



## COMPLETE AV BLOCK AND IDIOVENTRICULAR CARDIAC RHYTHM IN A DOG DURING 60-SECOND ECG RECORD

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### Summary

Rusenov, A. G., L. V. Lazarov & Ts. T. Hristov, 2025. Complete AV block and idioventricular cardiac rhythm in a dog during 60-second ECG record. *Bulg. J. Vet. Med.* (online first).

The present report describes a clinical case of a dog with severe cardiac arrhythmia. The alternating pattern of idioventricular rhythm with low-frequency ventricular extrasystoles and third-degree AV block in rhythm mode (60-second record with bipolar lead II) is a concurrent arrhythmia associated with disturbances in cardiac excitation and conduction. The case is interesting because ventricular escape complexes during the third-degree AV block occurred at a low rate – 27 beats per minute (bpm), whereas ventricular escape complexes during the idioventricular rhythm: at a higher rate (53 bpm), which occurs more rarely. Conservative treatment failed to produce an adequate effect. Therefore, pacemaker implantation was scheduled, but the dog died several days before the surgery. Pacemaker implantation is urgently important as any delay may result in cardiac death.

**Key words:** arrhythmia, cardiac death, dogs, ECG, third-degree AV block

The atrioventricular block (AVB) is a conduction abnormality along the atrioventricular node, which may result in various clinical outcomes depending on its aetiology and grade (Santilli *et al.*, 2016). Complete atrioventricular (AV) block occurs when AV conduction is completely and permanently absent, with no relationship between P waves and QRS complexes (complete AV dissociation) confirmed by electrocardiography. The depolarisation of ventricles is autonomic under the control of subsidiary pacemakers below the AV area. The slow regular ven-

tricular rhythm is called escape rhythm (Kaneshige *et al.*, 2007a; Machen *et al.*, 2008; Toaldo *et al.*, 2017). The ventricular contraction rate is considerably slower than that of atrial contraction because atria are depolarised by the sinus node. The most common clinical signs are rapid exhaustion, lethargy, syncope, congestive heart failure and sudden death resulting from decreased cardiac output and ventricular asystole (Kittleson, 1998; Miller *et al.*, 1999; Ettinger *et al.*, 2000; Kaneshige *et al.*, 2007b).

Arrhythmias associated to ventricular premature complexes (VPC) are classified into three groups depending on heart rate (HR). A series of three or more VPC that occur at a very high HR (HR > 160 beats per minute; bpm) is defined as ventricular tachycardia (VT), whereas accelerated idioventricular rhythm (AIVR) is present at a HR < 160 bpm. The third variant, when HR < 60 bpm, is the idioventricular rhythm (Santilli *et al.*, 2018; Santilli *et al.*, 2021).

The present report describes a clinical case of heart arrhythmia in a dog, in which a complete AV block at a low rate (27 bpm) of ventricular escape complexes alternated with an idioventricular rhythm of ventricular escape complexes at a higher rate (53 bpm), which is a less frequent and more unusual pattern.

#### *Case presentation*

A 12-year-old male mixed-breed dog, weighing 23 kg, was admitted to the Small Animal Clinic of the University Veterinary Hospital in June 2021. The dog was privately owned and lived freely in a mountain hut. Three months ago, the animal had experienced the first clinical signs. According to the owner's description, the clinical signs corresponded to those of cardiac syncope. Since that time, the dog had demonstrated a general weakness, had become lethargic, with selective appetite and more frequent syncope episodes. Two weeks before the referral, the dog could hardly stand up and stay upright.

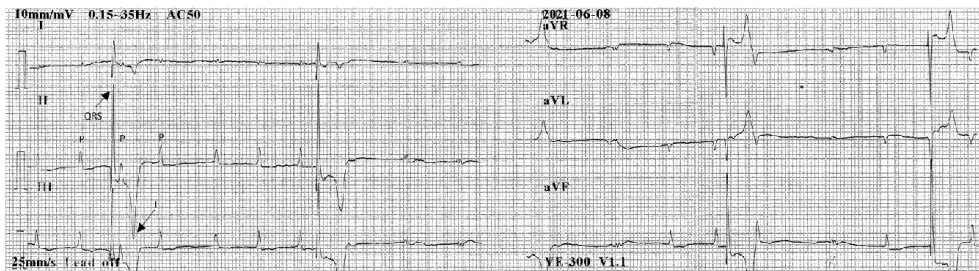
The physical examination revealed a medium body structure and relatively good body condition for the patient's age. The general condition indicated visible discomfort with apathy and lethargy. In standing position, the dog experienced multiple episodes of sharp onset of

tachypnea and falling down in lateral recumbency with hindlimb weakness, stretching and stiffening of forelimbs. The head and neck were in opisthotonus position, with obscured consciousness and diminished and slower reflexes.

