Assessment of Left Ventricular Function Using an Angiographic Method

SHIGETAKE SASAYAMA, M.D., HIROSHI NONOGI, M.D.
AND CHUICHI KAWAI, M.D.

Angiographic methods are by far the most frequently used for calculation of left ventricular volume, mass, forces acting within the ventricular wall or for analysis of contractile performance and diastolic property of the overall ventricle, as well as regional myocardial function in the presence of ischemia. A critical review of the theoretical basis and practical methods of calculation of these variables is presented. The left ventricular pressure-volume diagram is constructed which allows for an assessment of the inotropic state of the ventricle from the similar tension-length framework as obtained in the isolated papillary muscle. Computer technology of digital filtering and subtraction provides for an enhancement of the contrast of the ventricular image obtained with minimal doses of contrast medium. On-line methods for edge tracing and subsequent data processing are alleviating much of the tedious and laborious work of manual analysis of angiograms and accuracy of the calculation has been enhanced. Left ventriculography is most useful in diagnosing the presence of abnormal wall motion. Methods of assessing the magnitude and extent of the relative area of localized myocardial dysfunction are discussed. Quantitative information is crucial for evaluating prognosis and determining the application of therapeutic interventions.

SIZE and shape of the ventricles are important determinants of cardiac function. Several methods have now been developed which enabled measurement of cardiac dimensions in clinical settings. Among these methods angiography is by far the most reliable and reasonably accurate. Analysis of the dimension of the left ventricle has been directed at defining the ventricular performance within the classical framework of the length-tension relation and which involves calculation of the ventricular volume, mass, wall

stress in relation of Laplace's law, ejection characteristics of the ventricle, compliance of the chamber and muscle, and regional wall motion abnormalities. Knowledge of the interrelationship among various hemodynamic and angiographic variables provides details which are vital to the understanding of the circulatory status in respective diseases. This paper represents a review of the usefulness and limitation of quantitative analysis of left ventricular dimension by angiographic techniques. Also discussed are some of the potential areas for advancement of automated processing of the image in which a vast amount of work required for frame by frame analysis of ventricular dimension can be reduced and the accuracy in calculation of variables can

Key Words:

Cineventriculography
Left ventricular dimension
Left ventricular ejection
Automated image processing
Regional wall motion

The Third Division, Department of Internal Medicine, Kyoto University, Kyoto, Japan

This project was supported by Scientific Research Grant 444044 from the Ministry of Education, Science and Culture, Japan.

Address for correspondence: Shigetake Sasayama, M.D., The Third Division, Department of Internal Medicine, Kyoto University Hospital, Sakyo-ku Kyoto 606, Japan

Fig.1. Stroke volume calculated by left ventriculography compared with stroke volume obtained by the Fick method. Close agreement is seen in the two values.

be further enhanced.

Left Ventricular Volume

The left ventricular volume was first measured by radiographic techniques in humans in 1956 by Dodge and Tannenbaum,¹ and Soloff et al.² Subsequent to such pioneering work, Arvidson,³ Chapman et al.⁴ and Dodge et al.⁵ have established clinical methods which have now been widely used in every institute. Though the initial studies were performed using large cut films exposed in 6 frames/sec, currently single or biplane cineangiography with filming speeds of 30 to 60 frames/sec is more frequently used for quantitative analysis of the left ventricular dimension.

