Cardiac Mechanics Revisited
The Relationship of Cardiac Architecture to Ventricular Function

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Abstract—The keynote to understanding cardiac function is recognizing the underlying architecture responsible for the contractile mechanisms that produce the narrowing, shortening, lengthening, widening, and twisting disclosed by echocardiographic and magnetic resonance technology. Despite background knowledge of a spiral clockwise and counterclockwise arrangement of muscle fibers, issues about the exact architecture, interrelationships, and function of the different sets of muscle fibers remain to be resolved. This report (1) details observed patterns of cardiac dynamic directional and twisting motions via multiple imaging sources; (2) summarizes the deficiencies of correlations between ventricular function and known ventricular muscle architecture; (3) correlates known cardiac motions with the functional anatomy within the helical ventricular myocardial band; and (4) defines an innovative muscular systolic mechanism that challenges the previously described concept of “isovolumic relaxation.” This new knowledge may open new doors to treating heart failure due to diastolic dysfunction. (Circulation. 2008;118:2571-2587.)

Key Words: diastole □ heart failure □ muscles □ ventricles

Congestive heart failure, due usually to both left ventricular (LV) and right ventricular failure, is an increasing problem worldwide. In the United States, ≈5 million patients suffer from congestive heart failure, and each year ≈500 000 new patients develop the condition.1 Originally, this syndrome was believed to be due to failure of the LV to pump blood efficiently (ie, systolic ventricular failure). More recently, emphasis has been placed on diastolic ventricular failure, in which systolic function appears to be normal but diastolic ventricular function is impaired.2–4 Diastolic dysfunction, in fact, may be the cause of congestive heart failure in up to 50% of these patients.5,6 We have had treatment for systolic ventricular failure for many years, even if it is imperfect; at present, however, no agreement has been reached about the best ways of treating diastolic ventricular failure. To develop better treatments for congestive heart failure, both systolic and diastolic, we need first to understand the basic physiology of normal and abnormal ventricular contraction and relaxation.

The heart is a muscular pump that supplies blood to the body. This goal is achieved by electric excitation that produces sequential ventricular emptying and filling. Figure 1a demonstrates the physiological sequence of ventricular function: an isovolumic contraction phase to develop prejection tension, ejection, a postejection isovolumic phase, and then rapid and slow periods for filling. LV volume decreases rapidly early in systole and slowly thereafter, corresponding to the rapid early acceleration in the flow curve. The volume then increases rapidly in early filling and more slowly during late filling.

The information shown in Figure 1a is still correct, but it is only slightly more informative than the concept of William Harvey, who concluded, after dissecting cadaver hearts, that the heart squeezed by constriction to eject and dilated passively to fill. This accepted view of cardiac function has 3 main shortcomings.

1. It implicitly assumes that LV muscle is homogeneous, with all of its fibers contracting or relaxing simultaneously. This misconception stems from the ways in which we acquired information about LV function in the past. These were by angiography or echocardiography, which are 2-dimensional techniques, and, until recently, all we could see was a global change in shape of the cavity and the surrounding muscle.

2. The standard concepts of ventricular function made no attempt to consider the architecture of the LV with its complicated array of fiber angles and, in particular, the function of the spiral muscle bands that form a partial figure-8 loop at the apex.

3. The 2-dimensional techniques previously used prevented us from evaluating the twisting phenomena observed during emptying and filling of the beating heart (Movie I in the online-only Data Supplement).7 Ventricular twisting can be
explained by fibers that form a figure-8 clockwise and counterclockwise spiral muscle configuration. This muscle configuration was previously described by a spectrum of anatomists and is currently supported by tensor magnetic resonance imaging (MRI) but until recently has not been subjected to detailed anatomic–physiological correlations. Current interest in the role of the spiral fibers has led investigators to ignore the role of the dominant mass of circumferential fibers, and one of our objectives is to examine the interactions among these sets of fibers.

All of these deficits are beginning to be resolved by the introduction of newer imaging techniques such as high-resolution echocardiography to show regional strains, magnetic resonance myocardial tagging and speckle tracking by echocardiography to quantify torsion, and diffusion tensor MRI to photograph natural global motion and demonstrate movements that develop from the underlying complex LV architecture.

This report relates function to the underlying precisely described functional muscular anatomy that causes the ventricular directional motions of narrowing, shortening, lengthening, widening, and twisting (Figures 1b, 1c, and 2), thereby providing structural explanations for each of these contractile sequences.

Figure 1. a. Currently accepted time frames of systole and diastole, with measurements of intravascular pressure in the aorta, LV, left atrium (LA), and LV volume, together with their impact on the mitral and aortic valves. Aortic flow occurs between the 2 intervals that define ejection. The physiological phases of cardiac cycle that include isovolumic contraction, ejection, isovolumic “relaxation” (to be questioned in this report), rapid and slow filling, and atrial contraction are shown. b, Two-dimensional images of the LV in a longitudinal view that shows the normal sequence of narrowing, shortening, lengthening, and widening of the ventricular cavity during a normal cardiac cycle. Images were obtained by epicardial imaging in an open-chest porcine preparation. The phases of the cardiac cycle include end-diastolic state (bottom right), isometric phase (top left), ejection (top right), and isovolumic phase (bottom left). The broken-line markers are within the ventricular cavity and define the transverse (between the mid endocardial walls) and the longitudinal (from apical endocardium to a line across the mitral annulus) dimensions. Muscle thickness is shown by the dark area adjacent to these intracavity dimensional lines. The pale color is the cavity. The predominant changes exist with muscular thickening that narrows and widens the cavity rather than the external wall dimensional changes. Note progressive muscular thickening (evaluated by wider distance between epicardial and endocardial lines as myocardial mass narrows and shortens for ejection), together with maintained thickness as heart lengthens during the rapid filling phase before substantial widening. c, Twist of the heart: clockwise (below baseline) and counterclockwise (above baseline) motions of the base and apex, respectively, during the cardiac ejection and filling periods are represented in rotational degrees with the use of speckle tracking with marker placed at the LV endocardial surface (Echopac PC V 6, GE Healthcare, Milwaukee, Wis). The relationships between the initial uniform and then reciprocal twisting motions of the base and apex during the pre-ejection, ejection, and rapid and slow filling periods are explained in the text.
Basic Ventricular Function Updated

