"You are viewing this monthly SonoPath.com newsletter on hot sonographic pathology subjects, seen every day, owing to your relationship with a trusted clinical sonography service. This service has a working relationship with SonoPath.com, and sees value in enhancing diagnostic efficiency in veterinary medicine."

July 2011

DEMYSTIFING PULMONARY HYPERTENSION

Johanna Frank DVM DVSc
Diplomate ACVIM (Internal Medicine)
New Jersey Mobile Associates & SonoPath.com

Pulmonary hypertension is defined as an increase in pulmonary arterial pressures. Historically, this disease has been overlooked as a cause for respiratory signs but is becoming more commonly recognized with increased widespread availability of diagnostic techniques such as advanced echocardiography as well as increased clinical awareness. Therapeutic advances have also resulted in improvement in medical management.

Clinical signs of pulmonary hypertension include chronic cough, syncope, exercise intolerance and ultimately right-sided heart failure. Dogs with the above clinical signs and most specifically, chronic cough or increased respiratory effort non-responsive to traditional medical management, should be screened for pulmonary hypertension. Chronic left sided cardiac overload due to chronic valve disease is a common cause of insidious development of pulmonary hypertension. For this reason, especially in dogs with CHF who are failing traditional therapy, echocardiography is an important screening tool to identify concurrent pulmonary hypertension as a complicating factor, which would necessitate additional therapeutics (to be discussed below).

The main **causes of pulmonary hypertension** include primary respiratory disorders, such as chronic lung disease, chronic bronchitis/ COPD, Heartworm disease, pulmonary thromboembolism, pulmonary endarteritis, lung lobe torsion, chronic left sided heart disease and volume overload, high altitude disease, congenital heart disease (large left to right shunts – VSD and PDA) and brachycephalic syndrome if chronically hypoxemic [1-3].

Pulmonary hypertension is diagnosed most frequently with advanced echocardiography by a seasoned sonographer that is proficient in color flow and spectral Doppler. A tricuspid regurgitant velocity greater than 2.8 m/s and/or pulmonic insufficiency velocity greater than 2.2 m/s is considered to be abnormal and consistent with pulmonary hypertension [1 Other] sources have reported normal values up to 3.2 m/s (personal communication). The pressure gradient is then calculated using the modified Bernoulli equation $P= 4V^2$ (v= velocity). A normal pressure gradient is considered less than 30 mm Hg while mild PHT is considered when the pressure gradient is calculated between 31-50 mm Hg, moderate pulmonary hypertension between 51-75 mm Hg and severe PHT is considered above 75 mm Hg[4]. However, the gold standard of confirming pulmonary hypertension is via pulmonary arterial catheterization with direct measurement of pulmonary arterial pressures. Thoracic radiographs are imperative to evaluate the pulmonary parenchyma. Additional more advanced testing of pulmonary function can include arterial blood gas assessments (A-a difference calculation) or crudely, pulse oximetry reading (ideally by rectal probe in an awake dog). If thromboembolic disease is suspected, measuring pulse oximetry readings on and off oxygen is a crude way to assess diffusion capabilities. If the pulse oximetry reading is very low and does not improve with oxygen therapy, a VQ mismatch is suspected which would occur in the face of severe PTE. If the pulse oximetry reading normalizes with oxygen supplementation, this is more consistent with a diffusion disorder as occurs with parenchymal disease. Please note, this is a crude test to assess for PTE as a cause for pulmonary hypertension, and not other

causes. Ventilation perfusion scanning using nuclear scintigraphy is considered as a gold standard test.

Cor pulmonale is typified by right atrial enlargement secondary to various primary pulmonary disease states resulting in pulmonary hypertension [1,5] Right sided heart failure evidenced by the development of ascites can develop acutely if there is an abrupt change in pulmonary arterial pressures as can occur with acute severe thromboembolic disease, lung lobe torsion, acute respiratory distress syndrome [ARDS], acute inflammation, or rapidly progressive neoplasia. Alternatively, RHF can develop slowly over time due to chronic disease as occurs in the case of neoplasia, chronic inflammatory disease, asthma/bronchitis, pulmonary fibrosis, and brachycepahlic syndrome[6-10].

