Heart motion video tracking¹

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Heart motion video tracking is an atraumatic technique for analysing left ventricular wall abnormalities. The borders of the left ventricular silhouette as seen under an image intensifier are tracked at a number of points using two electronic markers, and the procedure is carried out in the posteroanterior, and right and left anterior oblique positions. This movement is converted into an analogue signal which is displayed linearly on an oscilloscope screen and permanently recorded onto multichannel paper. Alternatively the image can be recorded on videotape for subsequent analysis. The apparatus has been calibrated so that direct measurements of the amplitude and velocity of the cardiac movement can be made. Studies were made of 61 patients. The range of amplitude (2.0 mm to 10.5 mm) and velocity (3.5 to 77 mm/sec) of movement were determined on 13 normal subjects. In the remaining 48 patients, 11 had had a recent acute myocardial infarction, 30 had chronic coronary artery disease, and 7 had idiopathic cardiomyopathy. Abnormal tracings were found in 29 of these patients: 19 dyskinesia, 8 akinesia, and 20 asynergies or hypokinesia.

The apparatus has sufficient resolution to detect minor changes in left ventricular wall movements and can clearly show dyskinesia not apparent to the naked eye on direct vision of the television screen. It is concluded that heart motion video tracking is a valuable atraumatic technique for analysing left ventricular wall movements and has potentially many uses in cardiology.

In recent years several noninvasive techniques have been developed to assess left ventricular function. Unfortunately, most of these techniques are limited in value, and direct cardiovascular haemodynamic measurements and cineangiography still provide the most accurate means of assessment. Invasive techniques have a measurable risk and cannot be readily repeated to demonstrate serial changes (Sonnenblick, 1971; Rapaport and Scheinman, 1969). However, angiography has shown that localized abnormalities of left ventricular wall motion commonly follow impairment or obstruction of coronary blood flow, and this may impair the heart's function as a pump (Sonnenblick, 1971).

With the introduction of fluoroscopy using image intensification, closed circuit television monitors, and video tape recorders, dyskinetic movements of the left ventricular wall can now be observed directly. Accurate analysis of these movements, however, depends on ventricular cineangiography. Attempts by atraumatic means to assess left ventricular move-

ment have included roentgenokymography (Master *et al.*, 1940), electrokymography (Dack, 1955; Schwedel, Samet, and Mednick, 1950), kinetocardiography (Suh and Eddleman, 1959), and apex cardiography (Lane *et al.*, 1968), but none has gained popularity. New techniques recently introduced include echocardiography (Wharton, Smithen, and Sowton, 1971) and radioisotope studies (Zaret *et al.*, 1971). The value of these techniques has yet to be determined.

Heart motion video tracking is another atraumatic technique described by Schuette and Simon (1968), and used by Cohen *et al.* (1968) and Kazamias *et al.* (1971). The principle involves the direct tracking of the movements of the cardiovascular structures as seen under an image intensifier and projected onto closed circuit television monitors. The movements are detected by a radar loop tracking system (radarkymography) and then translated into reproducible linear graphic tracings. Only one point on the cardiac border can be tracked at a time, and analysis is performed by reference to an electrocardiogram taken simultaneously.

We have developed a heart motion video tracking machine which analyses the movements of the cardiovascular structures by a different technique

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but which are reproduced as linear graphic tracings in a similar way. Two areas can be tracked at the same time and compared directly from the oscilloscope. Tracking can be performed directly or from video tape recordings.

This report presents our experiences with this technique. The machine has been calibrated and the amplitude and velocity of recordings from the same area can be compared in a serial manner.

Methods

Heart motion video tracking needs only the video waveform from the image intensifier television chain in order to track two selected points on the cardiac or aortic border. It then produces an analogue output signal representing the horizontal movement of each of these selected points. In order to select the two points of interest, a vertical white cursor or reference line is superimposed on the television image. This is achieved by suitable counting circuits in the video tracker which use both the horizontal and vertical TV synchronous pulses. This cursor line is placed just to the left of the cardiac border and in this position the top of the line indicates one point on the cardiac border to be tracked while the bottom indicates the other (Fig. 1). The upper and lower extremities of the cursor are independently adjustable in the vertical axis, as well as in the horizontal position.

