Canine and Feline Caval Syndrome

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The caval syndrome is a serious complication of chronic heartworm (*Dirofilaria immitis*) disease in dogs and cats. The syndrome is characterized by acute anorexia, respiratory distress, weakness, right-sided cardiac murmur, anemia, hemoglobinuria, hepatic and renal dysfunction, signs of forward and backward heart failure, and, possibly, disseminated intravascular coagulation (DIC). Retrograde migration of adult heartworms from the pulmonary arteries to the right ventricle, right atrium, and venae cavae causes disruption of the tricuspid apparatus. Valvular insufficiency, with concurrent pulmonary hypertension, reduces cardiac output thus resulting in forward and backward heart failure. Additionally, red blood cells are traumatized and hemolyzed as they flow through the mass of worms. Therapy consists of supportive care and the removal of the heartworm mass from the right ventricular inflow tract. Caval syndrome in dogs and cats is associated with high mortality rates and generally has a guarded to poor prognosis.

Caval syndrome (CS), also referred to as venae cavae embolism, venae cavae syndrome, postcaval syndrome, acute hepatic syndrome, liver failure syndrome, or dirofilarial hemoglobinuria,[1] is a serious life-threatening complication of chronic heartworm disease (HWD). The syndrome associated with *Dirofilaria immitis* infection was first described in dogs by Adams[2] in the 1950s, and by Jackson et al[3] and von Lichtenberg et al[4] in the 1960s, and has since been extensively described. The syndrome has also been described in cats.[5]

Clinical features of CS include acute anorexia, dyspnea, weakness, onset of a right-sided systolic murmur, jugular vein distention and pulsation, weak femoral pulses, pale mucous membranes, anemia, hemoglobinemia, hemoglobinuria, hepatic and renal dysfunction, disseminated intravascular coagulation (DIC), as well as both forward and backward heart failure. This acute manifestation of chronic heartworm disease is probably associated with the movement of large numbers of heartworms from the diseased pulmonary arteries (PA) to the right ventricle (RV), right atrium (RA), and/or venae cavae (VC) which results in moderate to severe tricuspid regurgitation (TR).[1] Concurrent moderate to severe pulmonary hypertension exacerbates the hemodynamic effects of TR and results in right-sided heart failure and reduced right ventricular output. The pathophysiology, diagnosis, and treatment of caval syndrome in dogs and cats is reviewed.

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Pathophysiology and Genesis of the Caval Syndrome

The pathogenesis of heartworm disease-induced caval syndrome has been evaluated by several authors and is not fully understood. Caval syndrome occurs in up to 16% to 20% of dogs with spontaneous heartworm disease, and is seen in middle-aged (mean age of onset is 5 years; range of 1.5 to 10 years) male (75% to 90%) dogs in the spring and early summer.[6-10] Nine of 16 (72.5%) dogs experimentally infected with 200 infective *Dirofilaria immitis* larvae developed caval syndrome between 7 to 17 months postinoculation (mean interval to development of CS was 12.3 months).[11] Most studies indicate that males are predisposed, and that this predilection is not solely related to differences in exposure to infective larvae.[1,11] Dogs presenting with caval syndrome may be microfilaria positive or negative.

In cases of caval syndrome, worm burdens are usually heavy with most dogs having greater than 60 worms. Jackson et al[10] reported that in a series of 117 canine necropsies, dogs with asymptomatic heartworm infection had a mean of 25 worms, dogs with chronic heartworm disease had a mean of 58 worms, and dogs with caval syndrome had a mean of 101 worms. Atkins et al[11] investigated caval syndrome in dogs experimentally infected with *Dirofilaria immitis* and demonstrated no significant difference between dogs with CS and dogs without CS with regard to total worm burden, female worm burden, and relative worm burden (worms/kg body weight). However, factors other than a high worm burden are likely to be involved in the genesis of caval syndrome.

In caval syndrome, a large percentage of the worm burden redistributes to the right ventricle, right atrium, and/or the venae cavae. This atypical distribution was demonstrated in several studies in which 0% to 21% of the worm burden resided in the RA and VC in dogs without CS and 55% to 84% in dogs with acute CS.[6,9,11] The reason for the abnormal distribution of heartworms is currently unknown, although many hypotheses have been proposed. Potential causes of caval syndrome include the following: 1) high relative worm burden; 2) simultaneous migration of maturing worms in heavily infested dogs accounting for simultaneous arrival at the heart; 3) delayed migration of maturing heartworms accounting for simultaneous arrival at the heart; 4) retrograde migration of adult heartworms from the pulmonary arteries to the right atrium and venae cavae in response to hemodynamic changes such as acute worsening of pulmonary hypertension and reduced cardiac output; and 5) migration of adult heartworms from the pulmonary arteries potentially associated with administration of heartworm preventative or adulticide.[9,12-14]

Of the proposed hypotheses, retrograde migration of the adult heartworms from the PA to the RV, RA, and VC for some unknown reason was suggested by Atkins et al[9,15] as the most plausible explanation for the initiation of the clinical syn-

drome. Simultaneous arrival to the heart of maturing worms in heavily infested dogs is an unlikely cause of caval syndrome in dogs since several studies have demonstrated the retrograde migration of heartworms from the pulmonary arteries to the RA and VC.[16-18] Further evidence that simultaneous arrival is unlikely is that there are already signs of chronic heartworm disease (such as radiographic evidence of pulmonary parenchymal lesions, pulmonary arterial dilation and truncation, and right-sided cardiomegaly and hemodynamic alterations such as pulmonary hypertension) at the onset of CS.

