Torsion of the left ventricle during the ejection phase in the intact dog

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SUMMARY Torsion of the left ventricle (LV) is associated with rotation of the apex with respect to the base around the long axis of the LV. A mathematical model of LV mechanics, which relates torsion to transmural distribution of fibre shortening, was evaluated with two-dimensional echocardiography in nine anaesthetised closed-chest dogs. Torsion was calculated as the difference between the angles of rotation (radians) of echo-derived transverse cross-section projections of the LV obtained at the mitral valve and low papillary level, divided by the axial distance between these projections measured in a long-axis cross-section, and multiplied by the outer radius in a mid-papillary transverse projection of the LV. A shortening to torsion ratio (STR) was defined as the ratio of inner wall shortening to torsion occurring during ejection. In a series of 11 measurements, each based on frame-to-frame analysis of 15 cardiac cycles, STR was found to be $2.31 \pm 0.23$ rad$^{-1}$ (mean $\pm$SD), whereas the mathematical model predicted a STR value of $2.4$ rad$^{-1}$ over a wide range of preload, afterload and contractility levels. We conclude that two-dimensional echocardiography validates the presence of torsion in the normal canine left ventricle, as predicted by the model of left ventricular mechanics.

1 Introduction

Torsion of the left ventricle is a rotation of the apex with respect to the base around the long axis of the left ventricle. Torsion has been noticed directly in the intact heart and more indirectly by measuring shear deformation of the epicardial surface of the left ventricular free wall. In designing mathematical models of left ventricular wall mechanics torsion is often not taken into account. Such models focus either on accurate representation of left ventricular geometry, on matching observed fibre orientation, on transmural differences in mechanical loading, or on the contractile behaviour of the cardiac muscle. The mathematical model of left ventricular mechanics which we developed takes into account fibre orientation in the wall of the left ventricle, allows for anisotropy of the myocardial tissue and considers the physiological contractile behaviour of cardiac muscle. In particular the possibility of left ventricle torsion was incorporated into the model. Because of this emphasis and of the necessary simplifications in the model less attention is paid to detailed geometry; the left ventricle being assumed to be a thick-walled cylinder. The most important prediction of the model was a nearly uniform distribution across the left ventricular wall of muscle fibre stress, fibre shortening and contractile work during the ejection phase. In the present study, global deformation of the left ventricle during the ejection phase is assessed experimentally in the closed-chest anaesthetised dog, using two-dimensional ultrasonic echocardiography. The experiments were performed to evaluate the pattern of deformation of the left ventricle during the ejection phase, as predicted by our model of cardiac wall mechanics. For this evaluation we sought a dimensionless parameter associated with changes in left ventricular geometry during the ejection phase. Such a parameter should be essentially independent of the biological variance in left ventricular size, myocardial contractility and haemodynamic loading, which are difficult to control in most experimental settings. With the help of the model, these requirements were found to be met by the ratio of shortening of the left ventricular inner circumference to left ventricular torsion, both deformation parameters being measured during the ejection phase.
2 Mathematical model of left ventricular mechanics

Torsion and circumferential shortening of the left ventricle during the ejection phase were calculated using a mathematical model of left ventricular mechanics. The model was programmed in Fortran IV on a DEC computer (PDP 11/34). The principles of the model are briefly recapitulated below.

The left ventricle is assumed to be a thick-walled cylinder composed of eight concentric cylindrical shells. The myocardial tissue is modelled as an anisotropic structure consisting of contractile fibres embedded in a soft incompressible material. Different muscle fibre orientations for individual shells were chosen on the basis of previous studies. Shear deformation between layers in parallel with the wall is assumed to be zero everywhere in the cylinder. Both ends of the cylinder are closed by a plate which can rotate around the axis of the cylinder and allows radial displacement of the cylindrical wall. No external forces or torques act on the plates, which is also the case with the apex of the real left ventricle.

In such a plate the equilibrium of torques due to shear stresses in the wall accounts for one equation. The equilibrium between axial forces due to wall stress and due to left ventricular pressure accounts for the other equation. Solving both equilibrium equations renders torsion angle and the ratio of cylinder axis length to cylinder diameter. The mathematical description of contractile properties of myocardial tissue is based on in vitro measurements of mechanical characteristics of isolated cardiac muscle. The afterload of the left ventricle is simulated in the mathematical model by a diode (aortic valve) placed in series with an inerance, a resistance (aortic characteristic impedance) and an arterial capacitance in parallel with a second (peripheral) resistance.

