Anatomy and myoarchitecture of the left ventricular wall in normal and in disease

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KEYWORDS
Anatomy;
Myocardium;
Myoarchitecture;
Ventricular function

The normal left ventricle comprises an inlet, apical trabecular, and an outlet portion although these portions do not have discrete anatomical borders. The ventricular wall is thickest near the cardiac base and thins to 1–2 mm at the apex. Characteristically, the muscle bundles at the apical portion are thin, but there are also thicker bundles and very fine strands that may be mistaken on imaging as pathologies. Transmurally through the ventricular wall, the myoarchitecture has a typical arrangement of myocardial strands that change orientation from being oblique in the subepicardium to circumferential in the middle and to longitudinal in the subendocardium. The circumferential portion is the thickest with the longitudinal portion the thinnest. In the hypertrophied ventricle the circumferential portion is reduced. In combination with alterations in the quality and quantity of the connective tissue matrix, myoarchitecture impacts on myocardial function.

Introduction

This review focuses on the myocardial component of the left ventricle, leaving aside the aortic and mitral valves. First, the muscular structures and normal variants within this chamber are described and then the potential misinterpretation of normal structures is discussed. Secondly, the orientation of the myocardial strands in the normal and abnormal left ventricle is reviewed together with a consideration of the connective tissue matrix of the myocardium.

Anatomy of the left ventricle

The normal left ventricle comprises an inlet portion containing the mitral valve apparatus, an outlet portion leading to the aortic valve, and an apical portion containing fine trabeculations (Figure 1). From a simplistic viewpoint, the shape of the left ventricle approximates to a cone with the right ventricle hugging it. Consequently, the septal component of the ventricular wall is curved. Thus, when the heart is viewed from the anterior aspect, most of the left ventricle is hidden by the right ventricle. Furthermore, the left ventricular outlet overlaps the inlet. Normally, the left ventricular free wall is thickest at the cardiac base and it gradually becomes thinner towards the apex. At the very tip of the ventricle, the musculature is only 1–2 mm thick, even in hypertrophied ventricles (Figure 2). The normal thickness at the obtuse margin of the left ventricle for an adult heart is 12–15 mm, excluding trabeculations, when measured approximately 1.5 cm below the mitral hingeline (annulus). The endocardial aspect of the ventricle is characterized by a criss-crossing meshwork of thin muscle bundles (trabeculations) at the apical third of the ventricle. Not infrequently, fine muscular strands or so-called false tendons extend between the septum and the papillary muscles or the parietal wall (Figure 2). Autopsy series of normal hearts have reported an incidence of 55 and 62%.1,2 On echocardiography they may be mistaken for ruptured chords of the valve apparatus or vegetations. In contrast, the outlet portion of the septum is relatively smooth whereas relatively thick muscle bundles line the antero-superior, postero-inferior, and posterior walls (Figure 2). The latter, described as numbering three or fewer, are normal structures that when prominent may cause over-diagnosis of non-compaction of the left ventricular myocardium on echocardiography.3 The pathological entity of non-compaction cardiomyopathy, or spongy myocardium, is characterized by prominent and excessive trabeculations with correspondingly deep recesses in between within the hypertrophied wall.4,5 The left ventricular wall most frequently affected is the apical, mid-lateral, and mid-inferior portions.6–8 On heart specimens the affected segments have two components transmurally: a thin compact, or normal, layer in the subepicardium and a thicker subendocardial layer of trabeculations with deep recesses giving a thickness ratio of approximately 1:2. It remains unclear whether non-compaction is a distinct entity from hypertrabeculation where there are more than three prominent trabeculations and is often associated with extracardiac disorders.9

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The ventricular septum in the normal heart is curved, convexing into the right ventricular cavity. It is muscular except for a small portion immediately beneath the aortic valve which is a thin fibrous structure, the ventricular component of the membranous septum. Together with the right fibrous trigone, this thin component forms the central fibrous body that encases the atroventricular conduction bundle and is an anatomical landmark for the emergence of the conduction bundle onto the crest of the muscular septum (Figure 2). Over the age of 60 years, there is an increase in angulation of the basal part of the muscular septum, producing a sigmoid appearance on imaging that can produce features of hypertrophic cardiomyopathy.10,11

