Pulmonary hypertension is a complex phenomenon that develops secondary to many common disease processes that occur in small animals. However, pulmonary hypertension is often underdiagnosed in practice because of the varied clinical presentations and requirement for specialized diagnostic equipment such as echocardiography or pulmonary arterial catheterization. The diagnosis is usually made late in the course of disease and carries a poor prognosis when a reversible underlying cause is not found and corrected. It is important for veterinarians to recognize the causes of pulmonary hypertension and maintain an appropriate index of suspicion when these patients present to their hospital. Treatment for pulmonary hypertension is complex but is most rewarding if the underlying cause is found and appropriate treatment is initiated early.

The normal mean pulmonary arterial pressure ranges from 10 to 15 mm Hg in dogs. Pulmonary hypertension is defined as a mean pulmonary arterial pressure greater than 30 mm Hg. Systolic pulmonary arterial pressure ranges from 18 to 25 mm Hg, and normal diastolic pulmonary arterial pressure is 6 to 10 mm Hg. The normal pulmonary circulation is a low-pressure, low-resistance, high-capacitance vascular bed. The low resistance of the pulmonary circulation can be illustrated by the fact that the arteriovenous pressure difference is only 2 to 10 mm Hg (90 mm Hg in the systemic circulation). The pressure within the pulmonary circulation depends on the pulmonary venous pressure, right ventricular output, and pulmonary arterial resistance. Pulmonary arterial resistance is affected by the cross-sectional area of small vessels, total mass of the lung tissues, proximal vascular obstruction, extramural compression of small vessels, and blood viscosity. Vascular changes associated with pulmonary hypertension are vasoconstriction, smooth muscle and endothelial cell proliferation, and thrombosis. Morphologic changes that occur in the pulmonary vasculature include medial hypertrophy, intimal proliferation, and sclerosis of the small- to medium-sized arteries.

Many endothelium-derived mediators control pulmonary vascular tone (Table 1). These include prostacyclin, thromboxane A2, endothelin, nitric oxide, serotonin, and vasoactive intestinal peptide. Prostacyclin, nitric oxide, and vasoactive intestinal peptide cause vasodilation and have antiproliferative and antiplatelet effects. Endothelin may also contribute to vasodilation. Thromboxane A2, endothelin, and serotonin cause vasoconstriction and smooth muscle and endothelial proliferation and are platelet agonists. In pulmonary hypertension, there is an imbalance in these mediators, and many of the drugs used to treat pulmonary hypertension are directed toward these mediators.

The World Health Organization classifies human pulmonary hypertension into five groups based on the inciting cause (see box on page 7).

Pulmonary hypertension causes pressure overload on the right heart and can result in right ventricular enlargement (concentric hypertrophy or thickening). This is called cor pulmonale. Acute cor pulmonale, most often caused by massive thromboembolic disease, results in right ventricular dilation, tricuspid regurgitation, and signs of right-sided heart failure. The normal right ventricle cannot acutely generate a systolic pressure greater that 50 mm Hg while maintaining an adequate heart rate, and right ventricular failure may precede severe pulmonary hypertension in these cases. In chronic cor pulmonale, such as that caused by respiratory disease or left-to-right shunts, higher pulmonary arterial pressures are often seen. Pulmonary hypertension can be categorized as mild (right ventricular systolic pressure, 30–55 mm Hg), moderate (56–70 mm Hg), and severe (>70 mm Hg). Pulmonary hypertension associated with left-sided valvular or ventricular disease is often mild or moderate, but pulmonary hypertension associated with thromboembolic disease or chronic respiratory disease can be severe.

### Diagnostic Criteria

#### Historical Information

**Gender/Age/Breed Predisposition**
- Terrier breeds (e.g., West Highland white terriers) are predisposed to primary pulmonary disease (e.g., pulmonary fibrosis), which can predispose them to pulmonary hypertension.
- In one report of six dogs with idiopathic pulmonary hypertension, intact middle-aged female dogs were...
overrepresented.

• Pulmonary hypertension may be seen in any sex, age, or breed of dog or cat depending on the inciting cause.

**Owner Observations**

• If the animal is normally inactive or the owner is not observant, the patient may show no overt clinical signs with mild, moderate, or even severe pulmonary hypertension.

• Dyspnea or tachypnea.

• Lethargy.

• Exercise intolerance.

• Cough.

• Ascites if right-sided heart failure is present.

