SHORT COMMUNICATION

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Effect of medetomidine on left ventricular outflow tract velocity in cats: A Doppler echocardiography study

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Abstract

The purpose of the present study was to evaluate effects of medetomidine on left ventricular outflow tract (LVOT) velocity in domestic short-haired cats. Eighteen healthy adult male domestic short-haired cats were used for this study. All animals were client-owned. Echocardiography machine with 7.50 MHz transducer was used. Specific veterinary two-dimensional and pulse-waved echocardiogram images in apical five chamber right parasternal view were obtained and blood velocity in LVOT was calculated. After baseline echocardiographic recordings, 0.04 mg kg^{-1} of medetomidine was intramuscularly administered to each animal and LVOT velocity was calculated after 15 (T15), 50 (T30) and 80 (T80) min following drug administration. The LVOT velocity values (mean ± SEM) of cats in baseline were 1.06 ± 0.04 m sec^{-1}. There were significant differences between baseline and T15 and T30 regarding mean LVOT values. Age and weight had no significant effect on LVOT velocity values. The LVOT velocity values of T15, T50 and T80 were 0.77 ± 0.04, 0.80 ± 0.02 and 0.96±0.03 m sec^{-1}, respectively. Our findings revealed significant decrease in mean LVOT velocity up to 50 min following medetomidine administration. The present study determined normal LVOT velocity range for a small population of cats before and after intra-muscular medetomidine administration.

Introduction

Left ventricular outflow tract (LVOT) is a portion of left ventricle connecting to the aorta and passing blood to it.[1] Hypertrophic cardiomyopathy (HCM) is the most common cardiac disease in cats. The LVOT obstruction is a common finding in cats with HCM.[2] The LVOT obstruction leads to increase of left ventricular pressure and systolic wall tension.[3] Myocardial ischemia and sudden death were reported as consequences of LVOT obstruction in humans and cats.[2,4] Measurement of LVOT velocity is an useful index in monitoring of LVOT obstruction in cats.[1]

Medetomidine is a member of alpha-2 receptors agonists generally used in animals as pre-anesthetic or sedative agents. Medetomidine has side effects such as vomiting, bradycardia and decreased respiration.[5] It elevates systolic blood pressure temporary after administration; but, it decreases heart rate leading to decreased systolic blood pressure.[6,7] Use of sedation in cats for variety of procedures is not uncommon, especially in uncooperative cats. Effect of sedatives on Doppler echocardiographic parameters is important, especially when the procedure is performing in sedated cats.

A few studies have been performed on the effects of medetomidine on the echocardiographic variables in animals. Lamont *et al.* have been reported the effect of medetomidine on LVOT obstruction in cats.[1] In dogs, medetomidine has been reported to have little impact on echocardiographic parameters and maybe useful in uncooperative dogs.[8]

To the best of author's knowledge, effects of medetomidine on LVOT velocity in healthy cats have not been studied previously. The purpose of the present study was to evaluate the effects of specific dosage of medetomidine on pressure gradient in LVOT in domestic short-haired cats.

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Materials and Methods

Eighteen healthy adult male domestic short-haired cats were used for this study. All animals were client-owned. Consent form was signed by clients prior to enrollment of their cats in the study. A full physical examination, electrocardiography, complete blood count and serum biochemical profile were performed prior to the admission to the study.

After 12 hr food and fluids withholding, each animal was gently physically restrained in a right lateral recumbency. Body hair between 4th and 9th ribs was shaved and ultrasonography gel was applied. Specific veterinary echocardiography machine (Z5 Vet; Mindray, Mahwah, USA) with 7.50 MHz transducer was used. Two-dimensional and pulse-waved echocardiogram images in apical five chamber right parasternal view were obtained and blood velocity in LVOT was calculated (Fig. 1). After baseline echocardiographic recordings, 0.04 mg kg^{-1} of medetomidine (Zoetis, Parsippany, USA) was intra-muscularly administered to each animal and LVOT velocity was calculated after 15 (T15), 50 (T30) and 80 (T80) min following drug administration.

Statistical analysis was performed using a SPSS Software (version 23.0; SPSS Inc., Chicago, USA). A one-sample Kolmogorov–Smirnov was used to test the data for normality. A repeated measure ANOVA with Bonferroni post hoc correction was used to compare the means of LVOT values obtained. A *p*-value less than 0.05 was considered to be statistically significant.

