in the Purkinje cells, they fit well with the clinical signs and provide an explanation for the respiratory failure which was the immediate cause of the patient's death.

It is impossible to be certain whether this case should be regarded as one of acute idiopathic porphyria or whether it was toxic in origin, as we were unable to obtain enough urine for the porphyrin to be typed. Of the drugs which she received, the sulphadiazine would have been the most likely cause of her condition; Dobriner and Rhoads (1940) mention both clinical and experimental evidence that sulphonamides may cause increased excretion of porphyrins, and sulphadimidine (sulphamezathine) was one of the drugs administered in the fatal case reported by Abrahams *et al.* The difficulty remains that in all reported cases of toxic porphyria the predominant excretion product has been coproporphyrin.

In our case symptoms, though slight at first, preceded sulphadiazine administration. The balance of evidence therefore seems to us to be in favour of this being an acute idiopathic porphyria rather than a toxic one. The urinary findings and the rapidly progressive neurological picture would also make this the more likely diagnosis.

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CONVULSIONS IN CHILDHOOD

BY

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The article by Nathan and Ransford (1949) prompts me to record six cases of convulsions in children between the ages of 5 and 11 years. These were observed between 18 and 25 years ago and their subsequent medical history has been recorded. I attended the mothers of Cases 4, 5, and 6 at their confinements. The observations were made during an investigation of the cortico-thalamic reactions to visceral tensions, which may represent only a small group of a type of epileptic convulsion—the viscero-cortical. The neurological syndrome, however, with which these cases appear to be associated has since become the basis of certain obstetrical procedures. These observations were made before electroencephalography came into use, and there was then no knowledge of cortical electrical potentials and the variations of their rhythmical patterns.

Case 1

A boy aged 10 suddenly and for no apparent reason fell to the floor in a typical epileptic fit The fits recurred every 15 minutes for an hour and lasted two to three minutes. He had bitten his tongue and become incontinent. When I saw him he had just recovered from a fit, was lying on a bed, restless, unconscious, but frowning and mouthing his lips over closed teeth as if trying to free them of an unpleasant taste. There was no history of constipation. He was not cutting any teeth. He had had a large tea—it being Christmas Day. A few minutes after the fit he became conscious and I decided to empty his stomach. This was effectively done. The resulting vomit was enormous. After an hour's rest he was perfectly normal and had no memory of what had happened, except that he had been sick.

He is now 35 years of age, has never had a recurrence of these attacks, was an exceptionally brilliant student at school, and was an outstanding athlete. He is a member of a family whose names are well known in the athletic and industrial world.

Case 2

A boy aged 6 had a series of epileptic fits for no apparent reason. They occurred every five minutes and had been going on for over an hour when I saw him. He was well nourished and a good specimen of a curly-haired boy. His history was that he had not felt well that day. He was lying on the bed in a fit when I saw him, with his head thrown back, his back hollowed, his legs rigid, and his arms in tonic spasm extended in front of his abdomen. His breathing was stertorous, his face cyanosed, his teeth tightly clenched, and strabismus showed through widely opened eyelids. There followed a series of clonic contractions from which he relaxed with a groan. He had not bitten his tongue, but there was the puckered frown upon his forehead and mouthing similar to Case 1. Not having felt well, he had had only a few mouthfuls of bread and milk during the day. There were no history of injury, no pyrexia, and no evidence of the onset of an acute inflammatory condition.

Examination of the abdomen however, revealed a distended descending colon and sigmoid lying like a large rope stretching almost from the splenic area to the pelvis. He was at once given a rectal injection of 2½ oz. (70 ml.) of equal parts of glycerin and oil. A mass of scybala and constipated faeces was passed, being followed after a few moments by a large and well-formed evacuation of the bowel which in mass was out of all proportion to the size of the child.

This fine boy was killed in action as an officer of the Guards Brigade at the age of 25. He had never had any recurrence of his convulsion, but had grown up a scholar and athlete. His father had been an athlete of considerable accomplishment.