The observed functional patterns (Figure 2a) include an initial global counterclockwise twist (as seen looking toward the heart from the apex) and attendant narrowing or "cocking" in the isovolumic contraction phase before ejection,14,15 followed by continuing counterclockwise twisting of the cardiac apex (lower third of the heart) and clockwise twisting of the base (upper third of the heart as seen by MRI and speckle tracking echocardiography) as the ventricle longitudinally shortens during the ejection phase, and then a vigorous apical untwisting in the opposite direction as the ventricle lengths and slightly widens during an isovolumic phase interval when no blood enters or leaves the ventricular chamber.12,16–18 This apical untwisting motion is associated with a rapid ventricular pressure decay (tau),16 quantifiable rate of untwisting13 and continues into the rapid filling interval,7 when suction occurs together with a recordable intraventricular pressure gradient11,12 until finally a phase of relaxation occurs during diastole, when heart widening continues by slower filling, before the atrium contracts prior to the next organized beat.

Cardiac motion begins in the base to narrow and elongate the ventricular chamber before longitudinal shortening develops,19,20 a sequence that contradicts the presumed apex-to-base contractile pattern inferred from studies on the anatomic path of the Purkinje system.10 Under these conditions, ventricular cavity elongation occurs (ie, mitral valve billowing19), yet muscle contraction causes this effect. The observed differentiation between initial narrowing and later shortening opposes the concept of synchronous contraction21,22 with a uniform or concomitant strain field across the ventricular wall23,24 because inhomogeneous strain (deformation) develops during contractile wave propagation.25 Until now, evaluation of regional strain during global ventricular motion was limited by insufficient spatial and temporal resolution of imaging technology.

This report will describe mechanics with imaging tools that amplify dynamic function, portray areas where gaps exist in explanations from conventional causative concepts, survey models of structure and define their advantages and limitations, correlate a mechanical structure/function sequence that is linked to known elements of a double helical cardiac configuration that coexists with the circumferential and longitudinal fiber arrangement described in all cardiac anatomic models, relate this structure to movement based on multiple imaging modalities, and outline its implications.

Newer Aspects of Ventricular Function

Early studies on ventricular torsion with implanted markers23,26 concluded that torsion (angular difference in reciprocal twisting of apex and base) was required to equalize stresses across the ventricular wall. Newer diagnostic tools allow noninvasive monitoring of cardiac motion and thereby avoid invasive strategies that can distort cardiac structure and unintentionally vary underlying function.27 Two-dimensional strain imaging by the speckle tracking method defines longitudinal and radial strain fields on the basis of fiber direction independent of angle of insonation.28 High spatial and temporal resolution echocardiography (Movie II in the online only Data Supplement) displays global wall movements relative to changes in regional myocardial strain, where regional strain (or deformation) is defined by the Lagrangian formula: \[ \varepsilon = (L - L_0)/L_0 \], where \( \varepsilon \) is strain, \( L_0 \) is baseline...
length, and L is the instantaneous length at the time of measurement.

Consequently, positive strain occurs if the segment length exceeds its original length, and negative strain exists if the segment length is shorter than its original length. Strain occurs in radial (narrowing or compression and widening or expansion), longitudinal (shortening and lengthening), and circumferential (tangential) coordinates in the same manner as described by MRI analysis (Figure 2b). Rotation is angular cardiac motion around a vertical axis, and twisting or torsion is the difference between rotation of different segments (usually apex and base) as measured by 2-dimensional speckle tracking. Differences likely exist in findings of echocardiographic and MRI analysis based on the location and depth of the area evaluated. MRI ventricular analysis is transmural, and echocardiography evaluates transmural motion only if no architectural overlap is found between endocardial and epicardial segments that have counterclockwise motion. Conversely, when such overlap occurs, echocardiography predominantly identifies the resultant motion of the endocardial or epicardial segment within the chosen regions.

Function Versus Missing Gaps From Conventional Explanations

The traditional apex to base electric excitation–cardiac motion concept is contradicted by early basal motion before ventricular shortening. MRI studies support observations that initial counterclockwise rotation during isovolumic prejection cocking that narrows the ventricle makes the mitral valve billow out, rotates the left side of the cardiac base counterclockwise to the right, and produces strain to compress the ventricle and further reduce mitral annulus dimension before ejection. These observations are consistent with the findings of Roy and Adami in 1890 and more recently Armour and Randall, whereby the initial cardiac motion involved the base rather than apex. Recent multiple gated ventricular acquisitions and ultrasonic sonomicrometer crystal measurements confirm this motion, but the causative mechanical mechanism is uncertain.

Furthermore, the conical apex and wider base undergo transient bidirectional lengthening during the prejection phase (Figure 3a) despite sonomicrometer evidence of shortening of LV wall endocardial fibers. Similar narrowing and apical “thrusting” was reported by McDonald in 1970, and an apical counterclockwise motion was confirmed by placing an intracoronary wire around the apical tip. These observations show a lengthening motion despite a contracting subendocardial muscle and simultaneously underscore a discrepancy that exists between the MRI that displays transmural counterclockwise motion of the base and apex during the prejection isovolumic contraction phase versus speckle tracking rotational motions that show that the apex has a clockwise motion. Structural reasons for this disparity must be defined.