FIG. 1 Closed circuit television screen showing the cardiac silhouette in the posteroanterior position. To the left of the left ventricular border a white cursor is superimposed electronically onto the TV screen. The top and bottom of the cursor represent the areas on the cardiac border to be tracked. At the border the dark to light transition is detected by two small black markers which become 'locked on' to the border and follow its movement. This movement is then converted to an analogue output voltage which gives a linear tracing.

At the border of the cardiac silhouette, a dark (cardiac) to light (lung fields) transition indicates a voltage change in the video waveform and when detected the horizontal movement of the cardiac border is followed by a small black marker covering four lines of the video waveform, which becomes locked onto this point. The movement of the marker is converted to an analogue output voltage which gives a linear tracing of the cardiac border movement. Two such areas of tracking can be performed simultaneously and displayed with the patient's electrocardiograph on an oscilloscope and then recorded onto multichannel paper. The process can be reversed, that is, black cursor and white markers, if a white-to-black transition is required, as in tracking the posterior wall of the heart in the right anterior oblique position.

Method of calibration

Calibration of the heart motion video tracking machine involves two planes of measurement.

METHOD OF CALIBRATION

FIG. 2 Method of calibration. A lead template placed under the image intensifier (II) has two small reference notches exactly 1 cm apart cut out of its upper surface. On the right border of the template the markers are 'locked on' to the transition zone. By a system built into the tracker the white cursor is made to move a fixed distance which corresponds to the reference notches. A square wave shown below produced by the movement of the cursor appears on the recording paper. (For detail - see text.)

A) Horizontal movement of heart Because of the nonparallel x-ray path, the relative distances of the patient to the image intensifier must be standardized to take account of magnification effects. In horizontal calibration a small lead template, as shown in Fig. 2, is placed under the image intensifier at a height above the table corresponding to the average midchest position of a patient. Two small reference notches 1 cm apart are cut out of the template and are visible under the intensifier. The black markers are made to trigger on the edge of the template silhouette and the white cursor is positioned directly under the right hand reference notch. On pushing the 'calibration' button on the video-tracker the cursor moves to the left. Exactly 1 cm of movement occurs when the white cursor lies directly below the left-sided notch on the template. This is achieved for a given height of the image intensifier by adjustment of the internal calibration circuit. This elevation is marked on the supporting arm of the intensifier as position '1.0 calibration'. A square wavepulse produced by the movement of the cursor appears on the recording paper. The amplitude of this square wave is adjusted to some convenient deflection, for example 2.5 or 5 cm of square wave movement on the recorded are equal to 1 cm of actual cardiac wall movement.

B) Vertical level of image intensifier In almost all patients in most positions screened the optimum image intensifier position is at the '1.0 calibration position'. Very occasionally because of extreme obesity the height of the image intensifier lies above the '1.0 calibration position'. Thus '0.9 calibration' and '0.8 calibration' positions have been determined in a similar way to that already described. The fixed movement of the cursor will lie between two notches set 0.9 and 0.8 cm apart on the template. The height of the image intensifier representing these two positions is permanently marked on its supporting arm.

Measurement of overall frequency response of heart motion video tracking

An artificial sine wave was generated by means of an eccentric wheel rotated at varying speeds and televised onto a closed circuit television screen. The movements of the wheel were tracked in the conventional manner and the resultant sine wave frequency and amplitude measured. The usable band width was from 0.03 to 16 Hz.

Output filter

Using output filtering with an upper cut-off frequency of 20 Hz, there was considerable artefact due to quantal noise from the image intensifier tube, and this affected the triggering of the markers on the cardiac border. By reducing the cut-off frequency of the filter to 14 Hz on the lower marker and leaving the original filter on the upper marker, these two outputs were compared, tracking the same point on the cardiac border. There was less than 2 per cent variation in both amplitude and velocity of movement while the artefact was considerably reduced in the lower marker. As a result a filter with a 14 Hz cut-off frequency has been used.

Analysis of recordings

Fig. 3 shows normal tracings from the upper left ventricle and apex in the posteroanterior position. The downward deflection following soon after the QRS represents ventricular systole. At this point the cardiac silhouette and marker are seen to move inwards. During diastole the outward movement of the heart is seen as an upward deflection on the tracing. Immediately before systole, some changes in the shape of the upward deflection are usually seen.

