



FULL PAPER

Internal Medicine

Prominent J wave in cats with hypertrophic cardiomyopathy

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ABSTRACT. The J wave has never been documented in the electrocardiogram (ECG) of cats presenting with hypertrophic cardiomyopathy (HCM). The present study aimed to describe the presence, morphology, amplitude, and duration of J waves in cats with HCM. It included 20 apparently healthy cats and 45 cats diagnosed with HCM based on clinical, echocardiographic, ECG, and radiographic examination. The cats were of different breeds (Persian: 40, domestic short hair: 21, Siamese: 4), ages (6.01 ± 4.34 years), sexes (male: 33, female: 32), and weights (3.30 ± 1.51 kg). The J wave was absent in the ECGs of the healthy population, but was detected in 29 out of 45 cats with HCM (63%). The J waves were observed at the QRS-ST junction in more than one limb lead of the ECG. Only positive deflections with an amplitude ≥ 0.05 mV were included, as measured by an ECG ruler in three consecutive heart cycles. The J waves were mainly present in leads II (n=20) and III (n=16), with amplitudes of 0.06 ± 0.02 and 0.08 ± 0.03 mV; their mean (\pm SD) duration was 0.16 ± 0.05 msec in lead II and 0.18 ± 0.05 msec in lead III. They occurred in both notched and slurred morphologies, with the latter being more common. In conclusion, J waves were a common finding in the ECGs of cats with HCM.

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The J wave (Osborn wave) is a positive deflection found at the R-ST junction in the electrocardiograph (ECG) [13, 18, 19]. It has a characteristic morphology of slurring or notching [8, 18, 22], which can be partially or completely buried in the QRS complex [5, 22].

Previous reports have identified the presence of the J wave as a normal variant in humans, baboons, and dogs [2, 5, 19], while other studies have observed the J wave under pathological conditions, including hypercalcemia [16, 19, 23], ischemic heart disease [11, 15, 25], left ventricular hypertrophy [18, 25], and fatal arrhythmia. Characteristic J wave abnormalities have been reported in Brugada syndrome—an inherited arrhythmic condition characterized by ST elevation, negative T wave in the right chest leads, and no evidence of structural cardiac abnormalities [3, 14]—as well as in early repolarization syndrome, in which J waves are associated with life-threatening cardiac events [19, 20, 25], as they are in human patients with hypertrophic cardiomyopathy [11]. The J wave occurs as a normal variant in 43% of geriatric dogs without cardiovascular disease, as well as in 29.1% of geriatric dogs with cardiovascular diseases [16, 19].

There is no single explanation for the causal mechanism of J waves under normal and pathological circumstances. Previous reports have suggested that J waves are caused by a difference in action potential between the ventricular endocardium and epicardium during early repolarization, when a prominent transient outward current in the ventricular epicardium elicits a transmural voltage gradient [16, 21].

Hypertrophic cardiomyopathy (HCM) is the most commonly diagnosed heart disease in cats. It can lead to congestive heart failure, thromboembolism, and sudden death [1, 6, 9]. The ECG findings in cats with HCM include left anterior fascicular block, a pattern of left ventricular enlargement (QRS complex >0.04 sec and R wave >0.9 mV), intermittent ventricular premature complexes (VPCs), and sinus arrhythmia [1, 9, 10].

To our knowledge, no available literature describes the presence, morphology, amplitude, and duration of the J wave in cats with HCM. We hypothesized that, as in humans, prominent J waves could occur in the ECG of cats with HCM. The present study aimed to evaluate the overall presence of J waves in cats with HCM.

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MATERIALS AND METHODS

Animals

The present prospective study involved 65 preowned cats admitted to the Surgery Department at the Faculty of Veterinary Medicine, Cairo University, as well as to some private clinics in Cairo. The cats were of different breeds, ages, sexes, and weights. All were examined without sedation by physical examination, indirect blood pressure measurement, echocardiography, and blood sampling.

All study procedures were performed according to the guidelines of the animal use and ethical committee of the Faculty of Veterinary Medicine, Cairo University. The owners were aware that their cats would be used for research purposes and signed a consent form.

Inclusion criteria

Clinically healthy cats were included on the basis of normal physical examination, echocardiographic measurements, radiographic examination, and blood sampling. Cats with HCM were diagnosed according to guidelines for B-mode and M-mode echocardiographic measurements, as previously described [22, 24].

ECG examination

Six limb lead ECGs were recorded in each animal using a single-channel ECG machine (ECG 901A; Yasen, China). All cats were gently restrained in right lateral recumbency, with their limbs positioned perpendicular to the longitudinal axis of the body. Self-adhesive patch electrodes were attached to the palmar and plantar aspects of the fore and hind limb paws, respectively. The six standard limb leads I, II, III, aVR, aVL, and aVF were recorded at paper settings of 10 mm/mV and 50 mm/sec, with filter band settings of 0.05–150 Hz. The ECG parameters were evaluated and compared with previously reported values for cats [17, 26].

