a third at the right temporo-sphenoidal lobe. Small cerebellar cone round the brain-stem. Section of brain revealed congestion of grey matter and small congested vessels in the white matter. After fixation, sections of brain revealed a fair number of petechial haemorrhages, more marked in the white matter. Macroscopically the grey matter of the brain was congested, giving a pinkish hue. Many small congested vessels in white matter.

Microscopical Examination.—The lesions present were not numerous, therefore careful searching was necessary. Some blocks of brain tissue did not reveal any lesion. No particular part of the brain was especially involved, the lesions being irregularly distributed throughout the brain substance. They were perhaps more obvious at the junction of white and grey matter than anywhere else in the cortex. Main lesions in intracerebral vessels, consisting of endothelial swelling associated with ante-mortem thrombi, partially occluding the lumen of the vessels. Some of the thrombi were infiltrated with polymorphonuclear leucocytes, many of them eosinophils. Occasionally there was evidence of vascular rupture, with red blood cells and eosinophilic material in the Virchow-Robin spaces. No obvious change in nerve cells themselves, and there was absence of perivascular cuffing and other signs of encephalitis. Emulsions of the brain were injected into rabbits and mice with negative results.

Treatment

This consisted of repeated lumbar punctures. Adrenaline was given in doses of 1 c.cm. intramuscularly four-hourly, and sodium thio-sulphate was injected intravenously. Venesection was also done. Treatment to be successful must be started as soon as possible, and therefore it is of great importance that this complication of arsenical therapy be quickly recognized. In every case of sudden cerebral disaster in a young adult the question of previous arsenical treatment should be considered.

Discussion

This picture conforms to the usual type previously described in the literature—severe cerebral symptoms, with coma and epileptiform convulsions. The onset generally occurs after the second or third injection of an arsenobenzene preparation, and can happen as readily in the healthy subject as in the unfit. The main features in the present case are the haemorrhages, and extreme congestion of vessels of white matter coupled with blocking of cerebral capillaries and occasional rupture. There was no evidence of non-haemorrhagic perivascular areas of necrosis and demyelination as observed by Russell in her cases. This may have been due to the fact that this patient did not survive long enough for these findings to develop. Two of Russell's cases in which these changes were present survived for four days after onset of acute symptoms. The third of her patients died within six hours; evidence of necrosis and demyelination in the central nervous system was scanty.

The causal factor in arsenical encephalopathy is unknown, and seems to be unrelated to the arsenic radical, as poisoning by arsenic in an inorganic form does not give rise to such cerebral symptoms. It also seems unlikely that overdosage is at fault, as in most recorded cases symptoms have arisen shortly after the second or third injection. It is apparently not of the nature of a Herxheimer reaction, because cases are recorded (Glaser, Imerman, and Imerman, 1935) in which cerebral complications followed arsenical therapy in non-syphilitic subjects.

As symptoms occur most frequently after the second or third injection of an arsenical preparation it seems possible that the cerebral phenomena may be in the nature of an allergic reaction, although this does not seem to be of a permanent nature, as Lees mentions a case in which treatment with an arsenobenzene compound administered intramuscularly was resumed after recovery from haemorrhagic encephalitis without ill effects. There appears to be some relation between purpuric eruptions due to arsenobenzene preparations and haemorrhagic encephalitis (Winter, 1940). It may be that both are due to a toxic action of the benzene radical on capillaries. Organic arsenicals are well known as a cause of thrombocytopenic purpura, and it is possible that in some cases of haemorrhagic encephalopathy the toxic action is due to disintegration of arsenobenzene compounds in the blood, with resulting thrombocytopenia and capillary damage. The case described by Winter in which purpura was a feature could fit in with such a theory. I have been unable to find any reference in the literature to the platelet count in arsenical encephalitis.

The raised cerebrospinal-fluid protein in this case, with a normal cell content, is unusual, as was the increase in the C.S.F. pressure. In most cases described the protein has been

normal and the pressure also normal. In the present case, as in others described, the Wassermann reaction was positive in the blood but negative in the C.S.F.

In most cases previously recorded, cerebral complications of arsenobenzene therapy are referred to as haemorrhagic encephalitis. In view of the clinical picture and pathological findings, encephalopathy seems a more fitting description to apply to this complication.

Summary

A case of arsenical encephalopathy with pathological findings is described.

Attention is drawn to the need for early recognition of this condition, as treatment seems unavailing unless instituted in the first few hours after onset.

A short review of the literature is given. No adequate aetiology has been found to account for cerebral complications following arsenobenzene therapy.

I wish to thank Lieut.-Col. G. L. Montgomery, R.A.M.C., Assistant Director of Pathology, Scottish Command, for the post-mortem findings in this case, and Dr. A. A. F. Peel for advice and criticism; also the Department of Health for Scotland for permission to publish this report.

