Elastic modulus of the human intact left ventricle—determination and physiological interpretation*

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Abstract—*The left ventricle (l.v.) is represented as a shell of muscle whose performance is characterised in terms of the chamber pressure and stress/strain in the ventricular wall; the effective elastic modulus of the l.v. relates these performance variables, and hence represents the transfer function of the left ventricular physiological system. A method is presented for indirectly determining the effective modulus* E *for the left ventricle. The method employs a thick-walled mathematical model of the l.v. having a homogeneous isotropic medium. Instantaneous values of* E *are determined for subjects with heart diseases of varied etiologies, in order to assess the responses of the l.v. to chronic overloads of pressure and volume. Resulting values for* E *are used diagnostically to characterise the physiological state of the l.v. Normal values of* E, *at systole, indicate that the strength of contraction exercised by the l.v. is normal, and hence is an indication of the l.v. having adjusted to the heart disease.*

Keywords—*Elasticity, Human left ventricle, Modulus, Stress/strain, Thick-walled model*

Notation

L.V. = left ventricle
L, W, H = maximum length, calculated width and measured wall thickness of the left ventricular chamber—data quantities
P = pressure in the cavity of the left ventricle
d_1 = $W/2$, for the analytic model
d_2 = $(W/2)+H$, for the analytic model
t = time during a cardiac cycle
(x, y, z) = Cartesian co-ordinates
(r, y, w) = cylindrical co-ordinate system with y as the axis of rotation
$\bar{x}, \bar{r}, \bar{y}$ = $x/a, r/a, y/a$
a = half the length along which point dilatations are distributed
R = ratio of $\bar{y}$ to $\bar{r}$ intercept of a stress trajectory
$(\bar{r}_1, \bar{y}_1)$ and $(\bar{r}_2, \bar{y}_2)$ = intercepts of the stress trajectories of the inner and outer surfaces of the geometrically similar model with the $\bar{r}$ and $\bar{y}$ axes
R_1, R_2 = $(\bar{y}_1/\bar{r}_1)$, $(\bar{y}_2/\bar{r}_2)$, shape parameters of the model
σ^1, ε^1 = stress and strain for line dilatation
σ^2, ε^2 = stress and strain for a uniform hydrostatic stress system
B = strength or intensity factor for a uniform hydrostatic stress system (= magnitude of the uniform hydrostatic stress)
C = strength or intensity factor for a line dilatation system
E = instantaneous effective modulus of the left ventricle
ν = Poisson's ratio

1 Introduction

Characterisation of left ventricular performance: The ability of the heart to function as a pump can be characterised either in terms of hydraulic pressure/volume relationships (CHAPMAN *et al.*, 1959; DODGE *et al.*, 1966; BUNNELL *et al.*, 1965, RACKLEY *et al.*, 1967) or alternatively in terms of mechanical variables, namely, force, velocity, length and time (SONNENBLICK 1962; BRAUNWALD *et al.*, 1967; JEWELL and BLINKS, 1968). We will employ the latter approach and will characterise left ventricular performance in terms of stress and strain variables rather than in terms of force and length variables. However, instead of assessing l.v. performance by an analysis of just the measurable pressure and mechanical (stress and strain) variables, it is more

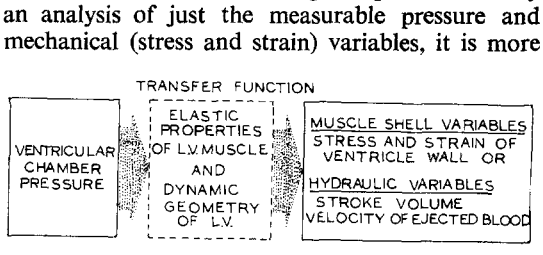

Fig. 1 Role of elastic properties of l.v. muscle

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pertinent to determine the intrinsic parameters that relate these variables for the shell of muscle representing the intact heart. This approach is summarised in Fig. 1. The intrinsic parameters are the effective elastic properties (namely the modulus and the Poisson's ratio) of the l.v. muscle shell and the dynamic geometry of the left ventricular chamber. One of the intrinsic parameters, namely the dynamic geometry of the l.v., is obtained from cineangiocardiography. Among the elastic properties, the modulus E (to be defined later) represents the stiffness or the elasticity of the left ventricular muscle shell, whereas the Poisson's ratio ν characterises the volume changes in the muscle during its activity. However, since the left ventricular muscle is incompressible (i.e. it does not change volume, during the heart cycle), ν is assumed to equal 0·5. Hence the modulus will be employed as the diagnostic parameter.

Significance of modulus: Our analytical model of the l.v. will only permit us to analyse the stress/strain response to a pressure loading, for a given set of ventricular dimensions. Based on our definition of the effective modulus of the l.v. (to be presented later), E governs the stress/strain response of the relaxed l.v. to a certain pressure in the ventricular cavity, and, in effect, characterises the material property of the relaxed l.v. muscle. In the contracted l.v. the effective elastic modulus of the l.v. reflects the strength of contraction (or the contractile effort) needed to maintain a certain cardiac output.

Left ventricular analytical model: Our thick-walled ellipsoidal-shaped analytic model is that of GHISTA and SANDLER (1969); the dimensions for the model are taken to be the instantaneous dimensions of the intact l.v. (obtained from cineangiocardiography). The model then determines the stresses in the left ventricular wall when it is loaded by the recorded l.v. pressure (obtained by cardiac catheterisation). The effective modulus relates this obtainable wall stress to the wall strain, calculated from the left ventricular dynamic geometry (obtained from cineangiocardigraphy); it can hence be readily obtained from data on the left ventricular chamber geometry and pressure.