NT-proBNP is a useful biomarker to help differentiate primary lung disease and primary cardiac disease; however, NT-proBNP has been documented to increase in cases of PHT [4,10].

Ultrasound, Additional Diagnostic Procedures, & PHT
Echocardiography is the practical standard procedure to rule in or rule out PHT by means of measuring TR and PR jets. However, on occasion, echocardiography fails to document increased tricuspid regurgitant velocities despite strong clinical suspicion for the disease process because the jet can be miniscule and difficult to document especially in a tachypneic patient that causes Doppler artifact that interferes with the evaluation. Regardless, pulmonary hypertension would be suspected even in the absence of high velocity TR jets especially if concentric hypertrophy of the right ventricle occurs in the absence of pulmonic stenosis, inferring the presence of elevated pressures, and potentially supported by the concurrent presence of hepatic vein dilation. In the absence of overt right-sided heart failure, the clinical signs of pulmonary hypertension often overlap with those of left sided heart failure and pulmonary edema. Given that left sided heart failure is often a co-morbid disease process, echocardiography is utilized to rule in/out the presence of left sided heart failure (LHF). If severe left atrial enlargement is present, therapeutic adjustments may be necessary to determine if the clinical signs are due to primary left sided heart failure with inadequate response to therapy or if they are due to concurrent untreated PHT. If left sided heart disease is ruled out, and primary lung disease is suspected, then additional work up may ultimately include a CT scan of the lungs, broncoalveloar lavage or even lung biopsy in some cases. It should be noted that thoracic ultrasound may be beneficial to assess for evidence of pulmonary infiltrates, lung lobe torsion and lung consolidation. Aspiration of focal lesions may be attempted if indicated. Abdominal ultrasound and blood work may be recommended to assess for concurrent disease processes that may result in PHT (such as hyperadrenocorticism, adrenal tumors, or neoplasia as a cause of thromboembolic disease or a primary neoplastic site).

Treatment for PHT initially revolves around the primary inciting cause. In the event of primary left sided heart disease with severe left atrial enlargement with resultant increased left sided pressures leading to PHT, ensure adequate management with appropriate doses of Lasix, benazepril (or enalapril) and Pimobendan the face of left sided failure. These medications are also indicated for therapy of right-sided heart failure in the absence of left sided disease. Pimobendan is an inodilator with both positive inotropic properties as well as vasodilatory effects in the pulmonary vasculature resulting in a reduction of pulmonary hypertension. This is the first choice for PHT in the face of left sided heart failure. The standard dose of pimobendan of 0.25 -0.3 mg/kg PO BID can also be increased to TID as needed to manage severe pulmonary hypertension. Sildenafil (Viagra) – a phosphodiesterase inhibitor is currently thought to be the treatment of choice for pulmonary hypertension especially in the absence of LHF and should be added as a second therapeutic in the face of CHF. The author up titrates the dose slowly over several weeks. The target dose is 1-2 mg/kg PO BID-TID. Begin with a low dose of 0.5 mg/kg PO BID for 2 weeks, and then increase to 1 mg/kg PO BID for 2 weeks, then 1.5 mg/kg PO BID for 2 weeks up to 2 mg/kg PO BID and up to TID a necessary. Supplemental oxygen therapy is needed in severe cases and can also be considered at home on an as needed basis in some patients.

Robyn Roberts RDMS
Appointments: 512-585-6698
Fax: 512-686-3149
Email: scheduling@mettasound.com

Prognosis for PHT is still guarded yet management has certainly improved with the advent of sildenafil and pimobendan in the past number of years. Monitoring clinical signs, respiratory rate and effort as well as periodic echocardiographic exams will be necessary. BUN, creatinine, electrolytes, radiographs and systemic blood pressure will also be recommended and tailored to the individual case depending on the underlying disease process and current medical management.

