STATE-OF-THE-ART REVIEW ARTICLE

Left Ventricular False Tendons: Anatomic, Echocardiographic, and Pathophysiologic Insights

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Left ventricular (LV) false tendons are chordlike structures that traverse the LV cavity. They attach to the septum, to the papillary muscles, or to the free wall of the ventricle but not to the mitral valve. They are found in approximately half of human hearts examined at autopsy. Although it has been more than 100 years since their initial description, the functional significance of these structures remains largely unexplored. It has been suggested that they retard LV remodeling by tethering the walls to which they are attached, but there are few data to substantiate this. Some studies have suggested that false tendons reduce the severity of functional mitral regurgitation by stabilizing the position of the papillary muscles as the left ventricle enlarges. LV false tendons may also have deleterious effects and have been implicated in promoting membrane formation in discrete subaortic stenosis. This article reviews current understanding of the anatomy, echocardiographic characteristics, and pathophysiology of these structures. (J Am Soc Echocardiogr 2013;26:582-8.)

Keywords: Left ventricular false tendons, Left ventricular remodeling, Myocardial infarction, Functional mitral regurgitation, Ischemic mitral regurgitation, Discrete subaortic stenosis, Echocardiography

Left ventricular (LV) false tendons were first described in 1893 by the British anatomist and surgeon Sir William Turner.[1] Although more than 100 years have passed since that initial description, the functional significance of these structures remains largely unexplored. Turner proposed that they retard LV enlargement (remodeling) by tethering the walls to which they are attached,[1] but there are few data to substantiate this. It has also been suggested[2] that false tendons reduce the severity of functional mitral regurgitation (MR) by stabilizing the position of the papillary muscles as the left ventricle enlarges. LV false tendons may also have deleterious effects and have been implicated in promoting membrane formation in discrete subaortic stenosis (DSS).[3] This article reviews current understanding of the anatomy, echocardiographic characteristics, and pathophysiology of these structures.

ANATOMY OF LEFT VENTRICULAR FALSE TENDONS

During embryologic development of the heart, two distinct myocardial layers can be identified: an outer condensed layer and an inner, less compact layer. The latter is composed of trabeculations that produce irregular ridges that protrude into the LV cavity and are separated from one another by intertrabecular recesses. False tendons arise from the inner trabeculated myocardial layer, but unlike trabeculations, these chordlike structures traverse the LV cavity.[4,5]

LV false tendons are found in about half of hearts examined at autopsy[6-9] and occur with equal frequency in normal hearts and in those with congenital malformations.[8] Autopsy and surgical series have demonstrated a slight male preponderance.[10] False tendons give attachment to the LV free wall, to the interventricular septum, or to the papillary muscles. On the basis of their sites of attachment, five types have been delineated,[7,9] as depicted in Figures 1 and 2. False tendons range in thickness up to about 3 mm[9] and contain varying amounts of fibrous and myocardial tissue as well as coronary vessels, which run the length of their shafts,[6,11] and Purkinje fibers, which are in continuity with the left bundle branch of the conduction system[12,13] (Figure 3). False tendons containing conduction tissue have been identified as substrates of intracavitary ventricular tachycardia[12,13] and have been successfully ablated.[13] In one study, such false tendons consistently extended from the inferoposterior LV wall to the septum and were the focus of ventricular tachycardia characterized by right bundle branch block morphology and left-axis deviation.[12] It is worth noting that the right ventricle harbors a solitary false tendon, commonly called the moderator band, which contains conduction tissue arising from the right bundle branch of the conduction system.[14]

ECHOCARDIOGRAPHIC FEATURES OF LEFT VENTRICULAR FALSE TENDONS

According to a number of early studies,[8,15,16] LV false tendons are found far less often echocardiographically than at autopsy, but detection rates appear to have improved with the advent of harmonic imaging.[17] In one study using pathologic specimens explanted at the time of cardiac transplantation as a standard, the preoperative sensitivity and specificity of echocardiography in detecting false tendons were 82% and 85%, respectively.[18] It should be emphasized, however, that conventional imaging planes are not well suited for detecting LV false tendons, and off-axis imaging is often required.[19] In general, longitudinally oriented false tendons can best be seen in parasternal or apical long-axis views, whereas transversely oriented false tendons are more readily visualized in the apical

