Left ventricular function in ischaemic heart disease. A review

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Summary

The clinical significance of the abnormalities seen at left ventriculography in ischaemic heart disease is discussed. Aneurysms may be recognized when left ventriculography is combined with coronary arteriography to show the characteristic obliteration of the supplying artery. Localized areas of abnormal contraction seen at rest are almost certainly indicative of infarcts. Similar areas provoked by exercise or atrial pacing represent the site of acute ischaemia. The ischaemic ventricle may be recognized by abnormal response to exercise, even in the absence of angina. The abnormal response may be reversed by successful revascularization surgery.

Introduction

Atherosclerotic coronary artery disease leads to focal myocardial ischaemia and consequently to focal abnormalities of left ventricular contraction. Proper understanding of the pathophysiology of ischaemic heart disease thus requires that such localized abnormalities of left ventricular movement be demonstrated. The most graphic method available is the ciné left ventricular angiocardigram and in this paper the significance is considered of various local abnormalities of left ventricular contraction which may be seen at left ventricular angiocardigraphy under various conditions. All the comments that follow are based on observations made on ciné ventriculograms filmed in the 30° right anterior oblique (RAO) projection, on full inspiration. In these angiograms, contrast medium was injected into the left ventricle using a catheter passed retrogradely through the aortic valve. All studies were made during the course of routine coronary arteriography performed for the investigation of suspected ischaemic heart disease.

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The RAO left ventriculogram is illustrated in Fig. 1. Contraction from diastole to systole occurs concentrically and equally towards aortic and mitral valves. To aid in description the left ventricular outline may be divided into segments, and the authors refer to anterior wall, apex, and inferior wall (Fig. 2), qualifying the location of any abnormality of wall movement within these broad categories. Abnormalities of wall movement have been divided into *dyskinesia*, when the abnormal wall segments moves paradoxically; *akinesia* when it remains immobile during contraction; and *hypokinesia* when it moves in similar direction to the rest of the heart but with a lesser excursion (Gorlin, Klein and Sullivan, 1967). The authors prefer the term *dyskinesia* to refer to any local abnormality of wall movement, visible as the ciné angiogram is played, and this is how the term is used in the text. In all their work the authors found complete agreement between three observers on the presence and locations of *dyskinesia* in any given left ventriculogram, though less agreement on the extent and severity of the abnormality.

Left ventricular aneurysm

The authors' interest in left ventricular *dyskinesia* began with an investigation into the angiographic appearance of left ventricular aneurysm (Raphael *et al.*, 1972). Patients with this condition usually present with left ventricular failure following a major myocardial infarction, more rarely with arrhythmias or angina. In the majority of such patients resection of the aneurysm may be performed with a relatively low mortality and marked relief of symptoms, so accurate diagnosis is of major importance. Raphael and his colleagues (Raphael *et al.*, 1972) reviewed the ciné angiographic findings in ten patients with proved left ventricular aneurysms, comparing them with the left ventriculograms of ten patients shown not to have left ventricular aneurysm at either surgery or post-mortem. The features (Fig. 3) common to patients with aneurysm were a dilated

FIG. 1. Normal ciné left ventriculogram in the right anterior oblique projection (a) diastole, (b) systole. Note the concentric contraction. The arrows indicate the position of the aortic valve.

FIG. 2. Division of the outline of the left ventricular image into areas. (Right anterior oblique projection.) (Reproduced by kind permission of the *British Heart Journal*.)

left ventricular cavity, a gross area of dyskinesia involving more than half of the left ventricular outline, and with the area just distal to the aortic and mitral valves always contracting vigorously. The functional transition from vigorously contracting subvalvar muscle to aneurysmal sac could always be recognized during ciné angiography, even in the absence of an anatomical neck to the aneurysm. Similar left ventricular appearances may be seen in patients who at surgical exploration did not have a discrete left ventricular aneurysm that was resectable (Fig. 4). Such patients have a patent, if obstructed, left anterior descending coronary artery

whereas patients with left ventricular aneurysm have an obliterated left anterior descending branch. Similarly, patients with total obliteration of the anterior descending coronary artery at coronary arteriography have aneurysm. The recognition of left ventricular dysfunction not associated with aneurysm led to the further investigation of its significance.

