Basic triage in dogs and cats: Part II

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Abstract

Background

Emergency cases can be presented at any time of the day or night. All small animal practitioners need to have the skills to triage and stabilize common emergency cases, even if cases are ultimately referred to another facility.

Objective and procedure

The second part of this 3-part review article series discusses animals that collapse at home as well as dogs and cats with bleeding. A stepwise approach to categorize and stabilize these cases is outlined, along with helpful tips to optimize the referral experience, if indicated.

Results

Having a robust and methodical approach to animals that collapse is important for many emergency cases, as the causes and treatment methods vary. Bleeding can lead to acute death if left untreated and knowing the steps to stop bleeding is important for patient stabilization.

Conclusion and clinical relevance

Do not refer emergent cases before completing basic stabilization. Many emergency cases do not require emergent referral and can be worked up by the primary veterinarian or sent to a referral clinic on an appointment basis after appropriate stabilization steps have occurred.

Résumé

Triage de base chez les chiens et les chats : Partie II

Contexte

Les cas d'urgence peuvent être présentés à toute heure du jour ou de la nuit. Tous les praticiens des petits animaux doivent avoir les compétences nécessaires pour trier et stabiliser les cas d'urgence courants, même si les cas sont finalement transférés vers un autre établissement.

Objectif et procédure

Le deuxième de cette série de trois articles traite des animaux qui s'effondrent à la maison ainsi que des chiens et des chats qui saignent. Une approche par étapes pour catégoriser et stabiliser ces cas est décrite, ainsi que des conseils utiles pour optimiser l'expérience de référence, si elle est indiquée.

Résultats

Avoir une approche robuste et méthodique face aux animaux qui s'effondrent est important dans de nombreux cas d'urgence, car les causes et les méthodes de traitement varient. Les saignements peuvent entraîner une mort aiguë s'ils ne sont pas traités et connaître les étapes à suivre pour arrêter le saignement est important pour la stabilisation du patient.

Conclusion et pertinence clinique

Ne référez pas les cas urgents avant d'avoir terminé la stabilisation de base. De nombreux cas d'urgence ne nécessitent pas de référence urgente et peuvent être traités par le vétérinaire initial ou envoyés à une clinique de référence sur rendez-vous après que les mesures de stabilisation appropriées ont été prises.

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Introduction

Primary care veterinarians in private general and emergency practice often carry out basic triage and stabilize animals from the "red" or "orange" groups [see Figure 1 in Part I of this review (1)] before referring them for definitive diagnostics and management. This article continues our outline of the most frequently encountered emergency situations [see Figure 2 in Part I of this review (1)] leading to referral in both dogs and cats and provides guidelines for how to stabilize animals before transport.

Section 1: Collapse

Acute collapse is defined as sudden loss of postural tone. The dog or cat may be able to sit but not stand (hind limb collapse) or be completely recumbent and unable to stand or sit (complete collapse). During a collapsing episode, the animal is fully awake and aware of its surroundings.

Collapsing should be differentiated from syncope (fainting). The latter is characterized by a transient period of loss of postural tone along with loss of consciousness. Syncopal episodes are short and the animal regains consciousness and postural tone afterwards. Most syncopal events are linked to arrhythmias [see Part III of this review (2)].

Immediate triage and care are required for a collapsed patient. These conditions are in 2 main categories: shock (cardiogenic, distributive, or hypovolemic) and non-shock.

Step #1: Is this animal having trouble breathing (*e.g.,* left-sided congestive heart failure or respiratory disease)? Does this animal have severe or end-stage heart disease?

If the animal is presented collapsed with signs of respiratory distress and auscultable crackles in the lung fields (*i.e.,* "loud auscultation"), severe heart disease might be suspected. Stabilize as detailed in the respiratory distress section [see Figure 3 in Section 1 in Part I of this review (1)].

Step #2: Is this animal cardiovascularly stable?

If the animal is not cardiovascularly stable, as manifested by clinical signs such as weak pulses, pale gums, hypotension, or sinus bradycardia/tachycardia:

- Evaluate ECG and measure blood pressure.
- Obtain a rapid point-of-care ultrasound (if available) of the thorax and the abdomen, looking for free fluid in the pleural space, abdomen, or pericardium.
  - Is there internal or external bleeding? (If yes, see Section 2.)
- Obtain baseline bloodwork, including at least glucose, lactate, packed cell volume/total protein (PCV/TP), sodium, and potassium (and ideally, a full CBC, chemistry, and electrolyte panel).
- Perform basic stabilization to end points as detailed in Box 1 and Box 2.

Box 1. How to perform basic stabilization of a hypotensive or hypovolemic dog or cat.

- Administer crystalloid fluids to treat shock and improve end points (see **Box 2**). Give 1/4 to 1/3 of the calculated shock dose (dog shock volume: 90 mL/kg; cat shock volume: 45 to 60 mL/kg) IV.
  - Bolus the fluids as quickly as possible in dogs. For example, in a 10-kilogram dog, give an initial bolus of 300 mL ($1/3 \times 90$ mL/kg) over ideally < 10 min.
  - In cats, administer calculated bolus over 10 to 15 min (slower if cat becomes nauseous).
  - Do not use a fluid pump in medium-to-large dogs; use a pressure bag or squeeze the fluid bag. A fluid pump can be used in most cats or in small dogs to give the fluids within the appropriate timeframe. Fluid pumps cannot give more than 250 mL within 15 min.
- Administer crystalloid fluids to end points (see **Box 2**).
- Do not worry about whether the fluids will worsen anemia; it is most important to deliver oxygen to tissues by increasing vascular volume and improving perfusion with crystalloids.
- Synthetic colloids may be considered [*e.g.,* VetStarch (Zoetis)].
  - Dogs: Administer IV in bolus volumes of 5 mL/kg given as quickly as possible (up to a total of 20 mL/kg).
  - Cats: Administer synthetic colloids in bolus volumes of 3 mL/kg over 10 to 15 min (up to a total of 10 mL/kg).
  - Give colloids with crystalloids to resuscitate to end points (**Box 2**). Colloids primarily lengthen the duration that crystalloids remain in the bloodstream rather than expand vascular volume.
  - Both human and canine studies have suggested that using synthetic colloids can lead to development of acute kidney injury and cause coagulopathies and platelet dysfunction (1–3).
- Is this animal possibly septic? (See Step #7 below for more information.)
  - If yes, beware of administering large volumes of fluids that can leak from the vasculature, cause edema, increase myocardial depression, and cause vasodilatory shock.
  - Administer broad-spectrum antibiotics as soon as sepsis is suspected.
- Transfusion of packed red blood cells or whole blood is rarely used for resuscitation. Their use is reserved for scenarios where the measured PCV/TP is < 15 to 20%[a] and:
  - total shock bolus amount of crystalloids has been given within the course of 1 h and the dog or cat is still hypotensive and/or tachycardic;
  - hypotension persists despite crystalloid and/or colloid administration;
  - blood pressure has normalized (typically, systolic blood pressure > 90 to 100 mmHg or mean blood pressure > 80 mmHg) and animal remains weak or is tachycardic.