The second phase of ventricular shortening and narrowing occurs during the reciprocal twisting of the apex and base during ejection (Movies I and II in the online-only Data Supplement), and these observations validate Borelli’s concept that blood is wrung out from the heart, just like wringing out a wet towel. This ballet-like progression pattern of muscular torsion involves thickening, circumferential and longitudinal shortening, and shearing, but the spiral muscular components responsible actions for these actions are described incompletely. Such force generation and torsion make use of a 60-degree angulation of ventricular wall fibers oriented together with spatially disposed counterwoven helices that act as “opposing force couples”; this angulation is identical to the angle determined by for optimal ejection in mathematical modeling.

The third phase is the isovolumic interval that precedes rapid filling, whereby the apical clockwise twist accompanies the 2 movements of lengthening and basal widening (Figures 2b and 3b). Rapid deceleration of LV pressure wave follows this sequential deformation pattern (Figure 1a), associated with a time constant of LV pressure decay as apical untwisting occurs to allow subsequent suction of atrial contents into the ventricle during the fourth rapid filling phase that includes similar lengthening, widening, and twisting motions.

In the past, lengthening during the isovolumic and rapid filling phases was thought to reflect restoring forces from recoil of muscular potential energy stored during systole and from release of the compressed titin coil. However, sonomicrometer crystal measurements record continuing subepicardial muscle shortening of the LV free wall (despite cessation of shortening on both sides of the cardiac base and subendocardial region) as shown in Figure 4. Echocardiography documents ongoing strain during this muscular contraction (Figure I in the online-only Data Supplement), and postsystolic contraction exists during this interval. This observation implies that ongoing contraction via an active muscular mechanism contributes to this motion, and the responsible muscle must be identified. Moreover, slight widening of the base and apex occurs during the isovolumic interval (Figures 2b and 3b) to cause a stretch that cannot result from the contracting muscle responsible for rapid lengthening.

Structural Mechanisms Underlying Ventricular Function

The heart requires an architectural design that allows the contractile apparatus to empty and fill with optimal mechanical efficiency, determined by integration of vectors of force generated by sarcomeres that can only shorten by active contraction. Ventricular thickening increases ~50% for only ~13% myocyte shortening, so that myocyte deformation from strain relative to fiber orientation influences these findings. The extracellular collagen matrix of the myocardium is an important scaffold in maintaining muscle fiber alignment, ventricular shape, and size. It forms a spiral fibrillar structure of endomysial collagen to support a spatial distribution of myocytes and myofibers that ensheathes the adjacent 3-dimensional reciprocal spiral arrangement pattern of muscle structure.

Many anatomic dissections of the musculature of the conical heart ventricles over the past 500 years confirm that the clockwise oblique fibers of the surface epicardial layer and counterclockwise oblique subendocardial layer fibers...
meet at the apical vortex and that a transverse layer surrounds the LV base. Histological studies document this arrangement, but controversy exists about the architectural form creating this 3-dimensional configuration and about how this dual helical configuration influences function.

Robb and Robb in 1942 described an arrangement of 4 distinct muscle bundles of superficial and deep bulbospiral and sinospiral fibers that resembled those previously defined by Mall and MacCallum, and they believed that each fiber bundle attached to the cardiac fibrous skeleton (Figure 5A). The deep circular bulbospiral fibers were traditionally thought to cause the dominant constriction motion during ejection, a movement that differs from the predominant twisting during the shortening motion during ejection and rapid filling. They did not consider how a myocardial syncytium without an obvious beginning or end could coordinate sequential motion, and the reproducibility of these tracts is questioned.

Rushmer et al, in 1953, looked at the whole ventricular mass, conceptually distinguished 3 layers of fibers (Figure 5B), postulated that these fibers twisted into a vortex at the apex, and implied an intimate connection of individual muscle layers that they believed was impossible to demonstrate by dissection of a real heart. Moreover, the transverse constrictor muscle was considered responsible for ejection by circumferential compression. Rushmer et al implied that simultaneous contraction of the oblique inner and outer muscles canceled each other out, suggested that tension developed between them generated stored potential energy that putatively establishes diastolic recoil that is currently thought to restore form during the rapid filling phase, and did not consider how transverse constrictor muscle influenced the recoil process.

In 1979, Streeter modeled the LV, using the work of Krehl and Torrent-Guasp, and described fibers that run like geodesics (shortest path on a curved form) on toroidal...
surfaces, superimposed like doughnuts as in a set of superimposed Russian dolls or a set of nested warped pretzels (Figure 5C). Each doughnut conformed to a ventricular cavity, with a tunnel at the center of each donut corresponding to the cavity chamber. Echocardiographic and MRI strain pattern recordings of narrowing before sequential twisting differ from the synchronous contraction developing from this architectural backdrop. Streeter’s work established the helical angular orientations of myocytes around the ventricular equator recently confirmed by diffusion tensor MRI recordings. However, his ventricular histology sections from the LV base displayed a more transverse central fiber orientation, whereas apical sections displayed only oblique fiber orientation, and either the left- or right-handed helix can form the endocardial component. These anatomic studies did not define the physiological implications of this architecture on LV mechanics.

The underlying structural concept of Streeter was endorsed by Greenbaum et al in 1981, but they disagreed with the symmetrical organization proposed by Rushmer et al, invoking the principle that the dissection method might have disrupted muscle fascicles, making it difficult to define the origin of the fibrous cardiac skeleton. This anatomic observation may be correct, but the functional objective is to show how the principal vector forces within the 3-dimensional cardiac structure influence cohesive integrated sequential fiber contraction to cause the observed motions during each beat.