Case 3

A boy aged 11 was sent into hospital with a history that he had epileptic fits. His doctor had never known him to be ill previously. He came of a good artisan stock and had several brothers and sisters. There was no history of illness in the family, epilepsy or allergy. Twenty-four hours before admission to hospital he had his first fit, which was followed by one approximately every hour. When he was admitted he was unconscious. His temperature was over 100° F. (37.8° C.) and he was considered to be suffering from acute infective encephalitis.

When I first saw him he was lying unconscious in bed with his head thrown back. A gentle effort to bring it forward precipitated a fit which lasted about two minutes. When the clonic movements ceased he lay frowning and mouthing as did the two previous cases.

Sir Henry Head pointed out to me, when I was his house-physician, that if convulsions were accompanied by mouthing a "visceral accompaniment" should be suspected. Between attacks this boy had complained of acute abdominal pain and put his hands over the lower abdomen. He lay fully stretched out upon the bed. Abdominal examination revealed a firm lump, apparently in his caecum. There was no tenderness or peritonism. His white cell count showed an eosinophilia with no rise of polymorphonuclear leucocytes. Assuming from these observations that his might be a case of ascaris toxaemia with visceral irritation, I gave him santonin followed by castor oil, which he was able to swallow. I refrained from doing an immediate lumbar puncture. Eight hours later he was sitting up in bed looking perfectly well, having no memory of what had happened. A jar was produced containing four large roundworms. In 24 hours the treatment was repeated and another bunch of large roundworms was expelled. At the end of three days he was sent home perfectly well.

He grew into a fine athlete and spent many years as a successful professional footballer. He was 29 when I last heard of him, about four years ago, and had never had a day's illness. I understand that his father had also been a good athlete.

Case 4

This case, that of a boy aged 6, was more difficult because it was complicated by the possibility of injury. He was riding a "fairy" cycle down a macadam road when he shot over the handlebars and landed on his forehead. He got up almost immediately and, although dazed, did not lose consciousness. He was taken home and I saw him within ten minutes. There was a very large swelling, apparently a haematoma, on his forehead, about 3½ in. (8.8 cm.) in diameter, protruding nearly 1 in. (2.5 cm.) from the frontal bone. A radiograph did not reveal a crack, depression, or fracture of the frontal bone. The injury was severe enough, however, to warrant careful treatment.

He was kept in bed and quiet in order that the swelling might subside. All went well for six days, when the necessity for this treatment was questioned by his parents. It was insisted, however, that no head injury should be taken lightly. On the evening of the sixth day I was summoned urgently because the boy was looking strange. Fortunately I was again able to be at the house in a few minutes, to learn that he had just had an epileptic fit. When I saw him he was in a drowsy condition and my efforts to examine him resulted in a second convulsion which was typically epileptic. There was nothing to suggest that it was Jacksonian in type. After a tonic phase there was a general convulsion which lasted well over a minute. It was very severe. His breathing was stertorous, he was cyanosed, and when he relaxed the mouthing that has been previously described was noted. There was no puckering of the forehead, however, because it was too swollen and tender.

In this case there was obviously more than one consideration. From the nature of the convulsion visceral influence appeared to be likely, but leaking subdural haematoma had to be taken into consideration. His bowels had not been open for 48 hours. On examining his abdomen a large palpable colon was found in the left iliac fossa. An injection of glycerin and olive oil was again resorted to. There was a copious movement of the bowels, followed 20 minutes later by a second evacuation quite abnormal in size. In a short time the boy became clear in his mind. He looked about him as a child looks around when wakened by light at night—at first a little dazed and then completely comprehensive. He called me by my name and asked why I was there. He never had a recurrence of this condition. He grew to be a good athlete with an exceptionally alert mind. He was killed while a pilot in the Royal Air Force at the age of 22. This boy's father was one of the greatest lawn tennis players of all time.

Case 5

I was asked to see a boy aged 5 because he was not feeling well. His symptoms were quite indefinite. He was by nature an extremely cheerful and sturdy boy. When I saw him he was sitting on his nurse's knee, which for him was quite unusual.