[a] In cases of acute traumatic blood loss, significantly higher PCV values may result in weakness, tachycardia, tachypnea, and weakness, still indicating a need for transfusion.

References

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Step #3: Could this dog have pericardial effusion?

If a dog is presented with sinus tachycardia, poor femoral pulse quality, and muffled heart sounds, especially if it is a large-breed dog (*e.g.,* Labrador or golden retriever), it may have pericardial effusion. Ideally, use point-of-care ultrasound to detect presence and quantity of pericardial effusion and presence/absence of cardiac tamponade (*i.e.,* collapsed right atrium). If ultrasound is

Box 2. End point resuscitation goals for animals.

Give fluids to restore vital signs (respiratory rate, mucous membranes, and capillary refill time) and pulse quality. Ideally, heart rate will also improve to closer to normal (10 to 15% decrease from baseline). Septic animals (see Step #7) often remain tachycardic despite appropriate resuscitation. Other resuscitation goals include:

- blood glucose > 80 mg/dL,
- restoration of blood pressure (systolic: > 90 mmHg and/or 10 to 15% increase from baseline),
- normalization of elevated blood lactate levels,
- improvement of lactate (at least trending towards normal).

Box 3. How to perform pericardiocentesis.

- Place peripheral IV catheter and connect patient to an ECG.
- Place animal in sternal or left-sided recumbency.
- Ideally, use an ultrasound probe to identify the best site for pericardiocentesis (*i.e.,* where there is obvious fluid). Without ultrasound, perform pericardiocentesis at heart location: right 4th to 6th intercostal space at costochondral junction.
- Shave and clean skin.
- Give butorphanol, 0.2 mg/kg, IV, for sedation, ± lidocaine locally at site of needle insertion.
- Using a sterile technique, insert a large, long, fenestrated catheter [*e.g.,* 14- to 18-gauge (depending on animal), at least 7 to 10 cm]. May need stab incision through skin.
- Once catheter enters the pericardial space, sanguineous or serosanguineous fluid will appear in catheter hub. Advance the catheter slightly (~1 cm) while holding the stylet. Remove stylet, connect catheter to extension set with 3-way stopcock, and drain fluid.
- Before removing large volumes of fluid, place a small amount in a red-top tube to ensure the fluid does not clot (characteristic of pericardial effusion). Clotting is suggestive that needle has entered the heart; remove catheter and try again.
- If an arrhythmia occurs when the needle is in pericardial sac, catheter may be touching the myocardium and need to be repositioned.
- If the arrhythmia is severe (usually a ventricular arrhythmia), give 2 mg/kg bolus of lidocaine slowly, IV, and start CRI if arrhythmia persists.
- Start IV crystalloids once pericardial fluid has been removed and there is no tamponade.
- Monitor ECG and blood pressure to ensure they have improved.

not available, use thoracic radiographs to detect a large, globoid heart (may be absent with limited pericardial effusion or acute pericardial bleeding). On an ECG, P-QRS complexes alternate height depending on heart movement (closer or farther from ECG leads; electrical alternans).

Background

During cardiac tamponade, fluid in the pericardial sac increases intrapericardial pressure, leading to diastolic collapse of the right atrium and/or ventricle. Blood cannot fill the right side of the heart and less blood is available to pump (decreased cardiac output), leading to cardiogenic shock which manifests as hypotension, pale gums, collapse, tachycardia, cold extremities, weak pulses, and muffled heart sounds. Ascites may also be present, as well as pulsus paradoxis (palpated pulse is stronger during expiration and weaker during inspiration). Immediate pericardiocentesis is needed to relieve pressure on the right side of the heart.

Pericardial effusion ± only mild tamponade manifests as collapse, lethargy, increased respiratory rate and effort, anorexia, and vomiting (3). Physical examination usually reveals a prolonged capillary refill time (CRT), pale gums, muffled heart sounds, weak pulses, possible arrhythmias, and tachycardia.

Causes for pericardial effusion include cardiac neoplasia (*e.g.,* hemangiosarcoma, chemodectoma, lymphoma, mesothelioma), idiopathy, secondary to a left atrial tear or secondary to coagulopathy, systemic inflammatory disease (4), or infectious diseases (5). Idiopathic or neoplastic causes are the most likely to produce volumes of fluid leading to tamponade. Depending on etiology, animals with pericardial effusion have a poor to guarded long-term prognosis.

Emergency stabilization

If the animal is collapsed or hemodynamically unstable, perform pericardiocentesis (Box 3) to reduce the risk of dying during transport to a referral clinic.

Practice tips

1. DO NOT perform pericardiocentesis if patient is stable (normal blood pressure, ambulatory, normal ECG or very mild arrhythmia) and can be transferred to a referral center within 20 to 30 min. Ideal echocardiographic visualization for disease such as a heart-based mass occurs with some pericardial effusion present.
2. Do not give diuretics or other medications, as they may cause hypovolemia and worsen clinical signs. Diuretics are only indicated with clear signs of pulmonary edema and left-sided congestive heart failure.
3. Obtain an echocardiogram with a cardiologist to further investigate the underlying cause and provide prognostic information.

Step #4: Is this cat suffering from an aortic thromboembolism?

