

Atropine responsive sinoatrial arrest in a Pug

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Abstract

A three-year-old male pug weighing 7.7 kg was presented to the Teaching Veterinary Clinical Complex, CVAS Pookode, Wayanad, with a history of inability to stand up for the last five days. Inappetence, cough, respiratory distress, lethargy and collapse were reported by the owner on detailed anamnesis. On physical examination, the animal was found to be weak and recumbent, with occasional episodes of syncope and cyanosis of the tongue. Arrhythmia was heard on cardiac auscultation. Electrocardiographic findings revealed irregular heartbeats with a pause, indicating the absence of P-QRS-T complexes, and the duration between the two complexes was exceedingly twice the previous P-P interval. The case was tentatively diagnosed as sinoatrial arrest. Laboratory investigations like haematology and serum biochemistry were found to be normal. Oxygen therapy was initiated as a primary treatment. Atropine sulphate at a dose rate of 0.04 mg/kg body weight, followed by adrenaline at a dose rate of 0.1 mg/kg body weight, was administered intravenously as an emergency treatment. Post-treatment electrocardiography revealed normal PQRST complex, and the case was confirmed as atropine responsive sinoatrial arrest.

Keywords: Adrenaline, Atropine responsive sinus arrest, Atropine, Sinoatrial arrest

Highlights

- A pug with clinical signs of respiratory distress, lethargy and collapse
- ECG findings indicating absence of P-QRS-T complexes
- Pause between two complexes exceeding twice the previous P-P interval
- The case responded to intravenous atropine administration at a dose rate of 0.04 mg/kg body weight.

Depolarisation of the heart is initiated in the sinus node (SA node), which sets the normal rate and rhythm, and hence it is called the pacemaker of the heart. It is indicated on an electrocardiograph by P wave followed by QRS complex. The rate of firing from SA node may vary from species to species and situation to situation.

Sinus arrest and blocking are impulse conduction disorders of SA nodes in dogs. They can be defined as the failure of an impulse to be formed within the sinoatrial node (SA node) due to the depression of nodal automaticity (Tilley, 1992). They are the most common manifestation of end-stage sinus node disease in canine patients (Hamlin *et al.*, 1972) and are uncommonly associated with myocarditis (Taylor and Sittnikow, 1968). The failure to discharge the impulses from the SA node at the correct time can cause fainting or even sudden death due to ventricular asystole. In brachycephalic dog breeds, sinus arrest may be found due to increased vagal tone during inspiration (Manoj *et al.*, 2023).

Sinus arrest can be considered an

electrocardiographic (ECG) abnormality with no heartbeats and the absence of P-QRS-T complexes. This may be due to a temporary failure of the sinoatrial node in electrical impulse production or conduction failure. The sinus arrest is characterized by the absence of p waves on ECG with a pause exceeding twice the normal P-P interval (Kumar *et al.*, 2011). Long periods of sinus arrest are usually terminated by a junctional or ventricular escape complex. Sinus block is also on ECG as there are no P waves or QRS complexes, but the pause is exactly twice the previous P-P interval (Scales, 2023).

The main aetiologies are atrial dilatation, infiltration or fibrosis, mitral valvular regurgitation (Woolley *et al.*, 2007), excessive vagal tone, drugs and electrolyte disturbances, especially hyperkalaemia (Kumar *et al.*, 2011). Weakness or fainting due to cerebral hypoxia is a major sign of sinus arrest and can be treated with an antimuscarinic agent, atropine sulfate and an artificial pacemaker if appropriate. Hence, the atropine test is validated in the confirmatory diagnosis of sinoatrial block or arrest

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in canine patients (Kumar *et al.*, 2011).

A 3-year-old pug was presented with a history of general weakness, inability to bear weight, inappetence, cough, respiratory distress, episodes of collapse, cyanosis of the tongue and lethargy (Fig. 1). On physical examination, the mucous membrane was congested and capillary refill time was more than two seconds. Rectal temperature was 100.4°F, and respiratory and heart rates were 20/min and 78 bpm, respectively. On detailed cardiac auscultation, heartbeats were found to be interrupted by a long pause after a few normal beats, giving an indication of cardiac abnormality.

On Electrocardiographic examination (ECG), heart rhythm was found to be irregular with a pause indicated by the absence of P-QRST complexes. The pause was greater than twice the previous normal R-R interval or P-P interval. On the basis of ECG findings, the case was tentatively diagnosed as sinus arrest (Fig. 2). The haematological findings were found within the normal reference range (Table 1). On serum biochemical analysis, sodium (142 mEq/L), potassium (4.9 mEq/L), BUN (24.4 mg/dL), creatinine (0.9 mg/dL), creatine kinase (84 IU/L) and ALT (26 IU/L) were found within normal reference limits.