2 Instantaneous effective modulus of the l.v.: *in vivo* determination and physiological significance

Data acquisition: All subjects included in this study are listed in Table 1 and were studied in a resting recumbent state, after premedication with 100-500 mg of sodium pentobarbital by transseptal and/or retrograde aortic catheterisation. Left ventricular chamber pressure was measured by a fluid-filled catheter and Statham P23Eb pressure transducer; the pressure was recorded immediately before or during angiocardiography in all cases. In the case of subjects V.B. and V.P., ventricular pressure was measured by means of an ultraminiature (1·0 mm diameter) manometer-tipped catheter (COON and SANDLER, 1967) passed through a transseptal catheter. Corrections for the onset of the pressure wave in the fluid-filled catheter system were obtained from studies using the manometer-tipped catheter and were used to correct all the measured ventricular pressures prior to comparison with the calculated ventricular dimensions.

Angiocardiography was performed by injecting 40–50 ml of 75% sodium diatrizoate into the left artrium or left ventricle. None of the subjects included in this study demonstrated a change in heart rate or pressure during the angiocardiogram. It has been found by using biplane angiocardiograms that calculated orthogonal chamber diameters are nearly identical (SANDLER and DODGE, 1968). These findings are used to justify the use of single-plane cine techniques which allow for beat-to-beat analysis of the chamber dimensions as used in subjects J.E., V.B. and V.P. of this study. Single-plane cineangiocardiograms were recorded in a posterioanterior projection from a 9 in image intensifier using 35 mm film at 48 frames/s. All other subjects were studied by biplane angiocardiography at 6-12 frames/s. The calculations of ventricular dimensions by radiographic or cine techniques are made by employing the procedures of DODGE *et al.* (1960) or COHN *et al.* (1967). The variables derived from the cineangiographic films are defined in Fig. 9; they consist of a measured maximum length or major axis L of the chamber, calculated chamber diameter or minor axis W, and measured lateral wall thickness H. All measurements are corrected for spherical and/or geometric distortion due to their respective recording systems (DODGE *et al.*, 1960; COHN *et al.*, 1967).

Analysis: The thick-walled analytic left ventricular model used in this investigation (illustrated in Fig. 9) has been derived and described mathematically by the authors (GHISTA and SANDLER, 1969). On its inner surface, the model is loaded with stresses equal to the measured l.v. pressure; the outer surface is stress-free.

Definition of the modulus of the l.v.: The modulus is used to describe the relationship of the stress in the left ventricular model to the associated deformation of the model. Consider a muscle strip, of unit cross-section and of resting length l_r, held fixed at one end and loaded at the other end with an instantaneous weight W_{rl}; let l_{rl} be the corresponding instantaneous length. Then the instantaneous strain ε_{rl} of the relaxed muscle in the direction of loading is $(l_{rl}-l_r/l_r)$. The instantaneous modulus of the relaxed muscle is defined as

$$E_{relaxed} = \frac{W_{rl}}{\varepsilon_{rl}} = \frac{W_{rl}}{(l_{rl}-l_r)/l_r} \quad . \quad . \quad (1)$$

For a contracted muscle, let W_{ct} be the instantaneous force developed in the muscle, l_{ct} be its instantaneous length and l_m the minimum length attained on shortening. The instantaneous strain ε_{ct} of the contracted muscle, corresponding to its length l_{ct}, is taken to be $(l_{ct}-l_m)/l_m$. The instantaneous modulus of the contracted muscle is defined as

$$E_{contracted} = \frac{W_{ct}}{\varepsilon_{ct}} = \frac{W_{ct}}{(l_{ct}-l_m)/l_m} \quad . \quad . \quad (2)$$

It is to be noted that, in defining the modulus as above, we are lumping all the different rheological components of the muscle (namely, the series elastic element, the parallel elastic element and the contractile element) into one unit. When we speak of the deformation of the muscle, it is the deformation of the overall unit. The modulus, defined by eqns. 1 and 2, is thus an overall effective modulus.

Let us note the significance of the instantaneous modulus, as defined by eqns. 1 and 2. For a relaxed muscle, the instantaneous modulus reflects resistance to stretch under a given loading. For a contracting muscle the force developed in the muscle (at a given instantaneous strain, as defined above) is representative of the strength of contraction of the muscle. The greater the force developed, the greater is the strength of contraction; also, by our definition of eqn. 2, the greater the force developed for a certain length l_{ct}, the higher the modulus. Hence, for a contracting muscle, the strength of contraction is reflected by the modulus.

The above definitions are given in terms of a one-dimensional muscle strip. When we apply these basic ideas to the 3-dimensional left ventricular shell structure, we have to consider the generalised 3-dimensional stress/strain relationship.

During diastole, the minimally loaded state (analogous to the length l_r in eqn. 1, when the chamber pressure is a minimum) serves as the reference state for measuring instantaneous diastolic strains; i.e. the instantaneous left ventricular dimensions are referred to this minimally loaded state in determining the instantaneous strains. From this reference state, the ventricular chamber enlarges as it is filled. During systole, the reference state will be taken to be the minimal dimensional state, attained towards the end of ejection.

Determination of the effective modulus of the l.v.: The method for determining the value of the instantaneous modulus of the l.v. muscle is described in detail in the Appendix. Here, we will qualitatively present the method.

The instantaneous strain in a central element of our analytical model (Fig. 5) in a principal direction, such as the hoop direction, is given by eqn. 3 (in the Appendix) in terms of the instantaneous left-ventricle chamber's pressure and dimensions and the hitherto unknown value of the modulus E.