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0894-7317/$36.00

http://dx.doi.org/10.1016/j.echo.2013.03.005

Abbreviations
DSS = Discrete subaortic stenosis
LV = Left ventricular
MR = Mitral regurgitation

four-chamber and short-axis views (Figure 4).[18] False tendons become more taut in diastole and more lax in systole[20] (Figure 5); LV enlargement may render them taut throughout the cardiac cycle.[21] When sufficiently taut and oriented more or less perpendicularly to the axis of blood flow, false tendons vibrate in much the same way as the strings of an Aeolian harp (Figure 6) when swept by the wind.[22] These vibrations can be seen as fine fluttering on M-mode recordings (Figure 7) and may be the cause of innocent (Still's) murmurs.[9,22-24] Identification of false tendons is enhanced in the dilated, thin-walled ventricle, which causes them to stand away from away from the endocardial surface of the heart.[25] LV false tendons are sometimes mischaracterized. Those near the LV apex may be confused with the edge of a thrombus,[26] and those closely applied to the septum may give the false impression that there is LV hypertrophy or hypertrophic cardiomyopathy.[27] Features that help in differentiating false tendons from other structures include the presence of echo-free spaces on both sides of the tendon and systolic laxity.[18] It is worth noting that LV false tendons frequently fan out, creating a broad base of attachment to the LV wall, as depicted in Figures 2F and 4B,[28,29] which can be mistaken for a papillary muscle, thrombus, or trabeculation. Rupture of a false tendon, whether spontaneous or in the setting of myocardial infarction, produces highly mobile intracavitary echoes that must be distinguished from vegetations, thrombi,[30] and ruptured chordae tendineae.[31]

Epidemiologic data from the Framingham Heart Study[20] revealed that individuals with echocardiographically identified LV false tendons are more likely to have lower body mass indexes, but, this finding may be a reflection of the superior image quality obtained in such individuals. The same study found that electrocardiographic criteria for LV hypertrophy were more common among individuals with LV false tendons. Finally, the Framingham study[20] concluded that the presence of LV false tendons on echocardiographic examination failed to impart any increase in mortality risk.

DO LEFT VENTRICULAR FALSE TENDONS LIMIT POSTINFARCTION REMODELING?

LV remodeling after myocardial infarction is an adaptive response designed to preserve stroke volume. Although this may work for a time, the increase in LV size augments wall stress, which, through the Laplace relationship, further increases LV size, leading to a vicious cycle in which "dilatation begets more dilatation." The inexorable rise in LV load precludes maintaining an adequate stroke volume, and LV failure eventually ensues.[32] Increased wall stress also upregulates metalloproteinase activity, which degrades the myocardial extracellular matrix, further promoting LV dilatation and failure.[33]

LV false tendons may retard remodeling through the restraining effects they exert on the LV walls to which they are attached. It is here proposed, allowing for variation, that LV false tendon types 1 to 3 (Figure 1) appear anatomically well situated to limit the extent of remodeling that follows inferoposterior myocardial infarction. Moreover, these false tendons may stabilize the position of the papillary muscles, reducing the likelihood that ischemic MR will develop

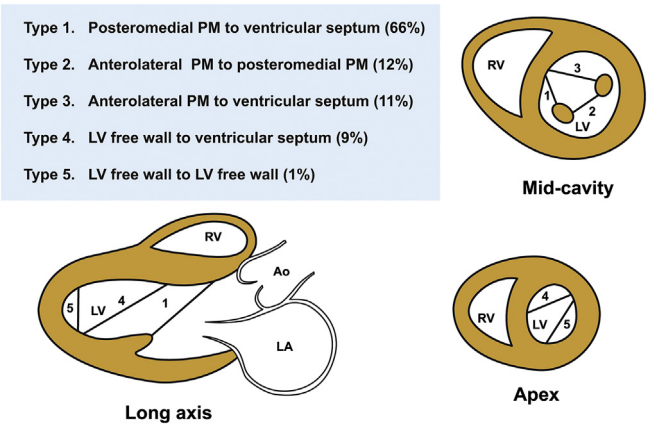

Figure 1 Five common types of LV false tendon connections. The numbers in parentheses represent the incidence of each observed at autopsy. *Ao*, Aorta; *LA*, left atrium; *PM*, papillary muscle; *RV*, right ventricle. Reproduced with permission from Luetmer *et al*.[9]

(see below). On the other hand, LV false tendon types 4 and 5 (Figure 1) appear anatomically well situated to limit remodeling that follows anterior myocardial infarction.