Most often, the contrast material is selectively injected into the left ventricular cavity. However, in early days, levophase cineangiograms with injection of dye into the right side of the heart were used. Ferlinz et al.⁶ pointed out the poor correlation of the two methods as well as significant difference between the two stroke volumes and the two ejection fractions, and suggested that levophase cineangiograms significantly underestimated the left ventricular ejection fraction when compared to standard (selective) left ventricular cineangiography. On the other hand, Krohn⁷ objected to this view and reported that there was no difference of clinical or statistical significance in the contractile measures obtained

Fig.2. Pressure-volume diagrams in one patient with coronary artery disease. Solid line represents the control state and the dashed line the loop during heart attack. Both loops rotate counterclockwise during ventricular systole with a small clockwise loop during atrial systole. During the attack, systolic volume reduction is markedly reduced, being followed by immediate volume expansion late in systole as ventricular pressure falls. The clockwise loop with atrial contraction is augmented with angina. Shown in the right upper corner are the superimposed end-diastolic and end-systolic left ventricular cavities in the both states.

with either method, and that two types of left ventriculograms could be used interchangeably. It has also been demonstrated that selective left ventriculography leads to progressive enlargement of left ventricular volume and prompt reduction in myocardial contractile force during the later cardiac stages of the angiogram, possibly due to a pharmacologic effect of contrast medium on the myocardium. Thus, it has been suggested that the dynamic analysis of selective cineventriculography should be derived from the first few cycles after opacification⁸

The left ventricle can be represented by a prolate ellipsoid which consists of a major axis and two minor axes. The latter was calculated using the directly measured major semi axis and the area of an assumed ellipse.^{3,5} Ventricular volumes then may be calculated by use of formula for an ellipsoid of revolution. The stroke volume

Fig.3. Calculation of left ventricular mass in patients with hypertrophic cardiomyopathy. On the right anterior oblique view of the end-diastolic silhouette, the lateral wall thickness can be readily identified. The septal thickness is then estimated with correction of free wall thickness by the echocardiographic septal/posterior wall thickness ratio (outlined by dashed line in the left panel). The left ventricular mass is represented as a bowl-shaped shell with opening at the aortic and mitral valve plane. The calculation of both chamber and total volumes is provided by Simpson's rule method. The left ventricular mass is obtained by the difference of the total and chamber volume multiplied by the specific gravity of the heart muscle.

can be calculated by subtracting the end-systolic from the end-diastolic volume.⁹ The statistical correction of calculated volumes has now been firmly established. The ratio of the stroke volume to the end-diastolic volume (ejection fraction) is considered to provide a measure of the myocardial contractility.¹⁰ The accuracy of angiographic measurements of left ventricular volume has been verified by comparing the stroke volume determined by the Fick principle to that obtained from angiographic methods in patients without regurgitant valvular disease (Fig. 1). Thus, the difference between the two measurements represents the regurgitant or shunt flow per beat. In certain disorders, such as hypertrophic cardiomyopathy in which the left ventricular chamber has a highly irregular shape, Simpson's rule is preferably used to compute volume.⁴

Pressure-Volume Relation

By relating instantaneous changes in left ventricular volume to the pressure throughout one cardiac cycle, a loop can be constructed

encompassing systole and diastole which provides additional information concerning the functional geometry of ventricular contraction. The mechanical work of the left ventricle can be determined from the planimetered area within the pressure-volume loop.^{11,12}

The systolic pressure and left ventricular volume at the end of ejection are independent of the end-diastolic volume and duration of ejection, and this relation approximates the active pressure-volume curve which can be generated when the ventricle is forced to contract isovolumetrically at a given diastolic volume. As in isolated muscle, this active pressure-volume curve provides the limit for the end-systolic volume of ejecting contractions.^{13,14} Thus, the end-systolic pressure-volume relation is shifted upward and to the left or downward and to the right with concordant changes in the inotropic state and may prove to be a useful addition for better measures of contractility.^{15,16} Figure 2 shows the pressure-volume loops in one patient with variant angina in the control state (solid line) and during anginal attack induced by ergonovine (dashed line). During angina, the wall motion of the anterolateral to apical portion is severely depressed and ejection fraction is decreased from 79 to 53% despite an increase in end-diastolic volume. The end-systolic pressure-volume relation during attack is shifted to the right of the control.

