Journal of Veterinary Cardiology (2025) **59**, 81—92

Journal of Veterinary Cardiology
ESVC

www.elsevier.com/locate/jvc

Occurrence and distribution of left ventricular bands and normal anatomical features in 78 feline hearts☆

N. Kiessling [a,*], **S. Rørvig** [b], **J. Häggström** [c]

[a] *VetGruppen Birkerød Veterinarian Hospital, Birkerød Hovedgaden 25, Denmark*
[b] *Pathologic Center, Rigshospitalet, Blegdamsvej, 2100 Copenhagen, Denmark*
[c] *Department of Clinical Sciences, Swedish University of Agricultural Sciences, Box 7045, 7507 Uppsala, Sweden*

Received 19 January 2024; received in revised form 3 March 2025; accepted 7 March 2025

KEYWORDS
False tendons;
Fibrous bands;
Cats;
Cardiology

Abstract *Introduction/Objectives:* Left ventricular bands (LVBs) are common in feline hearts. Their importance and general features are incompletely described. This study aimed to characterize LVBs in feline hearts based on anatomical location, quantity, histological features, and attachment sites.
Animals, Materials and Methods: Hearts from 78 domestic cats with or without heart disease were included in this study. Cardiac weight and dimensions were measured, and LVBs were categorized as singular bands or nets, with further characterization by location, length, appearance, and histological examination of attachment sites.
Results: Median cardiac weight was 4.34 g/kg (interquartile range: 2.1 g/kg). Left ventricular bands were present in all hearts, with 11% having only singular bands, 32% containing only nets, and 42% having nets covering the entire left ventricle (LV). The most common LVB attachment sites were the LV mid-region involving the posterior papillary muscle. Nets were most common in the mid-region including the papillary muscles (93%), followed by basilar (60%) and apical (59%) regions. All LVBs contained collagen, myocytes, adipose tissue, endothelial cells, and fibroblasts. No excess fibrosis, myocardial hypertrophy, or endocardial thickening at the attachment sites was identified.

☆ A unique aspect of the Journal of Veterinary Cardiology is the emphasis on additional web-based materials permitting the detailing of procedures and diagnostics. These materials can be viewed (by those readers with subscription access) by going to http://www.sciencedirect.com/science/journal/17602734. The issue to be viewed is clicked and the available PDF and image downloading is available via the Summary Plus link. The supplementary material for a given article appears at the end of the page. To view the material, go to https://doi.org/10.1016/j.jvc.2025.03.002.

* Corresponding author.
E-mail address: nathalie.birkeroed@vetgruppen.dk (N. Kiessling).

https://doi.org/10.1016/j.jvc.2025.03.002

Study Limitations: The study included mainly domestic stray cats aged 12 weeks to 15 years, with few purebred or diseased individuals. The hearts were examined by one person, which may introduce subjectivity.
Conclusions: Left ventricular bands are commonly found in the mid LV section of feline hearts, primarily involving the posterior papillary muscle, suggesting normal variation. Left ventricular bands contain myocytes, not Purkinje fibers, and are not fibrous tendons. Myocyte hypertrophy or excess fibrosis is absent at attachment sites.

Abbreviations

BW	body weight
HW	heart weight
IQR	interquartile range
IVS	interventricular septum
LV	left ventricle
LVB	left ventricular band
LVFW	left ventricular free wall
PPM	posterior papillary muscle

Introduction

Left ventricular bands (LVBs) are structures that transverse the left ventricle (LV) but are not associated with the mitral valve [1]. Some are string-like structures and referred to as singular bands in this study, whereas others are fused to form net-like structures [1]. Left ventricular bands are reported to be common in cats, but they are also found in other species [2] and were described as early as 1893 in humans [3].

Various terms are used to describe LVBs, including "moderator bands' and "false tendons' in veterinary medicine [1,4,5]. Consequently, using terminology such as moderator bands and false tendons may be misleading. However, this article encourages the usage of the descriptive term LVBs as opposed to false tendons or moderator bands. The term 'false tendons' is derived from a chorda tendinea-like appearance [6]. 'Moderator bands' typically describe intraventricular bands in the right ventricle, considered part of the conduction system [7]. Additionally, cats with endomyocardial fibrosis may exhibit similar bands macroscopically, resembling LVBs, but they are distinguished by pathological signs such as fibrosis [4,5]. It is currently unclear if, and how, LVBs and bands in cats with endomyocardial fibrosis are associated. In contrast to LVBs, bands in cats with endomyocardial fibrosis may hinder ventricular filling, potentially causing restrictive ventricular filling, previously termed "moderator band cardiomyopathy" [7] and more recently "endomyocardial restrictive cardiomyopathy" [8].

Left ventricular bands insert in the ventricular wall at two or more sites, where the ventricular myocardium may appear locally thickened [4,9]. These sites are sometimes referred to as "ectopic papillary muscles", and it is uncertain if they represent mild pathological hypertrophy or normal variation [3]. There are a few studies in cats describing LVBs by use of echocardiography [9]. However, two-dimensional echocardiography is limited in the sense that it usually only provides an image in one thin plane, and current three-dimensional modalities do not offer sufficient image resolution or frame rate [10—12]. Accordingly, two-dimensional echocardiography makes it difficult to identify and fully characterize LVBs [6]. Therefore, the characterization of endocardial bands, nets, and associated structures of LVBs is most accurately evaluated by a postmortem examination [13,14].

Studies concerning histological features of LVBs are scarce. Liu et al. [4] described them as being composed of Purkinje fibers, collagen, fibrous tissue, endothelium, and increased and immature fibroblasts at the attachment site. Kimura et al. [1] also described that they harbored muscle fibers, a finding well described in humans with LVBs [6]. The presence or absence of Purkinje or muscle fibers is important for understanding their function in the normal feline heart, whether they are part of the conduction system [15], contribute to myocardial contraction [16], or are functionally passive structures.

The aim of the present article was to characterize LVBs in the feline heart by describing their

anatomical location, quantifying their number, and characterizing their histological features in a subset of cats.

Animals, materials, and methods

Thirteen veterinarians at different clinics in Denmark contributed to this study by collecting hearts on a voluntary basis, and they received no compensation, except consumables necessary for harvesting the hearts of cats euthanized for reasons unrelated to this study. Cats were included, provided that the owner had signed an informed owner consent form, where the owner accepted that their cat's heart was donated for the present study. Veterinarians were asked to document the date of death, age, weight, health status, color, gender, home status, and the reason for and the method used for euthanasia. No restriction was made regarding age, gender, or health status. All veterinarians were asked to euthanize the cats with protocols not involving cardiac injection. In 39 cats, the age was unknown; the age was estimated from the appearance of the cat.

