BASIC SCIENCE

Linking left ventricular function and mural architecture: what does the clinician need to know?

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The myocardium has a unique architecture, which converts the linear pull of a striated but involuntary muscle into a constrictive action. The left ventricle has also to balance the need to restrict the diameter of its chamber, thereby minimising mural tension, while providing at the same time a wall thick enough to achieve systemic pressure within the cavity. The precise architectural arrangement of the cardiomyocytes that fulfils these requirements is currently a topic of considerable debate. There is a divide between proponents of a counter-wound, single myocardial band, 1 and those who describe an arrangement of clefts around thinner lamellar units. 2, 3 In seeking to contribute to this debate, we present here a description of the changes that occur in surface geometry of the ventricle. We will show how the strain indexes of the wall, including mural thickening, are mathematically bound together by this geometry, irrespective of the internal architecture of the wall. We will then relate these indexes to demonstrable features of cardiomyocytic orientation and function. 4 In so doing, we provide a relatively simple explanation for left ventricular (LV) twist that does not rely on the presence of a unique myocardial band. We will also reinforce the observation of MacIver and Townsend that hypertrophy of the left ventricle can falsely normalise its ejection fraction (EF) despite falling contractility. 5 We conclude by addressing other significant aspects of mural architecture.

THE VOLUMES OF THE WALL OF THE LEFT VENTRICLE AND ITS CAVITY

Initially, we will regard the LV myocardium as a structure of fixed mass and so, at physiological pressures, of fixed volume. It envelops the cavity, and its surfaces are illustrated in figure 1. The magnitudes of changes in the inner and outer dimension of the wall, and of the distance between them, which is the mural thickness, are linked mathematically by the geometry of this very simple arrangement. If the cavity diminishes, the outer envelope must move inward by the same stroke volume. If it were to move by less, then the wall would have increased in mass, which is not possible. In the clinical setting, the stroke volume can be estimated from epicardial movement.

As the outer envelope diminishes, so the unchanging myocardial mass has to be repacked within a smaller envelope, and therefore must increase its radial dimension. In other words, it must thicken. It follows directly that the wall cannot show overall thickening as an isolated event, because it must be accompanied by net inward movement of its outer surface. Were the wall to show focal thickening or thinning in the isovolumic phases of the cardiac cycle, there must be a balancing reverse action elsewhere. These constraints operate in a ventricle of any size, and with any pathology.

The situation is slightly modified if the myocardium loses a little volume as it contracts. 6, 7 We consider this change to be due to the expression of blood from the mural vascular pool, though it is generally called ‘mural compression’. 8 It will increase the net inward movement of the epicardial surface in systole. Estimates of total myocardial blood volume rarely exceed 10 mL/100 G, 9, 10 so 10% compressibility would be a reasonable maximal possible value. We will take 5% compressibility as our nominal value. This may be conservative, but if its effects are clear at this level then it is likely to have an increasingly significant effect during exercise.

Net inward movement of the outer surface is manifest by a combination of circumferential constriction and longitudinal shortening. The relative contributions of these parameters may differ in individual hearts, and also in the various pathological conditions that may alter the overall pattern of myocardial contraction.

A SAMPLE SLICE ACROSS THE SHORT AXIS OF THE LEFT VENTRICLE

We will generate representative values in order to illustrate the process described above. Rather than using a simplified model of the whole ventricle, we will take a sample of it. The lower panel of figure 1 shows the long axial projection of a tagged MRI. In it, we show a sample slice positioned at the clinical short axis. Note that the vertical boundaries of this slice retain a parallel configuration during systole. Given that the epicardial surfaces as shown in the upper panel are reasonably circular, 10 the dimensions of the slice, both in systole and diastole, can be accurately represented by a short tube. An immediate concern is that the endocardial surface, in reality, is not the perfect circle we depict. And, as there is a common boundary, the wall is not of uniform thickness. Despite these caveats, our point is that the sample will establish the relationships between mural deformation as a whole and changes in the volume of the cavity. The circle that represents the boundary between wall and cavity will have the same diameter as the mean diameter of an irregular outline that encloses the same volume.
Similarly, the single value for mural thickness will accurately reflect mean mural thickness.