The Coapsys device (Myocor, Maple Grove, MN0 might be regarded as a prosthetic false tendon. This device consists of epicardial pads connected by a flexible chord that traverses the LV chamber along its anteroposterior axis (Figure 8). The internal ventricular diameter of the chord can be adjusted by drawing the epicardial pads together.[34] Finite element analysis has demonstrated that the Coapsys device reduces LV wall stress,[35] which likely contributes to the reverse remodeling that is observed with its use.[36]

The foregoing observations raise a number of provocative questions that might well serve as the basis for future investigations into the functional role of LV tendons: (1) Do native LV false tendons reduce wall stress and promote reverse remodeling in a manner comparable with that observed with the Coapsys device? (2) Are individuals deficient in LV false tendons at greater risk for remodeling after myocardial infarction than those replete with these tendinous attachments? (3) Is the presence of LV false tendons a heritable trait, and if so, what is the mode of inheritance?

It is interesting to note that immunohistochemical studies performed in rats reveal that not only the atria but also LV false tendons are a source of atrial natriuretic peptide.[37] The secretion of atrial natriuretic peptide is triggered by increased LV filling pressure and promotes natriuresis, diuresis, and vasodilatation.[38] The resulting decrease in wall stress therefore appears to represent a humorally mediated mechanism by which LV false tendons impede remodeling. Atrial natriuretic peptide also retards LV remodeling through its inhibitory effects on endothelin-1, angiotensin II, and aldosterone, all of which promote remodeling by stimulating cardiac fibrosis.[39,40]

DO LEFT VENTRICULAR FALSE TENDONS REDUCE THE SEVERITY OF FUNCTIONAL MITRAL REGURGITATION?

Functional MR is caused by remodeling of the left ventricle and can occur after myocardial infarction (ischemic MR) and in the setting of idiopathic dilated cardiomyopathy (nonischemic MR). Regardless of the cause, as the left ventricle remodels, the papillary muscles

