to relative hypoglycaemia (normal ratio) and one due to glycolysis (lowered ratio) as occurs in tuberculous meningitis. We have found here that exceptions do occasionally occur: we have seen a normal ratio in tuberculous meningitis with a normal C.S.F. sugar, and we have further seen cases of aseptic meningitis with lowered ratios, C.S.F.-sugar levels being either normal or reduced.

We accept the view of Miller *et al.*² that a normal C.S.F. sugar must never be used as a major argument against a diagnosis of tuberculous meningitis, and consider that the importance of finding a low C.S.F. sugar in non-pyogenic meningitis is that it should prompt a thorough search of the C.S.F. for the tubercle bacillus, the identification of which is, after all, the only unequivocal evidence of tuberculous infection of the meninges. Failure to find the organism by direct examination should not delay the institution of anti-tuberculous chemotherapy (not intrathecally initially) in any case where the clinical suspicion of tuberculous meningitis is high.—We are, etc.,

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REFERENCES

- ¹ Hendry, E., *Arch. Dis. Child.*, 1939, 14, 159.
- ² Miller, F. J. W., Seal, R. M. E., and Taylor, M., *Tuberculosis in Children*, 1963, p. 401. J. & A. Churchill, London.

Dangers of Tetanus Antitoxin

SIR,—In the recent television programme "Postgraduate Medicine" (30 June), one of the contributors stated that provided no untoward effects followed a test dose of 0.1 ml. (150 units) of tetanus antitoxin made with horse serum within 30 minutes, it was safe to proceed with the usual dose of 1,500 units for tetanus prophylaxis in the injured patient. As no contrary opinion to this still widely held, though erroneous, view was expressed I feel that the case against the use of equine-derived tetanus antitoxin requires restating.

In 1963 I published full details of a patient who had developed serum neuritis after a test dose of 175 units of equine-derived tetanus antitoxin, and considerable muscle weakness and wasting with sensory loss was still present some eight months after this injection.¹ This case was not regarded as "a collector's piece" but was presented in support of your plea for universal active immunization with tetanus toxoid.² A few weeks later I saw a further patient who developed serum neuritis after a prophylactic injection of 1,500 units of tetanus antitoxin, though fortunately there are now no residual signs of neurological dysfunction. However, as this ex-Service man had previously received adequate tetanus toxoid for maintaining active immunity, it is obvious that this injection of tetanus antitoxin was completely unnecessary.

Further, in this television programme no mention was made of the serious neurological complications which may occur following a prophylactic injection of tetanus antitoxin made with horse serum. An extensive review of the literature undertaken by Bardenwerper in 1962 revealed 130 cases of serum neuritis.³ There is a predilection for involvement of the brachial plexus, particularly C5 and C6, though the lumbar plexus may also be affected, as was the case in my two patients. Ocular, facial, intercostal, and diaphragmatic

palsies have been reported and death may occur as a result of encephalomyelitis and respiratory paralysis. It is also well to recall that an estimated 15% to 30% of persons may develop other hypersensitivity reactions.³

It has been strongly recommended that the management of all tetanus-prone patients, including those who have never been given tetanus toxoid, should comprise adequate excision of wounds, antibiotics for a period of five to seven days, and active immunization with adsorbed tetanus toxoid, and that the use of tetanus antitoxin as a prophylactic should be discontinued.⁴ However, in view of the fact that a prophylactic injection of tetanus antitoxin made with horse serum is still being advocated and practised, I make no excuse for reiterating my view that adequate supplies of human tetanus antitoxin must be made available for general use in place of equine-derived tetanus antitoxin.¹ Human gamma-globulin is non-antigenic in man, so does not produce hypersensitivity reactions, and, although human tetanus antitoxin has been in use in certain centres for some years,⁵ supplies are still limited.