The main vital parameters were: rectal body temperature of 38.3 °C, heart rate of 35 beats per minute (bpm) and 32 respirations per minute (rpm). The visible mucosae (conjunctivae) were slightly pale, and the capillary refill time was within 4–5 seconds. The pulse was strong, full and rhythmic. Heart auscultation demonstrated a louder first heart sound and a substantially weaker second heart sound. In general, the heart sounds were smooth, continuous and clean. The simultaneous examination of heart sounds and arterial pulse did not show any pulse deficit. The chest auscultation showed no pathological breathing sounds.

ECG record was done in standard peripheral leads in auto mode (5-second record in leads I, II, III, aVR, aVL and aVF; Fig. 1) and rhythm mode (60-second record in lead II; Fig. 2). The paper speed was 25 mm/s, and the calibration voltage of the unit was 10 mm/1 mV. The rate of QRS complexes was calculated arithmetically; in auto mode it was 27 bpm, and that of P-waves, 125 (i.e. nine P-waves were registered vs only two QRS complexes). The AV conduction was entirely and consistently interrupted. A third-degree AV block was registered on the basis of presence of P-waves without QRS complexes, lack of constant PQ interval and slow ventricular rhythm of QRS complexes. There was a clear atrial P peak between the first QRS complex and the negative T wave. The cardiac electrical axis was 84° (Bailey's hexaxial system).

In rhythm mode (60-s record in second bipolar lead), a complex alternating pat-



**Fig. 1.** Third-degree atrioventricular block - auto mode ECG (5-second record in leads I, II, III, aVR, aVL and aVF). Complete AV dissociation: lack of association between atria (P waves) and ventricles (QRS complexes). Slow ventricular QRS complexes (escape rhythm).

tern of idioventricular rhythm of ventricular extrasystoles at a low rate (53 bpm) and third-degree AV block were observed. In the idioventricular rhythm interval, there was an atrioventricular dissociation (lack of association between P-waves and QRS complexes), and part of the P-waves were “buried” by ventricular complexes. Ventricular extrasystoles appeared as wide bizarre QRS complexes with duration of 0.12 seconds (Fig. 2); in most instances they were not preceded by P-waves, but followed by T-waves. The morphology of ventricular extrasystoles (in the idioventricular rhythm zone) was different from QRS complexes in the third-degree AV block zone.

The lateral chest radiography revealed a moderate cardiomegaly. The vertebral heart score (VHS), i.e. the sum of the long and short heart axes, was increased to 11.5 vertebrae (reference values from 8.5 to 10.5) (Ware & Ward, 2020). Neither dilation of pulmonary veins nor changes in the position and cross-section of the trachea were detected. The radiological lung pattern was normal broncho-interstitial and alveolar.

Echocardiographic measurements were done in the right parasternal short axis view with transventricular, transmitral and transaortic transducer position in B and M modes. The left ventricular internal-end

diastolic (LVIDd, wLVIDd) and end-systolic (LVIDs, wLVIDs) diameters, the fractional shortening (FS), the EPSS and the left atrial-to-aortic ratio (LA/Ao) were determined: LVIDd – 3.99 cm (wLVIDd – 1.765), LVIDs – 2.58 cm (wLVIDs – 1.141), FS – 35.4%, EPSS – 1.18 cm, and LA/Ao: 1.78.

The analysis of total and differential white blood cell counts (Table 1) demonstrated moderate leukopaenia with lymphocytosis. Blood biochemical analysis (Table 2) revealed mild hyperphosphataemia, prerenal azotaemia (increased creatinine concentration) and bilirubinaemia, as well as multifold increase in ALT activity.