Key to Table 1

MS = mitral stenosis
AS = aortic stenosis
MI = mitral insufficiency
AI = aortic insufficiency
S/D = at systole and diastole
ED = end diastole
ε_w = hoop strain
L = l.v. length
W = l.v. width
H = l.v. wall thickness
EDV = end diastolic volume
σ_w = peak hoop stress
SV = stroke volume
LVP = l.v. chamber pressure
E = effective modulus of the l.v.
LVM = l.v. mass
r.c.e.c. = reserve contractile effort capacity
c.e. = contractile effort

Thus (since the instantaneous dimensions and pressure are known) we can write the instantaneous strain in the model ε^M as a function of the modulus E only:

$$\varepsilon^M = \varepsilon^M(E)$$

Now, the numerical value of the instantaneous incremental strain (in the same principal direction) in the central element of the *simulated l.v.* is obtained from the dynamic geometry of the l.v. by employing eqn. 8; let us denote this value by ε^V. Since the model simulates the l.v., we equate the expression $\varepsilon^M(E)$ to the numerical value ε^V and solve the equation to get the value of the modulus E.

Physiological significance of the modulus of the l.v.: Keeping in mind the definitions given in eqns. 1 and 2 (and the interpretations given to these definitions), it is seen that during the diastolic phase of the cardiac cycle, the modulus E is a measure of the resistance of the muscle to stretch by the applied load, i.e. the filling pressure. A low value of the modulus, at diastole, enables the ventricle to dilate adequately before the muscle contracts; a high value of the diastolic modulus resists or prevents adequate filling.

As the left ventricular muscle starts contracting, it develops force; as indicated earlier, the force developed or the strength of contraction is reflected by the modulus of the l.v. Now, the contracting muscle tends to shorten against the almost incompressible blood in the l.v. chamber. Since both the valves of the chamber are closed, the contracting muscle, in turn, raises the chamber pressure. As the chamber pressure rises, the stress in the muscular wall of the l.v. must also increase (SANDLER and DODGE, 1963; HOOD *et al.*, 1968). When the strength of contraction or the modulus of the l.v. has increased sufficiently so as to raise the pressure in the left ventricular chamber above the pressure in the aorta, the aortic valve opens and ejection starts. Even during ejection, the degree to which, at any instant, the l.v. tends to the minimal dimensional state depends on the strength of contraction, i.e. the modulus of the l.v. Hence, we see that the value

Table 1

Subject and disease	LVP Aortic P (mm/Hg)	L/W (mm) ED / SYS	L/W S/D	LVM (g)	H S(D (mm)	H/W SYS	EDV/SV (cm^3)	ε_w Hoop strain (%)	σ_z Peak hoop stress ($\times 10^2$ N/m^2)	Modulus S/D ($\times 10^2$ N/m^2)	Remarks (l.v. muscle state)
E.S. (MS)	102/10 / 116/75	88/47 / 77·8/36·6	2·3/1·9	90	8·8/5·4	0·202	89/51	30	310	600/70	normal
J.E. (MS)	100/8 / 100/60	102/39 / 92·4/31	3·0/2·6	80	11/6	0·365	70/50	35	260	270/100	hypertrophied l.v.
V.G. (MS)	135/13 / 140/80	93·1/48 / 81·6/33·6	2·4/1·9	190	18/10	0·627	96/63	39	240	350/90	hypertrophied l.v.
S.S. (MS+MI)	115/7 / 125/85	98·4/51·2 / 87·6/37·4	2·35/1·9	210	17/10	0·417	120/68	28	210	275/65	hypertrophied l.v.
J.C. (MS, MI)	105/8 / 115/80	94/50 / 80/46	1·3/1·2	430	20/13	0·25	290/125	20	150	800/160	normal c.e.
M.N. (MS, MI)	115/9 / 120/80	105/65 / 99·2/51·4	1·9/1·6	180	8·5/6	0·165	191/159	48	398	450/70	high distensibility
H.G. (MS, MI, AI)	117/14 / 117/75	131/64 / 122·6/53·6	2·3/2·0	270	13/10	0·245	264/120	22	310	725/190	normal c.e.
J.K.K. (MS, MI, AI)	145/8 / 150/100	103/62 / 94·2/41·4	2·0/1·7	220	12/87	0·225	186/88	24	350	450/60	high r.c.e.c.
N.W. (MS, MI, AI)	162/10 / 160/80	112/54 / 103·4/46	2·55/2·1	190	16/9	0·265	168/87	26	430	1500/210	low r.c.e.c.
C.S. (MS, MI, AI)	140/8 / 140/80	114/67 / 108·4/55·2	2·0/1·7	300	15/10	0·234	257/119	22	350	1800/1000	low r.c.e.c.
P.B. (AS)	198/25 / 160/80	123/67 / 115·6/60·6	2·0/1·8	270	12/8·5	0·197	263/79	15	550	4000/1100	low r.c.e.c.
M.L. (IMH)	109/12 / 115/70	105/81 / 99/77·8	1·3/1·3	230	9·3/7·6	0·111	368/95	8·6	390	2200/240	low r.c.e.c.
V.B. (Post OPMV)	135/6 / 140/80	129/79 / 120/70	1·7/1·6	490	15/13	0·125	425/125	13	410	3500/200	low r.c.e.c.