In 1957, Torrent-Guasp discovered a helical heart structure by simple hand dissection. First, he unraveled the heart to identify an underlying midventricular spiral fold that changes the transverse fibers to an oblique configuration and that allows the unfolded heart to become a simple flattened longitudinal ropelike model extending from the pulmonary artery to aorta (Figure 5D; Movie III in the online-only Data Supplement). Refolding the heart into its natural biological configuration allows definition of 2 loops, termed the transverse basal and oblique apical loops (Figure 6a and 6b). The basal loop is circumferential and wraps around both the LV and right ventricle but does not involve the septum, a finding supported by recent diffusion tensor MRI analysis, but this observation needs further investigation. The apical loop is composed of a descending and ascending segment that conforms to the right- and left-handed helical arrangement described by anatomists over many years. Torrent-Guasp’s dissection introduces a “principal or dominant pathway” without defining individual fiber tracts and gives a road map to its configuration. The resultant surrounding external basal loop buttress (or transverse shell embracing the LV and right ventricle) covers the internal oblique helical or conical apical loop comprising reciprocally oblique fibers termed the descending and ascending segments with a figure-8 configuration that form a vortex at the cardiac apex. These architectural findings mirror the anatomic suggestions in Rushmer’s cartoon (Figure 5b) and concur with Grant’s ropelike model, but the physiological impact is questioned, together with recurrent concerns about reproducibility and importance of tracts disrupted during manual dissection.

Torrent-Guasp’s exposure of the midventricular fold and uncovering of dominant muscular pathways introduces spa-
tial macroscopic correlations between his dissections and the histological models of Streeter et al. Although the data of Streeter have been interpreted as showing a continuum of fiber angles across the wall, his images of the LV base (Figure 7a) also suggest 3 groups of fibers: inner and outer fibers that tend to run obliquely to the equatorial plane and a middle set that runs more horizontally. This explanation is supported strongly by diffusion tensor MRI that shows the same 3 sets of fibers in the intact heart (Figure 7b). Moreover, Streeter’s lower images of the LV free wall near the apex demonstrate the reciprocal oblique fibers of the ascending and descending segments of the helical apical loop that are also evident with diffusion tensor MRI (Figure 7a through 7c).

Many differences of opinion have been expressed about ventricular architecture. The notion of a continuum of fiber angles is at odds with the notion of discrete bands, particularly because histological boundaries of discrete bands have not been demonstrated. On the other hand, diffusion tensor MRI as well as studies with polarized light demonstrates 3 layers of the LV free wall, compatible with layers that are either discontinuous or separated by a narrow zone in which fiber angles change markedly over very short distances. The syncytial nature of cardiac muscle also argues against discrete bands, except that we know that connective tissue septa exist throughout the wall and that during severe dilatation of the ventricle these septa allow bundles of myocytes to slip past each other. The fact remains that different sets of ventricular muscle fibers exist that do not contract synchronously and are likely to play specific roles in contraction and relaxation.

Structure/Function Correlation and Proposed Mechanical Sequence

The common features of a circumferential muscle mass with predominantly transverse fibers and the oblique helical fiber arrangement of the inner and outer wall are documented by all anatomic descriptions and must be integrated to define a functional model that can explain how the heart fills and empties. Previously, investigators had concentrated on one or the other set of fibers, with little regard for their interaction. The improved newer imaging methods add a unique way to focus on regional natural motions, and an attempt will be made to functionally explain these movements with the interactions among these oblique and circumferential fibers. Although the details of the planes of fiber structure causing these natural motions are uncertain, the macroscopic correlations between his dissections and the histological models of Streeter et al. Although the data of Streeter have been interpreted as showing a continuum of fiber angles across the wall, his images of the LV base (Figure 7a) also suggest 3 groups of fibers: inner and outer fibers that tend to run obliquely to the equatorial plane and a middle set that runs more horizontally. This explanation is supported strongly by diffusion tensor MRI that shows the same 3 sets of fibers in the intact heart (Figure 7b). Moreover, Streeter’s lower images of the LV free wall near the apex demonstrate the reciprocal oblique fibers of the ascending and descending segments of the helical apical loop that are also evident with diffusion tensor MRI (Figure 7a through 7c).

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scopic motions are clear, and the subsequent role of the anatomist is to explain the 3-dimensional microstructure that causes these movements. Images that display cardiac motion are best understood in context of the dominant force created as a result of interactions among the simultaneously contracting wrapped circumferential fibers and oblique helical fibers. For example, within this architecture, the right-handed helix can move one way, the left-handed helix can move the other way, and the outcome is in concert with which movement dominates, unless the circumferential wrap is more powerful.

Preejection Phase

Ultrasonic crystal data show that, at the initiation of systole, the predominantly transverse circumferential muscle shortens almost simultaneously with the right-handed helix or descending segment (Figure II in the online-only Data Supplement) to compress the central left- and right-handed helices like a stiff outer shell and to cause the temporary longitudinal lengthening of the apex and base during the preejection interval (Figure 3a and Figure III in the online-only Data Supplement). This circumferential muscle is consistent with the horizontal fiber orientation displayed in the histology analysis of Streeter et al.\(^5^6\) and diffusion tensor MRI analysis\(^5^9\) and its motion corroborate the constriction function described by Ingels et al.\(^3^9\). The action of this established circumferential muscle mass has not been considered in current echocardiographic descriptions of muscular causes for motion.\(^1^0,1^1\) Although the inner shell is stimulated at the earliest interval by the direct Purkinje-myocyte fiber connection,\(^6^4\) transmural stimulation of the right-handed helix is insufficient to cause longitudinal shortening at this time. Figure III in the online-only Data Supplement displays a brief shortening, followed immediately by apical stretching due to the narrowing during the presystolic isovolumic contraction phase that is caused by the dominant motion of the circumferential fibers. The simultaneous consequence is rotating the entire heart in a counterclockwise direction on MRI recordings viewed from the apex and thereby explaining the cocking motion observed during the isovolumic preejection interval (Figures 2a and 8c). This radial shortening due to circumferential fiber contraction occurs in humans\(^1^9\) to compress the as-yet not fully contracted inner helix to account for the bidirectional cavity lengthening during this isovolumic