If there is concern that a cat is in acute pain and dragging 1 or more limbs with decreased pulses in that limb, the top differential diagnosis is an aortic thromboembolism [see Part III of this review (2)].

Step #5: Does this animal have a severe tachy- or bradyarrhythmia?

If a dog or cat is collapsed due to a severe cardiac arrhythmia with heart rate > 180 bpm (dog) or > 240 bpm (cat) or < 60 bpm (dog or cat), stabilize as detailed for arrhythmia [see Part III of this review (2)].

Step #6: Is there internal or external bleeding?

If the animal has collapsed due to severe bleeding, stabilize as detailed in Section 2 and Figure 1.

Step #7: Is this animal septic?

Sepsis is a clinical syndrome of systemic inflammatory response to a source of infection (bacterial, viral, fungal, or protozoal). Untreated, this can lead to septic shock, multi-organ failure,

Table 1. Common sources of infection in septic dogs and cats.

Type	Source
Gram-positive bacteria	Skin Injured soft tissue Intravenous catheters Respiratory system (pneumonia/pyothorax)
Gram-negative bacteria	Urogenital system (pyometra/pyelonephritis) Gastrointestinal system (most common in dogs and cats) Trauma Osteomyelitis Bite wounds Endocarditis

and possibly death. Early detection and rapid and aggressive treatment are critical.

Background

Sources for infection in septic animals are numerous (Table 1). In both dogs and cats, the most common source is leakage from the gastrointestinal tract due to obstruction, dehiscence of a previous surgical site or biopsy, or gastrointestinal neoplasia (6).

Depending on the stage of the condition (acute *versus* late), clinical signs of sepsis in dogs and cats include fever or hypothermia, tachycardia, bounding or poor pulse quality, injected or pale mucous membranes, rapid or slow CRT, hypotension (systolic blood pressure < 80 mmHg), and hypoglycemia (blood glucose < 60 mg/dL). Some animals have vomiting, diarrhea, weakness, lethargy, or anorexia.

Emergency stabilization

The initial resuscitative goal is to restore hemodynamic stability with crystalloids. To avoid fluid overload, "Give them as much [fluid] as they need and not a drop more" (7). The first 6 h after diagnosis of sepsis are critical; perform basic stabilization (Box 1) to end points (Box 2).

Practice tips

1. Many septic animals have abdominal effusion (*i.e.,* septic peritonitis), which gives a clue to underlying disease. Abdominocentesis (± ultrasound) will provide a fluid sample; compare glucose and lactate concentrations to those in peripheral blood.
   - If abdominal fluid glucose concentration is ≤ 20 mg/dL than peripheral blood, there is a very high chance of septic peritonitis (8).
   - If abdominal fluid lactate concentration is ≥ 2 mmol/L than peripheral blood, there is a high chance of septic peritonitis (8).
2. In-house fluid cytology identifying intracellular bacteria confirms sepsis.
3. After fluid therapy is initiated, obtain a quick ultrasound or repeat abdominal tap, especially if no obvious fluid is detected initially; fluid administration may produce more fluid.
4. When in doubt, treat an animal as if it has sepsis: carry out cautious fluid resuscitation, treat hypoglycemia, and start broad-spectrum antimicrobials.

Table 2. Clinical signs of anaphylaxis in dogs and cats.

System affected	Clinical signs
Cardiovascular	Sinus tachycardia Sinus bradycardia (cats) Collapse Pale mucous membranes and prolonged CRT Hypotension Hypothermia Arrhythmia
Respiratory	Dyspnea Tachypnea Bronchospasm Stridor (dog) Open-mouth breathing (cat)
Digestive	Vomiting Diarrhea (± hemorrhagic) Nausea Hypersalivation
Cutaneous	Pruritus Erythema/hyperemia Urticaria Facial edema
Neurologic	Weakness Seizures Incoordination Depression/stupor
Ocular	Blepharospasm Conjunctival hyperemia Chemosis

CRT — Capillary refill time.

Step #8: Is this animal in anaphylactic shock?

Anaphylaxis is a systemic, immediate hypersensitivity reaction caused by IgE-mediated release of mediators from mast cells and basophils. Anaphylaxis develops a few minutes after contact with the allergen and affects multiple organs. In dogs, "shock organs" are gastrointestinal and liver. In cats, anaphylaxis primarily affects pulmonary and gastrointestinal systems. In both species, cardiovascular, respiratory, neurologic, and cutaneous systems can be affected (Table 2). Signs of hepatic involvement manifest as increases in serum alanine aminotransferase, gallbladder edema, and sometimes fulminant liver failure (including elevated clotting times), most of which become apparent chronically.

Background

Clinical signs of anaphylaxis vary and depend on reaction severity, interval after reaction, and patient comorbidities. Multiple organ systems are affected but not all clinical signs will be present (Table 2) (9). Causes of anaphylaxis include ophthalmic antibiotics (cats) (10), vaccines, envenomation, insect bites, antibiotics, blood products, NSAIDs (dogs), opioids, contrast substances, and food (9).

Emergency stabilization

Provide immediate medical attention to a dog or cat with anaphylaxis as its condition can rapidly deteriorate.

- If respiratory distress is present, provide oxygen immediately. Severe upper airway edema may require intubation [see Section 1 in Part I of this review (1)].
- Place peripheral IV catheter as soon as possible.

- If hypotension is noted, see Box 1.
- If the animal is not yet hypotensive, start crystalloids at 120 mL/kg per day.
- Epinephrine, IV or IM, 0.01 mg/kg. Can repeat every 5 to 15 min (maximum: 0.5 mg/dog).
- Diphenhydramine, 2 mg/kg, IM + ranitidine, 0.5 to 2.5 mg/kg diluted, slowly IV (10 min).
- If bronchospasm is suspected, albuterol (1 or 2 puffs) may be given by metered-dose inhaler every 15 min, up to 3 doses. Alternatively, administer aminophylline, 5 to 10 mg/kg, IV, if albuterol is not available and the dog or cat is in respiratory distress due to bronchospasm.