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ETHYL CHLORIDE ANALGESIA

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The term "analgesia" implies the loss of pain sense without the loss of consciousness or the sense of touch (Guedel, 1937). It is generally accepted that during induction of anaesthesia by inhalation a phase of analgesia is passed through (Guedel, 1937). This phase starts at the beginning of induction with a relative blunting of the sense of pain (relative analgesia), and passes to a complete state of analgesia (total analgesia). The analgesic phase ends with the loss of consciousness of the patient, some drugs giving a longer and better analgesic period than others. These drugs have been used for a variety of purposes: chloroform analgesia in obstetrics; trichlorethylene analgesia for the relief of pain in tic douloureux (Striker *et al.*, 1935) and more recently for painful dressings; nitrous oxide in combination with air or oxygen in midwifery and dental work (Minnitt, 1934).

- The limitation of the method is the difficulty of maintaining the analgesic state without slipping into the second and most dangerous stage of anaesthesia with its attendant risk of ventricular fibrillation. Lincoln (1941) has made an advance in the production of analgesia by using ethyl chloride in combination with nitrous oxide by the nasal route. It was considered that it might be of advantage to investigate the effects of ethyl chloride alone without using nitrous oxide. This we have done with success in 90 cases, the majority being dental extractions. All these patients were soldiers, many of whom are anaesthetic-resistant subjects.

The Method

The apparatus required is two tubes of ethyl chloride, a dental prop, some vaseline, and a roll of gauze. The lips are smeared with vaseline and a dental prop is inserted. An oblong of gauze, about 2 in. by 6 in., is crumpled and placed between the teeth, but not so far back as to cause gagging. The patient is asked to breathe to-and-fro through his mouth alone. He is told that the vapour will not put him to sleep but will

prevent pain. Ethyl chloride is then sprayed on to the gauze on and off for 45 seconds. The amounts we have used vary between 4 and 9 c.cm., the average being about 6 c.cm. If the eyelids are seen to flicker the patient is ready; otherwise it is better to rely on the time period entirely. The gauze is pushed aside and the operation completed. The gauze is then removed and the patient asked to take one or two deep breaths; by the time he has rinsed his mouth out he is ready to leave the chair.

Of the 90 cases 70 were dental extractions and 20 were minor operative procedures, mainly incisions of abscesses. In all cases analgesia was achieved; there were no post-operative complications. The dental cases were not confined to single extractions of upper and lower incisors, as 33% were molars or pre-molars. In several cases a number of teeth were removed, in one case seven. The majority of cases were those in which local anaesthesia was contraindicated. Many of the cases were those in which the patient was a difficult subject, being uncooperative and fearful, as this was his first visit to a dental surgery.

Comment

The advantages of ethyl chloride are many. It is a safe and rapid agent (Hewer, 1930) requiring no apparatus. The method is simple to learn and easy to apply. By Lincoln's (1941) technique of oral breathing induction is pleasant; there is the psychological benefit of having no mask, and there is no struggling—not even by the anaesthetic-resistant patient.

Total analgesia lasts from 60 to 120 seconds; recovery is as rapid as with nitrous oxide. There are no after-effects: we have had none, and in a series of 2,200 cases only 6 complained of nausea (Lincoln, 1941).

It would seem that ethyl chloride analgesia might be useful, particularly in wartime, in the painless extraction of teeth and for such simple operative procedures as opening superficial abscesses. It is cheap, safe, and portable, with rapid induction and rapid recovery.

Summary of Conclusions

Ethyl chloride alone by oral breathing will produce analgesia—that is, a loss of sense of pain with the retention of consciousness.

The method is simple, safe, and rapid, and can for short procedures compare in every respect with nitrous oxide analgesia.

We wish to thank Dr. C. Langton Hewer, without whose assistance this article would not have been published; and Lieut.-Col. Ashley Daly, Adviser in Anaesthetics to the Army, for permission to publish it.

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Medical Memoranda

Post-scarlatinal Thrombophlebitis Migrans

Thrombophlebitis following scarlet fever is a rare event. The last reported case appears to be that of Rolleston and Ronaldson (1940). Previously Huenekens and Siperstein had recorded a case in 1923. These authors give a full review of the literature, in which they could find only 9 cases reported since 1882.

CASE HISTORY

A boy aged 11 was admitted to the East Malling Isolation Hospital on Feb. 13, 1942. He had complained of a sore throat for the past two days, and on admission showed the usual signs of mild scarlet fever. The temperature was 100°, the rash was typical, and there was marked circumoral pallor. The throat was sore, with exudate on the tonsils, and some cervical adenitis was present. The next day his temperature was normal, and he made a rapid recovery from the acute stage. He began to peel on the thirteenth day.