References

1) Johnson L, Boon J, Orton EC. Clinical characteristics of 53 dogs with Doppler-derived evidence of pulmonary hypertension: 1992-1996., J Vet Intern Med. 1999 Sep-Oct;13(5):440-7.
2) Glaus TM, et al: Clinical and pathological characterization of primary pulmonary hypertension in a dog. *Vet Rec* 154:786-789, 2004.
3) Glaus TM, et al: Non-invasive measurement of the cardiovascular effects of chronic hypoxaemia on dogs living at moderately high altitude. *Vet Rec* 152: 800-803, 2003.
4) Pulmonary Hypertension and N-Terminal Prohormone Brain Natriuretic Peptide in Dogs. Farace G, Ettinger SJ; Forney S et al Proceedings of the American Veterinary Internal Medicine Conference. Montreal 2009
5) Nauser, TD, Stites SW: Diagnosis and Treatment of Pulmonary Hypertension, Amer Fam Physi 2001;63:9:1789-1796.
6) D'Anjou MA, Tidwell AS, Hecht S.: Radiographic diagnosis of lung lobe torsion, Vet Radiol Ultrasound. 2005;46(6):478-84.
7) MacDonald KA and Johnson LR Pulmonary Hypertension and Pulmonary Thrombi. In Textbook of Veterinary Internal Medicine ed. Ettinger SJ and Feldman EC Elsevier 2008 p1284-1286.
8). Topal U, Ediz B.: Transthoracic needle biopsy: factors effecting risk of pneumothorax. Eur J Radiol. 2003; 48(3): 263-7.
9) Orton EC. Pulmonary Hypertension. In The Veterinary ICU Book. Ed Wingfield WE and Raffe MR. 2002. Teton NewMedia, Jackson WY. P524-539.
10) Rozanski E. Interstitial Lung Disease in Small Animals. Proceedings of the American Veterinary Internal Medicine Conference. Denver 2011.
11) Assessment of serum N-terminal pro-B-type natriuretic peptide concentration for differentiation of congestive heart failure from primary respiratory tract disease as the cause of respiratory signs in dogs. Oyama MA, Rush JE, Rozanski EA, et al. J Am Vet Med Assoc. 2009 Dec 1; 235(11): 1319-25.

Johanna Frank DVM DVSc
Diplomate ACVIM (Internal Medicine)
New Jersey Mobile Associates & SonoPath.com

Dr. Johanna Frank is my assistant director of operations in NJ Mobile with 15 years of experience in ultrasound diagnostics and internal medicine. Johanna is also my primary coauthor for our pending textbook Clinical Approach To Sonographic Pathology to be offered by SonoPath.com.
Best regards and feel free to look at our many case studies on cardiac and abdominal disease as well as many new developments and resources for the clinical sonographer and general practitioner alike at www.SonoPath.com.

Eric Lindquist, DMV (Italy) DABVP
Cert. IVUSS
Founder/CEO SonoPath.com
Director NJ Mobile Associates

For mobile appointments in the Austin, TX region contact:

Robyn Roberts RDMS
Appointments: 512-585-6698
Fax: 512-686-3149
Email: scheduling@mettasound.com

This Communication has Been Fueled By

SonoPath.com™
ENHANCING DIAGNOSTIC EFFICIENCY

SonoPath LLC. 31 Maple Tree Ln. Sparta, NJ 07871 USA
Via Costagrande 46, MontePorzio Catone (Roma) 00040 Italy ***Tel: 800 838-4268***
For Case Studies & More Hot Topics In Veterinary Medicine For The GP & Clinical Sonographer Alike, Visit
www.SonoPath.com

MettaSound
Veterinary Imaging

Robyn Roberts RDMS
Appointments: 512-585-6698
Fax: 512-686-3149
Email: scheduling@mettasound.com