Drugs such as thiopentone, cyclopropane, and curare are on the whole unsuited to the procedure under consideration, since, although they may or may not produce a rise of uterine tone, they certainly do not relax the uterus.

On the other hand, a spinal or extradural anaesthetic given to a height sufficient to abolish uterine sensation does cause a considerably "heightened reactivity" to stimuli of the uterus (Malpas, 1944; Lull and Hingson, 1948). As a result not only is the risk of trauma both to the foetus and to the mother greatly increased, but, in addition, foetal anoxia may arise owing to placental ischaemia. This form of anaesthesia is therefore entirely contraindicated in breech extraction.

Considerations with Regard to the Third Stage.—A possible argument against the use of ether in breech extraction is that the resultant lowering of uterine tone might predispose to serious post-partum haemorrhage. It is probable, however, that this complication has been over-emphasized in the past; certainly in our experience it has rarely been observed. This may be due to the fact that it has always been our policy to give 0.5 mg. of ergometrine intravenously following the birth of the head. At all events, the danger of a contracted uterus during breech extraction would seem considerably to outweigh that of a relaxed uterus after delivery has been completed.

Summary

Assisted breech delivery and breech extraction are two very distinct obstetric operations requiring different anaesthetic techniques.

The special considerations involved in anaesthesia for such deliveries have been reviewed.

In assisted breech delivery it is recommended that a perineotomy be performed under local infiltration analgesia, inhalation anaesthesia being limited to the delivery of the arms and head.

In breech extraction a high degree of uterine relaxation is necessary. For this reason a deep ether anaesthetic is the only one which may justifiably be employed.

Spinal or extradural anaesthetics are not the most suitable for assisted breech deliveries and are absolutely contraindicated in breech extraction.

Close co-operation between obstetrician and anaesthetist is essential if the best results are to be obtained in this branch of obstetrics.

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Conservancy Systems at Collieries is the title of a medical memorandum recently issued by the National Coal Board. It is intended as a general guide for colliery managers. After stating the legal requirements, the memorandum describes various installations for use above or below ground. The standard of sanitation in mines is, not surprisingly, below that in other industries. Both health and morale would benefit if it were improved.

DANGERS OF POLYPHARMACEUTICAL PREPARATIONS CONTAINING FOLIC ACID

BY

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The inclusion of folic acid in proprietary preparations was deplored by Davidson (1948), but, as can be seen from the examination of recent advertising matter, the practice continues. The purpose of this note is to draw attention to the difficulties which may arise when anti-anaemia treatment is undertaken without proper investigation of the case and with preparations of the "blunderbuss" type.

Case Report

A 46-year-old housewife first complained of stiffness of her knees in October, 1952. Treatment with short-wave diathermy was given after she had attended orthopaedic and medical dispensaries. In January, 1953, she developed a short febrile illness associated with vomiting, anorexia, and loss of weight, from which she did not fully recover. In February, at another medical dispensary, no abnormality was found on examination, with the exception of a haemoglobin level of 48% (Sahli). A course of iron was recommended, and the patient was requested to attend for further investigations. Her family doctor gave her a proprietary preparation of iron, and her general condition improved greatly. Her appetite returned to normal and her weight increased, but the stiffness of her legs began to worsen rapidly. She did not return for investigation, but in May, 1953, she fell and was admitted to hospital for treatment of an infected haematoma. After 10 days in bed she was found to be unable to rise or walk, and was transferred to a medical ward for investigation.

On examination the patient was found to have a spastic paraplegia with great muscular weakness, bilateral extensor plantar responses, and complete loss of posterior column sensation in both legs. There was no upset of the function of bladder or bowels. Histamine-fast achlorhydria was demonstrated. Blood examination showed: haemoglobin, 12.3 g.%; red cells, 3,600,000 per c.mm.; white cells, 8,000 per c.mm.; P.C.V., 38%; M.C.V., 105.6 μ^3 ; M.C.H.C., 32.1%. Sternal marrow biopsy showed appearances suggesting a partially treated anaemia resulting from deficiency of the haemopoietic principle. Lumbar puncture and radio-scopic of the lumbar spine showed no abnormality.

A diagnosis of subacute combined degeneration of the cord was made, and treatment with "anahaemin" and vitamin B₁₂ was started (4 ml. of anahaemin on alternate days, and 100 μ g. of "cytamen" daily). Haematological and neurological improvement was rapid, and five weeks later the patient was walking well enough to continue treatment as an out-patient. When last seen she had only very slight ataxia, and examination of the blood and marrow showed normal appearances.

It was found that she had been treated originally with capsules containing iron, vitamin B, extract of raw liver, and folic acid. The amount of folic acid present in each capsule was 1 mg., and she had consumed 3 mg. of folic acid daily for 38 days.

Discussion

The literature on the results of treatment of cases of Addisonian anaemia with folic acid was reviewed by Davidson (1948) and by Israëls and Wilkinson (1949). These authors concluded that folic acid had no place in the treatment of pernicious anaemia. The haematological state not

infrequently remains unsatisfactory (Wilkinson, 1948), and the neurological state is not prevented from deteriorating (Davidson, 1948; Israëls and Wilkinson, 1949). Since then Fuld (1950) and Schwartz *et al.* (1950) have reported poor haematological and neurological results with folic acid therapy. The latter reported 72 cases followed for three and a half years and treated with 5 mg. of folic acid daily: 23 cases developed cord degeneration, in 23 the red cell count and marrow showed relapse, and in 9 cases both occurred. Subacute combined degeneration of the cord has been reported as occurring with doses varying between 5 and 25 mg. daily. In the present case 3 mg. daily was enough to produce a considerable improvement in the blood condition, but did not prevent the rapid development of paraplegia.