Left ventricular dyskinesia at rest

In order to discover the significance of left ventricular dyskinesia in the resting ventriculogram the authors (Khattri *et al.*, 1974) reviewed one hundred consecutive coronary arteriograms performed in patients with ischaemic heart disease, after excluding patients with left ventricular aneurysms. Fifty of these patients had normal left ventricular contraction and only seven had ECG evidence of myocardial infarction. Forty-nine patients had one or more areas of dyskinesia and one had a diffusely impaired left ventricle. The relationship of dyskinesia to myocardial infarction is shown in Table 1. A high proportion of patients with dyskinesia had some evidence of previous myocardial infarction. There is a close correlation between inferior dyskinesia and inferior infarction, a variable relationship between ECG pattern and apical dyskinesia.

TABLE 1. Relationship of dyskinesia at rest to ECG findings

Localized Dyskinesia 49 patients (All had obstructive coronary artery disease.)		
Historical evidence of infarction 32 (65%)		
ECG evidence of infarction 29 (59%)		
Apical dyskinesia 32	Inferior dyskinesia 8	Apical and inferior dyskinesia 8
Evidence of infarction 19 (59%)	Evidence of infarction 5 (62%)	Evidence of infarction 5 (62%)
Antero-septal 9	Inferior 5	Inferior 5
Antero-lateral 4		
Inferior 10

FIG. 3. Left ventricular aneurysm. (a) Left ventriculogram in diastole, (b) left ventriculogram in systole. Arrows indicate the transition between actively contracting ventricle (towards the aortic valve) and akinetic distal area. (c) Left coronary arteriogram, right anterior oblique projection. The arrow indicates a completely obstructed anterior descending artery. There is no distal filling. (d) Right coronary arteriogram, right anterior oblique projection. There is no evidence of obstruction.

Eight of the forty-nine patients had single vessel coronary artery disease. Four of these had left descending coronary artery disease and apical dyskinesia and four had right coronary artery disease and inferior dyskinesia (Fig. 5). All the patients had obstructive coronary artery disease, and when more than one vessel was involved an association between anterior descending disease and apical dyskinesia, and right coronary artery disease and inferior dyskinesia, was always seen. Others (Hecht

et al., 1975) have demonstrated that dyskinesia is more likely to occur with severe or complete obstructions and relative lack of collaterals.

These findings led to the concept of the segment of left ventricular myocardium subserved by a regional artery of supply, and dependent for its functional integrity on this vessel. The anterior descending coronary artery supplies the anterior left ventricular free wall and most of the anterior septum. The posterior descending coronary artery, usually a

FIG. 4. Large apical dyskinesia. At surgery a full thickness infarct was seen. There was no significant aneurysmal dilatation. (a) Left ventriculogram in diastole, (b) left ventriculogram in systole. The arrows indicate the transition between actively contracting ventricle (towards the aortic valve) and dyskinesic distal area.

(c) Left coronary arteriogram, right anterior oblique projection. The arrow indicates the site of an anterior descending structure. The distal anterior descending vessel is patent.

branch of the right coronary artery, supplies the inferior free wall and lower septum. The free wall segments of the left ventricular outline are clearly seen at left ventriculography in the RAO projection and abnormalities of movement are easily detected. Obstructive coronary artery disease of the regional artery of supply may lead to abnormal movements of the part of the left ventricle it subserves. Involvement of coronary arteries supplying parts of the myocardium which are not normally silhouetted in the

right anterior oblique view usually produce some dyskinesia in that projection, revealing their presence. The use of the left anterior oblique projection, at right angles to the RAO, may increase appreciation of the extent of dyskinesia but does not reveal otherwise unsuspected dyskinesia (Cohn *et al.*, 1974).

