The Patient with Collapse Syncope versus Seizure and Much More!

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Elisa M. Mazzaferro, MS, DVM, PhD, DACVECC
Cornell University Veterinary Specialists, Stamford, CT, USA

Acute collapse is often an incredibly alarming situation for a pet owner and can represent a diagnostic challenge for the veterinary practitioner if the event is not witnessed or recorded. A careful history, as well as a thorough physical examination should be obtained in order to decipher the cause of acute collapse in dogs and cats. The term “collapse” is generally defined as a loss of postural tone with or without a loss of consciousness. One of the first questions to ask is whether the animal experienced a loss of consciousness. Some pet owners cannot answer that question accurately, as the animal usually keeps the eyes open, even when loss of consciousness has occurred. I often will ask whether the animal was responsive or unresponsive to verbal or tactile stimuli during the event. If the animal was not responsive, then I err on the side that there was a loss of consciousness until proven otherwise. If there is loss of consciousness, primary differential diagnoses include seizure or syncope, versus neuromuscular or metabolic causes of collapse. One other question to ask, particularly if there was not a loss of consciousness or a history of being unresponsive, is whether the collapse affected all four limbs, or affected only the front or rear, right or left side of body. This can lead to pursuing neuromuscular causes of collapse, or generalized weakness from metabolic disorders (hypoglycemia or hypokalemia for example) or anemia.

TRIAGE OF THE COLLAPSED ANIMAL

Any animal that has collapsed, or is nonambulatory at the time of presentation, should be prioritized on the triage list. Careful assessment of whether the animal is conscious or unconscious, is currently having a seizure, or is responsive to external stimuli should be performed very rapidly as personnel obtain permission for emergency stabilization and placement of an intravenous catheter. If a seizure is suspected or witnessed, placement of the catheter in the rear limb should be prioritized, to allow vascular access without compromising personnel safety during administration of an anti-convulsant in a front limb near an animal’s jaws and teeth.

The animal’s vital signs of temperature, heart rate/rhythm, pulse quality, respiratory rate and effort as well as peripheral perfusion should be assessed. If an animal has muffled heart sounds, pleural or pericardial effusion should be suspected, however, hemorrhage or fluid loss elsewhere in the body, such as from abdominal or retroperitoneal hemorrhage can cause loss of circulating blood volume/hypovolemia that results in microcardia and decreased or muffled heart sounds. All the above can cause decreased peripheral perfusion

and poor pulse quality, however, some animals will demonstrate pulsus paradoxus, with a fall in arterial blood pressure and pulse quality during inhalation with pericardial effusion/tamponade.

Once vascular access has been obtained, additional diagnostic testing in the form of blood pressure, pulse oximetry and cursory thoracic and abdominal ultrasound (TFAST/AFAST) can be performed. Diagnostic bloodwork should include a glucose, PCV/TS, venous blood gas/electrolytes and lactate, and samples obtained for blood chemistry, complete blood count/peripheral blood smear and urinalysis.

SEIZURES

Tonic-clonic, or grand mal seizures, are one of the primary differential diagnoses when a client reports that their animal has collapsed. In many cases, animals with seizures will have muscle twitching and rhythmic rigid contractions of the muscles of the face and limbs, often with hypersalivation, movement of the jaws and teeth, and paddling movement of the limbs. Urinary and fecal incontinence also may occur. Unlike syncope, animals often will display abnormal behavior before (pre-ictal) and after (post-ictal) the actual seizure event. There are numerous causes of tonic-clonic seizures that can include intracranial mass lesions or infection, increased intracranial pressure, exposure to toxin(s), hypoglycemia or cerebrovascular accident in addition to idiopathic or cryptogenic epilepsy.

