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Research Article

Comparison of in-hospital and 24-Hour Ambulatory Electrocardiography in Dogs with Heart Disease

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A B S T R A C T

Background/Aim: The purpose of the study was to compare the short and long term individual electrocardiographic findings in preclinical (asymptomatic) and clinical (symptomatic) dogs with heart disease.

Material and Method: 2 minute clinic ECG and 24-h Holter monitoring were performed in 30 dogs. Disturbances in impulse conduction, impulse formation and heart rate were assessed.

Results and Conclusion: A significant linear correlation was found between the average heart rates detected by clinic ECG and Holter monitoring in preclinical group: y= 45.33 + 1.023x (P<0.05, r=0.46) as well as in clinical group: y = -10,39 + 1,546x (P<0.05, r=0.73). Of 30 dogs, 19 had cardiac arrhythmias on 2 minute clinic ECG. 24-h Holter records revealed the presence of cardiac arrhythmias in 29 dogs. 11 dogs with normal sinus rhythm on clinic ECG had clinically significant arrhythmias on 24-h Holter monitoring. This study represents Holter-detected significant changes in cardiac arrhythmias. 24-h Holter monitoring provides the best assessment of the presence, frequency and complexity of the cardiac arrhythmias in dogs with heart disease.

Key Words: Arrhythmia, canine, ECG, holter, supraventricular, ventricular

Kalp Hastalıklı Köpeklerde Klinik Elektrokardiyografi ile 24 Saat Elektrokardiyografinin Karşılaştırılması

ÖZET

Özbilgi/Amaç: Bu çalışma ile; semptomatik ve asemptomatik kalp hastalıklı köpeklerde kısa ve uzun dönem elektrokardiyografik bulgularının karşılaştırılaması amaçlandı.

Materyal ve Metot: 30 köpekte 2 dakikalık klinik EKG ve 24 saat Holter kaydı gerçekleştirildi. İmpuls oluşum, iletim ve kalp atım sayısındaki değişiklikler değerlendirildi.

Bulgular ve Sonuç: Klinik EKG ve Holter kaydı ile belirlenen ortalama kalp atım sayıları arasında sırasıyla; preklinik grupta: y= 45.33 + 1.023x (P <0.05, r=0.46) ve klinik grupta: y = -10,39 + 1,546x (P <0.05, r=0.73) linear korelasyon belirlendi. 30 köpeğin 19'unda 2 dakikalık klinik EKG ile kardiyak aritmiler belirlendi. 24 saat Holter kaydı ise 29 köpekte kardiyak aritmileri ortaya koydu. Klinik EKG'de normal sinüs ritmi belirlenen 11 köpeğin 24 saat Holter kaydında klinik olarak anlamlı aritmiler tespit edildi. Çalışmada, kardiyak aritmilerde Holter ile belirlenmiş anlamlı değişiklikler görüldü. Kalp hastalıklı köpeklerde; kardiyak aritmilerin varlığı, frekansı ve kompleksitesini belirlemede 24 saat Holter kaydı en iyi değerlendirmeyi sağlamaktadır.

Anahtar Kelimeler: Aritmi, EKG, holter, köpek, supraventriküler, ventriküler.

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Introduction

Most of dogs are referred to veterinary hospitals in advanced stages of heart disease. However, dogs with early stage heart disease have the increased risk of cardiac arrhythmias (Petrie, 2005). Cardiac arrhythmias defined as a disturbance in impulse conduction and formation or heart rate are valuable markers of heart diseases in dogs (Petrie, 2005; Aptekman et al., 2010). Clinic electrocardiography (ECG) has often limitations in detecting underlying or early stage cardiac arrhythmias because of the short recording period and transient nature of the arrhythmias (Miller et al., 1999; Eastwood and Elwood, 2003). It has also been reported that arrhythmias detected by clinic ECG comprise only 0.14% of daily rhythm (Petrie, 2005). The number and types of arrhythmias in paralel with the progression of heart disease increases over time and following that dogs starts to show clinical signs. Detecting the cardiac arrhythmias in early and advanced stage of heart disease is essential for diagnostic and prognostic purposes (Calvert et al., 2000; Wess et al., 2010). The purpose of the current study was to compare the short and long term individual electrocardiographic findings in symptomatic and asymptomatic dogs with heart disease.

