ing day. If, therefore, only those patients who remained at rest are included, the incidence of muscle pains in this group is 5.6%—that is, 2 patients out of 36 examined.

Trial with Gallamine

In order to determine whether the abolition of visible muscle fasciculations prevented the muscle pains following suxamethonium a further series was attempted identical with group I (out-patients) except that the injection of suxamethonium was preceded by 40 mg. of gallamine triethiodide. This abolished visible twitching in every case. Of 15 cases examined, 6 (40%) complained of muscle pains. The significance of results based on such a small series is difficult to analyse, but the general impression was that following gallamine the incidence and severity of the muscle pains were diminished, though with this dosage they were not abolished. Further support is given to this suggestion by the finding in 12 in-patient cases (that is, confined to bed for 48 hours after operation), that if a dose of gallamine triethiodide (80 mg.) was given before the administration of suxamethonium there was no case of post-operative muscle stiffness.

Discussion

Suxamethonium has been widely used as a short-acting muscle relaxant in both clinical surgery and electric convulsion therapy. If it is given, however, to patients who are not confined to bed after treatment it may be followed by a degree of incapacity out of all proportion to the severity of the procedure. Severe stiffness can develop on the first day after operation, which has been likened to the muscle stiffness that often follows some violent physical exercise. If, on the other hand, the use of suxamethonium is confined to patients resting in bed the incidence of muscle pains is low.

It has been shown in man that decamethonium iodide causes muscle activity of apparent motor unit origin (Churchill-Davidson and Richardson, 1952) as opposed to single muscle-fibre potentials which might be anticipated from the use of a depolarizing drug. It has been suggested (Paton, 1952) that this fascicular twitching is due to centripetal antidromic impulses in the motor nerve axon bringing about a synchronous discharge of the whole motor unit (Masland and Wigton, 1940). The finding that gallamine triethiodide prevents the muscle fasciculations of suxamethonium but only reduces the incidence of "after-pains" suggests that these pains may not be due to the vigorous twitching of the muscles but to some curious association of depolarization by succinylcholine and normal muscular activity.

Suxamethonium, therefore, has two serious disadvantages. First, in company with all depolarizing drugs it has no satisfactory antidote, and the occasional case of prolonged action still occurs (Bourne, 1953; Evans *et al.*, 1952). Secondly, it is unsuitable for use as a muscle relaxant for out-patient procedures, because it may be followed by severe muscle stiffness.

Decamethonium enjoyed only a brief span of popularity in clinical surgery. It would seem that the longevity of suxamethonium must depend upon the time it takes to find a new muscle relaxant of brief effect acting by competitive inhibition (like D-tubocurarine chloride) but destroyed by plasma cholinesterase. In the event of a prolonged action neostigmine could then be given with confidence.

Summary

When suxamethonium is used as a short-acting muscle relaxant for out-patient procedures it is followed next day in a large proportion of cases by muscle pains and stiffness, the severity of which is out of proportion to that of the minor operation. These pains commonly persist for two to three days. The incidence in patients confined to bed is low.

The abolition of the fascicular twitching—a notable feature after the injection of suxamethonium—reduced the incidence and severity of the muscle pains.

The value of suxamethonium as a short-acting muscle relaxant is briefly discussed.

I am most indebted to Drs. Regina Mackay and David Chandler for their help in following up these cases, and to all the members of the department of anaesthetics who co-operated so generously. Miss Jean Davenport gave invaluable secretarial assistance.

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ACUTE TOXIC HYPOGLYCAEMIA IN THE VOMITING SICKNESS OF JAMAICA

BY

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The illness at present known as the vomiting sickness of Jamaica has a distinct clinical picture, which has been reviewed by Hill (1952). It occurs sporadically or in small, usually family, outbreaks during the colder winter months, when food is relatively scarce amongst the Jamaican peasant population. The onset is dramatically sudden, often with severe vomiting, followed by rapid prostration with a low blood pressure and tachycardia; the temperature is not elevated and diarrhoea does not occur. This may be followed by a latent period of apparent improvement lasting a few hours or may pass direct into the terminal phase of drowsiness, twitching, convulsions, and coma—sometimes associated with effortless vomiting. There is a high mortality, death occurring on an average after 12 hours. Cases that recover do so completely.

A fulminating clinical variant is also recognized in which no vomiting occurs, the illness presenting with spectacular abruptness, with sudden collapse, drowsiness, convulsions, stupor, twitching of the limbs, coma, and death. The whole episode may last only a few hours.