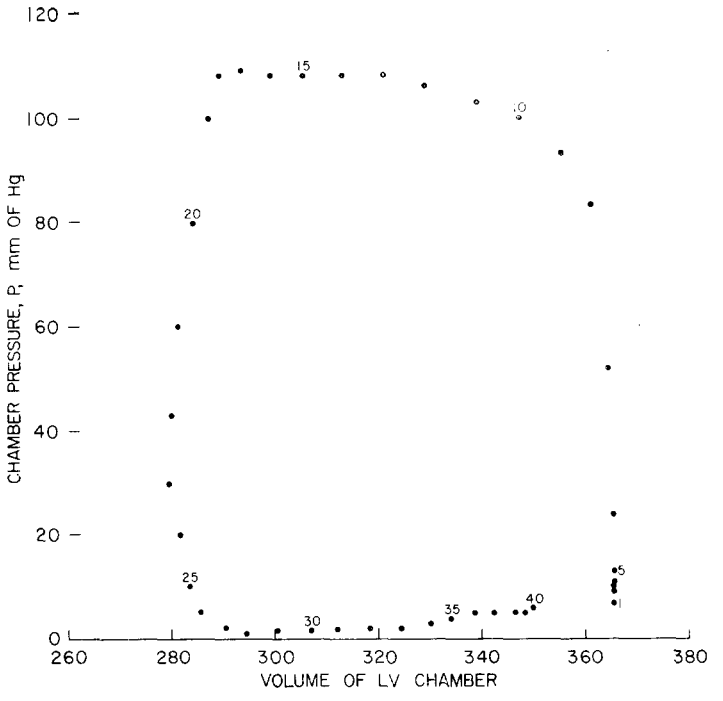

Fig. 2 Relationship between hoop stress and hoop strain during a cardiac cycle

of the modulus, at an instant of systolic phase (or the instantaneous systolic modulus E) is a representation of the effort put in by the cardiac muscle in performing its pumping function, which we call the contractile effort of the l.v.

3 Criteria for assessing the physiological state of the l.v.

Let us study the response of the l.v. to volume and pressure loads caused by valvular disorders, peripheral vaso-constriction etc. The response to volume load is ventricular dilatation. Now, in dilatation, the size increases; we have noted, from our analytical expressions (eqn. 3), that in the dilated left ventricular chamber an adequately high chamber pressure necessary to eject the normal complement of blood would require a considerable increase in the modulus E if the wall thickness remained the same. On the other hand it is noted from the analysis that, if the l.v. muscle hypertrophies so that the l.v. wall thickness increases just adequately relative to the increased size of the dilated chamber, then the modulus E would remain normal; i.e. normal effort would be required of the l.v. muscle to eject its normal complement of blood. Hence we note that our criterion for the l.v. having adjusted to the heart disease is based on the value of the instantaneous modulus E at systole (or the instantaneous systolic modulus).

Based on this discussion, we will now propose the following criteria for the physiologic state of the l.v.:

(*a*) If the value of the instantaneous systolic modulus is normal, the contractile effort of the l.v. muscle is normal. This means that there is provision for reserve contractile effort; i.e. under situations of abnormal physiological stress, there is still provision for the l.v. to exert a higher effort. Hence the l.v. can be said to have adjusted to the disease.

(*b*) If the value of the instantaneous systolic modulus is high, the l.v. is being subjected to a sustained high effort. Hence, the reserve contractile effort capacity is low. An abnormally high value of the systolic modulus is thus an indication of nonadjustment to the heart disease.

(*c*) Even the adjusted l.v. could deteriorate to a failing l.v. if, owing to excessive hypertropy (i.e. overcompensation), the oxygen utilisation of the l.v. is inadequate. Towards this end, we can employ an analytical method to determine the oxygen utilisation of the l.v., utilising the l.v. dimensions and pressure data (GHISTA and SANDLER, 1970).

We will now make some clinical applications of our studies and apply the observations that we have made so far, to diagnose the nature of l.v. response to heart diseases.

4 Results of clinical applications

The results for the 13 subjects included in this study are listed in Table 1. For each subject the

chamber pressure and dimensions are monitored at 20 ms intervals during the cardiac cycle; a typical set of pressure and chamber variations for a subject is shown in Fig. 2. The modulus E was similarly calculated for each 20 ms during the cardiac cycle, as indicated earlier (see Fig. 3 for an illustration of the modulus variation); E during systole was read from such a curve (for each subject) at the level of pressure necessary to open the aortic valves as determined from the diastolic pressure levels from aortic or brachial arterial tracings measured during angiocardiography. E during diastole was read at the point midway between the nadir of the l.v. pressure curve and the onset of systole (point of rapid rise of pressure curve). E remained fairly uniform during diastole for all subjects. During systole, E was noted to be almost continuously variable during the cardiac cycle.

The subject E.S., with a stenotic mitral valve, has a normal ventricle size and pressure fluctuation. Note, from the ratio H/W, that no compensation is required. The findings in this subject can serve as a standard for comparison with other subjects to detect abnormalities. For subjects J.E., S.S. and V.G., the chamber size, as assessed by end-diastolic volume (e.d.v.) and change in volume [stroke volume (s.v.)], were within normal limits for these subjects, as was the intra-cavitary pressure (l.v.p.). The systolic modulus varied from 2·5 to $4{\cdot}5 \times 10^4$ N/m^2 for these subjects. E during diastole was noted to range from 5 to 7×10^3 N/m^2, an eight-fold decrease from the systolic values for these same subjects. These three subjects have distinctly hypertrophied l.v.s, as denoted from col. 7 of Table 1. Under this condition, it is useful to determine the oxygen consumption of the l.v. in order to determine whether there has been insufficient oxygen delivery to the l.v. muscle as a result of overcompensative hypertrophy. Certain of the subjects listed in Table 1 had moduli which differed significantly from those of the above-mentioned subjects; subjects C.S., M.L., P.B. and V.B. had systolic moduli seven to 10 times greater than those listed above; and diastolic moduli four to 10 times over the above diastolic values. An abnormally high value for E in each case is an indication or index of noncompensation for the load imposed by the specific physiologic state under study.