On the basis of evidence submitted and in the knowledge that even a test dose of tetanus antitoxin made with horse serum may result in serious neurological complications with permanent sequelae, surely it is not high time that, having been justly accorded its rightful honoured place in the history of medicine, equine-derived tetanus antitoxin now be banished once and for all from hospital casualty departments and doctors' surgeries? —I am, etc.,

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REFERENCES

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- ² *Ibid.*, 1963, 1, 902.
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- ⁴ Hadgraft, J. W., Qvist, G., Ramsay, A. M., and Thomas, P. K., *Brit. med. J.*, 1964, 1, 768.
- ⁵ Cox, C. A., Knowelden, J., and Sharrard, W. J. W., *ibid.*, 1963, 2, 1360.
- ⁶ Ellis, M., *ibid.*, 1963, 1, 1123.

* For statement from Ministry of Health on prevention of tetanus see p. 243.—ED., *B.M.J.*

Treatment of Cyanide Poisoning

SIR,—We refer to the Medical Memorandum "Oxygen Therapy and Cyanide Poisoning" (4 July, p. 33). We are all engaged in whole-time industrial medical practice, and this case is of interest to us since we have a certain experience of cyanide poisoning. We think it would be unfortunate if any impression was to gain ground that oxygen *per se* was of any significant value in the treatment of acute cyanide poisoning. The correct treatment, as mentioned indeed by the authors of the article, is the nitrite and thiosulphate method described by Chen and Rose.¹

On our joint experience over a long period of years, this antidote is immediately effective and normally produces rapid recovery. We do not dispute, with mildly affected cases, that spontaneous recovery is obviously possible. In such cases, and without using the antidote, supportive measures as described in your memorandum would do no positive harm and might conceivably be of some use. One factor in the survival of the case de-

scribed is probably that the solution which he drank was not one of potassium cyanide but of potassium ferricyanide ($K_3Fe(CN)_6$). To the best of our knowledge potassium cyanide is never used in photographic reducing work. On the other hand, the ferricyanide salt is in common use, and in dilution is a yellow colour resembling lemonade. If this hypothesis is correct the stomach of the patient described contained the ferricyanide salt, which is very much less toxic than potassium cyanide and liberates the CN ion extremely slowly. This probably accounts for the long period of survival prior to specific treatment, and possibly for the long period to regain consciousness afterwards. It is probable, if the stomach had been washed out as soon as the patient was received, that he would have recovered much more quickly.

Cyanides are in increasing use in industry of all sorts, and we would make a plea that major hospitals in industrial centres should maintain a stock of nitrite/thiosulphate antidote. The antidote keeps for a long time (at least a year), and if a small stock is kept and turned over annually much more efficacious treatment of cases such as the one described would be possible.

Another important point is confirmation of the diagnosis of cyanide poisoning. It is our practice to collect a specimen of blood from a vein just before the antidote is given. Enough should be taken to completely fill a 20-ml. fluoride bottle. Home Office Forensic Science Laboratories in each region have facilities for reliable cyanide estimations. Although the results will not be available in time to assist in the immediate diagnosis and treatment, at least confirmation or otherwise that the case was in fact one of cyanide poisoning, with the blood-cyanide level, will later be known. This will be useful in assessing the value of the treatment given.—We are, etc.,

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REFERENCE

- ¹ Chen, K. K., and Rose, C. L., *J. Amer. med. Ass.*, 1952, 149, 113.

Annular Detachment of the Cervix

SIR,—Once again this fascinating condition was described in your pages by Mr. K. Amatayakul (20 June, p. 1613). Once again we were asked to believe that the cervix suddenly became responsible for obstructing the course of labour after dilating to three fingers in the face of normal co-ordinated uterine action.

Certain significant points emerged from a review of cases reported fully in the literature.¹ These have been listed for brevity: (1) Uterine action and dilatation of the cervix progressed normally until 2–3 fingers' dilatation. (2) Prolonged labour (over 24 hours) was the rule, but there were many exceptions. (3) Bearing down by the mother occurred before full dilatation of the cervix. (4) The detached cervix showed no histological abnormality. (5) Labour began at or beyond the fortieth week of pregnancy. (6) Babies were large (over 8 lb.—3.6 kg.). (7) Mothers were usually primigravidae, but not elderly (average age 29). (8) Cephalo-pelvic disproportion was undoubtedly associated with detachment of the cervix but not of the annular variety.