The role of pulmonary hypetension in the genesis of caval syndrome has been evaluated by several authors. Atkins et al[11] showed that mean pulmonary arterial pressures (PAP) increased with the progression of heartworm disease in dogs with and without CS. The mean pulmonary arterial pressures in dogs with CS (PAP of 60 (+18) torr) were significantly greater than those that did not develop CS (30 (+4) torr). The above findings have also been supported by other studies.[1,11 14,19] The cause of the pulmonary hypertension in dogs with HWD and CS is thought to be a combination of the progressive endarteritis and villous endothelial proliferation associated with chronic heartworm infection, and thromboembolic phenomena associated with the death of either adult heartworms or microfilaria. Additionally, decreased pulmonary arterial endothelium-dependent relaxation is thought to contribute to the development of pulmonary hypertension in dogs with HWD.[20] Increased mean pulmonary arterial pressure has been shown to correlate significantly with the severity of pulmonary thromboembolism.[14] Sasaki et al[19] examined 41 dogs with naturally acquired heartworm disease (28 cases of chronic heartworm disease and 13 cases of caval syndrome) in an effort to determine the relationship between pulmonary hypertension and lesions of the pulmonary arteries and parenchyma, and cardiac valves. Multiple correlation analysis revealed the highest correlation was between pulmonary hypertension and lesions of the pulmonary arteries and parenchyma, and cardiac valves. Multiple correlation analysis revealed the highest correlation was between pulmonary arterial pressure and thromboembolism, followed by mitral valvular lesion, tricuspid valvular lesion, and pneumonic lesion. There was no significant correlation between PAP and intimal lesions. They concluded that pulmonary thromboembolism following natural death of heartworms was the most important factor causing an increase in PAP. The presence of fresh pulmonary thrombi in dogs with caval syndrome has been noted in several other studies.[13 14,18] It is currently unknown whether the more severe pulmonary hypertension seen in dogs with CS is an initiating factor or merely a manifestation of the syndrome. It has been shown though that heartworm-infected dogs with severe pulmonary hypertension due to thromboembolism do not always develop CS.[14] Furthermore, the entire worm burden does not migrate from the pulmonary arteries to the RA and VC during CS which further suggests that pulmonary hypertension is not the sole cause of CS.

There are several studies that have demonstrated that changes in right-heart hemodynamics induce heartworm migration from the pulmonary artery toward the right atrium and venae cavae. Kitagawa et al[17] demonstrated heartworm migration in five of six dogs with spontaneous heartworm disease after the administration of the cardioselective, beta-1 antagonist metoprolol (8 mg/kg PO). Milbemycin D (1.5 mg/kg PO), a topically-applied analog of milbemycin oxime, caused changes in heart rate and right ventricular hemodynamics and induced heartworm migration 50 to 105 minutes after administration.[21] Additionally, the cardiopulmonary depressant, pentobarbital sodium (25 mg/kg IV) has been used to experimentally induced retrograde heartworm migration.[14] Most indices of right ventricular performance (cardiac output, stroke volume, cardiac index, stroke index) decreased before the migration of the heartworms. These findings suggest that changes in right-heart hemodynamics that result in decreases in heart rate and cardiac output are associated with heartworm migration in some dogs with HWD.

Retrograde migration of heartworms is thought to occasionally occur after the administration of heartworm preventatives, microfilaricides, and adulticides. Kitagawa et al[22] reported caval syndrome in 12 of 89 microfilarial-positive dogs after the administration of milbemycin oxime at both the heartworm prophylactic dose (0.25 mg/kg) and the hookworm anthelmintic dose (0.5 mg/kg). Caval syndrome secondary to the administration of dichlorvos in a heartworm-infected dog has also been reported.[23] Heartworm migration and caval syndrome occurs infrequently after administration of adulticides such as melarsamine dihydrochloride and thiacetarsamide. Heartworm migration has been induced after the insertion of dead heartworms, heartworm-like silicone tubes, or after intravenous injection of body fluid extract from a female heartworm.[18] Heartworm migration occurred immediately after intravenous injection of the heartworm body fluid extract. These results suggest that the death of a few adult heartworms or microfilaria may be associated with retrograde migration of heartworms from the pulmonary arteries toward the right atrium and venae cavae.