Subject P.B. serves as an example of a patient with aortic stenosis, enlarged chamber and relatively thin chamber wall. The elevated diastolic modulus in this patient, as well as C.S., V.B. and M.L., represents a stiff muscle. End diastolic volume was increased to at least two to three times normal values for these latter three subjects, but end-diastolic pressure was elevated significantly only

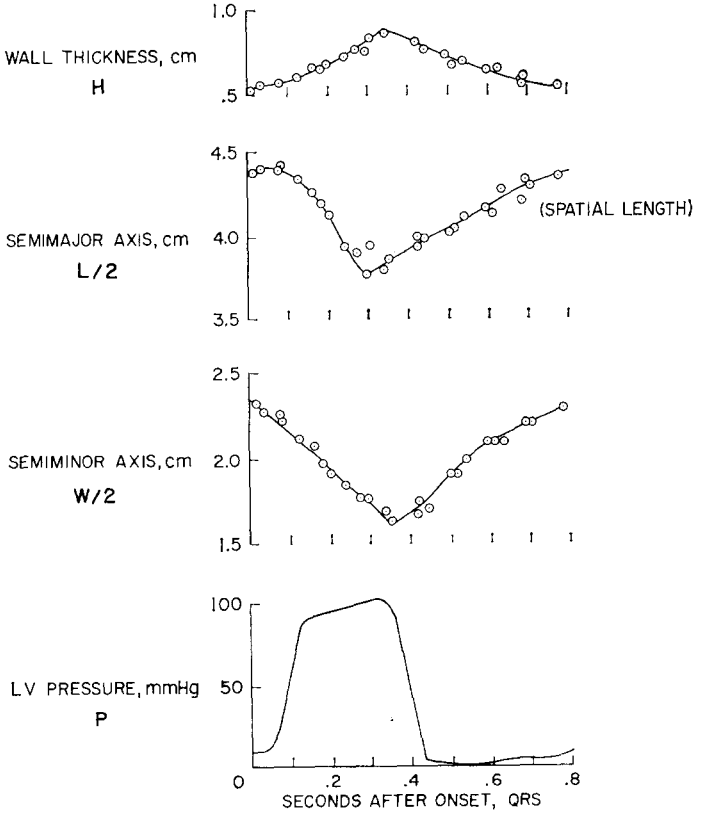

Fig. 3 Pressure and dimensions during a cardiac cycle

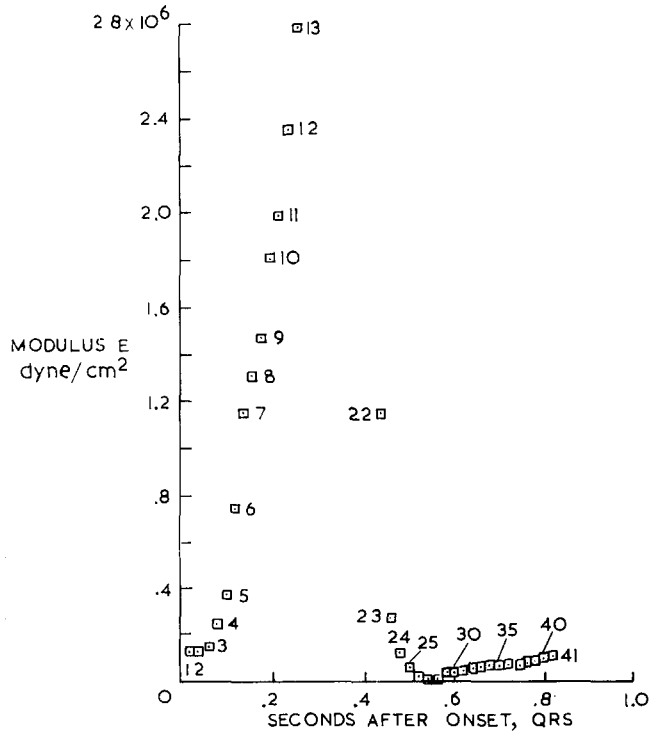

Fig. 4 Time course for the elastic modulus during the cardiac cycle

for P.B. The calculation of compliance* (CORSON *et al.*, 1963) or the use of end-diasatolic pressure (BRAUNWALD and ROSS, 1963) did not assist in characterising such ventricles with chronic heart disease. In contrast, H.G., J.K.K., J.C. and M.N. represent subjects with significant valvular regurgitation where appropriate changes in chamber size and shape have occurred, and, in spite of that, the values for systolic and diastolic moduli are normal—an indication of compensation to the disease.

Finally, let us examine the results for subjects C.S. and H.G. They both have enlarged chambers and high stroke volume; C.S. has a higher systolic pressure. Note that the values of their ratios of ventricular wall thickness to ventricular width (col. 7) are almost identical. On the basis of their thickness/width ratios, it would seem that both the ventricles have had compensative hypertophy to the disease; yet their moduli indicate that, whereas H.G.'s ventricle is exerting a normal contractile effort, that of C.S. is exerting an above normal effort and hence would have a low reserve contractile-effort capacity.

One should not attempt to use peak hoop stress diagnostically. As seen from eqns 3–4 in the

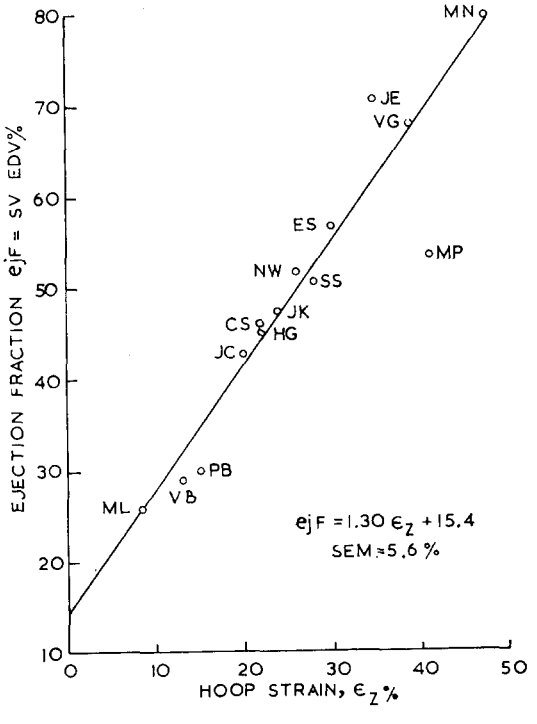

Fig. 5 Comparison of hoop strain and ejection fraction

**Compliance, as given by dV/dP, is by no means an intrinsic parameter of the l.v. muscle. In this form, it is a mere algebraic operation on the measurement data of pressure and volume, and takes no account of the internal mechanisms, due to elasticity of the muscle, contributing to the pressure/volume results. Hence, compliance reflects, in a general way, the overall dynamic characteristics of the l.v. and is not indicative of the l.v. muscle's material property.*