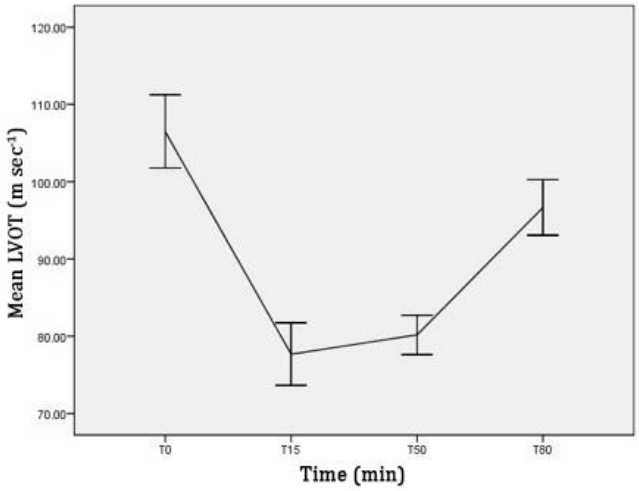

Fig. 1. Two-dimensional and pulse-waved echocardiogram images in long-axis five chamber right parasternal window for assessment of left ventricular outflow tract (LVOT) velocity in a cat. Error bars= ± 1 SE.

Results

Age and weight (mean ± SEM) of animals were 3.10 ± 0.20 year (ranging from 2-5 years) and 3.70 ± 0.10 kg (ranging from 3.00 - 4.90 kg), respectively. The LVOT velocity values (mean ± SEM) of cats in baseline were 1.06 ± 0.04 m sec^{-1}. There were significant differences between baseline and T15 and T30 regarding mean LVOT values ($p < 0.001$), (Fig. 2). Age and weight had no significant effect on LVOT velocity values ($p > 0.05$). The LVOT velocity values of T15, T50 and T80 were 0.77 ± 0.04, 0.80 ± 0.02 and 0.96 ± 0.03 m sec^{-1}, respectively. Figure 3 shows the effect of medetomidine on LVOT velocity in each age group.

The highest velocity at the baseline and greater reduction in velocity following medetomidine administration compared to the baseline were recorded in cat no. 2. Baseline velocity was evaluated as 1.38 m sec^{-1} and after 15 min, the recorded velocity in this cat was 0.72 (47.70% reduction in LVOT velocity). In some cats, the LVOT velocity restored to the baseline values after 80 min.

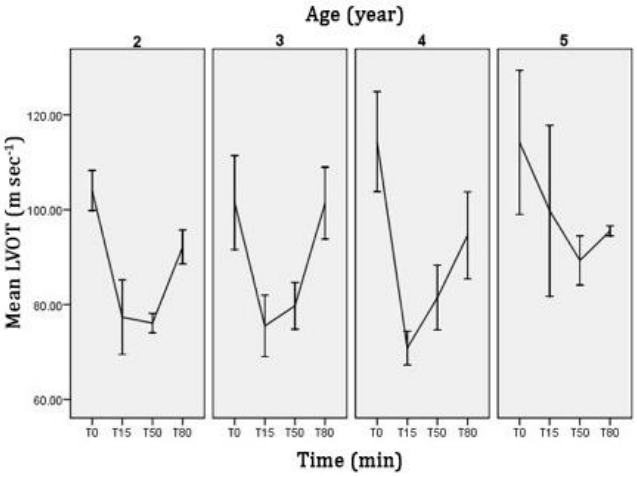

Fig. 2. Mean left ventricular outflow tract velocity (LVOT) in 18 adult male domestic short-haired cats at the baseline and 15, 50 and 80 min after intra-muscular medetomidine administration. Error bars= ± 1 SE.

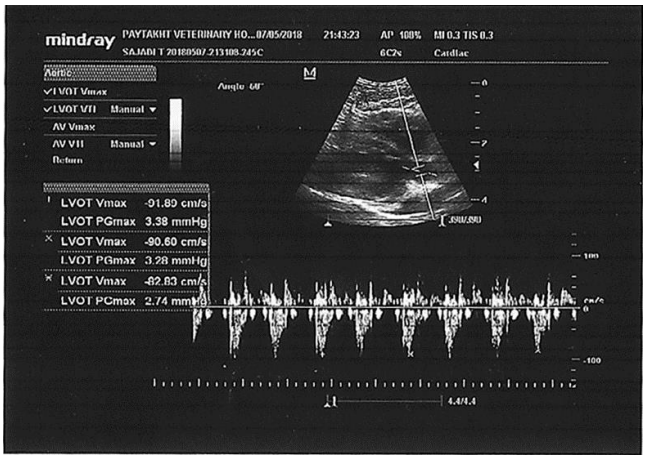

Fig. 3. Mean left ventricular outflow tract velocity in an adult male domestic short-haired cats based on the age at the baseline and 15, 50 and 80 min after intra-muscular medetomidine administration.

