LETTERS TO THE EDITOR

Mycoplasma pneumoniae

EDITOR,—The report from Thomas *et al* may lead us to alter our current understanding of the association of *Mycoplasma pneumoniae* and central nervous system dysfunction.[1] The suggestion, however, that *M pneumoniae* should be considered in all cases of encephalopathy, regardless of preceding respiratory infection, could potentially lead to considerable overdiagnosis for the following reasons.

Complement fixation serology, which is raised as a function of both IgG and IgM, may not be specific due to the use of a potentially cross reactive glycolipid antigen. Complement fixation titres may be raised in the diagnostic range in a small subset of normal individuals, and therefore a background of high titres should exist as well in patients with neurological disorders regardless of the diagnosis. Furthermore, specific increases have been previously found in some central nervous system disorders, for example, Kleemola and Kayhty previously demonstrated a fourfold change in complement fixation titres in 40·7% of paired sera from patients with bacterial meningitis and 10·3% of patients with bacteraemias.[2] Most current IgM assays will also yield positive results in approximately 2–10% of asymptomatic populations, and therefore a similar frequency is at least likely in all patient populations regardless of illness. The frequency of true disease in a patient population will affect the predictive value of a positive test. A positive serological test for *M pneumoniae* is more likely to be truly positive in patients who have had respiratory manifestations in contrast to those who have not. Therefore, if one were to adopt the position that all encephalopathies should be screened serologically for *M pneumoniae* despite the low frequency of true positives, there would be a good chance of false positives equalling or exceeding true positive assays. The problems here are analogous to those encountered in the use of screening tests for populations with low endemicity.

The potential for endemic seropositivity needs to be especially considered when association between a positive test and an uncommon disease is made. It would be preferable then to establish more definitively the diagnosis of *M pneumoniae* infection by adding independent methods such as direct bacterial isolation, IgM anti-P1 immunoblotting,[3] and/or direct polymerase chain reaction assay of respiratory secretions.[4]

The respiratory manifestations of *M pneumoniae* infection are variable and may range from a mild sore throat to atypical pneumonia. In the context of an acute encephalopathy, it would be easy to disregard a mild form of respiratory illness when the focus of history taking and physical examination is the neurological illness. The existing body of medical literature still indicates that the majority of *M pneumoniae*-associated central nervous system diseases are associated with a preceding or coexisting respiratory illness.

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1 Thomas NH, Collins JE, Robb SA, Robinson RO. Mycoplasma pneumoniae infection and neurological disease. *Arch Dis Child* 1993; **69:** 573–6.
2 Kleemola M, Kayhty H. Increase in titers of antibodies to Mycoplasma pneumoniae in patients with purulent meningitis. *J Infect Dis* 1982; **146:** 284–8.
3 Cimolai N, Cheong ACH. IgM anti-P1 immunoblotting: a standard for the rapid serologic diagnosis of Mycoplasma pneumoniae in pediatric care. *Chest* 1992; **102:** 447–81.
4 Bernet C, Garret M, DeBarbeyrac B, Bebear C, Bonnet J. Detection of Mycoplasma pneumoniae by using the polymerase chain reaction. *J Clin Microbiol* 1989; **27:** 2492–6.

Dr Thomas comments:

Dr Cimolai's suggestion that the diagnosis in *Mycoplasma pneumoniae* infection requires more specific tests than complement fixation methods highlights the uncertainty expressed by other authors as to the true prevalence of mycoplasma-associated neurological disease.[1] We would accept his view that complement fixation testing has the limitations outlined.

However, the very fact that the respiratory infection associated with *M pneumoniae* may be so mild as to be disregarded leads us to believe that neurological disease with *M pneumoniae* often appears to occur without a preceding respiratory infection.

1 Clyde WA. Neurological syndromes and mycoplasmal infections [editorial]. *Arch Neurol* 1980; **37:** 65–6.

Surface area is best estimated from weight alone: pocket calculators and nomograms are unnecessary

EDITOR,—In a study that compared the use of pocket calculators and nomograms to estimate body surface area (BSA) from height (H) and weight (W), Briars and Bailey demonstrated that their volunteers made serious errors with both techniques.[1] These authors reference Edith Boyd's monograph from 1935,[2] noting that it is the largest ever study of direct measurement of BSA. Unfortunately Briars and Bailey, along with other groups, have failed to note one of the most important conclusions of her meticulous monograph, namely that BSA is best estimated from W alone.