Practice tips

1. Steroids are *contraindicated in acute anaphylactic shock.* The dog or cat may receive a low dose of steroids once it is hemodynamically stable.
   - Steroids may increase gastrointestinal tract damage if the animal is not hemodynamically stable.
   - Steroids are largely used for anti-inflammatory effects with urticaria, facial edema, and pruritus. Their response is too slow (several hours) to be beneficial in acute anaphylaxis (9,11).
2. Many dogs with anaphylaxis have abdominal effusion consistent with blood (similar hematocrit to peripheral blood, low TP). It is VITAL not to confuse anaphylaxis with hemoabdomen and take the dog to surgery. Anaphylaxis-induced hemoabdomen commonly resolves on its own but in some cases may require red blood cell and/or plasma therapy if there is a coagulopathy induced by anaphylaxis (12,13). Consider a prothrombin time/activated partial thromboplastin time test to evaluate the coagulation system.
3. Point-of-care ultrasound may reveal hepatic congestion and gallbladder wall edema in dogs, which may help to suggest anaphylaxis (14).
4. Animals with anaphylaxis will have increased alanine aminotransferase (14), likely secondary to liver hypoxia.
5. Inform the owner that the dog may require a few days in the hospital for full recovery, as rebound reactions can occur 24 to 48 h after the first anaphylaxis episode.

Step #9: Is this dog (rarely cat) suffering from heat stroke?

Heat stroke is caused by exposure to a high environmental temperature or strenuous exercise. A core body temperature > 41.0°C is consistent with heat stroke (15). The severity of clinical signs is determined by duration and maximal temperature.

Severe hyperthermia causes a systemic inflammatory response potentially progressing to multi-organ failure, including circulatory collapse, encephalopathy, acute respiratory distress syndrome, acute liver and renal failure, coagulopathy, rhabdomyolysis, intestinal ischemia, and eventually death.

Background

Heat stroke is most frequent during summer when the weather is hot and humid. However, abrupt transition from cool to warm (*e.g.,* in the spring) can also lead to heat stroke. Working dogs and brachycephalic breeds are predisposed but heat stroke can occur in any dog confined in a hot or humid environment without proper ventilation and water. Upper airway disease (*e.g.,* laryngeal paralysis) or obesity impairs panting and predisposes to heat stroke.

Box 4. How to cool a hyperthermic dog (or cat).

- Put cool water on the body and neck and use a fan to improve convective heat loss.
- Provide IV fluid therapy with room-temperature crystalloid fluids, with rate based on needs (more if the animal is in shock) and presence/absence of pulmonary edema or respiratory distress.
- Apply ice packs to jugular veins and head only (peripheral vasoconstriction will slow cooling).
- Stop cooling measures when rectal temperature reaches 39.4°C (to avoid rebound hypothermia).
- If hyperthermia is severe and persistent, consider a cold-water retention enema (10 to 20 mL/kg of cold water in rectum *via* long red rubber catheter). This invalidates rectal temperature.

Clinical signs include, but are not limited to, a wide range of body temperatures (depending on stage of disease progression and attempted cooling measures), panting, tachycardia with weak pulses, hyperemic gums, a wide range of neurologic signs (from normal mentation to prostration and coma, ataxia, or seizures), hemorrhagic diarrhea, and petechia (16).

The history may provide information about heat stroke cause and duration. Prognosis varies based on severity of clinical signs, duration of heat, previous medical conditions, and secondary complications. Poor prognostic indicators are persistent neurologic deficits, hypoglycemia (17), severe or refractory hypotension, arrhythmias, disseminated intravascular coagulation, and acute renal injury (18). In a retrospective study, mortality was 50% (18).

Emergency stabilization

If the animal is hyperthermic, provide cooling measures immediately (Box 4). It is difficult to assess respiration or mentation until the animal has been cooled.

Many dogs with heat stroke are presented with respiratory difficulties. Since many hyperthermic dogs are brachycephalic or have underlying upper airway disease, ensure the airway is patent and the dog is able to ventilate. Dogs with heat stroke may be presented with another lung pathology that reduces oxygenation (*e.g.,* pulmonary edema, pulmonary bleeding, or a pulmonary inflammatory response due to heat-induced tissue and endothelial injury).

Provide oxygen while still allowing panting. Avoid placing the animal in an oxygen cage with poor ventilation or temperature control or using a tight oxygen mask; use a loose mask or flow-by oxygen. If respiratory distress continues despite oxygen therapy, treat as described for respiratory distress in Section 1 in Part I of this review (1).

Dogs with heat stroke are also in shock and can have arrhythmias. If the animal is cardiovascularly unstable (*i.e.,* low blood pressure, poor peripheral pulses, obtunded, tachy- or brady-arrhythmia), address any arrhythmias [see Part III of this

review (2)] and stabilize as described in Box 1 to end points (Box 2). Administer antibiotics to prevent sepsis (Table 1).

Ideally, do baseline bloodwork including CBC, chemistry with electrolytes, and coagulation profile. At a minimum, monitor blood glucose, lactate, and PCV/TP hourly until the animal is stable. Ideally if a patient is referred, its blood glucose should be > 90 mg/dL, lactate should be trending down towards normal, and PCV/TP should be > 20%.

Practice tips

1. Avoid alcohol on paws or ice water baths, as vasoconstriction traps hot blood centrally.
2. Avoid steroids or NSAIDs (gastrointestinal tract is already damaged).

Step #10: Is this dog or cat suffering from metabolic derangements (hypo-/hyperglycemia, electrolyte abnormalities, anemia/polycythemia, toxin ingestion)?

Background

Most metabolic diseases cannot be addressed immediately and require further diagnostics. Some metabolic derangements (hypoglycemia, hyperkalemia, severe anemia) lead to collapse and can be fatal if not addressed immediately.

In an emergency room, 28% of dogs and 16% of cats with hyperkalemia (potassium > 6 mEq/L) had muscle weakness (19), which can contribute to unwillingness to stand. Hyperkalemia will cause bradycardia and other cardiac arrhythmias, including atrial standstill and asystole (death), if untreated. Potassium concentration > 8.0 mg/dL causes the most profound cardiac changes, with arrhythmias and bradycardia occurring at lower concentrations. Hyperkalemia occurs most commonly in dogs and cats with urethral obstruction or anuric/oliguric renal failure, and in dogs with hypoadrenocorticism.