The mathematics we will use will now be based on the formula for the volume of a cylinder, when radius and length are the only variables. We are using lifelike dimensions. The radii will be the short axial measurements used in routine clinical imaging, and long axial shortening of the sample slice will approximate to clinical estimates of long axial strain.

**INITIAL RESULTS FROM THE SAMPLE**

We constructed a spreadsheet to convert the diastolic dimensions of the sample into its systolic configuration. The input dimensions were the diastolic epicardial and endocardial diameters and sample length. For convenience, the latter was given the value of 1 cm. From these values, we derived the volume of the diastolic cavity, along with the mural thickness and the volume of the wall. The functional input values were an epicardial circumferential strain of 10%, and a long axial strain of 14%. These values were averages from MRI scans in a cohort of normal volunteers held by one of us (SEP). In systole, the total volume of the wall plus that of the ventricular cavity was easily derived from the new length and epicardial diameter. The systolic mural volume was a known quantity, being the diastolic mural volume corrected for 5% systolic mural compressibility. Systolic cavity volume was then the total systolic volume minus the mural volume. Cavity radius followed, and so did systolic

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**Figure 1** MRIs of a normal left ventricle. In the upper panel, a ‘white blood’ sequence is shown in the two chamber (vertical long axial) and short axial views in diastole (upper panel) and systole (middle panel). The endocardial and outer (“epicardial”) margins have been outlined. The myocardium is shaded blue and the cavity pink. In the systolic images, the diastolic outer surface has been superimposed (arrows). If the myocardium is regarded as incompressible, then the green shaded volume must equal the stroke volume (see text). The lower panel shows the pattern of mural displacement as seen on tagged imaging in the four chamber plane. The evenness of long axial shortening is evident. The position of the sample slice is outlined. On the right, the slice is drawn to show that as its height (ie, short axial dimension) and length shorten, its wall thickens.
mural thickness, the endocardial circumferential shortening strain, and the ratio of mural thickening.

By replacing the endocardial diameter with the value for any particular depth within the wall, the same sequence generated an array of circumferential strains. We show such an analysis in figure 2, in which we used a nominal diastolic epicardial diameter of 55 mm. Two series were used, one with a diastolic mural thickness of 9 mm, and one of 12 mm. The wall was divided into four zones of equal diastolic thickness, and circumferential strain and mural thickening were calculated for each zone. The EF was the global figure, derived from the cubes of the short axial diameters.

For a mural thickness of 9 mm, the EF was 71%, total mural thickening 34%, and endocardial circumferential strain 25%. The calculations show the steady increase in circumferential shortening and zonal thickening from outer to inner parts of the walls. Indeed, this progression is rigid, and the circumferential strain at any depth can be used as an index value to calculate the strain at any other depth. We will return to this later. It can also be seen that the circle that lies at the middle of the wall in diastole is not in the middle in systole, as the inner half must thicken by a greater ratio than the outer.

Using the same outer size and external systolic change, increasing the diastolic mural thickness to 12 mm increased the EF to 94%, mural thickening to 56%, and inner shortening to 61%. This was despite a fall in slice stroke volume of 22%. This fall was entirely the consequence of 5% systolic mural compression. In the absence of compression, the stroke volume would have been identical.

The striking increase in circumferential strains reveals a weakness in the model. Keeping the epicardial value constant does not mean that underlying cardiomyocytic strain has been held constant, as all other circumferential strains are increased when diastolic mural thickness is increased. The index circumferential strain needs to be placed at some other depth, to provide more lifelike changes. Our slice sample can be improved to achieve this, requiring first a review of some aspects of mural architecture.

**EQUALISATION**

The cardiac sarcomeres throughout the wall show the same visible structure, albeit with some variation in expression of protein isoforms. They are all in the same inotropic environment, and during ventricular ejection depolarisation is universal and complete. They have a limited range of shortening, which is inherent in their striated architecture. When measured in isolation, mammalian cardiomyocytic shortening appears to be around 15%. We have taken this as our nominal value for the unstressed cardiomyocyte. Just how much it can increase with stress is not really known. A value of 20% has been observed in isolated murine cardiomyocytes, and 22% in cells from exercise trained rats. These observations, which were made on isolated cells, imply that there is a measure of inherent equalisation of cardiomyocytic shortening.