Figure 6. a, Unscrolling of Torrent-Guasp’s myocardial band model, whereby his unwrapped heart (e) contains an oblique centerfold that separates the basal and apical loops. Note (1) the transverse basal loop fiber orientation (b through e), representing circumferential fibers, and (2) the right- and left-handed apical loop helix with predominantly oblique fibers and reciprocal spiral (c) representing the right- and left-handed helix configuration, which (3) twists at basal and apical loop junction. The myocardial band extends between the pulmonary artery (PA) and the aorta (Ao). Note (a) the intact heart and (b) detachment of the right ventricle free wall with circumferential transverse orientation of right basal segment (RS). A genu adjacent to the septum separates the right and left ventricles, with (c) the detached rotated apical loop showing the left basal loop segment (LS) surrounding the inner helix configuration containing oblique right- and left-handed helical or descending and ascending segments. Note (d) unraveling of the helix to show unfolding of the descending segment (DS) and (e) the complete transverse myocardial band, with the central myocardial muscle fold to separate the basal and apical loops. The left segment is the transverse circumferential or basal loop, containing left and right segments, and the right segment is the unwrapped right- and left-handed helices of the apical loop containing a descending and ascending segment (AS). b, Architectural arrangement of the fiber orientation of the detached circumferential fibers (basal loop) that has predominantly horizontal fibers compared with the conical apical loop that contains right- and left-handed oblique fibers in a helical design, with these segments superimposed (top image); when the segments are separated (below), the right-handed helix or descending segment (lower left) is connected to the myocardial fold, and its oblique fibers aim toward the apex, whereas the overlying left-handed helix or ascending segment (lower right) is longer, and its oblique fibers course toward the fiber connection with the aorta.
contraction phase. The elongation of the double helix caused by cocking is also evident by ventriculography during the presystolic interval,19 confirmed by echocardiography (Figure 3a), MRI analysis (Figure 2b), and velocity vectors of directional motion (Figure IV in the online-only Data Supplement). This preejection stretch of the helix may “load” the muscles to produce a Starling effect via the titin mechanism65 for subsequent ejection.

Figure 7. a, Streeter’s analysis of LV free wall with T-shaped full-thickness specimens before removal of the anterior papillary muscle (APM) and posterior papillary muscle (PPM). The leg of the T is between papillary muscles, and the top is the base and the leg extends toward the apex (A). Fiber angles from 4 separate sites on the T top (base) in diastole are plotted as a function of wall thickness (B). Zero percent of wall thickness implies the endocardial surface. Average data of 5 hearts in systole and diastole suggesting fiber angle direction through the ventricular wall (C). Zero percent of wall thickness implies endocardial surface. Note all fiber angles in systole are increased through the wall by an approximate constant angle relative to their counterpart in diastole. Reprinted with permission from the publisher from Streeter et al.66 Copyright © 1969, the American Heart Association. b, Diffusion tensor MRI from the work of Zhukov and Barr69 showing the helical inner or endocardial (clockwise) and outer or epicardial (counterclockwise) fiber orientation (in purple and blue colors) and a central LV free wall in top chamber that is white to reflect a more horizontal or very small angle pitch that does not involve the septum. c, Diffusion tensor MRI from studies by Rohmer, Sitek, and Gullberg63 showing helical configuration of the right-handed helix endocardial (green) fibers obliquely coursing in a clockwise orientation toward the apex and left-handed helix or epicardial (blue) fibers displaying a counterclockwise orientation. Figure 7b and 7c reprinted with permission of the publisher from Rohmer et al.63 Copyright © 2007, Lippincott Williams & Wilkins.
Analysis of timing of the electric impulse shows that early activation of the right and left parts of the base and septum of the heart occurs through the moderator band (right ventricle) and from the left posterior fascicle (LV). Human endocardial mapping studies at this time interval show that the upper septum becomes activated 15 to 20 ms before the apical region. A clockwise motion is implied because multiple gated ventriculography analysis and ultrasonic crystals show that the sequence of initiation of contractile activation to produce strain and compression proceeds from right to left to constrict the base of the heart. However, the mechanical rotation sequence is counterclockwise, a finding that may reflect how the larger muscle mass of the left part of the circumferential muscle becomes dominant to govern global rotation.

The endocardial contraction does not exert sufficient force to cause the consistent longitudinal shortening movement that exists during ejection, as evident from radionuclide ventriculography recordings and speckle tracking imaging recordings showing evidence of that brief shortening that is subsequently overcome by lengthening during this interval (Figure IV in the online-only Data Supplement). The failure to shorten the chamber during the isometric phase implies that the counterclockwise rotation of the apex is due to circumferential muscle movement because the epicardial segment of the helix is not contracting during the pre-ejection period, as shown in Figure 4.

The apex is composed of the right- and left-handed helical fibers, and its motion is determined by how it is affected by both the helical fibers and their interconnection with the surrounding base of the heart. For example, the contrast between the MRI that displays counterclockwise motion of the transmural wall and the clockwise motion shown by speckle tracking that samples predominantly the endocardium may relate to the imaging evaluation tool because the superimposed epicardial segment is not contracting. Movie II in the online-only Data Supplement shows a transmural image and demonstrates how high-definition echocardiography can provide visual insight into counterdirectional forces of the right- and left-handed helical segments that are usually termed endocardium versus epicardium or descending versus ascending regions. The counterclockwise motion displayed by MRI recordings (Figures 2a and 2b) shows that the dominant rotational direction is governed by the more powerful circumferential muscle component. However, the capacity of speckle tracking imaging to recognize pre-ejection endocardial clockwise rotation introduces a mechanical reason for mitral valve closure before LV pressure generation because presystolic blood flow velocity is directed from the apex toward the base during early endocardial rotation to thereby establish a flow-related explanation. Conversely, initiation of left-handed helix contraction is delayed until ejection (Figure 4) when it is responsible for the ongoing counterclockwise twisting of the apex, as described in the next section.