The hearts were harvested by the attending veterinarian or the veterinarian responsible for investigating the hearts (N. Kiessling). All hearts were flushed with water or 10% phosphate-buffered formalin to remove the blood in the heart, before being placed in 10% phosphate-buffered formalin within 24 h. The veterinarian examining the hearts morphologically (N. Kiessling) was instructed on how to evaluate the anatomical features of the hearts by a human pathology specialist in heart and lung disease (S. Rørvig). The weight, width, length, and height of the hearts were measured according to predefined criteria, as shown in Figure 1.

The one-dimensional measurements of cardiac width, length, and height were body size normalized by dividing the numerical value by the cubic root of body weight (BW) according to the principle of allometric scaling [17,18]. This approach leads to a unitless number of cardiac dimensions as previously described for certain one-dimensional echocardiographic measurements in cats [18].

The LV was dissected, as illustrated in Figure 2A, by making an incision from the left atrium wall along the free ventricular wall to the apex. A second incision extended from the apex along the anterior interventricular septum (IVS) and through the left part of the anterior aortic wall. Finally, a vertical section was made from the apex to the base of the free wall, behind the posterior papillary muscle (PPM), to allow for comprehensive examination of the LV endocardium. The endocardium was then examined for LVBs, and findings were recorded into a predefined protocol (Tables 1—3). The apical region of the LV was defined as the area apical to the anterior and posterior papillary muscles, the mid-region as the region involving the anterior and posterior papillary muscles, and the basilar region as the section between the anterior and posterior papillary muscles and the mitral and aortic valve orifices (Fig. 2).

All hearts were photographed for documentation; if findings were equivocal, a decision was made after a discussion with the pathologist (S. Rørvig).

The LVBs were subdivided into singular bands and nets. A singular band was defined as a macroscopically white string-like structure with maximum three sites of attachment to the

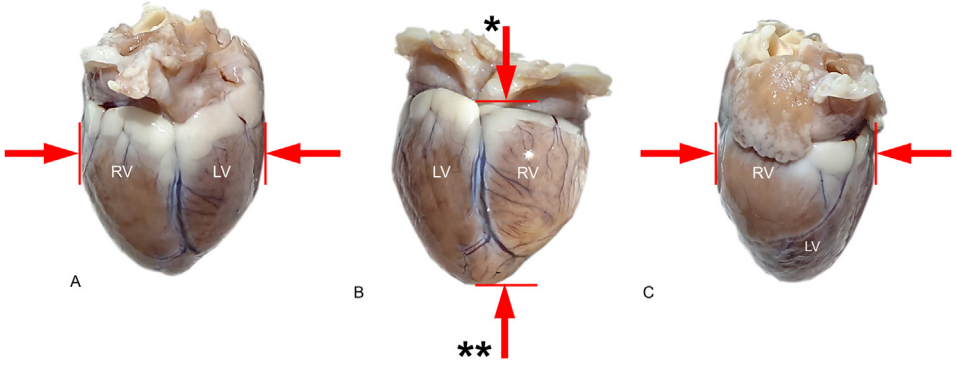


Figure 1 Measurements of the width, height, and length of the hearts:
A: Width, transverse distance measured on the inferior surface of the heart from the coronary sulcus*, and to the apex**. B: Length, longitudinal distance measured on the posterior aspect of the heart. C: Length, distance from the front (anterior) surface to the back (posterior) surface of the heart.
LV: left ventricle; RV: right ventricle.

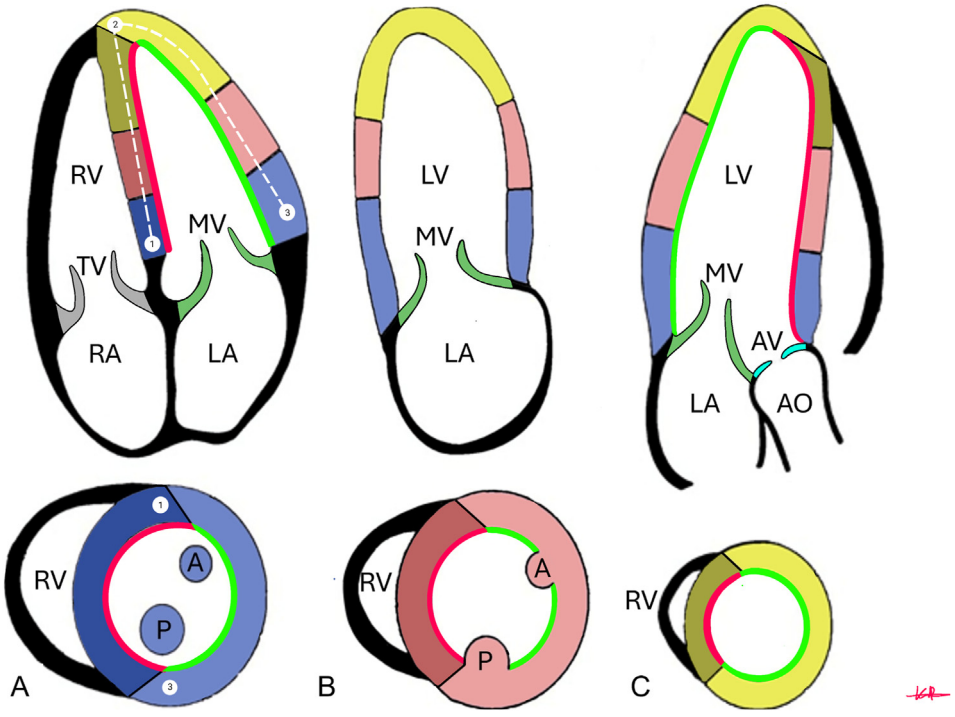
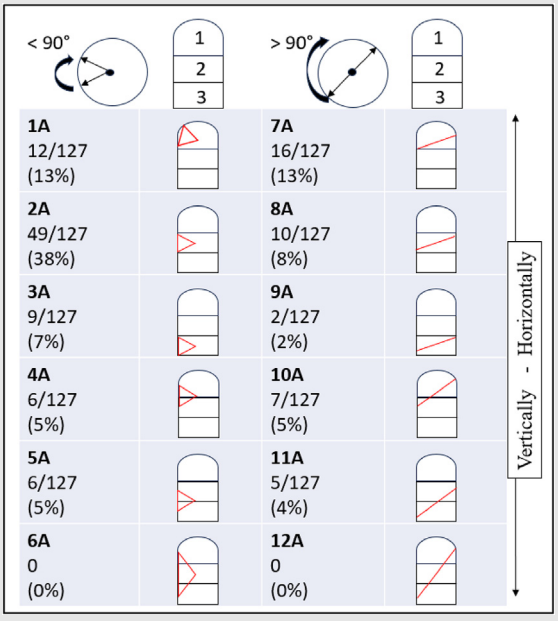


