The relevance of left ventricular bands

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Summary

Left ventricular bands (LVBs) were first described almost a century ago but were largely ignored until their 'rediscovery' by echocardiography. Renewed interest in LVBs and the demonstration of their association with clinical abnormalities have resulted in attempts to establish their causal relationship with various phenomena, especially the vibratory systolic murmur and premature ventricular contractions. Published articles on LVBs are reviewed with specific reference to nomenclature, embryological development, histological features, prevalence, demonstration in vivo and at autopsy, and alleged clinical importance. Current views on LVBs are evaluated and future research directions are suggested.

Left ventricular bands (LVBs) (Fig. 1) have been shown to be a common feature in the human heart. 1 They are even more common in animals. 2-4 Despite this, they receive little if any attention in current human anatomy and cardiac pathology textbooks 5-8 and no mention is made of their structure, prevalence or functional importance.

The recent in vivo demonstration of LVBs by echocardiography (Fig. 2) has resulted in a renewed interest in these previously ignored structures, 1,9-31 and conflicting reports have appeared regarding their association with clinical phenomena such as murmurs, 10,25 thrombi, 12 left ventricular aneurysm, 27 left ventricular dysfunction, 1,25,26 and atypical chest pain. 56

Fig. 1. Left ventricular band (arrow) passing from the basal interventricular septum to the posterior papillary muscle.

Fig. 2. Two-dimensional echocardiogram (modified apical four-chamber view) showing a left ventricular band (arrow) as a bright linear echo between the mid-portion of the interventricular septum and the apical free wall (LV = left ventricle; RV = right ventricle; LA = left atrium; RA = right atrium).

Reports on the prevalence and 'normality' of LVBs vary. Some investigators state that they are rare and only occur in about 0.2% 11 of the population. Other reports suggest that they are common and their prevalence may be as high as 61%. 26 Numerous terms are used to describe these structures, most of which imply that they are abnormalities of no functional importance. 6-12, 14-22,25,33,35 Some authors, however, regard them as an important part of the cardiac conduction system. 31,36,37

The apparent confusion in the literature regarding the structure, function, nomenclature, clinical importance and prevalence of LVBs prompted this review.

Nomenclature and definition

Various terms have been used to describe tissue bands crossing the left ventricle: trabeculae septomarginalis, 2,3 musculi transversi, 12 anomalous or aberrant left ventricular bands, 9,12,32 left ventricular false tendons, 10,19,20,22,27 false tendons, 10,19,20,22,27,33 false or anomalous chordae tendineae, 5,18,24,32 moderator bands, 13,39 chordal structures, 23 left ventricular chordae, 24 aberrant tendons, 28 abnormal left ventricular chordae, 34 anomalous bands, 13 left ventricular bands, 23,27,28,31 pseudotendons, 29 intracardiac strings, 12,27,28,33 aberrant fibrous bands and ventricular cords. 1 The use of terms such as 'abnormal', 'false' and 'aberrant' implies anomaly, whereas autopsy 1 and echocardiographic 26 studies confirm that these tissue bands occur frequently. The term left ventricular bands, as preferred by Gerlis et al., 1 is therefore used to describe all discrete tissue bands with a free intraventricular course which may pass from papillary muscle to papillary muscle, ventricular septum to papillary muscle, interventricular septum to free ventricular wall, or free ventricular wall to free ventricular wall. This term adequately describes these structures and will distinguish LVBs from truly anomalous bands, such as those which pass through two, or even three, cardiac chambers. 31,42
Embryology and comparative anatomy

The chambers of the early embryonic heart enlarge by a process of trabeculation1 whereby degeneration of subendocardial cardial cells results in expansion of the ventricles into the space occupied by the thick inner layer of the primitive myocardial coat.5,13,43,44 Local differences in the rate and extent of cell death result in retention of muscular and tendinous cords crossing the left ventricular cavity. These give rise to the trabeculae carnea and chordae tendineae.6,43,44 It would seem likely that LVBs develop in a similar fashion.

LVBs are a normal anatomical feature of canine, bovine, equine and feline hearts,1,29 and it is generally accepted that they form part of the conduction system.1,26,27,42 Standard human anatomy texts describe two structures which, by definition, could be regarded as LVBs: (i) those bands which resemble chordae tendineae in that they originate from a papillary muscle but are not attached to the mitral valve leaflets the 'false chordae'52; and (ii) those trabeculae carnea which have a free intraventricular course,5,6,37 thus forming bridges.4,49

As early as 1906 Keith and Flack39 suggested that LVBs play a role in a conduction. Histological transverse sections demonstrated the presence of Purkinje-type cells in LVBs examined in recent studies.17,37,39 Hudson1,7 does not support this finding and could not demonstrate Purkinje-type cells in the mid-portions of LVBs sectioned.

