

Isorhythmic Atrioventricular Dissociation in Labrador Retrievers

M. Perego, L. Ramera, and R.A. Santilli

Background: Isorhythmic atrioventricular dissociation (IAVD) is a rhythm disturbance in which atria and ventricles are driven by independent pacemakers at equal or nearly equal rates.

Objectives: To describe electrocardiographic and electrophysiologic features of IAVD in a group of 11 Labrador Retrievers and its possible correlation with focal junctional tachycardia (FJT).

Animals: Between December 2004 and October 2010, medical records of 11 Labrador Retrievers with surface electrocardiographic findings compatible with IAVD were retrospectively analyzed.

Methods: Twelve-lead surface electrocardiograms, thoracic radiographs, and echocardiographic findings of each dog and electrophysiologic mapping results of 3 dogs were retrospectively analyzed.

Results: In 10 of 11 dogs, the ECG pattern revealed the presence of IAVD with type I synchronization. In 5 of 10 dogs, IAVD with type I synchronization was interrupted by periods of junctional tachycardia with 1 : 1 ventriculo-atrial conduction. One of 11 dogs presented IAVD with type II synchronization. The ECG diagnosis of IAVD with type I and type II synchronization, and junctional rhythm with 1 : 1 ventriculo-atrial conduction was confirmed in 3 of 11 dogs with endocardial mapping in which the diagnosis of focal junctional tachycardia was made.

Conclusion and Clinical Importance: IAVD with type I synchronization is more common than IAVD with type II synchronization in Labrador Retrievers, and a correlation between IAVD and FJT can be hypothesized.

Key words: Arrhythmias; Cardiology; Dog; Focal junctional tachycardia; Isorhythmic atrioventricular dissociation.

Introduction

Isorhythmic atrioventricular dissociation (IAVD) is a rhythm disturbance in which atria and ventricles are driven by independent pacemakers at equal or nearly equal rates. A peculiar aspect of this arrhythmia is the apparent synchronization on the surface ECG of these 2 independent pacemakers over relatively long periods, in spite of different influences acting on both.¹ In 1946, Segers et al attempted to explain such synchronization on the basis of experimental models represented by isolated frog heart segments. These studies demonstrated that when separate and distinct cellular elements having no anatomic continuity and possessing inherently different rhythms are placed in contact with each other, they sometimes begin to discharge impulses simultaneously at a common rate. Furthermore, they showed that when 2 independent foci of impulse formation exist side by side in a biologic medium, they can interact with each other under certain circumstances in such a way that the focus with the lower rate of discharge accelerates to approximate that with the higher frequency.^{2,3} When this occurs, it is logically called synchronization.^{2,3} If the “hook-up” between cardiac sources is short-lived, lasting for only a few beats, after which the separate elements resume their

Abbreviations:

AH interval	atrium-His bundle interval
ECG	electrocardiogram
EPS	electrophysiologic study
FJT	focal junctional tachycardia
HV interval	His bundle-ventricular interval
IAVD	isorhythmic atrioventricular dissociation

original independent rhythms, Segers proposed the term “accrochage”.^{4,5} This term derives from the French word accrocher (to hook together) and it is related to relationship between the P waves and the QRS complex.^{4,5} The term synchronization refers to a longer period during which the 2 independent rhythms maintain the same rate.^{2–5} It must be emphasized that isorhythmic atrioventricular dissociation does not only mean the synchronous or near-synchronous contraction of atrial and ventricular myocardium, but the essence of synchronization is that atria and ventricles respond to their own separate pacemakers, which are, however, beating synchronously.⁵

During IAVD, P waves and QRS complexes are dissociated, and their relationship appears to fall into 2 distinct patterns. Isorhythmic atrioventricular dissociation with type I synchronization is characterized by a rhythmic fluctuation of the interval between the P waves and the QRS complexes, most often with the P waves oscillating gradually back and forth across the QRS complexes with periodically varying PQ interval. Typically, the P wave never moves through the S-T segment and T wave toward the subsequent QRS complex because the variation of rate between the 2 pacemakers is always very small. In IAVD with type II synchronization, the P waves and QRS complexes are in a relatively fixed position with respect to each other.⁶ Numerous theories have been proposed to explain the interaction between atria and