I went into the night nursery while he was being put to bed, with the idea of observing his behaviour and talking to him before examining him, when without any warning he suddenly became rigid, with his head flung back and his back arched, and he would have fallen to the ground if his mother had not caught him. He had a series of clonic movements of the arms and legs; his eyes were open, and there was strabismus and rigid opisthotonos. After about a minute, which seemed a very long time, the clonic movements ceased; he became relaxed and lay still on the bed with his fingers working in the palms of his hands and his mouth moving in the typical manner previously described. Two or three minutes later he was violently sick. He brought up his lunch and his tea, neither of which appeared to have been digested.

This boy had a congenital pyloric stenosis, for which Rammstedt's operation was performed when he was 5 weeks old. He grew up to be a normal intelligent boy and an outstanding athlete. He did not suffer from any illness or from any recurrence of convulsions. He is now about 24 years of age. His father was an all-round athlete of recognized ability, both at the university and afterwards, whose books are read in many countries.

Case 6

This patient was a girl aged 9, a heavily built sturdy child. She was staying in my house. Half an hour before breakfast she was playing happily in the nursery, feeling quite well, when without any warning she fell flat on the floor. I was with her almost immediately and found her lying with her eyelids open and her teeth clenched. There were strabismus, clonic movements of arms and legs, and deep stertorous breathing. The convulsion lasted an alarming time: it was the most prolonged that I have ever witnessed, probably three minutes before she relaxed. She remained unconscious. After a few minutes she was violently sick, having had nothing to eat that morning. I carried her to her bed and she vomited a second

Tabulated Record of Cases

	Past and Family History	Gastro-intestinal State at Time of Attack	No. of Fits	Fall	Type of Convulsion	Incontinence of Urine	Time Unconscious	Mouthing and Frowning Between or After Fits		Treatment	Result	After-Result	1949
								Mouthing	Frowning				
Case 1: Boy, 10 years	No epilepsy. No protein-sensitive diseases	Overloaded stomach	3 or 4 in an hour	Yes	Tonic spasm; opisthotonos; clonic movements; relaxation	Yes	2½–3 hours	Yes	Yes	Emptied stomach	Complete recovery very few hours	Excellent physical and mental development. No recurrence of fits	Aged 35
Case 2: Boy, 6 years	,,	Overloaded rectum	10 or 12	Lying down at onset	,,	,,	1–1½ hours	,,	,,	Emptied rectum	,,	,,	Killed in action, aged 25
Case 3: Boy, 11 years	,,	Mass of intestinal parasites (round-worms)	Between 20 and 30	No history	,,	Yes and faeces	36 hours approx.	,,	,,	Expelled parasites	,,	,,	Aged 35
Case 4: Boy, 6 years	,,	Overloaded rectum	2	Lying down at onset	,,	Yes	10–15 minutes	,,	No	Emptied rectum	,,	,,	Killed flying in R.A.F., aged 22
Case 5: Boy, 5 years	,,	Overloaded stomach with delayed digestion	1	Yes	,,	,,	1–2 minutes	,,	Yes	Emptied stomach	,,	,,	Aged 24
Case 6: Girl, 9 years	,,	,,	1	,,	,,	No	3 or 4 hours	,,	,,	,,	,,	,,	Aged 25

time. Both these vomits were copious: I estimated that the two of them were nearly four pints (2.27 litres) of stomach content. The food was undigested, her lunch and tea of the previous day being recognized.

She remained in a comatose state for approximately four hours and during that time the same frowning and mouthing was observed. When she regained consciousness she complained of headache. There was no previous history of any similar attacks, or any family history of convulsions or allergy, and she was not at the time cutting her 10-year molars.

She has grown into a strong healthy woman, weighing nearly 12 stone (76.2 kg.), though not excessively fat. She has had a fine athletic career and obtained the maximum credits in her School Certificate. She comes of a family who have combined fame in politics and science with considerable athletic distinction.

Discussion

In each case a short note on the family and stock from which these children came has been appended. The similarity of types is not without interest. A high degree of physical and mental efficiency and personal achievement is common to them all. Is there, therefore, any association between these factors and predisposition to cortical storms of this nature?