LVBs are usually single.2,8 The interventricular septum is the most common site of origin2,13 and the majority of LVBs insert in the posterior papillary muscle.1 A conical 'muscle base' may be noted at the attachment site.2,8 Sometimes LVBs are branched or even net-like.17 Most autopsy studies do not mention the dimensions of LVBs, although Nishimura et al.13 reported an LVB 1 mm in diameter and 23 mm in length.

Prevalence and demonstration in humans

Until recently LVBs could only be demonstrated at autopsy.1,2,26 Gerlis and co-workers examined 686 hearts for the presence of LVBs. Bands were found in 303 of the 636 paediatric hearts and in 26 of the 50 adult hearts examined. Similar findings were reported by others.31 LVBs have been reported to be demonstrable on ventriculography,1,2,42 however, other investigators10,22 reported that they had been unable to demonstrate LVBs by this method.

LVBs can be demonstrated on M-mode but are more accurately demonstrated on two-dimensional echocardiography. Echocardiographic reports on their prevalence range from as low as 0.2%11 to as high as 61.9%.39 This remarkable difference could be explained on the grounds of study design, the quality of the equipment used and the skill of the examiner. It can be expected that prospective studies by skilled examiners using high-quality equipment will yield results approaching those of autopsy studies (48 - 52%).1,11

Various authors11,13,16,20,23 have suggested that LVBs may be confused with pathological structures in the left ventricular outflow tract. In our opinion the most important conditions that may be confused with LVBs are asymmetrical left ventricular hypertrophy, left ventricular aneurysm, and thrombi, especially pendunculated forms.

Clinical implications

There are numerous reports of murmurs in the presence of LVBs.4,10,11,15,18,24,36,39 Before the advent of echocardiography the association of LVBs with a clinical murmur could only be made retrospectively at autopsy.12,25 In a recent study12 to investigate the association between the vibratory systolic murmur (VSM) and LVBs, a group of 34 subjects with the VSM was compared with a control group of 22 subjects who had no murmurs. The association between the presence of the VSM and LVBs in the left ventricular outflow tract was significant (P < 0.001). Some investigators have, however, reported LVBs in the absence of murmurs. Nishimura et al.13 observed no murmurs in a group of 5 patients known to have LVBs. Vered et al.18 reported 9 subjects with LVBs who had no murmurs.

The VSM (Still's murmur) has been adequately reviewed elsewhere.25,36 The cause is still unknown,65 but its musical nature suggests that it may result from the oscillation of some other structure within the LV. Furthermore, it has been shown to originate in the region of the aortic valve.6,26 Brenner et al.36 and Wessel et al.24 reported systolic flutter of an LVB associated with the VSM. We suggest that the murmur is the result of an aeolian oscillation64 (periodic wake fluctuations65) caused by blood flow around an LVB. The characteristics of the aeolian oscillation (regular vibration within a narrow frequency range23) are reminiscent of the musical nature65,66 of the VSM. It seems unlikely that turbulence could cause the VSM,57,61 although turbulence generates a broad spectrum of frequencies and, furthermore, careful Doppler examination of subjects with both the VSM and LVBs had failed to demonstrate any turbulence to which a murmur could be attributed.57

The association of LVBs and Still's murmur throws new light on studies65,66 performed on children with the VSM. These children have been shown to have shorter pre-ejection periods65 and larger QRS complexes66 than children without murmurs.

Premature ventricular contractions (PVCs) were present in 4 of the 31 patients with LVBs reported by Perry et al.19 Suwa et al.35 evaluated the coexistence of LVBs and PVCs in apparently healthy individuals and demonstrated LVBs in 35 subjects with PVCs. Proposals10,21,25 that stretching of LVBs may cause PVCs seem tenable in the light of evidence that stretching of canine LVBs increased the automaticity of the Purkinje cells in these bands.26 However, the high prevalence of LVBs in the human population1,24,29 demands larger studies before the association between LVBs and PVCs is established. LVBs have been demonstrated as a site for thrombus formation.8 Vibration is known to cause Raynaud's phenomenon.71 Analysis of the vibration of LVBs may help determine whether or not trauma could result and whether this in turn could have been the cause of reported rupture1 of LVBs as well as thromboses on LVBs.