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ventricles in man and dog during atrioventricular block, isorhythmic atrioventricular dissociation, and ventriculophasic sinus arrhythmia. For the most part, these theories considered mechanical effect, reflex mechanism, and electrotonic influence of stroke volume.⁵⁻¹¹ Because there are only 2 reports in the veterinary literature describing IAVD with accochage in a dog with ruptured chordae tendineae¹² and in a dog with a suspected atrioventricular junctional tachycardia producing atrioventricular dissociation,¹³ the aim of this study was to describe the electrocardiographic and electrophysiologic features of IAVD in a group of Labrador Retrievers and its possible correlation with focal junctional tachycardia (FJT) in this breed.

Materials and Methods

Medical records of 11 Labrador Retrievers with electrocardiographic findings compatible with IAVD, which underwent diagnostic evaluation at the Clinica Veterinaria Malpensa between December 2004 and October 2010, were retrospectively reviewed by 1 author (RAS). The study group included 10 males and 1 female with a median age of 7 years (interquartile range: 4.5 years) and a median body weight of 33 kg (interquartile range: 7 kg). Each dog underwent physical examination, 12-lead surface electrocardiogram (ECG) in right lateral recumbency with a previously described precordial lead system¹⁴ thoracic radiographs, and standard transthoracic echocardiography to evaluate underlying structural cardiac diseases. Electrophysiologic studies (EPS) were conducted in 3 of 11 dogs. The following surface ECG variables were analyzed during tachycardia: (1) heart rate, (2) QRS complex duration, (3) R-R interval regularity and duration, (4) P-P interval regularity and duration, (5) presence of IAVD with type I synchronization described as rhythmic fluctuation of the interval between the P waves and the QRS complexes, (6) presence of IAVD with type II synchronization described as the presence of P waves and QRS complexes in a relatively fixed position with respect to each other, (7) presence of periods of junctional rhythm with retrograde atrioventricular conduction identified by the presence of P' waves characterized by negative polarity in inferior leads (II, III and aVF) and positive polarity in aVR and aVL with similar voltage, (8) PQ interval duration, and (9) RP' interval duration.

Electrophysiologic studies were performed after premedication with midazolam^a 0.2 mg/kg IV, induction of general anesthesia with propofol^b 4 mg/kg IV bolus and maintenance with isoflurane^c (1-2%) and oxygen (100%). Dogs were placed in dorsal recumbency, and venous accesses were obtained using the modified Seldinger technique. Under fluoroscopic and intracardiac ECG guidance, a decapolar electrode catheter was inserted through the right external jugular vein into the coronary sinus. A quadripolar electrode catheter was inserted through the right femoral vein and placed near the His bundle. Several electrophysiologic features have been considered suggestive for the diagnosis of FJT: (1) narrow QRS complexes and normal ventricular activation originating from the His bundle, (2) His bundle electrograms always preceding the ventricular electrogram with an HV interval compatible with antegrade conduction, (3) evidence of atrio-ventricular dissociation (variable AH interval) when sinus rate and FJT rate are similar, (4) evidence of concentric retrograde atrial activation pattern when FJT rate exceeds the sinus rate.

Numerical data are presented in median and interquartile range and categorical data in percentage.

Results

Four dogs presented without clinical signs, 2 dogs presented with episodic weakness, and 5 dogs with clinical signs of left-sided congestive heart failure. Radiographic and echocardiographic findings were unremarkable at presentation in 5 dogs. In 1 dog, radiographic and echocardiographic findings included biatrial and biventricular enlargement with generalized left ventricular hypokinesia without evidence of congestive heart failure and in 5 dogs severe biatrial and biventricular enlargement with generalized left ventricular hypokinesia and evidence of left-sided congestive heart failure were observed.