Movements at the apex of the heart as compared to the upper left ventricle have a smaller amplitude, and the onset of the downward deflection is delayed.

Fig. 4 compares a tracing taken from the aortic and upper left ventricular borders. Aortic pulsations during ventricular systole are outwards. Consequently the tracking deflection is upwards and then downwards again as blood passes through this vessel to the peripheral arteries.

Amplitude and velocity

Measurements on the tracings include the amplitude and velocity. The *amplitude* (Fig. 5) of the cardiac movement during systole is measured from the peak of the tracing to the trough at the end of the downward deflection. The figure obtained is then converted to true cardiac movement knowing the calibration. Five complexes are measured and averaged. The *velocity* (Fig. 6) during systole is calculated from a line drawn along the major slope of the downward deflection. Again 5 complexes are measured and averaged. Corrections for amplitude

FIG. 3 *Heart motion video tracking. Normal tracings from the upper left ventricle and apex in the posteroanterior position. At the top is the electrocardiogram. Soon after the QRS of the cardiogram there is the downward deflection of the tracings indicating inward movement of the heart during ventricular systole. During diastole the outward movement of the heart is seen as an upward deflection on the tracing, and there are a number of minor changes in the shape of this upward deflection especially towards the end of diastole. The onset of the downward deflection at the apex is delayed compared with the upper left ventricle.*

FIG. 4 Heart motion video tracking. Normal tracings from the aorta and upper left ventricle in the posteroanterior position. At the top is the electrocardiogram. Soon after the QRS there is an upward deflection in the aortic tracing indicating the outward movement of the aorta during systole as it fills with blood. At the same time there is the normal systolic downward deflection of the upper left ventricle.

FIG. 5 Amplitude is measured from the apex of the tracing to the trough at the end of the downward deflection. This value is then corrected for true cardiac movement. The example shown is taken in the posteroanterior position with the electrocardiogram at the top. Below this is the tracing from the upper left ventricle and at the bottom is a tracing from the apex. The amplitude at the apex is less than at the upper left ventricle (see Table).

and velocity are made if the image intensifier position is not '1:0 calibration'.

Procedure

The patient lies on the x-ray table on one small pillow with the electrocardiographic limb leads connected. The tracker calibration is checked so that 1 cm movement of

the cursor line produces the appropriate deflection onto the recording paper. The position to be tracked is then found and the patient is asked to take a small breath. It is essential to avoid a Valsalva manoeuvre. Tracking is then performed immediately to avoid the physiological changes in cardiac function that occur with held inspira-

FIG. 6 Velocity is measured by drawing a tangent to the main downward deflection. The example shown is taken in the posteroanterior position with the electrocardiogram at the top. Below this is the tracing from the upper left ventricle and at the bottom the tracing from the apex. The velocity at the apex is less than at the upper left ventricle (see Table).

FIG. 7 Heart motion video tracking positions. Three main positions are used: posteroanterior (PA) for the aortic and left ventricular (LV) wall, right anterior oblique (RAO) for the anterior wall, and left anterior oblique (LAO). In the left anterior oblique position the posterior wall is best tracked from almost the left lateral position. The apex or low left ventricle is usually best tracked down from a 10° left anterior oblique position, especially if an apical fat pad is present.

tion. The positions tracked are shown diagrammatically in Fig. 7, and listed below.

1) *Posteroanterior aorta, upper left ventricle, high and low mid left ventricle, and low left ventricle.* Tracking of the apex in this position depends on the size of the apical fat pad. Tracking cannot be performed through this pad.

2) *Right anterior oblique* The anterior left ventricular wall is tracked from one to four positions depending on the length of border clearly seen. This position varies from slight right anterior oblique to right lateral (90°). An attempt may be made to track the back wall of the heart (left ventricle) using the black cursor line and white markers.

3) *Left lateral or left anterior oblique* Here the posterior wall of the heart is tracked in one or two positions depending on the position of the diaphragm. We have been unable to track the inferior or diaphragmatic surface, because of the similar density of the diaphragm.

4) *Slight left anterior oblique* The patient is turned to 10° left anterior oblique: this position has been found to be best for tracking the apex of the heart.

