Self Assessment

Emily Van Wetter DVM
Nicole Van Israël DVM CESOpht CertSAM CertVC Dipl ECVIM-CA (Cardiology) MSc MRCVS
EUROPEAN SPECIALIST™ IN VETERINARY CARDIOLOGY
ANIMAL CARDIOPULMONARY CONSULTANCY (ACAPULCO) BELGIUM

CASE PRESENTATION

A two year old male entire Hovawarth was presented with a three week history of lethargy, reduced appetite and weight loss.

On clinical examination, the dog was found to be in poor body condition (body condition score 3/9). He seemed fully hydrated and his mucous membranes were pink with a capillary refill time of less than 2 seconds. His heart rate was 44 beats per minute (BPM) with a strong and very regular apex beat. His pulses were strong without any deficits. The examination of his jugular veins showed marked canon a-waves (Fig. 1). No murmurs or abnormal lung sounds were audible. Abdominal palpation was within normal limits. The remainder of the physical examination was unremarkable.

QUESTIONS

1. What are the main clinical abnormalities of this dog?
2. How would you further assess the rhythm disturbance?
3. What is your differential diagnosis?
4. What further investigations would you perform?
5. Which treatment would you propose?
6. What is the prognosis of this case?

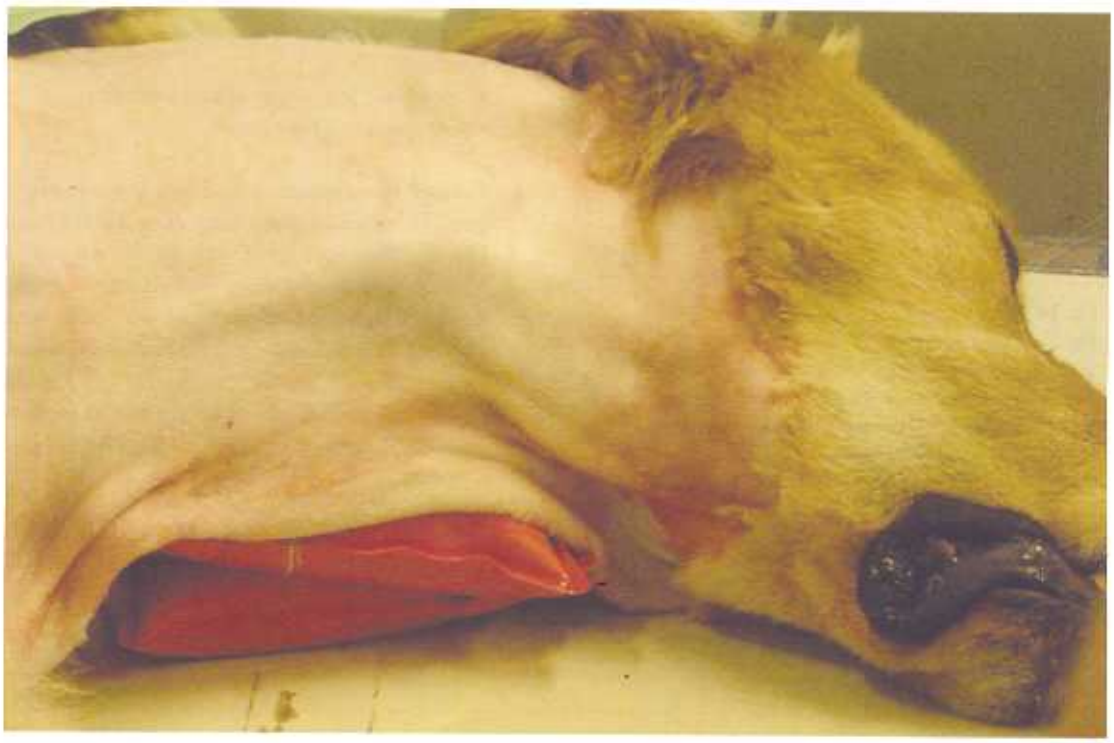

Fig. 1: *Jugular canon a-waves*

ANSWERS

1. This dog presented with a regular bradycardia. The two most likely causes of this bradycardia were either a reduced rate of sinus node firing (physiological or pathological) or reduced impulse conduction to the ventricles (third degree atrioventricular (AV) block). The jugular canon a-waves noted on clinical examination indicate the presence of a third degree AV block. These jugular pulses are caused by the back-flow of blood in the jugular veins when the right atrium contracts against a closed tricuspid valve (Fig. 2). Weight loss and reduced appetite were the other main clinical abnormalities in this case and were thought to be secondary to the reduced cardiac output caused by this dog's rhythm disturbance.