A strong argument has been made that the architecture of the myocardium should allow a significant degree of equalisation, or normalisation, of cardiomyocytic shortening throughout its mass, thus ensuring that all cardiomyocytes are in a position to respond equally to increases in preload and/or inotropic stimulus. The myocardial mesh does not require great precision of alignment to achieve this attribute. In this respect, Dorri and colleagues have argued that local modest departures from an ideal orientation, in health or in disease, result in only slight changes in overall strain values.

**THE HELICAL PROGRESSION**

Careful blunt dissection of the wall, as shown in figure 3, reveals a general orientation, or ‘grain’, of the aggregated individual cardiomyocytes. It is well established that there is a gradual and relatively even progression of helical angulation in this grain, from the well angled ‘left-handed’ outer myocytes through to a level in the middle part of the wall where they form a circumferential zone. Deep to this, the helical angle increases with depth, at the reverse ‘right-handed’ orientation to the outer zone. These observations fit well with slow changes of angle within a tightly knit mesh of branching cells. The definitions of the helical angle are shown in the left hand panels of figure 4. Transmural angulation is also defined, and this is discussed below. The depth and thickness of the circumferential zone is variable, but it does tend toward the middle of the wall. As the cardiomyocytes in this zone are aligned in circumferential fashion, the circumferential shortening ratio at this depth will approximate closely to equalised cardiomyocytic strain. This allows us to make the critical inference that the circumferential strain in the outer layers must be less than the value of equalised cardiomyocytic strain, and that the circumferential strain in the inner zone is greater.

Mid wall short axial circumferential strain is already a feature of measurements made by speckle tracking sonography and feature tracking MRI. This value is also calculable from routine cross-sectional imaging, as only the changes in epicardial short axial diameters, long axial strain, and the diastolic mural thickness are required.

Long axial shortening has a different pattern. In our chosen sample it has the same value across the wall, as the shape is a rectangle in both phases (figure 1). This will approximate to the shortening ratio of the subendocardial long axially oriented cardiomyocytes, which are at a right angle to the equator of the ventricular cone.

**RESULTS FROM THE SAMPLE USING A SINGLE VALUE FOR CARDIOMYOCYTIC STRAIN**

The considerations in the previous two sections led us to the position that the input values for myocardial strain in the sample could be replaced by a...
single value ascribed to both long axial strain and mid wall circumferential strain. This is an approximation, but arguably close enough to allow the values generated to reflect the quality of the changes in question.

Using this single value, and making a nominated stroke volume a target value for each curve, our spreadsheet was reorganised to yield patterns of greater physiological relevance. In figure 5, the horizontal axis is a linear progression of cardiomyocytic strain. If the rate of cardiomyocytic shortening is assumed to be linear with time, which may not be precisely the case, then the axis could also be regarded as a time scale. The graphs show that, as systole progresses, there is an accelerating increase in all the ratios of dimensional change, including mural thickening, which is most evident in the inner zones. The EF, however, shows a more linear increase. The only dampening factor in this process is mural compressibility, which at our nominal 5% causes a visible decrease in all ratios.

Mural hypertrophy augments these changes, with the notable exception of epicardial circumferential shortening, which decreases. Though the examples are derived from one particular set of values, by varying them we were able to show that the degrees of enhancement of inner mural thickening, endocardial circumferential strain, and EF are functions of the diastolic ratio of mural thickness to the internal diameter of the ventricular cavity (figure 6). This analysis can, therefore, be applied to ventricles of all sizes. The thicker the wall, the more the dimensional changes are enhanced.

These graphs strongly support the contentions of MacIver and Townsend, published in this journal with editorial support from Manisty and Francis. They described how mural hypertrophy can inflate the EF to be in the normal range, despite an underlying decrease in cardiomyocytic strain. Elements of this argument are also to be found in the works of Ingels, Dumesnil and Shoucri, Spotnitz et al, and Arts et al.

DO THESE OBSERVATIONS DEPEND ON MURAL ARCHITECTURE?

