

Unreviewed Reports

Facial numbness as presentation of parotid tumour

A 68 year old man presented with numbness in the distribution of his right mandibular nerve (3rd division of the trigeminal nerve). Taste and facial movements were normal. On examination there was a firm mass behind the angle of his jaw and a preauricular swelling; a biopsy specimen showed a squamous carcinoma. Tumours of the deep lobe of the parotid gland usually present with local pain or a mass inside the mouth.¹ They may infiltrate posteriorly across the base of the skull, but spread to the mandibular nerve has not been reported. A neurilemma of the mandibular nerve is the main differential diagnosis.—R MCR HIGGINS, The Brook Hospital, London SE18 4LW. (Accepted 17 April 1984)

¹ Baker DC, Congley J. Treatment of massive deep lobe parotid tumors. *Am J Surg* 1979;138:576-8.

Periorbital oedema caused by nifedipine

A 54 year old woman was started on a slow release formulation of nifedipine (Adalat Retard) as her blood pressure was uncontrolled by atenolol 100 mg and bendrofluazide 5 mg daily. After the first dose of nifedipine she developed severe periorbital oedema accompanied by facial flushing, paraesthesiae, headache, and dizziness which lasted six hours, recurring after her next dose. The patient was then given a single dose of 5 mg nifedipine, which caused identical symptoms. Clearly these were due to nifedipine itself and not to the slow release formulation. This side effect has not been reported.¹—P H SILVERSTONE, Gravesend and North Kent Hospital, Kent DA11 0DG. (Accepted 17 April 1984)

¹ Terry RW. Nifedipine therapy in angina pectoris: evaluation of safety and side effects. *Am Heart J* 1982;104:681-9.

Magnesium induced neuropathy in patient undergoing haemodialysis

A 42 year old woman undergoing regular dialysis developed crippling peripheral neuropathy, unresponsive to extended dialysis time. Her plasma magnesium concentration was 1.85 mmol/l (4.5 mg/100 ml) (normal 0.7-1.1 mmol/l (1.7-2.7 mg/100 ml)). She was therefore dialysed on magnesium free dialysis fluid, and within a month her symptoms cleared. The neuropathic features reappeared after she was inadvertently dialysed against her usual dialysis fluid (magnesium 0.85 mmol/l (2.1 mg/100 ml)). They cleared again, however, with magnesium free dialysis fluid and she remains well. Hypermagnesaemia may have a role in neuropathy in patients undergoing dialysis.¹—RASHEED AHMAD, Sefton General Hospital, Liverpool L15 2HE. (Accepted 30 April 1984)

¹ Steward WK, Flemming LW, Anderson DC, et al. *Proc Eur Dial Transplant Assoc* 1967;4:285-92.

Tuberculosis in a functioning gall bladder

Of the 49 reported cases of tuberculous cholecystitis, gall stones were present in 46 and cystic duct obstruction in the rest. The resistance of the intact gall bladder to tuberculous infection has been attributed to bile acid concentration sufficient to inhibit growth of tubercle bacilli.¹ A 48 year old Asian woman resident in the UK for nine years was investigated for chronic cholecystitis. Oral cholecystogram and ultrasonogram were normal, but because of persistent symptoms laparotomy was performed and a thick walled gall bladder removed. Histology confirmed tuberculosis with acid fast bacilli. Tuberculosis may thus occur in a functioning gall

bladder.—T I DAVIDSON, R GARNHAM, Mount Vernon Hospital, Northwood, Middx HA6 2RN. (Accepted 30 April 1984)

¹ Miggar MS, Kariholu PL, Bhat DN, Fazili F, Yousuf M, Muhajid S. Tuberculosis of gallbladder. *J Indian Med Assoc* 1980;74:196-7.

Mesothelioma due to domestic exposure to asbestos

Two sisters, who lived in adjacent mobile homes, developed pleural mesotheliomas. One recovered after undergoing pleurectomy, but in the other the diagnosis was made post mortem. The only history of exposure to asbestos was that both had some years previously cleaned an outhouse roof made of corrugated white asbestos cement to remove moss growth. This was done as a dry process (with a wire brush and paint scraper) and without wearing masks, and the roof was described afterwards as "lovely and white." Exposure to asbestos on domestic premises may constitute an unrecognised hazard.—G C FERGUSON, H WATSON, Northampton General Hospital, Northampton NN1 5BD. (Accepted 9 May 1984)

Inaccurate home blood sugar monitoring in Raynaud's syndrome

A 43 year old man with chronic pancreatitis had poorly controlled insulin dependent diabetes despite good motivation and finger prick monitoring at home with a Hypocount meter. On three occasions metered finger prick samples indicated hypoglycaemia (1.9, 0.2, 0.6 mmol/l (34, 3.6, 11 mg/100 ml)), but his only symptoms were of mild Raynaud's syndrome. Glucose concentrations in earlobe and venous blood were in the normal range (4.1, 3.6, 4.9 mmol/l (74, 65, 88 mg/100 ml)), and no subsequent earlobe blood showed any disparity with simultaneous venous samples. Raynaud's syndrome may cause intermittently falsely low finger prick sugar concentrations; this should be considered if therapeutic decisions are safely to be based on the results of metered non-venous samples.—J E MACSWEENEY, A FORBES, Middlesex Hospital, London W1N 8AA. (Accepted 10 May 1984)