The curved diastolic relationship of the pressure-volume loops is analyzed to determine compliance of the ventricle.^{17,19}

Left Ventricular Mass

The thickness of the left ventricular free wall can be measured at the wall segment slightly below the equator. A uniform wall thickness is assumed to surround the left ventricular chamber. The average wall thickness is added to the major and minor axes to calculate the volume of the chamber plus wall. Subtraction of the chamber volume from this total volume provides the shell volume of the left ventricular myocardium. The wall volume multiplied by the specific gravity of heart muscle (1.050) equals left ventricular mass.²⁰ Close agreement was evident between the angiographical calculations during life and the actual left ventricular mass determined at autopsy.²¹

However, in cases of localized hypertrophy of the left ventricular wall such as asymmetric septal hypertrophy, apical hypertrophy or hypertrophic cardiomyopathy with midventricular

Fig.4. A gradient image of the digitized left ventricular silhouette obtained by the spatial differentiation of gray levels. The left ventricular boundary is traced along the maximum value of spatial derivatives. The area of search enclosed within the window is determined manually by the operator and the starting point of the automatic edge tracing shown by the horizontal line is given by cursor. For details, see text. (Reproduced from *Circulation*²⁹ by permission)

obstruction, the shape of the ventricle is sufficiently different from the usual geometry reference figures of the prolate ellipsoid. Under these circumstances, we employ Simpson's rule method for determination of the chamber and myocardial shell volumes. The regional wall thickness which is not completely visualized by angiography can be corrected by the data obtained from two dimensional echocardiography (Fig. 3).

Left Ventricular Wall Forces

The forces acting within and upon the left ventricular wall are function of chamber dimensions, wall thickness and pressure. A force per unit of cross sectional area of the ventricular wall is termed wall stress. Sandler and Dodge²³ described an angiographic method for calculating mean wall stress acting within the left ventricle, on the basis of the Laplace relation modified to incorporate wall thickness. For this calculation, the left ventricle is assumed to be represented as an ellipsoid with a relatively thin wall. Hood et al²³ pointed out that the calculation of wall

stress using a thin walled ellipsoidal models overestimates the mean stress determined from the thick walled ellipsoidal model of Wong and Rautaharju²⁴ by 10% throughout the cardiac cycle. Subsequent investigators described more complicated models insisting that some reference figures may be more appropriate than others. However, Burns et al²⁵ compared directly measured left ventricular wall stress with a calibrated transmural auxotonic strain gauge and stress calculated from geometric reference figures. Their studies have shown that there are relatively minor differences between directly measured myocardial wall stress and wall stress calculated under the assumption of relatively simple geometric reference figures, a sphere or ellipsoid, and thin wall theory for the basis of the calculation.

Left ventricular wall stress is an important determinant of cardiac function of the myocardial level, and in chronic valvular heart disease with large changes in chamber size and wall thickness, calculation of systolic wall stress is desirable for an analysis of ventricular function

Fig.5. Left ventricular volume calculated manually (cross) plotted with respect to time of exposure and automated time-volume curve, in the same patient. (Reproduced from Am J Cardiol³⁰ by permission)

within the force-velocity-length framework²⁶⁻²⁸

Application of Computer Technology for Analysis of Angiogram

In order to reduce tedious and laborious tasks involved in the frame by frame analysis of consecutive cine film and to expedite analysis without observers' variation, we developed an automatic processing system using minicomputers.^{29,30} The left ventricular images on each cine film were scanned by a flying spot scanner and stored on magnetic disc which were then converted into 128 × 128 pixels (8 bit words) with gray levels of 256 values. The spatial derivatives of the gray levels were then obtained and the left

ventricular boundaries were automatically determined using a heuristic method to search for the local maximum gradient values. The algorithm of the boundary tracing consists of two major weight coefficients for direction and depth of search. The former enables avoidance of an abrupt change in the direction of edge tracing and the latter is to refer to the sequential information of several remote edge points. In addition, the boundary of the preceding frame is always referred to as a global guidance. These border algorithm closely imitates a visual detection of a boundary by the human eye^{29,30} (Fig. 4)

When the entire edge was detected, the position of the aortic valve was determined at the area where the separation of the segments across the outflow tract of the left ventricle is the shortest. The apex is determined at the point with the longest distance from the midpoint of the aortic valve.