In the simulations the following were calculated: 1) the time course of left ventricular pressure and volume as well as aortic pressure and volume flow; 2) the time course of inner and outer diameters, length and torsion of the cylinder; and 3) in each shell the time course of sarcomere length and stress, intramyocardial pressure and stroke work per unit of tissue volume. The torsion parameter ($\gamma$) was calculated as the gradient along the axis of the cylinder of the angle of rotation of the cylinder transverse cross-section, multiplied by the external radius of the cylinder (fig 1). Physically this torsion parameter is equivalent to the shear angle at the outer surface of the cylinder. At the reference geometric state $\gamma$ is supposed to be equal to zero.

Natural strain ($\varepsilon_{ci}$) of the inner circumference of the cylinder is calculated from the inner diameter of the cylinder ($d_i$) and its value ($d_{i0}$) at the reference state by:

$$
\varepsilon_{ci} = \ln \left( \frac{d_i}{d_{i0}} \right)
$$

The reference state is quite arbitrarily chosen to be the end-diastolic phase in the control state and is the same for all cardiac beats analysed. Choosing another reference state causes a shift of the deformation parameters $\varepsilon_{ci}$ and $\gamma$, but does not affect the magnitude of changes in these parameters during the ejection phase. Therefore, in the presentation of the results zero values of $\varepsilon_{ci}$ and $\gamma$ are not indicated.

Simulations of cardiac cycles were performed at various assumed end-diastolic values of aortic pressure ($p_{ed}$) and aortic volume flow ($Q_{ao}$). At different levels of end-diastolic left ventricular volume $V_{ed}$ (30 to 100 ml), control 60 ml and end-diastolic aortic pressure $p_{ed}$ (7 to 20 kPa or 53 to 150 mmHg; control 11 kPa or 83 mmHg).

**FIG 1** The left ventricle is modelled as a thick-walled cylinder. Torsion is indicated as a possible mode, and is quantified as the shear angle $\gamma$ at the outer surface of the cylinder. $h$ and $d$ are length and diameter of the cylinder. $\alpha$ is the angle of rotation of the upper transverse cross-section with respect to the lower cross-section.

**FIG 2** Model simulation: time course of left ventricular pressure ($p_{lv}$) and aortic pressure ($p_{ao}$), aortic volume flow, inner circumferential natural strain ($\varepsilon_{ci}$) and torsion ($\gamma$) at different levels of end-diastolic left ventricular volume $V_{ed}$ (30 to 100 ml; control 60 ml) and end-diastolic aortic pressure $p_{ed}$ (7 to 20 kPa or 53 to 150 mmHg; control 11 kPa or 83 mmHg).
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pressure (7 to 25 kPa [52 to 188 mmHg], control: 11 kPa [83 mmHg]; 1 kPa = 7.5 mmHg) and of left ventricular volume (20 to 100 ml, control: 60 ml), and without as well as with increased contractility. In the model computations, increased myocardial contractility was simulated by a 50% increase of the parameters controlling the contractile properties of cardiac muscle in the model. These parameters were contractile muscle stress at zero shortening velocity, and shortening velocity at zero load. Fig 2 shows the simulated time course of left ventricular and aortic pressure, aortic volume flow, inner circumferential natural strain ($\epsilon_{ci}$) as well as torsion (γ) during the ejection phase under some of these circumstances. The traces shown in fig 3 represent model calculations of circumferential natural strain of the inner circumference of the cylinder ($\epsilon_{ci}$) as a function of torsion (γ) during the ejection phase for beats with various values of end-diastolic left ventricular volume (preload) and aortic pressure (after-load). All data are close to an approximately straight line. Therefore, all slopes of the relation between $\epsilon_{ci}$ and γ determined within the individual beats, are similar. This slope is quantified as the shortening to torsion ratio (STR), which is calculated by the least square fit of:

$$\epsilon_{ci} - \epsilon_{cib} = \text{STR} (\gamma - \gamma_b)$$

with equidistant samples in time (sampling interval 20 ms) of $\epsilon_{ci}$ and γ during the ejection phase. The parameters $\epsilon_{cib}$ and $\gamma_b$ represent the values of $\epsilon_{ci}$ and γ at the beginning of the ejection phase. In fig 4 the calculated values of STR are plotted as a function of end-diastolic left ventricular volume (left panel) and aortic pressure (right panel). Thus, in the model STR appeared to be quite independent of haemodynamic loading, its value ranged from 2.4 to 2.6 rad$^{-1}$ when end-diastolic left ventricular volume varied between 30 and 100 ml with an end-diastolic aortic pressure of 11 kPa (83 mmHg) and also when the end-diastolic left ventricular volume was 60 ml with the end-diastolic aortic pressure varying between 7 and 25 kPa (52 and 188 mmHg). A 50% increase in contractility under the control end-diastolic circumstances caused a minor increase in STR from 2.40 to 2.43 rad$^{-1}$.