No link between acute pericarditis and HLA factors

Acute pericarditis is usually idiopathic or viral. It may also be secondary to various other diseases.¹ HLA factors help in modulating immune responses and may be important in determining susceptibility to viral infections. We studied 29 men and nine women with viral or idiopathic acute pericarditis. They were typed for 12 HLA-A, 20 HLA-B, and 5 HLA-C antigens. The distribution of antigens was compared with that found in normal healthy individuals. No significant differences were found; this excludes any strong association between known HLA-ABC factors and non-specific viral or idiopathic acute pericarditis.—REINHOLD BARTRAM, County Central Hospital of Frederiksborg, Denmark, ARNE SVEJGAARD, State University Hospital, Copenhagen Denmark, et al. (Accepted 14 May 1984)

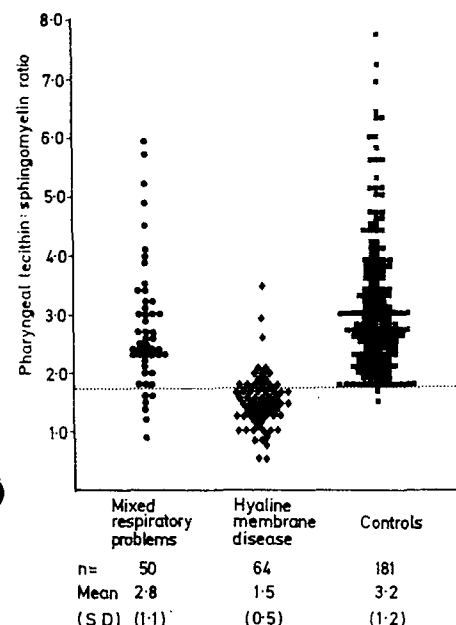
¹ Shabetai R. *The pericardium*. New York: Grune and Stratton Inc, 1981: 108-53 and 348-65.

"Unreviewed Reports" aims at publishing very brief findings quickly, without the usual external peer review. Each item should be no more than 100 words long, with a title of up to 10 words, only one reference, and no more than two named authors (*et al* is allowed). Authors of papers about side effects must have reported them to the Committee on Safety of Medicines and the manufacturers. Correspondence asking for further details about these items should be sent directly to the authors, who should be willing to supply answers.

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establishing the diagnosis of hyaline membrane disease.

This conclusion differs from findings that we have published based on 295 infants,¹ in which the lecithin:sphingomyelin ratio measured on pharyngeal secretions obtained within six hours of birth identified babies with hyaline membrane disease (figure).^{2,3}



Comparisons of pharyngeal lecithin:sphingomyelin ratio in babies with mixed respiratory problems (transient tachypnoea and pneumonia), hyaline membrane disease, and controls. There is a significant difference in the proportion of immature lecithin:sphingomyelin ratios (<1.8) between infants with hyaline membrane disease and those with mixed respiratory disorders (48/64:6/50, $\chi^2=44.6$, $p<0.0001$).

There was a highly significant difference in the proportion of immature lecithin:sphingomyelin ratios (<1.8) between infants with hyaline membrane disease and those with other respiratory disorders (predominantly transient tachypnoea and pneumonia) ($p<0.0001$). A lecithin:sphingomyelin ratio of <1.8 detected 75% (48 out of 64) of cases of hyaline membrane disease with a false positive rate of 3% (eight out of 231). Thus the sensitivity of the test is 75% and the specificity is 97% (223/231).⁴

It is not always possible to assign a respiratory diagnosis with certainty to a newborn infant managed on intermittent positive pressure ventilation from birth since this treatment frequently obscures both clinical and radiological diagnostic features. In such infants a measurement of pulmonary surfactant is of particular value in supporting the clinical diagnosis. It is unacceptable, in our view, however, to include such babies in any study aiming at evaluating the diagnostic reliability of a test of surfactant status. It is not clear how many babies in Dr James's series labelled as having hyaline membrane disease or congenital pneumonia were in fact ventilated from birth. None of the 64 infants independently identified as having hyaline membrane disease in our series were on intermittent positive pressure ventilation from birth onwards.

The lecithin:sphingomyelin ratio is ideally

suited for confirming the comparability of infants randomly allocated to trials of surfactant therapy,⁵ high frequency ventilation (unpublished results), and other treatments. We cannot agree with the conclusion of Dr James and colleagues that pulmonary surfactant deficiency is unreliable in establishing the diagnosis of hyaline membrane disease.

