Correspondence

Letters to the Editor should not exceed 500 words.

Rheumatic Fever and Rheumatoid Arthritis in Children

SIR,—The subject of rheumatic fever and rheumatoid arthritis in children is beset with so many problems, and there is so much that we do not know about them, that it would be wise to avoid dogmatic statements about the correct choice of treatment.

As for the use of corticosteroids in rheumatic fever, many would agree with Professor E. G. L. Bywaters (26 June, p. 1655) that they should be reserved for children with carditis. On the basis of controlled studies in Sheffield we believe that they can prevent the development of carditis, and so we prefer to give them in all cases. We would agree that not many children who come to hospital without signs of carditis subsequently develop carditis during their stay in hospital. But some do, and we have several times seen it develop in children receiving salicylates alone, while we have never seen it develop in children receiving corticosteroids.

I feel in addition that a warning about some of the treatments advocated by Professor Bywaters would be in order. He wrote, "Children do not develop peptic ulcers as adults do" with prednisone. I saw a child who required 20 pints (11.4 litres) of blood for her haematemesis as a result of corticosteroid therapy in another hospital. Her frequent complaints of abdominal pain had been ignored.

The dose of aspirin recommended by him for rheumatic fever (4 g. per day for a child of around 10 years old) is more than I personally would dare to recommend a family doctor to give. For rheumatoid arthritis Professor Bywaters recommends salicylates in a dosage sufficient to give a blood level of 20 to 30 mg./100 ml. It would be very difficult for a family doctor to have single or repeated serum-salicylate levels estimated. In any case the upper limit of the range (30 mg./100 ml.) is very near the toxic and dangerous level, and I would not dare to recommend that to a family doctor. Professor Bywaters recommends the use of phenylbutazone—without emphasizing the danger of this treatment.

As for the diagnosis of rheumatoid arthritis, Professor Bywaters wrote that "only a single . . . joint may be involved, and here it is imperative to do an open biopsy to distinguish the disease from tuberculosis." Surely the first step is to carry out a tuberculin test. If it is negative, the arthritis cannot be due to tuberculosis (unless the child has miliary or meningeal tuberculosis or other potent reason for anergy).—I am, etc.,

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Prevention of Rheumatic Fever

SIR,—I have just received a copy of the *Prevention of Initial Attacks and Recurrences of Rheumatic Fever* [see *B.M.J.*, 26 June, p. 1625], and would like to make some comments on same.

On page 2 it states "It is important to remember that sulphonamides, although useful as a continuous prophylaxis for the prevention of recurrences . . . are not effective for the prevention of initial attacks of rheumatic fever if they are given for the treatment of existing streptococcal infection."

For many years I have been swabbing and re-swabbing throats, and in my experience the haemolytic streptococci of the Lancefield groups are penicillin-sensitive, and in the great majority of cases sulphonamide-resistant, and if they are sulphonamide-sensitive they soon develop resistant strains. This never happens when the patient is given penicillin.

Further on the report contains the following statement, "It should be emphasized that in a majority of sore throat patients it is only by means of the bacteriological examination of a throat swab that it is possible to distinguish streptococcal infection from other causes." I am in full agreement with this. In my opinion sulphonamides should not be used in the treatment of the acute sore throat,

unless a swab shows that the causative organism is sensitive to sulphonamide. This so rarely happens that one is not justified in using them.—I am, etc.,

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J. G. COONEY.

REFERENCE

- ¹ Cooney, J. G., *Brit. med. J.*, 1960, 2, 387.

Hookworm Infections Acquired in Britain

SIR,—In an article entitled "Hookworm Disease in Immigrants" which appeared last year in the *B.M.J.*, Drs. S. N. Salem and S. C. Truelove described four cases of hookworm disease in immigrants resident in this country, and in their summary they suggested that in this connexion a public health problem exists on two counts: "First, that there may be many immigrants suffering from hookworm infestation who need to be treated for the sake of their own health. Secondly, the possibility of the spread of hookworm disease in Great Britain must exist while such cases go untreated." Regarding the first count, there is clearly a need for the treatment of infected persons for purely medical reasons; there is now evidence that their treatment is

imperative from the public health aspect as well, for we have recently diagnosed hookworm infection, by faecal examination, in two Pakistani children who were born in this country.

These children, aged 6½ and 7½ years, are resident in the Shires and have never been abroad. Hookworm eggs passed by them were cultured to the infective larval stage and identified as *Ancylostoma*, most probably *A. duodenale*. *Ancylostoma* and *Necator americanus* infections were also diagnosed in some of the adult occupants of the same house, but the parents of the two children were negative.