Figure 2 Images illustrating incisions made to open the left ventricle (LV) for left ventricular bands (LVBs) evaluation, showing the delineation of nets and singular bands' attachment sites. These were categorized into three subgroups: base (A), middle (B), and apex (C).
The base included the area above the papillary muscles, extending to the aortic orifice and mitral valve. The middle section encompassed the mid-level region of the papillary muscles. The apex extended from the base of the papillary muscles to the heart's tip. White circles in (A) represent the incision starting point (1), middle point (2), and end point (3) of the LV opening.
The inner red line follows the interventricular septum (IVS), and the inner green line follows the left ventricular free wall (LVFW).
AO: aorta; APM (A): anterior papillary muscle; LA: left atrium; LV: left ventricle; PPM (P): posterior papillary muscle; RA: right atrium; RV: right ventricle.

endocardium at either end (Fig. 3). Nets were macroscopically defined as a loose white or light net-like structure, with more than three sites of attachment to the endocardium at either end (Fig. 3). The following were documented in relation to the LVBs, singular bands, and nets: numbers, thickness, length, sites of attachments, and the presence of fibrous tissue at the attachment site. Fibrosis was defined by a white plaque-like thickening of the endocardium. The band thicknesses were subjectively classified by comparing findings in all the hearts. The length of the bands was measured under controlled tension to avoid rupture. All bands in the hearts were classified according to three different schemes, A to C (Tables 1–3), and areas are also illustrated in Figure 2. In scheme A, the bands were allocated into subgroups depending on their attachment site, as displayed in Table 1. In scheme B, as presented in Table 2, the bands were divided into groups by attachment site using the system by Kimura et al. [1], slightly modified by the addition of two more sites IVS and left ventricular free wall (LVFW) (IVS to IVS and LVFW to LVFW). In scheme C, the bands were allocated into subgroups according to the length of the bands, as shown in Table 3. Eight hearts were selected for histological evaluation by the study's primary investigator (N. Kiessling) and the pathologist (S. Rørvig). The hearts were selected to represent all ages, LVB locations, thin and thicker singular bands, nets covering partly or entirely the LV, and heart weight (HW) span, and details are presented in Supplementary Table A. The intraventricular septum and LVFW were measured just beneath the mitral valve in all hearts chosen for microscopic evaluation. Samples were

Table 1 Distribution of 127 singular bands in 78 hearts, grouped according to their attachment site (scheme A).



In groups 1A—6A, the attachment sites are less than 90° apart from the site of origin and are more parallel to the heart. In groups 7A—12A, the attachment sites are greater than 90° apart from the site of origin, and the singular bands cross the intraventricular lumen.

embedded in paraffin, sliced along the longitudinal axis of the LVBs in 5-μm sections on a microtome and stained with hematoxylin-eosin, Masson's trichrome, and periodic acid-Schiff for subsequent histological examination by light microscopy (S. Rørvig). Myocyte size was measured with the cellSens[d] imaging software, in the myocardium, nets, singular bands, and insertions sites. The width of the myocytes in the LVBs and myocardium was measured across the area of the nucleus and where cell borders were sufficiently visible.

Statistical analysis

Statistical analyses were performed using commercially available software[e]. Results are reported as median values with interquartile ranges (IQRs). The width of the myocytes in myocardium and in LVBs was compared by the Mann-Whitney U test. Potential associations between the presence and numbers of LVBs (nets or singular bands), age, and HW were investigated using linear or nominal logistic regression. Statistical significance was set at $P<0.05$.

Results

Of the included 78 cats, 23 were stray cats euthanized due to humanitarian reasons, six were euthanized because of car accidents where the owner could not be found due to lack of identification, four were euthanized due to deceased owner, and 45 cats were euthanized for other reasons (presented in Supplementary Table A). There were 47 male and 28 female cats, 64 domestic cats, and 14 cats of different pure breeds. The sex was unknown in three cats.

The median age was 4.5 years (IQR: 11 years) ranging from 12 weeks to 15 years. The median HW was 18 g (IQR: 11 g), which corresponds to a median HW per BW of 4.34 g/kg (IQR: 2.1 g/kg), 4.3 g/kg (IQR: 1.4 g/kg) for females and 4.6 g/kg (IQR: 2.5 g/kg) for males. The median heart height, width, and length (sulcus to apex) were 24.9 mm, 29.0 mm, and 30.0 mm (IQR: 5.4, 6.526.0—32.5, and 5.9, respectively). Body weight—normalized medians of height, width, and length were 15.6, 18.4, and 18.9 (IQR: 2.6, 2.7, and 2.6, respectively).

In this study, no cats were diagnosed with hypertrophic cardiomyopathy. Among the eight cats examined by histopathology, four had an HW per BW above the study's median (Supplementary Table A). Three of these eight cats were under one year of age. Younger cats (aged three months to one year) presented slightly higher HWs (6.8 g/kg) than older cats (aged >one year, 5.5 g/kg), which was concluded to explain this finding in these cats. The fourth cat had a lower HW per BW measure than the median for cats with hypertrophic cardiomyopathy [17,19] and showed no histopathological signs of hypertrophic cardiomyopathy [20,21]. Images showing the macroscopic and histopathological appearance of all eight cats are presented in Supplementary Figures I—VIII.

Left ventricular bands were found in all hearts. Eleven percent of the hearts had singular bands exclusively, 32% had only nets, and 57% had both singular bands and nets. In 33 of the 78 hearts, extensive nets covered all three LV regions (base, middle, and apex). The most common location of the nets was the middle section, as shown in Table 2.

In total, 127 singular bands were found: 18 cats had one, 13 had two, and 10 had three. The locations for attachment are presented in Table 1, showing that the most common attachment site for

[d] cellSens, Olympus Evident TruAI™.
[e] JMP Pro v16.0, Cary, NC.

Table 2 Distribution of singular bands and nets in 78 hearts where singular bands were grouped according to attachment site (scheme B) using the system by Kimura et al. [1], with slight modifications by the addition of two more sites (IVS to IVS and LVFW to LVFW) and nets by their anatomical location.

Group	Attachment sites	This study	Kimura et al.
B1	PPM-IVS	35/127 (28%)	25/25 (100%)
B2	APM-IVS	9/127 (7%)	22/25 (88%)
B3	PPM-LVFW	27/127 (22%)	(17/25) 68%
B4	APM-LVFW	8/127 (6%)	(16/25) 64%
B5	PPM-APM	1/127 (1%)	9/25 (36%)
B6	IVS-LVFW	5/127 (2%)	NA
B7	IVS-IVS	16/127 (13%)	NA
B8	LVFW-LVFW	26/127 (21%)	NA
Net	Base[a]	50/83 (60%)	NA
Net	Middle[a,b,c]	78/83 (93%)	NA
Net	Apex[c]	49/83 (59%)	NA
Net	Macroscopically thick	17/83 (20%)	NA
Net	Macroscopically thin	64/83 (77%)	NA

Note that this study defined singular bands as structures with a maximum of three attachment sites at either end. Structures with more than three attachment sites were referred to as nets, whereas Kimura et al. [1] did not differentiate between nets and LVBs. Additionally, the distribution of nets is included for comparison.
APM, anterior papillary muscle; IVS, interventricular septum; LVB, left ventricular band; LVFW, left ventricular free wall; NA, not applicable; PPM, posterior papillary muscle.
[a] Net attachment sites include the area below PPM/APM.
[b] Net attachment sites involve the area including APM and PPM.
[c] Net attachment sites are apical to the PPM/APM.