The authors have also seen five cases where proved myocardial infarction had occurred in patients subsequently demonstrated to have normal coronary arteries at coronary arteriography; dyskinesia was

FIG. 5. Extensive inferior dyskinesia. (a) Left ventriculogram in diastole, (b) left ventriculogram in systole. The whole of the inferior aspect of the left ventricle remains immobile in the transition from diastole to systole. (c) Right coronary arteriogram lateral projection. The arrow indicates the site of complete obstruction. (d) Left coronary arteriogram lateral projection. The arrow indicates retrograde filling of the distal right coronary artery. There is also disease of the anterior descending coronary artery.

seen in relation to ECG localization of the infarction in all.

The degree of overall functional impairment associated with dyskinesia is variable in that although many patients have left ventricular end diastolic pressure (LVEDP) within the normal range, the general level was higher with dyskinesia than with normal contraction and coronary artery disease (Fig. 6). Where areas of dyskinesia are large and multiple the LVEDP is always raised and the ejection fraction reduced (Hamilton, Murray and

Kennedy, 1972) leading to a condition of 'ischaemic cardiac failure' which is not due to a mechanical complication myocardial infarction (Keene and Raphael, 1970) and hence not amenable to surgery. Characteristically, only the upper anterior left ventricular wall appears to move properly when this condition is studied at left ventriculography (Fig. 7).

It is concluded from this study that dyskinesia represents objective evidence of local myocardial ischaemia, and is usually associated with previous

infarction, the site being localized by the left ventriculogram. The impairment of function is usually due to an obstruction in the regional artery of the supply of the segment of myocardium affected. These abnormalities lead to impairment of cardiac function and, when severe and numerous, produce ischaemic cardiac failure.

Many authors claim to recognize areas of abnormal contraction of the surface of the contracting heart when it is exposed at surgery and that

these areas correspond in location and type to abnormalities seen at left ventriculography (Cheng, 1971). The present authors' surgical colleagues have not noted such a consistent relationship. They have confirmed that large and gross abnormalities of contraction usually correspond to large, often full thickness, infarcts; they usually find some evidence of infarction, located to the angiographic site of abnormality, in lesser degrees of ventriculographic dyskinesia.

Induction of dyskinesia

Exercise

Since left ventricular dyskinesia is assumed to be the manifestation of regional function impairment due to ischaemic heart disease, its presence might be expected to be the rule in severe coronary artery disease. Its absence in the fifty patients with symptomatic, proved, coronary artery disease with normal left ventriculograms was therefore striking in that these patients had equally severe coronary artery disease as shown by coronary arteriography (Table 2). This suggested that the definitive defect might be intermittent as with angina pectoris. To attempt to define the functional abnormality of angina pectoris, left ventriculography was performed during pain induced by exercise, as well as left ventriculography at rest. Dyskinesia was observed to appear or extend in all the first eleven patients so studied. Of seven patients with normal left ventricular contraction at

FIG. 6. The association of end diastolic pressure and left ventricular dyskinesia. $P < 0.01$.

FIG. 7. Ischaemic cardiac failure. (a) Left ventriculogram in diastole, (b) left ventriculogram in systole. The arrows indicate the only part of the left ventricular wall which shows any significant movement.

TABLE 2. Relationship of severity of coronary artery disease to dyskinesia at rest

	Normal coronary arteries	Single vessel disease	Two vessel disease	Three vessel disease	Total
Normal LV contraction	12	5	13	20	50
Dyskinetic LV contraction	0	8	22	19	49

rest, six developed apical dyskinesia, and this was associated with an obstructed anterior descending coronary artery, and one developed inferior dyskinesia associated with an obstructed right coronary artery (Sharma and Taylor, 1975) an association similar to that of dyskinesia at rest. This suggested that acute ischaemia led to acute reversible localized impairment of left ventricular contraction and also raised the possibility that the site of production of angina might be localized by this method.