Boyd first compared estimates of measured BSA from W and H using standard equations, such as that of Du Bois and Du Bois,[3] to estimates using self adjusting power (SAP) equations. In these W and H are each raised to a power, but the exponent to which W is raised is varied according to its own value. The SAP formula produced significantly closer estimates of BSA than standard formulas. Boyd then demonstrated that the following SAP equation using W alone

$$BSA = 4{\cdot}688W^{0{\cdot}8168 - 0{\cdot}0154 \log W}$$

(where BSA=cm², W=g) produced estimates of BSA which were so accurate that the advantage of the SAP-W-H equation was 'practically reduced to insignificance'. Her SAP-W equation produced estimates of BSA that were significantly more accurate than conventional equations that use both W and H.

Using a formula relating BSA to W alone means that values for BSA can be read directly from a simple table, such as the one shown. This carries the advantages of speed and convenience and minimising the possibility of error, without any reduction of accuracy of the estimation.

Chart to estimate surface area from body weight

Weight (kg)	BSA (m^2)	Weight (kg)	BSA (m^2)	Weight (kg)	BSA (m^2)
0·4	0·049	8	0·422	40	1·27
0·5	0·058	9	0·458	42	1·32
0·6	0·066	10	0·493	44	1·36
0·7	0·074	11	0·526	46	1·40
0·8	0·082	12	0·559	48	1·44
0·9	0·089	13	0·591	50	1·48
1	0·096	14	0·622	52	1·52
1·2	0·110	15	0·652	54	1·56
1·4	0·123	16	0·681	56	1·60
1·6	0·135	17	0·710	58	1·63
1·8	0·147	18	0·739	60	1·67
2	0·159	19	0·767	62	1·71
2·2	0·170	20	0·794	64	1·75
2·4	0·181	21	0·821	66	1·78
2·6	0·191	22	0·848	68	1·82
2·8	0·201	23	0·874	70	1·85
3	0·212	24	0·900	72	1·89
3·2	0·222	25	0·925	74	1·92
3·4	0·231	26	0·950	76	1·96
3·6	0·241	27	0·975	78	1·99
3·8	0·250	28	0·999	80	2·03
4	0·259	29	1·02	82	2·06
4·5	0·282	30	1·05	84	2·09
5	0·304	32	1·09	88	2·16
5·5	0·325	34	1·14	92	2·22
6	0·345	36	1·19	96	2·29
7	0·384	38	1·23	100	2·35

A further advantage of Boyd's formula is that it allows data expressed per unit BSA and per unit W to be calculated easily one from the other, provided W is known. For example, this approach enabled the comparison of glomerular filtration measurements in newborns from 15 different studies, despite the fact that the data had been expressed variously as per m^2 or per kg, because body weight values were also available.[4] None of the studies gave body length measurements, so data conversion would not have been possible using conventional formulas for estimating BSA.

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1 Briars G, Bailey B. Surface area estimation: pocket calculator v nomogram. *Arch Dis Child* 1994; **70:** 246–7.
2 Boyd E. *The growth of the surface area of the human body.* Minneapolis: University of Minnesota Press, 1935.
3 Du Bois D, Du Bois E. A formula to estimate the approximate surface area if height and weight be known. *Arch Intern Med* 1916; **17:** 863–71.
4 Coulthard MG. Maturation of glomerular filtration in preterm and mature babies. *Early Hum Dev* 1985; **11:** 281–92.

Primary sclerosing cholangitis in childhood inflammatory bowel disease

EDITOR,—The strong association between primary sclerosing cholangitis and inflammatory bowel disease, ulcerative colitis in particular, was realised after the introduction of endoscopic retrograde cholangiopancreatography (ERCP).[1] Furthermore it is well known that primary sclerosing cholangitis is an important risk factor for neoplastic transformation in adults.[2] In Swedish adults with ulcerative colitis, primary sclerosing cholangitis is seen in 2·3–6·3%.[3 4] The occurrence of primary sclerosing cholangitis in children, however, is less well documented.[5]

Over the past four years we have treated 31 children with inflammatory bowel disease, 11 of whom were 10 years or younger at diagnosis. There were 14 with ulcerative colitis, eight with indeterminate colitis, and nine with Crohn's disease diagnosed