respectively, to be followed by adenovirus type 5 again. This must have been simultaneous double infection, and the laboratory result merely reflected that type 5 was normally dominant but occasionally the other types predominated. In a recent trial a similar picture was found when children were fed three types of poliovirus simultaneously (Hale *et al.*, 1961). One or two viruses would be isolated in the faeces, then the third would appear; this was followed by the irregular reappearance of all types.

In spite of the frequency of isolation of adenovirus the general health of the community remained high. Mild coryzas were common, but never seemed to be related to the appearance of any adenovirus in the stools of those affected. Adenovirus type 7, an organism usually regarded as an epidemic strain, did not spread or produce clinical disease. This virus was introduced by a nurse in November, appeared in a toddler in December, and, after being excreted by a baby a month later, disappeared.

Adenovirus type 5 was isolated from no less than 50% of the children in the home, but never seemed to be responsible for any clinical illness with the possible exception of the outbreak of bronchiolitis described above. There are a number of hypotheses which might account for this outbreak. A new type 5 strain of virus may have been introduced during December which, when encountered by the very young and not the older children, caused respiratory illness; alternatively, perhaps conditions at the time might have been such that if type 5 virus infection took place illness resulted. On the other hand, the bronchiolitis at this period might have been due to an agent undetectable by available methods, or to this agent in combination with adenovirus type 5 (Gardner *et al.*, 1960b). These cases, however, emphasize the difficulty in deciding the aetiology of a disease which may be due to a virological agent that can be carried by a large number of normal individuals showing no sign of disease.

As a result of a constant influx of new children and prolonged excretion by some residents, conditions for endemicity are established. Communities of this type, in which viruses are epidemic, may act as foci of infection for the general population.

Summary

A residential nursery has been studied over a period of a year for the excretion of adenovirus. Types 1-7 were found in stools, and in a number of children adenovirus type 5 was excreted for many months. Many children were capable of carrying more than one adenovirus, and the significance of this is discussed. The relationship of adenovirus excretion to the general health of the community is discussed, with special reference to an outbreak of bronchiolitis which occurred during the period of study.

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TABETIC GASTRIC CRISIS WITH FLUID LEVELS AND VISIBLE PERISTALSIS

BY

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Gastric crises are recurrent paroxysmal attacks of severe abdominal pain, with or without vomiting, occurring in patients suffering from tabes dorsalis. Their onset is usually sudden, and an attack may last hours, days, or weeks, and usually subsides rapidly and completely. The incidence of these attacks has declined during recent years with the decrease in cases of tabes dorsalis. However, the condition is still seen from time to time in different branches of medical practice, and the importance of considering the possible diagnosis of gastric crisis in patients complaining of severe abdominal pain cannot be stressed too frequently. Unnecessary surgical interventions in such patients occur so often that the presence of abdominal operation scars with pain has been alluded to by Tilney as one of the diagnostic signs of this syndrome.

The incidence of gastric crises in cases of tabes dorsalis appears to be about 12%. Bonar (1901), at Columbia University, found 36 cases of gastric crises among 286 cases of tabes dorsalis. Nuzum (1916) reviewed 1,000 cases of tabes and found that gastric crises were the initial symptom in 17%. The Cooperative Clinical Group in the United States (O'Leary *et al.*, 1938) studied 985 patients with tabes dorsalis, gastric crises occurring in 118.

The cause of gastric crises remains undetermined. Some authors regard the attacks to be of sympathetic origin. Others believe that they result from recurrent posterior ganglionitis in the lower dorsal segment of the cord. Others regard involvement of the vagus as responsible for the symptoms. Gowers (1900) showed that in cases of tabes with gastric crises there were marked lesions in the upper cervical segments of the cord. Eppinger and Hess (1917) regarded visceral crises as the outcome of irritation and later of paralysis of the autonomic nervous system. The only factors known to induce attacks are acute infection, emotional tension, and severe fatigue, but the majority occur

without obvious precipitating factors. Bennett (1925) thought that most crises occurred in the pre-ataxic stage of tabes.

The differential diagnosis of tabetic gastric crises includes a number of abdominal conditions. Peptic ulceration with or without perforation, gall-stones, cholecystitis, acute pancreatitis, appendicitis, renal colic, salpingitis, lead colic, peritonitis, and intestinal obstruction are the commonest intra-abdominal conditions which have to be considered. More rarely intrathoracic disease involving the diaphragmatic pleura, the pre-herpetic stage of herpes zoster, Henoch's purpura, acute porphyria, and dissecting aneurysm of the aorta have caused confusion.