• Syncope.

**Other Historical Considerations/Predispositions**

• Dogs with mild to moderate pulmonary hypertension often do not show any overt clinical signs.

• The clinical course can range from acute (as in cases of massive, acute pulmonary thromboembolism) with signs of right-sided heart failure to chronic (e.g., secondary to chronic interstitial lung disease) with progressive lethargy, tachypnea, exercise intolerance, and syncope.

• Clinical signs depend on the inciting disease process. For example, if the animal has pulmonary hypertension because of chronic respiratory disease (chronic interstitial lung disease, bronchitis) harsh lung sounds, pulmonary crackles, cyanotic mucous membranes, and coughing may be seen. If the patient's pulmonary hypertension is attributed to thromboembolic disease, lung sounds may be normal, but the patient may present in respiratory distress.

• Dogs with heartworm disease are particularly at risk for pulmonary thromboembolism and pulmonary hypertension 7 to 14 days after adulticide therapy but should be strictly cage confined for 6 weeks after treatment.

**Physical Examination Findings**

• Dyspnea or tachypnea.

• Cyanosis.

• Cardiac murmur: May be tricuspid, mitral, aortic, or pulmonic systolic. A split $S_2$ sound (“lub-dudub” instead of “lub-dub”) may also be heard and is the classic hallmark of delayed pulmonic valve closure associated with moderate or severe pulmonary hypertension of any cause.

• Ascites or jugular distension if right-sided heart failure is present.

• Harsh lung sounds, crackles, or rales.

• Tachycardia or bradycardia.

• Cough.

• May be normal.

**Laboratory Findings**

No specific laboratory findings are associated with pulmonary hypertension.

**Complete Blood Count $**

• Stress leukogram (neutrophilia, lymphopenia, monocytosis) or nucleated red blood cells as an indicator of hypoxia.

• Eosinophilia or basophilia if heartworm disease is present.

• An increase in packed cell volume in patients with reverse cardiac shunts (right-to-left shunting patent ductus arteriosus, atrial septal defects, and ventricular septal defects).

**Chemistry Panel $**

No specific abnormalities.

**Coagulation Panel $**

• If pulmonary hypertension is secondary to thrombosis caused by hypercoagulability, elevated fibrin degradation products (FDPs) or D-dimers may be seen, although these values may also be elevated in other disease processes.
• Antithrombin III levels may be decreased in patients with nephrotic syndrome.

**Arterial Blood Gas Analysis**
- Decreased partial pressure of oxygen (PaO₂) and elevated partial pressure of carbon dioxide (PaCO₂), especially with chronic respiratory disease.
- With pulmonary thromboembolism, PaO₂ is decreased and PaCO₂ is normal to decreased secondary to tachypnea.

**Pulse Oximetry**
- May be decreased (<96%) in patients with pulmonary hypertension, especially if caused by primary lung disease, indicating abnormal gas exchange.
- Caution should be exercised because small decreases in hemoglobin saturation indicate large decreases in arterial oxygen concentration.

**Heartworm Antigen Test (Dogs)**

**Other Diagnostic Findings**

**Thoracic Radiography**
- May be normal, such as in cases of acute pulmonary thromboembolism, despite clinical signs of respiratory distress.
- Cardiomegaly, interstitial or alveolar lung pattern, enlarged pulmonary arteries, pulmonary edema, pleural effusion, enlarged caudal vena cava.
- Cardiomegaly associated with pulmonary hypertension is typically right sided; less common is the finding of left- and right-sided (generalized) cardiomegaly, which occurs when pulmonary hypertension is caused by chronic mitral insufficiency, for example.
- Thoracic radiographs are nonspecific for diagnosing pulmonary hypertension.

**Electrocardiography (Dogs)**
- Right axis deviation or right bundle branch block only if substantial right-sided heart enlargement is present.

**Cardiac Catheterization**
- This is the “gold standard” for the diagnosis of pulmonary hypertension.
- It is rarely performed in veterinary medicine because of the cost, requirement of general anesthesia, and lack of technical experience.
- This procedure is used to diagnose pulmonary hypertension in human medicine and monitor the effect of short-acting intravenous vasodilator therapy. Improvement in pulmonary arterial pressure is seen as an indication to begin long-term vasodilator therapy.

