perfusion test is a useful procedure in patients who give an atypical history, and an unequivocally positive result would strongly support the diagnosis. A negative result would focus attention on other causes of the symptoms, irrespective of the radiological finding of oesophageal reflux. Endoscopic biopsy as used in this study added little information of diagnostic importance, perhaps because the criteria of abnormality were sensitive to trivial degrees of reflux. It seems that endoscopy and biopsy could be reserved for the small group of patients with radiological abnormality, evidence of blood loss, or dysphagia.

We thank Dr. A. M. Dawson for encouragement and criticism and for allowing us to study patients under his care; the many students and members of junior hospital staff who helped with the acid perfusion test; the nursing staff of the endoscopy unit; and the technical staff of the histology department. We thank the secretaries of the university department of medicine, Royal Hospital, Sheffield, for typing the manuscript.

G.E.S. was supported financially by the Wellcome Trust and J.M.T.W. by the Medical Research Council.

References

1 Pope, C. E., *Gastroenterology*, 1970, **59**, 460.
2 Ismail-Beigi, F., Horton, P. F., and Pope, C. E., *Gastroenterology*, 1970, **58**, 163.
3 Bennett, J. R., and Atkinson, M., *Lancet*, 1966, **2**, 1150.
4 Bernstein, L. M., Fruin, R. C., and Pacini, R., *Medicine*, 1962, **41**, 143.
5 Langley, R., *Practical Statistics*, p. 269. London, Pan Books, 1968.
6 Skinner, D. B., and Belsey, R. H. R., *Journal of Thoracic and Cardiovascular Surgery*, 1967, **53**, 33.
7 Bombeck, C. T., Battle, W. S., and Nyhuss, L. M., *Archives of Surgery*, 1972, **104**, 477.
8 Baron, J. H., Connell, A. M., and Lennard Hones, J. E., *British Medical Journal*, 1964, **1**, 89.
9 Tuttle, S. G., Belttarello, A., and Grossman, M. I., *Gastroenterology*, 1960, **38**, 861.
10 Skinner, D. B., *American Journal of Digestive Diseases*, 1966, **11**, 771.
11 Cocco, A. E., and Brantigan, O. C., *Annals of Surgery*, 1969, **169**, 857.
12 Palmer, E. D., *Archives of Pathology*, 1955, **59**, 51.
13 Thomas, E., Hislop, I. G., and Grant, A. K., *American Journal of Digestive Diseases*, 1973, **17**, 43.
14 Stanciu, C., and Bennett, J. R., *British Medical Journal*, 1972, **3**, 793.
15 Benz, L. J., *et al.*, *Gastroenterology*, 1972, **62**, 1.
16 Spencer, J., *Proceedings of the Royal Society of Medicine*, 1972, **65**, 30.
17 Earlam, R. J., *British Medical Journal*, 1970, **4**, 714.
18 Hadded, J. K., *Gastroenterology*, 1970, **58**, 175.

Contemporary Themes

Pathophysiology of Subendocardial Ischaemia

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British Medical Journal, 1975, **1**, 76-79

Summary

Most forms of heart disease cause myocardial damage which often is confined to the deep (subendocardial) layer of left ventricular muscle. Much clinical and experimental evidence suggests that subendocardial muscle is prone to ischaemic damage, and a physiological mechanism for this vulnerability is described. Furthermore, experiments suggest that pressures recorded at cardiac catheterization can help to assess if there is subendocardial ischaemia in a variety of lesions in man.

Introduction

During 1971 in England and Wales cardiovascular diseases caused half of all deaths, and in those dying under 65 years of age they caused 42% of all deaths in males and 30% of deaths in females. Irrespective of the aetiology of the heart disease most of these deaths were due to damage to and failure of heart muscle. Until the basic diseases can be prevented doctors can reduce the mortality and morbidity from myocardial failure by early and accurate diagnosis of the disability and selection of appropriate medical therapy. Hence it is essential to understand the pathophysiology of myocardial failure and, in particular, to appreciate how important a part subendocardial ischaemia plays in many types of heart disease.