3 In vivo experiments

3.1 METHODS

Experimental measurements were performed in nine mongrel dogs ranging in weight from 22 to 32 kg. The animals were premedicated with morphine (2 mg·kg$^{-1}$ iv). Anaesthesia was induced by sodium pentobarbital (30 mg·kg$^{-1}$ iv). After endotracheal intubation ventilation was kept constant with a respirator pump (Harvard 607). The ECG was derived from the limb leads and visualised on the video monitor screen of a two-dimensional ultrasonic echo scanner system (ATL MARK V). The animal was placed on its right side with the chest extending over the edge of the table.
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FIG 5  Long-axis projection of the left ventricle (LV) and left atrium (LA). Transverse intersections are indicated at mitral valve level (MV; fig 6), low papillary level (LP; fig 7) and mid-papillary level (MP; fig 8).

FIG 6  Transverse projection of the left ventricle at mitral valve level. The line connecting the anterior juncture of the right ventricular free wall to the left ventricle with the inferior commissure of the mitral valve is used for detecting rotation.
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FIG 7  Transverse projection of the left ventricle at low papillary level. The line connecting the right edges of the papillary muscle is used for detecting rotation.

FIG 8  Transverse projection of the left ventricle at mid-papillary level. The lines along which inner and outer diameters are measured, are indicated.
allowing recording of two-dimensional images with the ultrasonic transducer applied from below. ECG and echo images were recorded simultaneously on a video tape.

The two-dimensional echocardiographic protocol consisted of the following video recordings each of 15 s duration:

a) The aortic valve region was imaged and the dynamics of the valve leaflets observed to determine the instant of opening and closure of the valve, with respect to the simultaneously recorded ECG-QRS-complex.

b) Long axis cross-sectional images of the left ventricle and the mitral valve were obtained (fig 5).

c) A transverse cross-section of the left ventricle was imaged at the level of the mitral valve. This view was adjusted so that visualisation of the inferior commissure of the mitral valve as well as the anterior junction of the right ventricular free wall with the left ventricle were optimal throughout the entire ejection phase (fig 6).

d) A second transverse projection was imaged showing the cross-section of the low left ventricle near the apical region. The level of this section was chosen so that the bases of the papillary muscle were still sufficiently visible to serve as markers for detection of ventricular rotation (fig 7).

e) A third transverse cross-section of the left ventricle at the mid-papillary level served to visualise inner and outer diameter of the left ventricle at that site (fig 8).

Fig 9 shows the composition of the various cross-sections in three dimensions. The echocardiographical protocol was completed with a second set of recordings of the long-axis cross-section and of the aortic valve. The entire measuring procedure was repeated once in each experiment.

3.2 ANALYSIS OF RECORDED VIDEO DATA

Aortic valve movements were analysed in at least 10 beats at the beginning as well as the end of each two-dimensional echo measurement procedure. For this purpose, a time reference videoframe was defined in each beat as the frame corresponding to the upward slope of the R-wave of the ECG exceeding half of the peak-voltage. The number of subsequent video frames counted up to the opening and to the closure of the aortic valve were then registered. The time delay between the ECG-trigger and aortic valve opening or closure was determined as the corresponding average number of intervening video frames, divided by the frame rate (60 Hz). The ejection phase of the left ventricle was defined as the period between opening and closure of the aortic valve. Left ventricular transverse section rotation, and transverse diameter during the ejection phase were measured successively at five equidistant points in time, starting at the video frame preceding opening of the aortic valve and ending at or just after the frame preceding closure of the aortic valve. Thus the sampling interval was generally three frames, or 50 ms.