The overall functional impairment associated with the development of a dyskinetic area on exercise was determined by comparing the findings in a group of patients exercised to the level of angina, with the development of dyskinesia, with a group of patients found to have haemodynamically and anatomically normal hearts, being investigated for chest pain (Sharma *et al.*, 1976). A rise in LVEDP seen when angina developed was much greater in the ischaemic than in the normal heart (Fig. 8). Measurement of

FIG. 8. The effect of exercise (e) on the end-diastolic pressure in three groups of patients, r—patient at rest.

(a) These patients were found to have normal coronary arteries and normal left ventricular function during routine investigation.

(b) Symptomatic patients with demonstrated coronary artery disease, but in whom exercise did not bring on anginal pain during the study.

(c) Patients with proved coronary artery disease in whom exercise during the study produced anginal pain.

All three groups showed a rise in end-diastolic pressure on exercise.

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left ventricular volume showed a variable effect on the ejection fraction when angina developed; in the main it declined, often markedly, whereas in normal hearts it increased (Fig. 9). Diastolic volume increased dramatically when angina developed (Fig. 10) and so did systolic volume, both declining in

FIG. 9. The effect of exercise on ejection fraction.

The groups are divided up as in Fig. 8.

The ejection fraction increased in those patients with normal left ventricles. It did not change appreciably where ischaemic heart disease was present but exercise did not lead to anginal pain.

The ejection fraction was variably influenced by the development of angina.

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FIG. 10. The effect of exercise on the end-diastolic volume.

Groups divided as in Fig. 8.

Exercise did not influence the end-diastolic volume of patients with normal left ventricles or in those patients with ischaemic heart disease who failed to produce angina.

End-diastolic volume increased greatly when angina developed.

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FIG. 11. The effect of exercise on the end systolic volume.

The same division of patients as in Fig. 8.

All patients with normal left ventricles responded to exercise by reducing their systolic volumes and this is how they increase stroke volume and increase their ejection fraction.

In the presence of ischaemic heart disease, even if angina is not produced by that level of exercise, the systolic volume failed to reduce. When angina developed there was usually a major increase in the systolic volume.

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normal patients (Fig. 11). It was of particular interest to note that the systolic volume on exercise which always declined in normal hearts, failed to change significantly in the ischaemic left ventricle on exercise, even if pain did not develop. Thus the response of the systolic volume to exercise revealed a functional impairment in the ischaemic left ventricle which might by other criteria appear entirely normal. It is hoped that it will be possible to demonstrate that successful myocardial revascularization can reverse the abnormal haemodynamic response of the ischaemic left ventricle to exercise, and to provide confirmation that myocardial revascularization improves ventricular function as well as relieving the symptoms of ischaemic heart disease (Fig. 12).

Atrial pacing

Right atrial pacing has been used in the assessment of ischaemic heart disease for many years. Left ventriculography performed during anginal pain produced by atrial pacing is able to induce or extend areas of dyskinesia, although reports in the literature suggest that dyskinesia may be seen in only a proportion of patients even if angina develops (Dwyer, 1970; Pasternac *et al.*, 1972). In an effort

FIG. 12. The effect of successful reconstructive surgery on left ventricular function in ischaemic heart disease.

