# Downward Dog: Now What? A Criticalists Perspective

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When presented with a “collapsed” patient, the interest from a criticalist typically perks up. If the patient is subsequently shown to be an alert Dachshund clambering along with her front legs or a crazy Labrador with a previous stifle scar, our interest quickly wanes. When a stretcher is required to transport a patient into hospital for examination, the sound of the wonky wheels of a stretcher rapidly rolling on laminate flooring gets our attention once again.

As seen from the box below left, the causes for collapse in a dog are extensive. This list of differential diagnoses is clearly not exhaustive, however demonstrates that an abnormality in any body system could result in a collapsed patient.

| Causes of “Collapse” in the Dog | | |
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| Hypovolemia | Hypoglycemia | Seizures |
| Hemorrhage | Diabetic ketoacidosis | Vestibular events |
| Sepsis | Pheochromocytoma | Forebrain disease |
| Anaphylasix | Hyper/hypokalemia | Brainstem disease |
| Arrhythmia | Hyper/**hypo**calemia | Spinal cord disease |
| Syncope | Airway obstruction | Neuromuscular disease |
| Pericardial effusion | Pleural space disease | Polyarthritis |
| Myocardial failure | Pulmonary thromboembolism | Bilateral cranial cruciate disease |
| Cardiac valvular stenosis | Pulmonary hypertension | Drug or toxin induced |
| Hypoadrenocorticism | Acute pancreatitis | |

Three common causes of a collapsed dog presented for emergency and critical care to be discussed include spontaneous hemoabdomen, pericardial effusion, and the Addisonian crisis.

## Spontaneous Hemoabdomen

The cause of collapse due to a hemoabdomen is from hypovolemic shock, specifically from hemorrhage. Spontaneous hemoabdomen is defined as the accumulation of free blood within the abdominal cavity without known trauma as the predisposing cause. The majority of canine nontraumatic hemoabdomen is due to underlying neoplastic disease. The organ most affected is the spleen followed by the liver as the second most common source of bleeding. Geriatric, large breed dogs tend to be most affected with German shepherd dogs, Labrador retrievers, and golden retrievers found to be at higher risk than the general population for developing hemangiosarcoma and splenic hematoma.

Due to the disparate prognosis for patients with hemangiosarcoma (and other highly malignant tumors) versus most other causes of spontaneous hemoabdomen, full staging with thoracic radiographs, abdominal ultrasound, and echocardiography should be considered if time, based on patient stability, permits. The occurrence of concurrent splenic hemangiosarcoma and cardiac mass has been reported to range from 9 – 25%. Golden retrievers are reported to be much more likely (10 X) to present with splenic hemangiosarcoma and a cardiac mass. With unstable patients or those presenting after hours, minimal staging should including thoracic radiographs as the lungs are a common site of metastasis. Standard baseline diagnostic tests including complete blood count, serum biochemistry profile, blood gas analysis, and coagulation profile (prothrombin time and activated partial thromboplastin time) may give the clinician a sense to the magnitude of peri- and post-operative support required

Patient stabilization prior to surgical intervention should be aimed at hypotensive resuscitation. The goal of hypotensive resuscitation is to administer fluid volumes to target a mean arterial pressure of 60 – 70 mm Hg. At these blood pressures, tissue perfusion should be persevered while preventing clot disruption. Hypotensive resuscitation is implemented in people with uncontrolled cavitary hemorrhage, however surgical stabilization is expected within an hour of first response. This short transition time to the operating room is difficult to achieve in veterinary medicine. Prolonged hypotensive resuscitation requires vigilant monitoring so as to avoid the complications of poor tissue perfusion and organ dysfunction.

As the majority of patients with spontaneous hemoabdomen are large breed dogs, they may pose a high demand for blood products. Autologous blood transfusions (autotransfusion) may be the only reasonable method of supporting these patients. Autotransfusion of blood from ruptured neoplastic tissue is thought to be a relative contraindication, where the risk of autologous transfusion must be weighed against the risks of homologous transfusion or no transfusion. A canine study has reported the short term effectiveness, safety, and simplicity of

autotransfusion of blood collected from ruptured masses, however long term survival studies are lacking. Current data from the human literature suggest that autologous blood that is processed and transfused through a leukoreduction filter reduces bacterial load and virtually eliminates white blood cells and malignant cells.