Problems

1) **Respiration** Because of chest wall and diaphragmatic movements during respiration, the best recordings are obtained with held inspiration. The patient is instructed to take only a very small inspiration and to suspend respiration, without performing a Valsalva manoeuvre. The tracking curves are recorded immediately. A deep inspiration will result in decreased cardiac movements because of the decreased pulmonary venous return and rotational movements caused by the depression of the diaphragm. To compare tracking recordings in a serial manner the patient must achieve a similar held inspiration at each session. Dyspnoeic patients are therefore difficult to track.

2) **Rib interference** Occasionally the movements of the markers at the cardiac border are disturbed by the shadow of the overlying ribs, resulting in bizarre tracking patterns. The problem is overcome by adjustment of the contrast provided by the image intensifier tube.

3) **Apical fat pad** We have been unable to track the apex of the heart through an enlarged apical fat pad. The left anterior oblique 10° position occasionally overcomes this problem. A similar problem arises with pleural effusions.

4) **Reproducibility** Great care with respiration and the positioning of the patient and cursor will result in tracings which can be compared in a serial manner.

5) **Inferior surface** Though the posterior wall in the left anterior oblique, left lateral, right anterior oblique positions can usually be tracked, we have been unable to follow the movements of the inferior surface of the left

ventricle. This segment lies against the diaphragm, thus preventing a voltage transition from appearing on the screen.

Patients

Sixty-one patients were studied using heart motion video tracking. They were divided into two groups based on a simple clinical assessment.

Group A: Normal Thirteen subjects with an age range of 20 to 39 years, most in their early 20's.

Group B: Abnormal Forty-eight patients with cardiac disease were studied. They were further subdivided.

i) *Acute myocardial infarction* Eleven patients studied within 3 days of the onset of symptoms and the diagnosis proven by clinical history and typical electrocardiographic and serum enzyme changes.

ii) *Chronic coronary artery disease* Thirty patients with a past history of a proven myocardial infarction or diagnosed by selective coronary arteriography, without evidence of a recent acute myocardial infarction.

iii) *Idiopathic cardiomyopathy* Seven patients with clinical and radiographic evidence of an enlarged heart, with evidence of left and/or right ventricular failure without evidence of coronary, hypertensive, or valvular heart disease. Cardiac catheterization and selective coronary angiography showed left ventricular dysfunction only.

Three patterns of abnormal cardiac movements (asynergy) were recognized (Gorlin, Klein, and Sullivan, 1967).

FIG. 8 *Asynergy. Heart motion video tracking tracing from a patient in the posteroanterior position who had recently sustained an anterior myocardial infarction. The electrocardiogram (ECG) is shown above. Below this is the tracing from the upper left ventricle (LV) – amplitude 3.5 mm. At the bottom, the apical tracing shows asynergy (amplitude 2 mm).*

FIG. 9 *Akinesia*. Heart motion video tracking tracing from a patient in the posteroanterior position who had recently sustained an anterior myocardial infarction. The electrocardiogram (ECG) is shown above. Below this is the normal tracing from the upper left ventricle (LV). At the bottom the apical tracing is almost flat and the normal waveform pattern is lost.

a) *Asynergesis* (Fig. 8) The cardiac wall movements were diminished or inadequate, but the direction of movement was normal. This refers to a local disturbance. Hypokinesia was used to describe a generalized reduction in the normal degree of contraction.

FIG. 10 *Dyskinesia*. Heart motion video tracking tracing from a patient in the posteroanterior position who had recently sustained an anterior myocardial infarction. The electrocardiogram (ECG) is shown above. Below this is the normal tracing from the upper left ventricle (LV). At the bottom the apical tracing is dyskinesic; that is, during ventricular systole there is an upward movement which is the opposite to the upper LV and thus the ventricle moves paradoxically. Note, there is no apical delay in dyskinesia, the outward movement beginning during the isovolumetric contraction time.

b) *Akinesia* (Fig. 9) An absence of movement so that the patterns of the heart motion video tracking waves were lost.

c) *Dyskinesia* (Fig. 10) During systole the area of cardiac wall involved bulged outwards in a paradoxical fashion. The heart motion video tracking waves were opposite to the normal pattern, with an upward deflection during systole and a downward deflection during diastole. Such movements were seen in patients with ventricular aneurysms. Dyskinesia was also seen in a number of patients in the early stage of acute myocardial infarction well before typical radiological evidence of aneurysm formation.

