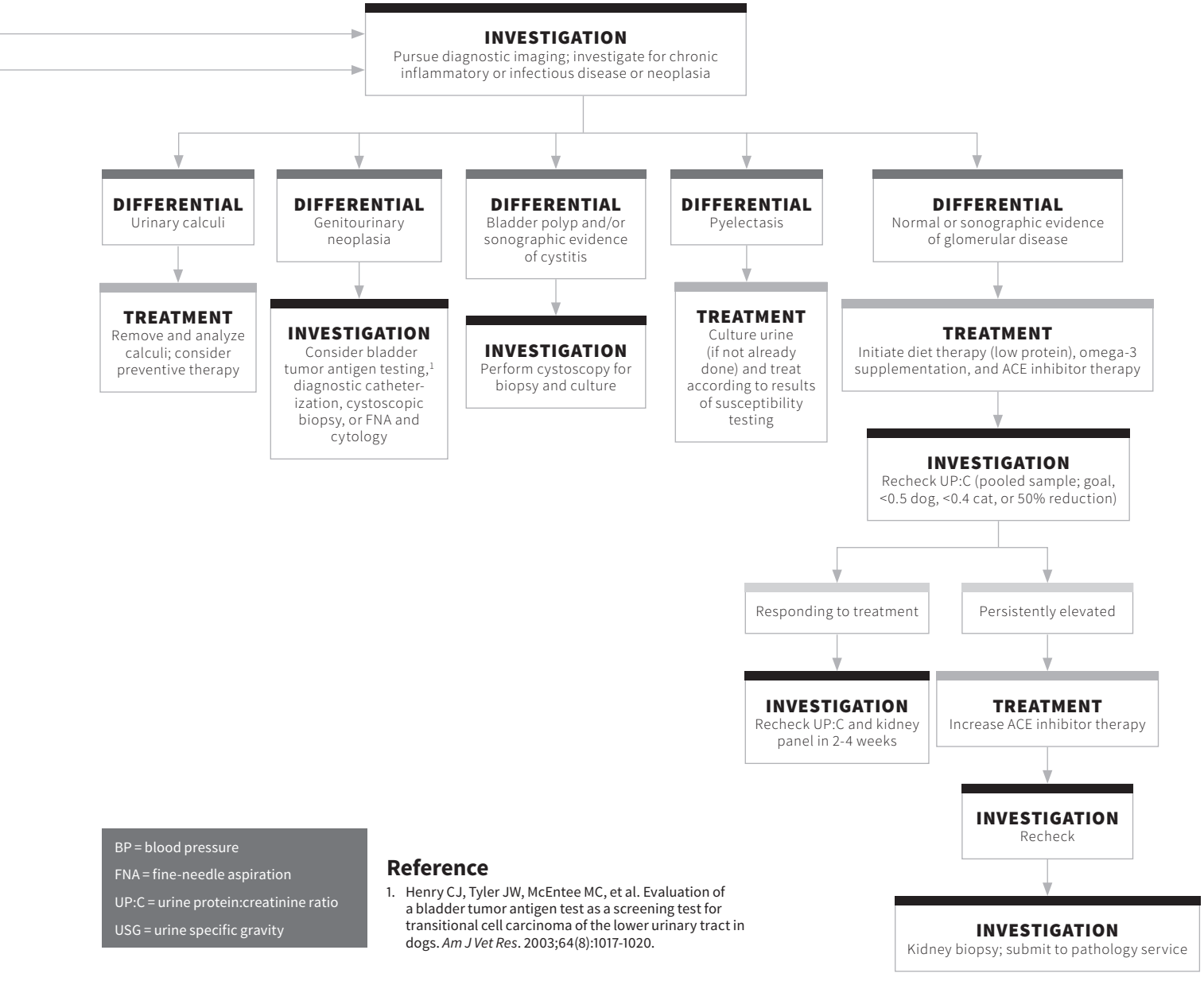
ADH = antidiuretic hormone
AKI = acute kidney injury
CKD = chronic kidney disease
GFR = glomerular filtration rate
MDB = minimum database
PU/PD = polyuria/polydipsia
UP:C = urine protein:creatinine ratio
USG = urine specific gravity

*Test can be harmful, as it must create a stimulus to concentrate urine (ie, mild dehydration)

PROTEINURIA

Jonathan Dear, DVM, DACVIM (SAIM)
University of California, Davis





BP = blood pressure
FNA = fine-needle aspiration
UP:C = urine protein:creatinine ratio
USG = urine specific gravity

Reference

1. Henry CJ, Tyler JW, McEntee MC, et al. Evaluation of a bladder tumor antigen test as a screening test for transitional cell carcinoma of the lower urinary tract in dogs. *Am J Vet Res*. 2003;64(8):1017-1020.

