ways than one, not least that most refuse to attend child guidance clinics or outpatients. They are not only school refusers but "clinic refusers" and do not come to light easily or early without the regular scrutiny of school attendance registers, sickness notes from parents to teachers, etc. Others are presented ab initio as posing an educational problem needing the advice of educational psychologist or specialist school doctor. Assessment from the developmental point of view, especially as to social maturity, is bound to be involved. Frequently delay is owing to medical or psychological factors. Cases may be classified on a medical basis as disease entities appearing in one form but perhaps remitting as another later.[2] Some children are almost chameleon-like in masquerading neurotic or physical symptoms as social problems. They are liable to obtain a good deal of perverse satisfaction by performing before a succession of professional audiences the mysteries of their case.—I am, etc.,

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Haemolytic Streptococcus Group R Infection

SIR,—We report a case of human infection by group R haemolytic streptococci, which brings the total number of cases described in the literature to five.

The patient, a 47-year-old, healthy butcher engaged in slaughtering pigs, was admitted to hospital acutely ill with fever and periodic confusion. He was semicomatose, with increased muscle tone and a bilateral Babinski's reflex. There was no stiffness of the neck. The temperature was 102·2°F (39°C), pulse rate 76, blood pressure 130/80 mm Hg, and respiration rate 28. The cerebrospinal fluid was turbid and yellowish with a cell count of erythrocytes 50/μl., polymorphonuclears 700/μl., and mononuclears 150 μl., and concentrations of protein of 8·6 g/l. and sugar of less than 0·3 g/l. The blood sugar was 1·5 g/l. Direct microscopy of Gram-stained spinal fluid showed a few bacteria thought to be pneumococci, but later streptococci, identified as haemolytic streptococci Group R, were cultered from both the C.S.F. and blood. They were sensitive to all antibiotics examined except sulphonamides. Primary treatment was with penicillin, streptomycin, chloramphenicol, and sulphamethizole.

Two days after admission respiratory arrest occurred: intermittent positive pressure ventilation was started and the patient placed in a refrigerated room. The condition was complicated by bilateral pneumonia. *Pseudomonas aeruginosa* was cultured from the sputum. Clinically and radiologically the pneumonia cleared in a few days and the patient recovered gradually. Artificial ventilation was stopped after six days and the temperature was normal after 12 days. Thirteen days after admission the patient responded to speech, but there was amnesia for events during the previous 21 days. The E.E.G. four days after admission was exceedingly abnormal with abundant diffuse ½-3 c.p.s. activity and slight suppression of the right hemisphere. One month later the E.E.G. was less abnormal but still characterized by diffuse activity of low frequency.

After eight weeks in hospital the patient was discharged. Psychometric tests indicated organic dysfunction of the brain. Neurological examination was normal. Cold agglutinin titre and parotitis, herpes, and psittacosis complement fixation tests were normal. Coxsackie viruses could not be isolated from the spinal fluid.

Group R streptococci were first reported in 1963,[1] isolated from septicaemic infections in pigs. The first report on streptococci of group R causing infection in man appeared in 1968.[2] Two cases of meningitis and one fatal case of sepsis were described. Evidence was given for the biochemical and serological identity of the streptococci isolated from man with de Moor's reference strain isolated from a pig. Since then one case of purulent meningitis caused by group R streptococci has been published.[3]

There are several similarities in all the cases described. The course of the disease was almost identical, four of the patients had meningoencephalitis, and the patients were men. In three cases the origin of the infection was possibly a skin lesion, and in three cases, including the one described here, there had been contact with pigs. It is remarkable that human infection with group R streptococci has been described only in Denmark. The reason for the small number of reported cases might be that the infection is thought to be due to streptococci of a different group or pneumococci, because of some similarities in the morphology of the colonies and overlapping serological reactions—We are, etc.,

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Toxic Amblyopia from Ibuprofen

SIR,—Two patients with ocular symptoms related to ibuprofen therapy have been reported.[1] The first had defective vision and a bilateral central field defect. Six months after withdrawal of ibuprofen visual acuity returned to normal and the field defects disappeared. The second had a visual disturbance which was thought to be cortical in origin and not a true toxic amblyopia. The same report recorded the findings in 38 patients taking ibuprofen, one of whom seemed to have a true toxic amblyopia. I report here a further case of toxic amblyopia caused by ibuprofen.

A woman aged 57 was referred in November 1971 complaining of failing vision for some months, which her optician had been unable to correct by a change of spectacles. She also described difficulty in distinguishing blues and greens and had made a glaring error in colour-matching when choosing clothes. She had been taking ibuprofen 1,200 mg daily since June 1971 for "fibrositis." The total dose consumed by the time of her first attendance was approximately 168 g. She had also had a short course of troxerutin and an occasional dose of Sonalgin. She took alcohol in moderation once a fortnight. She was a non-smoker. Six years before she had been treated for pulmonary tuberculosis and had developed a rash attributed to PAS.

Corrected visual acuity was 6/24 in each eye: she read N.8 print with difficulty. (Reference to her optician's records showed that her visual acuity in May 1970 had been 6/9 in each eye.) The media were clear and the fundi were normal. Visual field studies were unhelpful, because her concentration and comprehension were inadequate. Static perimetry, as advocated by Foulds and colleagues,[2] was not available. The patient was almost totally colour-blind by the Ishihara test. A grossly abnormal profile was recorded with the Farnsworth-Munsell 100-hue test. E.R.G. and E.O.G. tracings were within normal limits. Blood examination was normal.

Though reliable field studies were impossible, the combination of failing eyesight and an acquired colour vision defect with normal fundi pointed to a diagnosis of toxic amblyopia. She was taken off ibuprofen on 1 December. She did not return for examination until 21 March 1972 when she reported a gradual improvement in eyesight and colour vision. Visual acuity was 6/12 in each eye and she read N.6 print with ease. No errors were made with Ishihara's plates and the Farnsworth-Munsell test showed a pronounced improvement. On 3 May the visual acuity was 6/9 right and 6/12 left and she could read N.5 print. The Farnsworth-Munsell test showed a further improvement.

The diagnosis of toxic amblyopia rests on visual failure unaccompanied by visible retinal abnormality, a characteristic field defect, and an acquired defect of colour vision. Though reliable visual field studies were impossible in this patient the colour vision defect was easily demonstrated. In toxic amblyopia the Farnsworth-Munsell test characteristically demonstrates a concentration of errors in a bipolar fashion, the axis extending between the yellow/red (10 o'clock) and purple/blue (4 o'clock) regions. This characteristic profile was not seen in this case, probably because severer degrees of toxic colour-blindness affect many wavelengths. The typical bipolar shape may emerge only during the phase of recovery.[2,3] The recovery of vision in this patient was almost complete, but a continuing measurable improvement in the colour sense may be detected.

Drugs which can cause ocular side effects, including toxic amblyopia, have been listed.[4,5] Ibuprofen should now find a place in the list.

I am grateful to Mr. I. M. Strachan for interpreting the electrodiagnostic tests, to Mrs. M. Saynor for technical assistance, and to Mrs. C. Duffy for secretarial help.—I am, etc.,

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Payment by Colour

SIR,—Having followed with interest the letters on payment by colour I feel that there are one or two pertinent facts which should be brought to your notice, none of which have been adequately emphasized in the *B.M.J.* thus far.

Firstly, the Republic of South Africa is divided up into areas, each area being under the control of either part- or full-time district surgeons. This also applies to the Bantu homelands. These medical officers are paid by the Government and are responsible for the well-being of the population in their areas. A short summary of how medical services are run in my area, which borders on