Materials and Methods

Animals

The present study was a prospective, single institution, hospital-based study conducted at Small Animal Veterinary Teaching Hospital between July 2011 and April 2012. Thirty seven privately owned dogs with heart disease were prospectively enrolled in the study. The dogs were not receiving any medication at the time of referral investigation and during diagnostic applications. The dogs with heart disease were classified as preclinical (asymptomatic) and clinical (symptomatic) according to International Small Animal Cardiac Health Council (ISACHC) recommendations based on cardiac clinical signs and thoracic radiographs. ISACHC classification included the followings: I, asymptomatic dogs; Ia, asymptomatic dogs without cardiomegaly; Ib, asymptomatic dogs with an evidence of cardiac compensation (cardiomegaly) on radiographs; II, mild to moderate heart failure. Clinical signs evident at rest and mild exercise; III, advanced heart failure. Clinical signs of CHF are immediately obvious; IIIa, home care possible; IIIb, hospitalization required. Dogs were excluded if they had clinical signs of non-cardiac disease on history, clinical examination and routine blood works. All dogs were treated appropriately. Written owner consent was obtained and the ethics committee of the institution (report no: 2011 107 402) approved the protocol used in this study.

Examination procedure

All dogs were examined by a standardized protocol including physical examination, routine blood work, radiography, echocardiography, 2 minute clinic ECG and 24-h Holter monitoring. Two dimensional B-M mode, color-flow and spectral doppler echocardiography (Esaote Biomedica AU 5 Multi Frequency Ultrasound System, Esaote Biomedica, Firenze, Italy) were performed to identify the structural heart disease. Clinic 6-lead ECG (Cardiofax ECG 6851K, Nihon Kohden Corporation, Tokyo 161, Japan) was recorded for 2 minutes to specify heart rate (during 6-s period) and rhythmin all dogs lying right lateral recumbency. Clinic ECGs were performed before 30 Holter studies. A 5-lead/3 channel holter recorder (DR 200/HE Holter and Event Recorder, NorthEast Monitoring, Inc., Maynard, US) was attached to the dogs in a standard precordial placement. Before the electrode placement, the skin at the electrode sites was prepared by shaving and cleaning with alcohol. Specially designed dog vests (Dog vests, DogLeggs,

LLC, Reston, USA) were used to secure the holter recorder and leads. The daily routine in each case including sleeping-bed time, awakening and exercise were noted in a diary by the owners. At the end of the 24-h recording period; recorder and electrodes removed. Arrhythmia identification and the summary of minimum, average and maximum heart rate (HR_{min}, HR_{mean}, HR_{max}) were performed by a commercially available software (Holter LX Analysis Software, NorthEast Monitoring, Inc., Maynard, USA). Cardiac arrhythmias were evaluated as ventricular, supraventricular and bradyarrhythmias. Arrhythmia variables included the followings: Sinus bradycardia (SB), >4 sinus complexes at a HR <70 bpm (Edwards, 2000); sinus pause (SP), NN intervals >2.0 seconds; sinus arrhythmia, as a NN interval >180% longer than the previous NN interval (Hall et al., 1991); sinus tachycardia (ST), >3 sinus complexes at a HR>150 bpm; supraventricular premature complexes (SVPC), a premature beat with abnormal P wave conducting a normal appearing QRS complex; supraventricular tachycardia (SVT), >3 supraventricular complexes at a HR >150 bpm, where the first complex was premature, while the folloving have NN interval shorter than or equal to the previous NN inerval (Kittleson, 1998); ventricular premature complex (VPC), a premature wide and bizarre QRS complex, not associated with a P wave, accompanying a large T wave of opposite polarity (Kittleson, 1998) and ventricular tachycardia (VTAC), as a sequence of more than three consecutive QRS complexesat a HR> 180 bpm (Edwards, 2000). All holter records were manually checked by the same operator to confirm the arrhythmia identification. During the diagnostic examinations none of dogs were sedated.

