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Electrocardiographic changes in Obese Dogs

Harneet Kour[1], Ravdeep Singh[2] and Rajesh Agrawal[1]

[1]Department of Veterinary Medicine, [2]Department of Veterinary and Animal Husbandry Extension Education, Sher-e-Kashmir University of Agricultural Sciences and Technology, R.S. Pura, Jammu, PIN: 181102, Jammu and Kashmir, INDIA

Abstract

The prevalence of obesity has been progressively increasing in companion animals as well as in humans due to sedentary lifestyle and lack of exercise. Obesity itself is an independent risk factor for cardiovascular diseases but the relationship between overall obesity and heart disease is unclear till date in canines. This research aimed to evaluate the structural and functional cardiac changes by assessing the electrocardiographic variables (ECG). The study was conducted on 20 obese and 12 normal Labrador retriever dogs at Sher-e-Kashmir University of Agricultural Sciences and Technology, Jammu. The ECG findings revealed an increased ($P<0.05$) P_{dur}, PR_{int} and QT_{int} with lower ($P<0.05$) R_{amp} and T_{amp} in obese dogs. P_{amp} and QRS_{dur} were numerically higher in obese dogs. These data can be used as baseline for the interpretation of effect of obesity on the functional and morphological changes in cardiac status for further studies.

Key words: Obesity, Dogs, ECG, Labrador Retriever

In the recent past, obesity has become a major problem in humans as well as canines. Studies from various parts of the world have estimated the incidence of obesity in the dog population to be between 22 and 40% (McGreevy *et al*., 2005). Obesity is a volume expansion illness with high cardiac output, increased plasma and extracellular fluid, neuro-humoral activation, reduced urinary excretion of sodium and water, increased heart rate, elevated arterial pressure, systolic and diastolic ventricular dysfunction, and exercise intolerance (Alpert 2001). Obesity exhibits a wide variety of electrocardiogram (ECG) abnormalities, which often lead to cardiovascular events. ECG offers a quick, non-invasive clinical and research screen for the early detection of cardio vascular diseases (CVD). ECG is influenced by morphological changes like displaced heart, elevated diaphragm, hypertrophy, increased epicardial and subcutaneous adipose tissue. Hypoventilation, increased heart rate, cardiac output and blood pressure (Bach *et al*., 2007) are induced by obesity. ECG detects not only the disturbances of cardiac rate and rhythm but also cardiac enlargement, myocardial disease, ischemia, pericardial diseases, certain electrolyte imbalances and some drug toxicities (Gugjoo *et al*., 2013a). ECG changes include increased PR, QRS, QT interval, low QRS amplitude, ST abnormality, flattened T wave (Poirier *et al.*, 2006) and malignant arrhythmias due to increased sympathetic activity (Simonyi, 2014). These ECG changes are reversible with weight loss (Neto *et al*., 2010).The purpose of the study reported herein was to investigate possible structural and functional cardiac changes in obese dogs with electrocardiogram.

Materials and Methods

The study was carried out in the Division of Veterinary Medicine, Faculty of Veterinary Sciences and Animal Husbandry, SKUAST, Jammu, R.S. Pura and Private Veterinary Clinics in Jammu. About 20 obese and 12 clinically healthy Labrador dogs were included in the study. A complete history of patient's description regarding age, gender, deworming and vaccination status was gathered from owner. Primary concern of the owner about dog, main symptoms observed, other systemic diseases and symptoms having a potential influence on the circulatory system were also recorded. Examination of patient included present status of appetite, water intake, vomiting, defecation, urination, behaviour, confirmation, posture or gait, syncopal attacks, cyanosis, enlargement of thyroid gland, exercise intolerance etc. Heart rate and respiration rate were recorded. A 12-lead standard ECG recorder (BPL CARDIART-6208 VIEW, BPL Limited, India) under all standard prescribed conditions was used in the present study for evaluation of heart rate, amplitude of P, R and T waves, duration of P wave and QRS complex and PR and QT intervals and cardiac status by placing the dogs in right lateral recumbency.

*Corresponding author: kourharneet13@gmail.com

Estimation of obesity and body fat

The approximate ideal body weight of Labrador retrievers was set as 65 to 80 lb (29.48–36.28 kg) for males and 55 to 70 lb (25–31.75 kg) for females as per Burr *et al*. (2002). The dogs having body weight in excess of 20–25 % were considered as obese. Body fat percentage (BF%) of dogs was calculated using gender specific formula as given by Burkholder and Toll (2000) and Mawby *et al*. (2004).