Electrocardiographic Analysis

Median heart rate was 140 bpm (interquartile range: 9.5 bpm), and QRS complex duration was normal (median: 52 ms, interquartile range: 6 ms) with a regular ventricular cycle duration of 420 ms (interquartile range: 32.5 ms). In 10 of 11 dogs (90.9%), the ECG pattern revealed the presence of IAVD with type I synchronization characterized by P waves with an electrical axis compatible with an origin in the sinus node and rhythmic fluctuation of the interval between the P waves and the QRS complexes and with P waves oscillating gradually back and forth across the QRS complexes (Fig 1). With shorter P-P intervals, the PQ intervals were also reduced in duration and the P waves approached the QRS complexes, became superimposed on the QRS complexes, and finally appeared after the QRS complex as a consequence of the variation of sinus node discharge rate. Vice versa with longer P-P intervals, the P waves positioned after the QRS complexes began gradually to move toward the QRS complex, became superimposed on the QRS complexes and finally appeared before the QRS complexes with a progressively increasing PQ interval. The P wave never moved through the S-T segment and T wave toward the subsequent QRS complex. Five of 10 dogs presenting IAVD with type I synchronization revealed the same rhythm during the entire recording time, whereas 5 of 10 dogs of this group alternated periods of IAVD with type I synchronization with periods of junctional tachycardia with 1 : 1 ventriculoatrial conduction. In these 5 cases, during ventriculoatrial conduction, the ECG pattern was characterized by the absence of sinus axis P waves, narrow QRS complexes followed by negative P' waves detectable as negative deflections in leads II, III and aVF, and as equally positive deflections in aVR and aVL. In these cases, P' waves appeared as pseudo-S waves, being inscribed in the descending branch of the QRS complex with a very short RP' interval ranging from 19 and 57 ms (Fig 1). One of 11 dogs presented IAVD with type II synchronization characterized by constant duration of the PQ interval with P waves and QRS complexes in a relatively fixed position with respect to each other (Fig 1). In this case, the PQ interval duration was 34 ms.



Fig 1. Six-lead surface electrocardiograms (I, II, III, aVR, aVL and aVF) during sinus rhythm (A) and during focal junctional tachycardia with isorhythmic atrioventricular dissociation and type II synchronization (B), isorhythmic atrioventricular dissociation and type I synchronization (C), and 1:1 ventriculo-atrial conduction (D). Note the presence in electrocardiogram D of P' with inferior-to-superior axis on the frontal plane that appeared as pseudo-S waves (arrow). Paper speed 50 mm/s 1 mV = 1 cm.

Electrophysiologic Studies

Three of 11 dogs underwent electrophysiologic mapping because of the presence of cardiac failure and to test the possibility to ablate the arrhythmic substrate. In all dogs, electrophysiologic findings were consistent with an electrical impulse originating from the His bundle. His Bundle electrograms always preceded the ventricular deflection with a normal HV interval duration (range: 30–48 ms) compatible with antegrade conduction. In 2 of 3 dogs, atrial depolarizations were always unrelated to the His and ventricular depolarizations. In these patients, an atrial activation pattern spreading from the high right atrium and dissociated from the ventricular activation arising from the atrioventricular junction was recorded (Fig 2). In both cases, there were periods of variable AH interval (IAVD with type I synchronization) and periods of fixed AH interval (IAVD with type II synchronization). In 1 of 3 cases, periods of atrioventricular dissociation (IAVD with type I synchronization) alternated

with periods of retrograde atrial activation with 1:1 ventriculo-atrial conduction. In this dog, the atrial myocardium was captured in a concentric retrograde fashion by the impulse originating from the area of the His bundle (Fig 2).