Statistical analysis

Statistics were performed with commercially available software (Statistical Package Version 14.01, SPSS Inc, Chicago, IL, USA). Checking the normality of data was tested with shapiro-wilk test. The relationship between the heart rates on ECG and Holter was analysed with paired sample T-test. Mean differences between the average heart rates detected by ECG and Holter were calculated. Mean differences between the groups for the overal presence of arrhythmias on both ECG and Holter were evaluated with fisher exact test. Results were presented as mean ± standard error of the individual groups. Statistical significance was defined as a value of $P < 0.05$.

Results

Data were collected from 37 dogs including Terrier types (9), Kangal Dogs (4), Cavalier King Charles Spaniels (3), Labrador Retrievers (3), Boxers (3), Miniatura Pinschers (3), German Shepherd Dogs (2), Mongrel Dogs (2), Golden Retriver (1), Doberman Pinscher (1), Dalmatian (1), Irish Setter (1), Pekingese (1),Pomeranian (1), Beagle (1) and Shih Tzu (1). However, 7 dogs were excluded from the study because of excitation (1 Terrier type and 1 Kangal Dog), the diagnosis of Cushing's disease (1 Terrier type) and <24 hour data on Holter records (2 Terrier types and 2 Mongrel Dogs).

Structuralheartdiseasewasidentifiedin28dogs(9dogsinclinical group and 19 dogs in preclinical group). Structural abnormalities diagnosed in clinical group were Endocardiosis of the Mitral Valve (55.5%), Dilated Cardiomyopathy (22.2%), Tricuspid Valve Insufficiency (11.1%) and Hemangiosarcoma(11.1%). Dogs with structural heart disease in preclinical group had Endocardiosis of the Mitral Valve (38.09%), Dilated Cardiomyopathy (33.3%), Aortic Stenosis (4.76%), Tricuspid Valve Dysplasia (9.52%) and Unilateral Papillary Muscle Hypertrophy (4.76%). Two dogs had no evidence of structural heart disease.

A total of 60 electrocardiographic examinations, including 2 minute clinic ECG and 24-h Holter monitoring, were performed on 30 dogs. There were no clinically significant differences

Table 1. Presence of cardiac arhythmias on ECG and 24-h Holter monitoring in preclinical group.

Tablo 1. Preklinik grupta 24 saat Holter monitörizasyon ve EKG ile belirlenen kardiyak aritmilerin varlığı.

Gender (male/female)			14/7
Mean Age (years)			6.77±0.90
Mean BW (kg)			19.33±2.80
Mean BSA (m^2)			0.67±0.07

Cardiac Diagnosis (ISACHC class I)	Percentage of Total n (%)	Presence of Arrhythmias on Clinic ECG n (%) (ISACHC class)	Presence of Arrhythmias on 24hr Holter Monitoring* n (%) (ISACHC class)
SSS	2 (9.52)	2 (9.52) (Ia:1, Ib:1)	2 (9.52) (Ia:1, Ib:1)
DCM	7 (33.3)	5 (23.8) (Ia:1, Ib:4)	7 (33.3) (Ia:2, Ib:5)
MVI	8 (38.09)	3 (14.28) (Ia:2, Ib:1)	7 (33.3) (Ia:2, Ib:5)
UPMH	1 (4.76)	-	1 (4.76) (Ia:1)
AS	1 (4.76)	1 (4.76) (Ib:1)	1 (4.76) (Ib:1)
TD	2 (9.52)	1 (4.76) (Ia:1)	2 (9.52) (Ia:1, Ib:1)
Total	21 (100)	12 (57.14)	20 (95.23)

SSS: Sick Sinus Syndrome, DCM: Dilated Cardiomyopathy, MVI: Mitral Valve Insufficiency, UPMH: Unilateral Papillary Muscle Hypertrophy, AS: Aortic Stenosis, TD: Tricuspid Valve Dysplasia, BW: Body Weight, BSA: Body Surface Area.

between the groups for the presence of cardiac arrhythmias on both clinic ECG and 24-h Holter monitoring (P >0.05). The characteristics, cardiac diagnosis, ISACHC classification and the presence of arrhythmias in 30 dogs with and without cardiac signs were shown in Table 1 and 2.

