Life threatening conditions - cardiogenic pulmonary edema, cardiogenic shock, ventricular underfilling (pericardial tamponade), hemodynamically unstable arrhythmias, and arterial thromboembolism - require immediate interventions.

**Acute pulmonary edema (Congestive Heart Failure)**

In dogs CHF results most commonly from volume overload caused by chronic degenerative valvular disease (severe mitral regurgitation) or dilated cardiomyopathy. In cats diastolic heart failure associated with hypertrophic or restrictive cardiomyopathy is the predominant underlying condition. Less common etiologies include aortic insufficiency, left-to-right shunting (PDA, arteriovenous fistula), and high output states (thyrotoxicosis). Treatment requires aggressive measures to resolve the congestive state and improve cardiopulmonary function. Furosemide is given as IV boluses (2-4mg/kg q 30-60min) or by constant rate infusion. Vasoactive drugs are added to promote venodilation and/or arterial dilation. Typically, this may include nitroglycerin ointment for mild to moderate edema. In states of life threatening edema in the dog, the potent vasodilator sodium nitroprusside is administered by CRI (2-20ug/kg/min with constant arterial blood pressure monitoring). Alternatively, hydralazine, a potent arteriolar dilator, can be given (2mg/kg PO bid) when pulmonary edema results from mitral regurgitation. Inotropic support using dobutamine (5-15ug/kg/min constant rate infusion) is indicated when severe myocardial failure or cardiogenic shock is present (e.g., dilated cardiomyopathy). The potential role of pimobendan in this circumstance has not been clarified but may provide benefit as well. Digoxin is often considered (dog- 0.005-0.01mg/kg lean body weight q 12 hrs; cat- ¼ of 0.125mg tablet q 24-48 hrs), especially when right-sided heart failure or atrial fibrillation is present. Antiarrhythmic therapy is administered when needed to suppress or abolish ventricular tachyarrhythmias, or to control ventricular rates with supraventricular tachyarrhythmias such as atrial fibrillation. Supplemental O\textsubscript{2} administration is provided. Mechanical removal of effusion is performed if necessary. ACE inhibitors and pimobendan are included in chronic management strategies.

Reversible causes of heart failure should be treated if present. Myocardial failure has been associated with taurine and carnitine deficiency. Volume overload secondary to patent ductus arteriosus is correctable by surgical or occlusion techniques. Other systemic and metabolic disorders may cause or contribute to heart failure including endocarditis, myocarditis, pheochromocytoma, diabetes, and hyperthyroidism. Heartworm disease is a treatable cause of right-sided CHF.

With recurrent heart failure, upward drug titration may be necessary. Serum digoxin concentrations should be monitored. Diuretic resistance may occur as heart failure progresses. Some animals are likely to benefit from intravenous
Furosemide therapy which has higher bioavailability, or a second and third diuretic (e.g., thiazide, 5 to 20 mg daily, or spironolactone-12.5 to 25 mg once to twice daily). It is prudent to assess BUN, creatinine, electrolytes and blood pressure during chronic therapy.

**Cardiogenic shock**

Myocardial failure is most commonly associated with dilated cardiomyopathy. Less frequent etiologies include chronic volume overload (e.g., mitral regurgitation, left-to-right shunts) or sepsis. The principal hemodynamic feature of cardiogenic shock is systemic hypotension associated with reduced ventricular pumping (i.e., myocardial failure/systolic dysfunction). Pulmonary edema, systemic congestion, hypotension, and tissue hypoxia result. Acute management may require inotropes (dobutamine CRI), diuretics to reduce congestion, vasodilators such as sodium nitroprusside. ACEI, digoxin, pimobendan, and control of sepsis and arrhythmias.

**Ventricular underfilling**

Conditions which interfere with return of blood to the heart may result in decreased cardiac preload, compensatory neuroendocrine activation, and a clinical condition known as cardiac tamponade. This is generally associated with pericardial disease (typically neoplasia in dogs; or FIP or idiopathic effusions in cats). Less common causes include space occupying atrial or ventricular masses including blood clots or tumors. Initial management requires therapeutic pericardiocentes. Avoid using drugs that decrease preload or cause vasodilation.