Discussion

This study revealed significant decrease in mean LVOT velocity up to 50 min following medetomidine administration. Increased vascular resistance and heart rate decrease are possible reasons for reduction in LVOT velocity.[1,7]

Lamount *et al.* have administered 0.20 mg kg^{-1} of medetomidine intramuscularly to cats with LVOT obstruction. Peak LVOT velocity in their study was 1.10 ± 0.10 m sec^{-1}, which was significantly reduced in comparison with baseline values (4.70 ± 0.40 m sec^{-1}).[1] Cats in their study had ventricular hypertrophy. In our study, cats had normal cardiovascular function. The LVOT velocity after medetomidine administration was reported 80 min following drug administration as 0.90 ± 0.03 m sec^{-1}. This difference was not significantly different from baseline (1.06 ± 0.04 m sec^{-1}).

Saponaro *et al.* have administered 0.20 mg kg^{-1} of medetomidine intra-muscularly to 10 dogs and evaluated cardiovascular effects of this drug after 15, 50 and 80 min following drug administration.[8] In dogs, baseline LVOT velocity was reported as 1.25 ± 0.18 m sec^{-1}. Following medetomidine administration, LVOT velocity was reported as 1.08 ± 0.24, 1.13 ± 0.15 and 1.09 ± 0.24 m sec^{-1}, respectively. Peak LVOT velocity after medetomidine administration was observed 50 min following drug administration. In dogs, medetomidine was reported to increase right ventricular afterload.[8] Findings of our study and trends of LVOT velocity following medetomidine administration in the present study were in agreement with previous study in dogs.[8] Low dose of medetomidine was used in our study population. More decrease of LVOT velocity is expected in cats, in the case of using high dose of medetomidine as 0.20 mg kg^{-1}.

In normal golden retriever puppies and golden retriever puppies affected by sub-aortic stenosis , LVOT velocity was reported as 1.70 ± 0.26 and 2.63 ± 0.91 m sec^{-1}, respectively.[9]

Normal ranges for peak LVOT velocity in horses have been determined between 0.78 to 1.15 m sec^{-1}. This value was reported to increase up to 2.00 m sec^{-1} in disturbed blow flow conditions such as aortic insufficiency.[10]

In humans, peak LVOT velocity in healthy subjects was reported as 0.98 ± 0.16 m sec^{-1}.[11] Baseline values obtained in the present study were similar to previously published values in dogs and humans.[8,11]

Cats of our study were client-owned and living with human for long period of time. They were comfortable at the time of examination and restraint. No typical sign of stress was observed in our study population. Effect of stress on LVOT velocity in animals has not been reported formerly. In healthy humans, mean LVOT velocity was reported at rest and stress as 1.00 ± 0.10 and 1.70 ± 0.20 m sec^{-1}, respectively.[12] Stress increases the LVOT velocity.

In cats with LVOT obstruction, LVOT velocity was reported as high as 5.10 m sec^{-1}. This value may increase at the time of stress in cats. Fortunately, medetomidine completely eliminates LVOT obstruction and decreases LVOT velocity to 1.20 m sec^{-1}.[1]

The LVOT obstruction leads to increase of LVOT velocity. In cats, LVOT obstruction is a consequence of obstructive form of HCM.[13] In humans, hypertrophic obstructive cardiomyopathy, hypertensive left ventricular hypertrophy and post-open heart surgery are main reasons of LVOT obstruction and LVOT velocity increase.[14]

In a recent study, ivabradine and atenolol have been used to reduce LVOT velocity in cats with HCM reporting that atenolol is more potent to reduce velocity of LVOT in cats.[15] Mean reduction with ivabradine was reported as 0.32 m sec^{-1}, which was lower than what observed in the present study.