2. The investigation of choice to differentiate between the different causes of bradycardia is an electrocardiogram (ECG). The ECG of this dog (Fig. 3) shows a third degree AV block. There is no relationship between the P-waves (rate 140 BPM) and QRS complexes (rate 44 BPM) indicating a complete dissociation between atrial and ventricular electrical activity (AV dissociation). The ventricles depolarise according to a slow, regular, automatic rhythm called an escape rhythm. In this case, the escape rhythm is of left ventricular origin (wide negative QRS complexes in lead II, slow rate) and not junctional (narrow QRS complexes of nearly normal appearance, faster rate). This means that the block is localised lower than the AV node in the bundle branches. This is of importance for the aetiology and prognosis. Dogs with a junctional escape rhythm can show no clinical signs while dogs with ventricular escape rhythms almost always show clinical signs due to the very slow rate of their ventricular pacemakers. Classical clinical signs described include lethargy, reduced appetite, weight loss and syncopal events.

3. The differential diagnosis of a third degree AV block includes:
   - idiopathic third degree AV block
   - isolated congenital AV block
   - congenital defects: ventricular septal defect (VSD), atrial septal defect (ASD), atrioventricular canal defect
   - infiltrative disorders e.g. idiopathic fibrosis (often age-related), inflammation (Lyme disease myocarditis, *Toxoplasmosis myocarditis*, associated with endocarditis), amyloidosis or neoplasia
   - myocardial ischaemia
   - hyperkalaemia (e.g. secondary to hypo-adrenocorticism)
   - hypothyroidism
   - myasthenia gravis
   - systemic lupus erythematosus
   - trauma
   - iatrogenic (e.g. severe digoxin toxicity)
   - extreme vagal tone (rare)

4. Further investigations are aimed at excluding a possible 'treatable' underlying cause for the heart block. This consisted in this case of a haematology and serum biochemistry profile, serum electrolyte determination and total thyroxin and thyroid stimulating hormone assays. Echocardiography is indicated to assess myocardial and valvular function and to exclude congenital defects (unlikely in this case because

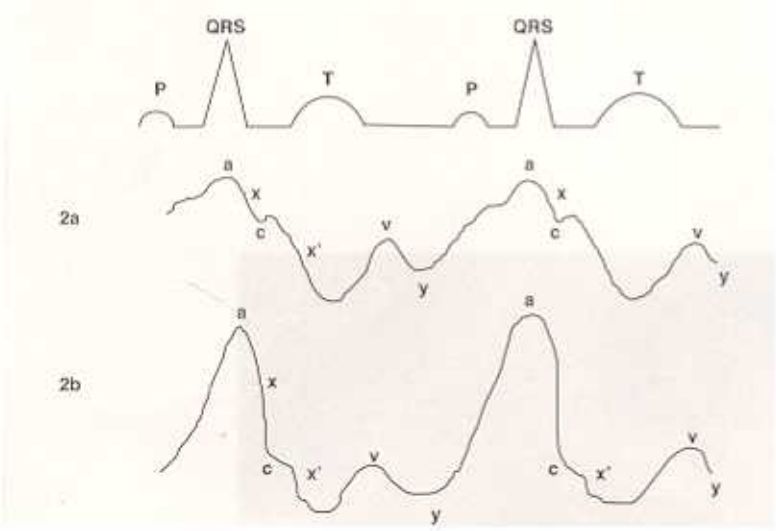

Fig. 2a: *Normal jugular pulse wave.*
Fig. 2b: *Canon a-waves; a: contraction of the atrium; x: relaxation of the atrium; c: closure of the tricuspid valve; x': the tricuspid valve annulus goes down during the ventricle systole; v: filling of the atrium; y: opening of the tricuspid valve and emptying of the atrium.*