Of 30 dogs, 19 (63.33%) had cardiac arrhythmias on 2 minuteclinic ECG. Analysis of 24-h Holter records revealed the presence of cardiac arrhythmias in 29 dogs (96.66%). The arrhythmias on clinic ECG consisted of atrial fibrillation (13.33%), ST (6.66%), SB (10%), SP (3.33%), atrioventricular block (26.66%), VPC (16.66%), SVPC (3.33%) and SVT (6.66%). The arrhythmias on Holter monitoring included atrial fibrillation (13.33%), ST (10%), SB (16.66%), SP (6.66%), atrioventricular block (26.66%), VPC (76.66%), SVPC (23.33%) and VTAC

Detailed description of cardiac arrhythmias in dogs with and without cardiac signs were shown in Table 3 and 4.

The HR_{mean} frequency recorded with clinic ECG in preclinical and clinical groups was 135.24±8.20 bpm and 154.56±16.28 bpm, respectively. The HR_{mean} detected by 24-h Holter monitoring in preclinical and clinical groups was respectively 87.90±3.63 bpm and 106.67±7.67 bpm. A significant linear correlation was found between the HR_{mean} detected by clinic ECG and Holter monitoring in preclinical group: y= 45.33 + 1.023x (P<0.05, r=0.46) as well as in clinical group: y = -10,39 + 1,546x (P<0.05, r=0.73). The ECG HR_{mean} in groups was significantly higher than HR_{mean} recorded with Holter monitoring. The 24-h Holter results showed no differences between the groups for HR_{min} and HR_{max},

Table 2. Presence of cardiac arrhythmias on ECG and 24-h Holter monitoring in clinical group.

Tablo 2. Klinik grupta 24 saat Holter monitörizasyon ve EKG ile belirlenen kardiyak aritmilerin varlığı.

Gender (male/female)			8/1
Mean Age (years)			10.55±1.2
Mean BW (kg)			26.61±6.24
Mean BSA (m^2)			0.84±1.40

Cardiac Diagnosis (ISACHC class II-III)	Percentage of Total n (%)	Presence of Arrhythmias on Clinic ECG n (%) (ISACHC class)	Presence of Arrhythmias on 24-h Holter Monitoring* n (%) (ISACHC class)
DCM	2 (22.2)	2 (22.2) (II:1, IIIb:1)	2 (22.2) (II:1, IIIb:1)
MVI	5 (55.5)	3 (33.3) (II:2, IIIb:1)	5 (55.5) (II:4, IIIb:1)
TVI	1 (11.1)	1 (11.1) (IIIb:1)	1 (11.1) (IIIb:1)
HS	1 (11.1)	1 (11.1) (IIIb:1)	1 (11.1) (IIIb:1)
Total	9 (100)	7 (77.7)	9 (100)

DCM: Dilated Cardiomyopathy, MVI: Mitral Valve Insufficiency, TVI: Tricuspid Valve Insufficiency, HS: Hemangiosarcoma, BW: Body Weight, BSA: Body Surface Area.

*Artefacts were excluded.