The syndrome usually occurs against a background of poverty and subnutrition, and those affected are often, but not always, underweight and frequently show signs of slight deficiency of the vitamin-B complex and protein—such as angular stomatitis, glossitis, mosaic skin, and hypochromotrichia. Families are often involved, the main incidence being in children of 3 to 10 years.

As has been stressed by Cicely Williams (1953) and Chambers (1953), a diagnosis of vomiting sickness is often loosely used in Jamaica to cover various undiagnosed illnesses in which vomiting or sudden death has occurred. Nevertheless, after taking this into account, there remains a definite group of typical cases showing a characteristic clinical picture.

Aetiology

The precise aetiology of the vomiting sickness of Jamaica is still very much in doubt and cannot be discussed here fully. The various hypotheses have been considered by Hill (1952). Meningitis, encephalitis, cerebral malaria, and other acute cerebral inflammatory diseases can be excluded both clinically and by post-mortem evidence. The present consensus of opinion is that the syndrome is due to the effect of an unknown poison, or possibly one of several poisons, upon subjects who are usually rather poorly nourished, especially children. Metallic poisons, botulism, "barracuda poisoning" (from fresh but diseased fish), and the staphylococcal toxin have all been suggested on occasion with no real evidence; while ill-prepared bitter cassava or unripe yams—both of which contain cyanogenic glucosides—are sometimes alleged to be responsible, although again there seems to be little proof.

The ackee (*Blighia sapida*), which is widely grown and eaten throughout Jamaica, has been shown to contain a toxic saponin when immature (Evans and Arnold, 1938). Scott (1916), in his investigation of this subject, concluded that all cases were due to the eating of unripe ackees, and he was of the opinion that the disease should be called "ackee poisoning." However, as Cicely Williams (1953, personal communication) notes: "In spite of the evidence produced by former workers that the ackee was responsible, it is difficult to reconcile this with the fact that most Jamaicans are eating ackees regularly. They eat them in large quantities. Some people eat them raw or eat the fruit before the 'pod' is opened. They even drink the 'pot-water' after boiling the ackees, without disaster in the majority of cases." Nevertheless, most observers feel that the unripe ackee should still be suspect. Finally, it may be noted that herbal infusions of "bush tea," which are universally used as beverages and medicinally, are made from a very wide variety of plants which may conceivably be sometimes misused, with toxic results.

An unusual feature of the syndrome of vomiting sickness is that it has not been described elsewhere in the world, even in the other Caribbean territories.

Actual field investigations of cases of vomiting sickness are usually inconclusive, owing to the difficulty of obtaining accurate medical histories and because of the large number of potentially poisonous plants—including the ackee tree—to be found in the bush around the average Jamaican peasant's home. The dietetic history of children under these circumstances is even more difficult to assess, as, although the family's diet may be known, during the "hungry months" ill-fed children may scavenge in the bush and experiment with wild berries, fruits, and plants.

During the first three months of 1953 a number of cases of vomiting sickness were admitted to hospital here showing a vivid and pathogenic clinical picture associated with severe hypoglycaemia. Clinical details of these cases are in the process of publication (Stuart, Jelliffe, and Hill), as is a discussion of the pathology (Hill and Bras). The following is the description of a case seen more recently.

Case Report

A 6-year-old Jamaican boy of African extraction had been quite well until 4 p.m. on the day before admission, when he had vomited once, bringing up a small quantity of undigested food. There was no abdominal pain or diarrhoea. Following this the child had appeared to be quite well and went to bed normally at 9 p.m. He passed urine during the night at 1 a.m. and 6 a.m., and on each occasion he was seen by his father, who thought he appeared to be quite well. At 7 a.m. the father called the child for his morning "bush tea," but could not wake him. The father thought the boy was in a deep sleep, but on trying to rouse him 15 minutes later the child was found to be unconscious, with his legs in rigid extension, arms tightly flexed on his chest, hands clenched, and jaw locked. This stiffness seemed to wax and wane, lasting about 10 minutes with longer intervals between.

The rest of the family, consisting of the father and a 3-year-old sister, were in no way affected, although they had all eaten the same meals together. The patient had eaten no ackees or other possibly toxic substances during the preceding 36 hours, so far as the father was aware, although there were plenty of ackee trees and other bushes around the house.

