The subject of ether convulsions has been discussed more than once at the Section of Anaesthetics, Royal Society of Medicine, and a good deal has been written about it in the medical journals, but in spite of this we have no knowledge as to the exact cause of the condition, or the treatment we should adopt when it arises, and so I need make no apologies for bringing forward the notes of two cases of this condition which occurred recently at the London Hospital.

For the notes of Case 1 I am indebted to my colleague, Dr. N. A. Gillespie, who also kindly called me from a neighbouring theatre to see the patient when the convulsions had developed. She was a married woman, 36 years of age, who had a laparotomy for obstructive jaundice on October 27th, 1936. In the previous January, that is nine months before, she had had Caesarean section performed under general anaesthesia at another hospital, chloroform being the agent employed, and there is a story of difficulty with the anaesthetic, rigidity and cyanosis persisting throughout. Following this there was a period of normal health for a few months, after which gradually increasing jaundice developed and she was admitted to the London Hospital.

At the operation in October the anaesthetic was ethyl chloride followed by ether on an open mask, atropine gr. 1/100 being the only premedication. She was not intubated; an ordinary Phillips' air-way was used. The patient had been having 5 c.c. of calcium gluconate twice a day intravenously for some weeks. The operative procedure was laparotomy with removal of a piece of liver for examination.

The induction and early stages of the anaesthesia were uneventful, but with the onset of deep anaesthesia, cardiac irregularity and later tachycardia developed. This was followed by typical ether convulsions just as the peritoneal
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Closure was completed. CO₂ and oxygen were given at once and the head and shoulders raised and the feet lowered, at first with no success, but after a few minutes the convulsions suddenly ceased just as a dose of evipan was about to be put into a vein. The convulsions did not recur. There was no post-anaesthetic vomiting, but slight bronchitis developed.

After-history. The patient gradually went downhill. After three weeks ascites developed and she died one month after the operation. Post-mortem examination revealed general fibro-sero-purulent peritonitis and acute yellow atrophy of the liver.

Points in this case to which attention may be drawn are:
1. The weather was not hot, but the patient had been running temperatures round about 104°.
2. She had had a previous abdominal operation, chloroform being used.
3. She was deeply anaesthetised at the time convulsions started.
4. The convulsions ceased spontaneously after the head had been raised for a few minutes and just before evipan was to be given.
5. She had been having calcium gluconate before operation.

Case 2. For the notes of this case I am indebted to Mr. B. B. Hickey, a House Officer at London Hospital. The patient was a male child aged 3½ years, admitted to hospital at 12.15 a.m. on March 3rd, 1937, with a three days' history of abdominal pain and vomiting. Eight hours before admission symptoms became worse, the breathing being rapid and embarrassed. On examination the child looked very ill, face flushed, temperature 101.4, pulse 140, respirations 32.

Diagnosis. Acute appendix.

Operation. 1 a.m., atropine gr. 1/150 at 12.40.


Operative findings. Acute perforated appendix. Pelvic peritonitis with abscess formation. Perforation of loop of small intestine which formed part of wall of abscess.

There was no trouble with the anaesthetic for 20 minutes when, as the peritoneum was being closed, the face twitched and the abdominal muscles became rigid. This lasted a few seconds and the child became cyanosed. The ether was at once shut off and the boy was given CO\textsubscript{2} added to oxygen. Breathing and colour quickly became normal and the operation was completed without difficulty.

On removing the mask the child again became cyanosed but this was at once corrected with pure oxygen. As the patient was about to be removed from the theatre cyanosis reappeared. The pupils dilated widely, there was spasm of the jaw muscles and general clonic convulsions began. Oxygen with about 12 to 15 per cent CO\textsubscript{2} was given, and after a few breaths of this mixture the convulsions ceased, only to recur again on removing the mask, but again to be abolished by inhalation of the mixture. A little later, however, a convulsion occurred whilst the mixture was being inhaled. These convulsions consisted of about 10 clonic spasms of the flexors of the limbs and digits, generalised facial twitchings with clonic movements of the neck and jaw muscles, synchronous with the flexor movements of the limbs. The convulsive movements were accompanied by short gasping inspirations. This was succeeded by a varying number of normal inspirations, after which the train of events recommenced. Cyanosis was avoided if the CO\textsubscript{2} oxygen mixture was given during the convulsions.

The individual movements of the convulsions were predominantly clonic contractions of the flexors; there was no pronation of the forearms and no rotation of the hips; the fingers became adducted but not to a greater extent than is normal in flexor movements; the thumb was not opposed, it was flexed with the rest of the fingers. It was agreed that the movements and postures of the limbs did not resemble those seen in tetany.

The CO\textsubscript{2} oxygen mixture was continued during the journey back to the ward, but on removing the mask to lift the patient into bed cyanosis at once reappeared. Morphia gr. ⅛ was given. The convulsions continued unchanged for about 40 minutes when the pulse, which, though rapid, had continued steady, became weak. Cyanosis persisted in
spite of oxygen, the convulsions became weaker, and the child died at 2.40 a.m.