(16.66%). 11 dogs (36.66%) with normal sinus rhythm (NSR) on 2 minute clinic ECG had cardiac arrhythmias on 24-h Holter monitoring. SVPCs were not frequent on clinic ECG, whereas 7 dogs (23.33%) had SVPCs on 24-h Holter records ranged from 2 to 15 beats. VPCs diagnosed in 5 dogs (16.66%) on clinic ECG were much more common on 24-h Holter monitoring (23 dogs, 76.66%). The total number of VPC (included R on T) ranged from 2 to 594 beats on 24-h Holter monitoring. The 24-h Holter findings of nonsustained VTAC were found in 16.66% of the total population. No dogs demonstrated sustained VTACduring 24-h Holter monitoring. Of 4 dogs with persistent atrial fibrillation on 2 minute clinic ECG, 1 had paroxysmal atrial fibrillation lasting 48 minutes. Clinic ECG and 24-h Holter records revealed the bradyarrhythmias in 8 dogs (26.66%). Of 30 dogs; in only 1, Holter monitoring could not documented cardiac arrhythmia.

but the HR_{mean} was significantly higher in clinical group of dogs (P<0.05).

Discussion

Cardiac arrhythmias in dogs are valuable markers of heart disease (Petrie, 2005; Aptekman et al., 2010). Few publications about the prevalence of clinically significant arrhythmias have been reported in dogs with heart disease (Detweiler and Patterson, 1965). In the present study, symptomatic and asymptomatic dogs with an evidence of cardiac abnormality have been evaluated for the presence of short and long term individual electrocardiographic findings. Electrocardiographic studies in detection of cardiac arrhythmias on clinic ECG have often limitations because of the short recording period and

Table 3. Association between clinic ECG and 24-h Holter records in preclinical group.
Tablo 3. Preklinik grupta klinik EKG ve 24 saat Holter kaydı arasındaki ilişki.

Breed	Body Weight (kg)	Age (years)	Sex	Cardiac Diagnosis	Arrhythmias on Clinic ECG	Arrhythmias on 24hr Holter Monitoring*
Golden Retriever	30	7	♀	SSS	SB (≤40 bpm), SP, Second-Degree AV Block (Mobitz Type I)	Tachycardia Bradycardia Syndrome (Min. HR: 38 bpm, Max. HR: 196 bpm), Second-Degree AV Block (Mobitz Type I)
Doberman Pinscher	37,3	10	♂	DCM	1 VPC (LBBB)	312 Multifocal VPCs
Boxer	26,3	13	♀	DCM	NSR	7 Multifocal VPCs, 3 RonT, 1 Wide Complex VTAC Episode (>3 VPC), 2 Narrow SVPCs
Miniature Pinscher	2,5	10	♀	AS	ST (≥150 bpm)	ST (Max. HR: 229 bpm)
Miniature Pinscher	4,5	1.5	♂	TD	NSR	ST (Max. HR:250 bpm), 4 Unifocal VPCs (RBBB)
Labrador Retriever	25,5	4	♀	DCM	NSR	SVT (≥150 bpm), 2 Unifocal VPC (RBBB), 9 Wide Complex VTAC Episode (>3 VPC)
Dalmatian	22	10	♂	DCM	2 Unifocal VPCs (LBBB)	440 Unifocal VPCs (LBBB, Trigeminy), 154 RonT
Miniature Pinscher	6	3	♀	MVI	NSR	406 Unifocal VPCs (RBBB, Trigeminy/Quadrigeminy)
Cavalier King Charles	5	2.5	♂	MVI	ST (≥150 bpm)	No Arrhythmia
Irish Setter	24	16	♂	MVI	NSR	113 Multifocal VPCs (Quadrigeminy), 1 Wide Complex VTAC Episode (>3 VPC), 5 Wide SVPCs
Labrador Retriever	36,5	4	♂	UPMH	NSR	3 Unifocal VPCs (RBBB), 1 RonT
Cavalier King Charles	8	5	♂	MVI	NSR	45 Unifocal VPCs (LBBB), 3 Wide SVPCs (Run)
Pekingese	4	10	♂	MVI	SB (≤70 bpm), First-Degree AV Block	SB (Mean HR:62 bpm), 1 Wide Complex VTAC Episode (>3 VPC), First-Degree AV Block
Boxer	38	8.5	♂	DCM	First-Degree AV Block	15 Unifocal VPCs (Couplet), First-Degree AV Block
Pomeranian	10	2.5	♀	TD	Second-Degree AV Block (Mobitz Type II)	SB (Mean HR:68 bpm), SP, Second-Degree AV Block (Mobitz Type II)
Cavalier King Charles	9,5	2	♂	MVI	First-Degree AV Block	First-Degree AV Block, 3 Unifocal VPCs (RBBB)
Kangal	25	0.25	♂	DCM	Fine AF with a Fast Ventricular Response (HR:196 bpm), 5 Unifocal VPCs (RBBB)	Persistent AF, 195 Multifocal VPCs (Bigeminy), 120 RonT
Labrador Retriever	32	9	♂	SSS	SB (≤40 bpm), First-Degree AV Block	SB (Min. HR: 20 bpm), SP, First-Degree AV Block, 6 Unifocal VPCs (RBBB) and Escape Beats
Terrier Type	12	8	♀	MVI	NSR	7 Unifocal VPCs (RBBB), 3 RonT
Boxer	38	7	♂	DCM	1 Unifocal VPC	145 Unifocal VPCs (Trigeminy), 26 RonT
Terrier Type	10	9	♂	MVI	NSR	3 Unifocal VPCs (RBBB), 15 Narrow SVPCs