## Pericardial Effusion

The cause of collapse in patients with pericardial effusion is cardiac tamponade causing decreased right ventricular filling. Cardiac tamponade is a form of cardiogenic shock as obstructed venous return results in reduced cardiac output. Effusions are commonly (85%) hemorrhagic in nature with differential diagnoses to include neoplasia (hemangiosarcoma, heart-based tumors, pericardial mesothelioma), idiopathic pericardial effusion, coagulopathy, and atrial tears. Hemangiosarcoma is the most common primary cardiac tumor in dogs and usually arises within the right side of the heart. Older dogs of many breeds can be affected, however the German shepherd dog, golden retriever, Afghan hound, English setter, American cocker spaniel, Doberman pinscher, Labrador retriever, and miniature poodle appear to have an increased prevalence. Seventy-five percent of dogs with cardiac hemangiosarcoma have evidence of metastatic disease with the most common site of metastasis being the lungs. Idiopathic pericardial effusion is found most often in medium to large dog breeds and the Saint Bernard and Labrador retriever may be predisposed. Transudative effusions are much less common and may be due to congestive heart failure (especially in cats), peritoneopericardial diaphragmatic hernia, uremia, and lymphoma. Exudative pericardial effusions are rare and usually caused by infectious agents.

Patients with pericardial effusion can present with a range of clinical signs from mild to life threatening depending of the rate of fluid accumulation and distensibility of the pericardial sac. Pericardial scarring and thickening can add an element of constrictive pericarditis. This variability in clinical presentation can be found among patients with any volume of pericardial effusion. To help clarify how some patients with moderate to severe pericardial effusion can present without any clinical signs of tamponade, despite echocardiographic evidence of cardiac chamber collapse; tamponade should be considered a continuum rather than an “all or nothing” phenomenon. The severity of hemodynamic compromise cause by pericardial effusion progresses from evidence of hemodynamic tamponade to echocardiographic tamponade culminating in clinical tamponade at its most severe form. Hemodynamic tamponade occurs when the intrapericardial pressure rises to equal the right atrial pressure. The circulation adapts to the increasing intrapericardial pressure by increasing central systemic and pulmonary venous pressures to equal the pericardial pressure, thereby preventing complete collapse of the cardiac chambers. Echocardiographic tamponade is defined as the collapse of either the right atrium and/or ventricle during the diastolic phase of the cardiac cycle. Clinical tamponade is easily

recognized in debilitated patients with dyspnea, tachycardia, jugular venous distension, pulsus paradoxus, and hypotension.

Echocardiography remains the most sensitive and non-invasive method for detecting pericardial effusion. Additionally, echocardiography can help to differentiate between patients with compensated effusions from those with tamponade physiology and may also be able to establish an etiology. Echocardiography has been found to be highly specific when a cardiac mass has been identified, however serial evaluations are recommended when the initial examination fails to detect an underlying problem. With the exception of infective pericarditis (which is rare), pericardial fluid cytology is infrequently able to provide a diagnosis. However, if an obvious mass has not been identified by echocardiography, fluid analysis may be helpful in determining further diagnostic and therapeutic steps. It has been suggested that elevated plasma cardiac troponin I (cTnI) may be able to distinguish pericardial effusion due to hemangiosarcoma from all other causes of effusion. This finding can be helpful as small cardiac masses can sometimes be difficult to identify during echocardiography. In dogs with pericardial effusion, elevation of cTnI > 0.25 ng/mL is likely to be due to cardiac hemangiosarcoma. This result will require careful interpretation in dogs with concurrent heart disease as identified on the echocardiogram. If cTnI concentrations are being considered, it is recommended to collect the blood sample prior to pericardiocentesis to avoid confounding the results through inadvertent cardiac trauma.