RECURRENT URINARY TRACT INFECTION

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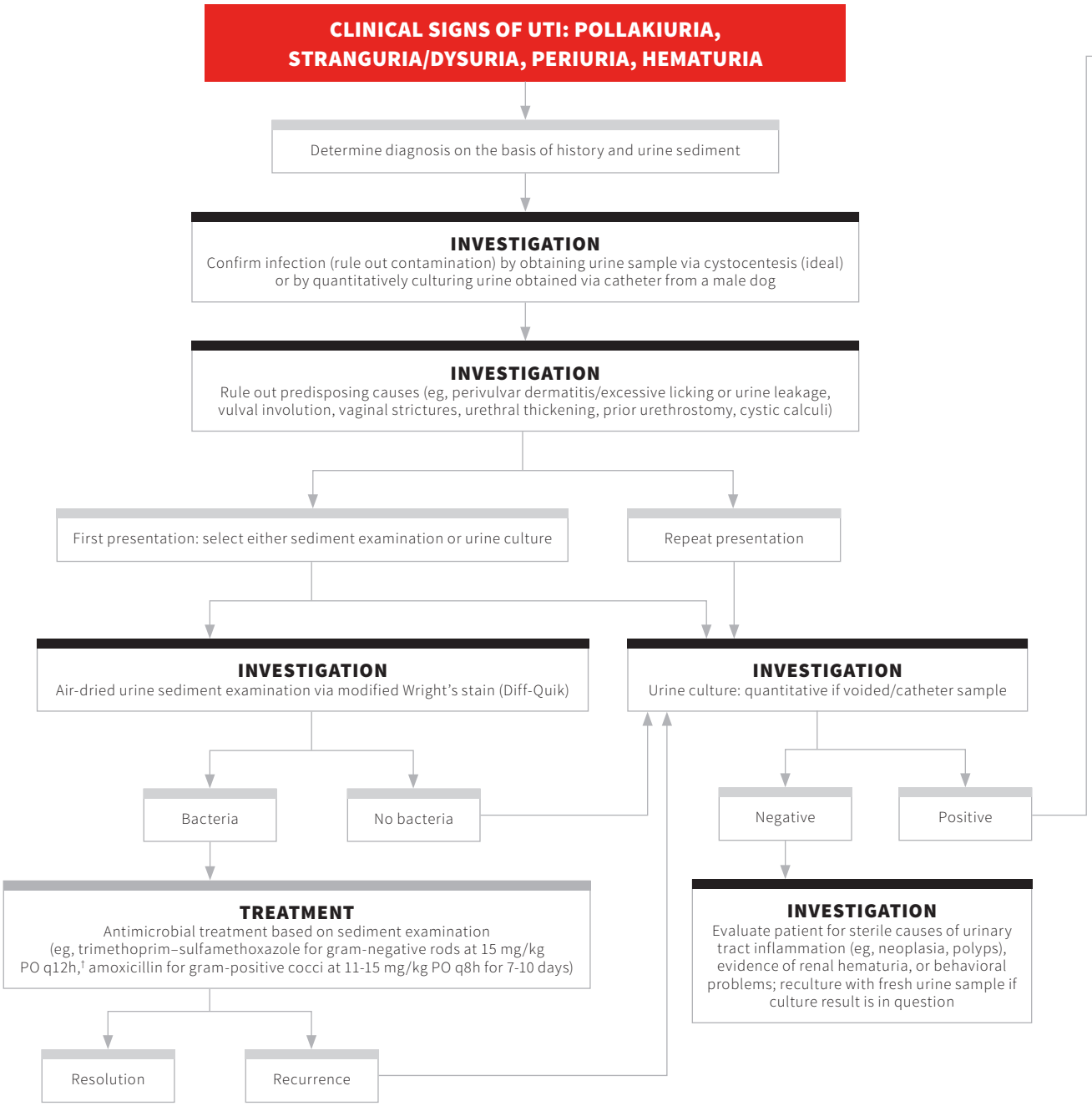
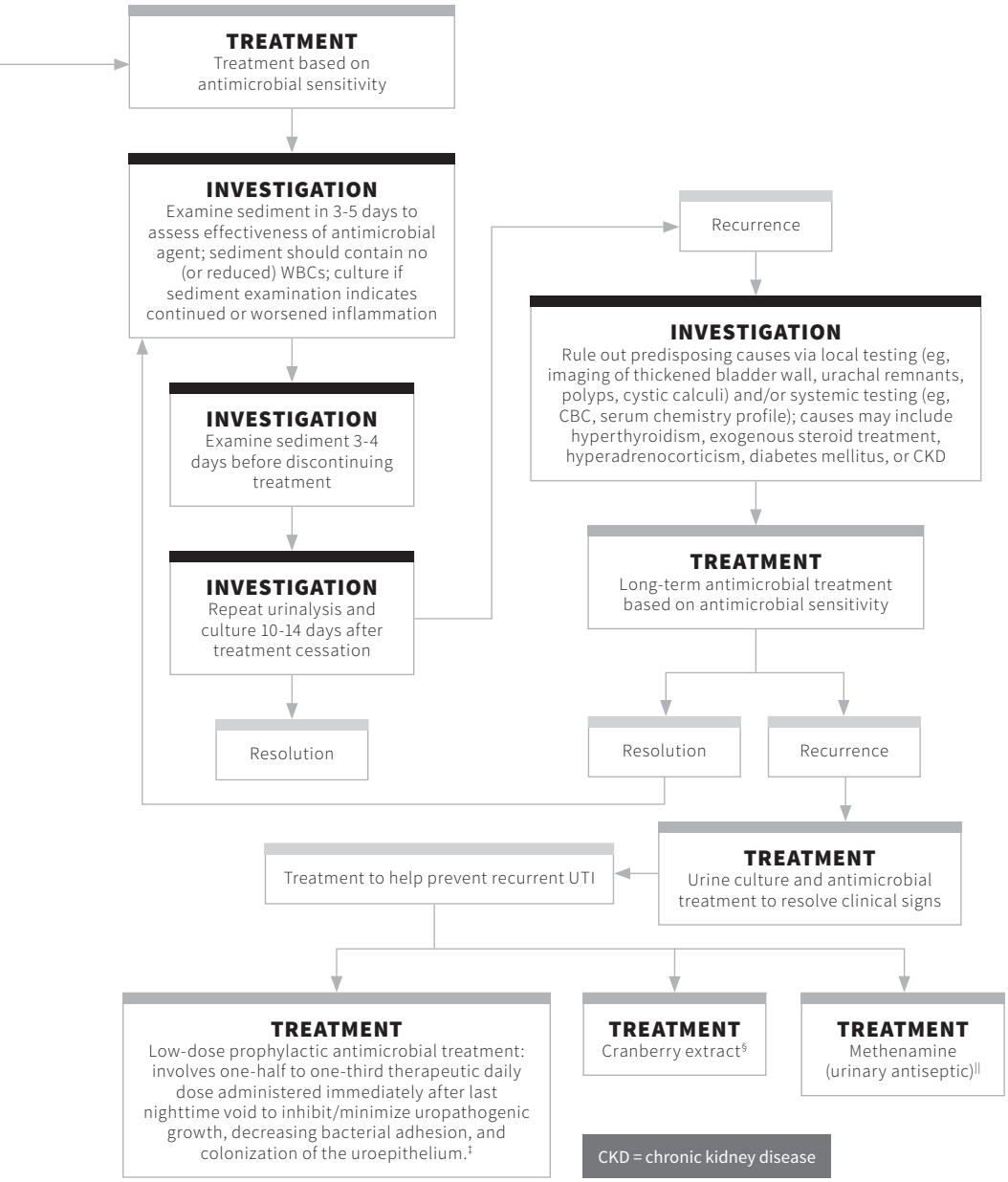


†Sensitivity of gram-negative rods is known to be unpredictable, as a large proportion of them will be *Escherichia coli*. If gram-negative rods are seen on urine sediment, culture and susceptibility testing is advised.

‡No studies have evaluated efficacy and adverse effects of these protocols in dogs. To avoid creating resistant infections, this administration should be reserved for dogs with reinfections when predisposing causes cannot be corrected. Treatment is initiated after standard dose of antimicrobial therapy has been successful. Other considerations for antimicrobial selection should include adverse events and culture and susceptibility test results. Protocols include fluoroquinolones, cephalosporins, or ß-lactam antimicrobials. This is typically recommended for ≥6 months and urinalysis/culture should be performed every 4-8 weeks. If UTI occurs during treatment, treat as a complicated UTI. Prophylactic, low-dose therapy can be restarted after reinfection resolves.[1]



§Canine studies evaluating efficacy of cranberry extract are limited; however, some in vitro data show promising results. One study demonstrated that *E coli* in urine from dogs receiving cranberry extract PO had decreased the ability to adhere to Madin-Darby canine kidney cells.[2] Based on this, PO administration of cranberry extract may help reduce *E coli* reinfections in patients with compromised host defense mechanisms. It may be beneficial to use products evaluated for canine use.