This is the first record of human hookworm transmission in Great Britain, apart from the well-known account of its occurrence formerly in Cornish tin mines, where climatic conditions underground, temperature, and humidity were favourable for the development of hookworm eggs to the infective larval stage. Hitherto it has been thought that the English climate (above ground) is unfavourable for the soil development of hookworms and other parasitic infections prevalent in tropical and subtropical countries. We must now revise our ideas about this, and we may infer from the present cases that a prolonged spell of warm summer weather, such as was enjoyed in 1964, could provide the conditions necessary for hookworm transmission. The circumstances of the transmission, however, have not been determined in the present instance. The house where the children live has been inspected, and it was found that infection indoors was most unlikely to have occurred; nor did the small backyard provide any clue. It seems most probable that the children became infected by contact with contaminated soil in public playgrounds or picnic sites frequented by parties of immigrants. These possibilities are now being investigated.

It would be of great interest to hear of further cases of hookworm infection in children born of immigrants in this country; such information might give some indication as to the extent to which hookworm transmission is taking place.—We are, etc.,

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REFERENCE

- ¹ Salem, S. N., and Truelove, S. C., *Brit. med. J.*, 1964, 1, 1074.

Bullous Lesions in Acute Barbiturate Intoxication

SIR,—It is interesting to recall, in relation to the correspondence on bullous lesions in acute barbiturate intoxication (Drs. G. W. Beveridge and A. A. H. Lawson, 27 March, p. 835; Dr. G. Warnock, 1 May, p. 1188; Drs. D. I. Freeman and M. Raza, 5 June, p. 1495) that during the war the erythematous areas corresponding to compression sites seen in patients rescued from bombed build-

ings with crush syndrome frequently progressed to blister formation and were mistaken for burns.¹ Depending somewhat on temperature (which determines the metabolic needs and the ischaemic death time of tissues) a quite small compressing force such as the weight of the body will produce ischaemic tissue damage and tissue death. Some cases of prolonged unconsciousness, besides showing skin damage and pressure palsies, may show muscle necrosis and secondary renal tubular damage; such a mechanism is responsible for some of the cases of "lower nephron nephrosis" seen associated with carbon-monoxide poisoning, barbiturate poisoning, prolonged anaesthesia, or other causes of prolonged unconsciousness. It was found that muscle necrosis and the release of myoglobin into the blood stream following return of the circulation seldom appeared under three hours' compression, but after four hours' compression was not uncommon.—I am, etc.,

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REFERENCE

- ¹ Bywaters, E. G. L., *J. Amer. med. Ass.*, 1964, **124**, 1103.

Respiratory Failure

SIR,—Dr. Moran Campbell is to be congratulated on a first-class Goulstonian lecture (5 June, p. 1451). I was surprised that no credit had been given to my work on the subgroups of irreversible airways obstruction first outlined in *Tubercle*.¹ Lack of laboratory facilities prevented me from pursuing this clinical observation until 1960, when, with the help of Drs. Heard and Laws, of the Postgraduate Medical School, we were able to confirm that my Type 2 patient, corresponding to Fletcher's Type B patient, might have little or no anatomical emphysema at necropsy.² Whilst in America in March of this year I showed a film I made in 1958 with Mr. Veronique, our photographer, setting out the two main subgroups and their clinical features. In Denver, Chicago, and New York it was acknowledged that I had anticipated their papers, and Dr. Briscoe, of Belle Vue, paid me the compliment of attempting to acknowledge my work in his paper, but unfortunately it was past the proof stage.—I am, etc.,

Chase Farm Hospital, Enfield, Middx. THOMAS SIMPSON.

REFERENCES

- ¹ Simpson, T., *Tubercle (Lond.)*, 1958, **39**, 307.
² — Heard, B. E., and Laws, J. W., *Thorax*, 1963, **18**, 361.
³ Briscoe, W. A., and Nash, E. S., *Ann. N.Y. Acad. Sci.*, 1965, **121**, 706.

SIR,—Dr. E. J. M. Campbell, in his Goulstonian lecture on respiratory failure (5 June, p. 1451), refers to the recognition "within the past few years" of two types of obstructive airways disease (sometimes labelled "Blue Bloaters" and "Pink Puffers"). He quotes five references between 1963 and 1965, of which, incidentally, only one was taken from the British literature. In fact, the clinical and physiological distinctions between the two types which are listed

by Dr. Campbell were first reported from the London Hospital some eight years ago¹ and subsequently published in 1959.² (One confusing difference is that Campbell refers to the London Hospital Type "A" as Type "B" and vice versa.) Simpson³ has also noted the clinical features of the two types. I confess that my motives for writing this letter comprise an admixture of self-interest and patriotism. I would not wish to detract in any way from Moran Campbell's superb lecture.—I am, etc.,

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REFERENCES

- ¹ Ogilvie, C. M., *Proceedings of the Thoracic Society*, July 1957.
² — *Thorax*, 1959, **14**, 113.
³ Simpson, T., *Tubercle (Lond.)*, 1958, **39**, 307.