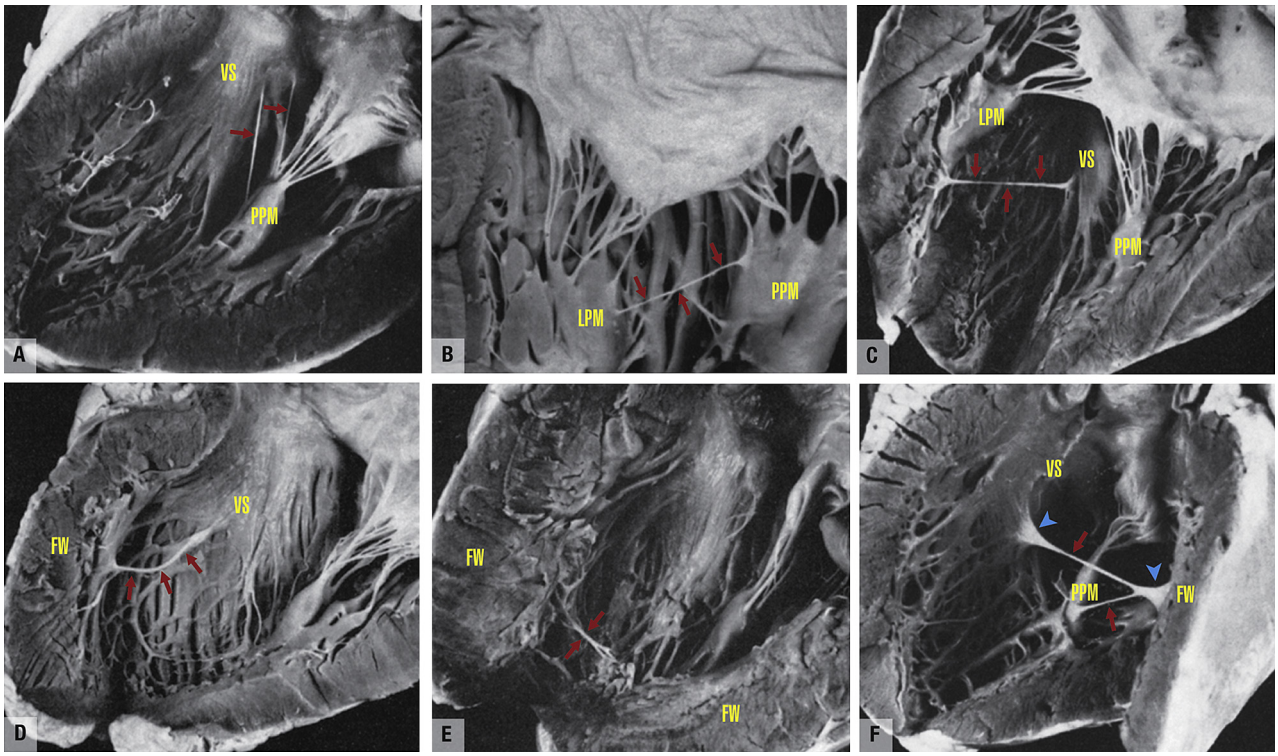

Figure 2 Gross pathologic specimens of LV false tendon connections. **(A)** Two false tendons spanning between the posterior papillary muscle (PPM) and the ventricular septum (VS). **(B)** False tendon connecting the lateral papillary muscle (LPM) and the PPM. **(C)** False tendon spanning between the LPM and the VS. **(D)** False tendon connecting the VS and the LV free wall (FW). **(E)** False tendon spanning between LV FWs. **(F)** A branching false tendon arising from the FW connecting to both the VS and the PPM. Note the broad base at the sites of attachment (*blue arrowheads*). Reproduced with permission from Luetmer *et al*.[9]

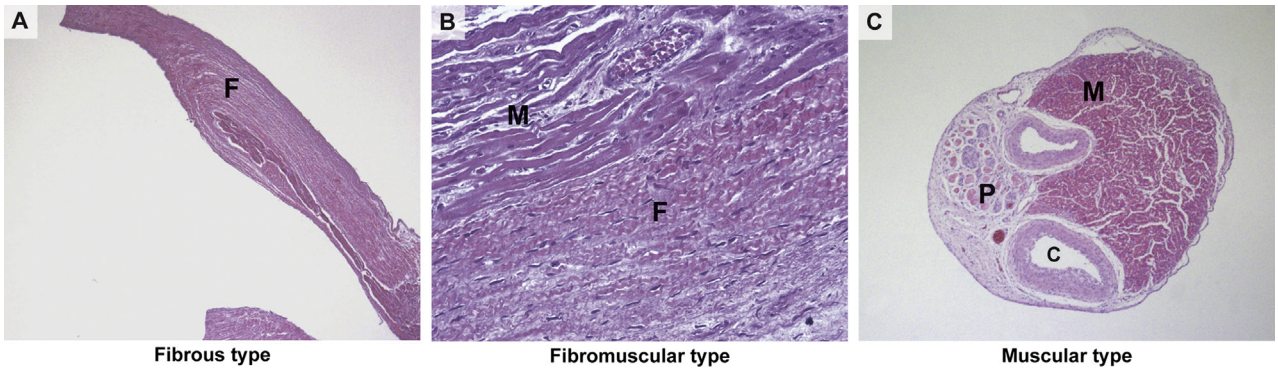

Figure 3 Histologic sections of LV false tendons. **(A)** Fibrous type. **(B)** Fibromuscular type composed of fibrous tissue (F) and myocytes (M). **(C)** Muscular type composed of myocytes, Purkinje cells (P), and coronary arteries (C). Reproduced with permission from Philip *et al*.[6]

become displaced away from the annular plane and, in so doing, exert traction on the chordae tendineae. The latter causes effacement (deformation) of the mitral leaflets resulting in incomplete valve closure and regurgitation.[41]

In one study[2] of patients with functional MR of both ischemic and nonischemic etiologies, those with transversely oriented midcavity LV false tendons were found to have significantly less regurgitation than those without such false tendons. The authors found that these patients had less mitral valve deformation (decreased tenting depth and area) and attributed this to the restraining effects these false tendons exert on the papillary muscles (Figure 9).