‖The most commonly used urinary antiseptics include methenamine, which may be considered in patients with recurrent infection or potentially untreatable/unresolved immunity breaches. For this to be effective, urine must be acidic.

Continues ▶

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REASONS FOR POOR THERAPEUTIC RESPONSE

- Use of ineffective drugs or insufficient therapy duration: **Relapse**
- Failure of owner to comply with prescription: **Relapse**
- GI disease, concurrent intake of food and drug (decreased absorption), or impaired renal concentrating ability (decreased antibiotic concentrations in urine): **Relapse**
- Impaired drug action, either because bacteria are not multiplying or because they are in an inaccessible area (eg, prostate, neoplasia, uroliths): **Relapse**
  For penetration into prostatic tissue, antimicrobial agents should be lipid soluble, not be highly protein-bound, and ionize at the tissue's pH. Fluoroquinolones, trimethoprim–sulfamethoxazole, and chloramphenicol can achieve prostate gland penetration.
- Failure to recognize and eliminate predisposing causes: **Reinfection**
- Presence of mixed bacterial infection in which antimicrobial therapy eradicates only one pathogen: **Relapse**
- Iatrogenic reinfection via catheterization: **Reinfection**
- Development of drug resistance in bacteria: **Relapse**

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ANESTHETIC HYPOTENSION

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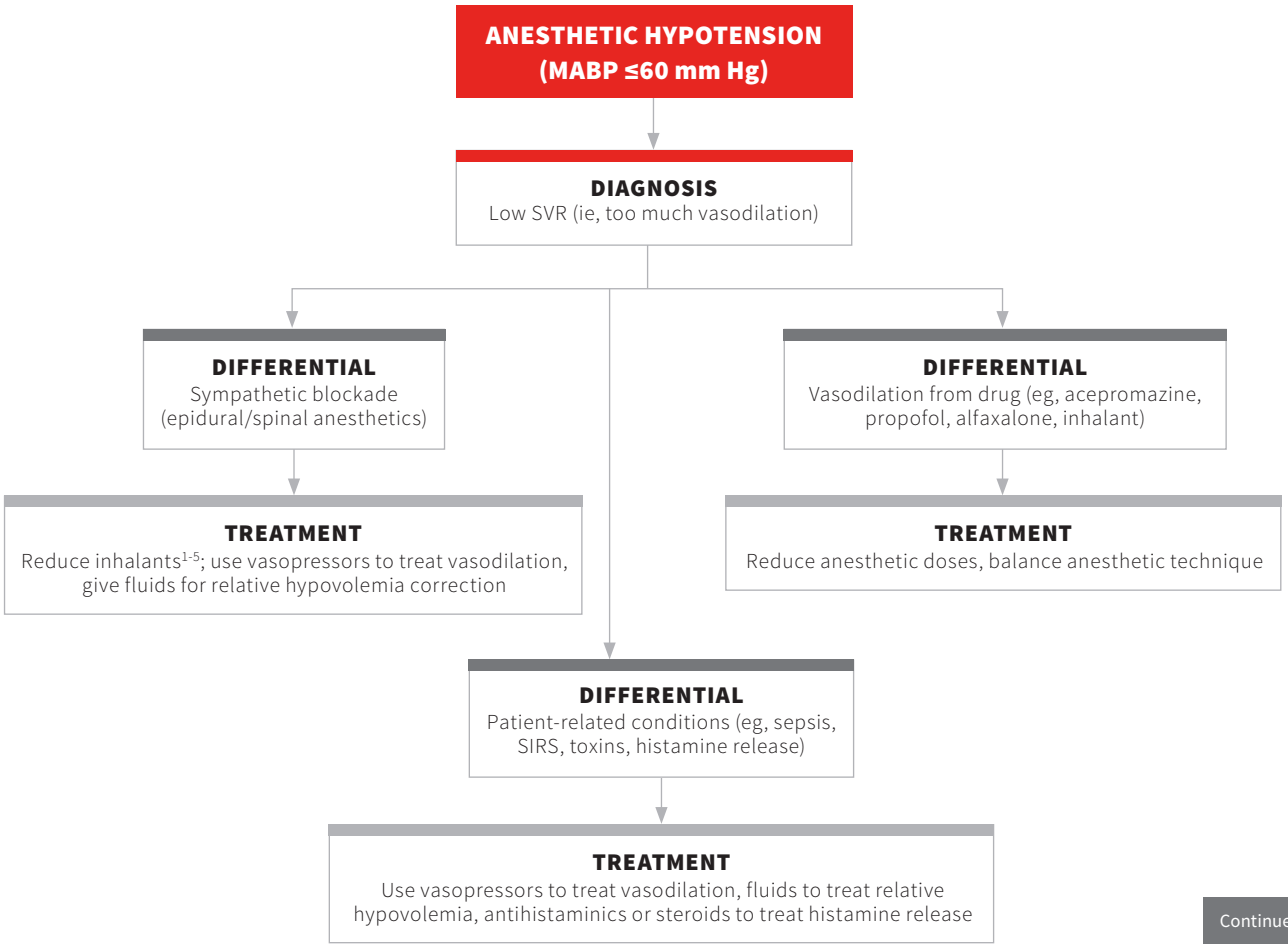

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PERTINENT CALCULATIONS

- Blood pressure = CO × SVR
- CO = SV × HR

CO = cardiac output
HR = heart rate
MABP = mean arterial blood pressure
SIRS = systemic inflammatory response syndrome
SV = stroke volume
SVR = systemic vascular resistance