Irrespective of the etiology of the seizure, the seizure itself should be controlled with use of rapidly acting anticonvulsant drugs in the emergency setting. The benzodiazepine drugs diazepam (0.5 to 1 mg/kg IV) and midazolam (0.2 mg/kg IV, IM) are considered the standard of care for abruptly stopping the ictus, or seizure event. If bloodwork shows hypoglycemia, dextrose (0.5 to 1 ml/kg 50% dextrose, or 0.25 to 0.5 g/kg 50% dextrose diluted 1:2 to 1:4 IV) should be administered to control seizures induced by neuroglycopenia. Once the seizure is controlled, additional diagnostic testing should be considered based on the patient's signalment, history (including possibilities of toxin exposure), and presence or absence of neurologic abnormalities in the inter-ictal period. Diagnostics such as an MRI to detect mass lesions or areas of inflammation/granulomatous disease, hydrocephalus, cerebrovascular accidents/infarcts may be required with or without cerebrospinal fluid analysis, cytology and infectious disease serology as possible inciting causes of the seizure. In the event that the seizure is secondary to hypoglycemia, careful questioning of possible exposure to xylitol, when a toy breed was last fed, whether the seizure or neurologic signs occur in relation to eating, or evaluation of other bloodwork abnormalities such as hypocholesterolemia, decreased sodium:potassium ratio or absence of a stress leukogram should be considered to do baseline screening and increase or decrease your index of suspicion for a

portosystemic shunt, hepatic microvascular dysplasia, or hypoadrenocorticism. Definitive diagnostic testing can include measurement of pre- and post-prandial bile acids, blood ammonia levels, abdominal ultrasound or contrast CT scan and an ACTH stimulation test. In an older dogs and cats without bloodwork abnormalities suggestive of hypoadrenocorticism, a citrated blood sample obtained at the time of glucose nadir, before administration of dextrose, can be obtained to evaluate an insulin:glucose ratio in combination with an abdominal ultrasound or CT scan to investigate for an insulin secreting tumor.

SYNCOPE

Syncope, more commonly known as fainting, occurs due to temporary lack of blood flow to the brain. Cerebral perfusion pressure is influenced by mean arterial pressure as well as intracranial pressure and vascular resistance within the brain. Any condition that decreases cardiac output or systemic vascular resistance or increases intracranial pressure can decrease oxygen delivery to the brain, and thus cause clinical signs of syncope when cerebral blood flow decreases by 30-50% of normal.

Syncope can be secondary to a variety of causes; however cardiac dysrhythmias are high on the list of differential diagnoses. In general, unless there is an ongoing cardiac dysrhythmia (sustained severe brady- or tachycardia), the episode of collapse secondary to syncope is short-lived, with a rapid recovery within seconds to a minute or less. At the time of presentation, it is useful to perform an ECG to investigate for sinus arrest, idioatrial rhythm (usually secondary to hyperkalemia, or can be lone atrial standstill in English Springer Spaniels), or high-grade AV block. Less commonly but noteworthy are tachyarrhythmias such as atrial fibrillation, atrial flutter, and ventricular and supraventricular tachycardia that decrease diastolic filling time and by Starling's Law of the heart, a decrease in contractility and cardiac output that can lead to hypotension, collapse, and/or and syncope.

Animals with third degree AV block can initially be screened using an atropine response test to help differentiate cardiac conduction abnormalities from vagally-mediated causes of AV block. To perform an atropine response test, administer 0.04 mg/kg IM atropine, and recheck the patient's ECG 20-30 minutes later. If the heart rate increases by more than 50% of that observed at baseline or is greater than 160 beats per minute, the AV block is likely secondary to conditions other than a cardiac conduction abnormality. In contrast, if the heart rate does not increase by more than 50% of that observed at baseline or if the heartrate remains less than 140 beats per minute, cardiac conduction abnormality should be considered. Some cardiac glycoside drugs (digoxin) and plants (Digitalis purpura or Foxglove, Lilies of the Valley) can cause AV block significant enough to cause collapse and syncope. If there is any possibly exposure to digoxin or plants that contain cardiac

glycosides, the treatment can be administration of Digibind (40 mg Digibind per 1 mg of digitalis ingested) in addition to atropine (0.04 mg/kg IV) until the toxin is cleared from the system. Temporary cardiac pacing may be needed in the most severely affected animals. If a cardiac conduction abnormality is diagnosed, in some cases, the animal may respond to oral administration of propantheline hydrobromide (7.5 to 15 mg PO Q8h, 3.75 mg PO Q8h in small patients) or may require implantation of a permanent cardiac pacemaker.