**Hemodynamically unstable arrhythmias**

Tachyarrhythmias may depress cardiac output, cause hemodynamic impairment or hypotension, and result in organ ischemia. Shortened diastolic filling decreases coronary blood flow, reduces myocardial oxygen supply, causes ischemia and results in more serious arrhythmias. Certain tachyarrhythmias may deteriorate by becoming electrically unstable. Hemodynamic impact of tachyarrhythmias are influenced by factors related to underlying cardiac disease and the particular type of arrhythmia (i.e., (a) loss of synchronized atrial systole, (b) altered ventricular activation sequence, (c) rapidity of ventricular rate, (d) timing of ectopic beats relative to preceding P-QRS-T complexes, (e) background vasomotor tone, (f) cardiac effects of antiarrhythmic drugs, and (g) underlying cardiac dysfunction or health). Because cardiac output = heart rate x stroke volume, sustained tachycardia may reduce cardiac output and arterial blood pressure. In atrial fibrillation with rapid ventricular response, ventricular filling shortens due to loss of atrial contraction, variation in cycle length and high ventricular rate. This is worsened by concurrent myocardial dysfunction (e.g., dilated cardiomyopathy) or exercise. Impulses originating in the ventricle (e.g., ventricular tachycardia) alter patterns of electrical activation and reduce stroke volume. Rapid, sustained ventricular tachycardia decreases cardiac output, results in hypotension and organ ischemia. Ventricular flutter causes precipitous deterioration and all circulation ceases with ventricular fibrillation.
Paroxysms of atrial tach with normal ventricular activation may not cause clinical consequences; multifocal atrial or ventricular tachycardia are more likely to compromise hemodynamics, especially if ventricular function is abnormal. Electrical instability is increased by rapid ventricular rates and multifocal impulse origination. Additional factors include timing of the ectopic impulse (i.e., the earlier the premature complex relative to the preceding T wave, the greater electrical liability). Depolarizations occurring within the preceding T wave are extremely dangerous. The underlying state of ventricular function, systemic and metabolic alterations, and concurrent drug or anesthetic agents influence electrical stability. Electrical instability is increased by rapid ventricular rates and multifocal impulse origination. Additional factors include timing of the ectopic impulse (i.e., the earlier the premature complex relative to the preceding T wave, the greater electrical liability). Depolarizations occurring within the preceding T wave are dangerous. The underlying ventricular function, systemic and metabolic alterations, and concurrent drug or anesthetic agents influence electrical stability. Tachycardia = ventricular rate >240bpm in cats; > 180bpm in small breed dogs; > 160bpm in large breeds, and >220bpm in puppies. With supraventricular tachycardias, vagal maneuvers may occasionally convert the arrhythmia. Supraventricular arrhythmias may be treated with digitalis glycosides, calcium channel blockers, beta blockers, and other agents. Acute management of ventricular tachycardia includes treatment of the underlying cause and lidocaine. Pacemaker implantation may be required to treat high grade AV block.

**Evaluating the critical patient**

Assessment of the unstable patient is aided by a careful history, complete general examination, and complete data base.

**Noninvasive Monitoring of Hypoxemia (Pulse Oximetry)** The saturation of hemoglobin with oxygen in arterial blood (SaO2) is a useful indicator of hypoxemia. Pulse oximetry is a noninvasive technique to allow continuous monitoring of arterial oxyhemoglobin saturation. Blood contains 4 species of hemoglobin (Hb): 1) oxyhemoglobin (HbO2), 2) reduced Hb, 3) methemoglobin (MetHb), and 4) carboxyhemoglobin (COHb). In healthy individuals, the latter 2 are in small concentration. Pulse oximetry measures functional hemoglobin saturation [SaO2= HbO2 divided by HbO2+Hb x 100], and thereby assesses arterial oxygenation. It does not assess ventilation (CO2 elimination). Hypoxemia may be a late onset sign of deterioration in some cases of respiratory failure, especially when compensatory tachypnea has maintained normal oxygen levels. Accurate pulse oximeter readings are not always possible in every animal owing to probe placement issues, thick or pigmented skin, movement artifact, and other factors. Thus, hemoglobin saturation determined by pulse oximetry should always be evaluated in light of the patients clinical condition. Arterial blood gas analysis should be considered whenever pulse oximetry estimation is in question.

**Noninvasive Blood Pressure Monitoring** Hypertension may predispose certain "target" organs to injury, particularly the eyes, kidneys, and cardiovascular and...
neurovascular systems. Hypotension is a common consequence of shock, dehydration, and certain drug toxicities. Systolic blood pressure >160 suggests hypertension; SBP>200 mmHg recorded on 2 occasions at least 24 hours apart indicate hypertension, unless the animal was excited. End-organ injury provides supportive evidence of hypertension. SBP <90 indicates hypotension.

Central Venous Pressure (CVP) CVP directly measures pressure in the great thoracic veins as blood returns to the right heart. Serial or continuous CVP measurement helps assess right heart filling and status of intravascular volume. Evaluation of the direction of change in CVP measurements over time is more relevant than basing diagnostic/therapeutic changes on isolated measurements. CVP generally decreases as venous return decreases. Low CVP suggests hypovolemia. Elevated CVP measurements suggest either right ventricular failure or intravascular volume overload.

Electrocardiography Assessment of heart rate and rhythm provides information about cardiac chamber enlargement, implies the presence of severe pericardial or pleural effusion, and can help assess certain suspected systemic and metabolic disorders (e.g., marked disturbances of potassium or calcium, ischemia, infarction). Continuous ECG monitoring, event recorders, or Holter recordings are useful to detect transient arrhythmias.

Radiography The radiograph 1) confirms disease suspected from the history and physical examination, 2) assesses disease severity, 3) distinguishes between cardiac and respiratory disease, 4) confirms tube/catheter placement, 5) screens for unsuspected conditions, 6) discovers complications, and 7) helps monitor (from repeated studies) response to therapy.

Echocardiography Diagnostic ultrasound assists cardiac examination when the heart is obscured by pleural effusion; diagnoses pericardial effusion; provides quantitative assessment of cardiac structure (valves; chamber dimensions, wall thickness); assesses systolic (contractile) and diastolic function; quantifies gradients via Doppler echocardiography; detects disturbances of blood flow; detects intracavitary masses (clots, tumors); and helps characterize congenital and acquired heart diseases.