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Cardiac damage occurs predominantly in the subendocardial muscle of the left ventricle. Many myocardial infarcts are subendocardial,[1 2] and patients with angina pectoris but no major infarct have subendocardial ischaemia, as shown by electrocardiography, and areas of necrosis and fibrosis scattered mainly in subendocardial muscle. Even when the coronary arteries are normal there may be electrocardiographic and pathological evidence of subendocardial ischaemia, as in patients with severe aortic regurgitation and stenosis,[3 4] rapid tachycardias,[5] and complicating acute haemorrhage[6] and shock.[7] Finally, many who die after open heart surgery for a variety of lesions have diffuse haemorrhagic necrosis of the left ventricular subendocardial muscle.[8 9] To explain how these lesions occur and might be prevented we will discuss the pathophysiology of coronary flow, show how animal experiments throw light on the causes of subendocardial ischaemia, and apply this information to a variety of human diseases.

Physiological Background

When the left ventricle contracts it compresses vessels in its wall and reduces coronary flow. The compressive force is greatest in subendocardial muscle, where it equals or exceeds pressure in the left ventricular cavity, and falls to near atmospheric pressure at the epicardial surface.[10 11] As a result, in systole no blood flows through subendocardial muscle, whereas the outer subepicardial muscle is perfused. In diastole, however, all the muscle is perfused, thus explaining the dominant diastolic flow pattern in the left coronary artery (fig. 1 A).

When the ventricle works harder and needs more oxygen and thus more coronary blood flow the coronary vessels dilate, lower vascular resistance, and permit more flow even with unchanged perfusing pressures. If, however, cardiac work increases until coronary vessels dilate maximally then coronary flow will depend upon the perfusing pressure, just as flow

through a pipe varies with the pressure head. When this happens subendocardial flow (which is entirely diastolic) will depend on (*a*) coronary arterial diastolic pressure which equals aortic diastolic pressure if the coronary arteries are normal, (*b*) the opposing pressure—either coronary sinus pressure or, if it is higher, left ventricular diastolic compressive force which is similar to left ventricular diastolic pressure,[10 11] and (*c*) the duration of diastole. Therefore, subendocardial blood flow at maximal coronary vasodilatation is represented by the area between aortic and left ventricular pressures in diastole—the dotted pressure areas in fig. 1. This area is termed the diastolic pressure time index (D.P.T.I.) and when multiplied by heart rate is an index of blood supply to left ventricular subendocardial muscle per minute.

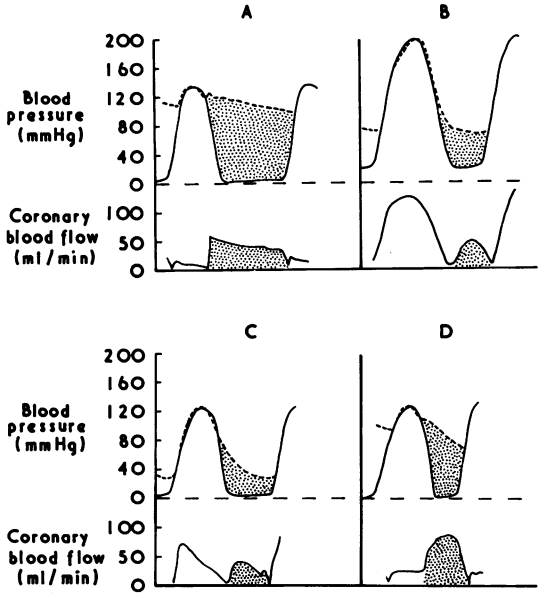

FIG. 1—Superimposed aortic and left ventricular pressure tracings (above) and phasic flow in branch of left coronary artery (below) showing (A) normal physiology, (B) supravalvar aortic stenosis, (C) aorto-atrial fistula (slow heart rate), and (D) aorto-atrial fistula (fast heart rate). Dotted pressure area is D.P.T.I. and clear area is S.P.T.I. Dotted area in flow tracing indicates diastolic flow.