LV dyssynergy is not uncommon in patients with functional MR and may contribute to its pathogenesis in a number of ways. Delayed activation of the anterolateral papillary muscle can cause uncoordinated papillary muscle contraction, resulting in malalignment of the mitral leaflets.[42] Dyssynchronous contraction can also blunt the rate of LV pressure generation (LV dP/dt). The resulting decrease in mitral valve closing force leaves leaflet tethering forces relatively unchecked, thereby increasing the mitral regurgitant orifice area.[43] Conduction through false tendons spanning the septum and lateral wall may shorten intraventricular conduction time in much the same way as biventricular pacing. Although the latter improves

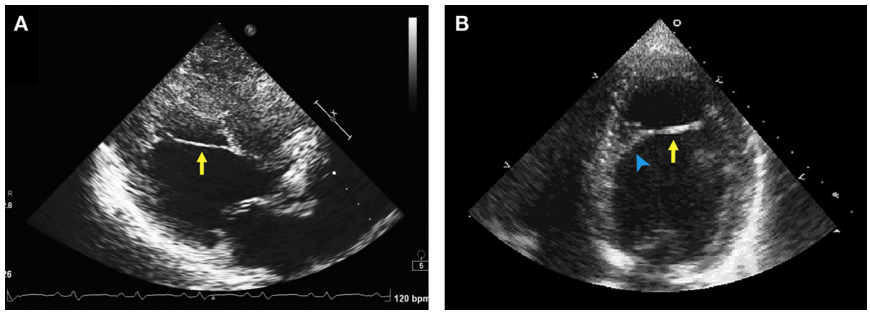

Figure 4 **(A)** Apical long-axis view of a longitudinally oriented false tendon (*arrow*). **(B)** Apical four-chamber view of a transversely oriented false tendon (*arrow*). Note its broad base of attachment to the interventricular septum (*blue arrowhead*).

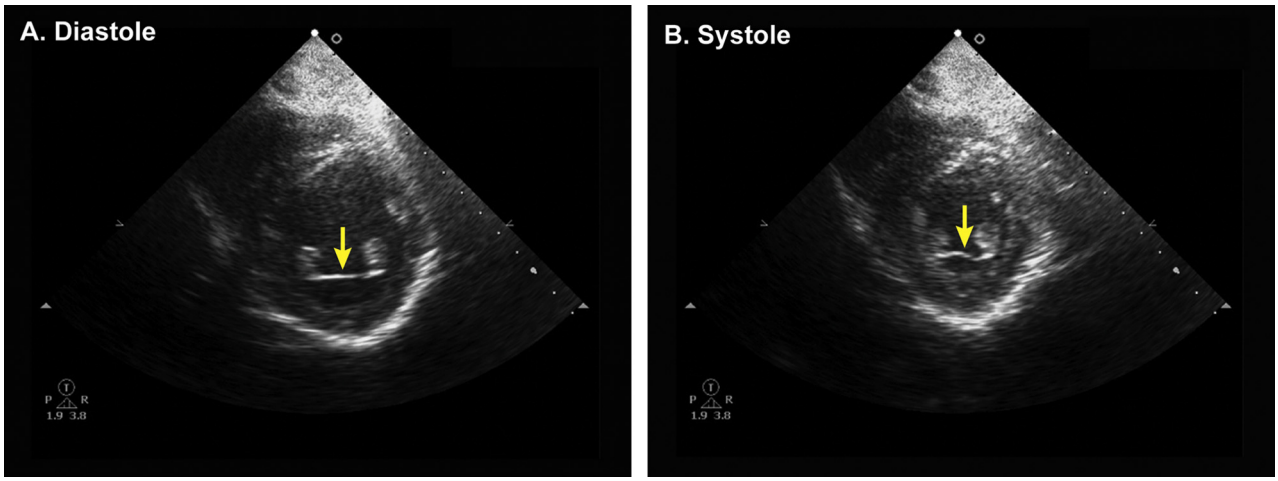

Figure 5 LV false tendon. **(A)** Diastolic frame showing a false tendon under tension spanning between the papillary muscles. **(B)** Systolic frame showing laxity of the same false tendon. Reproduced with permission from Ker.[31]