Table 3 Singular bands grouped by their length in 78 hearts (scheme C).

0—4 mm	4—8 mm	8—12 mm	12—16 mm	16—20 mm
58/126 (4%)	36/126 (28%)	24/126 (19%)	6/126 (5%)	2/126 (2%)

The length of the singular bands was measured while applying maximum tension without causing rupture. One singular band ruptured before measurement, resulting in 126 bands instead of 127.

singular bands (38%) was in the mid-region, without crossing the LV including the PPM. One of the 127 singular bands ruptured during measurement, leaving 126 for length measurement. The singular band length is presented in Table 3, showing that most of the singular bands were 0—4 mm in length (46%), followed by 4—8 mm (28%). No excessive fibrosis was found in any heart at the attachment sites of singular bands.

No association was found between age, cause of death, breed, reproductive status, gender, or fur color and the presence of LVBs or nets. Furthermore, no association could be found between extent of LVBs and the HW.

Histological examination of singular bands, as well as thin and thick nets, showed that they were covered by endothelium and composed of delicate peripheral connective tissue containing collagen fibers and scattered resting fibroblasts. The central part of the LVBs had longitudinal bands of one to four myocytes in thickness, separated by a varying amount of loosely arranged connective tissue and focally interspersed clusters of endomyocardial adipose tissue (Fig. 4). The chordae tendineae were composed of a core of dense collagen with a thin peripheral layer of spongiosa arising from the mitral valve to which it was attached, and there was no presence of myocytes or adipose tissue (Fig. 4). The sectioning along the longitudinal axis of the LVBs suggested that LVBs-myocyte bands extended unbroken from one insertion in the endocardium to another. The morphology of the myocytes in the LVBs seemed to be more loosely arranged but was not interpreted to be different from that of the myocardial cardiomyocytes or that at the sites of attachment (Figs. 5 and 6). The myocytes within the LVBs appeared slightly smaller than those found in the myocardium (Fig. 6). They exhibited striations representing sarcomeres, and no signs of Purkinje fibers or fibrin were observed in either the LVBs or the myocardium at

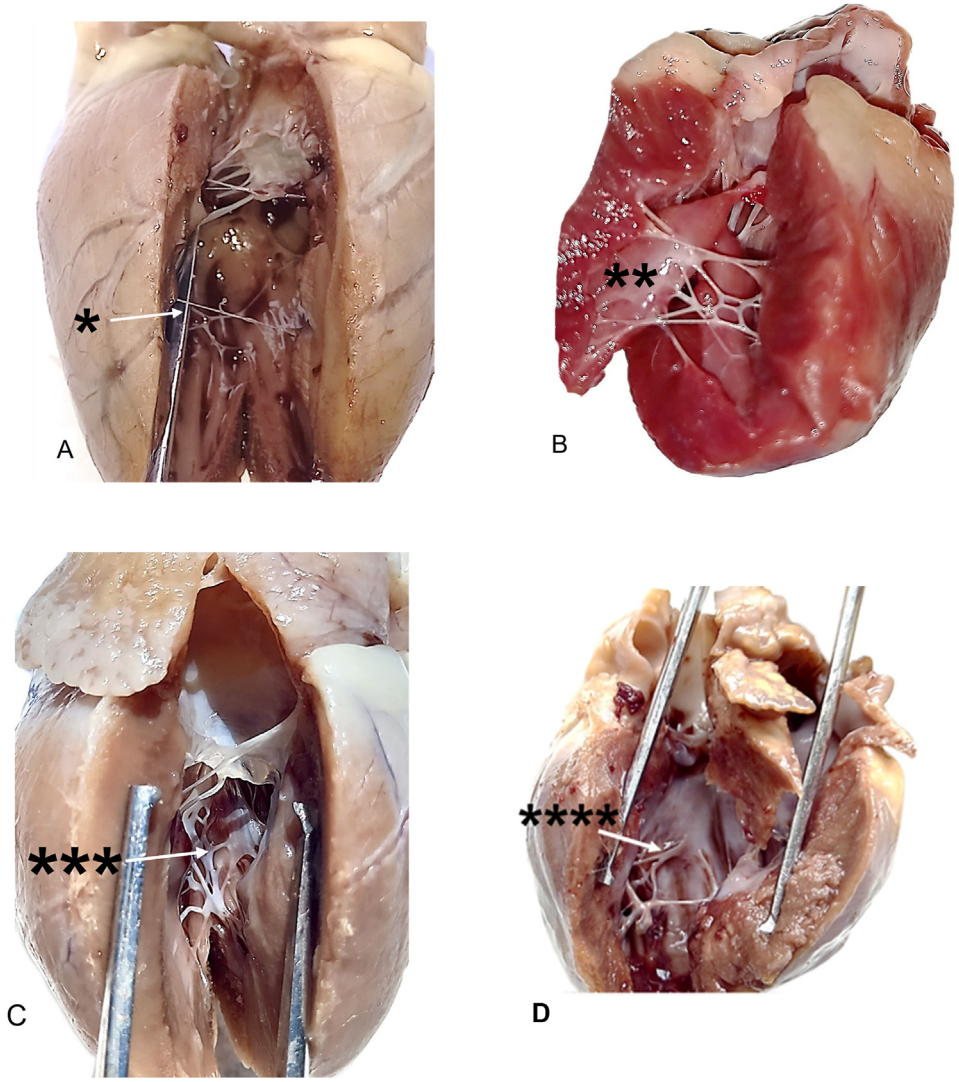


Figure 3 Macroscopic evaluation of four hearts with the left ventricle opened for left ventricular bands (LVBs) visualization and classification into subgroups. Heart A is from a 15-year-old female Domestic shorthair (DSH) cat, weighing 32 g (6.7 g/kg body weight [BW]); heart B is from a 5.5-year-old female DSH cat, weighing 15 g (4.8 g/kg BW); heart C is from a 4-year-old female cat, weighing 18 g (5.8 g/kg BW); and heart D is from a 1-year-old intact male DSH cat, weighing 27 g (4.3 g/kg BW). Images demonstrate how nets may extend across the entire ventricle, closely resembling myocardial adhesion while remaining distinct structures. * thin band; ** thin net; *** thick net; **** broad thick net.

attachment sites (Figs. 5 and 6). Additionally, intracellular glycogen was not observed to be increased with special stains, nor were there increased quantities of mitochondria when compared with cardiomyocytes of the LVBs or myocardium (Fig. 6A, B). Intercalated discs were observed between myocytes within the LVBs, as illustrated in Figure 6D.

A total number of 56 cardiomyocytes from two hearts and 63 LVB myocytes were measured, and the median width of cardiomyocytes was significantly greater than that of the LVB myocytes (10.5 vs. 9.6 μm, IQR: 2 μm and 1.3 μm, respectively, $P<0.0001$).