We have described the geometric linkage between the six indexes of dimensional change, namely epicardial and endocardial circumferential strain, mid wall circumferential strain, long axial strain, mural thickening, and EF. We have argued that if any two of them are known then the values of the other four are set. Any effect that twist may have on dimensional change is immersed in them. This interdependence does not, of itself, indicate whether the underlying motive force is one which causes a decrease in the axial dimensions, or one which directly powers mural thickening. The important corollary is that any force that directly causes mural thinning will cause the ventricle to dilate. In these respects the left ventricle displays...
As the motive force in the myocardium is provided by the contractile elements of the cardiomyocyte, the magnitude of mural thickening is ordained by the shortening strains. In this respect, it must be a passive result, and not powered by an independent mechanism of its own. Thickening of the constituent cells of the myocardium may contribute to overall mural thickening, but it is neither the driver of it, nor the determinant of its magnitude.

The hydraulic effect applies equally well to the whole ventricle, when the geometric linkage is still certain, but the mathematics are less simple.

**TWIST**

We have established that circumferential strain in the wall superficial to the circumferential zone is less than the equalised value for cardiomyocytic strain. If the cardiomyocytes in this zone can still achieve an equalised value, then this action is spent on more than circumferential and longitudinal shortening alone. Indeed, since the myocardium is a compound mesh of soft tissue, comprising the cardiomyocytes and their adjoined connective tissue, which only pulls on itself, it is unrealistic to expect deformation only to occur in the orthogonal directions. The local orientation of the cardiomyocytes must have consequences.

Figure 7 illustrates the general character of the geometric interplay between the three zones of the LV myocardium. A conservative range of 15–16% is assumed for equalised cardiomyocytic strain. For each zone, the change in shape of a diastolic reference square is shown, first with no twist and then with twist. Chains of cardiomyocytes sit in a left-handed helical angle of 45° in the subepicardial zone and a right-handed angle of 45° in the deep inner plane. The value for long axial shortening is 15% throughout, and this value also sets short...
axial circumferential strain in the ‘circumferential’ layer. Diastolic mural thickness is in the normal range, and the value for subepicardial circumferential strain is our ‘standard’ of 10%.

The central upper panel of figure 7 shows that, without twist, the outer cardiomyocytes conform to the systolic shape at a strain of only 13%. If these cells have an innate tendency to achieve values in the range of 15–16%, and since the short axial dimensional changes are fixed by their geometric relationship to the circumferential layer, the result of further contraction will be to pull the shape into a parallelogram, as shown on the right. Radially tagged magnetic resonance studies have shown that twist is transmitted in the same direction to the rest of the wall.\(^{19,20}\)

In the middle rank, at the circumferential zone, twist is seen to have no effect on cardiomyocytic strain, but some shear movement is consequent to it.

The crux of the matter is to be found in the lower rank (figure 7). This series is positioned deep in the inner zone, not quite at the subendocardial layer, but specifically at a distance from the mid point that yields 25% circumferential strain. The cardiomyocytes in this plane cannot lie at a zero angle as their strain would then have to be an impossible 25%. In the middle image, their right-handed helical angle is shown to lessen this demand to 21%, but this leaves no reserve for increasing preload. On the right, when twist is added, their strain falls to 15.5%. The inner cardiomyocytes do not produce a reverse twisting moment which would antagonise the outer zone.

This is a very simple illustration of the geometry, and does not include the effect of any intrusional angulation, which will further decrease the conformational strain, bringing both the inner and outer layers closer to 15%.\(^{18}\) A recent mathematical model study produced by some of us has confirmed that the main determinants for equalisation of cardiomyocytic strains are the helical distribution of cardiomyocyte orientation throughout the mural thickness and the associated twist of the myocardial...
The stroke volume was normalised.

Figure 6  The same changes as in figure 5 plotted against an increasing ratio of mural thickness to internal cavitory diameter and whose character therefore applies to a ventricle of any size. The stroke volume was normalised.

mass, but that transmural angulation is necessary to fully lower cardiomyocytic strain to physiological levels.4

The values we have used illustrate the geometry of the architecture and are not to be taken as a statement of the normal situation. The equalisation of inner cellular strain is the product of three attributes, namely helical angle, intrusional angle, and twist. These will have differing values from case to case, and from place to place. In addition, the position and thickness of the mid mural circumferential layer changes from place to place. The actual range of cardiomyocytic strain that represents achievable equalisation is not known. Indeed the range of cellular strain in life is not yet confidently known. For any particular combination in any particular individual, nonetheless, this arrangement is how a single muscle has oriented its contractile cells to ensure as far as possible that all cells make an equal contribution to overall contractility.