Multiple Sclerosis and Poliomyelitis

SIR,—In their paper published on 5 June (p. 1471) Drs. M. J. Eadie and others demonstrate an interesting positive correlation between the average notification rate for poliomyelitis (1921–50) and the death rate from multiple sclerosis (1951–8) for Queensland, New South Wales, Victoria, Tasmania, and New Zealand. This they regard as satisfying one of the prerequisites of the hypothesis of Poskanzer *et al.*,¹ for which "a resemblance between the geographical distributions of multiple sclerosis and clinical poliomyelitis" is necessary.

Whatever the merits of Poskanzer's hypothesis may be, it seems to depend upon the assumption that multiple sclerosis shares with poliomyelitis the remarkable biological characteristic that increasing age of infection is attended by increasing risk of clinical sequelae. Both, it is proposed, are enteric-virus infections which in communities with poor hygiene produce a universal but sub-clinical disease of infancy which goes largely undetected (thus explaining the rarity of the clinical manifestations of either disease in the tropics). In areas with better sanitation exposure occurs later, and neurological sequelae become more commonly manifest.

In order to demonstrate that the epidemiology of poliomyelitis and multiple sclerosis in Australasia are consistent with Poskanzer's hypothesis it is necessary to show that where multiple sclerosis is thought to be rare (Queensland and Western Australia) poliomyelitis tends to attack younger people than where multiple sclerosis is thought to be common (New Zealand and Tasmania). In these circumstances the use of notifications at all ages by Dr. Eadie and his colleagues seems to be an unsuitable correlate.

The Table shows a comparison between the notification rates by age from polio-

myelitis in Johannesburg and Western Australia (where multiple sclerosis is rare) and for England and Wales (where multiple sclerosis is common) for the epidemic year 1947–8. Figures for the nearest epidemic year in Queensland (multiple sclerosis rare) and for New Zealand (multiple sclerosis common) are also shown. Unfortunately age-specific notification rates are not available for Tasmania, nor is information about the proportions of paralytic and non-paralytic cases in each area. In addition to notification rates expressed per 100,000 population, the risk of attack in each age group relative to the average risk for persons of all ages expressed per cent. is shown in parentheses.

The Table demonstrates a wide range in notification rates for all age groups combined, the highest being for Johannesburg, followed by Queensland, New Zealand, Western Australia, and England and Wales in that order. When the relative risks by age are considered, it can be seen that England and Wales and Johannesburg share a similar pattern in which the youngest age group (0–4) had the highest risk. Western Australia, Queensland, and New Zealand shared a different pattern in which the highest relative risk was in the 5–14 age group. Thus there was no relationship immediately after the war between the pattern of relative risk of poliomyelitis by age and the local frequency of multiple sclerosis, such as seems implicit in Poskanzer's hypothesis. What is more, a further comparison made between the age-incidence pattern of the Queensland epidemic of 1937–8² with that of the New Zealand outbreak of 1936–7³ showed no important difference.

If multiple sclerosis is the result of a virus infection, there are some epidemiological reasons for supposing that it may be the occasional clinical consequence of a widespread subclinical infection in early life.⁷ Perhaps the analogy with poliomyelitis should be taken no further than this.—I am, etc.,

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REFERENCES

- ¹ Poskanzer, D. C., Schapira, K., and Miller, H., *Lancet*, 1963, **2**, 917.
² Millar, J. W. S., *J. roy. San. Inst.*, 1949, **69**, 154. (Population taken from the Census of the Union of S. Africa for 1951.)
³ Snow, D. J. R., personal communication to the author. (Population taken from the Census of Western Australia for 1947.)
⁴ Report of the Ministry of Health for the year ended 31 March 1948. On the State of the Public Health. H.M.S.O., London. (Population taken from the estimates of the Registrar-General for 1947.)
⁵ Patrick, P. R., *Med. J. Aust.*, 1963, **2**, 838, and personal communication.
⁶ Canghey, J. E., Poliomyelitis. Papers and discussion presented at the 3rd International Poliomyelitis Conference. Lippincott, Philadelphia. (Population taken from the Census of New Zealand for 1951.)
⁷ McAlpine, D., Lumsden, C. E., and Acheson, E. D., *Multiple Sclerosis, a Reappraisal*, chapters I and II, 1965. Livingstone, Edinburgh.

Poliomyelitis Notification Rates per 100,000 Population

Age	J'burg (Europeans) ² 1947–8	Western Australia ³ 1948	Queensland ⁵ 1951	New Zealand ⁶ 1948	England and Wales ⁴ 1947
0–4	585 (338)	122 (197)	174 (179)	128 (183)	68 (378)
5–14	350 (202)	155 (250)	220 (227)	185 (264)	49 (272)
15+	66 (38)	32 (52)	54 (56)	30 (43)	7 (39)
All ages ..	173 (100)	62 (100)	97 (100)	70 (100)	18 (100)
Multiple sclerosis frequency ⁷ ..	Rare	Rare	Rare	Common	Common

Figures in parenthesis are derived by dividing the appropriate age specific incidence rate by the rate for all ages and multiplying by a hundred.