Discussion

This study is hitherto, to the author's knowledge, the most extensive and detailed macroscopical and microscopical study of LVBs conducted in cats. This study showed that LVBs (singular bands and nets) are present in all cats and that they likely represent normal features of the feline heart. They are composed of myocytes, collagen, adipose tissue, and endothelial cells. No pathological features or alterations of the myocardium at the attachment sites could be associated with LVBs (bands or nets), even in the presence of the most extensive LVBs that occupied large parts of the left

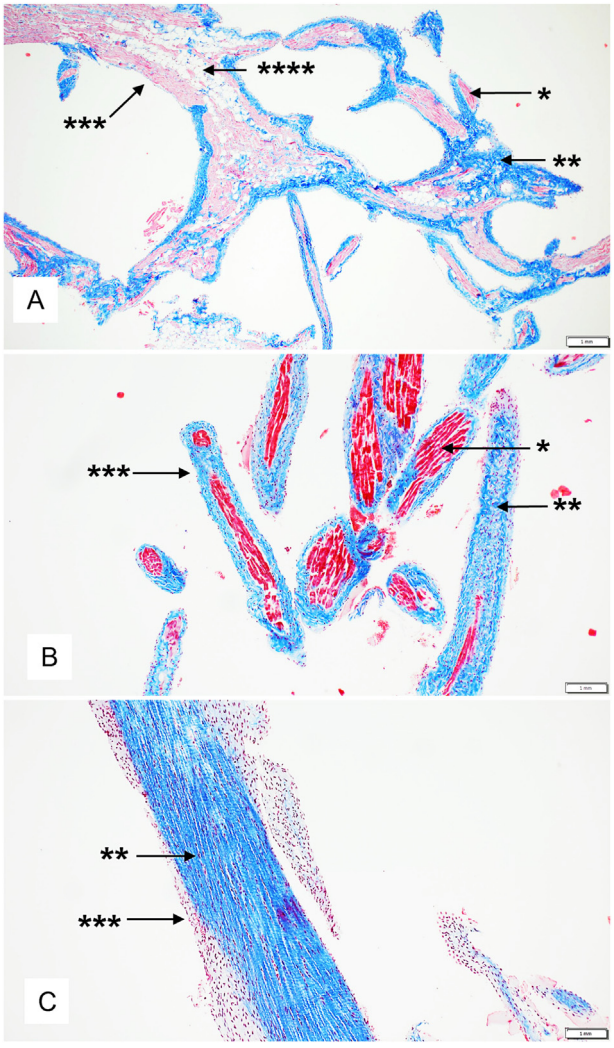


Figure 4 Representative histological sections of left ventricular bands (LVBs) and chordae tendineae (CT) stained with Masson's trichrome from two cats. Section (A) shows a thick net from a three-year-old female cat, illustrating the network extending from multiple insertion sites into the ventricular lumen. The muscle layers extend continuously from one attachment site to another. The net is composed of muscle fibers (*) and collagen (**), surrounded by a thin peripheral layer of endothelium (***) and occasional endomyocardial fat (****), maintaining a uniform cellular composition. Section (B) displays another thick net from a three-month-old kitten, with a similar cellular composition to that in (A). Section (C) presents a CT section from the same female cat as in (A), included for comparison. The CT lacks myocytes and endomyocardial fat but contains a higher collagen content (**), along with a thin peripheral spongiosa layer and endothelium (***) originating from the mitral valve. * muscle fibers; ** collagen; *** endothelium; **** endomyocardial fat.

ventricular lumen. Left ventricular bands were found in cats of all ages without any apparent histological difference between old and young cats. The finding of myocytes in the LVBs is suggestive that they are not passive structures and that they might have a role to play in the myocardial function of the feline heart, but this needs to be shown in further studies.

Previous studies have identified Purkinje cells in LVBs, using the same staining as this study [1,4,7]; thus, we considered the possibility that the LVB myocytes were Purkinje cells. Purkinje cells are characterized by having a larger cell and nuclear size than conventional cardiac myocytes [4,7,22]. Purkinje cells have a limited number of sarcomeres and contain a light, foamy cytoplasm due to a substantial amount of glycogen, making them strongly stainable by periodic acid-Schiff [23,24].

This study found no convincing presence of Purkinje fibers in the LVBs, using various staining methods. The presence of intercalated discs within LVB myocytes supports the conclusion that these structures are composed of working myocardial cells rather than conduction system cells. Purkinje fibers typically lack prominent intercalated discs and contain abundant glycogen, distinguishing them from ordinary cardiac myocytes [15,23]. The identification of intercalated discs within LVBs suggests that LVBs do not contain Purkinje cells [28,29]. The absence of Purkinje fibers suggests that the LVBs may not serve as a major component of the LV conduction system [15]. However, given the conflicting findings regarding Purkinje fibers in the LVBs, additional ancillary tests could be considered to further support their identification. These include immunohistochemistry [25—27] (e.g. anti-HSP27, and anti-PGP 9.5), Crossman's staining, and transmission electron microscopy [28,29]. On the other hand, the presence of myocytes indicates the potential for LVBs to play a role in LV myocardial function. If the muscle cells in the LVBs affect the heart's function, one can only speculate that they could potentially contribute to the myocardial contraction and perhaps help to stabilize the LV, considering the small size of the feline heart.

This study was not primarily aimed at comparing healthy to diseased hearts but aimed at providing an overview of LVBs in the general feline population and to make comparisons between groups based on the numbers and characteristics of the LVBs. However, a study by Liu et al. [4] compared LVBs in cats with and without cardiac disease, without excluding any heart diseases, and it was suggested that excessive LVBs could potentially restrict cardiac growth and LV filling. This study did not find any association between HW and presence and abundance of LVBs, which is in agreement with the findings of Kimura et al. [1], where no difference in the presence of abundance of LVBs was found between diseased and healthy hearts.

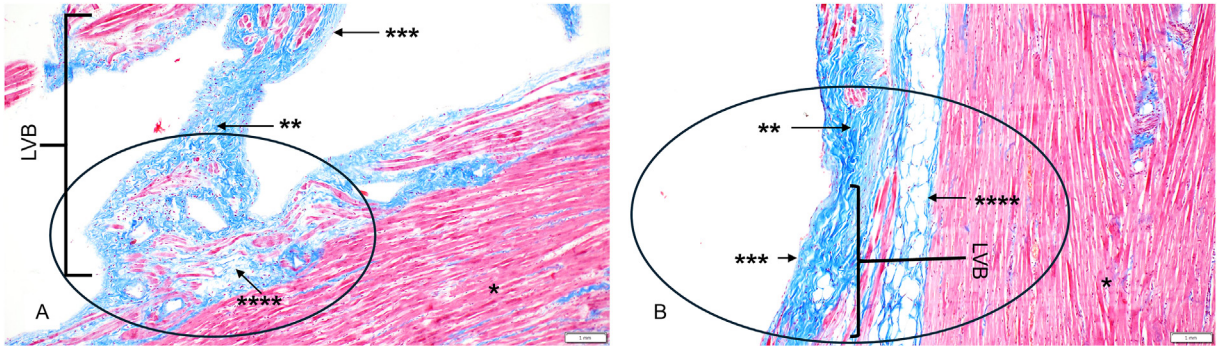


Figure 5 Representative histologic sections stained with Masson's trichrome, showing the left ventricular bands (LVBs) insertion site to the myocardium in two cats: (A) from a 12-year-old female cat and (B) from a one-year-old male cat. The insertion site is highlighted by a circle, and the LVBs are indicated by a bracket. * myocardium; ** connective tissue; *** endothelium; **** endomyocardial fat.