The collapsed patient with pericardial effusion is likely to have clinical tamponade and immediate pericardiocentesis is indicated. Electrocardiographic monitoring is recommended during the procedure as ventricular arrhythmias may occur when the needle or catheter contacts the heart. Pericardiocentesis is typically performed from the right to minimize the risk of trauma to the lungs and coronary vessels. Sedation is required for most patients especially those who are uncooperative. Complications of pericardiocentesis to inform clients about include the development of cardiac arrhythmia, cardiac puncture, lung laceration, and pneumothorax.

## Adrenal Crisis

The cause of collapse in the Addisonian patient is from severe hypovolemic shock. Addison's disease is truly a great pretender as patients often present with a constellation of nonspecific signs and symptoms. Although a typical signalment of a young to middle aged dog of certain breed (ie. poodles, Portuguese water dogs, bearded collies, west highland white terriers, Nova Scotia duck tolling retrievers, great danes) could heighten your index of suspicion; hypoadrenocorticism should remain on a differential diagnosis list of many critically ill patients.

Collapse is more often found in primary adrenal insufficiency (PAI) involving both mineralocorticoid and glucocorticoid deficiencies. Primary immune-mediated destruction of the adrenal cortex is thought to be the predominant cause of PAI and approximately 85 – 90% of

the glands are destroyed before clinical signs are obvious. Severe physiologic stress can rapidly deplete the limited cortisol reserve of an Addisonian patient leading to a crisis. Precipitating factors may include grooming, boarding, surgery, strenuous activity, or changes in the animals household.

Aldosterone is the most important mineralocorticoid produced in the zona glomerulosa of the adrenal cortex. It is responsible for maintaining sodium, potassium, and body water homeostasis. The primary site of aldosterone action is the principle cells of the cortical collecting ducts in the kidney, with additional target organs being the intestinal mucosa, salivary glands, and sweat glands. The two most important stimuli for aldosterone secretion are angiotensin II and an increased potassium concentration in the extracellular fluid. When secreted, aldosterone's effect will be to increase sodium and chloride reabsorption and enhance potassium and hydrogen excretion. Water absorption and thus expansion of the vascular volume occurs secondarily to the retention of sodium. An aldosterone deficiency will then cause renal sodium wasting which in turn severely reduces the plasma volume resulting in hypovolemia. A failure to maintain body water balance is compounded by the loss of urinary concentrating ability with Addisonian patients commonly presenting with isosthenuria in the face of a severe dehydration. Sodium is an important component of the renal medullary concentration gradient and chronic urinary sodium loss causes renal medullary washout. Although cortisol also is important for the maintenance of water balance, vascular volume, and blood pressure, adrenal crisis and collapse appears to occur less commonly in patients with atypical primary adrenal insufficiency and secondary hypoadrenocorticism. In these patients, aldosterone production is generally preserved.

The gold standard for diagnosis of hypoadrenocorticism remains the adrenal corticotropin hormone (ACTH) stimulation test. An ACTH stimulation test should be completed not only for those patients with classic electrolyte disturbances (hyponatremia, hyperkalemia, hypochloremia, Na:K ratio < 27), but also for patients presenting with the following scenarios:

- normal heart rate or bradycardia in the face of hypovolemic shock
- hypotension and shock refractory to fluid resuscitation and vasopressors
- waxing and waning course of disease
- previous response to empiric corticosteroids or fluid therapy
- absence of a stress leukogram during critical illness

Be aware that the ACTH stimulation test may be affected by previous administration of prednisone, prednisolone, and hydrocortisone for up to 2 months. Random serum cortisol has been proposed as a useful tool to exclude hypoadrenocorticism carrying a high negative predictive value. Patients with random cortisol levels > 55 nmol/L are unlikely to have hypoadrenocorticism.

The treatment of an Addisonian crisis is beyond the scope of this lecture, however generally should be targeted at fluid therapy and electrolyte stabilization, correction of hypoglycemia, glucocorticoid replacement therapy, treatment of gastrointestinal symptoms and hemorrhage if present, and mineralocorticoid replacement if required. Although uncommon, central pontine myelinolysis can occur with rapid correction of hyponatremia and serum sodium should not be increased by more than 0.5 mEq/L/h in severely hyponatremic patients.

References available upon request.