The patient arrived in the ward at 2 p.m., when he was found to be deeply comatose with stertorous respiration. He was afebrile, with a pulse of 120 and a blood pressure of 110/60. There were no signs of meningeal irritation. The eyes tended to rove about, with a transient squint. The fundi and pupils were normal. The limbs were flaccid at the first examination and all tendon reflexes were diminished. Painful stimuli produced no response, while the abdominal and plantar reflexes were absent. The child was well hydrated and moderately nourished, with only minor stigmata of malnutrition, in particular slight angular stomatitis and cheilosis. The liver was just palpable.

During the first three-quarters of an hour after admission there were repeated attacks of stiffening of the limbs, each lasting about one to two minutes, accompanied by a rigor-like shaking. During these bouts the limbs became hypertonic with greatly increased reflexes.

In view of experience gained with previous cases, a diagnosis of acute toxic hypoglycaemia due to vomiting sickness was made.

Investigations.—Investigations carried out immediately on admission showed: (1) Blood count: haemoglobin, 11.6 g. per 100 ml.; red cells, 3,500,000 per c.mm.; white cells, 17,000 per c.mm. (metamyelocytes 0.5%, neutrophils 85%, lymphocytes 14.5%); E.S.R., 6 mm. per hour; haematocrit, 32%; M.C.V., 91.4 μ^3; M.C.H.C., 36.3%; M.C.H., 33.1 $\mu\mu$g. (2) Liver-function tests: bilirubin (total), 0.6 mg. per 100 ml.; cephalin-cholesterol flocculation test, negative; gamma-globulin (Kunkel), 5.8 units; thymol flocculation, negative; thymol turbidity, 1.2 units; serum proteins, total 7.4 g.% (albumin 4.9 g., globulin 2.5 g.); cholinesterase

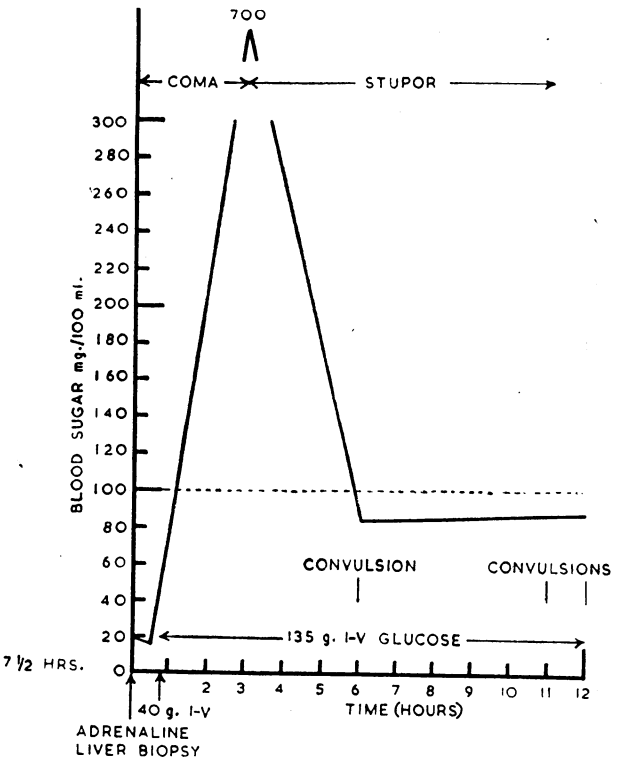

Chart of the case.

(Michel), 0.7 unit. (3) Blood urea, 45 mg. per 100 ml. (4) Serum calcium, 11 mg. per 100 ml. (5) Carbon dioxide combining power, 40 volumes per 100 ml. (6) E.C.G., normal. (7) Blood sugar, 19 mg. per 100 ml. (8) Liver biopsy (immediate): almost complete absence of liver glycogen (0.7%).

Progress in hospital is summarized in the accompanying graph. Adrenaline, 5 minims (0.3 ml.), was given subcutaneously on admission and the blood sugar was estimated after 15 minutes. This showed no real change (18 mg. per 100 ml.). Treatment was then begun with an injection of 40 g. of glucose intravenously, in a 50% solution, followed by a continuous intravenous drip of 9.5% glucose solution. By 4.25 p.m. the blood sugar had risen to 700 mg. per 100 ml. and was associated with a marked glycosuria. No dramatic clinical effect was produced, although the coma appeared to be definitely lighter during the afternoon and evening. The blood sugar had fallen to 84 mg. per 100 ml. by 8.30 p.m., and thereafter remained between 80 and 100 mg. The child continued in a stuporose condition, and during the night he had two generalized tonic-clonic convulsions. Next day a further fit occurred at 5 a.m., following which the child died, the respiration stopping while the heart continued to beat for 15 minutes afterwards.