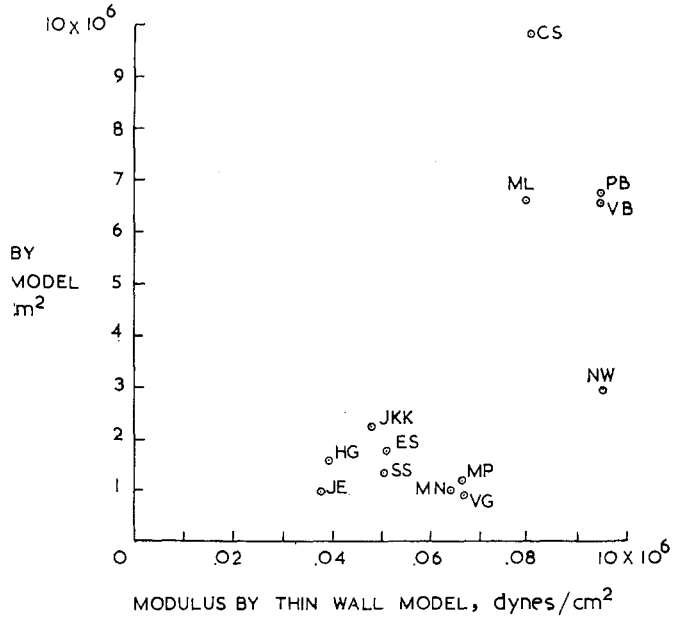

Fig. 6 Comparison of the modulus calculated by thick- and thin-wall models

Appendix, the stress merely depends on the l.v. chamber's dimensions and pressure, and is independent of muscle properties. Hence the stress does not reflect the state of the muscle at all. This is further indicated by the values of peak hoop stress (given in the Table), for the examined subjects, varying asynchronously with the extent and nature of the subjects' disorder.

Subjects (such as M.L., P.B. and V.B.) with a relatively small hoop strain (below 22%, say) had elevated moduli. Further, the hoop strain correlated closely with the ejection fraction for all subjects (as illustrated in Fig. 4); therefore we note that a high modulus (representing a stiff muscle) prevents a more complete ejection.

We have diagnosed the 'state of health' of the cardiac muscle of the subjects studied in Table 1 on the basis of the value of the muscle's modulus. Our diagnoses have correlated quite closely with the clinical course, to date, of the subjects studied; subjects with elevated values of modulus have fared poorly.

5 Discussion

Cardiologists have been continually looking for better and more pertinent cardiac indices. Reviews (including analyses and clinical applications) of the available cardiac indexes (and associated mechanical analyses) have been compiled by MIRSKY and GHISTA (1973) and GHISTA and MIRSKY (1973).

Since the early work of WOODS (1892), Laplace's law has been used to interpret and understand the effect of change in cardiac size and shape on the subsequent l.v. function. Theoretical and practical applications of these concepts to the diseased heart have been demonstrated by several investigators (BURTON, 1957; SANDLER and DODGE, 1963; BURCH *et al.*, 1952). The law relates the pressure supported or generated by the walls of a hollow organ to the tension within its walls and the associated curvature of that wall; this law is only applicable to thin-walled organs where the effect of shear in the walls is negligible.

It is known that the ratio of ventricular wall thickness to internal dimension is of the order of 0·1–0·25. It is also recognised that ventricular wall thickness is an important independent variable in determining cardiac performance (HAWTHORNE, 1966; GRANT *et al.*, 1965; FEIGL and FRY, 1964; HOOD *et al.*, 1968). Hearts of different antomical size have been demonstrated to have different wall thicknesses and changes in wall thickness during the cardiac cycle (SANDLER and DODGE, 1963). On the basis of these findings, the use of Laplace's law and thin-walled models is not justifiable for the l.v.

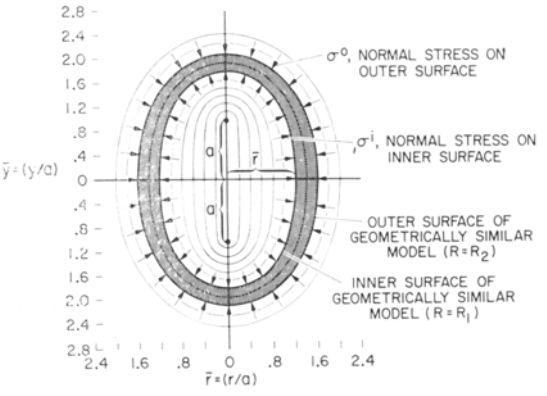

Fig. 7 Stress trajectories for the analytic model

Accounting for the thick wall in a mathematical model affects the stress distribution in the wall and the value of the modulus, as determined by the procedure indicated earlier. WONG and RAUTAHARJU (1968), MIRSKY (1969) and GHISTA and SANDLER (1969) have presented independent mathematical solutions for the stress distribution in a thick-walled ellipsoid of revolution; GHISTA and SANDLER's model has been employed in this study.