**Ejection**

During ejection, the circumferential fibers continue to shorten together with the oblique fibers in the right- and left-handed helices (or descending and ascending apical segments) that co-contract to shorten and thicken to empty the heart. The circumferential fibers produce a horizontal counterforce that
accentuates narrowing throughout ejection. Furthermore, these contracting transverse circumferential fibers provide a “buttress” to counteract the outward forces generated by the twisting and shortening oblique muscle of the inner right- and left-handed helical segments and thereby prevent an outward explosion at the cardiac base during ejection. The twisting segments move in clockwise and counterclockwise directions, as shown by recent echocardiographic observations as well as by tangential vectors by MRI and visually in Movie II in the online-only Data Supplement, which simultaneously allows comparison to underlying architecture to convey structural reasons for directional motions in areas with and without overlap of helical fibers (Figure 9a).

Strain is greatest in the right-handed helix (or subendocardium), accounting for its capacity to cause shortening and clockwise twisting of the cardiac basal region, but the simultaneous counterclockwise apical twist is due to the torque of the co-contracting left-handed helix (or epicardium) with a larger radius of curvature; their interaction is responsible for systolic torsion. The subendocardial muscle is formed by both the right- and left-handed helices (or descending or ascending segments) (Figure 9b), depending on their sampling site within the helical architecture. This structural arrangement explains why MRI (Figure 2a and 2b) and speckle tracking imaging display clockwise and counterclockwise shortening during ejection. Conversely, speckle tracking imaging displays transmural motion in regions without helical fiber overlap in areas of lateral and inferior walls and septum just below the aortic valve. As a result, no counterforces act on the endocardium of the left-handed helix (or ascending segment) in these areas (Figure 9b).

Sonomicrometer crystal tracings demonstrate sequential shortening of the descending, posterior, and ascending segments (Figures V and VI in the online-only Data Supplement) of the Torrent-Guasp model. However, a network of connected fibers exists within the deeper intermingled transverse and oblique fibers, so that transverse fiber interconnections may induce shearing by creating transmural torque, whereas oblique fibers exert directional shortening. The global rotational and directional forces are displayed by MRI in Figure 2a and 2b, which conveys how these co-contracting clockwise and counterclockwise forces create the radial velocities that define systolic compression of the LV. As the right-handed helix (or descending segment) contraction dominates
to shorten the ventricular chamber, it simultaneously pulls down the co-contracting left-handed helix (or ascending segment), whose fibers now become more horizontally oriented. Consequently, such helical co-contraction joins the circumferential contraction to accentuate chamber narrowing during the ejection phase to aid ventricular propulsion of blood.

Deformation is greatest toward the apex to achieve maximal reduction in chamber volume and may be explained by the fact that the right- and left-handed helical segments converge toward the apex to form the vortex of the double helical loop. The oblique right-handed helix initiates shortening and is joined 80 ms later by co-contraction of the left-handed helix that comprises the ascending segment of the apical loop (Figure 4, Figures VI and VII in the online-only Data Supplement). This shortening also thickens and twists the LV apex and exerts a torque in an opposite (counterclockwise) direction, as shown in Movie II in the online-only Data Supplement, by MRI in Figure 2a, and by velocity vector directional motion (Figure VIIIa in the online-only Data Supplement). This regional left-handed helical directional motion is evident by velocity vector analysis in which left-handed helical components in the lateral wall and upper septum have no helical overlap (Figure 10). However, the left-handed helical mechanical mechanism underlying counterclockwise apical motion during ejection differs from the pre-ejection phase apical rotation, which represents dominance of the circumferential fibers when contraction of the left-handed helix (or ascending segment) is absent.

Each spiral arm of the helix globally twists itself in opposite directions; the right-handed helix is dominant, directed downward, and its twist causes the observed clockwise rotation of the cardiac base. Conversely, the left-handed helix twists counterclockwise to produce apical reciprocal rotation. Although its longitudinal motion is directed upward, it moves downward during ejection because it is dominated by the right-handed helix, as shown by velocity vector analysis (Figure VIIb in the online-only Data Supplement). Evidence for the simultaneous elevation and straightening of the left-handed helix (ascending segment) muscle is delayed until the isovolumic interval, when its ongoing contraction now exists without right-handed helix (descending segment) contraction, resulting in upwardly directed velocity vector, as discussed in the next section (Figure VIIIc in the online-only Data Supplement).

These observations contradict prior suggestions that the epicardial muscle exerts the downward force during ejection, as well as recent suggestions that the epicardium (left-handed helix) twists within itself, so that its apex goes in one direction (counterclockwise) and its base in the opposite direction (clockwise), together with the suggestion that the endocardium (right-handed helix) has the reverse action. A limitation of this analysis is the absence of recognition that the endocardium can be formed by either the left- or right-handed helix, depending on their overlap. The left- and right-handed helices connect at the apical vortex that becomes the turning point for their reciprocal motion. Thereby, the helical arrangement of fibers shows that the entire right-handed helix component twists clockwise to shorten the chamber. Such early clockwise motion was evident by speckle tracking imaging during the preejection interval (Figure 1c), when transient shortening occurs (Figure III in the online-only Data Supplement), and from the continued clockwise motion of the cardiac base during ejection, as evident by MRI tangential motion (Figure 2a and 2b) and speckle tracking imaging. Furthermore, velocity vector imaging shows clockwise rotation of the endocardial segment (forming the LV septum endocardium) at all levels (apex, mid wall, and base) during ejection (Figure IX in the online-only Data Supplement).