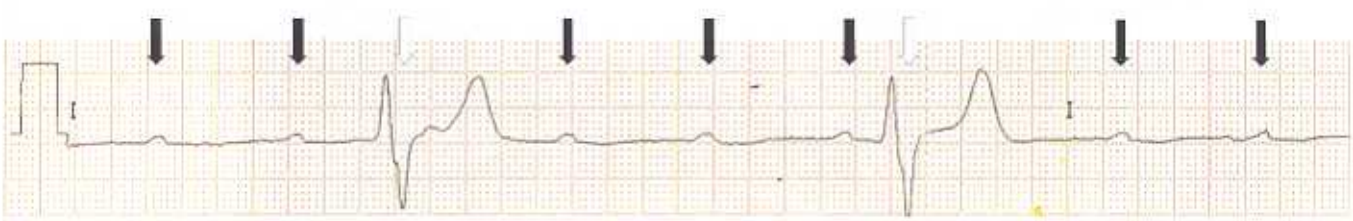

Fig. 3: *Electrocardiogram showing a third degree atrioventricular block with an atrial rhythm of 140 beats per minute (P-waves: black arrows) and an escape rhythm of 44 beats per minute (QRS-complexes: white arrows) (50mm/sec, 1cm/mV).*

of the absence of a murmur on auscultation). *Borrelia*, *Toxoplasma* and antinuclear antibody serologies, *Bartonella* PCR and haemoculture are indicated if there is a suspicion of an associated myocarditis. An ACTH stimulation test should also be performed to rule out hypo-adrenocorticoidism in cases with hyperkalaemia. All these tests were negative or normal in this dog. Thoracic radiographs showed a mild cardiomegaly of bradycardia but no signs of congestive heart failure. An atropine challenge test was also performed (0.02 mg/kg IV, in normal animals one can expect a 50-100% increase in heart rate depending on the initial heart rate). There was no heart rate modification after atropine administration in this dog.

After exclusion of most possible causes, the diagnosis of idiopathic third degree AV block was made.

5. The treatment of choice for third degree AV block is artificial pacemaker implantation. Medical treatment with parasympatholytics or sympathomimetics can be considered but is often not successful in the long term, particularly in cases with a negative atropine response test. The authors' preference for medical management is a slow-release preparation of theophylline (Corvental® 10-20 mg q 24h). Theophylline competitively inhibits phosphodiesterases (PDE) non-specifically, thereby increasing the intracellular levels of cyclic AMP (cAMP). Increased cAMP has a direct sympathetic effect by releasing epinephrine from adrenergic nerve terminals and the adrenal medulla and further contributes to an increase in intracellular calcium influx via phosphorylation of calcium channels. An increase in calcium influx in nodal tissue (sinus node and AV node) and myocardial tissue results in a positive chronotropic and inotropic effect respectively. However, this PDE mediated mechanism is controversial since theophylline

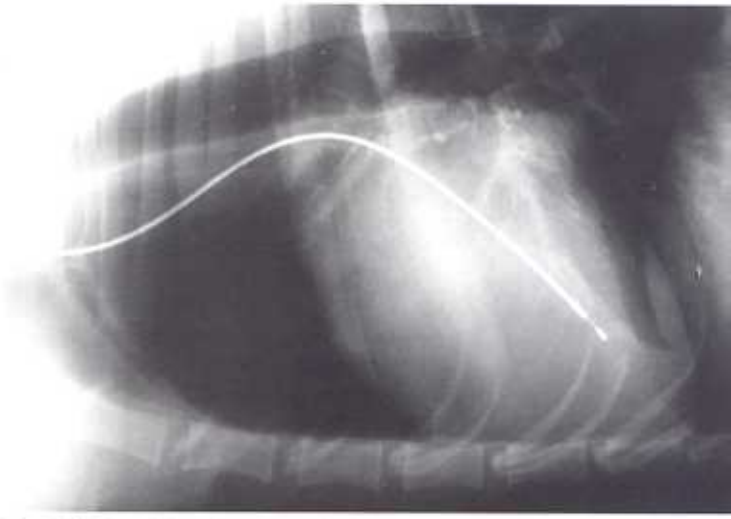

Fig. 4: *Right lateral thoracic radiograph after endovascular artificial pacemaker implantation showing the placement of the pacemaker lead in the right ventricle*

