Cost of Part II M.R.C.P.

Sir,—On scrutinizing application forms for entry to the part II M.R.C.P. (U.K.) examination I was astounded to note that the entry fee has just been increased to £60 “to cover rising cost of administration.” Since in 1972 the fee was £35 this represents an increase of 71%, far outstripping the national rate of inflation over these two years. I should be so lucky as to pass the examination then I would be accorded the dubious privilege of paying the colleges a further £30 for the diploma, making £90 in all for passing part II M.R.C.P.

The money you are charging the young doctors I find it a struggle to bring up my family, pay the mortgage, and yet remain financially solvent. Should the fees continue to escalate in this appalling manner then the financial deterrent to taking the M.R.C.P., which they will impose will mean that future aspirants to membership of the colleges will be confined to those with private means.—I am, etc.,

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Tuberculous Polyradiculitis

Sir,—An association of the Guillain-Barre syndrome with infectious agents is well documented,1-4 and Leneman1 found an association with infective disease in 638 out of 1,100 cases collected from the literature, but he noted a case in which tuberculous granulomata and the tubercle bacillus were demonstrated in the nerve roots at necropsy in a patient who presented with the salient features of the Guillain-Barre syndrome.

A 10-year-old boy who had suffered from a brief febrile illness without neurological or respiratory manifestations two weeks previously was admitted to hospital with difficulty in passing urine. On the following day he developed weakness of the right leg, but his surgical colleagues. The lower limbs were flaccid and areflexic, with absent plantar responses. The upper limbs showed no abnormality. He was very irritable and had generalized hyperesthesia which made sensory testing difficult. Lumbar puncture revealed a viscous, xanthochromic fluid with a protein content of 4-5 g/100 ml and 20 lymphocytes/mm³. During the next 36 hours he developed rapidly ascending paralysis with flaccid, areflexic upper limbs. Lumbar puncture was repeated and yielded C.S.F. with a protein content of 3-5 g/100 ml and 22 lymphocytes/mm³; there were no acid-fast bacilli. His condition remained unchanged for 36 hours but the next day he developed a tachycardia and died suddenly.

At necropsy the interpeduncular fossa and the spinal cord were covered with a thick, gelatinous exudate resembling coagulated plasma. The rest of the surface of the brain appeared normal. No abnormality was seen in coronal sections of the brain and spinal cord other than the subarachnoid coagulum enveloping the brain stem and spinal cord. In histological sections a few small tuberculous granulomata were seen in the exudate used in the interpeduncular and spinal subarachnoid space. The basal and spinal leptomeninges were sparsely infiltrated with mononuclear cells, mostly lymphocytes. The anterior and posterior nerve roots in the cervical, thoracic, and lumbar regions were swollen and infiltrated with mononuclear cells, mainly lymphocytes. Many of the nerve roots showed tuberculous granulomata (some with caseation) and swelling and destruction of myelinated nerve fasciculi with lipid phagocytes in relation to them (see figs). Small perivascular collections of lymphocytes and swelling and vacuolation of some ganglion cells were seen in the posterior root ganglia. Acid-fast bacilli, morphologically Mycobacterium tuberculosis, were seen in some nerve roots and in the exudate in the interpeduncular fossa.

There was no evidence of an inflammatory process in the peripheral nerves examined. Wallerian degeneration of axons and myelin in scattered fibres in peripheral nerves was seen in sections and teased nerve preparations. No evidence, macroscopic or histological, of tuberculous or other disease was seen in the lungs, heart, liver, kidneys, spleen, lymph nodes, or adrenals.

The occurrence of radiculomylopathy in tuberculous meningitis is not well recognized, but has been reported by Wadad.4 In the present case differential involvement of the nerve roots by tuberculous granulomata was unequivocally demonstrated and accounted for the rapid centripetal spread of paralysis simulating the Guillain-Barre syndrome. The possibility of such an association should evidently be borne in mind in the differential diagnosis of a puzzling radiculopathy in communities where tuberculous meningitis is not uncommon.—We are, etc.,

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1 Heymacker, W., and Kernohan, J. W., Medicine, 1949, 28, 19.
2 Miller, H., Proceedings of the Royal Society of Medicine, 1954, 47, 954.
5 Leneman, P., Archives of Internal Medicine, 1966, 118, 139.