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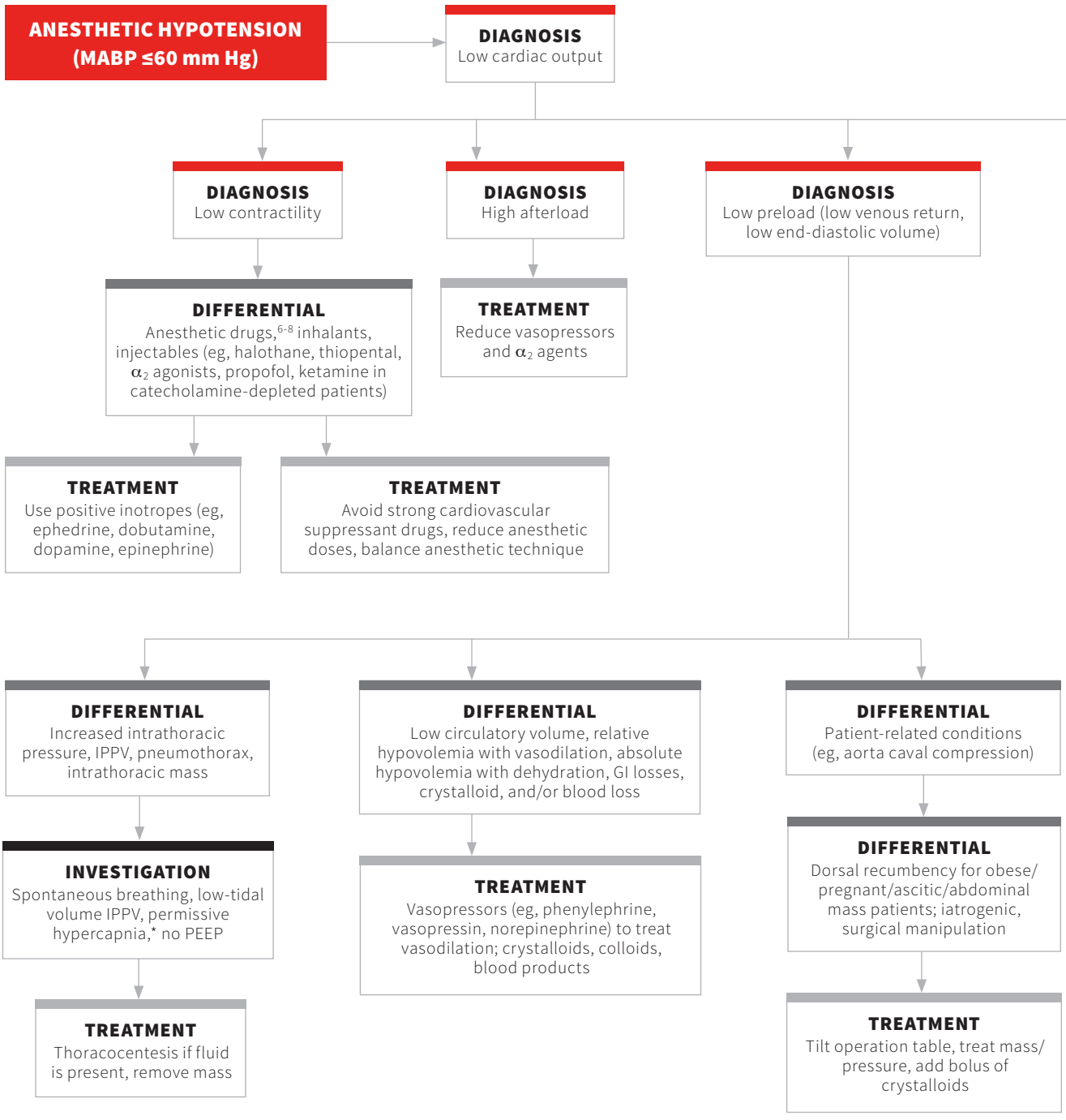


AV = atrioventricular

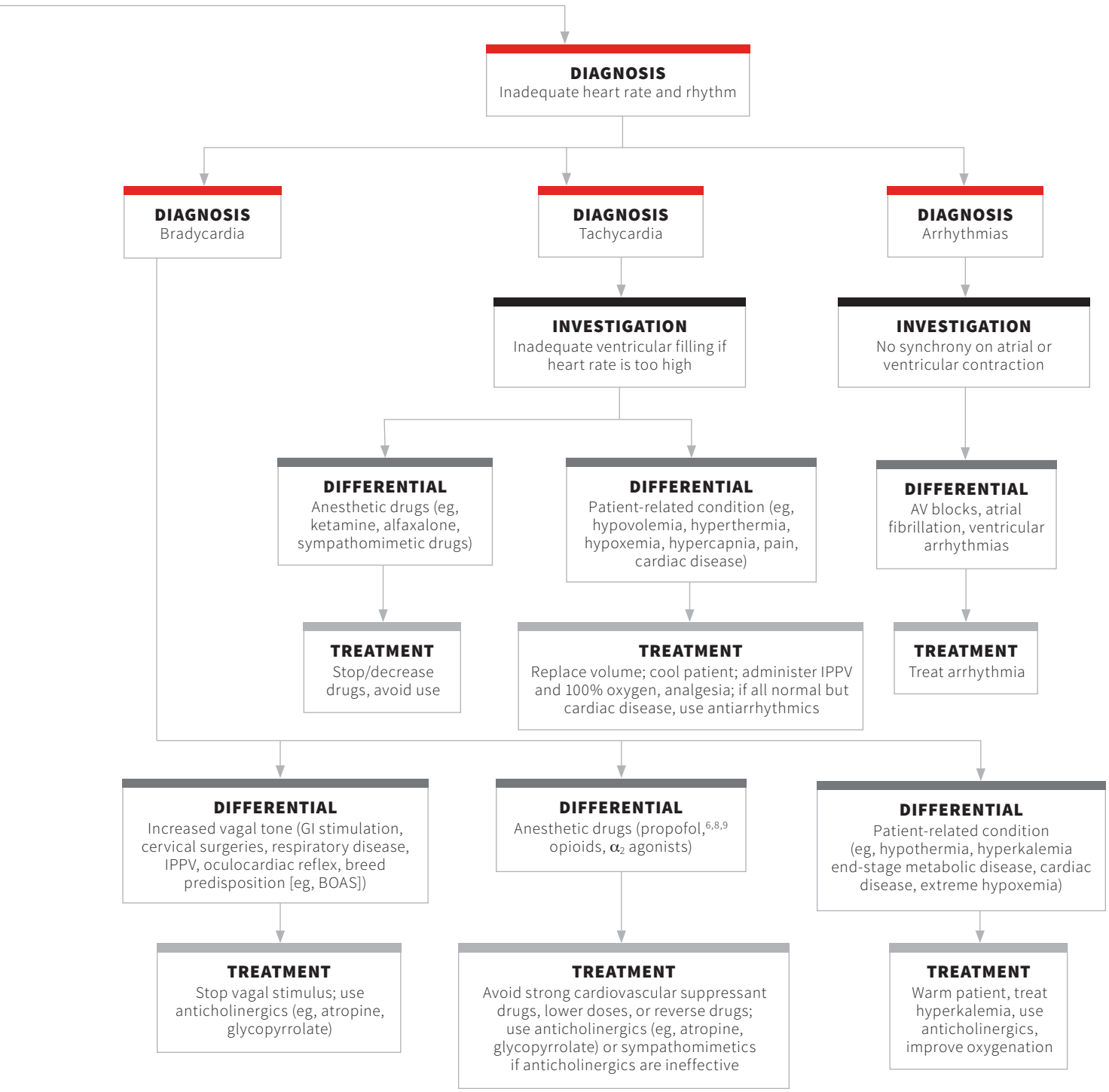
BOAS = brachycephalic obstructive airway syndrome

IPPV = intermittent positive pressure ventilation

MABP = mean arterial blood pressure

PEEP = positive end-expiratory pressure

*Permissive hypercapnia is a ventilation strategy in which oxygenation is prioritized over expired carbon dioxide. Higher than normal carbon dioxide levels are allowed because of low ventilation that preserves the lungs.