RATIO OF SUPPLY AND DEMAND

To assess adequacy of blood flow, however, we must know not only how much blood is supplied but how much is needed. The needs of a layer of left ventricular muscle for oxygen cannot be measured exactly but are roughly proportional to the area under the left ventricular pressure curve in systole;[12 13] double the area and myocardial oxygen needs about double. This systolic area (fig. 1) may be termed the systolic pressure time index (S.P.T.I.) and multiplied by heart rate it reflects the need of the left ventricle for oxygen per minute. The two areas thus reflect supply (D.P.T.I.) and demand (S.P.T.I.) and their ratio should give information about the adequacy of subendocardial blood flow. This adequacy can be assessed more directly by measuring subendocardial and subepicardial blood flow separately (see below). Many studies show that normally flow/g/min is about the same in the left ventricular subendocardial and subepicardial muscle[14 15] and that this similarity persists for moderate increases of cardiac work.[15] Since subepicardial blood flow occurs throughout the cardiac cycle and is not restricted by compressive forces it indicates roughly what flow is needed per unit of muscle. An appreciable fall in the ratio of subendocardial to subepicardial flow per gram is likely to indicate subendocardial ischaemia.

The hypothesis put forward now is that because of the uneven myocardial compressive forces in systole subendocardial blood flow is entirely diastolic and depends on D.P.T.I. when coronary vessels are maximally dilated. Since myocardial oxygen needs depend on S.P.T.I., a fall in the ratio D.P.T.I.: S.P.T.I. below a critical value should be associated with subendocardial ischaemia, with subendocardial flow less than subepicardial flow. At the same time there should be a smaller proportion of diastolic coronary flow.

Experimental Studies

Normal dogs and sheep were studied under anaesthesia.

We measured phasic flow in the left coronary artery with an electromagnetic flowmeter and pressures in the aorta, left ventricle, and both atria with catheters and strain gauges. Regional coronary blood flow was measured by injecting into the left atrium radioactive microspheres of 9 μm or 15 μm in diameter which are distributed to organs and regions within organs in proportion to local blood flow and are trapped in the tissues. Several different nuclides can be used and after the organs are removed and cut up the flows measured by each nuclide can be calculated to within 10% of true flows.[15] After making control measurements the ratio D.P.T.I.: S.P.T.I. was altered in various ways and new measurements of flows and pressures were made.

Constricting the descending aorta increased D.P.T.I. and S.P.T.I. so that their ratio changed little. Coronary flow increased but remained predominantly diastolic, and subendocardial and subepicardial flow increased equally. By contrast, constricting the aorta just above the aortic valves raised S.P.T.I., thus increasing myocardial oxygen needs, but lowered D.P.T.I. because tachycardia and prolonged systole reduced diastolic duration and because left ventricular diastolic pressure rose (fig. 1 B). Coronary flow increased but became predominantly systolic and, as expected, the increased systolic flow went to subepicardial muscle so that the subendocardial to subepicardial flow ratio fell to low levels.

Opening an aortoatrial fistula simulated aortic incompetence. It did not change S.P.T.I. but the marked fall of aortic diastolic pressure lowered D.P.T.I. (fig. 1 C). Once again coronary flow became predominantly systolic, and subendocardial flow fell, both absolutely and relative to subepicardial flow. Similarly, infusion of isoprenaline caused diastolic pressure and duration to fall, thus reducing D.P.T.I. At first coronary flow and myocardial contractility increased because β-receptors were stimulated, but larger doses caused further lowering of D.P.T.I. and D.P.T.I.: S.P.T.I. and reduced subendocardial flow and myocardial contractility.[16]