---

**WORLD HEALTH ORGANIZATION CLASSIFICATION OF PULMONARY HYPERTENSION IN HUMANS**

**Group I: Pulmonary Arterial Hypertension**
- Idiopathic (primary pulmonary hypertension)
- Familial pulmonary hypertension
- Congenital systemic to pulmonary shunts (PDA, VSD, ASD)
- Persistent pulmonary hypertension of the newborn

**Group II: Pulmonary Venous Hypertension**
- Left-sided atrial or ventricular heart disease and left-sided valvular disease

**Group III: Pulmonary Hypertension Associated with Chronic Alveolar Hypoxia**
- Pulmonary parenchymal disease
  - Pulmonary fibrosis
  - Chronic pneumonia
  - Tracheobronchial disease (tracheal collapse, laryngeal paralysis)
  - Pulmonary neoplastic disease
- High altitude

**Group IV: Pulmonary Hypertension Caused by Chronic Thrombotic or Embolic Disease**
- Hypercoagulability caused by systemic disease
  - Hyperadrenocorticism
  - IMHA
  - Nephrotic syndrome
  - Pancreatitis
  - Polycythemia
- Heartworm disease

**Group V: Miscellaneous Causes of Pulmonary Hypertension**
- Disorders of the pulmonary vasculature
  - Sarcoïdosis
  - Lymphangiomyomatosis
  - Pulmonary Langerhans’cell histiocytosis

- A Swan-Ganz or multipurpose single end-hole catheter is used.
- The procedure can measure central venous pressure, oxygen saturation, pulmonary arterial pressure, and pulmonary arterial oxygen saturation; with a Swan-Ganz catheter, it can also measure pulmonary capillary wedge pressure and cardiac output.

**Echocardiography**
- This is a noninvasive method for estimating pulmonary arterial pressure.
- Right ventricular free wall and interventricular sep-
tum thickening (concentric hypertrophy) secondary to pressure overload of the right heart may be seen. Dilation of the main pulmonary artery may also be seen. These two-dimensional findings generally occur only with moderate or severe pulmonary hypertension and are nonspecific.

- Paradoxic motion of the interventricular septum: Increased pressure on the right ventricle during diastole displaces the interventricular septum to the left, making it appear flattened.

- If tricuspid or pulmonic insufficiency is present, an estimate of the right ventricular systolic pressure can be made by measuring the peak velocity of the regurgitant jet and calculating the pressure gradient across the right ventricle using the modified Bernoulli equation and adding the right atrial or central venous pressure. For example:

  \[ \text{Pressure gradient (PG)} = 4V^2 \]
  \[ \text{Tricuspid regurgitant velocity} = 4.2 \text{ m/s} \]
  \[ \text{PG} = 4(4.2)^2 = 70.5 \]
  \[ \text{Normal right atrial pressure} = 5 \text{ mm Hg} \]
  \[ \text{Pulmonary arterial systolic pressure} = 75.5 \text{ mm Hg} \]

- Tricuspid or pulmonic regurgitant velocity above 2.5 m/s indicates pulmonary hypertension because the right ventricular systolic pressure (pulmonary arterial pressure) should normally not exceed 30 mm Hg. (A tricuspid regurgitant jet above 2.5 mm Hg suggests [but has never been proven to equal] pulmonary hypertension. Many cardiologists use 3 m/s as a cutoff. Pulmonic insufficiency above 1.8 m/s suggests pulmonary hypertension.)

- When pulmonic stenosis or right ventricular outflow tract (RVOT) obstruction is not present, right ventricular systolic pressure estimates systolic pulmonary arterial pressure. When one of these other two disorders is present, this technique is not valid for determining pulmonary arterial pressure.

- Pulmonary artery flow profiles and systolic time intervals may change with pulmonary hypertension.

Summary of Diagnostic Criteria

- History of lethargy, exercise intolerance, tachypnea, or syncope.
- Presence of one of the possible primary disease processes that can cause pulmonary hypertension.
- Evidence of cardiac or pulmonary disease present on thoracic radiographs.
- Echocardiographic high-velocity tricuspid regurgitant jet (>2.5 m/s) with or without evidence of right ventricular enlargement.

Diagnostic Differentials

RVOT Obstruction

- Pulmonic stenosis causes pressure overload on the right heart and results in right ventricular concentric hypertrophy and possibly high-velocity tricuspid regurgitation.
- Echocardiography should be performed to rule out RVOT obstruction before a diagnosis of pulmonary hypertension can be made.