Intravenous dobutamine (a sympathomimetic drug) was prescribed at a dose of 5 µg/kg/min dissolved in 5% dextrose in order to influence the idioventricular rhythm. The applied beta-agonist did not result in a steady positive therapeutic response, and the syncope episodes of the dog persisted. On the next day, atropine response test was carried out to evaluate the vagal effect on bradyarrhythmia. The intravenous administration of 0.04 mg/kg atropine sulphate (Atropine; Sopharma, Sofia, Bulgaria) did not alter the rate of ventricular complexes.

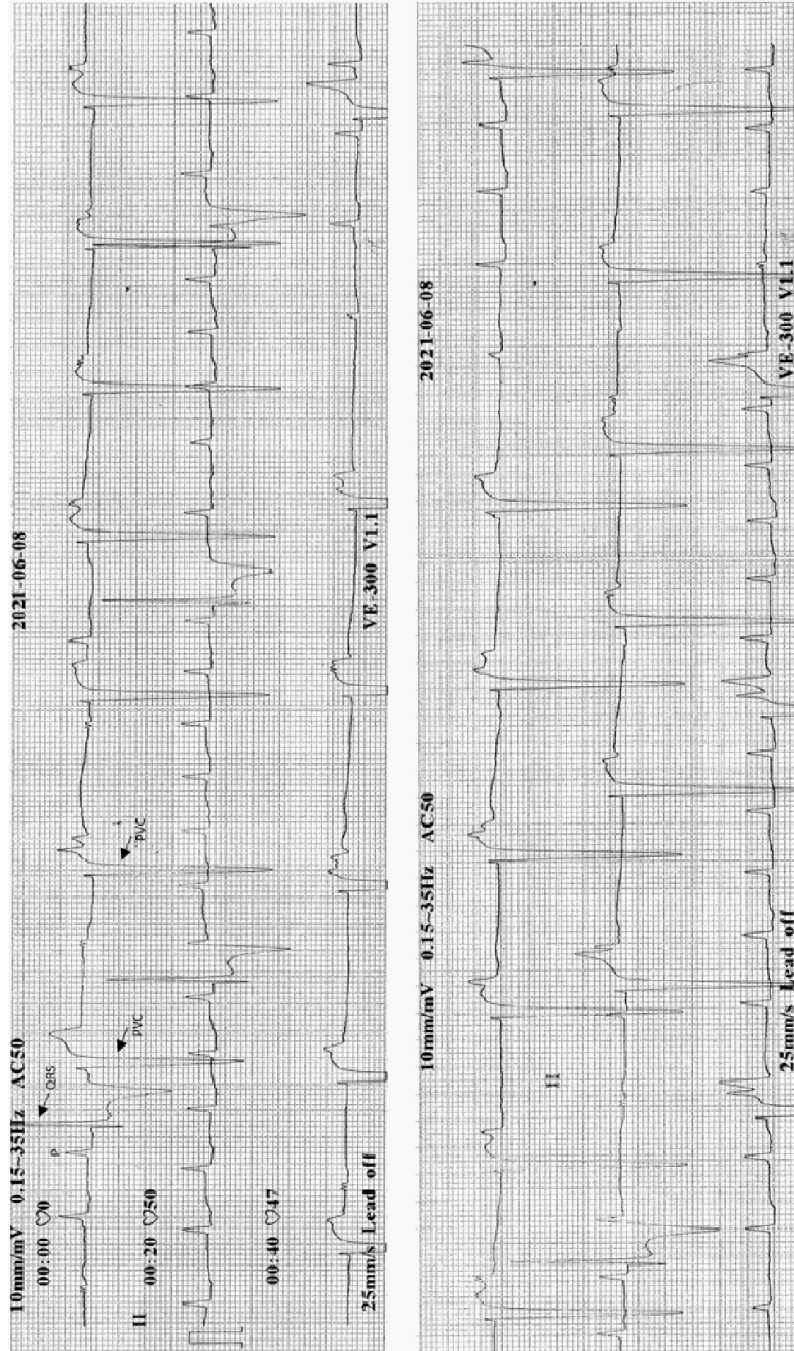


Fig. 2. Alternating pattern of third-degree AV block with idioventricular rhythm - rhythm mode ECG (60-second record in bipolar lead II): bizarre ventricular QRS complexes, without P waves at a slow rate.