Cardiopulmonary dysfunction in dogs with CS occurs as a result of valvular insufficiency with superimposed pulmonary hypertension. Disruption of the tricuspid valve apparatus with secondary moderate to severe valvular insufficiency results when the worm mass flows back and forth across the tricuspid valve. Pulmonary hypertension exacerbates the hemodynamic effects of the tricuspid regurgitation resulting in pressure and volume overloading of the right ventricle, and subsequent reduction in cardiac output. Furthermore, ventricular interdependence dictates that the left ventricle is affected by the pressure and volume overload in the right ventricle as a result of decreased right ventricular output, altered ventricular geometry, increased ventricular stiffness, and the associated decreased left ventricular inflow and reduced cardiac output.[15]

Invasive hemodynamic studies of dogs with CS have consistently revealed pulmonary and right ventricular hypertension, elevated central venous pressures (CVP) and right ventricular end-diastolic pressures (RVEDP), and decreased cardiac output, cardiac index (cardiac output/body surface area, m^2), and systemic blood pressure. Kitagawa et al[24] evaluated central venous pressures in 19 dogs with dirofilarial hemoglobinuria and found that the CVP ranged from 32 to 187 mmH_2O (mean 101.6 mmH_2O) before worm extraction and from -10 to 130 mmH_2O (mean 61.0 mmH_2O) after worm extraction. Inflow obstruction may or may not be involved in the pathogenesis of CS. Kuwahara et al[13] suggested that inflow obstruction (or tricuspid stenosis), as evidenced by elevated A-waves on right atrial pressure tracings, contributed to the decrease in right ventricular cardiac output. Others have suggested that the presence of increased mean right ventricular end-diastolic diameter, as determined by M-mode echocardiography, dis-

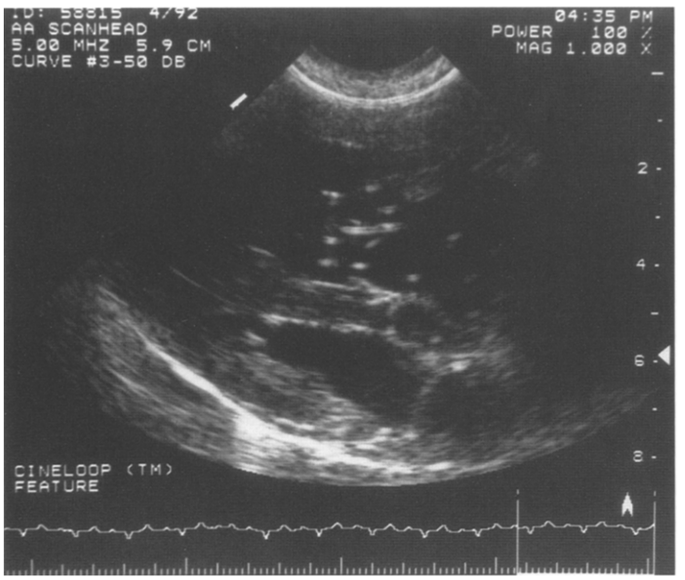

Fig 1. Two-dimensional long-axis view obtained from the right parasternal window of a dog with caval syndrome demonstrating heartworms (hyperechoenic parallel linear densities) spanning the tricuspid valve, right atrial and ventricular enlargement, and diminished left ventricular size.

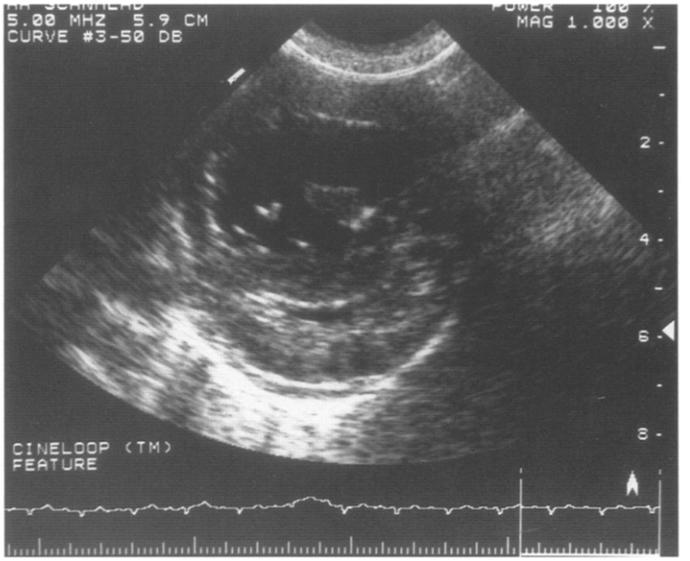

Fig 2. Two-dimensional (2D) short-axis view obtained from the right parasternal window at the level of the papillary muscles of a dog with caval syndrome demonstrating massive right ventricular dilation and hypertrophy, papillary muscle hypertrophy, hyperechogenic densities within the RV consistent with heartworms or chordae tendineae, severely diminished left ventricular size, and flattening and posterior displacement of the interventricular septum consistent with right ventricular pressure and volume overload. A small posterior pericardial effusion is also present.