Obtain a full chemistry panel, point-of-care analyzer panel, or, at a minimum, blood glucose and PCV/TP in collapse cases.

Emergency stabilization

Hypoglycemia. Correct hypoglycemia immediately when identified (Box 5).

Hyperkalemia. If a cat or dog is exhibiting changes to heart rate or rhythm or potassium concentration is > 7.5 mEq/L, correct potassium immediately to prevent death.

- 10% calcium gluconate, 0.5 to 1.5 ml/kg, IV over 15 min; this does not change potassium concentration but rapidly and transiently (30 to 60 min) corrects arrhythmias.
- Regular insulin, 0.25 to 0.5 U/kg, IV; then 4 mL of 50% dextrose/unit of insulin (diluted), IV. Insulin reduces potassium concentration (may take 20 to 30 min; lasts several hours).
- Treat underlying cause of hyperkalemia.

Anemia. DO NOT refer an animal without correcting hypotension (Box 1). Tachycardia occurs secondary to anemia, so crystalloid or colloid fluids will likely not fully correct tachycardia. Blood pressure should improve as fluid volume improves peripheral perfusion. Administer bolus fluids as needed to obtain systolic blood pressures ≥ 70 to 80 mmHg before sending an anemic animal to a referral clinic.

Box 5 How to correct hypoglycemia.

- Correct hypoglycemia immediately. If a peripheral venous catheter is present, give a bolus of 50% dextrose diluted at least 1:1 with saline to reduce osmolality and lessen chance of phlebitis and red blood cell hemolysis. If an intraosseous catheter is used, dilute the 50% dextrose 1:4 or 1:5 before administration.
- Doses vary widely, but a common starting dosage is 0.5 to 1.0 mL/kg of 50% dextrose, IV or IO (diluted at least 1:1 with saline) (1,2). It is extremely important to check blood glucose at 5 to 10 min after giving the dextrose bolus, to ensure the blood sugar has normalized. *Do not assume that a single dose of dextrose has corrected the hypoglycemia.*
- If the dog or cat remains hypoglycemic, do not hesitate to re-dose the dextrose (same or increased amount) and check again. Consider placing the animal on a CRI of crystalloid fluids with 2.5 to 5% dextrose to maintain normoglycemia. If the animal is hyperglycemic after the bolus, do not be concerned; endogenous insulin release normalizes the blood glucose. Transient iatrogenic hyperglycemia is not dangerous.
- If a catheter is not present, give sugar orally. Apply 50% dextrose, corn syrup, honey, or maple syrup to mucous membranes in the mouth. This may be less successful than IV treatment; oral glucose induces a greater insulin response than IV glucose administration *via* induction of gut incretins such as glucagon-like protein (GLP-1; *e.g.,* the incretin effect) (1–3). Recheck blood glucose 5 to 10 min after oral dextrose administration to determine if hypoglycemia has been corrected.

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Practice tips

1. If a dog or cat had clinical hypoglycemia at presentation and duration of travel to a referral institution is > 30 to 60 min, consider preparing the owner to give oral or IV dextrose *en route.*
2. When assessing PCV/TP to detect anemia, evaluate serum for icterus. This can suggest differential diagnoses (*e.g.,* anemia with icterus implies immune-mediated hemolytic anemia). If the animal is hyperglycemic, test serum for ketones (place serum on a urine dipstick ketone pad) to diagnose diabetic ketoacidosis. This information is important for prognostication and suggesting approximate costs of treatment before referral.

Step #11: Did this animal ingest a toxin?

Background

There are numerous sources of toxin ingestion (*e.g.,* prescribed and illicit drugs, household plants, insecticides, and herbicides). Following is a succinct, general approach to stabilization after ingestion of any toxin; for further information on a specific toxin, the reader is referred to other sources.

Clinical signs of toxicity vary based on age, underlying medical conditions, toxin type, toxin amount, and interval since ingestion. Obtain a thorough but rapid history including

name of drug and active substance, identity of item ingested, or name/picture of plant or mushroom ingested. If a drug was ingested, ascertain if it is extended-release, long-acting or sustained-release. Finally, determine if the animal exhibited clinical signs at home and if the owner administered medications or attempted to induce emesis.

Emergency stabilization

- Ensure the animal is stable (cardiovascular and respiratory).
- Induce emesis ONLY in asymptomatic patients with recent ingestion (< 4 to 6 h) or unknown time of ingestion.
  - — Dogs:
    - Apomorphine, 0.03 mg/kg, IV (compounded) or in conjunctival sac (tablet).
    - Ropinirole ophthalmic solution (dopamine agonist) approved for topical use.
  - — Cats:
    - Xylazine, 0.4 to 0.7 mg/kg, IM (20,21); dexmedetomidine 7 to 10 μg/kg, IM (22); or hydromorphone, 0.1 mg/kg, SC (23).
    - It is difficult to induce emesis in cats.
  - — If the animal is comatose or hyperthermic, has tremors or abnormal mentation, or otherwise is not a safe candidate for induction of emesis, consider gastric lavage.
- Administer activated charcoal with or without a cathartic.
  - — Use if ingested toxin binds to charcoal. Can give 1 to 3 g/kg, PO, once (24).
  - — Avoid activated charcoal with metals, caustic substances, or very small molecules (*e.g.,* xylitol, alcohol, iron, or zinc) (24).
  - — Avoid activated charcoal in dogs with megaesophagus, history of arytenoid lateralization surgery, or if predisposed to aspiration pneumonia.
- Administer appropriate antidote (if known).
  - — Consider 20% IV lipid administration for lipid soluble toxins that will be "trapped" in the "lipid sink" created by the lipid infusion. Examples include ivermectin, moxidectin, lidocaine and other local anesthetics, calcium channel blockers, pyrethrins, bromethalin, and cannabis (24).
  - — Administer lipids as 1.5 ml/kg bolus, followed by 0.25 mL/kg per minute for 30 to 60 min.
  - — If clinical signs persist, continue giving bolus doses of 1.5 mL/kg, q4h to q6h for 24 h. If clinical signs do not improve after 24 h, discontinue administration. Monitor serum for lipidemia every 4 to 6 h and discontinue administration if lipemia is noted.
  - — The use of intralipids is off label in animals. Reported side effects include pancreatitis, lipemia, corneal cholesterol deposits (in cats; resolved within 2 wk), fat-overload syndrome, coagulopathy, and hypersensitivity reactions.

