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Electrocardiographic abnormalities in dogs with pleural effusion and preserved left ventricular systolic function#

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Abstract

Pleural effusion in dogs is associated with cardiac and non-cardiac diseases, often occurring concurrently. Scientific literature has documented different aetiological factors related to cardiac as well as non-cardiac causes, however, any evidence to link intra-cardiac conduction abnormalities with pleural effusion in dogs is lacking. This study is an attempt to document changes on electrocardiography (ECG) including intracardiac conduction abnormalities in dogs with pleural effusion and preserved left ventricular systolic function. Fourteen dogs brought to the hospital with respiratory distress and confirmed to have pleural effusion and normal left ventricular systolic function were subjected to detailed study and the findings obtained were compared statistically with that of six apparently healthy dogs brought to the hospital either for vaccination or routine health check-up. The important electrocardiographic abnormalities were increased heart rate, increased P wave duration, reduced R wave amplitude, QRS electrical alternans, deep S-waves, ST elevation or depression, ST slurring and broad-based T waves. Rhythm alterations included non-respiratory sinus arrhythmia and supraventricular tachyarrhythmia. The intra-cardiac conduction abnormalities included a delay in conduction through atrial myocardium with normal AV nodal conduction. The minor delay in intraventricular conduction noticed in the affected dogs is not significant to consider it for a right or left ventricular block pattern. The electrocardiographic alterations recorded in the present study cannot be concluded as a cause of pleural effusion in dogs with preserved systolic function.

Keywords: *Pleural effusion, aetiology, electrocardiography*

Pleural effusion in dogs is associated with diverse spectra of aetiological factors including cardiac and non-cardiac causes, often occurring concurrently. Cardiac disorders are the most encountered underlying aetiology for pleural effusion in canines (de Oliveira *et al*., 2021). The major cardiac causes of pleural effusion were constrictive pericarditis, pericardial effusions and right-sided heart failure associated with chronic valvular diseases or congenital defects such as tricuspid valve dysplasia (Cote *et al*., 2024). According to Hawkins (2020), the non-cardiac causes of pleural effusions included hypoalbuminemia, diaphragmatic hernia, congenital thoracic duct leakage, pulmonary

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disorders such as pulmonary thromboembolism and lung lobe torsion, neoplastic conditions including mediastinal lymphoma and hemangiosarcoma.

Cardiac diseases are primarily diagnosed based on echocardiographic and electrocardiographic investigations. Echocardiography confirms structural abnormalities of the heart such as chamber enlargement, hypertrophy, valvular regurgitation, valvular stenosis, pericardial effusion and cardiac masses; and the functional characters such as cardiac output and cardiac contractility. However, the gold standard method for detecting various intra-cardiac conduction abnormalities is electrocardiography. Electrocardiography is a non-invasive, quick, widely available and economical diagnostic aid for detection of cardiac conduction defects, in a least stressful manner. Moreover, ECG is an invaluable tool in identifying myocardial ischemia (Ware and Ward, 2020).

Non-cardiac aetiologies could be diagnosed by employing various techniques such as microbial culture, cytology of pleural fluid, histopathology, thoracic radiography and thoracic ultrasonography according to Mellanby *et al*. (2002).

Although pleural effusion associated with structural abnormalities of heart is well documented, its association with intra-cardiac conduction disturbance has been poorly studied. Hence, the purpose of this study is to explore the possibility of finding an association between intra-cardiac conduction abnormalities and pleural effusion in dogs with preserved left ventricular systolic function.

Materials and methods

Dogs presented to the University Veterinary Hospital, Kokkalai and University Veterinary Hospital and Teaching Veterinary Clinical Complex, Mannuthy during the period July 2023 to July 2024 with clinical signs of exercise intolerance, tachypnoea, dyspnoea, open mouth breathing and cough were screened for the presence of pleural effusion by radiography and ultrasonography. The presence of anechoic or echogenic fluid between parietal and visceral pleura on thoracic ultrasonography and the radiographic signs of increased opacity of thorax, obscured cardiac silhouette and dorsal elevation of trachea were the criteria used for the diagnosis of pleural effusion as well as selection of study population. Fourteen dogs confirmed to have pleural effusion were selected for detailed investigation. Dogs presented with severe respiratory compromise were stabilised by supplementing oxygen at a flow rate of 300 mL/kg/minute (Murphy and Papasouliotis, 2011) prior to further evaluation.