Figure 6 Photograph of an Aeolian harp, designed to produce sound when wind, rather than the human hand, moves across its strings. The harp is named after Aeolus, the ancient Greek god of the winds.

functional MR by shortening the time to activation of the anterolateral papillary muscle[42] and by increasing LV dP/dt,[43] it remains to be determined if conduction through septolateral false tendons has similar effects.

DO LEFT VENTRICULAR FALSE TENDONS PLAY A ROLE IN THE PATHOGENESIS OF DISCRETE SUBAORTIC STENOSIS?

DSS is characterized by the formation of a progressive, obstructing, fibrous or fibromuscular lesion in the LV outflow tract. The outflow tract in DSS is frequently narrow and/or long.[44,45] The latter is

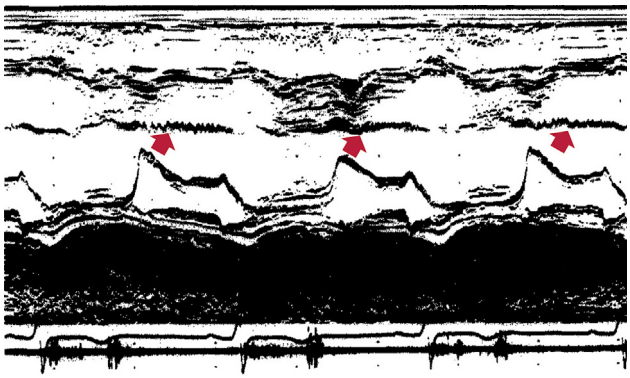

Figure 7 M-mode tracing of a false tendon in the LV outflow tract. Note the fine diastolic fluttering (*red arrows*), presumably produced by an anteriorly directed jet of aortic regurgitation striking it. Reproduced with permission from Nishimura *et al*.[24]

believed to result from widening of the aortomitral curtain, which increases the separation between the aortic and mitral annuli.[44] These geometric abnormalities are thought to play a role in the pathogenesis of DSS. Blood flow in the normal outflow tract appears laminar when imaged echocardiographically (Figure 10). When the outflow tract is narrow, however, the velocity of blood flow increases, which causes flow to become turbulent. Under such circumstances, surface irregularities, such as those produced by false tendons, are

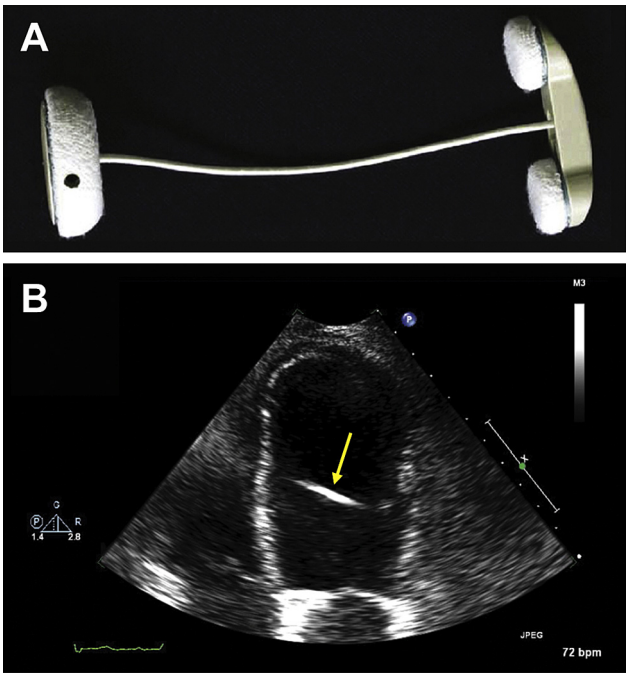

Figure 8 **(A)** Coapsys device. Note the flexible chord with attached epicardial pads. The double-padded side rests on the posterior wall of the left ventricle, and the single-padded side rests on the anterior wall of the left ventricle. **(B)** Apical four-chamber echocardiographic image showing the chord of a Coapsys device traversing the LV cavity (*arrow*).

more likely to become sources of flow destabilization and promote more turbulence. When present, elongation of the LV outflow tract further exacerbates these flow disturbances.[45] Figure 11 shows an echocardiographic image from a patient with a long and narrow outflow tract and an LV false tendon attached to the interventricular septum. Note the large cloud of turbulence originating at its site of attachment.