Noncardiogenic causes of syncope include pulmonary hypertension. Thoracic radiographs of animals with pulmonary hypertension can characteristically appear as a very heavy pulmonary interstitial pattern, sometimes with distinct tortuosity of the pulmonary vasculature. Right sided cardiomegaly and a backward D shape on a ventrodorsal radiograph also is characteristic of pulmonary hypertension, sometimes secondary to heartworm disease. A common concern is that the patient is experiencing congestive heart failure and pulmonary edema. Unlike an animal with left-sided congestive heart failure and pulmonary edema, an animal with syncope and respiratory distress secondary to pulmonary hypertension will not have dilation of the left atrium and will have primarily right sided cardiomegaly. Additionally, administration of furosemide to such patients will often result in worsening of clinical signs, as furosemide and other diuretic drugs can decrease ventricular preload to such an extent that ventricular preload is insufficient to flow against the increases in pulmonary arterial pressures such that cardiac output is adversely affected. In such cases, orthopnea and weakness will worsen, rather than improve with diuretic and oxygen therapy. Echocardiographic measurements of LA:Ao ratio will show a normal to mildly enlarged/dilated left atrium, as well as moderate to severe tricuspid regurgitation.

Questioning the owner about any event that preceded the episode of collapse/syncope can help reveal whether neurocardiogenic or vasovagal syncope has occurred. During an episode of syncope, an animal can demonstrate muscle rigidity with opisthotonos, or can be flaccid with little to no muscle tone. Urinary and fecal incontinence may also occur, making the episode difficult to discern from seizures.

Neurocardiogenic syncope occurs under certain situations that cause a sudden decrease in heart rate with vasodilation and increase in stimulation of the vagus nerve, causing acute vasomotor collapse and loss of consciousness. Situations that increase vagal tone, such as extreme excitement, coughing, vomiting or retching or urination/defecation can result in increased vagal tone with resultant syncopal collapse. Rare cases of syncope associated with swallowing have also been reported.

In addition to ECG monitoring, thoracic radiographs should be performed to evaluate for cardiomegaly, pulmonary pattern consistent with pulmonary hypertension or heartworm

disease, pulmonary nodules or intrathoracic masses. Echocardiography should also be considered to investigate for primary valvular or infiltrative cardiac disease. Depending on the findings, Holter or event monitoring may be recommended, in case the cardiac dysrhythmia is intermittent and not apparent at the time of presentation to you.

Anemia/Blood Loss

Weakness with subsequent collapse can be associated with loss of intravascular fluid volume from hemorrhage, as well as from acute or chronic anemia. A minimum database that includes a PCV to investigate for anemia should be performed to determine if anemia is present. In the event of acute hemorrhage, such as a bleeding intra-abdominal or retroperitoneal mass or acute pulmonary hemorrhage from neoplasia or Vitamin K antagonist rodenticide intoxication, peripheral PCV may not immediately reflect anemia due to splenic contraction. Even in the absence of anemia, a rapid AFAST/TFAST ultrasound examination should be performed to investigate for the presence of abdominal and/or thoracic effusion. While performing these examinations, the clinician can also rule out pericardial effusion/tamponade as another cause of acute weakness and collapse.