counts the importance of inflow obstruction.[15] Persistence of tricuspid regurgitation, pulmonary hypertension, and decreased cardiac output are negative prognostic indicators in dogs with CS. Persistence of TR after heartworm removal suggests a worse prognosis since regurgitation of contrast medium after a right ventricular injection typically resolves after HW removal in dogs that survive the syndrome. In 25 dogs with naturally-occurring CS and 4 dogs with drug-induced CS, nonsurvivors had significantly higher A- and v-waves and right ventricular end-diastolic pressures than survivors.[14] Although total pulmonary resistance and right atrial A- and v-waves decreased after heartworm removal in both survivors and nonsurvivors, nonsurvivors continued to demonstrate pressure tracings suggestive of congestive heart failure (ie, elevated A- and v-waves and mean right atrial pressure).

Echocardiographic evaluation provides much insight into the pathophysiology of heart failure associated with CS. In several studies involving dogs and cats with CS, two-dimensional (2D) echocardiography consistently revealed a mass of heartworms spanning across the right ventricular inflow tract which then "flowed" into the RV during diastole.[15,25,26] Mechanical disruption of the tricuspid valve apparatus results in moderate to severe tricuspid insufficiency which is demonstrable with Doppler echocardiography. The importance of the TR as a contributor to cardiac dysfunction is evidenced by the sudden increase in right ventricular end-diastolic dimension and reduced right ventricular output in association with the onset of the syndrome. In comparison, trivial TR does not cause appreciable changes in right ventricular end-diastolic dimension or cardiac output. Other echocardiographic findings include right ventricular dilation and hypertrophy, paradoxical septal motion, diminished aortic root, left atrial and ventricular size, and decreased ratio of left-to-right ventricular diastolic internal diameter size, as well as decreases in estimates of cardiac index and stroke volume. Interestingly, echocardiographic indices of left ventricular function (velocity of circumferential fiber shortening, fractional shortening, ejection fraction, and pre-ejection period) were considered within normal limits in dogs with CS.[15] Poor left ventricular output in combination with normal indices of ventricular function suggests a loading abnormality such as concurrent decreases in pre- and afterload (see Figs 1, 2, and 3).

The hemolytic anemia seen in caval syndrome is likely the result of traumatic mechanical shearing forces placed on fragile

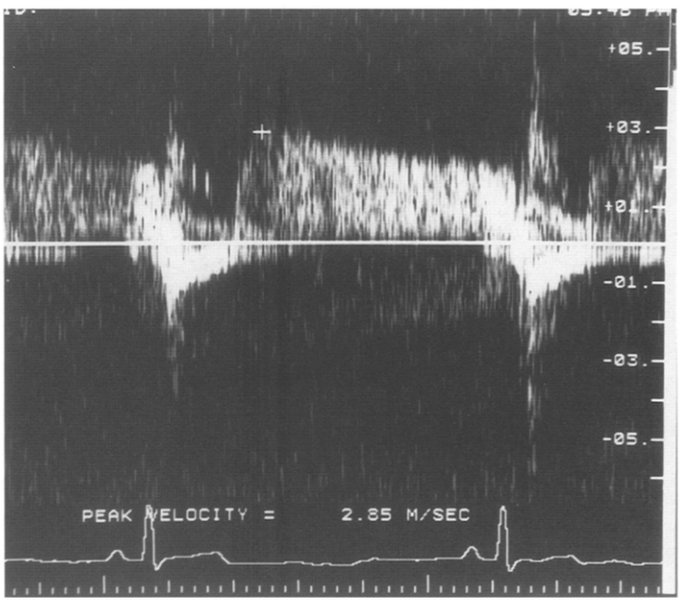

Fig 3. Continuous-wave Doppler echocardiographic study obtained from the right parasternal window demonstrating transpulmonic flow in a dog with caval syndrome. Note the high-velocity pulmonic insufficiency (maximum velocity approximately 2.85 m/s) during diastole suggestive of pulmonary hypertension.

erythrocytes as they flow through the mass of adult heartworms within the right ventricular inflow tract or potentially as a result of the microangiopathic processes associated with DIC.[12,27,28] Erythrocyte fragility is thought to be associated with membrane instability resulting from changes in serum-free and esterified cholesterol concentrations and lecithin aceyltransferase activity.[29] The role that immunologic factors play in the pathogenesis of anemia in heartworm disease appears to be of minor significance.[8,25]