Results

Group A: Normal The results of the 13 normal subjects are listed in the Table. Satisfactory recordings were made in the posteroanterior and right anterior oblique positions. It was found easier to track the posterior wall in the left lateral position rather than the left anterior oblique position. Though a number of different points could be tracked on the posterior wall there appeared to be no difference in the movement in this small zone and the results are grouped together. Tracking of the apex of the heart in the 10° left anterior oblique position was only performed in 7 subjects.

Amplitude There was a wide range of movements in all positions: range 2.0 mm to 10.5 mm. Both in the posteroanterior and right anterior oblique positions the movements in the upper left ventricle (posteroanterior 7.3 mm, right anterior oblique 6.0 mm) were greater than the low left ventricle (posteroanterior 5.3 mm, right anterior oblique 3.6 mm). However, the smallest movement in the posteroanterior position was found at the low mid left ventricle (4.5 mm).

Velocity There was a large range in the velocity of movements: 3.5 mm/sec to 90 mm/sec. In the posteroanterior and right anterior oblique positions the movements in the upper left ventricle (posteroanterior 47.8 mm/sec, right anterior oblique 32.3 mm/sec) were greater than the lower left ventricle (posteroanterior 35.3 mm/sec, right anterior oblique 23.1 mm/sec). However, the slowest movement in the posteroanterior position was found in the low mid left ventricle (27.6 mm/sec).

Apical delay (Fig. 3) The delay in the onset of the downward deflection at the low left ventricle compared with the upper left ventricle was measured in the normal subjects.

TABLE Results of amplitude and velocity of heart motion in normal subjects studied by heart motion video tracking

Position	No. of subjects	Amplitude (mm)		Velocity (mm/sec)	
		Mean	Range	Mean	Range
<i>Posteroanterior</i>					
Upper left ventricle	13	7.3	3.1-10.5	47.8	21.8-77.0
High mid left ventricle	13	6.1	2.9-8.6	30.2	18.8-58.0
Low mid left ventricle	13	4.5	2.7-8.0	27.6	6.4-69.0
Low left ventricle (apex)	11	5.3	3.1-7.7	35.3	23.0-52.0
<i>Right anterior oblique anterior surface</i>					
Upper left ventricle	12	6.0	3.3-9.0	32.3	9.4-51.4
Mid left ventricle	5	4.0	2.7-4.8	29.2	23.0-36.0
Low left ventricle	12	3.6	2.0-6.5	23.1	3.5-49.0
<i>Left lateral or left anterior oblique posterior surface</i>					
Posterior surface	13	5.6	2.6-9.8	38.3	15.0-90.0
<i>Left anterior oblique (5°-10°) Apex</i>					
Apex	7	4.9	2.8-6.4	35.0	24.0-58.0

Posteroanterior position average delay, 0.10 sec (n=11): range 0.03 to 0.16 sec.

Right anterior oblique position average delay, 0.15 sec (n=10): range 0.11 to 0.20 sec.

In the posteroanterior position low left ventricle tracking was unsatisfactory in two instances because of an apical fat pad. On three occasions tracking of the low left ventricle wall in the right anterior oblique position was unsatisfactory because of the proximity of the cardiac wall to the sternum.

Group B: abnormal This group was subdivided into those with acute myocardial infarction, those with chronic coronary heart disease, and those with idiopathic cardiomyopathy.

Acute myocardial infarction (11 patients) Five patients had normal tracking which included one patient with a subendocardial myocardial infarction. Five patients had dyskinesia, four on the anterior wall and one on the posterior wall close to the diaphragmatic surface. Two of these patients also had conspicuous asynergies. One patient had an akinetic segment with adjacent asynergies.

Chronic coronary artery disease (30 patients) Eleven patients had normal tracking; 13 had asynergies, 12 had dyskinesia, and 7 had an akinetic segment.

Idiopathic cardiomyopathy (7 patients) Three patients had normal heart motion video tracking and the other 4 had asynergies or hypokinesia. Two patients had an apparent dyskinetic segment close to the apex of the heart.

