ACVIM CONSENSUS RECOMMENDATIONS

Guidelines for Diagnosis and Treatment of CVHD (Stage A and B)

	Description	Diagnosis	Therapy
Stage A	Dogs at high risk for HF but without apparent structural abnormality	-Regular yearly evaluations (auscultation by rDVM) for small breed dogs and breeds with known predisposition -Owners of high risk dogs can choose to participate in yearly screening events conducted by cardiologists (usually at shows)	-No drug therapy -No dietary therapy
Stage B1	Dogs with structural abnormality indicating the presence of CVHD but have never had clinical signs of HF B1: hemodynamically insignificant MR	-Thoracic radiographs when asymptomatic -Blood pressure measurement -Echo **recommended in small breeds** with typical murmurs (*to answer specific questions regarding chamber enlargement if not answered adequately by auscultation and thoracic rads)*	B1 (normal LA, LV or both with normal LV systolic function, normal VHS; normotensive): -No drug or dietary therapy recommended -Re-evaluation buy radiology or echo with Doppler studies in 12 months (some recommend more frequen follow up in large dogs)
Stage B2	B2: hemodynamically significant MR	-Echo **indicated in large breeds** *(murmur of MR more likely related to other causes i.e. DCM)* -Basic lab work for all patients (hct, TP, creatinine, UA)	B2 (cardiac remodeling – enlarged LA, LV or both; normotensive): **No consensus re: drug or dietary recommendation**

Guidelines for Diagnosis and Treatment of CVHD (Stage C)

	Diagnosis	Therapy
Stage C: Dogs with structural abnormality and current or previous clinical signs of HF	-Clinical database (chest rads, echo, basic labs) -[Serum NT pro-BNP] -Signalment and PE can be helpful *Ex1: obese dogs with no history of weight loss are less likely to be in HF secondary to CVH* *Ex2: dogs with marked sinus arrhythmia and slow HR less likely to have HF secondary to CVHD* -CBC, Biochem, UA if therapy for CHF is anticipated	**<u>Acute (hospital-based)</u>** -Furosemide: dosing should be related to severity of CS and response (1-4mg/kg). Repeated IV boluses or CRI indicated for poorly responsive dogs -For life-threatening pulmonary edema (poor initial response to furosemide bolus in first 2 hours, expectoration of froth): furosemide administered as CI 1mg/kg/hr after initial bolus -Free access to water -Pimobendan 0.25-0.3mg/kg PO q12 *strongly supported by hemodynamic and experimental evidence* -O2 supplementation -Mechanical treatments: thoracocentesis or abdominocentesis for effusions sufficient to impair ventilation -Nursing care -Sedation: butorphanol + combinations -CRI of nitroprusside for up to 48 hours useful for life-threatening poorly responsive pulmonary edema
		<u>Chronic (home-based)</u> -PO furosemide to effect (2mg/kg q12 usually, range 1-6mg/kg IV q8) with attention to effects on renal function and electrolyte status -Start (or continue) ACEI (enalapril 0.5mg/kg PO q12 or equivalent) -Measure creatinine and electrolytes 3-7 days after initiating ACEI -Continue Pimobendan (0.25-0.3mg/kg PO q12) -Do not start beta blocker in face of active clinical signs of HF caused by CVHD -Follow up to ensure body weight, appetite, resp and HR monitoring
		<u>Dietary</u> -Maintain adequate calorie intake to minimize weight loss -Record weight at every visit -Ensure adequate protein intake and avoid low protein diets designed to treat CKD unless severe concurrent renal failure is present -Modestly restrict sodium intake -Monitor serum [potassium] and supplement diet with potassium from natural or commercial sources

Guidelines for Diagnosis and Treatment of CVHD (Stage D)

	Description	Diagnosis	Therapy
Stage D	-Refractory HF -Patients are receiving maximum recommended or tolerated dosage of furosemide, ACEI, and pimobendan	-Involves same diagnostic steps outlined for stage C PLUS failure to respond to treatments. -Chronic oral furosemide (>6mg/kg q12) needed to maintain patient comfort in the face of appropriate adjunct therapy indicates disease progression to Stage D	**<u>Acute (hospital-based)</u>** -Additional furosemide IV as a bolus of 2 mg/kg followed by additional bolus doses, OR furosemide CRI of 1 mg/kg/h **until respiratory distress has decreased, or for a maximum of 4 hours.** *Unless there is evidence of severe renal insufficiency (serum creatinine > 3mg/kg IV)* -Continue to allow patient free access to water -Fluid removal (abdominocentesis, thoracocentesis) as needed to relieve respiratory distress or discomfort -O2 supplementation + mechanical ventilation if necessary (allow time for meds to take effect) -More vigorous afterload reduction in patients that can tolerate arterial vasodilation. Sodium nitroprusside (starting at 0.5–1 **m**g/kg/min), hydralazine (0.5–2.0 mg/kg PO), or amlodipine (0.05–0.1 mg/kg PO) *Started at low dosage and titrated up hourly until clinical improvement and decrease of systolic blood pressure (5–10%).*
			<u>Chronic (home-based)</u> -Furosemide dosage increased as needed to decrease pulmonary edema or body cavity effusions, if use is not limited by renal dysfunction (should be monitored 12-48 hours after dosage increases) *Specific strategy and magnitude of dosage increase varied among panelists* -Spironolactone if not already started in stage C is indicated at this point -Beta blockade generally should NOT be initiated at this stage **<u>Home-based chronic dietary</u>** -Dietary considerations for stage C apply -Attempts to further decrease dietary sodium intake should be made if it can be done without compromising appetite or renal function