Tricuspid Dysplasia or Endocardiosis

- Tricuspid dysplasia or endocardiosis often causes tricuspid regurgitation, but the velocity should not be as high as in cases of pulmonary hypertension. Echocardiography is indicated to differentiate between regurgitation caused by pulmonary hypertension and tricuspid dysplasia.

Dilated Cardiomyopathy

- Pulmonary hypertension can occur in dogs with dilated cardiomyopathy. Thoracic radiography and echocardiography are indicated for diagnosis.

TREATMENT RECOMMENDATIONS

Initial Treatment

- Prevention of pulmonary hypertension is best. Early recognition of conditions that can lead to pulmonary hypertension and treatment of the primary cause can prevent or potentially reverse pulmonary hypertension.

Patent Ductus Arteriosus

- Surgical ligation or occlusion of the ductus if a left-to-right shunt is present. After a right-to-left shunt has developed because of elevated pulmonary arterial pressures, ductal closure can cause acute right heart failure and is not recommended.

Pulmonary Venous Hypertension

Treatment of heart failure:

- Diuretics
  - Furosemide: 2–4 mg/kg PO q12h.
  - Spironolactone (aldosterone antagonist): 1–2
mg/kg PO q12h as an addition if long-term furosemide is insufficient.

— Diuretics should be used with caution in patients with chronic lung disease because they can dehydrate bronchial mucosa and may lead to mucosal plugging of the airways and worsen gas exchange.

• Angiotensin-converting enzyme (ACE) inhibitors to reduce afterload and blunt the renin–angiotensin–aldosterone system. (ACE inhibitors are generally ineffective for affecting pulmonary arterial pressures.)
  — Enalapril: 0.5 mg/kg PO q12–24h.
  — Benazepril: 0.5 mg/kg PO q12–24h.

• Digoxin (if atrial fibrillation or other supraventricular tachycardia is present): 0.003–0.005 mg/kg PO q12h if the patient weighs less than 40 lb. If the patient weighs more than 40 lb, 0.25 mg/m² PO q12h should be given. Serum digoxin levels should be measured after 5 to 7 days (6 to 8 hours after administration). Therapeutic serum digoxin levels have not been established for dogs. Traditionally, 0.5 to 2.0 ng/ml has been used as the reference range. The patient should be monitored for signs of digoxin toxicity such as vomiting, diarrhea, anorexia, and other signs of gastrointestinal upset and for cardiac arrhythmias.

• Pimobendan, a positive inotrope and vasodilator (see discussion in later section): 0.15–0.3 mg/kg PO q12h administered 1 hour before feeding or 2 hours after feeding because food impairs absorption.

Thromboembolic Disease
Immune-mediated hemolytic anemia, hyperadrenocorticism, nephrotic syndrome, and so on: The primary goal is to treat the inciting cause. The secondary goal is to provide anticoagulant or antiplatelet therapy to prevent thrombus formation. In humans, pulmonary hypertension causes an increased risk of pulmonary thromboembolism, and preventative anticoagulants are given in all cases. Their role in veterinary medicine is unclear.

• Heparin
  — In humans, low-molecular-weight heparins (LMWHs) are used more commonly than unfractionated heparins because of the lower incidence of bleeding complications and less frequent dosing with LMWHs. The role of LMWH over unfractionated heparin is still being defined in veterinary medicine. The goal of heparin therapy is to achieve a partial thromboplastin time of 1.5 to 2 times normal.
  — Enoxaparin: 1 mg/kg (100 U/kg) SC q12–24h.
  — Dalteparin: 100 IU/kg SC q24h.
• Low-dose aspirin: 0.5 mg/kg PO q12h.

Heartworm Disease
• Monthly heartworm prevention should be given.
• Melarsomine can be used to treat heartworm disease. This is an adulticide. *Melarsomine should not be used in cats.*
  — Class I to III heartworm disease: Dogs should be staged before treatment. Staging generally includes a complete blood count, chemistry panel, urinalysis, thoracic radiographs, and echocardiography. A three-injection protocol is recommended for dogs exhibiting clinical signs or with radiographic changes, although it is widely believed that this protocol should be used for all dogs with heartworm disease.
  — Three-injection protocol: 2.5 mg/kg deep IM (lumbar epaxial muscles L3–5). Strict cage rest for 1 month should be prescribed. Then 2.5 mg/kg deep IM should be given twice 24 hours apart. Strict cage rest for 4 to 6 weeks should then be followed.
  — Controversy exists over the use of doxycycline to decrease the inflammatory response associated with *Dirofilaria* spp. If used, the dosage is 5 mg/kg PO q12h.
  — In addition to acute pulmonary thromboembolism, pulmonary hypertension, and right heart failure, side effects of melarsomine may include injection-site reactions, coughing, depression or lethargy, anorexia, fever, and vomiting.
  — Corticosteroids may be used to decrease inflammation associated with the worms in the pulmonary artery, if necessary. The dosage of prednisone is 0.5–1 mg/kg PO q24h.