Fig. 1. Animal with clinical signs

Table 1. Haematological parameters

Parameters	Results
WBC ($10^3/\mu\text{L}$)	10.0
Lymphocytes ($10^3/\mu\text{L}$)	2.20
Monocytes ($10^3/\mu\text{L}$)	0.50
Granulocytes ($10^3/\mu\text{L}$)	11.9
RBC ($10^6/\mu\text{L}$)	7.31
Hb (g/dL)	15.8
HCT (%)	49.9
MCV (fL)	68.3
MCH (pg)	21.6
MCHC (g/dL)	35.0
RDW (%)	13.4
PLT ($10^3/\mu\text{L}$)	312
Eosinophil (%)	1.7



Fig. 2. Electrocardiographic findings indicating sinus arrest

Radiographic (Fig. 3 and Fig. 4) and echocardiographic evaluations were also carried out (Fig. 5) and on lateral thoracic radiography, generalized cardiac enlargement was noticed. Vertebral heart score (VHS) was 12.6. Mitral regurgitation, thickened mitral valvular leaflets, and aortic regurgitation could be observed on Doppler echocardiography. E-point septal separation (EPSS) value of 0.75 cm (Fig. 6) and ventricular hypertrophy were also noticed, suggestive of mitral valve dysfunction and cardiac compensation.



Fig. 3. Lateral thoracic radiography indicating tracheal elevation and cardiac enlargement



Fig. 4. Dorsoventral radiography indicating left ventricular enlargement

The treatment was initiated with oxygen therapy. Atropine sulphate at a dose rate of 0.04 mg/kg body weight, followed by adrenaline at a dose rate of 0.1 mg/kg body weight, was administered intravenously as an emergency treatment. Post-treatment ECG findings were taken into account once the animal attained stability after the treatment (Fig. 7) and was

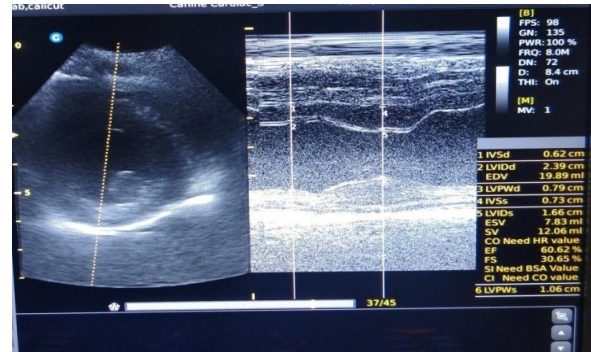


Fig. 5. Echocardiographic findings

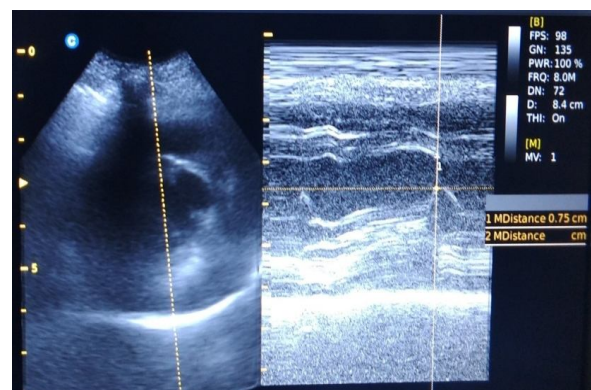


Fig. 6. Echocardiographic findings showing enhanced EPSS

found to be normal. Oral supplementation with Tab. enalapril maleate (Dilvas^R, 5 mg) at a dose rate of 0.25 mg/kg body weight twice daily, Tab. Cardiosupport^R and Nutrolin-B^R suspension were advised for 30 days. The animal started responding well to the therapy and was advised to review after a month.

Sinus arrest may happen due to neoplasm, accidental injuries, atrial pathology (dilatation, fibrosis and cardiomyopathy), excessive vagal tone, sick sinus syndrome (Crooks *et al.*, 2021), atrial myocarditis (Woolley *et al.*, 2007), electrolyte disturbances particularly hyperkalaemia, and drug induced toxicities especially with digitalis, beta-blocker, quinidine and calcium channel blockers (Santilli *et al.*, 2019). Exaggerated sinus arrhythmia in brachycephalic breeds causes long sinus pauses, especially during sleep.

There are reports on the atrioventricular block (AV block) in association with sinus arrest (Ogawa *et al.*, 2022). In this study, there was no AV block, and the P-R interval was found to be normal, suggesting normal anatomy of Bundle of His. Atropine sulphate, glycopyrrolate, isoproterenol, terbutaline, cilastazol (Ohmori *et al.*, 2022) and artificial pacemakers are indicated in the management of sinus arrest due to the



Fig. 7. Post-treatment ECG findings

reduced automaticity of the SA node.

Differentiation of sinus arrest from second-degree AV block is difficult on surface electrocardiograms because of the presence of prolonged pauses in the electrical activity of the heart. The prolonged duration of sinus arrest without evidence of escape beats was also suggestive of the pathology of additional areas of the conduction system. Normal subsidiary pacemakers, such as the atrioventricular node or Purkinje fibres,

should discharge at a rate of between 20 and 60 bpm in the presence of sinus arrest.

Conflict of interest: Authors have no conflict of interest in this study.

Author's contribution: STM: Carried out diagnosis of the case and clinical management. Involved in draft of the manuscript; VDP: Carried out diagnosis of the case and clinical management. Involved in draft and revision of the manuscript; SOK: Involved in draft and revision of the manuscript.

Data availability statement: All the data supporting the research findings have been presented in this paper. The corresponding author is willing to provide the raw data upon reasonable request.

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