Despite these findings, it has been speculated in several articles that LVBs might play a role in some diseased feline hearts [5,18,19,21,32]. In these articles, the hearts showed signs of cardiomyopathy such as cardiomegaly and fibrosis [5,30,31,32].

There are few studies that describe the normal macroscopic cardiac features in the feline population, and the available ones included small study groups or diseased cats [22,33,34]. The median HW was in agreement with other studies [35]. In the present study, the width, height, and length of the heart were measured, which, to our knowledge, have not been reported in the past. These measurements, besides weight, can be useful when assessing cardiac morphology at postmortem examination. The width, height, and length are one-dimensional measurements that optimally should be normalized to another one-dimensional measurement. However, several one-dimensional measurements, including echocardiographic dimensions, have been shown to correlate linearly with the BW raised to the power of approximately 1/3 [17]. Because no exact estimates were available for the scaling exponent for the cardiac dimensions of the present study, the measurements were normalized for body size using the cubic root of the BW, an approach that has previously been used for normalizing echocardiographic measurements in the absence of an exact scaling exponent [17].

The most common sites of LVB attachment were the PPM and septal free wall, followed by the IVS to IVS and LVFW to LVFW. These findings differ from those in the study by Kimura et al. [1], although that study did not include IVS to IVS or LVFW to LVFW as possible attachment sites. This study, as well as other studies [1,4], found that the most common site of attachment was the PPM, as well as the septal free wall. Interventricular septum to IVS and LVFW to LVFW were the third and fourth most common insertion sites, respectively, a finding that differs from that reported previously by Kimura et al. [1], although that study did not include IVS to IVS and LVFW to LVFW as possible attachment sites.

The origin of LVBs in cats and other species is unknown. Because LVBs were found in kittens, it can be assumed they are born with them. The difference in the prevalence of LVBs in cats and other mammalian species does indicate a difference in cardiac development [27].

Our study found LVBs in all kittens examined, suggesting they are congenital and form during fetal development. However, the youngest kitten was 12 weeks old, not a newborn, which, to some extent, leaves the question of when these structures first appear unanswered.

Variations in the prevalence of LVBs across species may reflect differences in cardiac development [2,36]. During fetal development, the left ventricular myocardium is highly trabeculated to support nutrient and gas exchange before the coronary vessels form [36—38]. As development progresses, these trabeculations typically undergo compaction, resulting in a smooth ventricular surface. While this process is conserved across species, the extent and timing can vary [36—38].

In some cases, incomplete compaction leads to left ventricular non-compaction, a condition where the myocardium remains spongy and trabeculated, extending into the ventricular lumen, which can impair cardiac function. This condition has been documented in humans [38—40], dogs [40], and cats [41,42]. It remains to be determined whether LVBs represent a form of non-compaction, remnants of normal development, or a species-specific trait. Further studies are needed to clarify their origin and significance [38—40].

Left ventricular bands have been studied by echocardiography. This study reported that most

