sunset resting or sleeping, so that energy expenditure is a lot less than normal.

Fortunately pregnant diabetics are usually quite willing not to fast, so their control is not affected during Ramadan. Many patients ask to be allowed to stop their insulin, and where this is not allowed it is difficult to be sure whether the patient is obeying instructions or not.

I think that it may in theory be dangerous to fast during Ramadan but in practice the patients seldom seem to get into any trouble.

J C DAVIDSON

Rumaillah Hospital,
Doha, Qatar

Mycoplasma pneumoniae infection and neurological complications

SIR,—The case reports of Drs J Aidan Twomey and M L E Espir (6 October, p 832), and Dr A B Jones (3 November, p 1144) prompt me to bring to your attention a survey carried out in this laboratory to investigate the association between *Mycoplasma pneumoniae* infection and neurological disease.

We tested sera from 800 patients, presenting with a wide variety of neurological syndromes, for complement-fixing antibodies to *M pneumoniae*. Three patients had rising titres and a further 14 had high (⩾256) static titres suggesting a current or recent infection with the organism. Of these 17 patients (2·1% of the total), 13 were 14 years or younger, and the remaining four were 17, 23, 42, and 49 years old. Six of these patients had meningism; in one case echovirus type 6 was isolated from the faeces and in another there was a measles complement-fixing antibody titre of 256. Four of the 17 patients had aseptic meningitis (one with echovirus type 4 isolated from the faeces) and two others possible aseptic meningitis. The table relates the cases by year of illness to the total cases of *M pneumoniae* serologically diagnosed in this laboratory.

The percentage of *M pneumoniae* infections associated with neurological disease is small (6·7% of the total) and is a constant fraction of the total for each year except in 1973, when we diagnosed only 22 cases. Because only 2·1% of the 800 "neurological" patients showed evidence of infection, it appears unlikely that *M pneumoniae* contributes significantly to the causation of serious neurological disease; in three of our patients there was also evidence of current or recent infection with potentially neurotropic viruses.

An aetiological association with minor neurological illness remains to be proved. Seven of our 17 patients had meningism, for which the causes in children are numerous, and it is not necessary for the organism to infect the central nervous system directly to cause it. In the 10 cases of actual or possible aseptic meningitis causation cannot be proved, but attempts to isolate the organism from CSF would help to resolve the difficulties of interpreting these serological findings. In any case it appears that if *M pneumoniae* does affect the CNS it does so infrequently and in our experience is associated only with mild disease.

G E D URQUHART

Regional Virus Laboratory,
Ruchill Hospital,
Glasgow G20 9NB

M pneumoniae infection and neurological disease 1970-4

Year	No of "neurological" cases tested	No (%) positive	Total No of *M pneumoniae* cases diagnosed	% positive neurological cases out of total diagnosed
1970 (Oct-Dec)	32	2 (6·3)	33	6
1971	216	7 (3·2)	98	7
1972	216	3 (1·4)	40	7·5
1973	214	0	22	0
1974	122	5 (4·1)	62	8
Total	800	17 (2·1)	255	6·7

"Positive" and "diagnosed" = fourfold or greater antibody rise or high titres (⩾256).

SIR,—We wish to add a further case report of neurological complications of mycoplasma infection to those already reported in your columns (6 October, p 832; 3 November, p 1144).

The patient was aged 16 when she presented in February 1977. In December 1976 she had developed a sore throat with swollen lymph nodes in the neck. She was treated with a short course of ampicillin, with complete resolution of her symptoms. In mid-January her symptoms recurred and again resolved with a short course of ampicillin. Her illness responded within 72 hours. In early February she complained of headache and neck stiffness and over a few hours developed nausea and photophobia and became irritable. At this time she was given a course of septrin. Two days later she complained of a "funny sensation" in her right arm and leg and had a grand mal seizure with a focal right-sided onset. Because of this she was admitted to a peripheral hospital, where she was found to be irritable with neck stiffness and a positive Kernig's sign. The cerebrospinal fluid examined at that time was reported as normal. During the following 48 hours she had further grand mal seizures and became comatosed. She was subsequently transferred to the neurology unit. At that time (18 days from the onset of her illness) she was comatosed, responding non-purposely to painful stimuli. Horizontal nystagmus was present on central gaze with intermittent opsoclonus. The brainstem reflexes were preserved. She was hypersalivating and showed forced trismus. Tone was symmetrically decreased and the plantar responses were extensor.