The distance between the centres of the left ventricular cavity at the mitral and low-papillary level was obtained by matching the transverse projections with landmarks seen in the long-axis cross-section projection. The basis of this matching procedure is the property of the long and short axis cross-sections having one intersection line in common throughout the entire cardiac cycle (fig 9). At the mitral level, the relative position of the mitral valve leaflets with respect to the wall of the left ventricle could be observed in the long axis as well as in the corresponding transverse section, generally providing a marker for satisfactory matching. At the low papillary level, inner and outer diameters of the transverse section were matched with the long-axis cross-section projections of these diameters. Thus the distance between the centres of the left ventricular cavity in the two transverse sections was measured in the long-axis projection. In this matching procedure, the use of hard copies of the video images (figs 5, 6, 7 and 8) proved of great help.

At each sampling time inner as well as outer left ventricular diameters in the transverse cross-section at the mid-papillary level were determined by averaging diameter measurements along the line of symmetry between the papillary muscles and along the line perpendicular to this symmetry line (fig 7), obtained from at least five heart beats. Dimensional changes in

FIG 9 Composition of long-axis projection (LA) and transverse projections at mitral valve (MV), mid-papillary (MP) and low-papillary (LP) level to a three dimensional picture of the left ventricle.
the papillary muscles were not used in this study of left ventricular torsion. The resulting time course of changes in measured left ventricular diameters was corrected by correcting wall thickness, so that the wall volume remained constant throughout the period of ejection (Appendix 1). This volume was set equal to the average wall volume calculated from the five pairs of outer and inner diameter determinations. Inner wall circumferential natural strain ($\varepsilon_{ci}$) was calculated from the actual inner diameter ($d_i$) at the mid-papillary level, and the value of this diameter at the beginning of the ejection phase ($d_{ii}$) by:

$$\varepsilon_{ci} = \ln \left( \frac{d_i}{d_{ii}} \right)$$

The angle of rotation of the mitral transverse section was defined so as to be zero at the beginning of the ejection phase. In the remaining four frames to be analysed, rotation of the line connecting the mitral valve leaflet below the inferior commissure to the anterior juncture of the right ventricular free wall and the left ventricle (fig 5) was considered to be representative of rotation of this transverse section. Similarly, rotation of the low papillary transverse section was assumed to be represented by rotation of the line connecting the right edges of the two papillary muscles (fig 6; ref 20). A reliable analysis of changes during the ejection phase required repeated observation of the ultrasonic images at slow video motion, to distinguish the true anatomical landmarks of the left ventricular wall from ultrasonic interference patterns, related to reflections from a variety of sources. The angles of rotation at the mitral ($\alpha_{mv}$) and low papillary ($\alpha_{lp}$) level were measured in at least five heart beats, and the results were averaged. According to its definition the torsion parameter ($\gamma$) is calculated by:

$$\gamma = (\alpha_{mv} - \alpha_{lp}) \cdot d/2h$$

where $h$ = axial distance between mitral and low papillary level and $d$ = outer diameter of the mid-papillary section.

The shortening to torsion ratio (STR) was calculated as the least squares fit for the slope of the relation $\varepsilon_{ci} = \text{STR} \cdot \gamma$ of the four measuring points obtained after opening of the aortic valve.

3.3 RESULTS

In the control state opening and closure of the aortic valve occurred 77±20 ms and 262±28 ms (mean±SD) respectively after half peak voltage was attained on the upward slope of the R wave of the ECG. The duration of the ejection phase in this study was 185±22 ms (mean±SD). Viewed from the apical direction the short axis projection of the left ventricle at the mitral level rotated 0.119±0.054 rad (mean±SD) clockwise while the section at the low-papillary level rotated 0.055±0.067 rad (mean±SD) counter-clockwise. Thus, the direction of torsion during ejection is as indicated in fig 1. At the beginning of ejection, inner and outer diameters of the transverse projection at the mid-papillary level were measured as 46.0±5.1 mm and 67.2±6.7 mm (mean±SD), respectively. The axial distance between the centres of the transverse projections at the mitral valve and low-papillary level was 42.4±6.7 mm (mean±SD). Fig 10 shows the time courses of inner circumferential natural shortening ($\varepsilon_{ci}$) and of torsion ($\gamma$) under normal loading conditions for the nine experiments. In experiments 6 and 7 $\varepsilon_{ci}$ and $\gamma$ were determined twice. Using the data presented in fig 10, $\varepsilon_{ci}$ and $\gamma$ during the ejection phase were calculated as $-0.283±0.062$ and $-0.125±0.027$ rad, respectively, and the shortening to torsion ratio (STR) as 2.31±0.23 rad⁻¹ (mean±SD). Circumferential strain of the outer wall of the left ventricle during the ejection phase was $-0.094±0.021$.