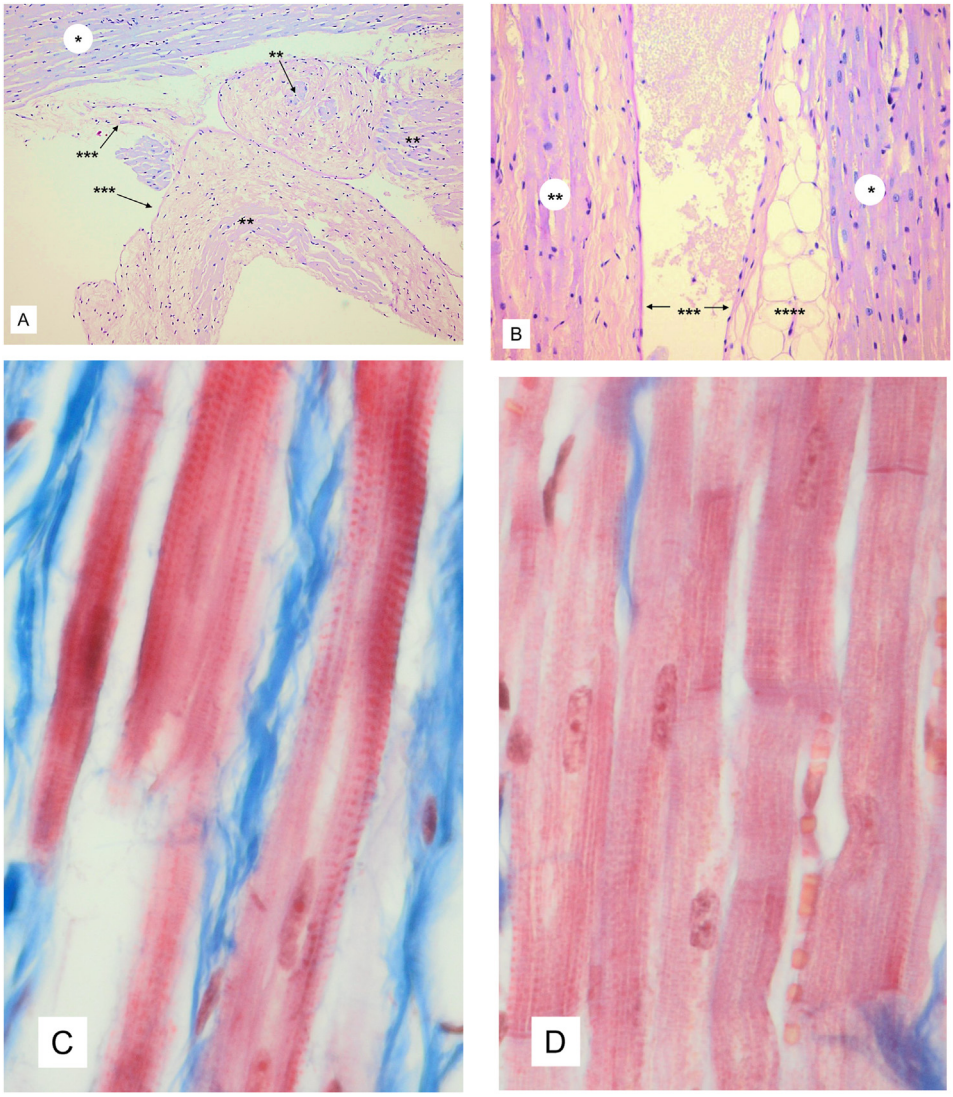


Figure 6 Representative histological sections comparing the myocytes in the left ventricular myocardium (*) and left ventricular bands (LVBs) (**), stained with periodic acid-Schiff (PAS). Images (A) and (B) demonstrate a similar appearance of the muscle fibers in the myocardium (*) and the in the LVBs (**). Sections from the left ventricular myocardium (C) and LVBs (D), stained with Masson's trichrome in higher magnification (x60) than in (A) and (B), provide a clearer comparison between LVBs and myocardial myocytes. The LVB myocytes were slightly smaller than those in the myocardium (median diameter: 9.6 μm vs. 10.5 μm). Striations, indicative of sarcomeres, are visible in both section (C) and (D), along with intercalated discs, which are more prominently visualized in section (D). Notably, no signs of Purkinje fibers or extensive fibrous tissue were observed in either the LVBs or the myocardium. * muscle fibers; ** collagen; *** endothelium.

LVBs were primarily parallel to the wall and were short in length. This location presumably leads to problems identifying some of them, compared to the ones that cross from one free wall to another. Therefore, when performing echocardiography in cats, it is important to know that LVBs are present in all cats and that echocardiography may not be the optimal method to fully characterize them [13,14].

In a study by Wolf et al. [9], LVBs were studied by echocardiography in 128 cats, and it was reported that IVS and LVFW wall thickness could be increased during the diastolic phase at the attachment sites, but this type of local thickening was not apparent in this postmortem study of feline hearts. In addition to the obvious difference between *in vivo* echocardiographic and postmortem studies, one important difference is also that echocardiography allows studies of cardiac architecture at normal cardiac pressures including during diastole. *Postmortem* studies do not accurately reflect *in vivo* hemodynamics as intracardiac pressures equilibrate to atmospheric levels after death and the heart ceases active contraction [43].

Limitations

Because all hearts were examined subjectively, by one person, it can be argued that results could have been different if one or more examiners also had examined the hearts. Although several young cats were included in the present study, it did not include any newborn kittens. It cannot therefore be concluded if cats are born with LVBs or if they develop after birth.

Given the challenge of consistently applying uniform tension to all bands and measuring them accurately, precise measurements for each band were difficult to obtain. Instead, length estimates were offered for each group. However, by providing a wide range in length for each measurement group, efforts were made to minimize variability in measurement techniques.

The study population was limited in that it included many comparably young stray cats. Purebreed cats, older cats, and cats with cardiac diseases were comparably few, and it cannot be concluded that LVBs are similar in these cats with respect to numbers and features. To confirm whether Purkinje fibers are present in feline LVBs, additional methods should be used.

Conclusions

Most cats have LVBs, of all ages, and their number was not associated with age, sex, BW or HW, or the cause of death. They are commonly found in the mid LV section of the feline heart, most commonly involving the PPM, and likely represent normal variation of the feline heart. Pathological hypertrophy or other alterations of the myocardium do not appear to be present at their site of attachment. The myocytes in the LVBs are slightly smaller than the myocardium, and Purkinje cells could not be identified. It cannot be ruled out that LVBs play a role in LV myocardial function.

Conflict of Interest Statement

The authors do not have any conflicts of interest to disclose.

Acknowledgment

The authors would like to acknowledge all veterinarians and veterinary nurses who helped in gathering hearts for the project, as well to the owners who donated their cat's heart for research purpose.

Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.jvc.2025.03.002.

References

[1] Kimura Y, Shimizu M, Kato T, Watanabe T. The morphology and histology of feline left ventricular bands: a post-mortem study. J Vet Cardiol 2015;17(2):111—9.
[2] Gerlis LM, Ho SY, Anderson RH. The incidence and morphology of ventricular myocardial bands in hearts with congenital heart disease. Br Heart J 1984;52(3):264—71.
[3] Turner A. The moderator band and ventricular trabeculations in human and animal hearts. J Anat 1893;28(3):275—89.
[4] Liu SK, Fox PR, Goldfine D. Structural features of feline endocardial bands and their role in cardiac function. Am J Vet Res 1982;43(4):654—60.
[5] Cavalcanti JS, Beltrão EI, Costa MC, de Lima E. Endomyocardial fibrosis and its relation to left ventricular bands in cats. J Vet Cardiol 2018;20(3):171—8.
[6] Silbiger JJ. Left ventricular false tendons: a review of their anatomy, physiology, and clinical implications. Cardiol Rev 2013;21(1):13—7.
[7] Liu SK. The feline heart: morphology, function, and disease. J Vet Pathol 1977;14(1):98—120.
[8] Fox PR. Endomyocardial restrictive cardiomyopathy: the evolving concept of feline restrictive cardiomyopathy. J Feline Med Surg 2004;6(3):183—90.
[9] Wolf R, Schober KE, Liese A. Echocardiographic assessment of left ventricular bands in 128 cats. J Vet Intern Med 2017;31(5):1425—33.
[10] Chetboul V, Serres F, Tissier R, Gouni V. Three-dimensional echocardiography in small animals: principles and clinical applications. Vet Radiol Ultrasound 2016;57(4):403—15.
[11] Giangrossi C, Ramalli A, Dallai A, Mazierli D, Meacci V, Boni E, Tortoli P. Requirements and hardware limitations of high-frame-rate 3-D ultrasound imaging systems. Appl Sci 2022;12(13):6562. https://doi.org/10.3390/app12136562.
[12] Aly D, Madan N, Kuzava L, Samrany A, Parthiban A. Comprehensive evaluation of left ventricular deformation using speckle tracking echocardiography in normal children: comparison of three-dimensional and two-dimensional approaches. Cardiovasc Ultrasound 2022;20(3). https://doi.org/10.1186/s12947-022-00291-5.
[13] Abdulla AK, Frustaci A, Martinez JE, Florio RA, Somerville J, Olsen EG. Echocardiography and pathology of left ventricular false tendons. Chest 1990;98:129—32.
[14] Maron BJ, Epstein SE, Roberts WC. Hypertrophic cardiomyopathy and intraventricular bands in felines. Am Heart J 1977;94(5):705—18.
[15] Moorman AFM, Webb S, Brown NA, Lamers WH, Anderson RH. Development of the cardiac conduction system: a matter of chamber differentiation. Cardiovasc Res 2002;58(1):1—10.