Increasing the heart rate had complex effects. D.P.T.I. and S.P.T.I. fell as the rate increased so that the D.P.T.I.:S.P.T.I. ratio fell slowly. Eventually at rates of 250-300/min the ratio fell below a critical level and only then did coronary flow become predominantly systolic and subendocardial ischaemia occur. If there was moderate aortic stenosis with a raised S.P.T.I. at resting heart rates then pacing reduced diastolic duration and D.P.T.I.:S.P.T.I. and produced subendocardial ischaemia at rates that would not cause it in the normal heart. By contrast, moderate tachycardia improved subendocardial blood flow when this had been reduced by opening an aortoatrial fistula (fig. 1 D). Though diastolic duration was reduced by pacing the increased mean aortic diastolic pressure produced a net increase in D.P.T.I. and D.P.T.I.:S.P.T.I. with resultant improvement in the coronary flow pattern and subendocardial flow.[17]

ANAEMIA

The relation between D.P.T.I.:S.P.T.I. and the ratio of subendocardial to subepicardial flow for these experiments is shown in fig. 2. During the various experiments D.P.T.I.: S.P.T.I. fell, but as long as the ratio remained above 0·5 subendocardial and subepicardial flows remained equal. Further stress on the heart lowered D.P.T.I.:S.P.T.I. below 0·5 and relative subendocardial flow fell because maximal vasodilatation had occurred. With anaemia, however, the critical level of D.P.T.I.:S.P.T.I. was variable.[18] Anaemia reduces the transport of oxygen per ml of blood and is compensated for

by coronary vasodilatation so that for any D.P.T.I.:S.P.T.I. ratio the coronary vessels were closer to maximal dilatation, when subendocardial flow would become entirely dependent on D.P.T.I. To allow for the effect of anaemia the D.P.T.I. is multiplied by the arterial oxygen content in ml per 100 ml blood to give an index of the total oxygen brought to the subendocardial muscle per minute. When this is done the critical ratio of oxygen content × D.P.T.I. to S.P.T.I. is about 10 and apparently scattered values lie on a common curve.[18] Presumably the same compensation can be used with arterial desaturation.

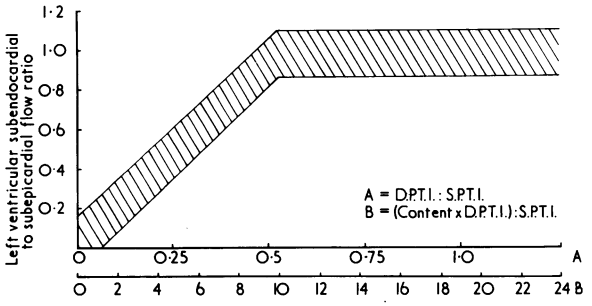

FIG. 2—Relationship of ratio of left ventricular subendocardial to subepicardial flow per gramme to the ratio D.P.T.I./S.P.T.I. (A) and ratio of oxygen content × D.P.T.I. to S.P.T.I. (B). Shaded area indicates values found in many experiments of varied types.

Clinical Implications

Anaemia.—Severe anaemia can cause subendocardial ischaemia[6] and infarction[19] even with normal coronary arteries and the probable mechanism is that just described. With coronary atheroma the harmful effect would be greater. In lesions such as aortic valve disease with lower D.P.T.I.:S.P.T.I. anaemia increases the risk of subendocardial ischaemia. It is thus important to avoid or treat anaemia when there is heart disease, both to lower cardiac output and to improve subendocardial blood flow and oxygen delivery.

Aortic Stenosis.—The theory explains why children with severe aortic stenosis have extensive subendocardial necrosis[4] and also explains the effects of tachycardia in this lesion. Clinically angina pectoris may occur with exercise despite an increased coronary flow,[20] isoprenaline infusion may induce anaerobic metabolism,[21] and T-waves in left precordial leads may invert with exercise.[22] Furthermore, children with severe aortic stenosis have adequate values of the ratio oxygen content × D.P.T.I. to S.P.T.I. and normal electrocardiograms at slow heart rates but at fast rates have low ratios and electrocardiographic signs of subendocardial ischaemia.[23] An exercise electrocardiogram is thus useful in assessing severity if the resting electrocardiogram shows normal T-waves.