THE VECTORS OF CARDIOMYOCYTIC FORCE

The constricting force in the ventricular wall has been likened to surface tension,7 in that the pressure in the cavity is the result of tension in the plane tangential to the curvature of the wall. To contribute principally to tangential tension, a cardiomyocyte needs to be mainly aligned to the tangential plane. More recently it has been appreciated that many cardiomyocytes are not precisely aligned in the tangential plane, having an angle of intrusion, also called a transmural angle.17 18 This feature is illustrated in figure 4. This introduces a radial vector (AR), which, as noted earlier, is a dilating force. At first, it seems strange that a cardiomyocyte should produce a vector that is antagonistic to its major task. It is only at 45° intrusional angulation, however, that the net effect of the cell would change from constriction to dilatation. At shallow intrusional angles, the dilating vector is small, and its concomitant diminution of the constricting vector in the tangential plane (AT) is slight. It is typical of a muscular hydrostat that the antagonistic force is at a right angle to the agonist.13 This is in contrast to skeletal antagonism, when the muscles are linearly opposed.

Lunkenheimer et al2 19 consider that the antagonistic vector causes a force that does not decay during systole—that is, it is auxotonic in nature. This is in contrast to the tangential constrictive force, which mirrors the fall in mural tension as the cavity shrinks. The functionality of auxotonic forces, and the mechanics of those cardiomyocytes that exceed 45° intrusion, are the subjects of ongoing debate.24 20 The auxotonic forces, despite causing modest antagonism, may well have a synergistic action that assists systolic deformation.

SYSTOLIC RESHAPING OF THE WALL

For tangential tension to be translated into emptying of the ventricular cavity, the ventricular wall has to repack itself into its systolic configuration, as indicated by considerable mural thickening. To do so, it must display sufficient plasticity. If the wall were to be a low viscosity fluid, then there would be no conceptual difficulty. In systole, however, it is a tensile three dimensional mesh, which will resist reformation. The cells of the wall, mainly the cardiomyocytes themselves, can thicken radially. Indeed, any three dimensional object with a regular shape that achieves shortening of 15% must show net thickening of 8.5% in the opposite axis. As sarcoplasm has fluid properties, the cell could thicken more in the radial direction than in the circumferential, and the result may well satisfy all of mural thickening where it is least, which is in the subepicardial region. For most of the wall this is insufficient, and a rearrangement of the cells must be the second mechanism. It is becoming accepted that there are clefts in the myocardium, devoid of branching connections and collagenous matrix, which aggregate the cardiomyocytes into lamellar units—albeit that the units themselves are of varying dimensions and have varying alignments within the walls. It is the sliding between the lamellar units that allows for local freedom of relative movement.9 This can lead to some aggregates steepening in a transmural direction.20 In contrast to this ‘mesh and cleft’ concept, the ‘unique myocardial band’ offers differing movement between the loops of the band as an explanation. In our opinion, however, there is a complete lack of anatomic evidence for the existence of the purported unique band.20

DISCUSSION

Limitations

Our aim has been to provide an outline of geometric mural function, and to relate it to well documented aspects of anatomy and physiology. Our
A description compares end-diastole to end-systole, and does not comment on transient movements during depolarisation or repolarisation, nor on the velocities of change, which are all clinically useful. We are able, nonetheless, to describe some general constraints on mural movement to which any suggested architecture must conform. These are listed in the key points box, and answer the question in the title.

If our argument that mural thickening is a passive phenomenon is acceptable, then the constrictive action of the wall can be regarded as a simple mechanism. On the other hand, the process whereby the individual cardiomyocytes become re-packed during systolic mural thickening remains far from fully understood. The functional relationship between the clefts between the lamellar units, and the role of the collagenous elements of the myocardium in packing together the cardiomyocytes, requires further investigation. The geometric necessity of repacking increases the significance of pathologic processes, such as fibrosis, which may inhibit it.

The endocardium

The endocardium is an important structure in clinical imaging, as it provides the boundary for measuring the volume of the ventricular cavity. Our

Figure 7 Representation of the way in which a combination of helical angulation and twist makes a major contribution to equalisation of cardiomyocytic shortening. The contribution by an intrusional angle is not included. The blue areas are the reference areas introduced in figure 3, and the dotted lines represent chains of cardiomyocytes. See text for full discussion.