Chronic Interstitial Lung Disease, Chronic Obstructive Pulmonary Disease, Chronic Pneumonia, and Tracheobronchial Disease
• Oxygen therapy, antibiotics (if secondary infection is present based on positive culture and sensitivity), nebulization and coupage (if indicated), corticosteroids to reduce sterile inflammation (if appropriate), bronchodilators (e.g., theophylline).
• Theophylline causes trivial vasodilation of the pulmonary arteries through nonspecific phosphodiesterase inhibition, leading to a clinically equivocal reduction in mean pulmonary artery pressure. In humans, theophylline produces sustained pulmonary vasodilation and improvement in right ventricular function in patients with chronic obstructive pulmonary disease and pulmonary hypertension.

Other Conditions
• Other conditions, such as pancreatitis, neoplasia, hyperadrenocorticism, sepsis, and hypothyroidism.
that may lead to a hypercoagulable state: Treatment of the underlying disease, cage rest, and low-dose aspirin are recommended as indicated.

Alternative/Optional Treatments/Therapy

Calcium Channel Blockers

- Calcium channel blockers (CCBs) are used in human medicine but are effective in less than 10% of human patients. Doses are much higher than used in standard treatment of heart failure. They are not thought to be clinically useful in veterinary patients. CCBs also have a negative inotropic effect and can cause systemic hypotension.
  - Amlodipine.
  - +/- Diltiazem.
  - +/- Nifedipine.

Phosphodiesterase V Inhibitors

- Sildenafil inhibits phosphodiesterase V–mediated breakdown of cyclic guanosine monophosphate (cGMP) and enhances nitric oxide–dependent, cGMP-mediated pulmonary vasodilation. It increases exercise tolerance and quality of life in humans with pulmonary hypertension. One retrospective evaluation in dogs revealed an 11% to 12% decrease in mean pulmonary artery pressure. Systemic hypotension was observed. Sildenafil may be cost prohibitive in large dogs. Compounding may be required for smaller dogs. Dosage: 1.9 mg/kg sid–bid.
  - Tadalafil (Cialis) is a new, longer-acting phosphodiesterase V inhibitor. One case report has been published in the veterinary literature. Pulmonary arterial pressure decreased and systemic hypotension was observed. Significant systemic hypotension was observed only when concurrent nitrate therapy was implemented. More veterinary studies are indicated in the use of phosphodiesterase V inhibitors, particularly with regard to the safe and effective dose in veterinary patients.

Pimobendan

- This drug is a positive inotrope (increases force of myocardial contraction) and systemic and pulmonary vasodilator.
  - Vasodilation is caused by phosphodiesterase III inhibition. Phosphodiesterase III breaks down both cAMP and cGMP, so inhibition increases concentrations of these vasodilators. Pimobendan may also have antiplatelet effects.
  - Pimobendan may be useful in combination with phosphodiesterase V inhibitors.
  - It is newly approved by the Food and Drug Administration for use in dogs. It has been available in Europe, Canada, and Australia for several years. It should be available in the United States in late August to early September 2007 (as a chewable formulation).
  - Other diagnostic differentials must be ruled out because they are relative (e.g., compensated tricuspid dysplasia) or absolute (pulmonic stenosis) contraindications to use of this positive inotropic drug.
  - Dosage: 0.15–0.3 mg/kg PO q12h administered 1 hour before feeding or 2 hours after feeding because food impairs absorption.

Prostacyclin Analogues

Prostacyclin is a potent pulmonary vasodilator and inhibitor of platelet aggregation and has antiproliferative properties. Studies in humans revealed an improvement in exercise tolerance, hemodynamics, and long-term survival. At this time, these are not used in veterinary medicine.