There appears to be general agreement that the prognosis is usually good. Holmes (1906) stated that "as a rule attacks usually become less frequent and severe and eventually cease as the disease advances." O'Leary *et al.* (1938) reported that gastric crises disappeared after treatment in 44% and were less severe in 25%. In 7% crises became worse.

Few radiological studies of the gastro-intestinal tract during tabetic gastric crises have been reported. Schlesinger (1918) administered a barium enema to a patient during a severe crisis and observed marked spasm of the descending colon. Fremont-Smith and Ayer (1925) reported abnormal x-ray findings of the stomach in 15 out of 24 cases of gastric crisis. Five were shown to have organic lesions of the stomach, while in 10 the abnormalities were thought to be due to spasm. Patrick and Reeves (1953) reported one case of abnormality of the peristaltic activity of the stomach during a gastric crisis which disappeared when the crisis subsided. In a detailed study of the literature we have been unable to find a report of fluid levels being found on straight x-ray examination of the abdomen during gastric crises.

This paper describes a case of tabetic gastric crisis in which visible peristalsis was observed on abdominal examination and fluid levels were found on x-ray examination of the abdomen.

Case Report

The patient was a 49-year-old man who worked as a caretaker. In 1955 he had suddenly developed severe epigastric pain and vomiting. The symptoms disappeared spontaneously in 24 hours and he was told that they were due to gastroenteritis. Two years later the epigastric pain and vomiting recurred, lasting for five days. He was admitted to hospital and a diagnosis of peptic ulceration was made. A year after this—in 1958—he again complained of the sudden onset of severe upper abdominal pain with vomiting. He was admitted to hospital, where signs of tabes dorsalis were discovered. His blood Wassermann reaction was positive and the cerebrospinal fluid showed 84 lymphocytes per ml., 42 mg. of protein, a positive Wassermann reaction, and a Lange curve of 0011210000. X-ray examination of the chest showed that the heart and aorta were normal. He was given penicillin treatment and recovered from the crisis in six days. In 1959, or six months before we saw him, he had a further gastric crisis. A straight x-ray film of the abdomen and a barium meal during the crisis showed the stomach to be dilated and to contain fluid and a large air bubble. No fluid levels were seen in the intestines. He recovered spontaneously in eight days.

On further questioning it was found that the patient had frequent attacks of abdominal pain occurring every few

weeks. These usually settled in a few days and did not require admission to hospital. His family doctor was in the habit of treating these attacks with pethidine.

In 1960 the patient was admitted to St. James's Hospital, Leeds, with abdominal pain and vomiting of seven days' duration. He had received repeated injections of pethidine and morphine from his family doctor, but these had not given him any relief. The pain was colicky in nature, most severe in the umbilical region, and radiated up to the epigastrium. There were no aggravating factors, and the pain was eased with vomiting greenish fluid, this occurring frequently. Anorexia was almost complete, his bowel habit was normal, and he had passed flatus on the day of admission.

On examination he was crying out with pain and requesting surgical intervention. He was pale and sweating. There was no abdominal distension, tenderness, or guarding, although he held himself rigid at the peaks of pain. Bowel sounds were present and rectal examination revealed nothing abnormal. The pupils were irregular and unequal, and reacted sluggishly to light and briskly to accommodation. There was mild bilateral ptosis. The tendon reflexes were absent at both ankles and sluggish at the knees. The plantar responses were flexor and there had been evidence of posterior-column loss on previous examination, but at the time of admission his pain was so great that co-operation was not sufficient to test sensation efficiently.

At this time straight x-ray examination of the abdomen revealed numerous fluid levels in the small intestine but no evidence of gross dilatation (see Fig.). When this x-ray film was shown to several surgeons who did not know the history of the case they all advised immediate laparotomy.

A Ryle tube was passed and repeated enemas were given with good results. A fluid-balance chart was begun, but it was not thought necessary to give intravenous fluids.

Next day he was noted to have visible peristalsis in the region of the umbilicus, and bowel sounds were present and augmented in bouts corresponding with the peaks of pain. A further straight x-ray film of the abdomen was normal and the fluid levels had disappeared. No further visible peristalsis was observed. On the third day he was symptomless, but on the fifth day he had a slight relapse

X-ray film showing fluid levels.

of his pain. This was rapidly relieved by two injections of pethidine. He then made a speedy and complete recovery and has been well until the time of writing.