NSR: Normal SinusRhythm, HR: Heart Rate, SB: Sinus Bradycardia, SP: Sinus Pause, ST: Sinus Tachycardia, VPC: Ventricular Premature Complex, LBBB: Left Bundle Branch Block Morphology, RBBB: Right Bundle Branch Block Morphology, SVPC: Supraventricular Premature Complex, SVT: Supraventricular Tachycardia, VTAC: Ventricular Tachycardia, AF: Atrial Fibrillation. *Artefacts were excluded.

transient nature of the arrhythmias. Recording of 2 minute rhythm on clinic ECG comprises only 0.14% of the daily rhythm strip at 100 bpm and stress induced activation of sympathetic tone suppresses the underlying arrhythmias (Miller et al., 1999; Petrie, 2005). In the present study, 11 dogs (36.66%) with NSR on 2 minute clinic ECG had significant changes in arrhythmias on 24-h Holter monitoring that confirmed the higher sensitivity of Holter monitoring in detecting cardiac arrhythmias. However, it was thought that the causes of NSR on clinic ECG were probably associated with stress induced sympathetic activation in hospital setting, transient nature of the arrhythmias or short recording period of the clinic ECG.

The presence of supraventricular and ventricular arrhythmias may play a significant role for cardiac symptoms and the risk of increased sudden death in dogs with MVI (Sisson et al., 1999; Crosara et al., 2010). Borgarelli et al. (2008) has reported the cardiac arrhythmias detected by clinic ECG in 17 (3%) of 558 dogs with MVI. Remaining dogs (97%) had no evidence of cardiac arrhythmias on clinic ECG. Of 558 dogs; 302 (54.1%) had asymptomatic disease (class I I SACHC) and 256 (45.8%) were symptomatic in class II and III ISACHC. In this study, 7 (53.8%) of 13 dogs with MVI had no evidence of cardiac arrhythmias on clinic ECG but those had significant changes in cardiac arrhythmias on Holter monitoring. Of 13 dogs with MVI; 8 (61.5%) had asymptomatic disease (class I I SACHC) and 5 (38.4%) were symptomatic in class II and III ISACHC. The differences in the present study compared to report of