FIG 10  Time course of inner circumferential natural strain ($\varepsilon_{ci}$) and torsion ($\gamma$), as measured in nine different experiments (two measurements in experiments 6 and 7). The experimentally determined duration of the ejection phase is indicated by a horizontal bar.

4 Discussion

4.1 TORSION OF THE LEFT VENTRICLE

In the present study our mathematical model of left ventricular mechanics was evaluated by comparison of torsion and circumferential shortening of the left ventricle as predicted by the model against direct measurement of these deformation parameters during the cardiac ejection phase in the closed-chest
anaesthetised dog. The value of the ratio of shortening to torsion (STR) was predicted to be 2.4 rad$^{-1}$ by the model before the present series of experiments on torsion were performed (fig 12 in ref 13). In the present experiments measured STR was 2.31±0.08 rad$^{-1}$ (mean±SE). The latter value is not significantly different from the model prediction despite the relatively narrow limits of the standard error. The values of STR are signed, ie the direction of torsion calculated for the model agrees with the direction found in the experiments. The agreement between calculated and measured STR values indicates the applicability of the basic principles of the model, which incorporates anisotropy of myocardial tissue, fibre orientation and torsion of the left ventricle.

In the model the sensitivity of the epicardial shear angle and epicardial circumferential shortening to changes in substituted values for fibre orientation angles has been discussed earlier. A 0.1 rad change in fibre orientation in one shell out of eight changes the torsion parameter $\gamma$ by ±3% and circumferential shortening by ±2%. Thus, the shortening to torsion ratio may vary by ±4% if both changes are independent. However, when assuming a uniform distribution of stress and fibre shortening across the wall, a unique distribution of fibre orientation across the wall could be found. In finding this uniqueness a continuous, monotonous change of the fibre angle across the wall was assumed. Mathematically, the mirror image of the solution is also a solution, but this symmetric counterpart is never reported to exist in anatomical studies. The distribution of fibre orientations thus calculated was not significantly different from anatomical measurements. When using this distribution of fibre angles, the results noted in this paper are found.

According to its definition the torsion parameter $\gamma$ is equal to the shear angle at the epicardial surface during the cardiac ejection phase, assuming a uniform distribution of deformation across the left ventricular epicardial surface. In an earlier study, local epicardial shear, and circumferential and base-to-apex shortening were measured with an inductive gauge system placed on the outside of the anterior left ventricular free wall in the open-chest, anaesthetised dog. The derived slope of the relation between shear and shortening during the ejection phase agreed with the slope as predicted by the model within 10%. Because both local measurements of shear and shortening as well as global measurements of torsion and circumferential shortening are found to agree with the model calculations, shear and shortening should be approximately uniformly distributed across the epicardial surface of the left ventricle. Recently deformation of the wall of the left ventricle has been studied using ultrasonic crystals. In that study, shortening of the epicardial surface was found to be 9.2% along the fibre direction and 7.3% along the circumference. On the basis of our measurements these numbers were 13.2%±2.4% and 9.08%±2.0% (mean±SD), respectively. Thus in our measurements the left ventricle shortened more, which might be due to the anaesthesia used in our experimental set-up. However, in comparing both studies for the ratio of circumferential shortening to estimated fibre shortening at the epicardium no significant difference was found.

In measuring torsion from landmark displacements we assumed that a straight line drawn through the centre of a transverse section of the left ventricle remains straight during cardiac contraction. This assumption is supported by earlier findings on systolic changes in the angle between the epicardial surface of the left ventricle and a needle pierced perpendicularly into the wall. During the ejection phase this angle was found to change as little as 0.02 rad without preferential direction indicating a low degree of bending of the straight line mentioned above.