The methods of treatment tried in this case were: (1) Varying mixtures of CO₂ and oxygen. It was found that a mixture containing 12 to 15 per cent CO₂ in oxygen had most effect. (2) Changing position of patient. He was definitely worse when foot of bed was raised. Very slight improvement was gained with high Fowler position. (3) Injection of morphia. Examination of the ether employed showed no impurity.

Here, then, we have two patients of different ages and sex showing the same symptoms, though of different severity, at the end of an abdominal operation under deep ether anaesthesia.

In the British Journal of Anæsthesia for July 1936 there are two papers dealing with the subject. In one paper Hoseason puts forward the idea that the condition is due to calcium deficiency, and he quotes a case of ether convulsions which responded dramatically to an intravenous injection of calcium, but in the first case which I report, the patient had been having intravenous calcium twice a day for weeks and can hardly have been suffering from calcium deficiency.

In the other article Vaughan Hudson reports two cases and quotes freely a paper published in the Lancet by Woolmer and Taylor. Hudson's first case was a typical one, a child with a gangrenous appendix who developed fatal convulsions towards the end of the operation. It was noticed in this case that the child's skin was extremely hot and the axillary temperature was found to be 106°.

Intravenous calcium was given without effect.

The second case was that of a child of two months old who was operated on for inguinal hernia under local anaesthesia, novocain .75 per cent being used. The convulsions occurred during a fairly severe surgical stimulus, viz. the separation of the cord from the wall of the sac. In this case the operation was rapidly completed and chloroform was given. This caused both respiration and convulsions to cease, but gentle pressure on the chest started the respirations and the convulsions did not recur, the child making a good recovery.
Hudson puts forward the view that the cause of the convulsions is surgical trauma in the presence of too light a degree of anaesthesia, and he suggests that with the onset of convulsions all forceps and instruments likely to provoke stimuli should be removed, and that the anaesthetist should deepen the anaesthesia by the most rapid means at his command, preferably with evipan, or failing this, with chloroform.

As regards the first suggestion it is my experience and the experience of a good many people I have spoken to that where convulsions come on the anaesthesia has always been very deep, in fact too deep, after showing that jerky breathing which one associates with over-deep ether anaesthesia. It would seem to me to be a very dangerous procedure to give the patient chloroform; fatalities have been recorded from this method of treatment. In a rigid, blue patient, taking occasional gasping breaths, it must be very difficult to regulate the intake of chloroform.

Evipan would appear safer, as it would be easier to regulate the dose, though the difficulty of finding a way into a vein in these patients must be considerable.

I should prefer to try methods which aim at rapid elimination of the anaesthetic, which relieve cerebral congestion and lower temperature.

In provoking discussion I would suggest that there are many factors which cause this condition, some of which are (i) sepsis, (2) pyrexia, (3) hot weather or very hot theatre, (4) deep anaesthesia, (5) cerebral congestion, (6) I have an idea that atropine may have something to do with it, but I shall be told that atropine has been given to hundreds of thousands of cases without causing convulsions. I would like, however, to ask these two questions: (i) Has anyone had a case of convulsions in which atropine had not been given? (ii) Has anyone had a case of convulsions when morphia had been added to the atropine?

I think it was about 1908 or 1909 that atropine began to be used, and certainly before that no case of convulsions had been reported.

(7) Surgical trauma. Most cases seem to occur when the peritoneum is clipped with forceps preparatory to being
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sewn up. As regards treatment I suggest: (i) Withdraw anaesthetic and hasten elimination with CO₂ and oxygen; remove forceps. (ii) Raise head and lower feet to relieve cerebral congestion (steeply). (iii) Apply cold pack to head and neck to lower temperature. (iv) If these measures fail give intravenous evipan.

ETHER CONVULSIONS CONTROLLED BY EVIPAN.

By H. Grantham Dodd, M.B., B.S., D.A.

T.C.D., male, aged 37 years, a steel-worker of average muscular development, but with a large, prominent muscular, and deep abdomen, was admitted to the Llanelly General Hospital during the evening of May 25th, 1937.

He had had an attack of abdominal pain about a fortnight previously, without vomiting; this had eased off, and he had been at work since. At five o’clock on the afternoon prior to admission the pain had recurred, commencing at the xiphisternum and going down to the umbilicus, and had gradually got worse; he went to work as usual on nightshift. On the morning of the 25th he took a fruit laxative, and his bowels were opened. The pain got much worse at 3 p.m.; and he vomited slightly thrice.

He was admitted to hospital with a temperature of 100.2, pulse-rate 96, respiration 26, and a furred tongue. There was general rigidity and tenderness of the abdomen: there appeared to be definite loss of liver dullness, but owing to the configuration of the abdomen this sign was not considered to be of much value: there was gurgling on auscultation to the left of the middle line over the gastric area.

He was taken to the operating theatre at 10 p.m. for operation by Mr. D. McLennan, F.R.C.S.

The Anaesthetic. A dental prop was inserted between the lower teeth and the edentulous upper jaw. Induction was by C₁₂E₄, C₁₂E₃₂. Ether sequence with an Ogston’s mask. There was no difficulty during induction; no obstruction nor cyanosis; deep surgical anaesthesia was obtained, with the pupils definitely commencing to dilate. At this stage a No. 8 Magill’s rubber endotracheal tube was