DIAGNOSIS
Inadequate heart rate and rhythm
DIAGNOSIS
Bradycardia
DIAGNOSIS
Tachycardia
DIAGNOSIS
Arrhythmias
INVESTIGATION
Inadequate ventricular filling if heart rate is too high
INVESTIGATION
No synchrony on atrial or ventricular contraction
DIFFERENTIAL
Anesthetic drugs (eg, ketamine, alfaxalone, sympathomimetic drugs)
DIFFERENTIAL
Patient-related condition (eg, hypovolemia, hyperthermia, hypoxemia, hypercapnia, pain, cardiac disease)
DIFFERENTIAL
AV blocks, atrial fibrillation, ventricular arrhythmias
TREATMENT
Stop/decrease drugs, avoid use
TREATMENT
Replace volume; cool patient; administer IPPV and 100% oxygen, analgesia; if all normal but cardiac disease, use antiarrhythmics
TREATMENT
Treat arrhythmia
DIFFERENTIAL
Increased vagal tone (GI stimulation, cervical surgeries, respiratory disease, IPPV, oculocardiac reflex, breed predisposition [eg, BOAS])
DIFFERENTIAL
Anesthetic drugs (propofol,[6,8,9] opioids, α_2 agonists)
DIFFERENTIAL
Patient-related condition (eg, hypothermia, hyperkalemia end-stage metabolic disease, cardiac disease, extreme hypoxemia)
TREATMENT
Stop vagal stimulus; use anticholinergics (eg, atropine, glycopyrrolate)
TREATMENT
Avoid strong cardiovascular suppressant drugs, lower doses, or reverse drugs; use anticholinergics (eg, atropine, glycopyrrolate) or sympathomimetics if anticholinergics are ineffective
TREATMENT
Warm patient, treat hyperkalemia, use anticholinergics, improve oxygenation

SEPARATION ANXIETY IN DOGS

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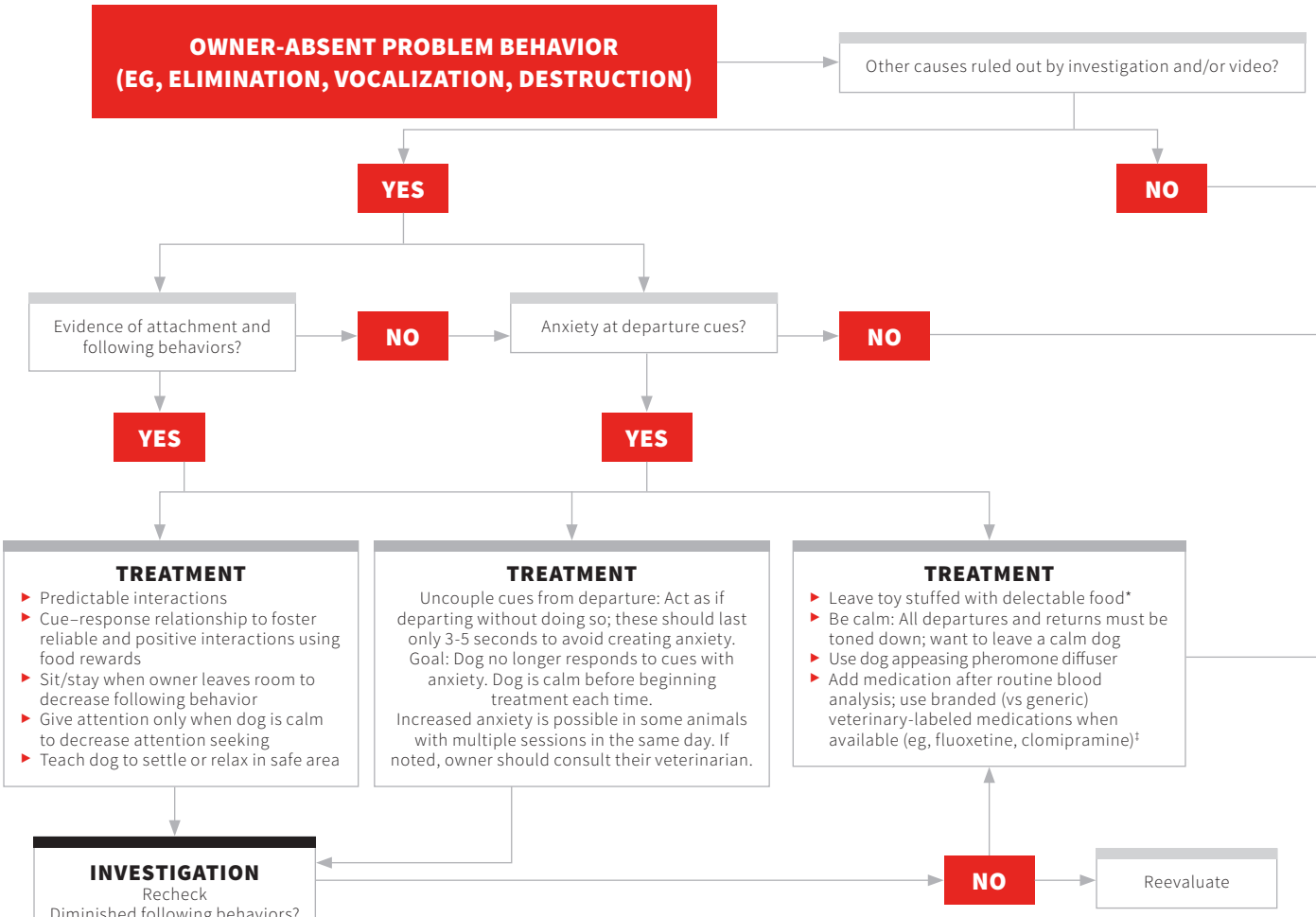
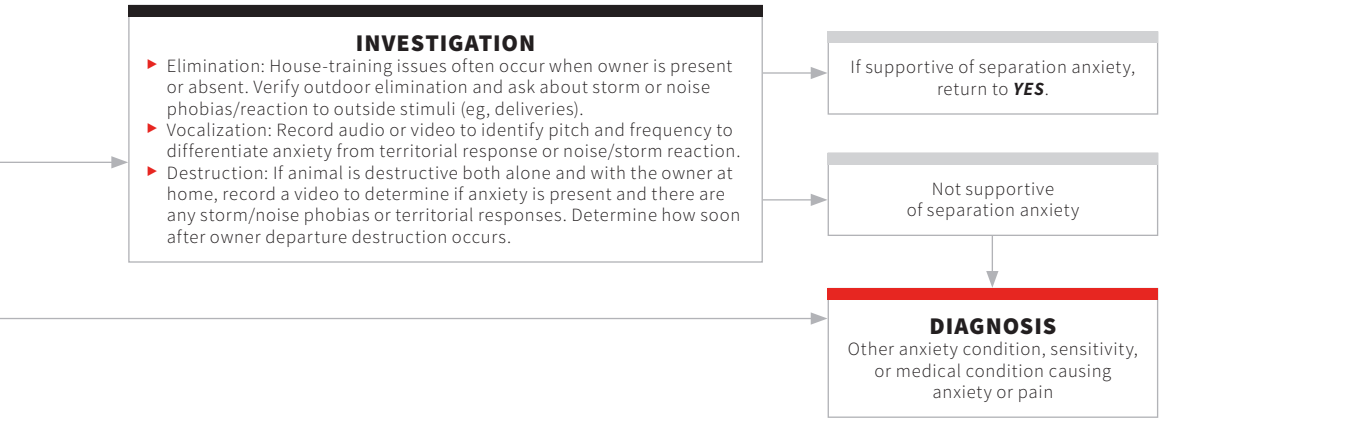