The selected dogs belonged to the age group of six months to two years (n = 3), two to six years (n = 7), and seven to 11 years (n = 4). The population belonged to different breeds of dogs, *viz.* Labrador Retriever (n = 3), non-descript (n = 3), crossbred (n = 3), Spitz (n = 2), German Shepherd (n = 1), Lhasa Apso (n = 1) and Doberman (n = 1).

Electrocardiogram was recorded in a quiet room with the animal held in right lateral recumbency on a wooden table with the limbs held perpendicular to the body without allowing them to contact each other (Fig. 1). A single-channel 12 lead ECG recorder (BPL CARDIART- 6108T) with standard bipolar limb leads (I, II, III) and augmented unipolar limb leads (aVR, aVL, aVF) was used to record electrocardiogram at a sensitivity of one centimetre for one millivolt and at a paper speed of 25 mm/s. Lead II was used to obtain various electrocardiographic measurements. The amplitude and duration of P wave, QRS complex, and T wave; the duration of PR interval and QT interval; and other abnormalities in wave pattern were determined (Tilley and Smith, 2008).

For echocardiographic evaluation, the right thoracic region was shaved from behind the forelimb to the sixth intercostal space from the costochondral junction to the sternum and on the left side, from behind the forelimb at the level of costochondral junction to just behind the last rib in a rectangular shape. Echocardiography was performed by laying the animal in right or left lateral recumbency according to the imaging plane required. Esoate MyLabX8 eXP ultrasound scanner with multi-frequency phased array cardiac probe was used for echocardiography. Two-dimensional, M mode, colour flow doppler and continuous wave doppler echocardiography were utilised to assess systolic function, identify valvular regurgitations, measure valvular regurgitation velocities and to determine parameters *viz*. left atrium to aortic root ratio (LA: Ao) and E-point to septal separation (EPSS) (Boon, 2011).

Six apparently healthy adult dogs brought to the hospital for routine vaccination or health check-up served as control to obtain normal electrocardiographic and echocardiographic measurements. The data was statistically compared using independent student's t-test. Statistical analysis was performed using SPSS Version 24.0.

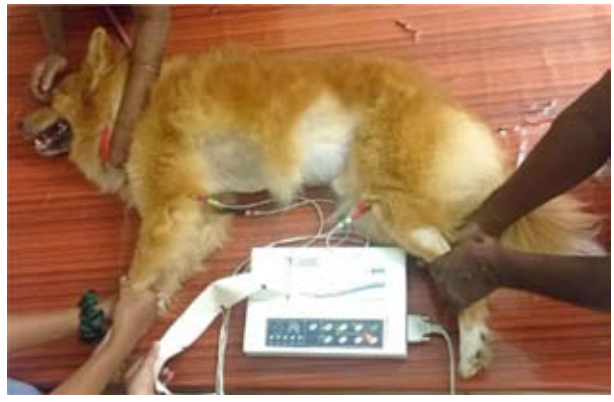

Fig. 1. Recording ECG with the animal positioned in right lateral recumbency

Results and discussion

Electrocardiographic measurements

Rate

A significant increase ($p \leq 0.01$) occurred in the mean heart rate (HR) of diseased animals (142.69 ± 9.70 bpm) compared to that of healthy animals (98.00 ± 6.67 bpm). Heart rate of more than 140-180 bpm was appreciated in 50 per cent of animals in the current study which could be associated with anaemia (haematological parameters given in Table 3), hypoxia, and congestive heart failure in those animals (Tilley and Smith, 2008). Tachycardia was reported in 25 per cent dogs with pleural and mediastinal effusions (Mellanby *et al*., 2002) and was regarded as an appreciable clinical finding in dogs with pleural effusion (Sanders and Sleeper, 2004) especially when associated with congestive heart failure (Mahendran *et al.,* 2021).

Tachycardia associated with large volume pleural effusion resulted from hypoxia due to limited expansion of thoracic cavity and reduced tidal volume. In the present study, tachycardia could be noticed even in animals with low-grade pleural effusion in many instances. Therefore, the reasons for tachycardia could be multifactorial, possibly anaemia, cardiac congestion, reduced lung parenchymal elasticity and low-grade lung oedema, in addition to restricted expansion of thoracic cavity as well as reduced tidal volume.

Rhythm

Rhythm was of sinus origin and regular in 10 animals (71.42 per cent). Of the remaining four animals, respiratory sinus arrhythmia (Fig. 2) was seen in two animals (14.28 per cent) while one animal each (7.14 per cent) exhibited non-respiratory sinus arrhythmia and supraventricular tachyarrhythmia (Fig. 3).