The left ventricular volumes (V) were calculated by a modification of Kennedy's formula:

$$V = 0.687 \times C^3 \times A^2 / L + 1.9 \text{ ml}$$

where A is the area of the ventricle, calculated from the amount of pixels surrounded by the left ventricular boundary; L is the longest measured length between the midpoint of aortic valve and the apex; C is the linear correction factor for the magnification of a unit of length (one pixel), which was derived from the comparison to the known area of 1 cm² grid filmed in parallel to the tube at the position of the heart.

Figure 5 shows the conventionally determined left ventricular volumes plotted with respect to time of exposure and superimposed on the

Fig.6. Original digitized image of the left ventricular end-diastolic silhouette obtained with 5 ml of the contrast material (A). The reference image for subtraction (B). The extracted left ventricular image using the subtraction technique (C). (Reproduced from Am J Cardiol³⁰ by permission)

Fig. 7. Left ventricular volume curve obtained in the same patient with small doses of contrast material (left) and with conventional large doses (right). (Reproduced from *Am J Cardiol*³⁰ by permission)

automated time volume curve of the same patient. As there was close agreement in these two values, our computerized method of volume calculation proved to be valid.³⁰

Subtraction Method for Left Ventriculogram with Minimal Doses of Contrast Medium

For the dimensional analysis of the left ventricular cavity, conventional methods of cine-angiography necessitate the injection of large amounts of dye. However, the intracardiac injection of contrast medium has been shown to cause profound hemodynamic changes.³¹ To minimize these untoward effects of contrast medium, we obtained left ventriculograms using only 5 ml of the dye. All the consecutive images containing dye and the corresponding reference images were transferred to a computer, as described above. Then, the digitized ventricular images were subtracted by the reference image to eliminate any irrelevant background and enhance only the object containing the dye. The extracted ventricular image has sufficient contrast to allow for subsequent computer processing for automatic boundary detection³⁰ (Fig. 6).

Using this small dose of dye, the elevation of the left ventricular end-diastolic pressure which consistently occurred one to 3 min after injection of the conventional large doses, could be avoided. Values for dimensional and contractile measures calculated from the small doses angiogram were similar to those obtained when large doses were used (Fig. 7).

Thus, the technique described permits to achieve precise detection of boundary of cardiac chamber with even less dye and circulatory

disturbances.

Quantitative Assessment of Regional Wall Motion

Although the development of systolic bulge following ligation of the coronary artery supplying that area has been known since the original description of Tennant and Wiggers,³³ it has not been until recent years that an assessment of similar regional myocardial phenomenon became possible in clinical settings.

An important consideration in precise identification of localized myocardial dysfunction resulting from coronary artery disease is the reference system employed. First, it is necessary to achieve proper superimposition of sequential ventricular images. The reference systems currently used for this purpose are 1) the long axis of the left ventricle from the midpoint of the aortic valve to the apex and its bisecting perpendicular,³⁴ 2) long axis of the left ventricle and the midpoint of the aortic valve,³⁵ and 3) fixed external reference markers^{29,30}. Of these three reference systems, the regional wall motion abnormalities have been shown to be most accurately represented as predicted by the location of myocardial infarction by a reference system employing fixed external markers (Fig. 8).

