Laboratory Findings

Stool specimens from 24 of the 25 affected children were sent to the laboratory on the first or second day of illness. It was noticed at once that 17 of these were unusually pale in colour, and most were fluid. Fourteen were given careful microscopical study as well as a bacteriological examination; 10 were examined bacteriologically only. The bacteriological examination included a special search for heat-resistant strains of *Cl. welchii*, and for *Bact. coli* of the possibly pathogenic D 433 and "Aberdeen beta" types; but these organisms were not found; nor were shigellae or salmonellae. All organisms isolated were recorded; but not one organism of any kind was common to even a majority of cases. It thus appeared that no help could be obtained from the bacteriological examination; and attention was redirected to the microscopical findings. No cysts, worms, ova, or protozoa had been found, and no leucocytes or red cells; but globules of neutral fat (staining with Sudan) had been seen in 9 of the 14 cases examined microscopically. These globules were mostly from 10 to 30 μ in diameter, and were numerous in every microscopical field.

Castor oil, paraffin oil, and cod-liver oil were considered; but the first two had not been given; and faeces specimens from children from another nursery, in which a similar diet and the same amount of cod-liver oil were being given, did not show this undigested fat.

It therefore seems that steatorrhea was an abnormality associated with the outbreak of gastro-enteritis described, and that the characteristic stool was pale or putty-coloured, fluid, and acellular, with, in most cases, much undigested fat recognizable microscopically.

Discussion

A great deal of gastro-enteritis occurs in the community, and often no attempt is made to obtain an explanation. When multiple cases occur within an institution, however, a single cause is reasonably suspected and it is usual for some investigation to be made.

In the majority of such outbreaks referred to this laboratory shigellae, salmonellae, or *Giardia* have been incriminated; but in four recent incidents of the kind, including that described above, no pathogen was isolated from the faeces and the circumstantial evidence did not suggest food-poisoning. An additional search for heat-resistant *Cl. welchii* and for *Bact. coli* of types associated with infantile enteritis was without result. It was noticed, however, that the stools of these patients were often of a pale putty colour, and that on microscopical examination leucocytes were almost always absent, however fluid the faeces, while globules of undigested fat were quite often present in specimens from children.

The occurrence of multiple cases of this condition, some of which exhibited fever over periods of four days, suggested an infective agent, but the negative results of many bacteriological examinations imply that the organism, if it exists, is not easily cultivated, and perhaps a virus aetiology should be considered.

The disorder would seem to have affected the digestion of fat in the small intestine, since it started as central abdominal colic with flatulence and was often followed by the passage of pale fatty stools. The absence of leucocytes indicated that there could not have been much inflammation of the large bowel mucosa.

In addition to the minor epidemic incidents referred to, sporadic cases of gastro-enteritis with similar features have been encountered, but in these isolated cases many explanations are possible, and such conditions as subicteric infective hepatitis, idiopathic steatorrhea, and unsuitable diet have to be considered in diagnosis. It would be of value in such cases, and especially when they occur in outbreaks, to

have a biochemical analysis of the stools, and to examine blood and urine for bile factors. Bile has not been found so far in a few urine specimens from similar cases.

The syndrome can be recognized. Further observations are needed to determine whether it is rare or common, and to throw light on its cause or causes.

DEATH FOLLOWING A BARIUM ENEMA IN A CHILD WITH HIRSCHSPRUNG'S DISEASE

BY

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It is generally recognized that barium enemata must be given with caution in cases of megacolon, because of the danger of precipitating intestinal obstruction. The following is an account of a case in which a child suffering from megacolon died after a barium enema, but from a different cause.

Case Report

The patient, a boy aged 3 years and 10 months when he was admitted to hospital on November 28, 1949, was the third child of healthy parents. The family history revealed nothing of any significance. There was no previous history of fits in the patient or in the family. He had been constipated since birth and had regularly been given soap suppositories. With this stimulation a hard brown stool was passed every three or four days.

Examination showed that the abdomen was grossly distended and tympanitic. In contrast, his limbs were wasted. His weight was 35 lb. (15.9 kg.). No other abnormalities were discovered. Before a barium enema he was given soap enemata daily. Less than 1 pint (570 ml.) of fluid was given on each occasion without ill effect. His bowel appeared to have been satisfactorily cleared of faeces on the day of the investigation, although the abdomen was still distended.

Four pints (2.3 litres) of barium emulsion was introduced and the x-ray films showed the typical appearance of Hirschsprung's disease with a short aganglionic segment. An attempt was made to siphon back the emulsion without result, and on return to the ward it was noticed that the child's condition was unsatisfactory. He was cold, collapsed, and sweating. In an effort to retrieve the enema emulsion, a soap enema of 1½ pints (850 ml.) was introduced, but this too was retained. The sweating persisted and he began to vomit dark-coloured fluid. About seven hours later he lapsed into coma, became cyanosed, and began to have generalized convulsions. This condition persisted through the night and sedatives were given repeatedly. It was noticed that large quantities of very dilute urine (S.G. 1001) were being passed. Examination of the central nervous system at this stage showed no abnormal physical signs apart from the unconsciousness, dilated pupils which reacted sluggishly to light, and bilateral Babinski reflexes. He eventually died in a convulsion 20 hours after the enema had been given.

Permission for necropsy was not obtained, so the cause of this sudden death cannot be proved with certainty, but it seems probable that it was due to water intoxication.

Discussion

In this connexion Rowntree (1926) demonstrated certain phenomena in man and in experimental animals. If a patient with diabetes insipidus began treatment with pituitrin but did not at the same time limit his excessive water intake

he developed weakness and vomiting. If large amounts of water were given to normal dogs by stomach tube they developed retching, vomiting, and violent convulsions, ending in death. The only abnormality found after death was oedema around the cerebral blood vessels. Rowntree also found that the convulsions could be prevented by cerebral decompression operations.

