effect on the locomotor muscles. The respiratory muscles might be expected to hypertrophy under the increased demand imposed by disease, but surprisingly what are usually found are increased fatigability and atrophy. In particular, fatigue of the inspiratory muscles may be important in limiting exercise tolerance.²⁷

How, then, may the respiratory muscles be trained? The answer is breathing against resistance, which increases tidal volume, decreases the respiratory rate, and increases effort tolerance. Respiratory muscle endurance training can also increase exercise capacity in chronic obstructive airways disease, sometimes by as much as half, but generally less.^{8,22} Nevertheless, some patients deteriorate during exercise because of severe fatigue of the diaphragm. The selection of appropriate patients on clinical grounds is difficult: it requires the services of elaborate muscle function laboratories. For that reason the recent report of Sonne and Davis is of interest.⁸ Six patients with moderate to severe airways obstruction entered a six week training programme using a resistance "whistle" with variable orifice, one way valve, and mouthpiece. Training was aimed at the inspiratory muscles and was performed for 30 minutes daily. The maximal work rate increased by 37% and $\dot{V}_{O_2}$ max by 15%. Clearly some patients derive considerable benefit from simple training programmes of this type. Further studies of respiratory muscle function in patients with chronic obstructive airways disease should be performed to maximise benefit from such training programmes.

T W EVANS
Research fellow

P HOWARD
Reader in medicine

Academic Division of Medicine,
University of Sheffield,
Sheffield S10 2JF

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True left ventricular aneurysm

The pathologist defines a true left ventricular aneurysm as "a protrusion of a localised portion of the external aspect of the left ventricle beyond the remainder of the cardiac surface with simultaneous protrusion of the cavity."¹ Angiographically, a left ventricular aneurysm is described as a motion disturbance of the myocardium where a part of the left ventricular wall shows localised akinesia or dyskinesia during the systolic phase of a cineangiogram.^{2,3}

The most common cause of left ventricular aneurysm is myocardial infarction but it may be due to a congenital defect, trauma, bacterial endocarditis, myocardial abscess, syphilis, or Chagas's disease,⁴ or be of the African idiopathic mitral subannular type.⁵

The incidence of left ventricular aneurysm in survivors of acute myocardial infarction depends on the definition used: figures are quoted from 4% in necropsy series to 40% in angiographic series.^{6,7} Furthermore, the interval from myocardial infarction to clinical presentation with a left ventricular aneurysm may be as long as three years.^{8,9} Patients who present only weeks after myocardial infarction have an especially poor prognosis.¹⁰

Symptoms that should arouse suspicion of a left ventricular aneurysm include persistent left ventricular failure and angina pectoris and, less commonly, recurrent ventricular tachyarrhythmias and thromboembolism. There are no diagnostic physical signs, though an expansile or double apex beat may be highly suggestive.¹¹ The heart is usually enlarged on the chest x ray film, but an aneurysm bulge is unusual.¹¹ Calcification may be seen in the wall of the aneurysm. Most patients have a persistent rise in the ST segment on the electrocardiogram,^{8,11} and a dominant R wave in lead aVF may be a helpful pointer.⁶

Confirmation of a clinical suspicion of left ventricular aneurysm relied entirely on left ventricular cineangiography until the advent of non-invasive techniques. M mode echocardiography is of limited value, but it is available in many peripheral general hospitals and diagnostic criteria for left ventricular aneurysm have recently been defined in a series of patients with severe cardiac failure and previous transmural myocardial infarction.¹² If the left ventricular cavity at the level of the mitral ring is greater than 6 cm at end systole or if fibre shortening (end diastolic diameter—end systolic diameter) divided by end diastolic diameter is less than 15%, then a left ventricular aneurysm is unlikely. Over 90% of patients with left ventricular aneurysm have fibre shortening greater than 20% and opening of the mitral valve delayed by 80 ms or more. Further evaluation of these measures is warranted.