With the advent of new instrumentation for the measurement of cardiac dimensions in animals and from the use of angiocardiographic techniques in man, it has been demonstrated that the shape of the l.v. and the change in shape during the cardiac cycle are more closely approximated by an ellipsoid (SANDLER and DODGE, 1968; HAWTHORNE, 1966; HAWTHORNE, 1961; RUSHMER, 1955; RUSHMER and THAL, 1951). Recent studies in man have also demonstrated that the minor axes of the l.v. chamber are nearly identical, and this has led to the use of an ellipsoid of revolution for the model geometry. The GHISTA and SANDLER model (1969) resembles a thick-walled ellipsoid of revolution and allows for a variation in shape during a cycle which is consistent with observations of the dynamic geometry of the human l.v. as analysed from biplane or cineangiocardiographic films. The model becomes less oval shaped as the internal cavity gets bigger, as during diastole, and becomes more oval shaped as it becomes smaller, as during systole. The model medium is isotropic and allows for variation in wall thickness. Exact elasticity has been employed in developing this model; the model provides for (i) shear in the ventricle wall, (ii) normal stress in the ventricle wall and (iii) variation of stresses across the wall in three dimensions. The use of our thick-walled model, in preference to a thin-walled model (SANDLER and DODGE, 1963) makes an appreciable difference to the value of the calculated modulus. The modulus of the l.v. was calculated for the subjects studied in Table 1, by the two models, at the instant of attainment of peak hoop stress (by the procedure described earlier); the results are compared in Fig. 5.

Comparison of the values found for E in the intact heart from this study with values for cardiac muscle determined by other methods is difficult, owing to the marked differences in the methods employed. Classic values of E for postmortem cardiac muscle have ranged from $0 \cdot 3$ to 5×10^4 N/m^2 for relaxed isolated cardiac muscle fibres and a tenfold increase of modulus on contraction. ANLIKER (1968) has reported findings for the systolic modulus of $2-4 \times 10^5$ N/m^2 in open-chested dogs using a method of artificially generated volume perturbation, and measuring the corresponding pressure perturbations. The values of E of subjects studied herein (by our method) are of the same order as the values reported by LUND (1944) and ANLIKER for experimental animals.

6 Appendix: Determination of the modulus of the l.v. muscle

Strain in the model: For a central, equatorial wall element of the analytical model, the model strain ε^M in a principal direction (hoop or meridianal direction, say) is a function of the known dimensions and pressure and

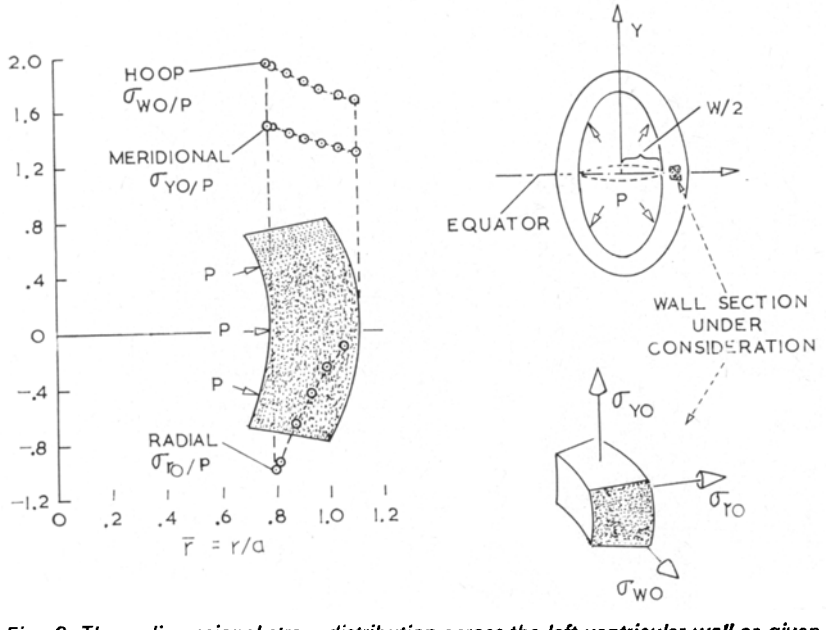

Fig. 8 Three-dimensional stress distribution across the left ventricular wall as given by the Ghista–Sandler model

the hitherto unknown modulus of the l.v. muscle, and is given by (GHISTU and SANDLER, 1969)

$$\left.\begin{aligned}\varepsilon^{M}_{ww' \text{ equator}} &= \frac{P(1-2\nu)}{EK_2} \\ &\quad + \frac{4P(1+\nu)}{EK(W_c/a)^2\{(W_c/a)^2+1\}^{1/2}} \\ \varepsilon^{M}_{\beta\beta' \text{ equator}} &= \frac{P(1-2\nu)}{EK_2} + \frac{4P(1+\nu)}{EK_1\{(W_c/a)^2+1\}^{3/2}}\end{aligned}\right\} \quad (3)$$

Strain in the l.v.: For the purpose of calculating the strain in the l.v. at an instant i, we characterise the instantaneous geometry of the l.v. by a thick-walled ellipsoid of revolution whose internal major and minor axes and wall thickness equal L_i, W_i and H_i, respectively. Now in an ellipsoid co-ordinate system given by the following transformation equations:

$$r = \sin\alpha \cos\beta, \quad y = \cos h\alpha \sin\beta \quad . \quad . \quad . \quad . \quad (4)$$

the hoop strain ε_{ww} and the meridional strain $\varepsilon_{\beta\beta}$, at the equator $\beta = 0$, are given by LOVE (1944):

$$\varepsilon_{ww,\ equator} = \frac{(U_\alpha)\ \beta = 0}{\pi_0}, \quad \varepsilon_{\beta\beta,\ equator} \frac{\pi_0 (U_\alpha)\ \beta = 0}{{y_0}^2} \quad (5)$$

where (i) r_0 and y_0 are the undeformed lengths of the semiminor and semimajor axes, respectively, of an elemental surface, and (ii) U_α is the displacement in the direction normal to the ellipsoidal surface α = constant.