Torsion of the left ventricle in the same direction as found in the present study has been described before. In the study by Ingels et al left ventricular torsion during the ejection phase was detected in humans by biplane cineangiography, using radio-opaque markers implanted in the mid-myocardial layer of the left ventricle. Using their data the torsion parameter $\gamma$ was calculated by us as follows. In fig 11 of the above mentioned paper the difference between the angles of rotation of basal and more apically situated transverse cross sections was 0.218 rad. Assuming their fig 9 to be representative, the ratio of axial distance between the centres of both transverse projections to mid-myocardial wall diameter at the mid-papillary level was calculated as 0.878. Further, it is assumed that the end-systolic ratio of left ventricular cavity volume to wall volume equals 0.20, which results in a value for the ratio of outer wall diameter to mid-wall diameter of 1.43. Thus, the ratio $d/h$ from equation (1) equals 1.43/0.878=1.63. According to equation (1) the torsion parameter $\gamma$ is calculated as 0.178 rad, which is not significantly greater than the value found in our experiments (0.125±0.027 rad; mean±SD). From their table 3, mean systolic circumferential shortening in the mid-myocardial layers was reported to be 18.7%. Assuming: (a) constancy of wall volume during contraction; (b) an end-diastolic ratio of left ventricular cavity to wall volume of 0.54; and (c) shortening from base to apex of 4.7%, natural shortening of the inner circumference ($\varepsilon_i$) of the left ventricle during ejection is calculated to be 0.529 (Appendix 2). Thus STR, based on alternate measurements, as computed by us is 0.529/0.178
rad=2.97 rad\(^{-1}\), which is somewhat greater than the value found in our experiments (2.31 ±0.23 rad\(^{-1}\)). The latter difference can easily be explained by the limited accuracy of the assumptions made by us in relation to the study of Ingels et al.

The technique of two-dimensional ultrasonic echocardiography introduces several possible errors in the measurement of torsion and shortening. The resolution is limited to approximately 1.5 mm. During the cardiac cycle, reflections can change in brightness or even totally disappear at some locations due to angulation or movement of reflecting structures out of the ultrasonic beam sector. Interference phenomena may occur in relation to reflecting objects, resulting in marked changes in brightness along a reflecting surface. However, a two dimensional echocardiographic image of the dog heart is generally of much better quality than human images due to the heart being closer to the chest wall and to less attenuation of ultrasound by air-filled lung tissue. Determination of geometry and rotation can be affected by the system of image processing. Sector lines are renewed continuously, so that after approximately 35 ms the entire image is renewed. Thus, a fast moving image may be distorted due to different phasing of sector lines in one picture. Furthermore, analysis of the video recordings requires steady state conditions of ventricular geometry as far as phasing with the ECG is concerned, and constancy of haemodynamic loading during the entire period of recording of the different projections required in one set of echocardiographic measurements. In observing the heart with ultrasound, the window between ribs and lung structures is generally relatively small, which limits the possibility of obtaining transverse projections in parallel planes at different left ventricular levels. The effects of angulation of these projections on the measurement of rotation can be minimised by choosing in the cross-sections those naturally appearing markers which are mutually opposite with respect to the centre of the transverse projection and at approximately the same distance from the transducer. Fortunately, most of the potential errors of two-dimensional echocardiography mentioned have a random character, so that their influence can be decreased by averaging over five heart beats, as we did here.

The papillary muscles served as useful markers even though they were subsequently excluded from the measurement of internal radius because of their differing function compared with the rest of the wall of the left ventricle. Their fibre orientation is directed from base to apex which results in a force acting on the leaflets of the mitral valve, rather than contributing to left ventricular pressure development by circumferential stress components in the wall.

4.2 TORSION IN RELATION TO TRANSMURAL DISTRIBUTION OF FIBRE MECHANICS

Torsion of the left ventricle is associated with the transmural course of fibre shortening. Ventricular contraction without torsion results in more shortening of the fibres in the inner than in the outer layers of the wall of the left ventricle. Torsion without contraction, however, has the opposite effect. According to our model study a certain amount of contraction is associated with a certain amount of torsion (fig 3) so that the transmural distribution of fibre shortening is homogeneous. This predicted uniformity is supported by findings of Grimm et al., who observed a fairly uniform transmural distribution of changes in sarcomere length when cavity volumes of postmortem left ventricles were changed. Uniformity of fibre shortening suggests uniformity of fibre stress, assuming no transmural differences in muscle contraction properties. Uniformity of fibre stress, which is an important determinant of oxygen consumption, may be inferred from transmural uniformity of coronary perfusion under normal circumstances. When assuming absence of torsion, fibre stress could also be calculated to be uniformly distributed across the wall, but only at the cost of assuming a more compliant muscle behaviour in the inner layers of the wall. Experimental data in the present study, however, show the existence of sufficient torsion during the ejection phase to allow fibre stress to be quite uniformly distributed across the wall of the left ventricle.

Conclusions

1. Global deformation of the left ventricle during the ejection phase, determined with two-dimensional echocardiography, is in agreement with the predictions of a model of left ventricular wall mechanics.