Respiratory sinus arrhythmia is regarded as normal in dogs which can be explained in terms of vagal stimulation on heart. During expiration, intra-thoracic pressure increases which in turn stimulates the vagal nerve endings. Stimulation of vagus nerve inhibits heart rate. The opposite happens during inspiration. The end result is relatively slower heart rate during expiration and faster during inspiration which is referred to as respiratory sinus arrhythmia (Sisson and Ettinger, 1999). This could be the reason for respiratory sinus arrhythmia in dogs studied here. Moreover, this variation in heart rate during inspiration and expiration does not occur when the heart rate is too high. Supraventricular tachyarrhythmia recorded in one dog can be a consequence of pressure on atrial myocytes associated with mitral valve insufficiency (Abbott, 2008) as evident from increased LA/Ao of 1.66.

Tables 1 and 2 compare electrocardiographic measurements and echocardiographic parameters in diseased and healthy animals, respectively.

P amplitude

No significant increase in P wave amplitude could be appreciated in the diseased dogs compared to healthy dogs.

P duration

P duration was significantly prolonged ($p \leq 0.01$) (Fig. 4) in diseased animals (0.049 ± 0.002 sec) in comparison with the mean value of healthy animals (0.04 ± 0.00 sec). While P wave could be slightly prolonged in giant breed dogs (up to 0.05 sec), a P duration of more the 0.04 was found in 50 per cent (7/14) of the patients which were small and medium-sized breeds. Increased P duration was generally associated with left atrial

Table 1. Comparison of electrocardiographic measurements in diseased and healthy animals

Parameters	Diseased dogs (n = 14) Mean ± SE (Range)	Healthy control (n = 6) Mean ± SE (Range)	t-value	p-value
HR (bpm)	142.69 ± 9.70 (83 - 188)	98.00 ± 6.67 (80 - 125)	2.95**	<0.01
P amplitude (mV)	0.107 ± 0.009 (0.05 - 0.2)	0.17 ± 0.03 (0.15 - 0.3)	1.84	0.12
P duration (sec)	0.0492 ± 0.002 (0.04 - 0.07)	0.04 ± 0.00 (0.04)	3.21**	<0.01
PR interval (sec)	0.0977 ± 0.005 (0.07 - 0.12)	0.11 ± 0.004 (0.10 - 0.12)	1.34	0.20
R amplitude (mV)	0.4885 ± 0.113 (0.1 - 1.7)	1.09 ± 0.26 (0.4 - 1.8)	2.53*	0.02
QRS duration (sec)	0.0615 ± 0.002 (0.05 - 0.08)	0.06 ± 0.004 (0.05 - 0.08)	0.31	0.76
QT interval (sec)	0.193 ± 0.008 (0.14 - 0.26)	0.22 ± 0.01 (0.2 - 0.28)	1.72	0.10

**Significant at p ≤ 0.05 and ** Significant at p ≤ 0.01*

Table 2. Comparison of echocardiographic parameters in diseased and healthy animals

Parameters	Diseased dogs (n=14) (Range)	Healthy control (n=6) (Range)	t-value	p-value
EF (%)	67.36 ± 3.32 (51 - 85)	62.83 ± 1.19 (50 - 67)	1.28	0.22
FS (%)	37.07 ± 2.45 (21 - 51)	33.50 ± 1.74 (30 - 41)	0.90	0.38
EPSS (mm)	3.23 ± 0.48 (0.79 - 6.9)	4.10 ± 0.54 (2 - 6.1)	1.07	0.30
LA/Ao	1.38 ± 0.08 (1.01 - 1.99)	1.09 ± 0.01 (1.06 - 1.17)	2.32*	0.03

**Significant at p ≤ 0.05*

Table 3. Comparison of haematological parameters between diseased and healthy animals