A. Male body Fat (%) = -1.4 (HS) + 0.77 (PC) + 4

B. Female body Fat (%) = -1.7 (HS) + 0.93 (PC) + 5

PC (Pelvic circumference) and HS (Hock to Stifle)

The normal reference ranges for body fat percentage were set at 15%–22% for male (neutered/non-neutered) dogs and at 15%–25% for female (nonspayed/spayed) dogs. Body fat percentage exceeding 22% in males and 25% for females was considered overweight and obese (Li *et al*., 2012).

Results and Discussion

Electrocardiographic variables

Electrocardiogram (ECG) abnormalities are associated with an increased risk of adverse cardiovascular outcomes, including high resting heart rate (HR, beats per min), prolonged PR interval, QRS duration and QT interval and abnormal shift in electrocardiographic axes (O'Neil *et al*., 2010).The mean values of the vital parameters and ECG variables for control and obese dogs are presented in Table 1 and Table 2 (Fig. 1 and Fig 2), respectively. In the current study, it was recorded that the heart rate (102.90±4.29 vs. 79.58±1.64) in obese dogs was higher (P<0.05) as compared to normal dogs. Similar changes like increased heart rate have been reported earlier by various workers (Alpert *et al*., 2001; Rhamouni *et al*., 2005; Fraley *et al*., 2005; Mutiso *et al*., 2014). A prospective study has reported that resting heart rate has a significant positive association with cardiovascular and all-cause mortalities (Okamura *et al*., 2004). A high heart rate is a strong indicator of cardiovascular mortality while a low heart rate is associated with an improved outcome. A non significant (P>0.05) increase in the mean value of P_{amp} was observed in obese dogs (0.3400±0.01000 vs. 0.3125±0.01754 mv) suggestive of right atrial enlargement (Tilley, 1992). P_{dur} showed a significant (P<0.05) increase in obese dogs (0.0475±0.00250 vs. 0.0400± 0.00 sec). P wave duration was longer in obese dogs that might be indicative of left atrial enlargement leading to atrial fibrillation. Wang *et al*. (2004) reported that left atrial enlargement is associated with 50% risk of atrial fibrillation and flutter. In obese patients, left atrial enlargement and electrical instability may be caused by elevated plasma volume, ventricular diastolic dysfunction and enhanced neurohormonal activity. These findings coincided with Suthar *et al*. (2016), Russo *et al*. (2008) and Neto *et al*. (2010) who also reported an increase in P_{dur} in obese dogs.

A highly significant (P<0.05) longer PR_{int} was observed in obese (0.1215±0.00499 vs. 0.0938±0.00308 sec) dogs. This is in accordance with the study of Alpert *et al*., (2000), Alpert (2001) and Sun *et al*. (2013). They noted progressive increase in PR interval duration with increasing severity of obesity. A 10% increase in obesity was manifested in an increase in PR interval of 0.5 ms (Fraley *et al*., 2005). Prolonged PR interval indicated atrioventricular (AV) block which refers to alteration of impulse conduction through the AV node from the atria to the ventricles. The conduction time is increased and is recognized on an ECG as an increased PR interval (Kittleson, 2015). It is associated with an increased risk of heart failure (HF), incident atrial fibrillation (AF), endothelial dysfunction and activation of vascular repair (Magnani *et al*., 2013; Cheng *et al*., 2009; Chan *et al*., 2013). QRS_{dur} although non-significant was higher in obese dogs (0.0510±0.00176 sec) as compared to normal ones (0.0500±0.00213 sec). Similar results were observed by Sun *et al*. (2013). QRS duration prolongation is a potential

Table 1: Vital parameters in normal and obese dogs

Parameter	Normal dogs (n= 12)	Obese dogs (n = 20)	P value
Heart rate (beats/ min)	79.58±1.64[a] (72 - 93)	102.90±4.29[b] (81 – 145)	0.000
Respiration rate (breathe/ min)	34.92±1.68[a] (26 - 45)	51.30±2.15[b] (30 – 70)	0.000

Figures in parenthesis indicate range, Different superscripts [a,b] indicate significant difference at P<0.05 within a row

Table 2: Electrocardiographic variables of normal and obese dogs

ECG variables	Normal dogs (n= 12)	**Obese dogs (n = 20)**	**P value**
P_{amp} (mv)	0.3125±0.01754[a]	0.3400±0.01000[a]	0.152
P_{dur} (sec)	0.0400±0.00000[a]	0.0475±0.00250[b]	0.028
PR_{int} (sec)	0.0938±0.00308[a]	0.1215±0.00499[b]	0.000
R_{amp} (mv)	2.4583±0.12995[a]	1.9000±0.07797[b]	0.000
QRS_{dur}(sec)	0.0500±0.00213[a]	0.0510±0.00176[a]	0.725
QT_{int} (sec)	0.1433±0.00310[a]	0.1747±0.00699[b]	0.002
T_{amp} (mv)	0.4750±0.01569[a]	0.2555±0.02167[b]	0.000