Hemoglobinemia, hemoglobinuria, and renal/hepatic dysfunction are frequently noted in cases of caval syndrome.[29] Hemoglobinuria in conjunction with the appropriate clinical signs has been considered pathognomonic for caval syndrome for many years.[3] In one report, all 36 cases of CS had hemoglobinuria.[30] Hemoglobinuria occurs when intravascular hemolysis results in destruction of erythrocyts at a rate that exceeds the liver's capacity for conversion of hemoglobin to bilirubin.[31] The relationship between the presence of a heartworm mass within the tricuspid valve apparatus and plasma hemoglobin concentrations was evaluated and the authors observed in some dogs with CS the simultaneous development of hemoglobinemia with that of a heartworm mass in the tricuspid valve apparatus.[32] Furthermore, plasma hemoglobin concentrations dramatically decreased within 30 minutes after the removal of the worm mass from the right ventricular inflow tract and were almost normal 20 hours after heartworm extraction. These findings further implicate erythrocyte trauma associated with the mass of adult heartworms as the cause of anemia in cases of CS. The pathogenesis of hepatorenal dysfunction is thought to be associated with poor tissue perfusion, passive congestion, and hypoxia of these organs. Hepatomegaly, centrolobular necrosis and fibrosis, and cavernomatous venous dilation and thrombosis, are among the pathological findings found at necropsy and on histopathology, respectively.[32] Kidneys from dogs with caval syndrome frequently demonstrate tubular necrosis, tubular heme casts, and hemosiderosis.[32]

In summary, the likely course of events surrounding the development of caval syndrome are as follows: 1) acquisition of a heavy heartworm infestation; 2) hemodynamic alteration (acute pulmonary hypertension and/or reduced cardiac output); 3) retrograde heartworm migration from PA to RV, RA, and VC; 4) valvular dysfunction and further reduction of cardiac output; 5) intravascular hemolysis; 6) extracardiac complications associated with poor tissue perfusion and hypoxia such as prerenal azotemia, passive congestion of the abdominal organs, DIC, etc; 7) progressive disease; and 8) usually death (within 24 to 72 hours) (see Fig 5).

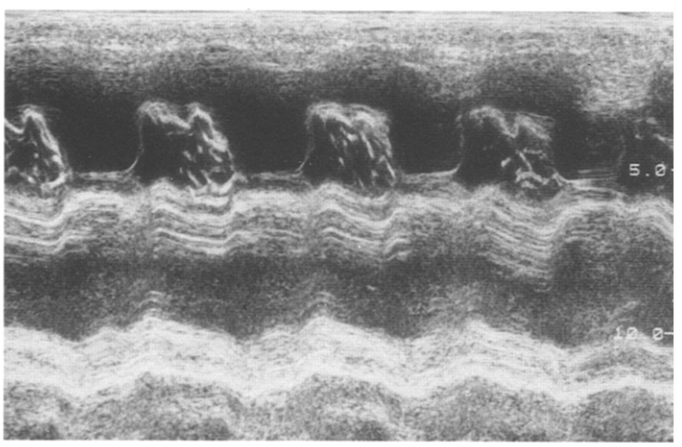

Fig 4. M-mode tracing obtained from the right parasternal window at the level of the tricuspid valve of a dog with caval syndrome demonstrating hyperechogenic parallel linear densities within the tricuspid valve during diastole, right ventricular enlargement, paradoxical septal motion secondary to right ventricular pressure and volume overload, and diminished left ventricular size.

Diagnosis

The clinical manifestations of the caval syndrome are characterized by a new cardiac murmur of tricuspid regurgitation, regenerative anemia, hemoglobinemia, hemoglobinuria, renal and hepatic dysfunction, signs of forward and backward heart failure, and possibly DIC. The most common observations by the owner include an acute onset (1 to 2 days) of inappetance or anorexia, lethargy, weakness, respiratory distress, and usually dark, red-colored urine. Coughing is less commonly reported by the owner.[4] Many dogs and cats have no history suggestive of heartworm disease (for example, coughing) prior to the onset of signs. Physical examination reveals signs of low cardiac output such as mucous membrane pallor, prolonged capillary refill time, weak femoral pulses, weakness, tachypnea, tachycardia, and signs of right-sided heart failure such as jugular distention, jugular pulsations, engorgement of peripheral veins, hepatosplenomegaly, and potentially abdominal distention from hepatosplenomegaly or ascites. Occasionally, mild jaundice is noted. Careful thoracic auscultation may reveal a murmur of tricuspid insufficiency, splitting of the second heart sound (split S2), and cardiac gallop rhythms in addition to increased bronchovesicular lung sounds.