Supravalvar Aortic Stenosis.—Here the coronary arteries are usually proximal to the stenosis and exposed to high pressure. Nevertheless, patients with this lesion have subendocardial ischaemia because good perfusion in systole does not increase subendocardial flow, while in diastole the low D.P.T.I.:S.P.T.I. ratio produces subendocardial ischaemia.[24] Ischaemia occurs earlier in those who have coronary arterial lesions as well.

Aortic Incompetence.—In this lesion angina pectoris occurs with effort but is more often noted at night,[25] possibly because the slow heart rate during sleep lowers D.P.T.I.:S.P.T.I. and causes subendocardial ischaemia. Furthermore, the effects of pacing in increasing subendocardial blood flow in dogs with arteriovenous fistulae[17] may in part explain the improvement found in eight patients with intractable heart failure from gross aortic incompetence when they were paced.[26]

Isoprenaline Infusion.—Prolonged massive infusions of isoprenaline may be associated with subendocardial necrosis and haemorrhage.[27] While many factors might produce these changes the ischaemic damage could be due mainly to the raised oxygen needs and low D.P.T.I.:S.P.T.I. ratio that isoprenaline produces. Therefore, isoprenaline infusions to treat low output states should be controlled to obtain optimal D.P.T.I.:S.P.T.I. ratios.

Tachycardia.—A rapid heart rate per se does not cause subendocardial ischaemia unless rates are over 300/min. If there is anaemia, coronary arterial disease, or aortic valve disease then even moderate tachycardia could lower D.P.T.I.:S.P.T.I. and cause subendocardial ischaemia.

Exercise.—Sudden maximal exertion in young healthy people often causes for the first 30 seconds definite S-T depression suggestive of myocardial ischaemia.[28] These electrocardiographic changes are associated with the decrease of D.P.T.I.:S.P.T.I. to under 0·5. After 30 seconds the ratio rises and S-T changes disappear. This finding suggests that sudden maximal effort should be avoided in older people with possible coronary arterial narrowing and a D.P.T.I.:S.P.T.I. ratio beyond the obstruction that is already low at rest.

Coronary Atheroma.—In patients who come to surgery because of coronary atheroma mean arterial pressures beyond the obstructions are 10-50 mm Hg whether there is good collateral flow or not.[29] There is thus a low D.P.T.I.:S.P.T.I. ratio beyond the obstruction at rest. Effort, tachycardia, or anxiety increase myocardial oxygen needs and decrease D.P.T.I. so that angina pectoris results.

Acute Myocardial Infarction.—In this condition aortic blood pressure may fall and left ventricular diastolic pressure may rise while there is often tachycardia. These changes may lower D.P.T.I.:S.P.T.I. so much that generalized subendocardial ischaemia is added to the focal ischaemia of the infarct. Left ventricular function is thus depressed more than expected for the size of the infarct and, in addition, the reduced subendocardial blood flow might jeopardise the viability of the peri-infarct region and cause the infarct to extend. This sequence might explain why experimentally tachycardia and isoprenaline infusions cause an increase in infarct size[30 31] and why isoprenaline and noradrenaline make myocardial metabolism and function worse after infarction in man.[32] By contrast, the same study[32] showed improvement after propranolol, which reduces myocardial oxygen needs, slows the heart rate, and increases D.P.T.I.:S.P.T.I.

Cardiac Surgery

Over half of those dying after open heart surgery have subendocardial necrosis and haemorrhage,[8 9] and many who survive might have lesser degrees of damage. Most of the patients studied had long cardiopulmonary bypasses with ventricular fibrillation, and there are several possible reasons for myocardial damage. All pump systems produce numerous small emboli which go predominantly to the subendocardial muscle. Secondly, even though the heart is fibrillating and not working there is still a high compressive force in subendocardial muscle[33] so that the pressure head from aorta to subendocardial muscle is low. It may be only 20 mm Hg when aortic pressure is 100 Hg. For adequate perfusion there would have to be maximal vasodilatation, and possibly even then the amount of oxygen reaching subendocardial muscle might be inadequate for its needs, especially if haemodilution is used.