When attempting to quantify the extent of the regional wall motion abnormalities, the fixing of a reference point to which the inward wall motion can be related is more controversial. To evaluate regional wall motion, three models have been considered: 1) radial system, 2) chord system and 3) area system. Karsch et al.³⁷ compared the three quantitative methods in the 30° right anterior oblique and 60° left anterior

Fig.8. Three common reference systems for determining left ventricular wall motion disturbance are compared. Upper rows show superimposed end-diastolic and end-systolic silhouettes in which 128 radial grids are drawn from the center of gravity of the end-diastolic image. Lower rows show the percent shortening of each radial grid over the entire ventricular perimeter. The reference systems employed for superimposition are 1) fixed external reference markers (left panel), 2) midpoint left ventricular long axis (middle panel) and 3) midpoint aortic valve plane (right panel).

A: In a patient with total occlusion of left anterior descending artery at segment 6, a reference system employing external markers displays apical dyskinesia and anterior wall motion abnormalities which were not detected by two other methods.

B: An example of wall motion analysis by the three methods in a patient with total occlusion of the right coronary artery at segment 1. Inferior wall dyskinesia is displayed only by the method with external reference markers.

Fig.9. Effect of rapid cardiac pacing in one patient with three vessel disease.

A: superimposed end-diastolic and end-systolic ventricular silhouettes, 128 radial grids are drawn from the center of gravity of the end-diastolic image. (left, control state; right, post-pacing state)

B: length-time plots of three specific grid lines. (left, control; right, post-pacing state)

C: percent shortening plotted over entire ventricular perimeter (dotted line, control state; solid line, post-pacing state)

D: three dimensional display of wall motion by stacking length-time plot of each radial length over the entire ventricular perimeter (left, control state; right, post-pacing state). (Reproduced from *Circulation*²⁹ by permission)

oblique projections and concluded that the radial method provided best separation of normal and abnormal regions and best reflected symmetric uniform motion of the ventricular silhouette with intact coronary arteries. On the other hand, Gelberg et al.³⁸ reported that area method was the most accurate in the assessment of local wall motion abnormalities. However, in this area system, the ventricular silhouette was divided into 5 areas in the right anterior oblique view and 3 areas in the left anterior oblique view. Therefore, though the wall motion disturbance in each area could be represented by the abnormality of systolic reduction of the respective area, such large grouping of segments is not likely to sufficiently characterize the extent of abnormal wall motion.

It has been widely accepted that the direction

of ventricular wall motion is much closer to natural behavior for all areas, if it is expressed along radial lines. In our study, the sequential ventricular silhouettes automatically determined were superimposed by using external reference markers. The geometric center of end-diastolic silhouette was chosen as the fixed reference point to which concentric wall motion was related. The position of center of gravity was examined in each ventricular silhouette in 5 normal subjects and its spatial movement proved to be minimal. If there is asynchronous contraction, this point will shift towards that area.²⁹

One hundred and twenty-eight radial grids were then drawn from the center of gravity of the end-diastolic silhouette to the margin of the ventricular cavity (Figs. 2, 8 and 9). Measurement of the length of each grid line throughout the

cardiac cycles provided a quantitative description of the segmental centripetal motion over the entire circumference. Figure 9-B reproduces the instantaneous length of the specific segment plotted against time, which allowed for characterization of contraction and relaxation of the regional myocardium, as shown in animal experiments with ultrasonic dimension gauges.^{39,40} Stroke excursion of each radial grid from end-diastole to end-systole corrected by end-diastolic length was expressed as percent shortening. The segmental wall motion expressed as percent shortening was plotted over the entire circumference (Fig. 9-C). Such a computer aided multi-axis approach allowed for more precise quantification of the extent and magnitude of regional wall motion disturbances.²⁹