The entire left-handed helix (epicardium) twists counterclockwise; this motion prevails at the apex and appears as a leftward direction of upper septum motion by velocity vector analysis (Figure 10) that correlates movement with the anatomic wraparound configuration. Simultaneously, the left-handed helix thickens during co-contraction to compress the cavity, but its effort to elevate the ventricle is offset by the prevailing dominance of the right-handed helix (descending segment) contraction, as demonstrated by downward velocity vectors during ejection (Figure 10, Figure VIIb in the online-only Data Supplement). The contraction-related elevation of the left-handed helix only becomes apparent during the isovolumic phase, when right-handed helix contraction stops (Figures 2b and 3b; Figure VIIIc in the online-only Data Supplement).
Supplement). From a mechanical standpoint, the shortening motion during ejection reflects the dominant force of the right-handed helix rather than the constrictive motion of the circumferential fibers that predominantly causes compression.

Isovolumic Phase

After ejection, the right-handed helix (or descending segment) stops contracting but maintains stiffness and tension\(^{17}\) and thus may act as a fulcrum for left-handed helix (ascending segment) straightening because this segment continues to contract in an unopposed fashion for an additional \(\approx 90\) ms (Figure 6).

When a helix is compressed, its 2 ends approach each other, and the internal coils (or springs) become more horizontal, and when it is stretched, the 2 ends move apart, the coils become more vertical, and the helix becomes straighter. These changes represent rearrangement of the coils as a whole and can be separated from changes of length of individual segments (myocytes) of the coil. For example, in the postsystolic isovolumic period, the left-handed helix (or ascending segment), which was compressed and more horizontal, continues to contract and straighten because it is no longer opposed by contraction of the right-handed helical spiral.

The contraction of the left-handed helix is the only force for the abrupt change to an upward velocity vector direction (Figure VIIc in the online-only Data Supplement) during this phase. Cavity widening also occurs without blood inflow (Figures 2b and 3b; Figure VIIc in the online-only Data Supplement) and is likely related to recoil of circumferential muscle that has stopped contracting. The discrepancy between a fixed volume and increases in width and length is probably due to the use of a 2- rather than 3-dimensional measurement. The oblique right- and left-handed helices demonstrate a spatial and temporal relationship during the initiation and completion of their shortening that interacts with the predominantly transverse circumferential muscle.

The predominant motion is rapid clockwise untwisting of the apex and mid wall, together with accentuation of the clockwise motion of the base (Figure 2b). These movements occur before the end of systole and exist only while the left-handed helix is still contracting (Figures 2b and 4; Figures I and VI in the online-only Data Supplement).

Moreover, the widening of the cardiac base shown by echocardiography in Figure 3b, MRI (Figure 2b), sonomicrometer crystals (Figure II in the online-only Data Supplement), and velocity vector analysis (Figure VIIc in the online-only Data Supplement) is associated with a lengthening of the basal area (Figure 2b).

The untwisting motion during elongation creates a negative pressure and potential vacuum\(^{70}\) that continues into the phase of rapid cavity filling after the decelerating ventricular pressure falls below atrial pressure. Its origin is likely from titin-related recoil\(^{43}\) of the noncontracting circumferential fibers that exerted a counterclockwise motion during prejection. The right-handed helix straightens as the left-handed helix maintains strain, continues to shorten (Figures 4 and 11; Figures I and VI in the online-only Data Supplement), and elevates, so that the left-handed helix (ascending segment) cannot be the cause of untwisting. This observation contradicts a recent report’s comment that untwisting is due to the epicardium because this reversal of twisting is not possible in a region that is still contracting.\(^{12}\)

Moreover, the ongoing...
left-handed helix shortening and strain now provide a muscular explanation for the origin of postsystolic contraction.45

Most likely, the clockwise rotation of the apex is governed by recoil of the circumferential fibers (or basal loop), which also stop contracting, and is the opposite movement from its preejection counterclockwise motion (Figure II in the online-only Data Supplement). Although the apex continues its counterclockwise motion because of the left-handed helix, its radial velocity forces are markedly diminished (Figure 2b) so that the observed net clockwise motion reflects untwisting of the apex from recoil of the circumferential fibers (or basal loop). Additionally, this decreased force of contraction is a potential reason for the later lengthening during the isovolumic interval recently reported by Notomi et al.12,16 An added mechanical factor is that the prominent untwisting shown in Figure 2b may also imply an unwinding of the taut right-handed helix (or descending segment), which now returns to its original helical starting point (Figure 6b, lower left).

Conversely, an opposite action exists for the circumferential muscle in preparation for ventricular filling, as untwisting and widening develop a potential intraventricular vacuum for subsequent suction during unopposed straightening of the left-handed helix or ascending segment. MRI studies show that the cardiac base widens and the chamber cavity lengthens (Figures 2b, 3b, and 11) and remains thick,71 so that the counterforce of the stiff outer rim circumferential muscle prevents potential implosion of the base during lengthening. The circumferential muscle thereby exerts the balancing action of preventing explosion during ejection and avoiding implosion before and during rapid filling and has the predominant action of governing narrowing and widening motions. In contrast, the oblique fiber orientation of the right- and left-handed helices governs the shortening and lengthening motions.