During diastole, the deformed state of the l.v. is measured with respect to the state of the ventricle when the pressure P is a minimum; during systole, the reference state is the instant when the chamber size is a minimum. Thus for the central muscle wall strip [whose instantaneous dimensions are $W_c = (W + H)/2$ and $L_c = (L + H)/2$], the undeformed lengths r_0 and y_0 and the displacement $(u_\alpha)_{\beta=0}$ of eqn. 5 are given by

$$\begin{aligned}\pi_0 &= (W_c)@P_{min} \text{ during diastole} \\ \pi_0 &= (W_c)_{min} \text{ during systole} \\ y_0 &= (L_c)@P_{min} \text{ during diastole} \\ y_0 &= (L_c)_{min} \text{ during systole} \quad . \quad . \quad . \quad . \quad . \quad . \quad (6)\end{aligned}$$

$$\begin{aligned}(U_\alpha)_{\beta=0} &= (W_c)_i - (W_c)@P_{min}, \text{ during diastole} \\ &= (W_c)_i - (W_c)_{min}, \text{ during systole} \quad . \quad . \quad (7)\end{aligned}$$

Hence the experimentally obtained instantaneous strains for a central element in the equatorial plane of the l.v. are given, from eqns 5–7, as follows:

$$\varepsilon^{V}_{ww \text{ equator}} = \frac{(W_c)_i - (W_c)@P_{min}}{(W_c)@P_{min}}, \text{ during diastole}$$

$$= \frac{(W_c)_i - (W_c)_{min}}{(W_c)_{min}}, \text{ during systole} \quad . \quad . \quad (8)$$

$$\varepsilon^{V}_{\beta\beta \text{ equator}} = \frac{(W_c)@P_{min}[(W_c)_i - (W_c)@P_{min}]}{({L_c}^2)@Q_{min}}$$

during diastole

$$= \frac{(W_c)_{min}[(W_c)_i - (W_c)_{min}]}{({L_c}^2)_{min}}, \text{ during systole}$$

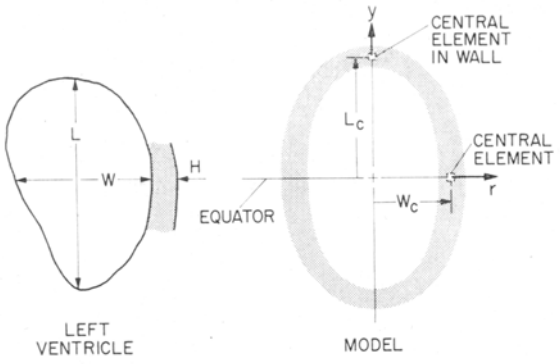

Fig. 9 Comparison of left ventricle with corresponding model

We now equate the expression $\varepsilon_{ww}{}^{M}$ or $\varepsilon_{\beta\beta}{}^{M}$ (eqn. 3)—a function of the modulus E—to its numerical values $\varepsilon_{ww}{}^{V}$ or $\varepsilon_{\beta\beta}{}^{V}$, as the case may be, calculated from eqn. 8; the equation is solved for the unknown E. A cyclic continuous time variation of the modulus E can be obtained by representing the chamber pressure and dimensions as periodic functions of time by Fourier series (GHISTA and VAYO 1969).

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Module d'élasticité du ventricule gauche intacte Détermination et interprétation physiologique

Sommaire—Le ventricule gauche (VG) est représenté par une enveloppe musculaire dont la performance est caractérisée par la pression de la chambre et la contrainte et la déformation sur la paroi ventriculaire; le module d'élasticité ventriculaire effectif du VG décrit la relation entre ces variables de la performance et représente donc la fonction du transfer du système physiologique du ventricule gauche. On présente une méthode de détermination indirecte du module effectif E du ventricule gauche. La méthode fait usage d'un modèle mathématique à paroi épaisse du VG et ayant un milieu isotropique homogène. On détermine des valeurs instantanées de E pour des sujets atteints de maladies cardiaques à étiologies variables, de façon à évaluer la réponse du VG aux surcharges chroniques de pression et de volume. Les valeurs résultantes de E sont utilisées diagnostiquement pour caractériser l'état physiologique du VG. Des valeurs normales de E à la systole indiquent que la force de la contraction exercée par le VG est normale et qu'il s'est donc ajusté à la maladie cardiaque.

Elastische Module des intakten linken menschlichen Ventrikels —Bestimmung und physiologische Interpretation

Zusammenfassung—Das linke Ventrikel (LV) wird als Muskelschale dargestellt, dessen Leistung durch Kammerdruck und Beanspruchung der Ventrikulärwand beschrieben wird. Das effektive elastische Modul des LV bringt diese Leistungsvariablen aufeindander in Bezug und stellt daher die Übertragungsfunktion des linken physiologischen Ventrikelsystems dar. Es wird ein Verfahren zur indirekten Bestimmung des effektiven Moduls (E) für das linke Ventrikel dargestellt. Das Verfahren verwendet das starkwandige mathematische Modell eines LV mit einem homogenen isotropen Medium. Sofortige E-Werte werden für Fälle mit Herzkrankheiten verschiedener Ursachen betimmt, um die Reaktion des LV auf chronische Überbelastung durch Druck und Volumen zu beaurteilen. Die sich für E ergebenden Werte werden diagnostisch dazu verwendent, den physiologischen Zustand des LV zu bestimmen. Normale Werte für E bei Systole bedeuten, daß die vom LV ausgeführte Kontraktion normal stark ist, was bedeutet, daß sich das LV auf die Herzkrankheit eingestellt hat.