Discussion

In the veterinary literature, IAVD has only been described in 2 case reports. The first report described a case of isorhythmic dissociation with accrochage in a dog with ruptured chordae tendinae,¹² whereas the second described a case of suspected atrioventricular junctional tachycardia producing atrioventricular dissociation.¹³

The present study describes the electrocardiographic and electrophysiologic features of IAVD with type I and type II synchronization and its possible correlation with JFT in a group of Labrador Retrievers. Isorhythmic atrioventricular dissociation can be present during various rhythm disturbances, such as third

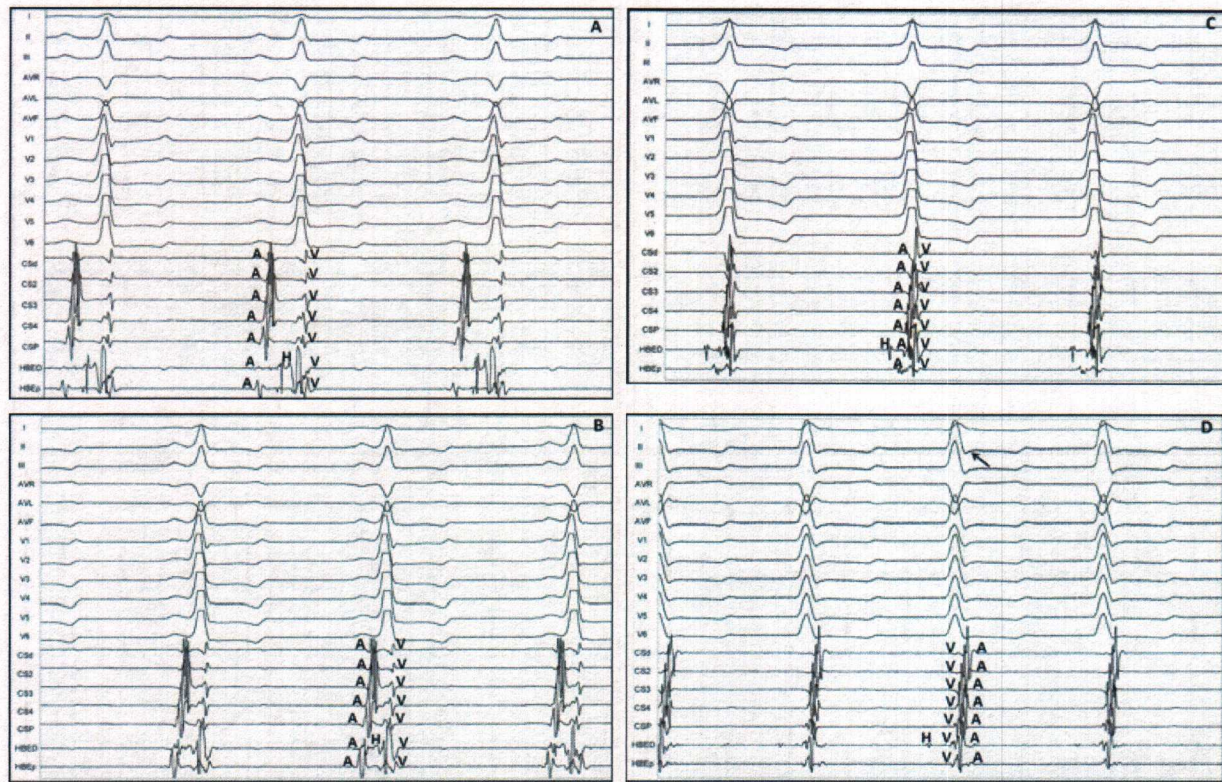


Fig 2. Twelve-lead surface electrocardiograms and intracardiac electrograms during sinus rhythm (A), focal junctional tachycardia with isorhythmic atrioventricular dissociation and type II synchronization (B), focal junctional tachycardia with isorhythmic atrioventricular dissociation and type I synchronization (C), and focal junctional tachycardia with 1:1 ventriculo-atrial conduction (D). Coronary sinus proximal, CS_p; coronary sinus distal, CS_d; His bundle proximal, HBE_p; His bundle distal, HBE_d; atrium, A; ventricle, V. Paper speed 150 mm/s. 22.5 mV = 1 cm.