Discussion

Since Tennant and Wiggers (1935) first showed cardiac wall motion abnormalities after experimental myocardial ischaemia, there have been many attempts to produce equipment to detect and analyse cardiac wall movements. The historical development and value of these machines have been reviewed by Cohen *et al.* (1968) and Kazamias *et al.* (1971). Heart motion video tracking, a relatively new technique, has been found to be an accurate atraumatic means of measuring the amplitude and velocity of cardiac wall motion. The systolic wave of the heart motion video tracking tracing in the normal subject is a steep downward deflection, the velocity of which tapers off towards the end of the contraction (Fig. 3). The diastolic upward deflection has a number of alterations in its shape. The initial rapid upstroke corresponds to the 'rapid filling phase' of the ventricle. The velocity then decreases, 'the slow filling phase', and this is followed by a second rapid upswing resulting from the contribution of atrial systole. Finally, a further upward deflection heralds the onset of ventricular systole. The shape of the diastolic curve is also modified by rotational movements, especially if the patient is tracked during normal respiration.

The terminal upstroke always follows after the onset of the QRS complex of the electrocardiogram and is thus coincident with the initial rise of ventricular systolic pressure during the isovolumetric contraction phase. Rushmer (1956) used variable inductance gauges installed across the left ventricular cavity and variable resistance gauges sutured to or encircling the left ventricle, and showed similar

changes in the diastolic curves to those seen with heart motion video tracking. The terminal outbulging of the heart during the isovolumetric contraction time was found to be associated with shortening of the longitudinal axis of the ventricular chamber. This asynchronous contraction resulted in the lateral walls bulging outward so that the chamber assumed a more spherical configuration.

Calibration of the heart motion video tracker enables patients to be studied in a sequential manner in order to assess changes in wall movements after acute myocardial infarction. Gorlin *et al.* (1967) and Herman *et al.* (1967) have described a mechanistic nomenclature to describe regional cardiac wall motion abnormalities seen on left ventriculography. We have accepted this nomenclature to describe the abnormalities of cardiac movement as seen on heart motion video tracking.

In patients after acute myocardial infarction, Dubnow, Burchell, and Titus (1965) found the postmortem incidence of ventricular aneurysm in patients to be 3.5 per cent, with 80 per cent of the pathological aneurysms lying on the anterior wall of the left ventricle. However, the incidence and site in surviving patients have been dependent on ventriculography. Large anterior aneurysms can be confirmed by chest radiography or fluoroscopy (Baron, 1971). Cheng (1971) analysed the results of left ventriculography in 100 consecutive patients with coronary artery disease and reported the incidence of ventricular aneurysm to be 35 per cent, with 75 per cent of these on the anterior wall or at the apex. Gorlin *et al.* (1967) also found a high percentage of aneurysms (90%) to be anterolateral or apical and thus probably detectable using heart motion video tracking. Aneurysms may be overlooked on viewing a chest radiograph or using fluoroscopy, especially if there is a dilated ventricle resulting in decreased overall cardiac contractility with difficulty in differentiating the abnormal pulsations (Baron, 1971).

Eddleman and Langley (1962) divided ventricular aneurysms into two types.

- 1) True anatomical aneurysm.
- 2) Physiopathological aneurysm with paradoxical or dyskinetic pulsation. The latter may occur immediately after an acute myocardial infarction and be associated with a normal chest radiograph. In some cases fluoroscopic examination will reveal dyskinetic pulsation, but, as identification depends on the skilled eye of the observer, such abnormal areas may be overlooked. Heart motion video tracking allows quantitation in such patients.

Kazamias *et al.* (1971) have shown that heart motion video tracking can be used as a prognostic

index in patients with acute myocardial infarction. In patients with persistent paradoxical pulsations, 29 per cent died within a 6-month follow-up period, compared with 7 per cent in whom no wall motion abnormalities were recorded at all or who lost their paradoxical motion during the follow-up period.