Practice tip

Call either of these centers for a detailed veterinary toxicology consultation (fee per call):

- ASPCA Animal Poison Control: 888-426-4435.
- Pet Poison Helpline: 855-213-6680.

Step #12: Was the dog or cat electrocuted?

Background

Electrocution is very rare and usually is caused by chewing on an electrical cord. Depending on current intensity and type (alternating current causes more damage than direct current) (25), the severity of clinical signs will vary.

Electrocution may cause muscle spasms, loss of consciousness, arrhythmias, respiratory distress, or cardiorespiratory arrest. The most frequent clinical sign is respiratory distress due to either upper airway edema or noncardiogenic pulmonary edema [see Section 1 in Part I of this review (1)]. Dogs and cats can also have superficial- to full-thickness burns on the tongue, lips, or roof of the mouth.

Emergency stabilization

- Stabilize respiratory distress [see Section 1 in Part I of this review (1)].
- Provide pain medication (ideally, full mu-agonist opioid; *e.g.,* methadone, 0.2 mg/kg, IV).
- Due to concerns for worsening noncardiogenic pulmonary edema, give small volume of IV fluids or hypertonic saline (3 mL/kg of 7.2% NaCl diluted 1:4 with sterile water, slowly over 10 min) rather than larger boluses of crystalloid fluids.
- Treat any cardiac arrhythmias [see Part III of this review (2)].
- Treat seizures [see Section 2 in Part I of this review (1)].

Practice tips

1. Check electrolytes in electrocution cases; hyperkalemia occurs due to tissue necrosis or persistent muscular convulsions during electrical shock.
2. Urinalysis may indicate myoglobinuria or hemoglobinuria secondary to muscle necrosis. Do not worry if you cannot obtain a urine sample immediately.
3. Animals may require prolonged hospitalization, feeding tube, or reconstructive surgery.
4. Some dogs develop cataracts months after electrocution.

Step #13: Did this animal collapse due to orthopedic or neurologic disease (*e.g.,* fractures, spinal cord disease)?

Evaluate as detailed in Part III of this review (2).

Preparing to refer a dog or cat that is presented collapsed

- The time to refer the animal is *after* stabilization of vital signs. Treat respiratory distress and normalize heart rate, blood pressure, mucous membrane color, and other vital signs.
- Treat hypoglycemia.
- Perform pericardiocentesis, if required, to relieve cardiac tamponade.
- Cool a dog with heatstroke or electrocution to 39.4 to 40.0°C.
- In a septic animal or dog with heatstroke, give the first dose of antibiotics.
- In an animal with anaphylactic shock, give at least 1 dose of epinephrine.

- Decontaminate dogs and cats with toxin exposure by inducing emesis (or gastric lavage) ± activated charcoal.
- Treat any pain.
- Inform the owner that the animal may decompensate during transfer, especially in cases of pericardial effusion, sepsis, aortic thromboembolism, or anaphylaxis.
- Ensure the animal has a patent IV catheter, if possible.
- Advise referral institution of times and doses of all drugs administered.
- Call ahead to the referral institution to alert them and discuss the case.

Section 2: Acute bleeding in the dog or cat

Anemia is commonly a reason for referral, but chronically anemic animals rarely require immediate cessation of bleeding. Acute bleeding identified during triage is addressed immediately (Figure 1).

Background: Reasons for acute bleeding

There are 2 major reasons for bleeding: physical damage to a blood vessel or coagulopathy.

Non-coagulopathic hemorrhage. Most cases of a damaged vessel causing hemorrhage are due to trauma (blunt force or secondary to neoplastic cells eroding a nearby blood vessel) or occur post-surgery. Theoretically, bleeding will stop when a clot can form.

Coagulopathic hemorrhage. Dogs with coagulopathies bleed due to an inability to form a clot and may not stop bleeding until the coagulopathy is addressed. Coagulopathies are rare in cats but behave similarly to those in dogs.

Coagulation involves interactions among the vascular endothelium, platelets, and coagulation factors. The coagulation system has 2 parts: primary (platelet-based) and secondary (coagulation factor-based) hemostasis.

Primary hemostasis. Platelets provide a surface for a clot to form and have granules containing factors and proteins that aid in coagulation. They are covered in surface glycoproteins that mediate adhesion to the endothelium and bind to other platelets, mediating signaling between platelets and other cells.

Platelet plug formation is divided into 3 phases: adhesion, activation, and aggregation. Damaged endothelium can attract and adhere to platelets. These platelets become activated, changing shape, altering surface charge, and releasing granule contents to promote platelet aggregation and create a platelet plug. These processes occur in tandem and concurrent with the secondary coagulation cascade (Figure 2). Inadequate platelets (thrombocytopenia) or dysfunctional platelets (thrombocytopathy) lead to primary coagulopathies.

Secondary hemostasis. The secondary coagulation cascade is mediated by hepatic-sourced clotting factors, is activated by the same tissue endothelial damage that causes platelet adhesion, and occurs on the surface of platelets (Figure 2). The initial phase (initiation phase) produces a limited amount of thrombin (factor II), whereas the subsequent amplification and propagation phases culminate in large amounts of thrombin and fibrin. Bleeding occurs with deficiencies in ≥ 1 coagulation factor(s).

Step #1: Stop the bleeding

Emergency stabilization

External hemorrhage might be arterial or venous and commonly occurs from the nose, mouth, or peripheral wounds. Internal hemorrhage [abdomen, thorax, gastrointestinal tract, or around fractures (fracture hematoma)] is more difficult to identify on physical examination. When you have identified external hemorrhage or are faced with a weak or collapsed animal (Section 1) in which you suspect internal hemorrhage or have ultrasonographically identified free fluid (abdomen, thorax, or pericardium), take immediate steps to stop bleeding.