degree or complete atrioventricular block with idioventricular rhythm, accelerated idioventricular rhythm, ventricular pacing, ventricular tachycardia, or focal junctional tachycardia. Isorhythmic atrioventricular dissociation is characterized by the presence of atrial depolarizations arising from the sinus node independently from ventricular depolarization originating from the atrioventricular junctional area or from ventricular structures. The relationship between P waves and QRS complexes can be described as synchronization type I and synchronization type II. Isorhythmic atrioventricular dissociation with type I synchronization was observed in 10 of the dogs included in this study. This type of synchronization has been previously described in the human^{2,3,6,7} and veterinary literature,^{12,13} and occurs consistently in dogs with experimentally induced AV block in which ventricular pacing was performed.⁶ Different mechanical or electrical theories have been proposed to explain IAVD with type I synchronization.¹⁻⁸ Observations in people and in experimental animal models strongly suggest that the primary mechanism responsible for the observed synchronization originates from cyclic fluctuations of arterial blood pressure.⁸⁻¹⁰ When the P wave "marches" to the right and appears after the QRS complex, the systemic blood pressure falls and

the right atrial pressure rises. The fall in arterial blood pressure and aortic flow during these periods result from the loss of the atrial contribution to ventricular filling. As a consequence of these hemodynamic changes, accelerating forces develop that increase the sinus rate and cause the movement of the P wave to the left of the QRS complex. Sinus acceleration therefore reestablishes a more physiologic AV activation sequence and normalizes aortic pressure and flow. These hemodynamic events reduce the cardio-acceleratory drive, and therefore the sinus rate slows down and the P wave moves back into the QRS complex and the cycle repeats itself.^{6,7} The rate of discharge of the dominant pacemaker (ie, the sinus node) varies inversely to the arterial pressure, primarily via reflexes that originate from the sino-aortic baroreceptors.⁷ However, other studies proposed that a direct influence of pressure in the sinus node artery on sinus node function should be taken into account.¹⁵ Another hypothesis has been proposed to explain the occurrence of IAVD with type I synchronization when both atrial and arterial pressures are kept constant. When atria and ventricles contract simultaneously, the content of the right atrium cannot be emptied into the right ventricle, the tricuspid valve being closed. Under these circumstances, the shortening of the muscular elements in the

atrial wall leads to distention of the noncontractile fibrous parts of the right atrium, and probably affects the very site of the sinus node (junction of the caval veins and the atrium). As a result, the sinus node is stretched, and distention of the pacemaker fibers results in acceleration of their spontaneous rate of depolarization.⁷ Isorhythmic atrioventricular dissociation type I becomes evident when sinus node discharge rate and ectopic rhythm discharge rate are quite similar. If the rate of discharge of the sinus node decreases, then the atrial myocardium can be captured by the retrograde depolarization wavefront originating from the atrioventricular junctional area, and is therefore activated in a concentric retrograde fashion with a 1 : 1 conduction.^{1,6,7} The position of the retrograde P' wave inscribed either in the descending branch of the R wave, the ST segment or in the ascending branch of the T wave, depends upon the retrograde conduction velocity along the atrioventricular junctional area.^{6,7} Isorhythmic atrioventricular dissociation with type II synchronization was recorded in one of the dogs included in our study, and is characterized by the presence of P waves in a fairly constant position relative to the QRS complexes. In this type of IAVD, P waves usually precede, are coincident with and therefore hidden by the QRS complex, or are located on the ST segment or the first half of the T wave.⁶ As atrial activation occurs simultaneously with or after ventricular activation, small changes in PQ or QP intervals are unlikely to significantly affect systemic blood pressure. Therefore, the mechanism proposed to explain IAVD with type I synchronization cannot account for IAVD with type II synchronization.⁶ Several other theories have been proposed to explain such type of synchronization. The most widely accepted hypothesis is the one proposed by Segers, who demonstrated the presence of electrical interaction between dissociated atrial and ventricular pacemakers.³ Furthermore, mechanical pulsations in the sinus node artery have been shown to exert a synchronizing effect on its pacemaker cells. Arterial pulsations could also produce a synchronizing effect on pacemaker cells in the atrioventricular junction area, thereby contributing to the synchronization of atria and ventricles.¹⁶ It is also possible that some reflex mechanisms are involved and different studies demonstrated that bursts of impulses in the efferent cardiac vagus nerve tend to synchronize the pacemaker activity in the sinus node with the rhythmic neural action potentials. These bursts of activity in the efferent vagal pathways could originate from the effect of ventricular ejection on arterial baroreceptors and could thereby synchronize atrial contraction with ventricular ejection.^{17,18} The presence of a very similar atrial and ventricular rate with P waves preceding the QRS complex at a fixed and short PQ interval can lead to erroneous diagnosis of ventricular pre-excitation, suggestive of the presence of an accessory pathway with antegrade conduction. Well-known features of this electrocardiographic entity are short PQ interval, prolonged duration of the QRS complex with