The papillary muscles supporting the mitral valve are an integral component of the left ventricular wall. There are usually two groups of papillary muscles disposed in antero-lateral and postero-medial locations of the left ventricle when the ventricle is seen in short-axis cuts. The antero-lateral group is often a single pillar or base whereas there is a more variation in the posteromedial group (Figure 2). Each pillar may have one head or multiple heads into which the tendinous cords insert. Occasionally, there is muscularization of cords with the muscular tip inserting into the undersurface of the leaflet.12 The belly of the papillary muscle may be finger-like or broad, long or short, or nearly sessile with very short free length. There may be multiple bellies that interlink or are arranged in two tiers.13 In between the two groups, there may be additional papillary muscles. Relating to the mural (posterior) leaflet of the mitral valve, there are basal cords that arise directly from the free wall or via small papillary muscles to insert to the under-side of the leaflet. All in all, the bases of the papillary

Figure 1  (A) and (B) Two parts of the same heart sectioned longi-tudinally. The broken lines divide the left ventricle into three por-tions. Note the cross-over relationship between right and left ventricular outflow tracts and the smooth upper part of the ventric-ular septum leading to the aorta. RVOT, right ventricular outflow tract.

Figure 2  (A) The papillary muscles in the left ventricle, fine apical trabeculations, a long false tendon (green arrows), and broad muscle bundles (triangles). The asterisk marks the membranous septum. Note the thin musculature at the apex (arrow). (B) Simu-lated ‘four-chamber’ cut through a heart with a dilated left ventri-cle. The bases of the papillary muscles are continuous with the trabeculations. This heart shows multiple fine strands of false tendons crossing the cavity, and trabeculations also on the posterior and postero-inferior walls. AL, anterolateral papillary muscle; PM, postero-medial papillary muscle.
muscles are attached to the middle to apical thirds of the ventricular wall but there are wide variations. But, the basal parts of the papillary muscles are not solid muscle (Figure 2). Multidetector-row computed tomography imaging in recent years has provided a novel observation that the bases are attached to muscular trabeculations rather than directly to the compact myocardium.14

**The myocardium**

The bulk of the myocardium is formed by the contractile cardiac myocytes. The adult myocyte is usually 120 μm long and 20–30 μm in diameter. The ends of each myocyte branches and adjoin adjacent myocytes to produce a complex three-dimensional network, or syncytium, of anastomosing fibres. In any attempt to relate structure to function, however, one should not ignore the network of interstitial connective tissue (Figure 3A). A fine network of fibrocollagenous connective tissue, the endomysium, surrounds each myocyte providing it with the supportive frame work. The endomysial weave coordinate the transmission of force and prevent slippage between cells. A network of thicker connective tissue surrounds groups of myocytes. Known as the perimysium, this weave bears the shearing forces between groups of cardiomyocytes and its lateral strands prevent malalignment between bundles.19 Abnormal accumulation and/or change in the quality of the connective tissue increases myocardial stiffness.16

**Ventricular myoarchitecture**

Myoarchitecture has been and remains a contentious issue right from the early days. Indeed, Keith,17 in his Harveian lecture in 1918, commented that such meticulous work was dismissed by Hunter in the phrase 'Much more pains than were necessary have been taken to dissect and describe the course and arrangement of the muscular fibres of the heart'. Based on investigation techniques, some argue that there are discrete systems of myocyte arrangements in each ventricle while others are insistent of a single rope-like configuration that encompasses both ventricles.18–20 Even in more recent years, using advanced reconstruction techniques and imaging tools there is much discussion on this subject.21–23 Despite obvious limitations of gross dissections, the myocardial strands (previously termed myocardial or muscle fibres) that are revealed using this basic technique provide a guide to the general longitudinal orientation of the myocytes and serve as an overview of myocyte arrangement, the myoarchitecture.24–27

**Normal left ventricle**

On gross dissection, the left ventricular wall comprises three 'layers' according to the longitudinal alignment of the myocardial strands: superficial (subepicardial), middle, and deep (subendocardial). Importantly, these ‘layers’ represent changes in orientation of the myocardial strands transmurally. They are not separated by cleavage planes or sheets.

![Figure 3](https://academic.oup.com/ehjcimaging/article-abstract/10/8/iii3/2396960/fig3)
of fibrous tissue since strands of one ‘layer’ interconnect with strands of the next ‘layer’ in a continuum.