Discussion

Fluid levels in straight x-ray films of the abdomen are usually thought to indicate intestinal obstruction. Shanks and Kerley (1950) state that "the cardinal signs in plain films of intestinal obstruction are gas-distended coils of gut and fluid levels." The finding of visible peristalsis on abdominal examination makes the diagnosis of subacute intestinal obstruction even more probable. However, in this case the fluid levels had disappeared when the abdomen was x-rayed 24 hours later and the visible peristalsis was not observed again. The patient has been symptom-free and in good health for six months since this episode.

The course of the illness, with the sudden disappearance of the physical signs and the complete symptomatic recovery, suggests that the cause of the subacute obstruction was a reversible process. Severe localized spasm of the intestine appears to be the most likely explanation. Spasm of the large intestine and of the stomach and pylorus has been noted by previous observers (Schlesinger, 1918; Fremont-Smith and Ayer, 1925). Moreover, relief of symptoms in gastric crisis can often be obtained by giving atrophic sulphate intravenously (Alajouanine and Horowitz, 1932). The evidence therefore suggests that the fluid levels seen on the x-ray film of the abdomen were caused by intestinal spasm. A similar mechanism is probably responsible for the visible peristalsis, the vomiting, and, in some degree, for the abdominal pain.

Cases of tabetic gastric crises are sometimes seen in casualty departments and in surgical services where straight x-ray examination of the abdomen is often carried out routinely on all patients with acute abdominal pain. Attention is drawn to the fact that the finding of fluid levels on the x-ray film and of visible peristalsis on abdominal examination does not exclude the diagnosis of gastric crisis.

Summary

A patient known to have tabes dorsalis and to have suffered previous gastric crises was found to have fluid levels on straight x-ray examination of the abdomen and visible peristalsis on abdominal examination during a severe gastric crisis.

The significance of these signs in diagnosis and a possible mechanism for their production are discussed.

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Preliminary Communications

Haptoglobin Types in Natives from the Kuru Region and Other Parts of Melanesia

At an early stage in the study of the fatal neurological disorder kuru among Fore natives in the Eastern Highlands of Australian New Guinea, Bennett, Rhodes, and Robson (1958, 1959) reported the occurrence of distinctive family patterns in respect of the incidence of the disease. After a detailed examination of the family data which had been obtained by Robson and Rhodes during their field investigations in January, 1958, I was led to put forward very tentatively, and as basis for further study, a single gene model for kuru (Bennett *et al.*, 1958, 1959). This suggestion was that kuru might be under the control of autosomal alleles *Ku* and *ku* such that, in females, homozygotes *KuKu* and heterozygotes *Kuku* would be potential early-onset and late-onset victims respectively, while in males the homozygotes *KuKu* would be potential kuru victims with the heterozygotes clinically normal. Such a possible genetic basis for kuru, though rather improbable from some points of view, was formally consistent with the immediate family data available.

Extensive field studies carried out subsequently have shown that the distinctive family patterns in respect of this disease occur generally in the kuru region (Bennett, Gray, and Auricht, 1959). It is not certain, however, that the familial nature of kuru has its only explanation in this genetical basis. Other possible explanations—for example, a toxic factor with a familial component—must be borne in mind. In the present state of knowledge it is particularly important, therefore, to seek data of a type that may furnish a critical test of this genetical hypothesis.

In this connexion, the report by Curtain (1959) of a marked association of kuru with the haptoglobin gene *Hp²* appeared worthy of attention, for it seemed to me that it would be very difficult, if not impossible, to reconcile such an association with the genetical model for kuru. Consequently, it was important to carry out an independent test for this association. This has been done and the results have been placed on record (Bennett, Auricht, Gray, Kirk, and Lai, 1961). Our material did not give any support to the suggested association; the frequencies of the haptoglobin types in kuru victims and in the other Fore natives without kuru chosen for comparison were almost identical.

Curtain, Gajdusek, and Zigas (1961) have now reported their work in some detail and have included results of the retyping by Dr. R. L. Kirk, of Perth, of some of their original sera from kuru victims for the haptoglobins. There is no longer any significant difference in the values for the frequency of the *Hp²* gene in their kuru and "normal" groups, and the gene frequencies which they give are in fact close to those reported by Bennett *et al.* (1961). Though these gene frequencies are close together, however, there are very marked differences between the frequencies of the three haptoglobin types reported by Curtain *et al.* and those given by Bennett *et al.* Both series of typings are summarized in Table I. It also includes the haptoglobin frequencies for a further group of 29 "normal" Fore individuals typed recently in this laboratory by D. W. Cooper, using the method of vertical starch-gel electro-