Borgarelli et al. (2008) were related to the population rate. The preliminary data obtained the study of Borgarelli et al. (2008) has indicated that ventricular arrhythmias detected by Holter monitoring were more common than thought in dogs with MVI. In addition, Falk et al. (2006) has reported that sudden death is uncommonin dogs with MVI. In the study presented here, the presence of ventricular arrhythmias (range 2-406) recorded with Holter monitoring in all dogs with MVI who had previosly detected NSR on clinic ECG corroborated the previous reports. We did also not observed the sudden death in dogs with MVI during the study similar to the report by Falk et al. (2006). Although cardiac arrhythmias are not common in dogs with early stages MVI (Olsen et al., 1999), increased frequency of arrhythmias regardless of the class of heart failure have been reported in dogs with MVI (Crosara et al., 2010). However, several studies in dogs with MVI and DCM have shown that volume overload resulting from progressive heart disease causes atrial remodeling and supraventricular arrhythmias (Verhuele et al., 2003; O'Grady and O'Sullivan, 2004). In the study presented here, 3 asymptomatic dogs with MVI had SVPC on 24-h Holter monitoring, but in the same dogs clinic ECG revealed NSR. SVT detected by 2 minute clinic ECG was also present in 2 symptomatic dogs with MVI. Of 13 dogs with MVI, only 1 from preclinical group had no cardiac arrhythmias on Holter monitoring. However, 1 dog in preclinical group and 3 dogs in clinical group presented with persistent atrial fibrillation on clinic ECG. Of 4 dogs, 1 with DCM in clinical group had paroxysmal atrial fibrillation lasting 48 minutes on Holter

Table 4. Association between clinic ECG and 24-h Holter records in clinical group.
Tablo 4. Klinik grupta Klinik EKG ve 24 saat Holter kaydı arasındaki ilişki.

Breed	Body Weight (kg)	Age (years)	Sex	Cardiac Diagnosis	Arrhythmias on Clinic ECG	Arrhythmias on 24hr Holter Monitoring*
Shih Tzu	6	15	♂	MVI	SVT (≥150 bpm)	11 Narrow SVPCs (1 Couplet)
Kangal	54	13	♂	DCM	Fine AF (HR:120bpm), 2 Narrow SVPCs (Couplet)	Persistent AF, 176 Unifocal VPCs (LBBB, Bigeminy)
Terrier Type	12	13	♂	MVI	SVT (≥150 bpm)	10 Multifocal VPCs, 1 Wide Complex VTAC Episode (>3 VPC)
Terrier Type	10,5	6	♂	MVI	NSR	2 Unifocal VPCs (RBBB), 3 Narrow SVPCs
Beagle	26	5	♀	MVI	NSR	75 Multifocal VPCs (Bigeminy)
German Shepherd Dog	50	7	♂	HS	First-Degree AV Block, 5 Unifocal VPCs (2 Couplet, LBBB)	First-Degree AV Block, 137 Multifocal VPCs, 3 Wide SVPCs
Kangal	45	13	♂	TVI	Fine AF with a Fast Ventricular Response (HR:195 bpm), St Depression	Persistent AF, 190 Multifocal VPCs (Trigeminy)
Terrier Type	10	12	♂	MVI	Second-Degree AV Block (Mobitz Type II), St Depression, Fascicular Block Pattern	Second-Degree AV Block (Mobitz Type II), SB (Mean HR:59 bpm), Fascicular Block Pattern
German Shepherd Dog	26	11	♂	DCM	Coarse AF with a Fast Ventricular Response (HR:240 bpm)	Paroxysmal AF (lasted 48 minutes), 250 Multifocal VPCs

NSR: Normal Sinus Rhythm, HR: Heart Rate, SB: Sinus Bradycardia, SVT: Supraventricular Tachycardia, VPC: Ventricular Premature Complex, LBBB: Left Bundle Branch Block Morphology, RBBB: Right Bundle Branch Block Morphology, SVPC: Supraventricular Premature Complex, VTAC: Ventricular Tachycardia, AF: Atrial Fibrillation. *Artefacts were excluded.

records.