The apparent high incidence of wall motion abnormalities and aneurysms after myocardial infarction, i.e. 35 per cent by ventriculography (Cheng, 1971), 73 per cent by fluoroscopy (Master *et al.*, 1940), and 79 per cent by heart motion video tracking (Kazamias *et al.* 1971), emphasizes the importance of objective diagnosis after infarction. When a patient is being considered for a coronary artery surgical technique such as a saphenous vein graft or an internal mammary artery implant, documentation of left ventricular wall function assumes great importance. The diagnosis and site of an aneurysm known before operation will aid the surgeon in determining what procedures should be performed. In the postoperative phase video tracking is a safe, convenient, and reproducible means of following the progress of the patients and also in assessing the value of the surgical procedure. It can also be used as an atraumatic means of evaluating drugs such as beta-adrenergic blocking agents in both normal subjects and in patients with asynergy. The technique is simple, easy to perform, and causes little inconvenience to the patient. The length of action of these drugs and their effects on wall motion, especially in dyskinetic areas, can now be evaluated.

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References

- Baron, M. G. (1971). Postinfarction aneurysm of the left ventricle. *Circulation*, **43**, 762.
- Cheng, T. O. (1971). Incidence of ventricular aneurysm in coronary artery disease. *American Journal of Medicine*, **50**, 340.
- Cohen, L. S., Simon, A. L., Whitehouse, W. C., Schuette, W. H., and Braunwald, E. (1968). Heart motion video-tracking (radarkymography) in diagnosis of congenital and acquired heart disease. *American Journal of Cardiology*, **22**, 678.
- Dack, S. (1955). The ventricular pulsations in myocardial infarction; a fluoroscopic and kymographic study. *Diseases of the Chest*, **27**, 282.
- Dubnow, M. H., Burchell, H. B., and Titus, J. L. (1965). Postinfarction ventricular aneurysm. *American Heart Journal*, **70**, 753.
- Eddleman, E. E., Jr., and Langley, J. O. (1962). Paradoxical pulsation of the precordium in myocardial infarction and angina pectoris. *American Heart Journal*, **63**, 579.

- Gorlin, R., Klein, M. D., and Sullivan, J. M. (1967). Prospective correlative study of ventricular aneurysm. *American Journal of Medicine*, **42**, 512.
- Herman, M. V., Heinle, R. A., Klein, M. D., and Gorlin, R. (1967). Localized disorders in myocardial contraction. *New England Journal of Medicine*, **277**, 222.
- Kazamias, T. M., Gander, M. P., Ross, J., Jr., and Braunwald, E. (1971). Detection of left-ventricular-wall motion disorders in coronary-artery disease by radarkymography. *New England Journal of Medicine*, **285**, 63.
- Lane, F. J., Carroll, J. M., Levine, H. D., and Gorlin, R. (1968). The apexcardiogram in myocardial asynergy. *Circulation*, **37**, 890.
- Master, A. M., Gubner, R., Dack, S., and Jaffe, H. L. (1940). The diagnosis of coronary occlusion and myocardial infarction by fluoroscopic examination. *American Heart Journal*, **20**, 475.
- Rapaport, E., and Scheinman, M. (1969). Rationale and limitations of haemodynamic measurements in patients with acute myocardial infarction. *Modern Concepts of Cardiovascular Disease*, **38**, 55.
- Rushmer, R. F. (1956). Initial phase of ventricular systole: asynchronous contraction. *American Journal of Physiology*, **184**, 188.
- Schuette, W. H., and Simon, A. L. (1968). A new device for recording cardiac motion. *Medical Research Engineering*, **7**, 25.
- Schwedel, J. B., Samet, P., and Mednick, H. (1950). Electrokymographic studies of abnormal left ventricular pulsations. *American Heart Journal*, **40**, 410.
- Sonnenblick, E. H. (1971). Non-invasive evaluation of regional ventricular dysfunction. *New England Journal of Medicine*, **285**, 114.
- Suh, S. K., and Eddleman, E. E., Jr. (1959). Kinetocardiographic findings of myocardial infarction. *Circulation*, **19**, 531.
- Tennant, R., and Wiggers, C. J. (1935). The effect of coronary occlusion on myocardial contraction. *American Journal of Physiology*, **112**, 351.
- Wharton, C. F. P., Smithen, C. S., and Sowton, E. (1971). Changes in left ventricular wall movement after acute myocardial infarction measured by reflected ultrasound. *British Medical Journal*, **4**, 75.
- Zaret, B. L., Strauss, H. W., Hurley, P. J., Natarajan, T. K., and Pitt, B. (1971). A noninvasive scintiphotographic method for detecting regional ventricular dysfunction in man. *New England Journal of Medicine*, **284**, 1165.

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