BEHAVIOR MODIFICATION TREATMENT PLAN

Begin planned training departures

- Establish safety signal† (eg, music, TV, air freshener, novel location)
- Determine minimum departure time: Generally very short (1-3 minutes), perhaps only walk to the door
- Departure training must simulate real departures: If owner always uses a car, train up to use cars for longer departure simulations.
- Place dog in safe area
- Deploy safety signal†
- Depart and remain away only for predetermined period of time (usually 1-2 minutes)
- Return, ignore dog except to matter-of-factly walk for elimination
- Dog must be calm before repeating. Assess anxiety level of the dog; it is often not practical or helpful to do more than 1-3 training departures per 24 hours
- **Never use safety signal on regular departures with unregulated length of absences**



*Must also be provided at other times to avoid toy becoming departure cue.
†A distinctive action owner takes just prior to training departures (eg, putting dog in novel location; out of confinement), employed to help the dog associate these departures with quick returns and no anxiety.
‡The decision to use drugs should be based on a combination of severity of problem and owner's tolerance of behaviors. In some cases, medication should be prescribed on the first visit. Additional medications (eg, benzodiazepines) prior to departure may be necessary for some animals in addition to daily medication to help with panic and extreme anxiety associated with departure.

ADDRESSING ANIMAL SEXUAL ABUSE

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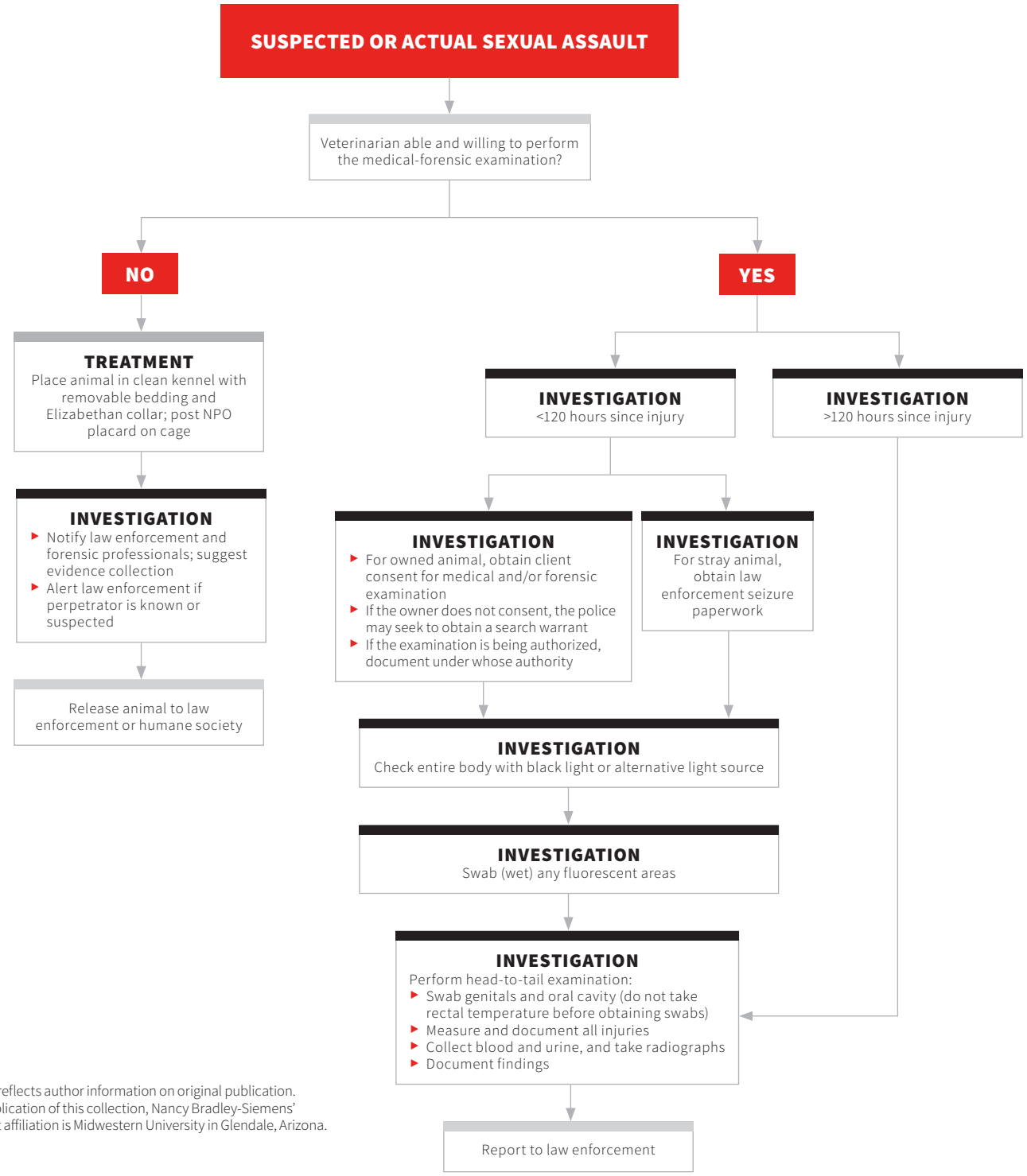

*Byline reflects author information on original publication. On publication of this collection, Nancy Bradley-Siemens' current affiliation is Midwestern University in Glendale, Arizona.