In skilled hands two dimensional echocardiography can allow differentiation of left ventricular aneurysm from diffuse left ventricular dilatation^{7,11} and also detect mural thrombi.¹¹ False left ventricular aneurysm (rupture of the left ventricular wall, the haematoma being contained by the adherent pericardium and extracardiac structures) has a poor prognosis for the defect often ruptures completely.¹³ The important differentiation between true and false aneurysms is aided by two dimensional echocardiography: true left ventricular aneurysms have a wide mouth while false aneurysms often extend behind the intact left ventricular wall and have a narrow neck.¹⁴

Non-invasive radionuclide techniques provide another range of methods for accurate screening for left ventricular

aneurysm, assessment of the contractile segment, and differentiation from diffuse left ventricular hypokinesis.^{13 15} A giant aneurysm of the anterior wall may be incorrectly diagnosed, however—an error made less likely by additional scintigraphic views and two dimensional echocardiography.¹⁶

Untreated, patients with a left ventricular aneurysm have a 90% chance of dying within five years of their myocardial infarction.^{17 18} Such global figures must be qualified by the statement that prognosis depends on several factors and, in particular, the overall left ventricular performance. Death usually results from recurrent myocardial infarction with or without left ventricular failure.

Patients should be referred for consideration of surgery when the maximum appropriate medical treatment has failed.¹⁹ All patients, especially those with severe left ventricular failure, must be fully assessed with attention paid to the function of the non-aneurysmal contractile segment of the left ventricle, the severity of occlusive coronary artery disease, and the presence and severity of mitral regurgitation.²⁰ Both the variable operative mortality (4-50%) and the clinical prognosis depend on these factors.^{21 22} Radionuclide angiography and two dimensional echocardiography are playing an increasing part in the preoperative (and post-operative) assessment of left ventricular function.²³

Many patients with severe left ventricular failure are much relieved after combined aneurysmectomy and coronary artery bypass surgery.^{19 24} Some, however, have disappointing results despite apparently good contraction of the non-aneurysmal segment on preoperative assessment. Indeed, a paradoxical inverse relation has been reported between the extent of any improvement in postoperative left ventricular function and the function of the remaining viable segments.²⁵ In patients who are not in failure aneurysmectomy with coronary artery bypass for refractory angina carries a low operative mortality (4%) and a five year survival of about 75%.²⁶

Mural thrombi are present in about half of all left ventricular aneurysms. The incidence of clinically recognised embolism is low at 5%, but necropsy studies show a higher incidence, of about 30%.²⁷ The presence of mural thrombus correlates inversely with the duration of treatment with anticoagulants, and the effect of anticoagulants should be assessed before considering aneurysmectomy for peripheral embolism.

Aneurysmectomy for ventricular tachyarrhythmias may help many patients, but the results are unpredictable, and the operative mortality approaches 60% within a month of acute myocardial infarction.^{19 28} The site of origin of such arrhythmias may be distant from the aneurysm. Electrophysiological mapping may help in its location: an encircling ventriculotomy or endomyocardial resection may then be beneficial.²⁹ Clearly a thorough trial of medical treatment should be given before surgery.

What has emerged from recent research is that patients with left ventricular aneurysms after myocardial infarction form a heterogeneous group. The main determinant of the clinical course and outcome of surgery is the state of the coronary arteries, which in turn determines the function of the non-aneurysmal segment of the left ventricle and the size of the left ventricular aneurysm. More studies of clearly defined subgroups of these patients are needed.

DAVID A TIBBUTT

Consultant Physician,
Worcester Royal Infirmary,
Worcester WR1 3AS

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Deputising services

Facts have been in short supply during the recent debate on problems associated with deputising services in general practice—but opinion has abounded and has at times been based on wishful thinking. If rational decisions on future policy are to be made the facts need to be distinguished and recognised.

Evening, night, and weekend visits make up only 1% to 3% of general practice consultations,¹⁻³ but they arouse strong emotions in both doctors and patients.⁴ Over 40% of night visits are currently carried out by deputising services.⁵ When a call has been received deputising services usually respond by visiting the patient.^{6 7} General practitioners are much more likely to respond with telephone advice.¹⁻³ A paper by Sheldon and Harris (p 474) confirms a previous report that use of deputising services was associated with a substantial increase in night visiting.⁸ No evidence exists to suggest which approach to calls represents good clinical practice.

On the whole, those deputising services which have been studied appear to respond to calls reasonably quickly.^{6 9} The appropriateness of delays is, however, difficult to judge from published work: patients are more likely to complain that the delay in visiting has been longer,¹⁰⁻¹² or longer than