Many clinicopathologic abnormalities are present in dogs and cats with caval syndrome.[1,4,5,9,25,26] Hemogram changes may include regenerative anemia with polychromasia, reticulocytosis, increased plasma hemoglobin, mean corpuscular volume (MCV), nucleated red blood cells, schistocytes, and thrombocytopenia. The leukogram may demonstrate eosinophilia, basophillia, and increased numbers of band neutrophils suggesting inflammation. Serum chemistry abnormalities include elevated blood urea nitrogen (BUN), alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), creatinine kinase (CK), total bilirubin, glucose, and alpha-and gammaglobulins, and decreased albumin concentration. Urinalysis demonstrates bilirubinuria, hemoglobinuria, proteinuria, and the presence of casts which signals tubular damage. A coagulation profile may be normal or may demonstrate prolonged one-stage prothrombin, partial thromboplastin, and activated clotting times, and elevated fibrin degradation product (FDPs) concentrations if DIC is present. Blood gas analysis usually reveals decreased PaO_2, $PaCO_2$, arterial pH, and bicarbonate levels.[34] In addition, an increased alveolar-arterial gradient ($AaDO_2$) and serum lactic acid levels have been reported. In a comparison of blood gas analyses one week after heartworm removal, survivors had increased PaO_2 and $PaCO_2$ concentrations and decreased $AaDO_2$ while nonsurvivors did not show relative improvement in their blood gas parameters suggesting that blood gas analysis may be useful as a prognostic indicator.[34]

Electrocardiographic findings in dogs with caval syndrome may include evidence of a rightward shift in the mean electrical axis (deep S-waves in leads I, II, III and aVF with a mean MEA

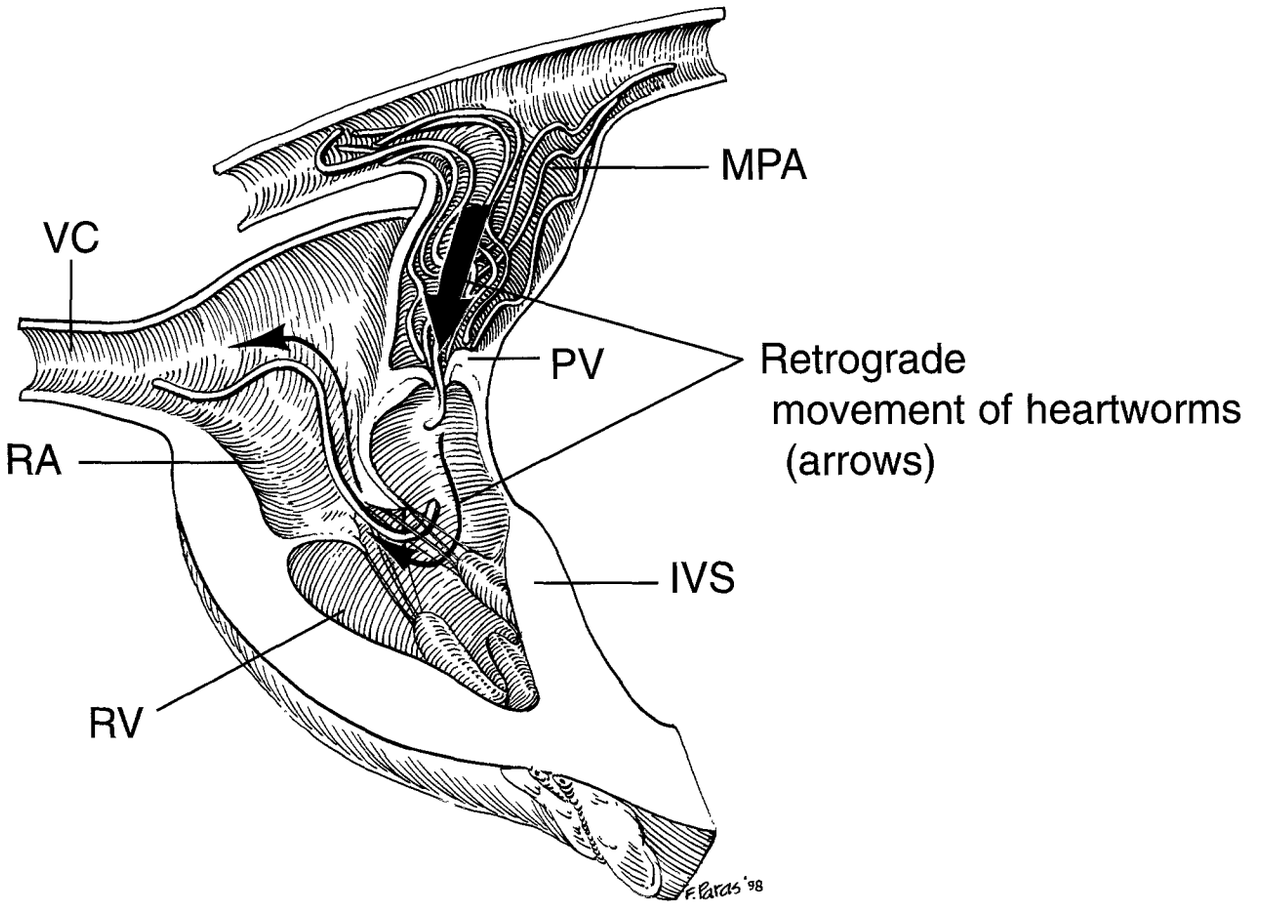

Fig 5. Reterograde movement of heartworms: VC—vena cava; RA—right atrium; RV—right ventricle; PV—pulmonic valve; IVS—interventricular septum; MPA—main pulmonary artery.

of +129°), sinus tachycardia, and premature atrial and ventricular complexes.[1] Thoracic radiography usually displays signs of chronic heartworm disease such as right ventricular enlargement, dilation of the main pulmonary artery, increased pulmonary arterial size when compared to their respective veins, distended caudal vena cava, and pulmonary arterial tortuousity and truncation in some severe cases. A caudal vena cava to descending aorta ratio of >1.50 may be helpful to define right heart disease.[35] Patchy mixed interstitial/alveolar infiltrates may be present in some cases with parenchymal disease or possibly pulmonary thromboembolism.