Ventricular arrhythmias detected by Holter monitoring in dogs predisposed to cardiomyopathy have been commonly reported (Meurs et al., 2001a; Martin et al., 2010). However, occult cardiomyopathies in dogs are identified by early ventricular arrhythmias detected by Holter monitoring (Calvert et al., 2000; Martin et al., 2010; Wess et al., 2010). Most affected dogs with occult cardiomyopathy have often a normal ECG and echocardiographic examinations during occult phase but, can be at risk of sudden death (Meurs, 2004). Therefore, early recognition of dogs with occult cardiomyopathy has an importance for both therapeutic course and breeding programmes. However, increased complexity of ventricular arrhythmias may predict sudden death in dogs with DCM (Yamaki, 2008). Martin et al. (2010) has reported that the presence of VPCs compared to atrial fibrillation and SVPC were significantly associated with survival in dogs with DCM. In the study presented here; although survival were not evaluated, 24-h Holter monitoring compared to 2 minute clinic ECG defined the significant changes in cardiac arrhythmias of Boxers, German Shepperd Dog, Doberman Pinscher, Dalmatian and Labrador Retriever with DCM. Of 30 dogs, 7 (23.33 %) with DCM in preclinical group and 2 (6.66%) with DCM in clinical group had significant changes in VPCs detected by Holter monitoring. In both groups of dogs with heart disease, 24-h Holter monitoring revealed a high number of VPCs with increased complexity and periods of VTAC. Considering clinic ECG recordings, no dogs in the present preclinical and clinical groups demonstrated nonsustained VTAC but the Holter findings of nonsustained VTAC episodes were found in 16.66% of the total population. In addition, this study provides the first evaluation of long term cardiac arrhythmias in Kangal dogs. Kangal dog (from preclinical group) with 5 unifocal VPCs on clinic ECG had 315 multifocal VPCs on Holter monitoring. In clinical group of dogs, 2 Kangal dogs with no VPC on clinic ECG had significant changes in VPCs on Holter monitoring.

The prognostic role of VPCs/24 h in dogs with DCM have been described (Meurs et al., 2001a; Meurs, 1999). Many cutoff values of VPCs have also been suggested to differentiate the affected and nonaffected dogs. Boxer dogs with at least 50 (Meurs, 1999), 100 (O'Grady and O'Sullivan, 2004) or 1000 VPC/24 h (Palermo, 2011) have been considered affected in the studies. However; in a study of 50 healthy dogs, 24-h Holter records revelaed 1-24 VPCs (Meurs, 2001b). In the study presented here, 3 asymptomatic Boxers with DCM had 10, 15 and 171 VPCs/24 h (included R on T), respectively. The data obtained from 3 Boxers were consistent with the reports previously described. Although the presence of 1 VPCs on clinic ECG is a strong indicator for further evaluation including Holter in dogs with occult cardiomyopathy, clinic ECG could not replace Holter monitoring in detecting occult cardiomyopathy (Wess et al., 2010). In the present study; of 7 (23.33%) asymptomaticdogs with DCM, 4 (13.33%) had at least 1 VPC on clinic ECG. During the Holter studies, VPC frequency ranged from 171 to 594 in the same dogs.

The ECG HR_{mean} in groups was significantly higher compared to HR_{mean} recorded with Holter monitoring. It is not a surprising result because of the stress in hospital setting during the clinical examination. Similar results have been reported by Miller et al. (1999) in a retrospective study in dogs. However, Holter results showed no differences between the groups for HR_{min} and HR_{max}, but the HR_{mean} was significantly higher in clinical group (P<0.05). Raising of heart rate associated with the severity of heart disease have been reported for the reasons of triggered activity and high frequency of sinus depolarization (Opie and Solaro, 2004; Song et al., 2006). In the present study, higher HR_{mean} recorded with Holter monitoring in clinical group

corroborates the previous reports.

Holter monitoring documented the increased arrhythmia detection and significant changes in cardiac arrhythmias in dogs with heart disease when the clinic ECG is not diagnostic. Similar findings have been reported in dogs (Miller et al., 1999; Wess et al., 2010). The major limitation in the study was the small population size of dogs with various heart disease. Population size of dogs with various heart disease may effectthe HR and arrhythmias in groups.

In conclusion, this study represents Holter-detected significant changes in cardiac arrhythmias. 24-h Holter monitoring provides the best assessment of the presence, frequency and complexity of the cardiac arrhythmias in symptomatic and asymptomatic dogs with heart disease.

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