Helwig, Schutz, and Curry (1935) reported the case of a woman aged 50 who by mistake received 9,000 ml. of water by proctoclysis in 30 hours. She became ill with sweating, vomiting, and pain in the neck. She passed into a stupor about 20 hours from the beginning of the symptoms and died 13 hours later after a series of convulsions. At necropsy the lungs and cardiovascular system were normal; the dura was very tense and the brain oedematous, with convolutional flattening. Microscopy showed perivascular oedema.

More recently, Hiatt (1951) noticed that patients with megacolon suffered from sweating, anorexia, and weakness following enemata. He showed that the rate of absorption of water from the colon was dependent on the intraluminal pressure, hence the symptoms were precipitated by straining, and investigations of the plasma electrolytes revealed that these symptoms were accompanied by a sudden marked drop in the sodium and chloride concentrations.

In the absence of any other satisfactory reason for death, it appears probable that this child died from water intoxication. The weight of Helwig and Schutz's patient is not stated, but if, for the sake of argument, it is assumed to have been 140 lb. (63.5 kg.) the lethal amount of water in her case was 9,000 ml. given in 30 hours. The equivalent amount for a child of 35 lb. (15.9 kg.) would be 2,250 ml. The child here reported received 3,124 ml. in a few hours. The symptoms and signs were those of water intoxication. The possibility of barium poisoning was investigated; no soluble barium salt could be found in the mixture that was used, and the solubility of barium sulphate is so slight that a toxic dose could not be dissolved in 4 pints (2.3 litres) of water.

The danger of water intoxication must be remembered when enemata are given to children with megacolon. The volume of the enema fluid must be limited, and every effort be made to retrieve it. Hiatt suggests using isotonic salt solution, which, if absorbed, will not tend to cause cerebral oedema; but an increase in the circulating blood volume will result, which could conceivably produce pulmonary oedema. He suggests that the fluid could be made hypertonic by adding some innocuous non-diffusible substance, say 7% gelatin.

The symptoms of water intoxication are due to the increase in interstitial and intracellular fluid. Treatment of an established case should aim at causing a shift of this fluid back into the circulatory system. The treatment recommended is the transfusion of hypertonic saline, and the amount of sodium ion that is needed can be calculated if the volume of absorbed water is known.

If circulatory failure occurred owing to a rapid increase in circulating volume a venesection would be necessary.

I wish to express my grateful thanks to Professor W. F. Gaisford for his helpful criticisms.

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W.H.O. and the Rockefeller Foundation have sponsored a pilot survey, to be carried out in England and France, on the types of health and welfare workers best qualified to meet the needs of the family (International Hospital Federation *News Bulletin*, March, 1952). The survey has begun in Bedfordshire, and will be extended to London shortly; work is also progressing in France.

Medical Memoranda

Spontaneous Rupture of a Leukaemic Spleen

Although Kaufmann (1929) mentions that a leukaemic spleen may rupture spontaneously such cases are rare enough to warrant being placed on record.

CASE REPORT

A Sinhalese man aged 56 was admitted with the history of pain in the upper part of the abdomen of two months' duration. The pain was continuous and unrelated to meals, but he vomited occasionally after taking food.

On examination he was pale but fairly well nourished. His pulse was 76 and temperature 98.4° F. (36.9° C.). No abnormality was found in the cardiovascular and respiratory systems. There was a hard smooth mass in the left hypochondrium, reaching to the umbilicus, which appeared to be an enlarged spleen. No lymphatic nodes were palpable. X-ray examination after a bismuth meal did not reveal any abnormality in the gastro-intestinal tract. The stomach appeared to be in the normal position, but the left kidney was pushed downwards. The leucocytes totalled 26,200 per c.mm., and the differential count showed 16% polymorphs and 84% lymphocytes. Most of the lymphocytes were mature and belonged to the small variety. No malaria parasites were seen in the peripheral blood.

As the pain became more severe and was unrelieved by any medication, it was decided to explore the abdominal cavity. This was done two days after admission, by a left paramedian incision. There was a fair quantity of blood in the peritoneal cavity. The spleen was found to be considerably enlarged, and several ruptures exposing the splenic pulp were noticed. The lymphatic nodes at the hilum and alongside the splenic artery were also enlarged. The liver was slightly enlarged, but palpation failed to reveal any nodules. He was given 10 oz. (280 ml.) of blood, and splenectomy was carried out by Mr. R. Anthonis.

* A blood count one month after the operation showed: erythrocytes, 3,595,000; Hb, 60%; leucocytes, 30,000 (polymorphs 25%, lymphocytes 69%, eosinophils 6%).

Description of Specimen.—The spleen measured 22 by 18 cm. and weighed 1,800 g. There was an extensive rupture exposing a large area of splenic pulp on the visceral surface and adjacent part of the diaphragmatic surface. Two irregular linear ruptures extended from this area towards its upper border, and a triangular-shaped rupture was seen towards its lower pole. The rupture over the diaphragmatic surface involved almost the whole breadth of the viscus (see illustration). Two linear ruptures extended from the splenic notch towards the diaphragmatic surface. The sectioned surface of the spleen appeared nodular. Localized areas of perisplenitis were found on the diaphragmatic surface. Microscopically the pulp was seen to be overrun with closely packed lymphocytes, which were uniformly distributed, obliterating the Malpighian corpuscles. Numerous mitoses were seen. The lymph sinuses were packed with lymphocytes.

Diaphragmatic surface of spleen.