J waves were defined as an elevation at the J point or the QRS-ST junction. In canine species, the J wave is considered prominent if its amplitude is ≥ 0.1 mV in more than one limb lead. In the present study, the J wave was identified based on previously established reports in dogs, which classified the morphology of the J wave as notched, slurred, or undetermined [2, 18].

The amplitude of the J waves was measured using an ECG ruler in three consecutive heart cycles, and the mean for each cat was calculated. The J wave was considered prominent if the amplitude was ≥ 0.05 mV, starting from the baseline. The mean J wave duration was measured in three successive cycles, starting from the beginning of the transition for slurred waves and from the beginning of the positive deflection for notched waves. The start of the ST segment was considered the end of the J wave. The duration of the J wave is expressed in msec [16, 18].

Statistical analysis

Demographic data, echocardiographic data, and ECG data, including the J wave amplitude and duration, were expressed as mean \pm standard deviation (SD) and analyzed using IBM SPSS version 22.0.0.0.

RESULTS

The study population consisted of 65 cats (20 healthy [30.8%] and 45 HCM [69%]). The demographic, echocardiographic, and ECG data of the healthy and HCM groups are summarized in Tables 1 and 2, respectively. The ECG of the healthy population showed normal rhythm and P-QRS-T wave characteristics. All ECG parameters of the cats with HCM were within normal ranges, except for QRS complex duration, which was significantly longer than in the healthy cats (P<0.05). The J wave was not detected in the ECG of the healthy population, but it was identified in 29 of the 45 cats with HCM. The J wave was commonly detected in leads II and III, as shown in Table 3, and its amplitude and duration were expressed in mV and msec, respectively. The J wave was observed in both the slurred and notched morphologies (Fig. 1). The variations in J wave morphology among the six limb leads are presented in Table 4. Four cats showed both morphologies in different leads. Left anterior fascicular block, tall R waves, and sinus arrhythmias were also detected in the ECGs of the HCM population.

DISCUSSION

The present study found prominent J waves in 29 out of 45 cats with HCM (63%). These waves were completely absent in the healthy population. J waves have occasionally been associated with hypothermia in other animal species, but have not been reported in cats [4]. Previous studies have reported that HCM in cats is only represented by ECG findings related to chamber enlargement and conduction disturbances, namely left anterior fascicular block, wide QRS complexes, tall R waves, intermittent VPCs, and sinus arrhythmias [11, 15], but no previous studies have found J waves in cats with HCM. Moreover, the J wave has been detected under pathological conditions in humans, including those with HCM [25].

All ECG parameters in the healthy controls and cats with HCM were within the reference ranges [17, 26], except for QRS complex duration, which was longer in the HCM population, as in previous studies [11, 15]. The ECG parameters of the healthy population were within the normal ranges. The HCM population showed increased left ventricular dimension in diastole, greater left ventricular wall thickness in diastole, or both, as well as elevated left atrial to aortic root ratio, as in previous literature [22, 24].

While the J wave is a normal finding in healthy geriatric dogs, it was not present in the ECGs of five cats aged >7 years [16,

Group	Healthy cats	Cats with HCM
Profile data		
Number	n=20	n=45
Sex (M/F)	8/12	25/20
Weight (kg), mean \pm SD	3.06 ± 1.61	3.60 ± 1.4
Age (years), mean \pm SD	4.8 ± 4.01	7.8 ± 2.90
Breeds (n)		
Persian	10	30
DSH	6	15
Siamese	4	0
Echocardiographic data (mean ±	ESD)	
LVDD (cm)	1.40 ± 0.14	1.26 ± 0.06
LVDS (cm)	0.8 ± 0.48	1.08 ± 0.55
IVSD (cm)	0.38 ± 0.08	0.62 ± 0.11
IVSS (cm)	0.48 ± 0.08	0.95 ± 0.14
LVWD (cm)	0.42 ± 0.06	0.79 ± 0.13
LVWS (cm)	0.50 ± 0.04	0.95 ± 0.11
LA/Ao	1.12 ± 0.08	1.58 ± 0.26

 Table 1. Profile and echocardiographic data of the healthy controls and cats with hypertrophic cardiomyopathy (HCM)

n: number of cats; SD: standard deviation; DSH: domestic short hair; LVDD: left ventricular end-diastolic dimension; LVDS: left ventricular end-systolic dimension; IVSD: interventricular septum end-diastolic thickness; IVSS: interventricular septum end-systolic thickness; LVWD: left ventricular wall end-diastolic thickness; LVWS: left ventricular wall end-systolic thickness; LVWS: left ventricular wall end-systolic thickness; LA/Ao: left atrial to aortic root ratio.