Frieri et al.37 observed LVBs in 5 patients with left ventricular aneurysm. Distortion of the left ventricle has been observed in association with LVBs,21,26,26 Of the 31 subjects studied by Casta and Woll21 7 are reported to have had an 'hour-glass' configuration of the left ventricular cavity. Beattie et al.36 reported 10 cases of atypical chest pain in which LVBs were the only 'abnormalities' observed. The majority of these bands were perpendicular to the left ventricular outflow tract, and the investigator proposed that this position rendered the LVBs more likely to activate myocardial pain fibres.

Since pulsus parvus of LVBs in the conduction process in the left ventricle should be borne in mind during invasive procedures such as cardiac surgery and catheterisation.37 It does not seem unlikely that a catheter could snare and rupture an LVB. Possible consequences could include thrombosis, embolism, conduction disturbance and infective endocarditis.

Conclusion

The development of echocardiography has allowed the demonstration of LVBs in vivo and the subsequent association of these structures with numerous clinical phenomena. There is
considerable evidence in support of the suggestion that LVBs can cause a vibratory systolic murmur. Only further research will demonstrate how many of the other associations are significant.

In summary, we emphasise the following as important research areas: (i) the prevalence of LVBs; (ii) the macro- and microscopic anatomy of LVBs; (iii) the relationship between the type of LVB and the nature of the associated vibratory systolic murmur; (iv) an evaluation of the suggestion that the vibratory systolic murmur is caused by an aeolian oscillation of LVBs; and (v) the association between LVBs on the one hand and thrombosis, increased QRS-complex amplitude and decreased pre-ejection period on the other.

Because of their high prevalence LVBs should be expected in approximately half of most groups examined echocardiographically. Investigators should bear this in mind when attempting to establish a significant relationship between LVBs and clinical observations. Regardless of the validity of the abovementioned clinical implications, LVBs remain an important differential diagnosis for less benign structures in the left ventricle. Further investigation in these directions will elucidate the clinical importance of LVBs.

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REFERENCES

Review Article

Primary arteritis of the aorta and its branches

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Summary

Primary arteritis of the aorta and its branches is a single clinicopathological entity affecting one or more segments of the aorta and resulting in a variety of symptom complexes. The prevalence is not known, but it is an important cause of renovascular hypertension in the black population of South Africa. Recently published articles on primary arteritis of the aorta and its branches are reviewed, and an attempt is made to draw conclusions regarding its cause, clinical features and treatment.

Primary arteritis of the aorta and its branches was known as Takayasu's disease, aortic arch syndrome, pulseless disease or brachial neuritis. Any part of the aorta may be affected. Type I is confined to the arch and its branches, type II involves the thoracic descending and abdominal aorta, type III is a combination of types I and II, and type IV involves the aortic arch in addition to the aorta. Various series have been described, the largest from Japan. Others are from Singapore, Thailand, the Philippines, India, South Africa, Latin America and Europe.

Causation

The cause of primary arteritis of the aorta is unknown. In India tuberculosis has been considered a cause. The largest series in India is in Bombay, where a registry for the disease shows that 70% of patients have tuberculosis elsewhere in the body. Primary arteritis is more common in the lower socioeconomic group. Active or quiescent tuberculosis may be present, and a tuberculin test is invariably positive. In view of the high prevalence of tuberculosis in Asian countries, this association may be coincidental. There is some evidence that the disease has an auto-immune cause. A genetic factor has been suggested.

Pathological features

There is irregular thickening of the walls of the aorta with dilatation or alternating dilatation and stenosis. The extent of the disease varies, but autopsy or aortography often reveals involvement of more than one site. There is considerable thickening of the aortic wall with focal or superimposed intimal thrombosis. In many cases the thrombotic process extends to involve a renal artery. Microscopically the lumen of the aorta is often markedly narrowed by both old fibrous intimal thickening and recent antemortem thrombus. The adventitia is fibrosed and shows heavy focal infiltration by plasma cells and lymphocytes. The elastic tissue of the media is fragmented where inflammatory cells extend into it. Much of the vasa vasorum shows endarteritis. In the most severely affected parts there is replacement fibrosis of the elastic tissue and muscle by dense acellular collagen. In the presence of hypertension left ventricular hypertrophy is seen, and if congestive cardiac failure has been present there is passive congestion of the remaining organs. Occasionally the para-aortic lymph nodes show evidence of a granulomatous reaction resembling tuberculosis, and it has been suggested that a hypersensitivity reaction to the tubercle bacillus in these lymph nodes may set up a reactive endarteritis of the vasa vasorum.

Differential diagnosis

Atheroma of the aorta causes the pulseless syndrome but occurs in older persons. The changes are primarily adventitial and medial, however, in contrast to the predominantly intimal...