It has widely been accepted that it depends on the size of abnormally contracting segments whether the patient manifests symptoms of left ventricular failure, however, hypokinesia defined by the ventriculogram does not always represent myocardial scar.^{41,42} Therefore, an assessment of additional response to cardiac stress as rapid pacing is often required to evaluate whether these abnormalities are permanent or are dynamic and potentially subjected to ischemia during a stress state.^{29,43,44} Figure 9 shows the representative changes in ventriculographic patterns induced by atrial pacing. The patient was a 50 year old man with previous history of myocardial infarction. Coronary arteriogram 2 months after an acute episode revealed complete obstruction of the right coronary artery and 75% stenosis of both left anterior descending and circumflex branches. The upper left panel(A) illustrates the superimposed end-diastolic and end-systolic left ventricular images. The control state is shown on the left and post pacing state on the right. The upper right panel(B) is the plot of instantaneous segment length at three specific grid lines on the anterior(1), apical(2) and inferior wall(3). In the control state(left panel), the magnitude of excursion is markedly depressed in the inferior wall, while active shortening is maintained in the other two segments. After termination of pacing, the end-diastolic dimension is increased by 10% in the anterior and apical segments and by 28% in the inferior wall segment. Percent shortening decreased from 42 to 24% in the anterior wall and from 59 to 40% at the apex. The inferior wall became completely akinetic. The lower left panel(C) shows percent shortening on each grid line over the entire ven-

tricle, control shortening by the dotted line and the post pacing shortening by the solid line. The vertical axis shows the percent shortening and the horizontal axis represents areas from the anterior to the inferior wall. The wall motion is depressed in the entire myocardium after pacing. Actually, active contraction is replaced by passive expansion during systole in the inferobasal area. The lower right panel(D) is the three dimensional display by stacking serial plots of wall motion against time for each radial grid. On the horizontal axis is shown the time of the cardiac cycle, on the vertical axis is the length of each grid line, and the third axis at right angles to both axes represents the area of the left ventricle; proximal parts correspond to the anterior wall and distal parts to the inferior wall (left, control state; right, post pacing state). This presentation enabled assessment of the size of the area which involved the disturbed wall motion.

Thus, it is concluded that in this patient, active contraction had been depressed over 25% of the total circumference in the inferobasal area, while normal contraction was present at rest in other portions of the ventricular wall, however, significant hypokinesia was induced over the entire wall by the temporary ischemia provoked by increasing the oxygen demand beyond the coronary reserve which had been critically limited.

From a practical standpoint, the size of the abnormal wall motion is an important determinant of heart failure and prolonged ischemic stress in such patients may easily lead to acute left ventricular failure. Alternatively, reduction in oxygen requirements may serve to improve myocardial failure. An assessment of the potential ischemia of the ventricular wall may be crucial for determining indications for bypass surgery.

Acknowledgement

The authors are in debt to Drs Michiyoshi Kuwahara and Shigeru Eiho, Automation Research Laboratory, Kyoto University, for their cooperation in data processing. We also thank M. Ohara for her help in preparation of this manuscript.

REFERENCES

1. DODGE HT, TANNENBAUM HL: Left ventricular volume in normal man and alterations with disease. *Circulation* 14: 927, 1956
2. SOLOFF LA, ZATUCHNI J, STAUFFER H: Angiocardiographic observations of intracardiac flow in the normal and in mitral stenosis. *Circula-*