Table 2. Electrocardiographic data of the healthy controls and cats with hypertrophic cardiomyopathy (HCM)

	ECG parameters							
Group	Heart rate - (bpm)	P wave		QRS complex		DD interval	OT interval	МЕА
		Amplitude (mV)	Duration (sec)	Amplitude (mV)	Duration (sec)	(sec) (sec)	(sec)	(°)
Healthy cats	172.14 ± 17.29	0.14 ± 0.06	0.028 ± 0.01	0.28 ± 0.18	0.026 ± 0.01 **	0.07 ± 0.01	0.16 ± 0.02	71.60 ± 10.38
	(145.00–200.00)	(0.05–0.20)	(0.02–0.04)	(0.05-0.60)	(0.02 - 0.03)	(0.06 - 0.08)	(0.12–0.18)	(58.00-85.00)
Cats with HCM	189.63 ± 19.24	0.16 ± 0.05	0.03 ± 0.01	0.28 ± 0.19	$0.045 \pm 0.01 ^{\ast\ast}$	0.07 ± 0.01	0.16 ± 0.05	63.75 ± 9.46
	(167.00–214.00)	(0.10-0.20)	(0.02–0.04)	(0.15–0.55)	(0.03–0.06)	(0.06–0.08)	(0.10-0.20)	(50.00-70.00)

ECG: electrocardiogram; bpm: beats per minute, MEA: mean electrical axis. *Data are expressed as mean ± SD, minimum-maximum value. **QRS duration showed significant difference between the groups.

Table 3. Amplitude and duration of J waves observed in cats with hypertrophic cardiomyopathy at each lead			
Lwava	Amplitude	Duration	

J wave	Amplitude (mV)	Duration (msec)
Lead I (n=9)	0.07 ± 0.03	0.13 ± 0.06
Lead II (n=20)	0.06 ± 0.02	0.16 ± 0.05
Lead III (n=16)	0.08 ± 0.03	0.18 ± 0.05
Lead aVR (n=5)	0.08 ± 0.03	0.13 ± 0.06
Lead aVL (n=7)	0.1 ± 0.00	0.2 ± 0.12

HCM: hypertrophic cardiomyopathy; n: number of cats. *Values are expressed as mean \pm standard deviation.

19]. However, a study should be conducted in a larger population to confirm that the J wave is not a common finding in geriatric cats. In the cats with HCM in the present study, the highest prevalence of the J wave was detected in leads II and III, as has been found in canine species [18]; however, this was inconsistent with another study in dogs reporting that the J wave was absent in lead III [22]. Indeed, the J wave has been discovered in the same leads in both humans and dogs, in whom the mean electrical axis is transmurally oriented across the left ventricle and septum [2, 23].

In the present study, the amplitude, duration, and morphology of the J wave were evaluated as in previous dog studies, as there is



Table 4.	J wave morphology observed in cats with
hypert	ophic cardiomyopathy at each lead

J wave	Slurring n (%)	Notching n (%)
Lead I (n=9)	100	0
Lead II (n=20)	57	14
Lead III (n=16)	67	33
Lead aVR (n=5)	100	0
Lead aVL (n=7)	100	0

n: number of cats.

Fig. 1. Echocardiographic (ECG) leads of six different cats diagnosed with hypertrophic cardiomyopathy. The traces show both slurred morphology (A, B and C) and notched morphology J waves (D, E and F). Paper settings were adjusted at 50 mm/sec and 10 mm/mV.

a paucity of data regarding the J wave in cats [2, 19]. However, an amplitude of 0.05 mV was used as the starting point for J wave evaluation because QRS values normally show lower voltage in cats than in dogs [26]. All cats showed either slurred or notched J waves, and four displayed both in different leads.

J waves have been found in human patients with HCM, in whom intraventricular conduction delay is often observed. Myocardial scarring usually occurs in patients with HCM, causing conduction disturbances that can lead to J wave development [25]. Previous morphometric studies in cats with HCM have detected histopathological findings comparable to those of human HCM, particularly increased interstitial fibrosis [7, 12], which may cause interventricular conduction disturbances that lead to J wave development.

The main limitation of the present study was the small population of normal cats and cats with HCM, as well as the absence of patient follow-up for future adverse cardiac events, such as fatal arrhythmias and sudden deaths that were potentially related to the J wave. Further studies should correlate the presence of J waves with different HCM stages and cardiac events in different breeds of cats with HCM.

In conclusion, the present study documented an association between J waves and HCM in cats, while J waves were absent in apparently healthy cats. Specifically, 63% of cats with HCM had J waves, which were most common in leads II and III.

POTENTIAL CONFLICTS OF INTEREST. The authors have nothing to disclose.

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