Treatment was started with epanutin 300 mg daily and prednisone 120 mg daily. The only abnormal finding at that time was the presence of symmetrical delta activity on the electroencephalogram. A lumbar puncture on day 20 of her illness showed 12 lymphocytes with normal sugar and protein concentrations. Over the next 21 days she slowly recovered. She had no recollection whatsoever of her illness. A repeat lumbar puncture on day 37 showed protein of 0·64 g/l, 1 lymphocyte, and normal sugar. The EEG returned to normal apart from the presence of moderate bilateral theta and delta components, most marked posteriorly. Titres of complement-fixing antibodies to *Mycoplasma pneumoniae* were (for blood): day 20—1/20; day 37—1/160; December 1977—1/160; June 1978—1/160; December 1978—1/80; (for CSF): day 37—1/20; December 1977—1/20.

Over the two and a half years since she was discharged from hospital, the patient has continued to have grand mal seizures. These were frequent in the first 18 months, but in the past year reasonable control has been obtained with a combination of valproate and carbamazepine. Recent computed tomography scans have been normal. Within a month of her discharge from hospital, however, it was apparent that she had sustained major intellectual impairment, Ten months after her illness her verbal IQ (89) was still only within the dull normal range, even though she had passed five O levels, including English literature and language. Wide variations in subtest results were seen, low scores being noted on general knowledge, attentional span, and mental arithmetic. The subtest scores for reasoning and vocabulary were within the normal range. The performance IQ (99) was found to be average, though this was considered low given that she had been receiving a sixth-form education. Again variations in subtest scores were noted, relatively poor results being found in relation to speed of visual perceptual-motor functioning and visual associative reasoning. Although the patient did not display short-term memory deficits she experienced great difficulty when attempting to learn new material. Her incidental retention of material from short-term memory was similarly extremely poor. The Halstead-Reitan neuropsychological test battery provided evidence of widespread cortical dysfunction, with moderate abnormalities noted bilaterally in the posterior cortical areas. No dysphasic symptoms were encountered. Repeat neuropsychological investigations 24 months after her illness showed no significant or consistent improvements.

In this patient the persistent intellectual difficulties and postencephalitic epilepsy contrast with the favourable outcome of neurological damage resulting from mycoplasmic infection previously reported in the *BMJ* and elsewhere.[1 2]

W J K CUMMING

Department of Neurology,
Manchester Royal Infirmary,
Manchester M13 9WL

C E SKILBECK

Avon Neurological/Stroke Rehabilitation Unit,
Frenchay Hospital,
Bristol BS16 1LE

[1] Taylor, M J, *et al*, *Journal of the American Medical Association*, 1967, **199**, 149.
[2] Jachuck, S J, *et al*, *Postgraduate Medical Journal*, 1975, **51**, 475.

Stress and premature labour

SIR,—I would like through your columns to answer the questions raised by Drs Judith Lumley and Robin Bell (10 November, p 1222) about our paper (18 August, p 411).

We are well aware of the difficulties involved in gestational assessment and we drew evidence from a variety of sources, considering the uterine size on booking, subsequent biparietal diameter on ultrasound scans, bone age, and assessment after delivery before allocating the baby to a particular study group. We demonstrated a continuous trend through three study groups (not merely a difference between two groups), linking prematurity with higher levels of maternal stress. It is unlikely that an error in assessment of a week either way would interfere with this trend.

About 25 women at term were excluded as their labour was induced. In the preterm groups we excluded four women who were sporting Shirodkhar sutures, three with multiple pregnancies, and three Asian ladies from Manchester who spoke no English. No other woman in the preterm group had an obvious "obstetric" cause for premature labour and there seems to be little scope here for a control group as suggested by Drs Lumley and Bell. Besides, we were relating levels of stress to duration of pregnancy and a control group of mothers with "obstetric" causes for preterm labour would be quite inappropriate to this question.