The figure shows the resting and exercise parameters in the patient re-studied 6 months after two patent bypass grafts were put in. Pre-operatively the exercise measurements were made in the presence of frank angina pectoris, postoperatively a higher heart rate could be achieved without the development of angina. Operation did not significantly affect the end diastolic pressure response. Postoperatively the systolic volume on exercise showed a reduction, the normal response, as compared to the pre-operative finding of a rise in systolic volume with exercise, the ischaemic response. This qualitative observation strongly suggests that there has been a normalization of the response of the left ventricle to exercise following successful surgery. The volume column refers to both diastolic and systolic volumes; EF, the ejection fraction; r, rest; e, exercise; — indicates pre-operative measurements; - - - indicates postoperative measurements. LVEDP=left ventricular and diastolic pressure.

to compare the efficacy of atrial pacing and exercise as methods of stressing the left ventricle, the authors (Sharma *et al.*, 1976) performed both investigations in a series of twelve patients under investigation for ischaemic heart disease. All twelve patients showed the development of extension of dyskinesia, together with ventricular dilatation both in diastole and in systole, when studied during pain induced by exercise. Only nine patients in whom pain was induced by pacing showed the development of dyskinesia. In all patients the site of dyskinesia was related to obstructive coronary artery disease following the pattern recognized previously. The volume response of the left ventricle to angina induced by pacing differed qualitatively from that of exercise (Fig. 13). Anginal pain induced by exercise came on at a much lower heart rate than anginal pain produced by pacing. Angina induced by exercise led to an increase in left ventricular diastolic volume, angina

FIG. 13. Comparison of the response of the left ventricle to the stress of pacing (p) and exercise (e). Pain comes on at a much lower heart rate on exercise than with pacing. The systolic volume shows a slight increase during angina produced either by atrial pacing or by exercise. The diastolic volume shows a significant increase during exercise-induced angina and a major reduction during pacing-induced angina. This is associated with a marked reduction in the ejection fraction (EF) at pacing and a much lesser change in the ejection fraction on exercise. Both pacing and exercise lead to an increase in the left ventricular end-diastolic pressure when (LVEDP) pain is produced. r = rest.

induced by pacing to a reduction. Angina induced by exercise led to an increase in systolic volume whereas pacing had a variable effect on the systolic volume. Both led to an increase in the end diastolic pressure. Thus the haemodynamic responses of the ischaemic left ventricle to pacing did not differ significantly in direction from those of the normal left ventricle. It is concluded that left ventriculography performed during atrial pacing, to the level of anginal pain, was a relatively convenient way of attempting to induce or locate left ventricular dyskinesia, and useful in those situations where the nature of chest pain may be in doubt. It played little part in the haemodynamic assessment of the ischaemic left ventricle and the results of surgery.

Augmentation left ventriculography

Successful coronary artery reconstructive surgery can cause areas of dyskinesia to disappear, with consequent improvement in left ventricular function. Considerable effort has been devoted to determining in which patients this will occur, and also in which patients adequate left ventricular functional reserve is present, so that surgical results may be defined. Left ventriculography performed under various forms of inotropic stimulation usually shows a

smaller and more vigorously contracting chamber than at rest and, occasionally, small areas of dyskinesia may disappear. It is claimed (Bodenheimer *et al.*, 1976) that those areas which disappear under stimulation with glyceryl trinitrate may also disappear after successful surgery. The authors consider this a rare occurrence. Larger areas of dyskinesia do not resolve with successful surgery, presumably because they represent full thickness infarcts.

Conclusion

Localized areas of abnormal contraction seen on the resting left ventriculogram represent the site of myocardial ischaemia, usually due to myocardial infarction. The size and severity of the abnormal movement indicate the size and severity of the process. Small areas, possibly representing partial thickness infarcts, are not associated with impaired ventricular emptying and may be reversible with successful revascularization surgery. Gross dyskinesia usually represents full thickness infarction, impaired emptying is often present, and the abnormality is not reversible. The grossest dyskinesia, left ventricular aneurysm, is associated with markedly impaired function that may be treated by surgical excision. Left ventriculography demonstrates the reversible loss of muscle function associated with acute ischaemia in angina pectoris—it enables the haemodynamic abnormality of ischaemic heart disease on exercise to be defined, and contrasted with the abnormality produced by atrial pacing.

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