Figures in parenthesis depict range, Different superscripts [a,b] indicate significant difference at P< 0.05 within a row

marker of cardiac structural and functional abnormalities, including left ventricular systolic dysfunction, that may predispose individuals to an increased risk of heart failure (Dhingra *et al*., 2005; Ilkhanoff *et al*., 2012). Similarly QT_{int} in obese dogs was also significantly (P<0.05) higher (0.1747±0.00699 vs. 0.1433±0.00310 sec) which is caused by heightened sympathetic activity (Mathew *et al*., 2008). This prolongation in the present study was seen in agreement with earlier studies conducted by various researchers (Papaioannou *et al*. 2003; Fraley *et al*., 2005; Calejo, 2013; Simonyi, 2014). The reason for this could be that central obesity particularly is associated with delayed ventricular repolarisation as designated by prolongation of the QT interval (Girola *et al*., 2001; Seyfeli *et al*., 2006, Russo *et al*. (2007)). Several mechanisms have been suggested for QT prolongation: autonomic system imbalance and autonomic neuropathy (Zipes and Miyaki, 1990), mutations of genes affecting cardiac ion channels involved in cardiac repolarization (Roden and Spooner, 1999), nonconducting scar tissue resulting from myocardial infarction, high glucose level, obesity and ventricular hypertrophy. In obese dogs, R_{amp} and T_{amp} were significantly lower (1.9000±0.07797 vs. 2.4583±0.12995 mv and 0.2555±0.02167 vs. 0.4750±0.01569 mv, respectively). The decrease in R wave and T wave amplitude was attributable to changes related to impaired myocardial depolarisation and repolarisation as well as changes in cardiac morphology as supported by Fraley *et al*., (2005); Poirier *et al*., (2006) and Mutiso *et al*. (2014). The decrease in R wave amplitude in the present study along with prolonged QRS and PR intervals could be due to high serum potassium concentrations in obese dogs. The electrocardiographic changes in the present study were in line with Bhide *et al*. (2014) who reported a statistically significant increase in heart rate, PR interval, QT interval and decrease in QRS axis in overweight and obese individuals as compared to normal individuals. Because obesity is linked to left ventricular hypertrophy, diastolic dysfunction, and aortic stiffness, all independent predictors of cardiovascular mortality (Sutton-Tyrrell *et al*., 2001; Bhatia *et al*., 2010) and increased mortality, this hypertrophic response provides a potential mechanism

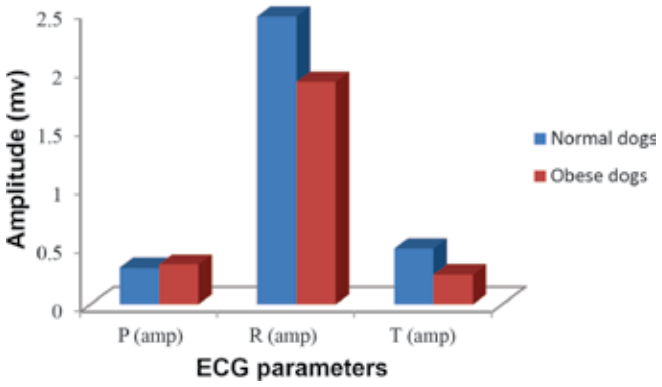

Fig. 1: Comparison of P_{amp}, R_{amp} and T_{amp} in normal and obese clinical cases

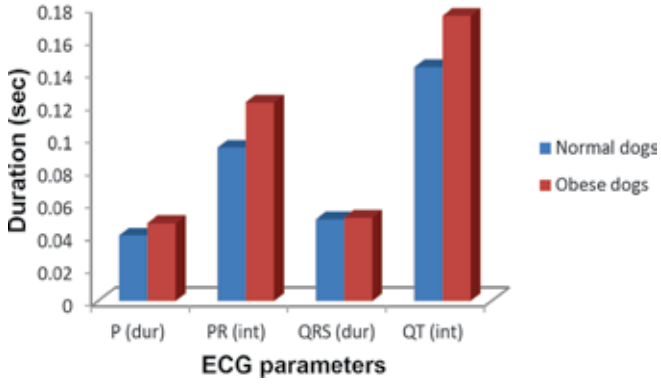

Fig. 2: Comparison of $P_{dur,}PR_{int}$, QRS_{dur} and QT_{int} in normal and obese clinical cases

by which obesity modulates cardiovascular risk.

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