Echocardiography is the most important diagnostic test since visualization of the heartworm mass "flowing" across the tricuspid valve during diastole is considered pathognomonic for this syndrome (see Fig 3). Additionally, right ventircular dilation and hypertrophy, abnormal septal movement, diminished left atrial and ventricular dimensions, and pulmonic and tricuspid insufficiency as demonstrated by Doppler echocardiography are usually present.

Treatment

Because CS is a multi-systemic disorder, many preoperative considerations must be taken into account. The prognosis for CS is guarded to poor and mortality rates still remain high (up to 42%) even in the face of successful heartworm removal. Accurate assessment of the severity of complicating factors (such as anemia, hypovolemia, hypotension, cardiac, renal, and hepatic dysfunction, coagulopathy, etc) will dictate the therapy. The appropriate data should include a thorough physical examination, complete blood count, chemistry profile, urinalysis, electrocardiography, thoracic radiography, echocardiography, coagulation profile including FDPs, blood gas analysis, and central venous pressures prior to heartworm embolectomy. Removal of the heartworm mass should be performed as soon as it is practical and probably should not be delayed solely for the acquisition of laboratory data.

Supplemental oxygen therapy is extremely important in the treatment of thromboembolism and pulmonary hypertension because hypoxia-induced vasoconstriction is thought to occur secondary to lung disease in dogs with HWD. Rawlings and Tackett suggested that oxygen therapy could reduce PAP and right ventricular afterload.[36] Fluid therapy consisting of crystalloids such as 0.45% NaCl with 2.5% dextrose is indicated for the treatment of metabolic acidosis, and decreased cardiac output and tissue perfusion. However, fluid therapy must be administered judiciously since most dogs and cats with CS have elevated CVP. Maintenance of a short jugular catheter (placed in the left external jugular vein) and evaluation of serial CVP measurements will facilitate monitoring of fluid therapy. Shock doses of fluids (up to 80 to 90 mL/kg in dogs and 50 to 55 mL/kg in cats during the initial hour) may be necessary in some animals but is contraindicated in animals with CVPs greater than 10 to 20 cmH_2O. Only severe metabolic