Because conservative therapy failed to produce an adequate therapeutic effect and the frequency of syncopal episodes increased, a procedure for pacemaker implantation was scheduled. However, several days later, the dog was found dead nearby the mountain hut. Necropsy was declined by the owner.

**Table 1.** Red and white blood cell counts

Parameter	Result	Reference*
WBC, G/L	4.0	5.0–14.1
Lymph, G/L	3.0	0.4–2.9
Mon, G/L	0.3	0.1–1.4
Gran, G/L	0.7	2.9–12.0
Lymph %	75.5	8.0–21.0
Mon %	7.7	2.0–10.0
Gran %	16.6	58.0–85.0
RBC, T/L	6.85	4.95–7.87
HGB, g/L	163	119–189
HCT %	47.8	35–57
PLT, G/L	267	211–621

\* Fielder (2024a).

**Table 2.** Blood biochemical profile

Parameter	Result	Reference*
TP, g/L	65.7	54–75
ALB, g/L	31.9	23–31
GLO, g/L	33.8	27–44
Ca, mmol/L	2.57	2.3–2.9
P, mmol/L	2.08	0.9–1.7
GLU, mmol/L	5.57	4.2–6.6
BUN, mmol/L	6.94	2.9–10
Amylase, U/L	568	226–1063
CHOL, mmol/L	7.05	3.5–7.2
ALT, mmol/L	524	10–109
TBIL, $\mu$ mol/l	11.23	0–5.1
ALP, U/L	96	1–114
CRE, $\mu$ mol/l	161	44–150
CK, U/L	218	42–368
K, mmol/L	4.8	3.9–5.1
Na, mmol/L	144	142–152
cTnI, ng/mL	0.08	0.00–0.11

\* Fielder (2024b).

The third-degree AV block diagnosed in auto recording mode is a severe arrhythmia associated with impaired cardiac conduction. In one-third of cases, it results in neuron-mediated syncope manifested as episodes of transient conscience loss (Martin & Corcoran, 1997; Perego *et al.*, 2020). This arrhythmia is characterised with complete and permanent interruption of the impulse at the AV node level. Ventricular depolarisation is autonomic, with slow, regular escape rhythm from the AV node (Martin & Corcoran, 1997; Kane-shige *et al.*, 2007a; Machen *et al.*, 2008; Toaldo *et al.*, 2017). The idioventricular rhythm of low-frequency ventricular extrasystoles (53 bpm) alternating with third-degree AV block is a concurrent arrhythmia associated with disturbances in both cardiac excitation and conduction (Martin & Corcoran, 1997).

In general, the escape rhythm arises in the AV node (junctional escape rhythm) but it may also originate from ventricles (ventricular escape rhythm). The pattern of junctional escape systoles partly corresponds to the normal reference range (junctional ectopy), whereas ventricular escape systoles show an abnormal and bizarre shape (ventricular ectopy) (Gallay *et al.*, 2011). The rhythm with continuous junctional escape systole (from the AV node) occurs at a heart rate of 60–70 bpm, whereas the rhythm with continuous ventricular escape systole occurs at a rate of less than 40 bpm. Both may be seen in complete AV block, and this rhythm is known as escape rhythm (Martin & Corcoran, 1997; Martin *et al.*, 2009; Toaldo *et al.*, 2017). In the presented clinical case, the patient's ECG demonstrated ventricular escape complexes; their morphology was different due to their different ectopic origin (Martin & Corcoran, 1997). An interesting finding was that the mor-

phology of the ventricular escape complexes during the third-degree AV block was close to normal but their rate was low (27 bpm). During the idioventricular rhythm, there were ventricular escape complexes, abnormal and of bizarre shape, yet at a higher rate (53 bpm).