- Epoprostenol and treprostinil must be given as either a continuous IV (epoprostenol) or SC (treprostinil) infusion and are extremely expensive, thus prohibiting their use in veterinary patients. They are associated with a risk of sepsis and thrombosis caused by an indwelling catheter.
- Iloprost is a stable prostacyclin analogue delivered via an inhaler. It must be given six to 12 times a day because of its short duration of action. No long-term studies have been done, and it is not currently approved in the United States. (It is approved in Europe.) It is also expensive.
- Beraprost is the first oral prostacyclin analogue. It must be administered four times a day. It is currently involved in clinical trials. It is also expensive.

Endothelin Receptor Antagonists

Endothelin (ET) levels are increased in dogs with heartworm disease and other cardiopulmonary diseases. Endothelin receptor antagonists are not currently used in veterinary medicine.

- Bosentan is an oral ET_A and ET_B receptor antagonist associated with improved exercise tolerance, decreased pulmonary arterial pressure, and pulmonary vascular resistance in humans. It is extremely expensive (~$2,970/month). It may induce an increase in hepatic enzyme activity. There have been no reports of liver failure in humans.
- Sitaxsentan and ambrisentan are selective ET_A receptor antagonists. They are currently under investigation in human medicine.

Nitric Oxide

- This is a potent endogenous, endothelium-derived vasodilator that stimulates guanylate cyclase and increases production of cGMP, which causes vasodilation. It is inhaled and has an extremely short half-life. It is more useful as a diagnostic tool.
Supportive Treatment

- **Oxygen therapy** should be instituted in all suspected cases of severe, decompensated pulmonary hypertension, regardless of the underlying cause. Oxygen is a potent vasodilator of the pulmonary arterial bed. Alveolar hypoxia is the dominant cause of pulmonary vasoconstriction; therefore, it is critical to identify and reverse hypoxemia. In some cases, hypoxic pulmonary vasoconstriction is believed to be a compensatory response to match pulmonary perfusion to alveolar ventilation. Vasoconstriction occurs in areas with low PaO₂, which results in less deoxygenated blood returning to the systemic circulation. In the normal lung, hypoxic vasoconstriction results in only a mild increase in pulmonary arterial pressures. When pulmonary vascular changes are present, hypoxic vasoconstriction can cause a dramatic increase in pulmonary arterial pressures. Chronic alveolar hypoxia is also a stimulus for pulmonary vascular remodeling. Oxygen can be given nasally (flow-by or oxygen cage) at a flow rate of 1 L/9 kg (20 lb)/min.

- Pulmonary hypertension is a complex disorder, and there is no specific treatment that is uniformly successful. **Combination therapy**, such as a phosphodiesterase V inhibitor and pimobendan or an endothelin receptor antagonist, may be more successful by targeting different pathways, but such approaches remain speculative.

Patient Monitoring

- Arterial blood gas or arterial oxygen saturation should be monitored.
- Systemic blood pressure should be monitored after vasodilator therapy is initiated to avoid systemic hypotension, especially during up titration of drug.
- Serial echocardiography should be used to evaluate the velocity of the tricuspid regurgitant jet and pulmonary arterial pressures to measure response to therapy. Thoracic radiography should be repeated if pulmonary disease or pulmonary edema is present.
- Improvement in clinical signs, such as increased exercise tolerance or resolution of tachypnea or dyspnea, both at home and in the hospital, can be strongly supportive of a response to treatment.

Home Management

- Activity should be limited, and stressful situations should be avoided (e.g., long road trips, new pets in the household). The animal should preferably be kept indoors to avoid extreme temperatures. Owners should monitor for signs of respiratory distress.
- Dogs should undergo strict, absolute cage rest for a minimum of 4 to 6 weeks after heartworm adulticide therapy.

Treatment Contraindications

- Diuretics should be used judiciously. They are indicated when pulmonary edema is present but are contraindicated for many other pulmonary parenchymal diseases.
- β-blockers should be used with caution in patients with heart failure because they can cause a decrease in cardiac output.
- Sildenafil and other pulmonary artery vasodilators should not be given concurrently with nitrates because this results in systemic hypotension.

PROGNOSIS

**Favorable Criteria**

- Mild to moderate pulmonary hypertension.
- Prognosis ultimately depends on the primary cause and ability to reverse the factors causing pulmonary hypertension. After irreversible structural changes have occurred, especially if they are associated with overt clinical signs such as syncope or right-sided heart failure, prognosis is poor.

**RECOMMENDED READING**