EMESIS INDUCTION

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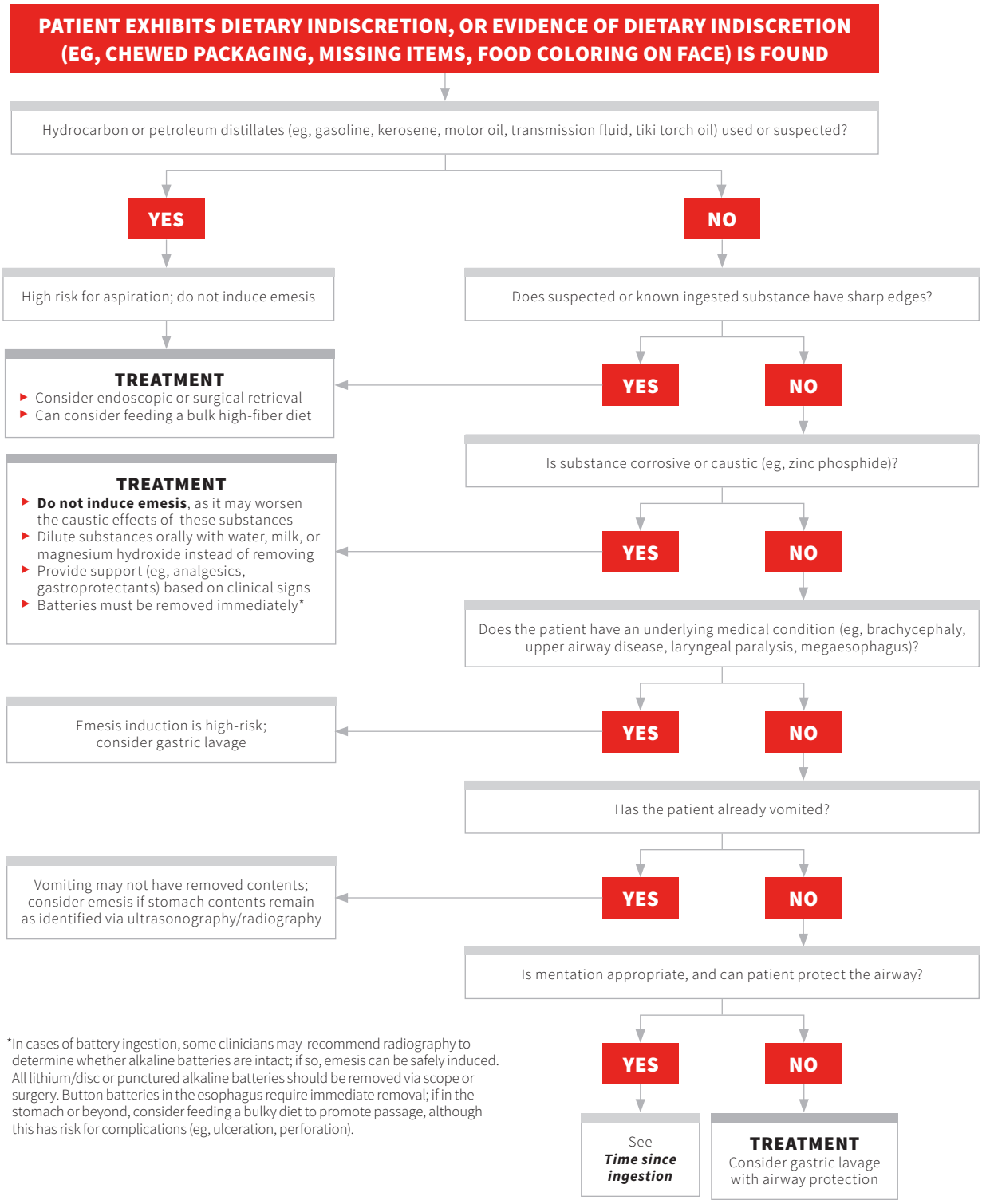
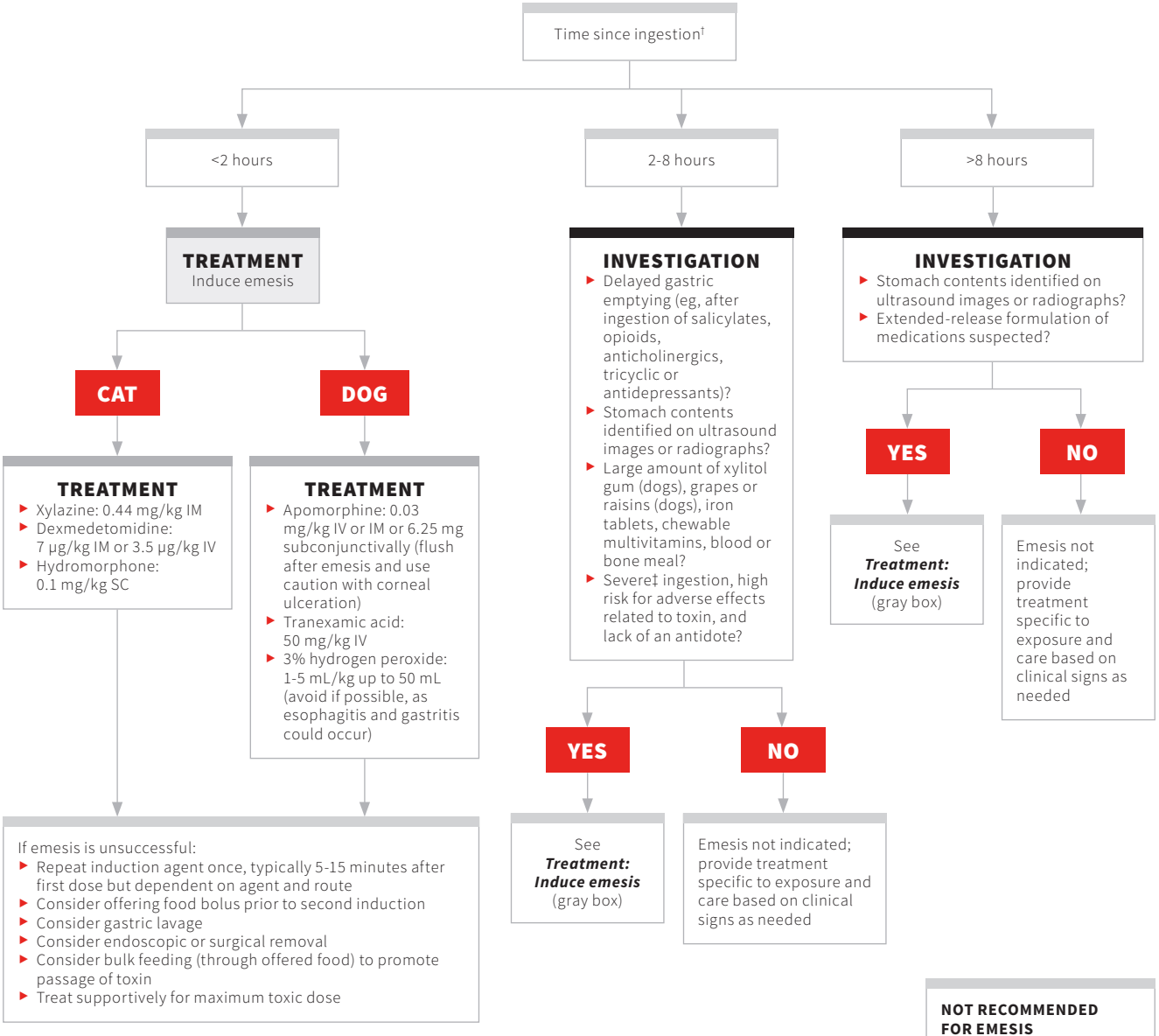
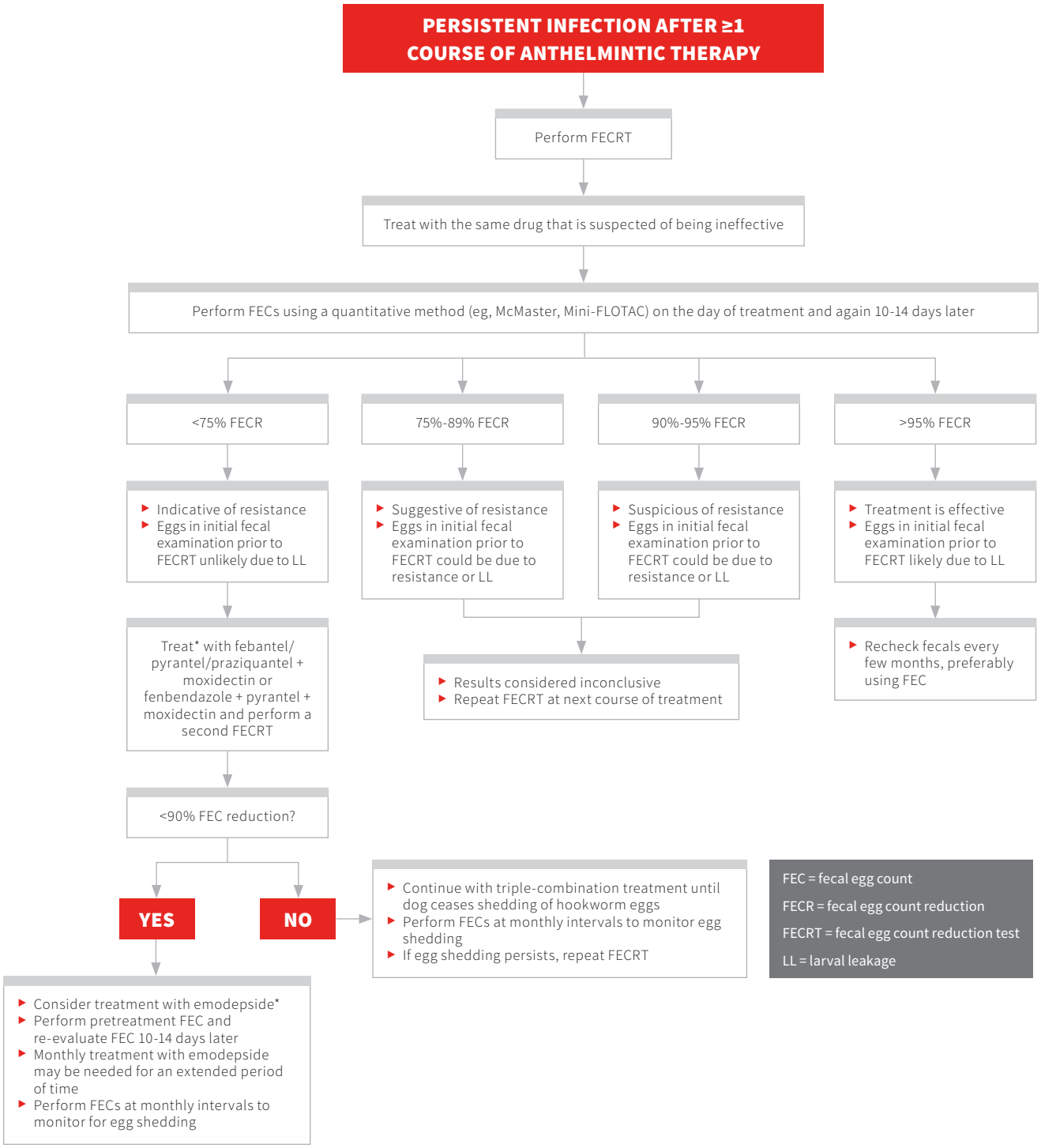


*In cases of battery ingestion, some clinicians may recommend radiography to determine whether alkaline batteries are intact; if so, emesis can be safely induced. All lithium/disc or punctured alkaline batteries should be removed via scope or surgery. Button batteries in the esophagus require immediate removal; if in the stomach or beyond, consider feeding a bulky diet to promote passage, although this has risk for complications (eg, ulceration, perforation).



†Due to the lack of a specific timeline recommendation in veterinary medicine, the authors provide these time frames based on time passed since suspected ingestion.
‡Ingestion is considered severe if the amount or type of toxicant ingested leads to higher risk for more serious toxicity.

NOT RECOMMENDED FOR EMESIS
- Liquid soap
- Ipecac syrup
- Salt
- Digital induction
- Mustard powder
- Hydrogen peroxide (cats)

PERSISTENT HOOKWORM INFECTIONS IN DOGS

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