Metabolic Causes of Weakness/Collapse

Animals with diabetes mellitus or chronic kidney disease could develop severe hypokalemia that contributes to muscle weakness. Other potential causes of muscle weakness and collapse include thiamine deficiency in aged cats. A complete biochemical and electrolyte panel can help determine whether hypoglycemia is contributing to signs of weakness, collapse, and/or seizure activity. Severe hypothyroidism also can cause severe weakness, lethargy, and somnolence. In such instances, the animal may appear to have facial edema secondary to myxedema, as well as clinical signs of being overweight and having a poor haircoat.

Exercise Induced Collapse

Collapse can occur secondary to exercise and can be associated with a genetic defect in ability to process calcium. This has been reported in lines of Labrador retrievers. Exercise induced collapse also occurs in some lines of Border Collies. Phosphofructokinase (PFK) deficiency, noted rarely but primarily in English Springer Spaniels, can be associated with acute intravascular hemolysis, weakness and collapse after periods of exercise or excitement/panting or stress. Laryngeal paralysis, laryngeal obstruction, brachycephalic airway disease, as well as hyperthermia secondary to high ambient humidity and temperatures can also result in collapse and heat-induced illness or heat stroke.

Anaphylaxis

Severe reactions to insect or arachnid stings, administration of vaccinations or drugs such as beta-lactam antibiotics, antivenom and blood products can result in an anaphylactic or anaphylactoid reaction. In such instances, massive release of histamine in addition to prostaglandins and other vasoactive mediators cause an acute global vasodilation with a concomitant drop in systemic vascular resistance and blood pressure. Clinical signs of anaphylaxis can vary from minor pruritus, angioedema, urticaria, and erythema to vomiting and diarrhea, or more serious manifestations such as respiratory distress, tachycardia and acute death. If an inciting cause of collapse is not known, an AFAST examination can show gall bladder wall edema, a common finding in animals with anaphylaxis. Gall bladder wall edema also is recognized in animals with pericardial effusion. If gall bladder wall edema is present in an acutely collapsed patient, a TFAST should be performed to rule out pericardial effusion or tamponade. Spontaneous abdominal hemorrhage has been documented secondary to Hymenoptera envenomation. Care must be exercised in such cases to differentiate hemorrhagic peritoneal effusion from anaphylaxis versus a bleeding abdominal mass, as surgical intervention would be unwarranted and dangerous if a bleeding abdominal mass was not present.

The gold standard of therapy for anaphylaxis is epinephrine (2.5 to 5 mcg/kg IV, 10 mcg/kg IM). Once vascular access is obtained a constant infusion of epinephrine (0.05 mcg/kg/min IV CRI) appears to be more effective at improving cardiovascular stability and can be titrated according to patient response and effect. Because of the risk of epinephrine causing an increase in myocardial oxygen consumption and ischemia, this author reserves this therapy for patients with hypotension. In animals with vasomotor collapse, combination of crystalloid and colloid fluids is useful in re-expanding intravascular fluid volume and replacing fluid losses observed with severe vomiting and diarrhea. Antihistamines are also useful in the treatment of pruritus and erythema associated with anaphylaxis. Combination therapy with diphenhydramine (0.5 to 1 mg/kg IM/PO) with famotidine (0.5 to 1 mg/kg IV/IM/PO) can be used to decrease pruritus as well as gastric acid secretion. Glucocorticoids may be useful in reducing the severity of later-phase anaphylactic reactions and are commonly used (dexamethasone 0.25 to 0.5 mg/kg IV/IM) in combination with antihistamines in patients with anaphylaxis.

Narcolepsy

One of the rare forms of collapse and loss of consciousness is narcolepsy. Animals with narcolepsy will collapse spontaneously, usually while in the process of a normal activity, then wake up and immediately resume its normal activity. Having a client video an episode

and keep a diary can potentially help make a diagnosis of narcolepsy. While there is no cure for narcolepsy, some drugs to treat hyperactivity or tricyclic antidepressants may decrease the frequency of episodes.

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