- tion 13: 334, 1956
3. ARVIDSSON H: Angiographic determination of left ventricular volume. *Acta Radiol* 56: 321, 1961
 4. CHAPMAN CB, BAKER O, REYNOLDS J, BONTE FJ: Use of biplane cinefluorography for measurement of ventricular volume. *Circulation* 18: 1105, 1958
 5. DODGE HT, SANDLER H, BALLEW DW, LORD JD Jr: The use of biplane angiocardiology for the measurement of left ventricular volume in man. *Am Heart J* 60: 762, 1960
 6. FERLINZ J, HERMAN MV, COHN PF, GORLIN R: Comparison of selective left ventriculograms with levophase ("forward") ventriculograms in patients with coronary artery disease. *Am Heart J* 91: 721, 1976
 7. KROHN BG: Interchangeability of left ventriculograms. *Am Heart J* 93: 130, 1977
 8. CARLETON RA: Change in left ventricular volume during angiography. *Am J Cardiol* 27: 460, 1971
 9. DODGE HT, HAY RE, SANDLER H: An angiographic method for directly determining left ventricular stroke volume in man. *Circ Res* 11: 739, 1962
 10. BARTLE SH, SANMARCO ME, DAMMANN JF Jr: Ejection fraction: An index of myocardial function. *Am J Cardiol* 15: 125, 1965
 11. RACKLEY CE, BEHAR VS, WHALEN RE, McINTOSH HD: Biplane cineangiographic determinations of left ventricular function: Pressure-volume relationships. *Am Heart J* 74: 766, 1967
 12. BUNNEL IL, GRANT C, GREENE DG: Left ventricular function derived from the pressure volume diagram. *Am J Med* 39: 881, 1965
 13. DOWNING SE, SONNENBLICK EH: Cardiac muscle mechanics and ventricular performance, force and time parameters. *Am J Physiol* 207: 705, 1964
 14. SONNENBLICK EH: Instantaneous force-velocity-length determinants in the contraction of heart muscle. *Circ Res* 16: 441, 1965
 15. GROSSMAN W, BRAUNWALD E, MANN T, McLAULIN LP, GREEN LH: Contractile state of the left ventricle in man as evaluated from end-systolic pressure-volume relations. *Circulation* 56: 845, 1977
 16. SASAYAMA S, KOTOURA H: Echocardiographic approach for the clinical assessment of left ventricular function: The analysis of end-systolic pressure (wall stress)-diameter relation and force-velocity relation of ejecting ventricle. *Jpn Circul J* 43: 357, 1979
 17. DODGE HT, HAY RE, SANDLER H: Pressure-volume characteristics of the diastolic left ventricle in man with heart disease. *Am Heart J* 64: 503, 1962
 18. MIRSKY I, COHN PF, LEVINE JA, GORLIN R, HERMAN MW, KREULEN TH, SONNENBLICK EH: Assessment of left ventricular stiffness in primary myocardial disease and coronary artery disease. *Circulation* 50: 128, 1974
 19. GROSSMAN W, STEFADOUROS MA, McLAURIN LP, ROLETT EL, YOUNG DT: Quantitative assessment of left ventricular diastolic stiffness in man. *Circulation* 47: 567, 1973
 20. RACKLEY CE, DODGE HT, COBLE YD Jr, HAY RE: A method for determining left ventricular mass in man. *Circulation* 29: 666, 1964
 21. KENNEDY JW, REICHENBACH DD, BAXLEY WA, DODGE HT: Left ventricular mass: A comparison of angiocardiology measurements with autopsy weight. *Am J Cardiol* 19: 221, 1967
 22. SANDLER H, DODGE HT: Left ventricular tension and stress in man. *Circ Res* 13: 91, 1963
 23. HOOD WP, THOMSON WJ, RACKLEY CE, ROLETT EL: Comparison of calculations of left ventricular wall stress in man from thin-walled and thick-walled ellipsoidal models. *Circ Res* 24: 575, 1969
 24. WONG AYK, RAUTAHARJU PM: Stress distribution within the left ventricular wall approximated as a thick ellipsoidal shell. *Am Heart J* 75: 649, 1968
 25. BURNS JW, COVELL JW, MYERS R, ROSS J Jr: Comparison of directly measured left ventricular wall stress and stress calculated from geometric reference figures. *Circ Res* 28: 611, 1971
 26. SASAYAMA S, ROSS J Jr, FRANKLIN D, BLOOR CM, BISHOP S, DILLEY RB: Adaptations of the left ventricle to chronic pressure overload. *Circ Res* 38: 172, 1976
 27. SASAYAMA S, FRANKLIN D, ROSS J Jr: Hyperfunction with normal inotropic state of the hypertrophied left ventricle. *Am J Physiol* 232: H418, 1977
 28. TAKAHASHI M, SASAYAMA S, KAWAI C, KOTOURA H: Contractile performance of the hypertrophied ventricle in patients with systemic hypertension. *Circulation* 62: 116, 1980
 29. FUJITA M, SASAYAMA S, KAWAI C, EIHO S, KUWAHARA M: Automated processing of cine-ventriculograms for analysis of regional myocardial function. *Circulation* 63: 1065, 1981
 30. SASAYAMA S, NONOGI H, KAWAI C, FUJITA M, EIHO S, KUWAHARA M: An automated method for left ventricular volume measurement from the cineventriculography with minimal doses of contrast medium. *Am J Cardiol* 48: 746, 1981
 31. FRIESINGER GC, SCHAFER J, CRILEY JM, GAIRTNER RA, ROSS RS: Hemodynamic consequences of the injection of radiopaque material. *Circulation* 31: 730, 1965
 32. KRUGER RA, MISTRETTA CA, HOUK TL, KUBAL W, RIEDERER SJ, ERGUN DL, SHAW CG, LANCASTER JC, ROWE GG: Computerized fluoroscopy techniques for intravenous study of cardiac chamber dynamics. *Invest Radiol* 14: 279, 1979
 33. TENNANT R, WIGGERS CJ: The effect of coronary occlusion on myocardial contraction. *Am J Physiol* 112: 351, 1935
 34. HAMILTON GW, MURRAY JA, KENNEDY JW: Quantitative angiocardiology in ischemic heart disease. *Circulation* 45: 1065, 1972
 35. HERMAN MV, HEINLE RA, KLEIN MD, GORLIN R: Localized disorders in myocardial contraction: Asynergy and its role in congestive heart failure. *N Engl J Med* 277: 222, 1967
 36. CHAITMAN BR, BRISTOW JD, RAHIMTOOLA SH: Left ventricular wall motion assessed by using