Parameters	Diseased dogs (n=14) Mean ± SE (Range)	Healthy control (n=6) Mean ± SE (Range)	t-value	p-value
Hb (g/dL)	11.60 ±1.21 (4.8 - 19.4)	15.16 ± 0.73 (12.6 - 16.6)	2.51*	0.02
TEC (10^6/µL)	5.05 ± 0.50 (2.7 - 8.38)	5.76 ± 0.18 (5.01 - 6.15)	1.31	0.20
VPRC (%)	33.10 ± 3.44 (13.9 - 55.5)	38.45 ± 0.98 (34.8 - 41)	1.49	0.16
TLC (10^3/µL)	19.85 ± 2.64 (6.1 - 42.1)	9.33 ± 1.12 (6.2 - 12.8)	2.53*	0.02
Neutrophil (x 10^3/µL)	15.40 ± 2.51 (4.05 - 33.74)	6.72 ± 1.13 (3.8 - 10.62)	3.15**	<0.01
Lymphocyte (x 10^3/µL)	2.53 ± 0.77 (0.44 - 9.8)	2.18 ± 0.08 (1.92 - 2.44)	0.45	0.66
Monocyte (x 10^3/µL)	1.69 ± 0.41 (0 - 6.11)	0.28 ± 0.09 (0 - 0.13)	2.18*	0.04
Basophil (x 10^3/µL)	0.04 ± 0.01 (0 - 0.15)	0.02 ± 0.02 (0 - 0.13)	0.80	0.44
Eosinophil (x 10^3/µL)	0.21 ± 0.05 (0 - 0.67)	0.11 ± 0.06 (0 - 0.38)	1.05	0.31
PLT (x 10^5/µL)	2.47 ± 0.31 (0.68 - 5.28)	2.03 ± 0.15 (1.6 - 2.66)	0.90	0.38

**Significant at p ≤ 0.05 and ** Significant at p ≤ 0.01*

enlargement which was evident on echocardiography as increased LA/Ao (Martin, 2015). In the present study, high LA/Ao ratio suggestive of atrial enlargement was observed in 79 percent dogs (11/14) as against an increased P duration in only 50 percent dogs (7/14). In other words, structural abnormality was seen in 11 dogs, whereas atrial conduction abnormality was seen in only seven dogs. This strongly suggested the development of structural alterations prior to electrical disturbances in this study population.

PR interval

There was no significant change in PR interval in diseased dogs compared to the healthy animals. This indicates a normal cardiac conduction through the AV nodal fibres.

R amplitude

There occurred a significant reduction in mean R amplitude of diseased animals (0.49 ± 0.11 mV) compared to that of healthy animals (1.09 ± 0.25 mV) and R amplitude was less than 0.5 mV in 10 animals (71.42 per cent) (Fig. 5). Mohan *et al*. (2019) reported low voltage R wave in dogs with pleural effusion, which was a significant finding in the current study also. Low R amplitude typically occurs when cardiac electric impulse needs to traverse fluid in the cavities (Tilley and Smith, 2008).

Electrical alternans of QRS complex (Fig. 6) was observed in one animal (7.14 per cent) with significant pericardial effusion on echocardiography, which could be associated with the change in cardiac axis due to the movement of the heart within pericardial fluid (Martin, 2015), while severe pleural effusion could also have

Fig. 2. Respiratory sinus arrhythmia and ST slurring (red arrow) (Lead II; 25 mm/s; 10 mm/mV)

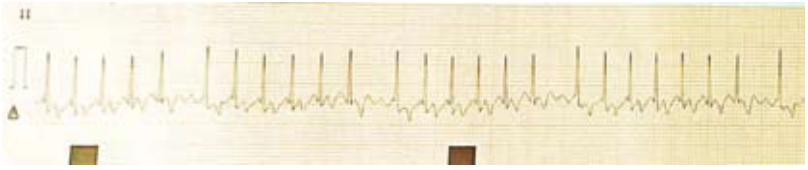

Fig. 3. Supraventricular tachyarrhythmia (Lead II; 25 mm/s; 10 mm/mV)

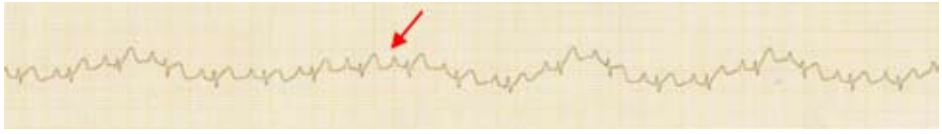

Fig. 4. Increased P duration (red arrow) (Lead II; 25 mm/s; 10mm/mV)

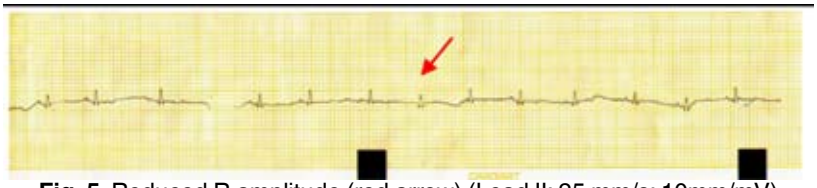

Fig. 5. Reduced R amplitude (red arrow) (Lead II; 25 mm/s; 10mm/mV)