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- ¹ Jenkins PA, Baum JD. Respiratory distress syndrome. In: Wald NJ, ed. *Antenatal and neonatal screening*. Oxford: Oxford University Press, 1984:298-313.
- ² Gluck L, Kulovich MV, Borer RC, Brenner PH, Anderson CG, Spellacy WN. Diagnosis of the respiratory distress syndrome by amniocentesis. *Am J Obstet Gynecol* 1971;109:440-5.
- ³ Barr PA, Jenkins PA, Baum JD. Lecithin/sphingomyelin ratio in hypopharyngeal aspirate of newborn infants. *Arch Dis Child* 1975;50:856-61.
- ⁴ Anonymous. Glossary of terms. In: Wald NJ, ed. *Antenatal and neonatal screening*. Oxford: Oxford University Press, 1984:552-4.
- ⁵ Wilkinson AR, Jeffrey JA, Jenkins PA. Controlled trials of dry surfactant in preterm infants. *Arch Dis Child* 1982;57:802.

Treatment of myopia

SIR,—I want to try to correct some of the exaggeration of Mr Thomas Stuart-Black Kelly's letter (2 June, p 1692). Firstly, myopia is not a disease and cannot be regarded as anything more than an error of refraction except in its higher degrees, when there may indeed be retinal and other complications. It is the fundus changes rather than the degree of myopia that is the disease, and although only likely with errors in excess of 7 dioptres (a small minority of myopes) such changes can be absent in higher myopes and can occur in low myopes or rarely hypermetropes.

Secondly, myopia is a useful condition, especially for ophthalmic surgeons or others doing fine, close work. Myopes come into their own in middle age when others need reading glasses.

Finally, the rationale of Mr Kelly's alternative medicine is that bifocals will abolish the need for the growing child to accommodate—that is, to hinder a most useful function of the eye which has not been proved to be a cause of high myopia. All I would advise is that the growing child wears the normal correction for his myopia so that he sees the blackboard clearly and does not screw up his eyes. Also, so that he does not bury his nose in his book, he should wear the glasses even for reading, thereby building up the normal relation between convergence and accommodation. Otherwise exophoria or divergent strabismus may ensue.

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Mesothelioma due to domestic exposure to asbestos

SIR,—The unreviewed report by Dr G C Ferguson and Dr H Watson (2 June, p 1654) of two sisters who both developed pleural mesotheliomas is of considerable interest. Several occurrences of two or more cases of mesothelioma within the nuclear family have been reported, and this is probably not simply due to a shared source of asbestos exposure.^{1,2}

Before either the diagnosis or aetiology can be accepted, however, further information is required. Was the diagnosis confirmed in both cases by a pathologist with particular experience in mesothelioma histology? In a recent review of cases diagnosed as mesothelioma by local pathologists in the USA, only 26% could be accepted as definite, probable, or even possible by a member of the mesothelioma reference panel.³ The importance of accurate diagnosis is underlined by the prolonged survival of one of the sisters after pleurectomy, as surgery has rarely been found to influence survival of patients with diffuse mesothelioma.

Dr Ferguson and Dr Watson suggest that the mesotheliomas arose from wire brushing of a white asbestos cement roof. This is to ignore the strong epidemiological evidence now accumulating that chrysotile, as distinct from amphibole asbestos, plays little or no part in mesothelioma aetiology. Acheson and Gardner in their 1983 report to the UK Health and Safety executive stated: "Subsequent evidence has supported our previous view that peritoneal mesothelioma for practical purposes never, and pleural mesothelioma rarely, has occurred in man in relation to exposure to chrysotile alone."⁴ Was a microscopic study of the lung fibre burden carried out in the fatal case?

In 1976 Milne reported the cases of two sisters living in Australia who died of mesothelioma in which most careful questioning failed to find any exposure to asbestos.⁵ For most of us nowadays some casual contact with asbestos is inevitable. But post hoc does not always mean propter hoc, and not all cases of mesothelioma are due to exposure to asbestos.

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- ¹ Browne K. Asbestos-related mesothelioma: epidemiological evidence for asbestos as a promoter. *Arch Environ Health* 1983;38:261-6.
- ² Wright WE, Sherwin RP, Dickson EA, Bernstein L, Fromm JB, Henderson BE. Malignant mesothelioma: incidence, asbestos exposure, and reclassification of histopathology. *Br J Ind Med* 1984;41:39-45.
- ³ Acheson ED, Gardner MJ. *Asbestos: the control limit for asbestos*. London: HMSO, 1983.
- ⁴ Milne JEH. Thirty two cases of mesothelioma in Victoria, Australia: a retrospective survey related to occupational asbestos exposure. *Br J Ind Med* 1976;33:115-22.

* * * Dr Ferguson and Dr Watson reply below.—ED, *BMJ*.

SIR,—Lack of space prevented us from stating that the relation between asbestos exposure and the development of mesothelioma in these two sisters had not been definitely established, particularly because a study of the lung fibre burden was not carried out in the fatal case.

In both of our cases the diagnosis of mesothelioma was made by our consultant pathologists, and reference to the failure of local American pathology services to diagnose this condition correctly may not be appropriate. We would, of course, be happy to arrange for histological slides from these cases to be submitted to any reference pathologist.

Prolonged survival with or without surgery does not rule out mesothelioma—Law *et al* found two patients who survived up to eight years from diagnosis.¹ The basic question is whether short term heavy non-occupational exposure to asbestos is a causative factor in