2. In modelling the transmural course of left ventricular muscle fibre mechanics consideration of torsion appears to be essential.

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Appendix 1

CORRECTION OF VENTRICULAR INNER RADIUS FOR CONSTANT WALL VOLUME

During ventricular contraction wall volume is approximately constant. With two-dimensional
echochardiography, inner (ri) and outer (ro) radius are measured independently, resulting in apparent changes in wall volume. In this appendix a method is described in which the inner diameter is estimated from measured inner and outer diameter, and left ventricular wall volume based on the average of geometric data obtained during the whole period of ejection.

For a cylindric left ventricular wall volume \(V_w\) it holds

\[ V_w = \pi h (r_o^2 - r_i^2) \]  
(A1.1)

with \(h\)=length of cylinder. During ventricular contraction shortening of base to apex segment length, which is proportional to \(h\), is 65\% of shortening of an outer circumferential segment length which is proportional to \(r_o\). Thus

\[ V_w = C_1 (r_i^2 - r_i^2) \]  
(A1.2)

where \(C_1\) is a constant.

From a certain video frame the average \(r_o\) of measured inner and outer radius is calculated by

\[ r_o = \frac{r_i + r_o}{2} \]  
(A1.3)

Rewriting equation (A1.3), and substituting \(r_o\) in equation (A1.2) by \(2r_o - r_i\), it holds

\[ \frac{V_w}{C_1} = 4r_o(2r_o - r_i)^{0.65} (r_o - r_i) \]  
(A1.4)

where \(V_w/C_1\) is calculated by

\[ \frac{V_w}{C_1} = \frac{1}{n} \sum_{i=1}^{n} r_{o,i}^{0.65} (r_{o,i} - r_{i,i}) \]  
(A1.15)

where different values \(i\) denote different sampling moments of inner \(r_{i,i}\) and outer \(r_{o,i}\) radii of the ventricle at the mid-papillary level. Solving equation (A1.4), \(r_i\) can be calculated.

Appendix 2

CALCULATION OF INNER CIRCUMFERENTIAL NATURAL SHORTENING FROM MID-WALL SHORTENING

Define parameter \(a\) by

\[ r_{o,ed}/r_{i,ed} = 1 + 2a \]  
(A2.1)

where \(r_{o,ed}\) and \(r_{i,ed}\) are end-diastolic outer and inner radius, respectively. Assuming cylindrical geometry of the left ventricle, the latter ratio of radii depends on the ratio of ventricular wall volume \(V_w\) to end-diastolic cavity volume \(V_{1,ed}\)

\[ (r_{o,ed}/r_{i,ed})^2 = 1 + V_w/V_{1,ed} \]  
(A2.2)

Substituting \(V_{1,ed}/V_w = 0.5414\) into equation (A2.2) and using equation A2.1, it follows \(a = 0.344\). From equation (A2.1) it can be derived that

\[ r_{i,ed}/r_{o,ed} = 1/(1+a) \]  
(A2.3)

where \(r_{o,ed}\) is the end-diastolic mid-wall radius.

For the end-systolic mid wall radius \(r_{a,es}\) it holds

\[ r_{a,es}/r_{a,ed} = b \]  
(A2.4)

where the value of parameter \(b\) is reported to be 0.813.\(^1\) During the ejection phase, the base to apex distance shortens 4.7\%,\(^2\) resulting in an increase in wall cross-sectional area. Thus, it holds

\[ \frac{r_{a,ed} - r_{i,es}^2}{r_{a,es}^2 - r_{i,es}^2} = c \]  
(A2.5)

where \(c = 100\% - 4.7\% = 0.953\). Dividing nominator and denominator in equation (A2.5) by \(r_{a,es}^2\), and using equation (A2.3) and (A2.4) it follows

\[ 1 - \frac{1}{(1+a)^2} = c \]  
(A2.6)

Solving equation (A2.6) for \(r_{i,es}/r_{i,ed}\), for the natural shortening of the inner circumference \(\epsilon_{ci}\) it follows

\[ \epsilon_{ci} = \frac{1}{2} \ln \left( \frac{r_{i,es}}{r_{i,ed}} \right)^2 \]  
(A2.7)

Substituting \(a = 0.344\), \(b = 0.813\) and \(c = 0.953\) it follows from equation (A2.7): \(\epsilon_{ci} = -0.529\).

References

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