The similarity of many features of the tabulated record of these cases suggests that a certain type of convulsion, whether recurrent or not, may be initiated by visceral tensions, irritations, and spasms. In children whose thalamic function is highly sensitized to sensory stimulus a visceral sympathetic assault may disturb integration of the normal thalamo-cortical relationship. Instead of protective, balanced motor responses an extensive cortical storm is precipitated by uncontrolled neural discharges, either causing or resulting from paroxysmal cerebral dysrhythmia. The integrity of the cortical response depends upon the intensity of tension or irritation, to sensitivity of the visceral nerve endings, the conductivity of the sensory neurones, and the quality of the thalamic receptivity. Abnormality of any one of these factors may predispose to convulsions. Later observations have shown that clinical manifestations of visceral tension result in variations of cortical discharge compatible with this theory. It has been possible to observe this during childbirth, and a large number of cases have provided evidence that the prevention and treatment of visceral tension greater than the normal mean may preserve the integrity of the viscero-thalamo-cortical syndrome.

In the course of the years I have recorded three cases of sudden epileptic fits during the late first stage of labour. None of these women showed any signs of toxaemia. There was no raised blood pressure and no albuminuria or excess of acetone in the urine. All three were primiparae. Two, however, had suffered from attacks of petit mal and the third had spent some months in a home with a severe nervous breakdown. All three were highly emotional—a state which results in resistance by the circular muscles of the uterus to the dilatation of the cervix. This in effect is the contraction of two opposing groups of muscles at the same time, giving rise to abnormal tension.

The explanation of these fits, which had no relation to eclampsia, was that excessive tension in the lower uterine segment overloaded the hypersensitive afferent sympathetic fibres from that area of the uterus. The thalamic receptors were already disturbed by emotional imbalance, with the result that a convulsion or cortical storm was precipitated by uncontrolled neural discharges. It is significant that all three remained well during the second stage of labour, when, in the absence of abnormality, there is relaxation of the lower uterine segment.

The possible relationship of this syndrome to certain types of convulsion in pregnancy and labour cannot be overlooked, and it seems reasonable to suggest that a high percentage of the non-recurrent epileptic fits in children may be explained upon this hypothesis.

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ENDOTHELIOMA OF THE PLEURA

REPORT OF A CASE

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[WITH PHOTOGRAVURE PLATE]

Since the original account by Wagner (1870) there has been much argument about the pathological entity of endothelioma of the pleura. Some pathologists (Robertson, 1924) denied its existence. There does seem, however, to be a small group of cases (Klemperer and Rabin, 1931), comprising about 0.2% of all necropsies (Saccone and Coblenz, 1943), in which such a diagnosis must be made. The clinical picture is fairly constant, the main features being increasing dyspnoea and pain in the chest associated with great thickening of the pleura and massive pleural effusion (Banyai and Grill, 1933). The reason for reporting this case is that the clinical and radiological signs were unusual and the diagnosis unexpected.

Case Report

The patient, a married woman aged 41, was first seen by one of us (A. W.) on November 24, 1947, when she complained of lassitude, dry cough, and low-grade fever of one month's duration. She was born in Germany and had lived there and in North-west Europe until her arrival in this country just after the war. There were no significant facts in her past or family history. On examination she looked well, was adequately nourished, and no abnormal physical signs were found. Radiographs of the chest showed two rounded homogeneous shadows on the left side, one large and one small, with, in the lateral view, two more small shadows lying anteriorly (Plate, Fig. 1).

These appearances suggested multiple hydatid cysts or metastases possibly from a hypernephroma. On December 12 she was admitted to the Middlesex Hospital for investigation, and on further interrogation she denied contact with dogs. She now complained of vague pain in the left chest, and there was some note impairment over the left upper chest anteriorly. No evidence of a primary growth was found in the thyroid, breast or pelvis. The temperature varied between 99 and 100° F. (37.2 and 37.8° C.), the pulse between 80 and 100, and the respirations remained at 20; the white cells numbered 8,000 (neutrophils 74%, lymphocytes 18%, monocytes 7%, eosinophils 1%); stomach washings for tubercle bacilli were negative; the Mantoux test was positive at 1 in 1,000; the Wassermann reaction and Casoni and hydatid complement-fixation tests were negative; and an intravenous pyelogram was normal. An artificial pneumothorax showed that the tumours were attached to the parietal and visceral pleurae.

On January 21, 1948, Mr. T. Holmes Sellors performed a thoracotomy, at which many rounded pinkish tumours varying