Post-mortem examination was mainly negative save for lymphoid hyperplasia in the mesentery and ileum, together with fatty change in the liver.

Discussion

The very low blood sugar and almost absent liver glycogen found in this case have also been noted in other examples of vomiting sickness investigated during the acute phase of the illness. Full clinical and pathological details are to be published elsewhere, but results from five cases, including the present one, are summarized in the Table. The rapid

Details of Five Children Admitted with Acute Toxic Hypoglycaemia in the Vomiting Sickness of Jamaica

Case No.	Age in Years	Sex	Mental Condition on Admission	Blood Sugar (on Admission) (mg. per 100 ml.)	Known Length of Coma (Hours) before Treatment	Response to Large Doses I.V. Glucose
1	6	M	Drowsy	6	—	Rapid recovery
2	5	F	Comatose	3	2	Slow recovery (32 hours)
3	8	F	Drowsy—comatose (fluctuant)	5	4	Rapid recovery
4	4	F	Comatose	15	11	Slight improvement. Died
5*	6	M	,,	19	7½	,, ,,

* Present case.

onset, with or without vomiting, of various grades of cerebral depression—varying from drowsiness to stupor to coma—have been observed. Some early mild cases have responded dramatically and immediately to large doses of intravenous glucose, while others have recovered after some hours of treatment.

The present case can be regarded as a fulminant example of so-called vomiting sickness—coma coming on in only one hour, if the father's story is to be believed, while vomiting was virtually absent. Clinical response to a large dose of intravenous glucose was very slight, possibly because of the relatively long period—seven and a half hours—elapsing between the onset of coma and the beginning of treatment. The prognosis in these cases seems directly related to this interval, possibly because irreversible changes can be produced if hypoglycaemia is prolonged.

The toxin responsible for this dramatic clinical syndrome is unknown, but is very probably an accidentally ingested poison, possibly from some wild plant used in one of the "bush teas." The biochemical mechanism producing the hypoglycaemia is also as yet undetermined, but it seems likely that some form of enzyme blockage may be produced, possibly in the enzyme systems responsible for hepatic gluconeogenesis. Whatever this may be, it is temporary and potentially reversible, as if recovery occurs, either spontaneously or following intravenous glucose, it is complete.

While hypoglycaemia is the most striking biochemical finding, there may be other upsets of blood chemistry as yet undiscovered. However, the presence of very low blood sugars in these cases does offer a possible immediate line of therapy, and it seems logical that cases of vomiting sickness should now be treated as medical emergencies with intravenous injections of concentrated glucose solution as soon as possible after the onset of symptoms.

Our thanks are due to Dr. G. Bras for carrying out the necropsy, and to Dr. S. J. Patrick for analysing the liver for glycogen.

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CONGENITAL ABSENCE OF BOTH KIDNEYS

A REPORT OF FOUR CASES

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Agenesis of both kidneys is a rare condition, only 171 cases having been reported since 1663. The four cases occurring at the West Middlesex Hospital described by us bring the total to 175.

The largest number reported was 20 by Potter (1946a, 1946b). These she found in a series of 5,000 necropsies on foetuses and newborn infants, and she estimated the incidence to be 0.3 per 1,000 births. At the West Middlesex Hospital the incidence was four cases amongst 9,940 deliveries (that is, 0.4 per 1,000 births).

The absence of both kidneys is usually accompanied by other gross or multiple deformities (Hinman, 1940). The sex incidence is predominantly male. Bilateral agenesis is compatible with intrauterine life, and does not appear to affect the quantity of liquor amnii. Survival after birth is short; the average duration of life in Potter's cases was 1½ hours. One case is reported to have lived for as long as 21 days, and another for 11 days.

Case 1

The mother, aged 40 years, had four previous normal pregnancies. The last child, aged 6 years, had an undescended right testicle. The other children, one boy and two girls, were normal and healthy. There was no history of congenital deformity in either the mother's or the father's family.

The early antenatal period appeared normal, although the mother later admitted that six weeks after the last menstrual period she had taken about 2.3 g. (35 gr.) of a quinine salt in one day, followed by 85 ml. (3 fl. oz.) of an ergot mixture and two tablets of an unknown substance.