In at least two large series folic acid has been given to normal subjects and to cases of anaemia other than pernicious anaemia (Weissberg *et al.*, 1950; Harvey *et al.*, 1950). There was no subsequent development of cord degeneration, and it is thus unlikely that folic acid has any direct toxic effect. The following reports of authors who have had the opportunity of observing large numbers of cases of Addisonian anaemia contain statements which suggest that they have not rejected the possibility that folic acid actually precipitates cord disease. Thus the explosive character of the onset of cord disease, as well as its rapid progression, have been more often noted after folic acid therapy, and have been contrasted with the more insidious disease previously described (Davidson, 1948; Wilkinson, 1948; Ross *et al.*, 1948; Jacobson *et al.*, 1948). The present case also showed these more acute features.

The experience of Conley and Krevans (1951) is interesting, being more akin to the present case. These authors saw 10 patients in 1950 who were suffering from undiagnosed Addisonian anaemia. Five had only neurological symptoms with no associated anaemia, and on this account various diagnoses, such as disseminated sclerosis and syphilis, were entertained. It was established that four out of the five had been given multivitamin preparations containing folic acid. The patients were not aware that they had received anti-anaemic treatment, and in two cases the physician prescribing the capsules was unaware that they contained folic acid. In all cases an excellent response to vitamin B₁₂ was obtained. It was therefore suggested that in all doubtful cases a marked improvement in nervous system signs and symptoms following the injection of vitamin B₁₂ provides strong support for a diagnosis of Addisonian anaemia.

The arguments against the inclusion of folic acid in polypharmaceutical preparations may be summed up as follows: (1) Folic acid temporarily improves the blood picture, and may restore it to normal. The physician is then faced with a disease of the central nervous system, the misdiagnosis of which is likely, as subacute combined degeneration of the cord without anaemia is very uncommon. (2) The relief of the general symptoms of pernicious anaemia often reassures the patient and the doctor, and masks the deterioration of the nervous system. (3) Folic acid deficiency occurs infrequently, and there is no need for this substance in multivitamin or iron preparations. (4) The question whether folic acid actually precipitates cord degeneration is still uncertain.

Summary

A case is described in which a proprietary preparation of iron containing folic acid was given to a patient with an anaemia which had not been accurately diagnosed. In fact, the patient was suffering from Addisonian anaemia. Deceptive improvement in the blood condition occurred, but was accompanied by the rapid development of combined degeneration of the cord.

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Medical Memoranda

Carbon Dioxide Retention Simulating Curarization

Numerous reports have appeared of persistent curarization after the use of one or other of the relaxants; the following case therefore seems worthy of description.

CASE REPORT

The patient, a child aged 6, weighing 42 lb. (19 kg.), had undergone a laparotomy for the removal of a polyp from the sigmoid colon.

The premedication consisted of pentobarbitone sodium, 2 gr. (0.13 g.), and atropine, 1/100 gr. (0.65 mg.), following which the child arrived at the theatre asleep. Anaesthesia was maintained with nitrous oxide and oxygen and a trace of ether, a size 3 endotracheal tube having been inserted. The flow was at the rate of 3 litres of nitrous oxide and 2 litres of oxygen a minute. No absorber was incorporated. Just before opening the peritoneum 2 mg. of decamethonium iodide was given intravenously. After a brief period of apnoea respiration returned and was accompanied by a satisfactory degree of relaxation. (The respiration was assisted throughout.) Some 15 minutes after the first dose a second injection of 2 mg. of decamethonium iodide was given. This time the apnoea persisted for 15 minutes, and at the time of the skin closure only minimal diaphragmatic respiration was present.

After an interval of five minutes, respiratory activity, although minimal, appeared just sufficient to keep the child a good colour, provided pure oxygen was given through a T-piece connected to the endotracheal tube. The child was therefore returned to bed with a flow of 5 litres of oxygen a minute passing through the T-piece, and inflation by means of a bag was carried out for one-minute periods every five minutes.

Respiratory activity did not increase, although the child at all times remained a very good colour, there being no evidence of suboxygenation. After two hours it was noted that, if anything, respiratory activity was decreasing again, but that there was some improvement after each period of ventilation. There was no response at all to an intravenous dose of 2 ml. of nikethamide. The delay in recovery of respiration had until this time been attributed to a residual effect of the decamethonium iodide; but it was noted that such respiratory effort as was present was not marked by any urgency, neither was it accompanied by a tracheal tug, both of which features are usually present in cases of residual curarization.

Three hours after the injection of the second dose of decamethonium iodide, on the supposition that carbon dioxide accumulation might be the underlying cause of the respiratory depression, the child was vigorously hyperventilated through an absorber for a period of three minutes. The absorber very rapidly became intensely hot, indicating considerable elimination of carbon dioxide. Promptly following this hyperventilation the child's respiration was noted