On Feb. 25, 12 days after admission, the cervical glands became more enlarged and there was slight pyrexia. He was given sulphapyridine 2 days later, and the temperature became normal in 48 hours. The sulphapyridine was continued for a further 5 days until a total of 11 g. had been given. At the end of this time the swelling of the glands was much less and the general condition satisfactory. Two days after this, on March 7, his temperature rose again, but there were no signs or symptoms to account for it. However, 48 hours later a painful red swelling appeared on the dorsum of the left foot, extending from the base of the great toe for 3 in. upwards.

This was 25 days after the onset of the rash. He was again given sulphapyridine. Three days later inflamed veins could be seen and felt extending from the inflamed area on the left foot up to the knee. The leg was oedematous below the knee, and the popliteal vein was tender. The tongue became furred and the boy looked ill. The next day the patient was better. The temperature was normal and there was less oedema of the left leg. A further 11 g. of sulphapyridine had been given, and the dose was reduced to 0.5 g. t.d.s.

Twenty-four hours later, however, on March 14, the temperature rose again to 100°. On examination the left leg was still improving, but the right popliteal vein was tender and there was obvious phlebitis of the superficial veins below the right knee. On the following day there was oedema of the right leg below the knee, and the right internal saphenous vein was tender below the groin. The temperature rose to 101° and the patient was seriously ill. Antiscarlatinal serum, 3,000 U.S.A. units (B.W. & Co.), was injected intramuscularly. The next morning he was better, having had a good night, and his temperature had fallen to 100°. The left leg had recovered, but the whole of the right leg was oedematous. The girth of the middle of the right thigh was $2\frac{1}{2}$ in. greater than that of the left. The right internal saphenous vein was tender and inflamed along the whole of its course, and there was tenderness in the right iliac fossa.

The patient by now had taken 33 g. of sulphapyridine since the beginning of his illness, and a blood count was made by Dr. Griffiths of the Kent County Laboratory. This showed: R.B.C., 3,860,000; Hb, 71%; C.I., 0.92; W.B.C., 15,800—polymorphs 90%, lymphocytes 7.5%, monocytes 2.5%. A blood culture proved sterile. Sulphapyridine was continued (3 g. a day), and on March 18 the girth of the right thigh was 3 in. greater than that of the left. The oedema had spread upwards over the right iliac fossa and the right loin. Enlarged superficial veins were seen over this area. The next day the patient was better. The temperature was falling and the tongue was getting cleaner. Three days later, on March 22, the temperature was normal and the oedema over the right loin and iliac fossa was much less.

On March 24 sulphapyridine was discontinued after a total of 41 g. had been given in 3 weeks without toxic effect. The patient was improving rapidly. The tongue was clean, and there was no pain or tenderness in either leg. The girth of the right thigh was only $3\frac{1}{4}$ in. greater than that of the left. A fortnight later all oedema had gone and the patient was very well. After a further fortnight's convalescence he was allowed to get up, having had a normal temperature for a month. He went home one week later, on April 28, having been in hospital 10½ weeks.

When seen on Oct. 7, 1942, he was in excellent health. The only complaint was an occasional feeling of "heaviness" in both legs, especially the right, but he was able to run and play games. There was no oedema, but the girth of the right thigh was still $1\frac{1}{2}$ in. greater than that of the left.

COMMENT

A case of thrombophlebitis migrans occurring on the twenty-fifth day after scarlet fever is described. The veins of both legs and the right external iliac vein were involved. Improvement was noted after the administration of antiscarlatinal serum, but sulphapyridine in the doses given did not appear to modify the course of the disease.

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An Unusual Retrocorneal Phenomenon

The following note on an unusual occurrence may be considered interesting enough to warrant publication.

Having removed a foreign body from the left cornea of a man aged 33, my attention was drawn to his right eye, where I saw a bright pink globule of about 1.5 mm. diameter, situated in the middle of the posterior surface of the cornea, the eye otherwise being quite normal. On examination under the slit-lamp the globule had—to my surprise—disappeared, but instead there was a faint reddish glow at the limbus about 12 o'clock. When the patient was asked to lie down again the spot was seen migrating slowly from the limbus towards the middle of the cornea, and always rose to the highest point of the anterior chamber as the eyes were turned in different directions.

The patient's record card showed that he had attended the hospital in Sept., 1942, when a foreign body deeply embedded in the stroma of the right cornea was removed and perforation with escape of aqueous occurred. Prontosil ointment 1% was instilled and a pad and bandage applied. The eye developed a mild iritis but healed in a week's time. A month later the patient complained again of photophobia and irritation, which quickly settled.

It would appear that owing to the pressure of the bandage a small portion of the prontosil ointment was forced into the anterior chamber, where it remained undissolved and unchanged without causing any trouble or inconvenience to the eye up to date (eight months). Neither the patient nor his friends have ever observed this striking appearance, since it is not visible when the patient stands upright.

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