a slow rising onset (delta-wave), and secondary alteration of the ST segment and the T wave.¹⁹⁻²¹

In 3 of 11 dogs that underwent electrophysiologic studies, endocardial mapping findings were consistent with FJT, characterized by ventricular activation originating from the His bundle and dissociated from the atrial electrical activity with normal and fixed HV interval and variable (IAVD with type I synchronization) or constant (IAVD with type II synchronization) AH interval and periods of 1 : 1 ventriculo-atrial conduction with concentric retrograde activation. Focal junctional tachycardia is a narrow QRS complex tachycardia caused by abnormally rapid discharges from the atrioventricular junction area. Although it is believed that a re-entrant mechanism utilizing several separate longitudinally oriented strands is the most likely electrophysiologic basis for the tachycardia, it is not possible to exclude an automatic focus in a component of the atrioventricular junction area.²² In human beings, FJT is a rare arrhythmia mostly observed in pediatric patients, presenting as a congenital or postsurgical form. Its incessant nature is responsible for a poor prognosis even when FJT is occasionally observed in adulthood.²²⁻²⁷ The electrocardiographic features of FJT in human beings include a heart rate ranging from 110 to 250 bpm, narrow QRS complexes, and the presence of atrioventricular dissociation although 1 : 1 retrograde conduction may be observed.²⁶ In our study, we observed a narrow QRS rhythm with median heart rate of 140 bpm. Despite a heart rate within the reference range for a dog, we define this rhythm as a tachycardia, depending upon the fact that in dogs, the physiologic discharge rate of P cells located in the atrioventricular junction area ranges from 40 to 60 bpm.²⁸ In this study, IAVD was observed in 5 of 11 dogs presented with evidence of congestive heart failure and left ventricular hypokinesia. We speculate that in these dogs, the increased sympathetic tone could have enhanced the automatism of the atrioventricular junctional area. Furthermore, the secondary occurrence of IAVD in these dogs could have worsened the cardiac hemodynamic. In fact, it has long been demonstrated that important changes in cardiac dynamics follow interruption of normal sequential atrioventricular electrical and mechanical activity both in experimental animal models and in humans.²⁹ The so-called atrial "booster pump action" accounts for 15-20% of end-diastolic ventricular volume and is partially or completely lost when AV synchrony is impaired the atrial contribution may also be more significant in the presence of myocardial dysfunction.^{29,30} By far, the most important changes are produced when the AV contraction sequence is reversed, so that the atrium contracts against a closed atrioventricular valve, as we can observe during IAVD.^{29,30}

Limitations of this study include its retrospective nature and the limited number of dogs enrolled. Furthermore, only 3 of 11 dogs underwent EPS to confirm the exact sequence of atrial and ventricular activation, and a possible influence of propofol infusion on the underlying heart rhythms should be considered.

Conclusion

This study describes the electrocardiographic appearance of IAVD based on endocardial mapping and its possible correlations with FJT in Labrador Retrievers. According to our results, FJT could be associated with IAVD with type I and type II synchronization and, in some cases, periods of 1 : 1 ventriculo-atrial conduction. This rhythm disturbance can deteriorate cardiac output in dogs with concomitant left ventricular systolic failure.

Footnotes

^a Midazolam, PHT Pharma, Milan, Italy

^b Rapinovel, Schering-Plough, Milan, Italy

^c Isoflurane, Merial Italia Spa, Assago, Milan, Italy

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