The combination of carvedilol-disopyramide has been also used to decrease LVOT velocity in a cat with HCM. This combination reduced LVOT velocity by 0.70 m sec^{-1} after 141 days of therapy.[16]

Reportedly, atenolol decreased LVOT velocity by 1.70 m sec^{-1}, which was a significant reduction in velocity of LVOT. However, the blood pressure was reported to remain unchanged.[17]

The present study determined normal LVOT velocity range for a small population of cats before and after administration of intra-muscular medetomidine. Furthur studies regarding evaluation of LVOT velocity in cats with different cardiopulmonary diseases are warranted.

Conflict of interest

The authors do not have any potential conflicts of interest to declare.

References

1. Lamont LA, Bulmer BJ, Sisson DD, et al. Doppler echocardiographic effects of medetomidine on dynamic left ventricular outflow tract obstruction in cats. J Am Vet Med Assoc 2002; 221(9):1276-1281.
2. Kittleson MD, Meurs KM, Munro MJ, et al. Familial hypertrophic cardiomyopathy in maine coon cats: an animal model of human disease. Circulation 1999; 99(24):3172-3180.
3. Sherrid MV. Dynamic left ventricular outflow obstruction in hypertrophic cardiomyopathy revisited: significance, pathogenesis, and treatment. Cardiol Rev 1998;6(3):135-145.
4. Maron BJ, Fananapazir L. Sudden cardiac death in hypertrophic cardiomyopathy. Circulation 1992; 85(1 Suppl): I57-63.
5. Plumb DC. Plumb's veterinary drug handbook. 9th ed. Ames, USA: Wiley-Blackwell; 2018; 854-857.

6. Golden AL, Bright JM, Daniel GB, et al. Cardiovascular effects of the alpha2-adrenergic receptor agonist medetomidine in clinically normal cats anesthetized with isoflurane. Am J Vet Res 1998; 59(4):509-513.
7. Lamont LA, Bulmer BJ, Grimm KA, et al. Cardiopulmonary evaluation of the use of medetomidine hydrochloride in cats. Am J Vet Res 2001; 62(11):1745-1749.
8. Saponaro V, Crovace A, De Marzo L, et al. Echocardiographic evaluation of the cardiovascular effects of medetomidine, acepromazine and their combination in healthy dogs. Res Vet Sci 2013; 95(2):687-692.
9. Javard R, Bélanger M-C, Côté E, et al. Comparison of peak flow velocity through the left ventricular outflow tract and effective orifice area indexed to body surface area in Golden Retriever puppies to predict development of subaortic stenosis in adult dogs. J Am Vet Med Assoc 2014;245(12):1367-1374.
10. Blissitt K, Patteson M. Evaluation of cardiac murmurs in horses 2. Echocardiography. In Pract 1996; 18(9):416-426.
11. Kupari M, Koskinen P. Systolic flow velocity profile in the left ventricular outflow tract in persons free of heart disease. Am J Cardiol 1993;72(15):1172-1178.
12. Thorstensen A, Dalen H, Amundsen BH, et al. Peak systolic velocity indices are more sensitive than end-systolic indices in detecting contraction changes assessed by echocardiography in young healthy humans. Eur J Echocardiogr 2011;12(12):924-930.
13. Silva AC, Muzzi RAL, Oberlender G, et al. Feline hypertrophic cardiomyopathy: an echocardiographic approach. Arch Med Vet 2013; 45:1-6.
14. Kobayashi S, Sakai Y, Taguchi I, et al. Causes of an increased pressure gradient through the left ventricular outflow tract: a West Coast experience. J Echocardiogr 2018;16(1):34-41.
15. Blass KA, Schober KE, Li X, et al. Acute effects of ivabradine on dynamic obstruction of the left ventricular outflow tract in cats with preclinical hypertrophic cardiomyopathy. J Vet Intern Med 2014; 28(3):838-846.
16. Hori Y, Fujimoto E, Nishikawa Y, et al. Left ventricular outflow tract pressure gradient changes after carvedilol–disopyramide cotherapy in a cat with hypertrophic obstructive cardiomyopathy. J Vet Cardiol 2020; 29: 40-46.
17. Jackson BL, Adin DB, Lehmkuhl LB. Effect of atenolol on heart rate, arrhythmias, blood pressure, and dynamic left ventricular outflow tract obstruction in cats with subclinical hypertrophic cardiomyopathy. J Vet Cardiol 2015; 17 Suppl 1:S296-305.