Perfusion of the fibrillating dog heart for one to two hours does not damage the subendocardial muscle unless a strong maintained electrical stimulus is used,[34] but even spontaneous fibrillation in dogs with hypertrophied left ventricles leads to massive subendocardial necrosis after 30 to 60 minutes of perfusion.[35] In both these circumstances there might be abnormally high intramyocardial compressive forces which further limit flow to subendocardial muscle. There could also be intermittent obstruction of myocardial arteries by local muscle

contraction. Though there is no synchronized contraction in ventricular fibrillation all the muscle does contract intermittently and might retard for an instant flow in a vessel passing through it. For perfusion to reach subendocardial muscle a vessel will need to be open through the whole thickness of the wall. If the fibrillating stimulus is strong enough and frequent enough it could reduce the time available for blood to flow to the subendocardium; if the heart is hypertrophied the subendocardial muscle is further away from the surface and there is more chance that blood will be obstructed on its way to deeper muscle. In effect, diastolic time available for perfusion of subendocardial muscle is reduced by stronger fibrillation or hypertrophy. Finally, some workers (J. Utley, personal communication) have suggested that the vented left ventricle at surgery is smaller than usual and that the remoulding of muscle that must take place distorts and compresses the subendocardial veins. This would cause myocardial oedema and further reduction of inflow. That this might be important is shown by the improvement of coronary flow in dogs and man when after a long bypass mannitol is given intravenously.

Postoperative care.—Possibly a heart not badly damaged by surgery might acquire subendocardial ischaemia if during the postoperative period there is a low D.P.T.I.:S.P.T.I. for long periods.[9] Measurement of left atrial and aortic pressures and attention to the D.P.T.I.:S.P.T.I. ratio might thus be useful in preventing ischaemic damage. At times, however, pharmacological manoeuvres might not be able to raise D.P.T.I.: S.P.T.I. without causing other problems. In these instances balloon aortic counterpulsation could be very useful.[36] The procedure lowers systolic pressure, thus reducing myocardial oxygen needs, and raises aortic diastolic pressure, thus increasing D.P.T.I.:S.P.T.I. and improving subendocardial perfusion. The procedure is not easy and has complications but potentially offers so much that it is being used with increasing frequency.

Conclusion

Not all myocardial damage is confined to subendocardial muscle, and there are many causes of myocardial damage other than that described here. Nevertheless, we believe that the studies of many workers have shown that there is a common and fundamental pathophysiological mechanism that can explain many of the clinical and pathological findings in patients with ubendocardi al ischaemia. More work is needed to define how closely human disease follows the animal models and, in particular, what happens when there is ventricular hypertrophy and during various types of cardiopulmonary bypass. We hope that this review will stimulate others to provide the information.

This study was supported by Program Project Grant HL-06285 from the National Heart and Lung Institutes of Health, Bethesda, Maryland, and a N.I.H. Special Fellowship HE-42853 to Dr. Buckberg.