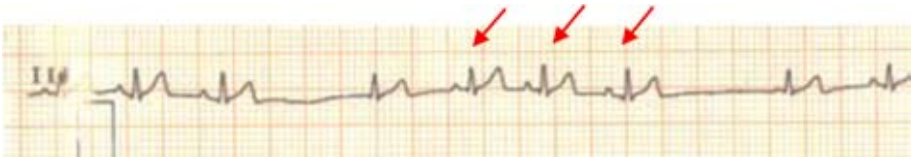

Fig. 6. Electrical alternans of QRS complex (red arrows) (Lead II; 25 mm/s; 10mm/mV)

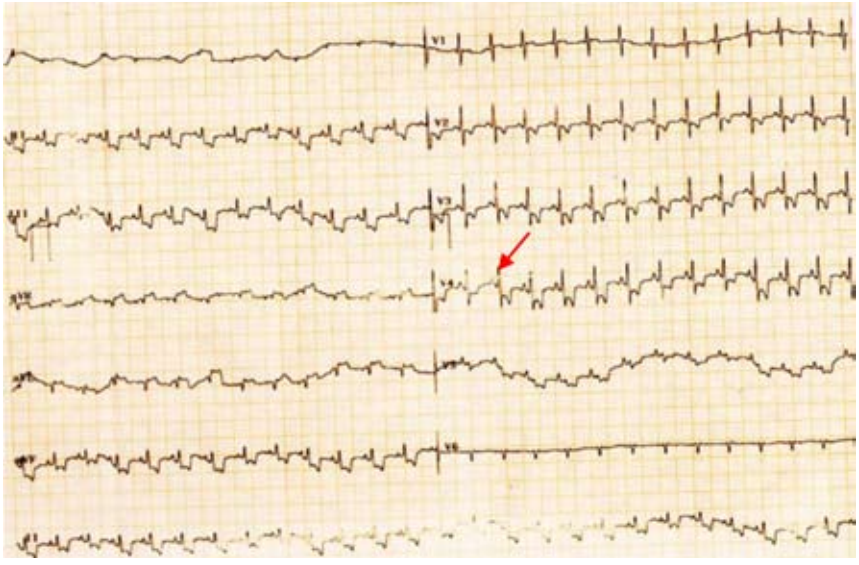

Fig. 7. ST depression, prominent in V4 (red arrow) (25 mm/s; 10 mm/mV)

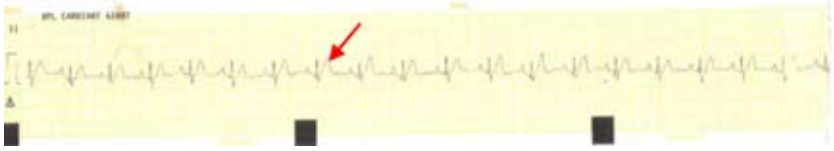

Fig. 8. Tall and broad-based T waves (red arrow) (25 mm/s; 10mm/mV)

resulted in electrical alternans (Tilley and Smith, 2008).

QRS duration

All the dogs except two had a QRS duration normal to the species. Two dogs had a QRS duration of 0.08 sec which is over and above the reference range for the breeds studied (Martin, 2015). However, a QRS duration of 0.08 sec is not regarded as a major intraventricular conduction defect and a delay of > 0.1 sec is considered significant (Tilley and Smith, 2008). In the present study, intra-ventricular conduction delay is not significant and hence cannot be attributed as a reason for pleural effusion in dogs.

S wave

Tilley and Smith (2008) described deep S wave as the presence of S wave in leads I, II, III, and aVF, with amplitude of S wave more than 0.05 mV in lead I and more than 0.35 mV in lead II. According to Martin (2015), deep S wave could be associated with right ventricular (RV) enlargement, pulmonic stenosis and left anterior fascicular block while deep and slurred S wave could be indicative of right bundle branch block.

In the present study, S waves in lead II, III and aVF with amplitude of S wave more than 0.35 mV could be observed in two animals (14.28 per cent). Absence of wide QRS complexes rules out right and left bundle branch block in these dogs. In both the animals with deep S wave, pulmonary hypertension and associated right ventricular enlargement could be appreciated on echocardiography. Hence, deep S wave in these dogs can be attributed to right ventricular enlargement rather than a conduction abnormality.