It has been suggested that in genetically predisposed individuals, the presence of turbulent flow can alter the phenotypic expression of endocardial cells of the LV outflow tract, resulting in the formation of the characteristic fibroproliferative lesion of DSS.[46] The process is thought to be initiated by the activation of endocardial cell mechanoreceptors by the changes in the magnitude and direction of the vector of septal shear stress that accompany outflow tract turbulence. Activated mechanoreceptors, in turn, are thought to trigger conformational changes within cytoskeletal proteins (mechanotransduction). At the level of the nucleus, these proteins interact with a number of mechanosensitive genes, culminating in membrane formation.[46,47]

The notion that outflow tract turbulence contributes to the pathogenesis of DSS has been criticized, in part, because the literature generally characterizes turbulence using color Doppler imaging criteria, which are not necessarily equivalent to formal hydrodynamic criteria (increased Reynolds number) and may therefore not be biologically relevant.[45,48,49] Nevertheless, some surgeons advocate removal of protruding mass lesions in the subaortic region as an adjunct to conventional decompression procedures (i.e., membranectomy and myectomy).[50]

CONCLUSIONS

LV false tendons are seldom sought during routine echocardiographic examinations. In light of the foregoing, it is suggested that consideration be given to this practice. The role of three-dimensional imaging in echocardiographic identification of LV false tendons remains to be established.

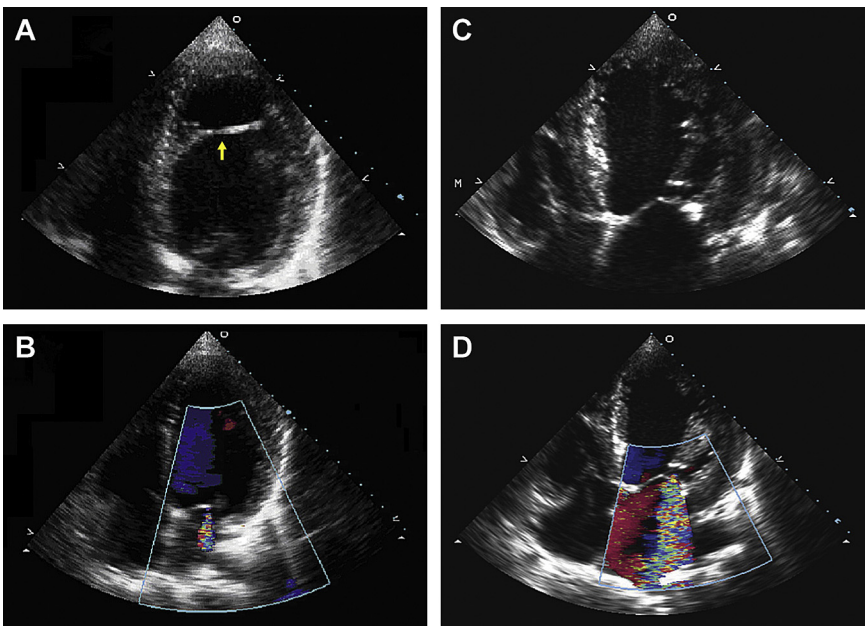

Figure 9 Echocardiographic images from two patients with dilated cardiomyopathy (LV ejection fraction <30%). The images in **(A)** and **(B)**, from a patient with a transverse midcavity false tendon (*arrow*), reveal mild MR and a coaptation depth of 0.9 cm. By contrast, the images in **(C)** and **(D)**, from a patient without a transverse midcavity false tendon, reveal severe MR and a coaptation depth of 1.8 cm. Reproduced with permission from Bhatt *et al*.[2]