does not inhibit PDE at therapeutic concentrations. Figs. 4 and 5 show respectively a right lateral thoracic radiograph and an ECG tracing of the dog after surgery.

6. The prognosis after artificial pacemaker implantation is fair. Major complications include cardiac arrest during implantation, pacing lead dislodgement, generator failure and infection. Minor complications include seroma formation, muscle twitch when a unipolar pacing device is used and inconsequential arrhythmias.

The condition of this dog dramatically improved after pacemaker implantation and two years afterwards he is living a normal and very active life.

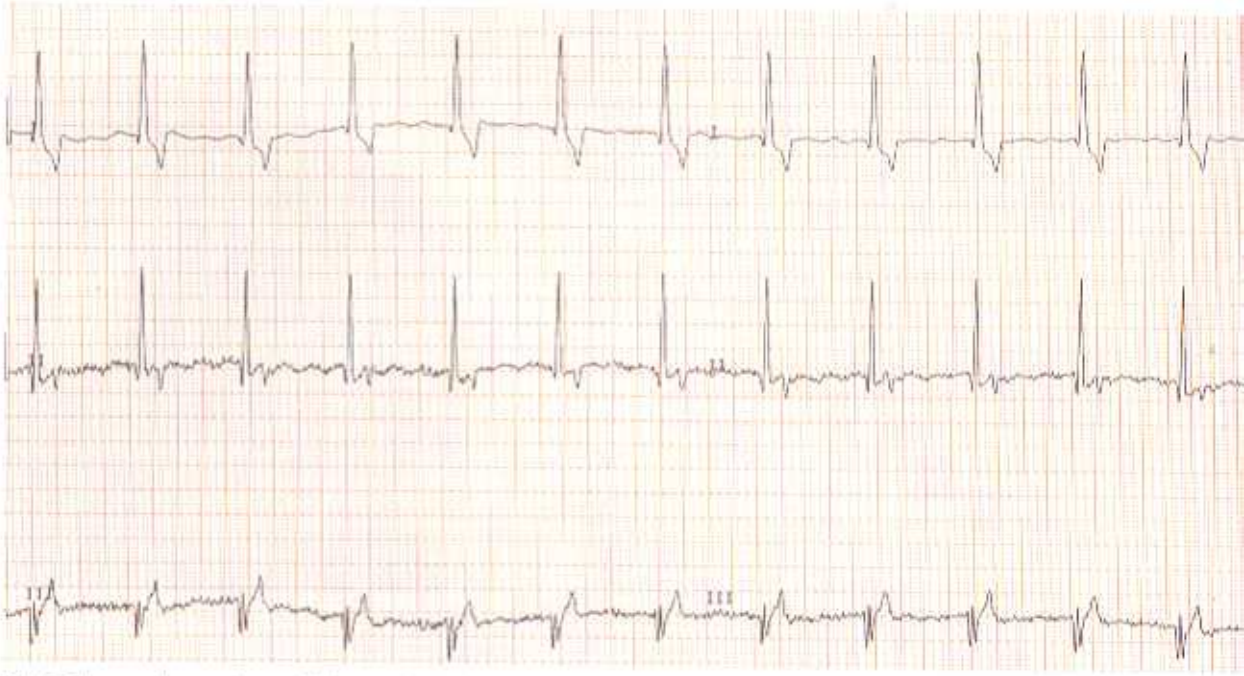

Fig. 5: *Electrocardiogram after artificial pacemaker implantation showing pacing spikes followed by QRS complexes at a rate of 70 beats per minute (50mm/sec, 1cm/mV)*