References

1 Miller, R. D., Burchell, H. B., and Edwards, J. E., *Archives of Internal Medicine*, 1951, **88**, 597.
2 Fulton, W. F. M., *British Heart Journal*, 1964, **26**, 1.
3 Friedberg, C. K., and Horn, H., *Journal of the American Medical Association*, 1939, **112**, 1675.
4 Moller, J. H., Nakeb, A., and Edwards, J. E., *Circulation*, 1966, **34**, 87.
5 Scherf, D., *American Heart Journal*, 1944, **28**, 332.
6 Master, A. M., *et al.*, *Circulation*, 1950, **1**, 1302.
7 Hackel, D. B., *et al.*, *Archives of Pathology*, 1964, **77**, 575.
8 Najafi, H., *et al.*, *Annals of Thoracic Surgery*, 1969, **7**, 550.
9 Buckberg, G. D., *et al.*, *Journal of Thoracic and Cardiovascular Surgery*, 1972, **64**, 669.
10 Brandi, G., and McGregor, M., *Cardiovascular Research*, 1969, **3**, 472.
11 Baird, R. J., *et al.*, *Journal of Thoracic and Cardiovascular Surgery*, 1970, **59**, 810.
12 Sarnoff, S. J., *et al.*, *American Journal of Physiology*, 1958, **192**, 148.
13 McDonald, R. H., Taylor, R. R., and Cingolani, H. E., *American Journal of Physiology*, 1966, **211**, 667.
14 Moir, T. W., and DeBra, D. W., *Circulation Research*, 1967, **21**, 65.
15 Buckberg, G. D., *et al.*, *Circulation Research*, 1972, **30**, 67.
16 Buckberg, G. D., and Ross, G., *Cardiovascular Research*, 1973, **7**, 429.
17 Buckberg, G. D., *et al.*, *Cardiovascular Research*. In press.
18 Brazier, J., Cooper, N., and Buckberg, G. D., *Circulation*, 1974, **49**, 968.
19 Elliott, A. H., *American Journal of the Medical Sciences*, 1934, **187**, 185.
20 Rowe, G. G., *et al.*, *Circulation*, 1965, **32**, 251.
21 Fallen, E. L., Elliott, W. C., and Gorlin R., *Circulation*, 1967, **36**, 480.
22 Halloran, K., *Pediatrics*, 1971, **47**, 31.
23 Lewis, A. B., *et al.*, *Circulation*, 1974, **49**, 978.
24 Vincent, W. R., Buckberg, G. D., and Hoffman, J. I. E., *Circulation*, 1974, **49**, 326.
25 Harvey, W. P., Segal, J. P., and Hufnagel, C. A., *Annals of Internal Medicine*, 1957, **47**, 27.
26 Judge, T. P., *et al.*, *Circulation*, 1971, **44**, 355.
27 Ferrans, V. J., *et al.*, *American Heart Journal*, 1964, **68**, 71.
28 Barnard, R. J., *et al.*, *Circulation*, 1973, **48**, 936.
29 Smith, S. C., jun., *et al.*, *Journal of Clinical Investigation*, 1972, **51**, 2556.
30 Shell, W. E., and Sobel, B. E., *American Journal of Cardiology*, 1973, **31**, 474.
31 Maroko, P. R., *et al.*, *Circulation*, 1971, **43**, 67.
32 Mueller, H., *et al.*, in *Myocardial Blood Flow in Man*, ed. A. Maseri, p. 347. Torino, Minerva Medica, 1972.
33 Baird, R. J., Goldbach, M. M., and de la Rocha, A., *Journal of Thoracic and Cardiovascular Surgery*, 1971, **64**, 635.
34 Hottenrott, C., Buckberg, G. D., and Maloney, J. V., *Surgical Forum*, 1972, **23**, 200.
35 Hottenrott, C., *et al.*, *Journal of Thoracic and Cardiovascular Surgery*, 1973, **66**, 742.
36 Feola, M., Haiderer, O., and Kennedy, J. H., *Chest*, 1971, **59**, 68.

General Practice Observed

The General Practitioner and Changes in Obstetric Practice

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British Medical Journal, 1975, 1, 79-82

Summary

Compared with the 1960s fewer general practitioners today are obtaining a postgraduate diploma in obstetrics, and the future more stringent criteria for practitioners wishing to undertake this will probably restrict the numbers of family doctors wishing to practise in this field. More deliveries are being performed in institutions —either in consultant or general-practitioner units. Moreover, within a decade probably few G.P.s will attend during normal labour or delivery, which can and should be conducted by midwives. In future, therefore, G.P.s should have a new role in obstetrics, being respon-

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