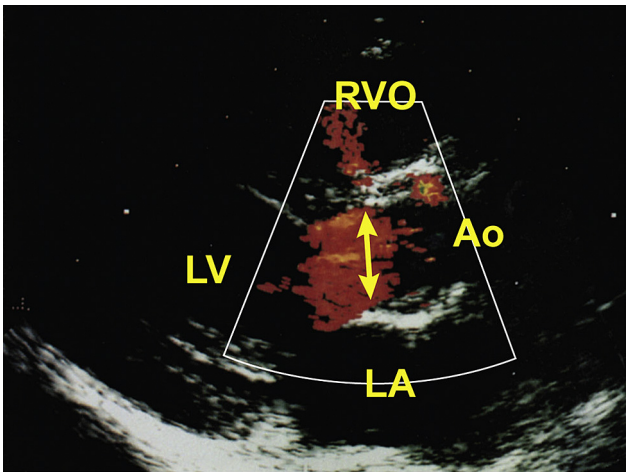

Figure 10 Long-axis echocardiographic image with color Doppler showing homogeneous *red* color in the LV outflow tract (*double-sided arrow*), suggesting that flow is laminar. *Ao*, Aorta; *LA*, left atrium; *RVO*, right ventricular outflow tract. Reproduced with permission from Gewillig *et al*.[45]

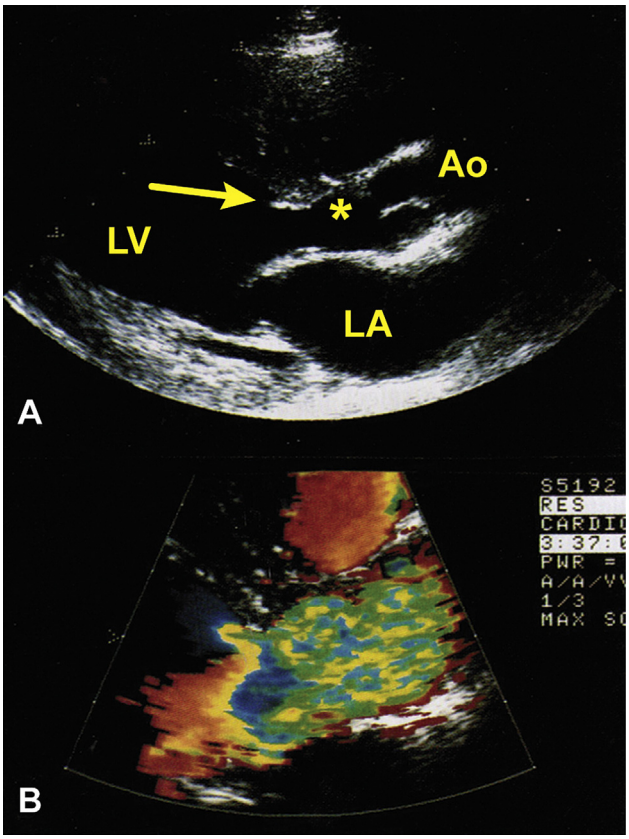

Figure 11 Long-axis echocardiographic images. **(A)** The *arrow* points to the insertion site of a false tendon. The *asterisk* indicates the site of prior membrane resection. **(B)** Note the large cloud of turbulence originating at the site of attachment of the false tendon, which fills the subaortic region. The turbulent cloud comes into contact with the site of prior membrane resection (*asterisk*) and may therefore represent a potential stimulus for membrane recurrence. *Ao*, Aorta; *LA*, left atrium. Reproduced with permission from Gewillig *et al*.[45]

It is here proposed that individuals whose hearts harbor LV tendons may be at some teleologic advantage because of their potential to retard postinfarction remodeling and to limit the extent of mitral valve deformation. Should this supposition be borne out, it is likely that future research efforts to address the genetic basis of false tendon hereditability will prove invaluable. It is also reasonable to assume that a greater understanding of the role that abnormal shear stress plays in DSS is likely to influence future treatment strategies of this disorder. New therapeutic targets might include endocardial mechanoreceptors, cytoskeleton proteins, and mechanosensitive genes. An improved understanding of the functional significance of LV false tendons may allow us to develop newer therapies and to achieve better patient outcomes.

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