A five-year-old girl was admitted to Hammersmith Hospital with a diagnosis of a perforated appendix. Her mother stated that she had complained of pain in the R.I.F. for 12 hours prior to admission, that during that time she had vomited periodically and was “breathing very fast.” The pain had become severe in the evening and she was brought up to the Casualty Department.

Physical examination revealed a well developed child in slight distress and looking very toxic. Her temperature was 102.6°F pulse rate 140 min, respiration rate 30 min. There was no history of previous illness or fits.

It was decided to operate and premedication of atropine (0.6 mg) was given 40 minutes before operation. The temperature in the operating theatre was 86°F and outside there was a thunderstorm. Anaesthesia was induced with N₂O and O₂ at 2 a.m., ether being added slowly in a semiclosed circuit. On establishing second plane anaesthesia an intravenous drip of 5 per cent Dextrose was set up and the skin incision made 15 minutes later. At 2.30 a.m. the peritoneum was lifted to open, very mild twitching of the eyelids and facial muscles was noted; these rapidly spread to the trunk and limbs, followed by generalized convulsions. The ether was turned off at once, the surgeon stopped his manipulations, the bag on the anaesthetic machine was emptied and pure oxygen was administered. At the same time 3 mg of d-tubocurarine were put into the drip and within three minutes all convulsive movements ceased.

The child felt excessively hot and sponging with ice cold water was carried out for 20 minutes to the upper and lower limbs, axillae and groins. Oxygen administration was continued. Pronounced movements started again at about 3.50 a.m. and this time 4 ml of calcium gluconate (5 per cent) were given intravenously. There was no effect from this but another 25 mg of thiopentone caused the movements to cease. This time she remained quiet for about 45 minutes, and when movements did return they were much less severe. A final dose of 2 ml of calcium gluconate was given and at 5.30 a.m. she opened her eyes and soon after that began to cry.

She was sent back to the ward at 6 a.m. and

geon removed the perforated appendix and closed the abdomen 25 minutes later. Respiratory exchange was adequate by this time. Pharyngeal toilet was carried out and much thick mucus was removed. It was considered advisable to keep the child under further observation and she was kept in the operating theatre.

At 3.10 a.m. (15 minutes after completion of surgery) she was about to be returned to the ward when slight clonic movements of the arms and legs were noted: these gradually became more pronounced; the reflexes were hypertonic, opisthotonos was present to such an extent that a closed fist could be placed easily under the arched back. At the first of these signs oxygen and a little CO₂ (300 ml for 1 min) was given, the head of the table raised and 1 ml of 2½ per cent thiopentone was injected into the drip. The CO₂ was given because it was thought that with the assisted respiration her alveolar CO₂ content might be low.

These measures had no effect on the carpopedal spasm and so a further 2 ml of thiopentone with 0.5 mg of d-tubocurarine were given; this reduced the movements. The child felt excessively hot and sponging with ice cold water was carried out for 20 minutes to the upper and lower limbs, axillae and groins. Oxygen administration was continued.

Pronounced movements started again at about 3.50 a.m. and this time 4 ml of calcium gluconate (5 per cent) were given intravenously. There was no effect from this but another 25 mg of thiopentone caused the movements to cease. This time she remained quiet for about 45 minutes, and when movements did return they were much less severe. A final dose of 2 ml of calcium gluconate was given and at 5.30 a.m. she opened her eyes and soon after that began to cry.

She was sent back to the ward at 6 a.m. and
was fully conscious by 9 a.m. and able to respond to questions. Apart from slight (? voluntary) shaking of the head, no other movements were noted and she appeared quite normal, subsequently she made an uneventful recovery and was discharged from the hospital ten days later.

DISCUSSION

From this case report it will be seen that quite a number of the so-called predisposing factors were present, namely: pyrexial patient, hot theatre, humid atmosphere, sepsis (pus in peritoneum), vomiting (? hypocalcaemia), and fast respirations (hypocarbia), atropine as premedication (causing a further rise in temperature and preventing sweating), and finally ether as an anaesthetic agent. One might say that the stage was set for convulsions. One or two points should be noted:

1. The character of the first convolution was different from the subsequent ones, i.e. it was a convolution in which the movements had no particular characteristics, whereas the latter ones were tetanic like with carpopedal spasm, opisthotonos and plantar flexion.

2. At the start of the operation the pupils were medium dilated and slowly returned to normal by the time the skin was sutured. Over the next two hours they gradually enlarged and remained almost fully dilated for some hours after return to the ward. They reacted very sluggishly to light.

3. The convolution occurred at the same time as the pulling of the peritoneum—this may have been coincidence.

4. The dose of calcium gluconate could have been larger.

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