Option 1: Apply pressure wrap and/or tourniquet

The most common way to control bleeding is to apply pressure using a hand, gauze, or hemostatic dressing (26). Ideally, a circumferential wrap, including inner layers of rolled gauze and conforming gauze with an outer layer of self-adherent bandage material, is applied and pulled tightly (27). Ensure the wrap does not impede breathing if placed around the head, neck, or thorax. If possible, elevate the bleeding area 15 cm above the heart (27).

A pressure wrap can be applied around the abdomen or around fractures, using material similar to that for wrapping an extremity (28). Abdominal pressure wraps should extend from the hind limbs to above the diaphragm. Usually, folded towels are placed between the hind limbs, in the inguinal region bilaterally, and on the ventral abdomen to equalize pressure and facilitate wrapping (28). Increased intra-abdominal pressure may slow bleeding from organs or veins but may not suppress arterial bleeding. Wraps surrounding a fracture should extend from the joint above to the joint below the fracture and are placed as a tighter modified Robert Jones bandage or splint.

A tourniquet can be used judiciously for distal extremity or tail wounds, but due to concerns about direct or ischemic tissue damage (28), use this only if pressure wraps are not practical or ineffective and bleeding is life-threatening. Tourniquets should be > 5 cm wide and applied proximal to the bleeding, tightly enough to eliminate a distal palpable pulse. More than 1 tourniquet can be used if bleeding and/or a pulse continue (27,28). Avoid tourniquets over joints, as they cannot be tightened sufficiently and will cause long-term damage.

If there is any strikethrough of the bandage material, DO NOT remove the existing material (this disturbs the clot) but add additional dressing and self-adherent bandage material (28).

Option #2: Other methods of achieving hemostasis

When bleeding is not amenable to a wrap, application of pressure, or a tourniquet (*e.g.,* oral or nasal bleeding), other options must be employed.

Sedation. Sedation is helpful in hemostasis as it can i) allow safe access to places such as the mouth to facilitate application of pressure or hemostatic agents, and ii) reduce peripheral blood pressure. Common drugs include dexmedetomidine (dog: 2 to 10 μg/kg, IV or IM; cat: 5 to 10 μg/kg, IV or IM) or acepromazine (dog or cat: 0.05 to 0.2 mg/kg, IV or IM). Dexmedetomidine has peripheral vasoconstrictive effects, rapid

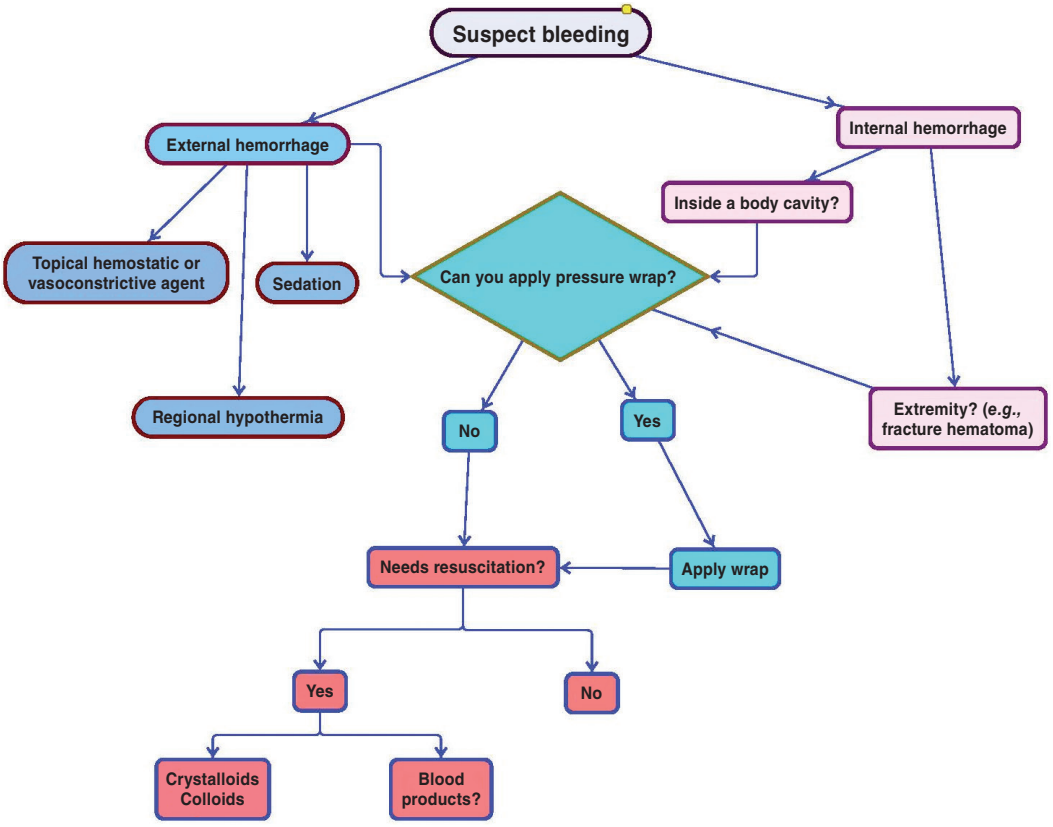

Figure 1. Flowchart illustrating the approach to a bleeding patient.

onset, and is reversible. Acepromazine has peripheral vasodilatory effects but a slower onset. An opioid is often combined with either drug for analgesia and sedation. General anesthesia (gas or injectable) can be used to vasodilate and slow blood flow plus facilitate application of pressure or other interventions.

Hemostatic agents are used for external hemorrhage; their most common components are chitin/chitosan, zeolite, and kaolin. Chitin/chitosan exists in arthropod skeletons and is produced by algae (29). It causes vasoconstriction to hold platelets and clotting factors locally and promote clot formation (29); it also has antibacterial activity (29). Zeolite agents [*e.g.,* QuikClot (Teleflex)] contain minerals that absorb blood and fluid and promote clot formation (29). Kaolin-based agents activate factor XII and thus intrinsic and common pathways of coagulation (Figure 2). These were shown to be efficacious in human clinical (30) and *in vitro* (31) studies.