The causes for the complete (AV) block may be functional or, more often, structural damage. Functional causes include hyperkalemia, digitalis toxicity, treatment with beta-adrenergic blockers or calcium channel blockers. Structural damage may be congenital heart defects (including aortic stenosis and ventricular septal defect), inflammation (bacterial endocarditis, borreliosis, Lyme carditis, traumatic myocarditis), endocrine disorders (hypothyroidism), heart base neoplasms (e.g. in the aortic region) or degenerative changes (physical damage derived from cardiomyopathy, endocardiosis or fibrosis) (Martin & Corcoran, 1997; Panciera, 2001; Kaneshige *et al.*, 2007a; Kaneshige *et al.*, 2007b; Schuller *et al.*, 2007; Gallay *et al.*, 2011; Stern *et al.*, 2012; Toaldo *et al.*, 2017; Romito *et al.*, 2021). Nevertheless, the primary cause of the AV block in most cases remains unclear (Schuller *et al.*, 2007; Kittleson & Kienle, 1998).

FS (35.4%), LVIDd (wLVIDd) and LVIDs (wLVIDs) were within the reference range for dogs with similar body weight (Brown *et al.*, 2003; Cornell *et al.*, 2004). These parameters may be associated with degenerative mitral valve disease (DMVD), but are not compliant with dilated cardiomyopathy (DCM) (Brown *et al.*, 2008). The high left atrial-to-aortic ratio (over 1.5) was also suggestive of DMVD. On the other hand, the EPSS increase over 0.8 cm in dogs (1.18 cm in the present case) is one of the mandatory echocardiographic signs of heart dilation

(Broschk & Distl, 2005). In our belief, the cause of heart dilation during a complete AV block (bradycardiomyopathy) was most probably bradyarrhythmia (Schuller *et al.*, 2007).

The results from the blood biochemical analysis were non-specific. Normal sodium, potassium, and cTnI values excluded a large group of causes leading to such arrhythmia. The observed prerenal azotaemia and increased activities of some liver enzymes (ALT) may be attributed to renal and liver hypoxia and congestion secondary to bradyarrhythmia (Glinska *et al.*, 2005; Schuller *et al.*, 2007).

In this clinical case, there was neither DCM nor congestive heart failure, digitalis toxicity, endocarditis, myocarditis and potassium disequilibrium. Thus, the cause of the severe arrhythmia remained unclear (idiopathic). We hypothesise that it was probably due to AV node fibrosis from organic heart damage at an early age. Our hypothesis is supported by a previous study that outlined lymphocytic myocarditis as an aetiological cause of complete AV block in 12 dogs (Sasaki *et al.*, 2020). The authors described intense infiltration of mononuclear cells, primarily lymphoid ones in AV structures, along with associated degeneration and necrosis of the adjacent cardiomyocytes, as well as replacement fibrosis. They suggested that such total or subtotal destruction of the AV conduction system has set the stage for blocking AV conduction of cardiac impulses.

To exclude the possibility about vagal origin of the patient's complete AV block, an atropine response test was performed. Following injection of atropine, dogs with vagally mediated AV block are expected to demonstrate increased frequency of P waves followed by improved AV conduction and normal sinus rhythm (Schuller *et*

*al.*, 2007). In this patient, the lack of improvement of AV conduction after the atropine injection suggested that increased vagal tone was probably not the cause of the AV block.

The most appropriate therapeutic approach in dogs with clinically manifested advanced second-degree AV block and all dogs with third-degree AV block is pacemaker implantation, as antiarrhythmic therapy in such cases is contraindicated (Martin & Corcoran, 1997; Schrope & Kelch, 2006; Johnson *et al.*, 2007). According to the authors, pacemaker implantation was successful in reduction or elimination of clinical signs in more than 90% of dogs with complete atrioventricular (AV) block, and this correlated strongly and positively with survival. Pacemaker implantation in dogs with third-degree AV block should not be deemed an ultimate solution, as sometimes it may progress to more severe pathology (Santilli *et al.*, 2016).

This case report has some limitations; for example, the absence of the full set of examinations. Despite the limitations, it clearly demonstrates the urgency of the pacemaker implantation procedure. The case is interesting because ventricular escape complexes (in the third-degree AV block interval) occurred at a low rate (27 bpm), whereas ventricular escape complexes (in the idioventricular rhythm interval), at a higher rate (53 bpm), which happens more rarely.

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Paper received 19.05.2025; accepted for publication 13.10.2025

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