[16] Côté E, Manning AM, Gordon SG, Saunders AB. Feline cardiology: diagnosis and treatment of cardiac disease in cats. 2nd ed. Elsevier; 2021.
[17] Cornell CC, Kittleson MD, Torre PD, Häggström J, Lombard CW, Pedersen H, Vollmar A, Wey A. Allometric scaling of M-mode cardiac measurements in normal adult dogs. J Vet Intern Med 2004;18:311—21.
[18] Häggström J, Andersson ÅO, Falk T, Nilsfors L, Olsson U, Kresken JG, Höglund K, Rishniw M, Tidholm A, Ljungvall I. Effect of body weight on echocardiographic measurements in 19,866 purebred cats with or without heart disease. J Vet Intern Med 2016;30:1601—11.
[19] Kittleson MD, Fox PR, Basso C, Thiene G. Naturally occurring biventricular noncompaction in an adult domestic cat. J Vet Intern Med 2017;31:527—31.
[20] Fox PR, Keene BW, Lamb K, Schober KA, Chetboul V, Fuentes VL, Wess G, Payne JR, Hogan DF, Motsinger-Reif A, Häggström J, Trehiou-Sechi E, Fine-Ferreira DM, Nakamura RK, Lee PM, Singh MK, Ware WA, Abbott JA, Culshaw G, Riesen S, Borgarelli M, Lesser MB, Israël NV, Côté E, Rush JE, Bulmer B, Santilli RA, Vollmar AC, Bossbaly MJ, Quick N, Bussadori C, Bright JM, Estrada AH, Ohad DG, Fernández-Del Palacio MJ, Brayley JL, Schwartz DS, Bové CM, Gordon SG, Jung SW, Brambilla P, Moïse NS, Stauthammer CD, Stepien RL, Quintavalla C, Amberger C, Manczur F, Hung Y-W, Lobetti R, De Swarte M, Tamborini A, Mooney CT, Oyama MA, Komolov A, Fujii Y, Pariaut R, Uechi M, Ohara VYT. International collaborative study to assess cardiovascular risk and evaluate long-term health in cats with preclinical hypertrophic cardiomyopathy and apparently healthy cats: the REVEAL study. J Vet Intern Med 2018;32(3):930—43. https://doi.org/10.1111/jvim.15093.
[21] Liu SK, Maron BJ, Tilley LP. Feline hypertrophic cardiomyopathy: gross anatomic and quantitative histologic features. Am J Pathol 1981;102(3):388—95. PMID: 7193978.
[22] Liu SK, Peterson ME, Fox PR. Hypertrophic cardiomyopathy and hyperthyroidism in the cat. J Am Vet Med Assoc 1984;185:52—7.
[23] Boyett MR, Inada S, Yoo S, Li J, Liu J, Dobrzynski H. The specialized conduction system of the heart: role of Purkinje fibers in rhythm regulation. J Mol Cell Cardiol 2000;32(6):1019—30.
[24] Henry CG, Lowry OH. Enzymes and metabolites of glycogen metabolism in canine cardiac Purkinje fibers. Am J Physiol 1985;248(5 Pt 2):H599—605. https://doi.org/10.1152/ajpheart.1985.248.5.H599.
[25] Sawamoto O, Harada M, Kurisu K, Nakashima Y, Nakayama M. Histopathological changes in Purkinje fibers in the heart of experimentally thiamine-deficient beagle dogs. Exp Toxicol Pathol 2009;61:288—93.
[26] Jacobsen B, Kreutzer M, Meemken D, Baumgärtner W, Herden C. Proposing the term purkinjeoma: protein gene product 9.5 expression in 2 porcine cardiac rhabdomyomas indicates possible Purkinje fiber cell origin. Vet Pathol 2010;47:738—40.
[27] Ateş S, Karakurum E, Takcı L, Başak F, Kürtül I. Morphology of the atrioventricular valves and related intraventricular structures in the wild pig (Sus scrofa). Folia Morphol 2015;76:650—9.
[28] Viragh S, Challice CE. The structure of the conducting system in the mammalian heart. J Anat 1977;124(3):633—53.
[29] Anderson RH, Ho SY, Redmann K, Brown NA, Benson DW. The anatomical arrangement of the myocardial cells making up the ventricular mass. Ann Thorac Surg 2005;79(1):179—88.
[30] Wray JD, Gajanayake I, Smith SH. Congestive heart failure associated with a large transverse left ventricular moderator band in a cat. J Feline Med Surg 2007;9:56—60.
[31] Cony FG, Bianchi MV, Argenta FF, Oliveira CR, Stefanello C, Pavarini SP. Congestive heart failure in a young cat with excessive moderator bands (false tendons) in the left ventricle. Ciência Rural 2021;51(10). https://doi.org/10.1590/0103-8478cr20200727.
[32] Tilley LP, Lui SK, Gilbertson SR, Wagner BM. Primary myocardial disease in the cat. J Am Vet Med Assoc 1977;170(5):548—53.
[33] Wilkie LJ, Smith K, Fuentes VL. Cardiac pathology findings in 252 cats presented for necropsy: a comparison of cats with unexpected death versus other deaths. J Vet Cardiol 2015;17:329—40.
[34] Joseph S. Studies on the normal and pathological anatomy of the feline heart. Vet Pathol 1908;9(4):431—50.
[35] Sedmera D, Pexieder T, Vuillemin M, Thompson RP, Anderson RH. Developmental patterning of the myocardium. Anat Rec 2000;258(4):319—37.
[36] Chin TK, Perloff JK, Williams RG, Jue KL, Mohrmann R, Miner PD. Isolated noncompaction of left ventricular myocardium. Circulation 1990;82(2):507—13.
[37] Finsterer J, Stöllberger C, Towbin JA. Left ventricular noncompaction cardiomyopathy: cardiac, neuromuscular, and genetic factors. Nat Rev Cardiol 2017;14(4):224—37.
[38] Towbin AJ, Jefferies JL. Cardiomyopathies due to left ventricular noncompaction, mitochondrial and storage diseases, and inborn errors of metabolism. Circ Res 2017;121:838—54.
[39] Vilcu M, Scurtu I, Ohad DG, Papuc I, Scurtu L, Tabaran F. Canine infantile left ventricular noncompaction. BMC Vet Res 2020;16:255.
[40] Miller MW, Gordon SG, Saunders AB, Roland RM. Noncompaction cardiomyopathy in a feline population: echocardiographic and pathologic findings. J Feline Med Surg 2007;9(3):185—93.
[41] Kittleson MD, Fox PR, Basso C, Thiene G. Naturally occurring biventricular noncompaction in an adult domestic cat. J Vet Intern Med 2017;31:527—31.
[42] Maron BJ, Henry WL, Roberts WC, Epstein SE. Comparison of echocardiographic and necropsy measurements of ventricular wall thicknesses in patients with and without disproportionate septal thickening. Circulation 1977;55(2):341—6. https://doi.org/10.1161/01.CIR.55.2.341.
[43] Basso C, Michaud K, d'Amati G, Banner J, Lucena J, Cunningham K, Leone O, Vink A, van der Wal AC, Sheppard MN. On behalf of the Association for European Cardiovascular Pathology. Cardiac hypertrophy at autopsy. Virchows Arch 2021;479(1):79—94. https://doi.org/10.1007/s00428-021-03038-0.