ST segment

ST-segment depression (Fig. 7) and elevation were evident in one (7.14 per cent) and two animals (14.28 per cent) respectively. ST slurring (Fig. 2) was identified in two animals (14.28 per cent). The animal with ST depression succumbed to death within a period of four weeks. ST elevation, ST depression as well as ST slurring has been associated with myocardial hypoxia, myocardial inflammation and myocardial infarction (Ford and Mazzaferro, 2012; Oyama *et al*., 2014). ST segment elevation myocardial infarction (STEMI) is an important factor causing morbidity and mortality in humans (Choudhury *et al*., 2016), which should also be considered in dogs under the study.

The presence of ST elevation, depression and slurring in the study population indicated the presence of varying levels of hypoxia, inflammation or infarction in the affected dogs, but a differential of these is beyond the scope of this study.

T wave

T wave was positive in six animals (42.85 per cent), negative in four animals (28.57 per cent) and positive, negative or biphasic in two animals (14.28 per cent). Two dogs had tall and broad-based T waves (Fig. 8) and one had (7.14 per cent) broad-based T waves. The dog with broad-based T wave succumbed to death by the third month and necropsy revealed severe hepatic cirrhosis.

T wave morphology is highly variable in small animals, the reasons for all the changes being not fully explained except for some changes *viz*. narrow spiked T waves seen in hyperkalaemia (Martin, 2015). Tall and broad-based symmetrical T waves in humans is said to be an early sign of acute ischemia (Levis, 2015) which can also be suspected in the dogs under the present study.

Echocardiographic parameters

There was no significant difference in EF, FS and EPSS between diseased and healthy animals. This indicated that the systolic function of left ventricle is within normal range. Left ventricular chamber enlargement and dilated cardiomyopathy was ruled out owing to the normal EPSS in all the studied dogs.

The mean LA/Ao ratio was significantly elevated ($p \leq 0.01$) in diseased animals (1.38 ± 0.08; Range: 1.01 - 1.99) when compared to healthy controls (1.09 ± 0.01; 1.06 - 1.17). However, the mean LA/Ao of diseased animals was within the normal population range for the species (Boon, 2011). Analysing on a case-by-case basis, 11 dogs had elevated LA/Ao ratio. The elevated mean LA/Ao could be attributed to left atrial enlargement associated with mitral insufficiency and pulmonary hypertension. Mild and moderate mitral valve regurgitations were found in two dogs each (14.28 per cent), while severe mitral valve regurgitation in three animals (21.42 per cent) based on jet size in colour flow doppler echocardiography (Boon, 2011).

Four animals (28.57 per cent) were concluded to be having pulmonary hypertension based on pulmonary and tricuspid valve regurgitation velocities found on continuous wave echocardiography (Johnson *et al.*, 1999).

Briefly, the important electrocardiographic abnormalities observed in dogs with pleural effusion and preserved systolic function were increased heart rate, increased P wave duration, reduced R wave amplitude, QRS electrical alternans, deep S-waves, ST elevation or depression, ST slurring and broad-based T waves. Rhythm alterations included non-respiratory sinus arrhythmia and supraventricular tachyarrhythmia. The electrical conduction abnormalities included a delay in atrial myocardial conduction with normal AV

nodal and intraventricular conduction. The documented electrocardiographic abnormalities were not consistent in all the affected dogs. The consistent electrocardiographic features included tachycardia and reduced R amplitude, in which tachycardia is a compensatory mechanism and reduced R amplitude developed consequent to the presence of fluid in thoracic cavity. The mean duration of P wave, suggestive of left atrial enlargement, was significantly different from the control dogs, however on a case-by-case analysis, the P duration was above normal reference level in only 50 percent of the affected dogs. The same feature of left atrial enlargement as assessed by the echocardiographic parameter of LA/Ao ratio revealed that 71 percent of the dogs had large atrial chambers. An extrapolation of increased P duration and increased LA/Ao ratio suggests that the atrial chamber enlargement preceded atrial electrical alterations in the study population. To conclude, electrocardiographic alterations were not significant enough to cause pleural effusion in dogs with preserved left ventricular systolic function and hence other ailments such as structural involvement of heart, hepatic insufficiency, thoracic masses, and coagulation defects should be closely monitored in all cases of pleural effusion in dogs.

Conclusion

Cardiac conduction disturbances recorded in dogs with preserved left ventricular systolic function under present study cannot be concluded as a cause of pleural effusion.

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Conflict of interest

The authors declare that they have no conflict of interest.

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