Hemostatic agents can be used in granular form or impregnated in gauze and applied to extremity wounds before pressure bandages (30). For a bleeding nose or mouth or other region not amenable to pressure, continued bleeding, saliva, or the tongue can remove granules; however, impregnated gauze packed into or held on the site may be effective (sedation is likely necessary).

Other. Bleeding may be controlled by epinephrine or phenylephrine on gauze applied topically to surfaces such as the nose (sedation is likely necessary). Chilling (ice or cold water) can cause vasoconstriction and slow bleeding.

In adolescent humans with cancer-related bleeding who were receiving conventional therapy, topical Yunnan Baiyao improved hemostasis (32). No veterinary studies have explored topical use of Yunnan Baiyao, but anecdotally, the author (LI) feels that topically applied powder from Yunnan Baiyao oral capsules induced hemostasis. Oral use of Yunnan Baiyao in dogs is controversial, with 1 study (in healthy, non-bleeding dogs) suggesting changes in thromboelastographic variables consistent with improved hemostasis (33), another reporting no change in thromboelastographic variables (34), and a 3rd reporting no change in clinical bleeding in dogs with pericardial effusion (35). Healthy cats given oral Yunnan Baiyao had no changes in thromboelastographic variables (36). The appropriate dose and interval for slowing or stopping bleeding with oral Yunnan Baiyao in cats and dogs are unknown.

Step #2: Resuscitate the dog or cat

If substantial blood loss has occurred or bleeding is internal and cannot be immediately stopped, treatment is needed. Hypovolemia (causing hypotension and shock) is a more emergent issue than anemia. Feline shock is identified by hypotension, hypothermia, and bradycardia (in contrast to tachycardia in dogs). See Box 1 for treatment and stabilization of hypovolemia and hypotension. In some cases where ongoing hemorrhage is occurring while awaiting definitive repair (such as with a bleeding splenic mass), permissive hypotension is allowed. Thus, resuscitation goals are to keep the systolic blood pressure high enough to deliver oxygen, but not in the normal range. No consensus exists as to the exact blood pressure goals, but an example might be systolic blood pressures between 60 to 90 mmHg

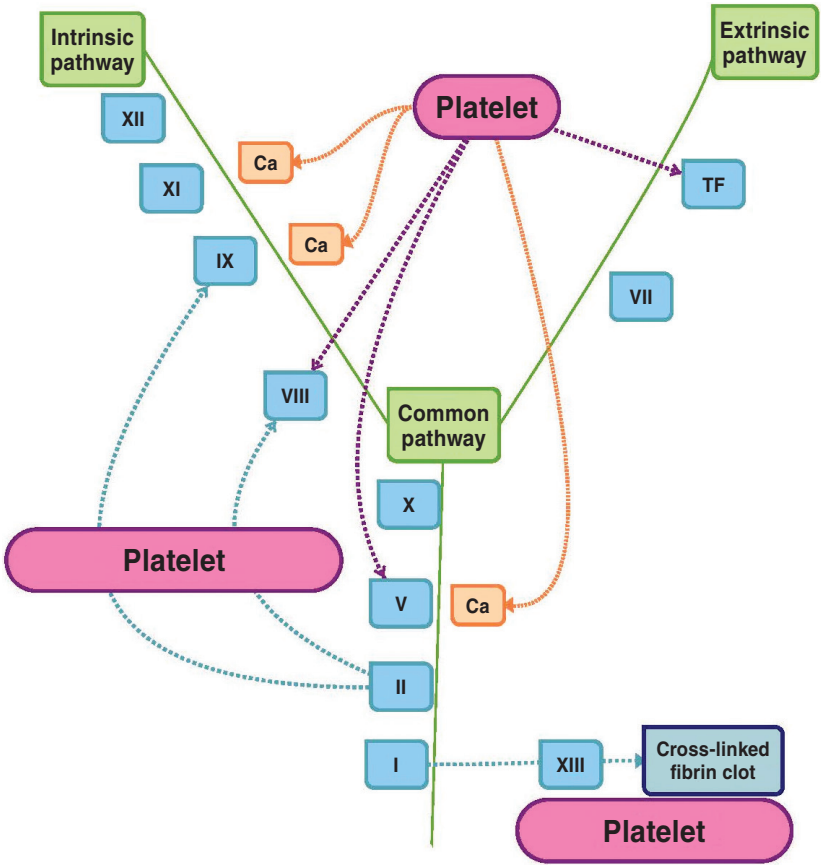

Figure 2. The secondary coagulation cascade.

rather than in the normal range (*i.e.,* > 120 mmHg) until the bleeding has been stopped (37).

Practice tips

1. Differentiation of primary *versus* secondary coagulation disorders is not important during triage. Stopping bleeding (if possible) and resuscitation (if needed) are the most important first steps.
2. Continue monitoring after treatment. Animals may go back into shock [tachycardia (or bradycardia in cats), hypotension, declining mentation], implying continued bleeding. Repeat resuscitation with fluids, colloids, and/or blood products while reexamining for a source of active hemorrhage. This occurs mostly with internal hemorrhage (*e.g.,* splenic masses or coagulopathies) for which control of bleeding is difficult.
3. Definitive treatment for internal hemorrhage requires transfusion to provide platelets and/or clotting factors (coagulopathy), or surgery (*e.g.,* splenectomy).
4. A CBC, blood smear evaluation, and prothrombin time/activated partial thromboplastin time test are the first steps to differentiate primary *versus* secondary coagulation disorders.

Preparing to refer a cat or dog that is bleeding

- External hemorrhage: Refer animal after controlling active external hemorrhage with a wrap and/or tourniquet, plus resuscitation as required.
- Internal hemorrhage: Refer animal after initial resuscitation to improve hypotension. Stopping internal hemorrhage is difficult without a specific diagnosis.
- Communicate to referral institution times and doses of all fluids, blood products, and drugs administered.
- Call ahead, especially if you expect bleeding to occur during transportation.

Refer to Part III of this review [Basic triage in dogs and cats: Part III (2)] for information on the approach to arrhythmias and stabilization of animals that are emergently unable to stand or walk. CVJ

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