When traced from the base to the apex, the superficial layer extends from one ventricle to the other (Figure 3B). The myocardial strands arise from the insertions of the cardiac valves at the cardiac base. The superficial strands on the sternocostal aspect run obliquely crossing the interventricular groove, sweep leftward over the obtuse margin and descend towards the cardiac apex. The strands arising from the mitral insertions continue to the diaphragmatic aspect rightward where they cross the inferior (posterior) interventricular groove. At the obtuse margin, they are at an angle of 10–20° to the long axis of the left ventricular inlet. The superficial ‘layer’ occupies approximately 25% of the wall thickness. At the vortex of the left ventricle, the myocardial strands invaginate in a spiral pattern to give rise to the subendocardial ‘layer’. There is a similar continuity between superficial and deep ‘layers’ at the base of the ventricle.

The middle ‘layer’ occupies approximately 53–59% of the ventricular wall thickness. It is thicker in the elderly. The myocardial strands are more circumferentially arranged, nearly parallel to the plane of the mitral orifice (Figure 3B). They do not insert into the mitral or aortic valves, nor to the ventricular apex. This layer is thickest near the base, thinning out towards the cardiac apex. At the base, it encircles the inlet together with the outlet portions.

From the apex of the left ventricle, the deep ‘layer’ of myocardial strands radiate in longitudinal fashion in the subendocardium, to insert into the aortic and mitral valves and the membranous septum (Figure 3B). They fuse with the trabeculations and broader muscle bundles that line the ventricular cavity, and also continue into the papillary muscles. Consequently, the deep myocardial strands form a meshwork determined by the trabeculations at the apical third, muscle bundles on the middle, and the valvar insertion at the basal third. This is the thinnest ‘layer’, accounting for <20% of the wall thickness.

The myoarchitecture of the ventricular septum reflects the parietal wall of both right and left ventricles, the major contribution coming from the middle ‘layer’ of the left ventricle except at the apical portion where the middle ‘layer’ is lacking.

Abnormal left ventricle

There are only a few anatomical studies on abnormal myoarchitecture. A study comparing hearts from normal patients with hearts from patients with history of hypertension without clinical heart failure, and patients with both ventricular overload and congestive failure showed the orientation of the myocardial strands is not altered. Instead, there was an increase in connective tissue content with the greatest increase in the failing heart. Another study, an experimental study on dogs in which histological sections were sampled only at the equator of the left ventricle revealed the myoarchitecture in the pressure, volume, and exercise-overloaded hearts, was remarkably similar to normals. However, these investigators reported an increase in the proportion of longitudinally aligned strands in the subepicardial and subendocardial regions of the wall thickness, with corresponding decrease of circumferential strands in the middle.

The ‘classical’ heart with hypertrophic cardiomyopathy presents with asymmetric thickening of the ventricular septum, with endocardial thickening representing mitral impact lesion in approximately a third of cases at autopsy. Septal thickening can exceed twice that of the parietal wall. Many hearts with hypertrophic cardiomyopathy, however, show symmetric hypertrophy presenting with an evenly thick-walled left ventricle and a reduced cavity (Figure 4). Cross-sectional slices often display a macroscopic whorl-like appearance reflecting a combination of myocyte disarray and fibrosis. Mid-ventricular cross sections of human heart specimens with hypertrophic cardiomyopathy were sampled in the study by Kuribayashi and Roberts. They reported extensive loss of the circular orientation of myocardial strands and deep endocardial clefts in the junctional regions of the parietal wall with the septum. The affected regions showed marked disarray and usually, but not always, accompanied by increased interstitial and focal fibrosis. There is an increase in the thickness of the longitudinal deep ‘layer’, an observation also reported in a study carried out using cardiac diffusion and strain magnetic resonance imaging. It is known that the connective tissue matrix supporting the myocardium is altered in disease. In the hypertrophied heart, there is an increase in the diameter of the perimysial tendons and density of the weave leading to a loss of intercellular contact and conductivity. In contrast, the cross connections are lost in dilated cardiomyopathy resulting in slippage and realignment of adjacent bundles of myocytes and wall thinning.

Figure 4 (A) Reduced left ventricular cavity in a heart with hypertrophic cardiomyopathy. (B) Histological section showing myocyte disarray (red) and fibrosis (green). Masson’s trichrome stain.
Conclusions
The normal left ventricular wall has characteristic muscle bundles and myoarchitecture. The myoarchitecture may be altered in ventricular hypertrophy by fibre disarray and/or increased proportion of longitudinally orientated myocardial strands. Myoarchitecture in combination with alterations in the connective tissue matrix provide the structural basis for abnormalities in myocardial function.

Conflict of interest: none declared.

Funding
The Cardiac Morphology Unit at the Royal Brompton Hospital receives funding support from the Royal Brompton and Harefield Hospital Charitable Fund.

References