Observations that document the muscular role of the left-handed helix during this interval include sonomicrometer crystal evidence of ongoing contraction (Figure 4), continuing strain (Figure 11, Figure I in the online-only Data Supplement), MRI evidence of radial velocity showing contraction (Figure 2b) and thickening,71 and an abrupt change in the velocity vector toward elongation as the isovolumic phase begins (Figure VIII in the online-only Data Supplement). The elevation component from ongoing contraction of the left-handed helix mirrors the spatial alteration that exists when a cobra elevates as its muscles continue to contract in the interval before striking. For the heart, this cardiac chamber elevation motion is due to the now unopposed contracting muscular force of the left-handed helix that becomes more vertical and is not “recoil from stored potential energy” because shortening (negative strain) and thickening exist during this phase of cardiac motion. Moreover, this active muscular effort is the reason that the ventricle returns to the previous “neutral” longitudinal position during the isovolumic phase, a motion that contradicts the conventional concept of recoil, which occurs at the ventricular base for widening and untwisting. The presence of active contraction during the period previously considered to be “isovolumic relaxation” suggests that this term is ambiguous and should be avoided unless qualified by accurate muscular descriptions.

The physiological untwisting action is caused by a geometric dynamic change in shape and has been characterized by a series of hemodynamic changes that include measuring the rate of untwisting,13 as well as tau (change in time related to change in deceleration in LV pressure),72,73 and also by measuring the intraventricular pressure gradient that is maximal just after the mitral valve opens,12 which is considered in the next section.

Rapid Filling and Suction

The suction phase for rapid filling occurs after the ventricular pressure falls below atrial pressure and is associated with a further rapid accentuation of untwisting of the apex in a clockwise direction (Figure 2b); Dong et al13 showed that 40% of untwisting occurred before the rapid filling phase. Continued untwisting is caused by elastic recoil of compressed titin coils within the left-handed helix fibers, in a manner similar to circumferential muscle (basal loop) widening and recoil for clockwise rotation during the isovolumic phase. Although the recoil mechanism is responsible for rapid filling, the vital role of active and unopposed left-handed helix contraction to cause straightening during the isovolumic phase is an important component because suction is impeded or avoided if this motion is opposed by prolonged descending contraction, as discussed in the next section.74,75 As the contractile phase dissipates and all the muscle segments become relaxed, the rapid titin-related “unwinding” of the apex to its original position creates the suction required to cause rapid ventricular filling, which occurs as the ventricular pressure drops below the atrial pressure. Further widening and lengthening develop from the hydraulic effects resulting from rapid and then passive filling after the apical reciprocal twisting action has stopped.

The contributions of different segments of the helical ventricular myocardial band toward these 4 phases of physiological motion response are identified by placing sonomicrometer crystals into the right and left segments of the basal loop, as well as into the descending and ascending segments of the apical loop of the LV free wall.64 During the pre-ejection isovolumic contraction phase, shortening occurs in 3 regions of the right and left basal segments and the LV endocardium. With ejection, co-contraction exists in the descending and ascending segments, together with ongoing shortening of the both basal loop segments, so that all 4 areas are shortening. Conversely, during the isovolumic intervals, active shortening occurs in only the 1 ascending segment because contraction has stopped in both segments of the basal loop and in the descending segment. Recoil after completion of contraction in the ascending segment produces the apex clockwise motion responsible for early rapid filling, an action that requires the isovolumic phase to display a temporal hiatus between descending and ascending apical loop segment contraction.

Implications

Excitation/contraction events during the pre-ejection sequence may have clinical utility to understand cardiac resynchronization therapy. During pre-ejection, the circumferential basal loop rather than the left- and right-handed helical apical loop
observed after transient ischemia to produce diastolic dys-
function and therefore remedy the diastolic dysfunctional mechanism for cardiac dynamics during the phase of rapid lead to selection of new drugs that can modify a contractile process that simply disappears or becomes recoil and passive dilatation as the cause of changing ven-
tricular volume. This architectural alteration changes the geometric position of the posterior papillary muscle to limit its tethering of the mitral leaflet by the bulging septum and thus improves leaflet coaptation to reduce mitral regurgitation, but without restoring the twisting motion of the septum.

The interaction of timing of contraction of left- and right-handed helices (or ascending and descending segments of the apical loop) governs the efficiency of apical untwisting that determines suction, thereby introducing a paradigm shift in conventional thinking. The traditional concept of elastic recoil and passive dilatation as the cause of changing ventricular volume before filling becomes changed to include an active contractile process that simply disappears or becomes limited when descending segment contraction is prolonged. The impact of prolonged systolic forces during ejection was observed by Stuber et al., employing tagging MRI studies in patients with aortic stenosis (Figure 12) and also in patients with dilated cardiomyopathy. Systolic contraction extended into the isovolumic phase (previously termed early diastole), and a similar pattern was observed after transient ischemia to produce diastolic dysfunction (Figures Xa, Xb, Xc, and XI in the online-only Data Supplement). Sonomicrometer crystal studies demonstrated that extended descending segment contraction limits the normal hiatus (~80 ms interval) between cessation of descending and ascending segment shortening to generate an abnormal pattern that is remedied by sodium-hydrogen exchange inhibitors. Recognition of this active process may lead to selection of new drugs that can modify a contractile mechanism for cardiac dynamics during the phase of rapid filling and therefore remedy the diastolic dysfunctional component of congestive heart failure that affects ~50% of patients.

Conclusions

Keith, in 1918, presented a currently unfulfilled challenge by stating, “We cannot claim to have mastered the mechanism of the human heart until we have a fundamental explanation of its architecture.” We conclude that comparison of functional images against several structural models showed that the helical ventricular muscular band model of Torrent-Guasp provides a functional anatomic model that explains the observed directional and twisting sequential motions. Further testing of these spatial anatomic concepts is needed because the architectural coordination of structure and function, if properly confirmed, may adhere to Keith’s challenge and allow accurate understanding of the mechanisms of cardiac dynamics.

Disclosures

Dr Buckberg consults with Helical Heart Company LLC (www.helicalheart.com), which makes a spatial heart model of helical ventricular myocardial band anatomic configuration. The authors report no conflicts.

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