Figure 8 The endocardium is easy to identify in the diastolic images to the left. In the upper series, several trabecular structures merge with the upper wall in systole, masking the true position of the endocardium. In the lower set, the papillary muscle merges with the endocardial surface.

geometric approach suggests that it is of less importance in other respects. It is certainly not an index of myocytic contractility. In hypertrophic myopathy, high values for endocardial shortening, and hence high EFs, belie severe contractile dysfunction (figure 5, lower right panel). ‘Shortening’ of the endocardial surface is more a matter of progressive trabecular crowding, and inter-trabecular obliteration, as shown in figure 8.

**Circumferential versus long axial strain**

The architecture of the wall throws a visual emphasis on to the circumferential shortening in the short axis. Krehl described the mid wall as the ‘powerhouse’ of systolic emptying. Relatively few cardiomyocytes are oriented along the long axis, and they are found on the inner border of the wall. There are enough of them that arterial insufficiency, which affects the inner wall earlier than the outer, is the typical cause of preferential long axial ‘dysfunction’. However, it should be pointed out that shortening of the wall occurs in all directions and as much effort is needed in the long axial direction as in the short. Any helical angle will endow the cardiomyocyte with a vector in the long axial direction.

**Twist, hypertrophy, and pathology**

In a hypertrophied heart without contractile dysfunction, the relative distance from the circumferential to the subepicardial zones will be increased, and epicardial strain lessened (figure 5). The result will be a greater degree of twist, as has been observed. Circumferential strain in the inner zone will be increased, but the increase in transmitted twist will help maintain equalisation.

In figure 7, twist has no constrictive action. It can be argued that the long axial side of the parallelogram should be the same length as the untwisted rectangle. This would result in twist making a contribution to long axial shortening. As twist is only around 8°, this contribution is slight, although it is made at greater mechanical efficiency than for the linear strain. We note that pericardiectomy diminishes twist without diminishing stroke volume, and it has been reported that a degree of untwisting can occur in isovolumic relaxation.

One of the superficial attractions of the concept of the unique myocardial band is that it provides an explanation of twist. As we have already emphasised, in our opinion there is no anatomic substance to this concept. Instead, we have argued that twist is more likely to be the natural consequence of outer helical geometry, no matter which model of architecture is espoused. If twist diminishes without any change of shape, then the outer cardiomyocytes are probably not achieving equalisation of shortening with the rest of the myocardium. As they lie in the region of greatest mural tension, they may be the first to display reduced contractility with the onset of disease. This would explain why diminished twist can be a sensitive indicator of early contractile dysfunction.

### Education in Heart

**Linking left ventricular function and mural architecture: key points**

The wall of the left ventricle is relatively thick when compared to the size of its cavity, and has finite volume. As a direct consequence, in a ventricle of any size:

- **Mural thickening, endocardial circumferential strain, and ejection fraction have values that are greater than cardiomyocytic shortening.**
  - The greater the ratio of mural thickness to diastolic cavity diameter, the more these indexes are inflated—this can mask underlying contractile dysfunction.
- **Systolic myocardial compressibility, should it occur, has a significant dampening effect on the dimensional indexes.**
- **There is no direct relationship between the magnitudes of mural thickening and myocytic thickening.**
- **The wall cannot thicken independently of other dimensional change.**
- **Short axial circumferential strain is at a minimum at the epicardial surface, and a maximum at the endocardium.**

Combining the previous point with the known progression of helical angulation of the cardiomyocytes, and presuming that cardiomyocytic strain is equalised across the wall, we argue that:

- **The cardiomyocytes of the outer wall have a local circumferential strain that is less than their own strain, the difference being the generator of twist in the direction of their helical angulation. There is no need to postulate a unique myocardial band in order to explain twist.**
- **Twist can thus be a sensitive indicator of contractile function in the outer zone, but does not make a great contribution to overall ventricular function.**
- **This configuration can also explain the increase in twist in uncomplicated left ventricular hypertrophy.**
- **In the inner wall, circumferential strain is greater than the cardiomyocytic strain. The cardiomyocytes of this zone lie at a reverse helical angle to that of the outer zone, but the direction of twist is the same as in the outer zone. This arrangement, together with varying degrees of transmural angulation, allows normalisation of their shortening ratio. The inner zone does not generate a significant opposing twisting force.**
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