with and budgeted for by social services for private property and by the housing department for council homes, the actual work usually being contracted out privately. For major structural adaptations costing over £500-£1000—for example, concrete ramps—council tenants receive a grant or partial grant from the department of environmental health, whereas those in privately owned property may receive a grant after undergoing a means test. The means test is applied to everyone living in the household. Those who “fail” the means test and pay for the adaptation themselves will have the payment they have made held in their favour should the need arise for an application to be made for a second adaptation in the future. Again, the actual work is contracted out privately. For a publicly used building, such as a church, the council may provide half the cost of a ramp, but local arrangements vary. No value added tax is payable on privately bought ramps if the user is chronically sick or disabled.

Ramps and rails provide the keys to accessibility to many disabled people. Their value seems obvious, but evaluation of different types has been scarce. Sweeney *et al* studied several portable ramps available in the United Kingdom and found that subjects’ preferences depended on whether they propelled themselves or were pushed by an attendant, the type of wheelchair, the visual ability of user and attendant, and their social needs.⁶ They advised individual testing by each user of any ramp in the environment in which it would be used before purchase. A visit to the nearest disabled living centre should ensure that the most appropriate purchase is made.

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Appendix

FURTHER READING

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Lesson of the Week

Bilateral diaphragmatic paralysis presenting with orthopnoea and apparent radiological evidence of pulmonary embolism

A Nisbet, W Kinnear, M J Ward

Bilateral diaphragmatic paralysis may be misdiagnosed as pulmonary embolism

Bilateral diaphragmatic paralysis is a rare but well recognised cause of breathlessness, classically presenting with orthopnoea.¹ Less common presenting symptoms are breathlessness on exercise or immersion.² Physicians without a special interest in respiratory muscles may not be alert to the possibility of this diagnosis in a patient presenting with breathlessness.

We describe two cases of bilateral diaphragmatic paralysis presenting to a district general hospital within a few months of each other. The first patient was treated for pulmonary embolism for several weeks before the correct diagnosis was made whereas the second was correctly diagnosed at the time of presentation. Both patients described profound orthopnoea developing within seconds of lying flat.

Case 1

A 50 year old miner presented with breathlessness of gradual onset over four months. This was worse on exercise or lying flat, but there were no other associated symptoms. He had previously been well, with no history of recent viral illness or vaccination, and was not taking any medicines. On examination the only abnormal findings were decreased breath sounds and dullness to percussion over both lung bases. His electrocardiogram and haemoglobin concentration were normal, but bilateral basal plate atelectasis was seen in the chest radiograph (figure). His standing

Chest x ray appearances in case 1

forced expiratory volume in one second (FEV₁) was 1.48 l and his forced vital capacity (FVC) 1.8 l (predicted 3.1 and 4.14 l respectively). The FEV₁:FVC ratio was 85.4%. A ventilation-perfusion lung scan showed unmatched perfusion defects at both lung bases. A diagnosis of pulmonary embolism was made, and the patient was treated with anticoagulants.

The patient was discharged home but returned after several weeks with no symptomatic improvement. On

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examination paradoxical inward motion of the abdomen was noted during inspiration, and his FVC fell from 1.45 l standing to 0.8 l supine. Lung function tests showed (in addition to the restrictive changes indicated by the high FEV₁:FVC ratio) a total lung capacity reduced to 67% of predicted, but the gas transfer per unit of lung volume was 108% of predicted.

Neurological findings were normal, but on further questioning the patient recalled that during the week before the onset of his breathlessness he had noticed stiffness in both shoulders associated with tingling in the tips of the fingers of both hands. During sniffs there was no descent of the diaphragm on radiological screening. Transdiaphragmatic pressure studies with transducers in the oesophagus and stomach showed that during sniffs the intragastric pressure remained negative. There was no detectable diaphragmatic contraction when the phrenic nerve was stimulated in the neck, and results of axillary and median nerve conduction studies were normal.

Serum electrolyte concentrations and thyroid function were normal, antinuclear antibodies were not detected, and a Tensilon test produced no changes. x Ray appearances of the cervical spine were normal. Gas exchange during sleep was normal. A diagnosis of bilateral diaphragmatic paralysis was made, probably related to neuralgic amyotrophy.

Case 2

A few months after case 1 a second 50 year old miner presented to the hospital with breathlessness and orthopnoea. During questioning he admitted that he also had pain in his shoulders and weakness in his arms. There were no other respiratory or neurological symptoms. Examination of the cardiovascular system showed nothing abnormal. On lying flat he rapidly became dyspnoeic and was noted to have pronounced paradoxical motion of the abdominal wall. There was winging of the right scapula. Spirometry recorded a fall in FVC from 3.0 l standing to 1.9 l supine. Bilateral basal plate atelectasis was noted in the chest radiograph. A ventilation-perfusion lung scan was normal.

Transdiaphragmatic pressure studies showed that during sniffs the patient was unable to generate a measurable positive intragastric pressure. Transcutaneous stimulation of the phrenic nerve in the neck produced no detectable diaphragmatic contraction. Nerve conduction studies in the arms showed nothing abnormal. Muscle enzyme activities, thyroid function, Tensilon test results, and x ray appearances of the cervical spine were all normal. Bilateral diaphragmatic paralysis was diagnosed.

Discussion

These cases showed the cardinal features of bilateral diaphragmatic paralysis.¹ These are orthopnoea soon after lying down, paradoxical (inward) movement of the abdominal wall on inspiration, a large fall in FVC on lying down, and small lung fields radiologically with basal atelectasis. The first patient was initially misdiagnosed as having pulmonary embolism.

Bilateral diaphragmatic paralysis is usually seen as part of a more generalised muscular or neurological condition such as motor neurone disease, myasthenia

gravis, or muscular dystrophy.¹ It may also occur in association with malignancy³ or hypothyroidism,⁴ after receiving tetanus antitoxin,⁵ or after a viral illness.⁶ Probably in both our cases weakness of the diaphragms was due to neuralgic amyotrophy associated with painful brachial neuritis.⁷

Investigations have classically included radiological screening of the diaphragms, but the test is not specific as paradoxical movement may be due to contraction of the abdominal muscles during inspiration.^{8,9} Diaphragmatic function is best assessed by measuring the change in transdiaphragmatic pressure. During sniffs there is usually a fall in oesophageal pressure and a rise in intragastric pressure; the gradient is large. In our cases the intragastric pressure remained negative and no pressure gradient was obtained, changes indicative of diaphragmatic paralysis.⁹

In the absence of weakness of other muscles bilateral diaphragmatic paralysis is usually associated with only mild nocturnal hypoxaemia.^{2,10} Provided that the patient can sleep propped up assisted ventilation at night is not usually necessary.

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Update box for *Oxford Handbook of Clinical Medicine* (2nd ed), p 200

Cheaper tuberculosis treatment is better than none

Rifampicin is acknowledged to be "the best" anti-tuberculosis drug available, and it forms part of all combined treatment regimens. It is also a drug that many countries find difficult to afford, and it is known that much tuberculosis remains untreated. It is therefore important for such communities to know that daily rifampicin is not necessarily needed and that twice weekly rifampicin (600 mg by mouth, combined with isoniazid 900 mg) is a reliable and cheaper alternative.¹

This regimen should also be considered when attempts to give daily doses are likely to fail. — J M LONGMORE

Principal source

- 1 Castelo A, Jardim JRB, Gohman S, et al. Comparison of daily and twice-weekly regimens to treat pulmonary tuberculosis. *Lancet* 1989; ii:1173-6.

Anyone may submit an update box; all boxes are peer reviewed.