- fixed external reference systems. *Circulation* **48**: 1043, 1973
37. KARSCH KR, LAMM U, BLANKE H, RENTROP KP: Comparison of nineteen quantitative models for assessment of localized left ventricular wall motion abnormalities. *Clin Cardiol* **3**: 123, 1980
38. GELBERG HJ, BRUNDAGE BH, GLANTS S, PARMLEY WW: Quantitative left ventricular wall motion analysis: A comparison of area, chord and radial methods. *Circulation* **59**: 991, 1979
39. THEROUX P, ROSS J Jr, FRANKLIN D, KEMPER WS, SASAYAMA S: Regional myocardial function in the conscious dog during acute coronary occlusion and responses to morphine, propranolol, nitroglycerin, and lidocaine. *Circulation* **53**: 302, 1976
40. SASAYAMA S, FRANKLIN D, ROSS J Jr, KEMPER WS, McKOWN D: Dynamic changes in left ventricular wall thickness and their use in analyzing cardiac function in the conscious dog. *Am J Cardiol* **38**: 870, 1976
41. BODENHEIMER MM, BANKA VS, TROUT RG, HERMANN GA, PASDAR H, HELFANT RH: Local characteristics of the normal and asynergic left ventricle in man. *Am J Med* **61**: 650, 1976
42. HUTCHINS GM, BULKLEY BH, RIDOLFI RL, GRIFFITH LSC, LOHR FT, PIASIO MA: Correlation of coronary arteriograms and left ventriculograms with postmortem studies. *Circulation* **56**: 32, 1977
43. DWYER EM Jr: Left ventricular pressure-volume alterations and regional disorders of contraction during myocardial ischemia induced by atrial pacing. *Circulation* **42**: 1111, 1970
44. PASTERNAK A, GORLIN R, SONNENBLICK EH, HAFT JJ, KEMP HG: Abnormalities of ventricular motion induced by atrial pacing in coronary artery disease. *Circulation* **45**: 1195, 1972