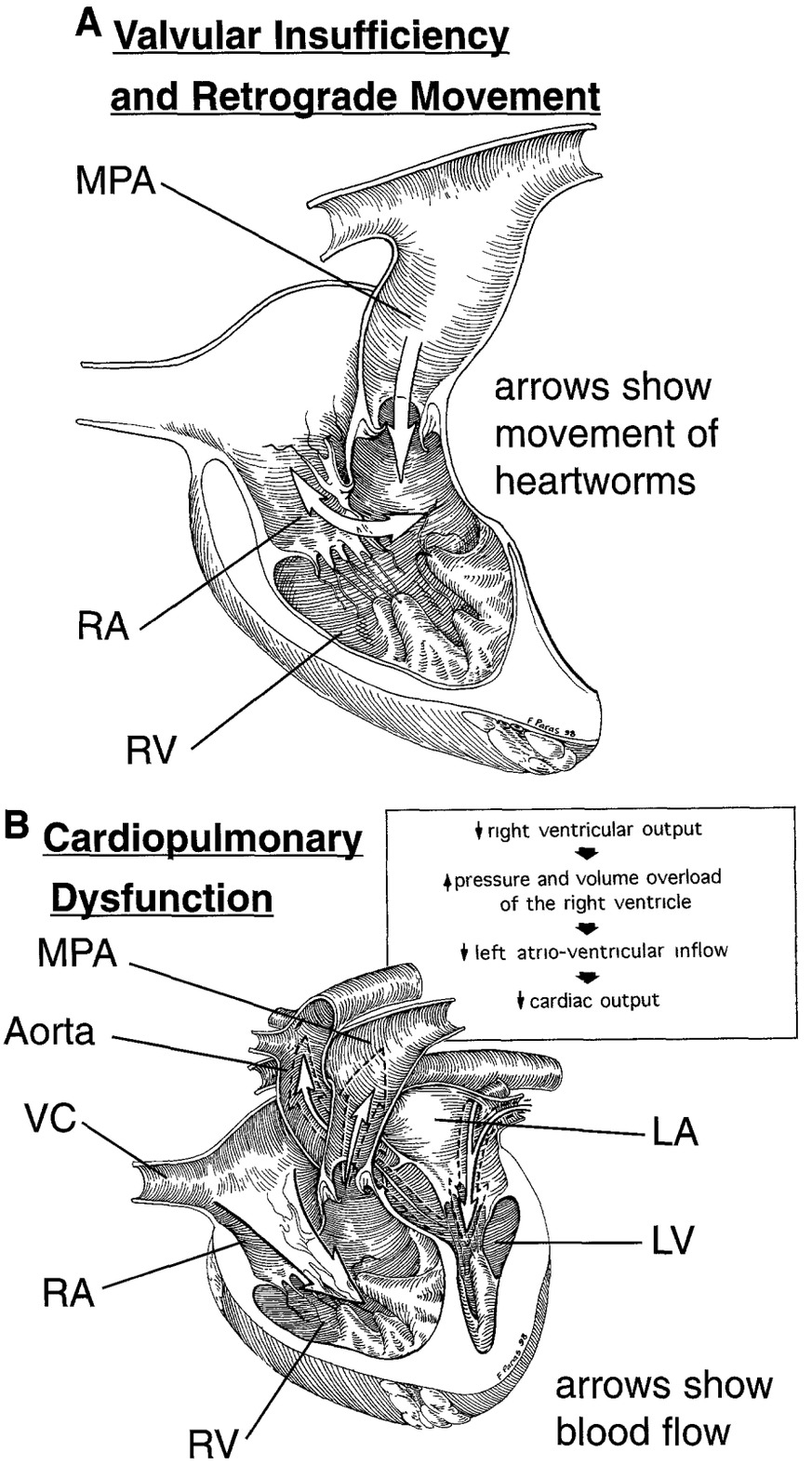

Fig 6. Valvular insufficiency.

acidosis (ie, arterial pH < 7.2) and severe anemia (ie, pcv less than 10% to 15%) necessitate therapy with bicarbonate or whole blood transfusion, respectively. Other therapeutic considerations might include broad-spectrum antibiotics, aspirin to reduce thromboembolic phenomena, diltiazem or hydralazine to treat pulmonary hypertension, and cage rest.[37] Disseminated intravascular coagulation (DIC) may be present in dogs and cats presenting with CS. As with any patient with DIC, efforts should be made to correct the underlying problem (ie, supportive care and heartworm embolectomy). Heparin therapy (200 U/kg SC TID for chronic, low-grade DIC and up to 500 U/kg SC TID for acute DIC) may be beneficial.[1] Plasma therapy may also be beneficial if antithrombin III levels are diminished.

Surgical intervention is indicated by the presence of hemoglobinuria, low output heart failure, anemia, and echocardiographic demonstration of heartworms within the tricuspid valve apparatus.[8] The technique for removal of heartworms from the right heart and venae cavae via jugular venotomy was first described by Jackson et al.[10] Sedation may be necessary if

the patient is not severely debilitated, although cats generally require general anesthesia. The patient is restrained in left lateral recumbency and the right cervical area is clipped and surgically prepared. Local infiltration of 2% lidocaine followed by a jugular vein cutdown procedure isolates the right jugular vein. Umbilical tape is placed proximally and distally to control hemorrhage and a venotomy is performed. Any one of several worm retrieval devices (rigid or flexible 20 to 40 cm alligator forcep,[38] endoscopic basket retrieval device[1,5,25,39] or a Tayama-type string brush used in cats[5,26]) is advanced to a pre-measured distance at the level of the right atrium. Attempts to retrieve heartworms are made until five consecutive attempts are unsuccessful. The jugular vein is ligated both proximally and distally, and skin closure is completed in routine fashion. In addition to the transvenous approach developed by Jackson et al several other methods have been described. Kuntz et al described the use of a cannula in a modified surgical approach to the right atrium in small dogs that had heartworms entangled in the chordae tendineae of the tricuspid valve while others have reported open-heart surgical removal of heartworms.[40,41]

Resolution of the caval syndrome is indicated by clearance of the hemoglobinuria, reduction in the intensity of the TR murmur, and hemodynamic improvement supported by a decrease in the CVP and an increase in cardiac output. A postoperative echocardiogram may be useful in documenting the success of heartworm removal. A poor prognosis is associated with persistent hypoxemia, reduced cardiac output, severe pulmonary hypertension, hypothermia, ascites, and elevations in CVP. Dogs that survive should undergo staged adulticide treatment with melarsamine dihydrochloride approximately 2 to 3 weeks after stabilization. In cats, adulticide therapy should be used with extreme caution and is generally not recommended. Adulticide therapy should be followed by strict cage rest for at least 4 weeks Microfilarial positive animals should receive microfilaricide therapy approximately 4 to 6 weeks after the completion of adulticide therapy and heartworm preventative is initiated after the completion of microfilaricidal therapy.

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