her cervical smear must lie with the doctor who took the smear." Over the past six months there have been statements from the medical defence societies and our local family practitioner committee to this effect.

Two questions follow: Is cervical cytology different from all other investigations or are we expected to inform patients of the result of all investigations? Why are patients assumed to be unable to take the responsibility and to return to receive the result when so requested? If patients are physically or mentally disabled from asking for the result of a test we can and do act to protect them. If we are aware that a patient has failed to come for an important result we will take action. We cannot plan to see that every patient is always told all abnormal results.

Our duty to our patients is to see that they know how and when to obtain the results of our tests so that we can respond with the results; it is not our duty to promise that we will see that the result reaches them come what may. We believe that many other doctors have a similar policy to ours and we appeal to the GMSC to support us.

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Motor neurone disease presenting as respiratory failure

SIR,—Dr B Al-Shaikh and others (17 May, p 1325) describe a case of motor neurone disease presenting with respiratory failure. A similar case presented to the respiratory department of the Western Infirmary.

A 58 year old crofter had first attended hospital six months earlier complaining of breathlessness. Inquiry showed exertional dyspnoea and orthopnoea, and examination showed mild hypertension (150/100 mm Hg). Neurological examination was normal; the chest radiograph showed basal atalectasis; and pulmonary function tests revealed a mild restrictive defect. The presumed diagnosis at that time was left ventricular failure secondary to hypertension. He failed to respond to treatment for this and six months later was referred to the respiratory unit.

His symptoms were similar but more severe. He could not lie supine without developing severe dyspnoea. Examination showed poor entry at the lung bases. Weakness and fasciculation were present in the muscles of both hands. Peak expiratory flow rate was 77 litres supine and 240 litres standing (predicted 520 litres). Electromyography confirmed the diagnosis.

He was allowed home in May 1985 and attempts were made to organise a rocking bed. He was readmitted in a terminal state in October 1985 before the rocking bed was available and deteriorated quickly, dying three days later. Necropsy showed complete atrophy of the diaphragm muscle in keeping with the diagnosis.

This patient, unlike the one reported on by Dr Al-Shaikh and colleagues, had no history of respiratory disease. Despite this the diagnosis was not considered on his first referral to a medical clinic. Lying and standing peak flow measurements are a simple screening test of diaphragmatic weakness in a patient who complains of dyspnoea on lying flat; they are easily carried out in the outpatient clinic and confirm the need for more detailed studies of diaphragmatic function.

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SIR,—The report by Dr B Al-Shaikh and colleagues (17 May, p 1325) has drawn attention to an important and frequently unsuspected problem; respiratory failure may also be the presenting feature in patients with myasthenia gravis. Since the pattern of muscles affected in myasthenia is variable,[1] the respiratory muscles, and in particular the diaphragm, may be affected selectively.[2]

In a study of 18 patients with myasthenia gravis, in whom we measured transdiaphragmatic pressures, significant diaphragm disease was found in five. Two of these patients, who had presented with acute respiratory failure requiring intubation and assisted ventilation, had no obvious sign of peripheral muscle weakness, apart from minimal ptosis. Myasthenia gravis was confirmed by the presence of acetylcholine receptor antibodies in one and reduced size of miniature end plate potentials on intercostal muscle biopsy in the other. Diaphragm strength improved considerably after plasma exchange and immunosuppression therapy. Both patients were able to return to their previous daily activities.

It is important to consider a diagnosis of myasthenia gravis in patients with dyspnoea and in those presenting with acute hypoventilation, since masthenia is a treatable condition, and one which may easily be overlooked.

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1 Simpson J. Myasthenia gravis and myasthenic syndromes. In: Walton JN, ed. *Disorders of voluntary muscle*. 4th ed. Edinburgh: Churchill Livingstone, 1981: 585-624.
2 Mier A, Havard CW. Diaphragmatic myasthenia in mother and child. *Postgrad Med J* 1985;61:725-7.

Sugar and facts

SIR,—I would like to draw your attention to serious inaccuracies in the article by Mr Geoffrey Cannon in your Medicine and Media column (7 June, p 1520).

There is no truth in the statement by Mr Cannon that "The 'news' item was constructed from a public relations handout." The story was distributed by the country's leading and most respected news agency—the Press Association—who are impartial with no axe to grind.

Mr Cannon is also incorrect in saying that the handout was "entitled 'Obesity' and (smaller letters) 'Putting Sugar in Perspective' and (even smaller letters) 'produced and distributed by The Sugar Bureau.'" The only press release distributed was headed "Sugar and Obesity—No Firm Link."

More than 200 journalists were invited to "the discreet press conference" as described by Mr Cannon and, yes, he was there, very much so.

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AUTHOR'S REPLY—The Press Association has confirmed that the source of its own story was the Sugar Bureau. After Professor Durnin's press conference PR handouts were given out; and, as I said, "produced and distributed by the Sugar Bureau" was printed in small letters at the bottom of the handout. Mr Bithell says that this handout was not distributed. It was.

He has also overlooked a Sugar Bureau "News Bulletin" with the title "Sweet News—Sugar Does Not Make You Fat," which goes on, "Sweet news for slimmers and healthy eaters—a leading international expert on obesity today revealed that sugar does not make you fat." This press release ends "for further information contact Gerard Bithell."

Readers with a special interest in the information the public receives about sugars can obtain a copy of a 561 page report with 426 references from the library of the Health Education Council (A Quick *et al*, unpublished).

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SIR,—Mr Cannon's article is such a gay knockabout it is hard to distil the message, but it seems to be that journalists are irresponsible to be uncritical of scientific statements based on research funded by industry. Mr Cannon's anxiety stems from his concern about sugars, their role in obesity, and the doubts that that role is adequately substantiated. In justification he refers to the Royal College of Physicians' report on obesity, the NACNE discussion document, and the BMA report on diet, nutrition, and health, each of which, he claims, recommended that the consumption of simple sugars be reduced by half.

In fact the Royal College of Physicians' report made no such recommendation. The NACNE document did so but this was conditional on modified patterns of consumption not being achieved by other means. The BMA report, a kind of lay version of NACNE, made the recommendation with no supporting evidence, as did a recent report from the Health Education Council. The last three documents read as if they were written by the same hand—or by hands tutored in the same school perhaps.

Mr Cannon and his colleagues seem to suggest that a scientist's results should be judged according to the source of his funds. Given the state of research funding in the United Kingdom I suggest it would be better to continue to judge results by their scientific quality even if this does mean having to tolerate plausible nonsense from time to time.

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Missed malignant melanoma

SIR,—Our reply to Professor Mackie's and Dr Doherty's question (7 June, p 1524) of how we explain the "good results" of the series from Queensland and New Mexico is provided in the last paragraph of our letter of 10 May (p 1270). We would reiterate that the inclusion in any series of patients with incidental and asymptomatic lesions could inflate the five year survival of that particular series by simply including melanomas of limited or no biological potential to metastasise.

We do not doubt the prognostic significance of Breslow's thickness of the primary tumour on survival of patients with malignant melanoma; this is why we take great pains to review the histopathology and record the thickness of the primary lesion of all patients referred to our unit. We do not, however, confuse the dimensional concept of Breslow's tumour thickness, which reflects the biological state of the disease, with the chronological concept of "late or early" presentation for surgery of the primary lesion.

Nowhere in Dr Doherty's and Professor Mackie's paper (12 April, p 987) can we find any scientific evidence that these two concepts, on which they base their educational campaign, are congruent. Our data (p 1270) and those of