ACUTE DILATATION OF THE STOMACH

LOUIS SOLOMON, M.B., CH.B.

Upington

Acute dilatation of the stomach has long been recognized as a clinical entity, yet it is only during the last few years that its pathogenesis has come to be understood, and too often it is still attributed to mechanical obstruction, 'post-operative hypersecretion', or simply post-operative aerophagy. Though not frequently met in general practice its importance must not be underestimated. It may occur when least expected, sometimes after the most trivial of operations, and occasionally unassociated with any operation at all. It is a serious condition (Starr[1] reports a mortality of 40%) in the best hands. In a country practice, unaided by laboratory reports on the state of the plasma electrolytes, it is frankly dangerous. Only early diagnosis, a thorough understanding of its pathogenesis, and bold treatment can avert disaster.

Two cases have been encountered during the last year, differing in their development, treatment and outcome, and illustrative of several aspects of the condition.

CASE 1

Mr. S. was a healthy European male of 54 years, over 6 feet tall and weighing approximately 240 pounds. He had undergone an operation for repair of a small umbilical hernia. The anaesthetic consisted of pentothal, nitrous oxide, oxygen, ether and Flaxedil, and was administered by myself. The immediate post-operative course appeared in no way unusual, but on the 2nd day, when the patient had not passed flatus, a purgative was given. On the 3rd day there was still no passage of flatus and an enema was administered, with a negative result. By the 4th day the abdomen was somewhat distended and the patient began to vomit—at first small amounts of greenish fluid, increasing steadily in quantity throughout the day and later of a turbid, brownish colour.

When the patient was seen on the 5th post-operative day it was immediately evident that his condition was poor. He was slumped in bed, listless and apathetic, complaining of thirst. His tongue was dry and he was clearly dehydrated. The pulse rate was 88 per minute; the blood pressure 105/60 mm. Hg. The temperature was normal. His abdomen was slightly distended and bowel sounds were absent. Urinary output was diminished (800 ml. in the ensuing 24 hours) and the specific gravity high. Fantus' test for chlorides[2] was not performed as the reagents were not available. Plasma-electrolyte concentrations were not measured at any time as the results could not have been received in less than 4 days.

A diagnosis of acute dilatation of the stomach was made, and confirmed radiographically. Gastric suction, with intravenous fluid-replacement, was commenced forthwith; during the ensuing 24 hours 2½ litres of brown fluid were aspirated and 4½ litres replaced intravenously (3 litres of 5% dextrose in water, and 1½ litres of normal saline).

On the 6th day his condition had improved slightly. The blood pressure was now 115/85 mm. Hg (still lower than normal for him) and his tongue was not as dry as before. Urinary output, however, was only 700 ml. in 24 hours, and dark, foul-smelling fluid was still being aspirated from the stomach.

In spite of the diminished urinary output potassium chloride (KCl) was now added to the intravenous fluids. It was estimated at this stage that he required about 8 litres of fluid for re-hydration, about 160 mEq. of potassium and a similar amount of chloride (12 g. KCl). Neither serum nor Darrow's solution was available, and only 4 g. of KCl solution could be found in the town. This was given at the rate of 1 g. per litre of fluid.

Under the circumstances it appeared to be dangerous to give 8 litres of fluid over the next 24 hours, and this assumption was confirmed on the 7th day when, in spite of receiving only 3½ litres, he developed bilateral pulmonary oedema. Two litres of faeculent fluid were aspirated during that day. The urinary output fell to 400 ml.

In the next 24 hours he received 3 litres of fluid (including 1 litre of dextrose in saline) intravenously, but over 3½ litres of faeculent gastric fluid were aspirated. Though grossly dehydrated his lungs were very moist now, and at times he became cyanotic.

At this stage his relatives insisted on having him transferred to Cape Town by air. During the flight he lapsed into coma and died as we stood by powerless to assist him in any way.

CASE 2

Ten months later the second case was seen. On 2 August 1954 Mr. M., a European male of average build, 56 years old, developed what appeared to be a typical attack of gastro-enteritis, with pyrexia, vomiting and diarrhoea. Treatment with sulphaguanidine and a bismuth mixture brought a prompt recovery and 3 days later he was feeling quite well. That evening, however, he started vomiting again, and by the next day the vomitus was copious, dark in colour and foul-smelling. There was no sign of improvement during the day, and early the next morning he was admitted to hospital, where 900 ml. of faeculent fluid were aspirated from the stomach.

On examination he presented a listless appearance, could not concentrate on conversation and at one stage actually got out of bed with the intravenous apparatus *in situ*. He was dehydrated but his tongue was not completely dry. The pulse rate was 74 per minute; the blood pressure 125/85 mm. Hg. Temperature normal. There was slight distention of the epigastrium, but no abdominal tenderness. Auscultation elicited occasional faint propulsive bowel sounds, and 4 times during the next day a small amount of flatus was passed per rectum. The urinary output was good (1,200 ml. during the ensuing 24 hours), the specific gravity was low (1·008), and tests for albumen, sugar and ketones were negative. Fantus' test showed a chloride concentration of less than 1 g. per litre. Gastric fluid was being aspirated by Wangenstein's apparatus, and during the ensuing 24 hours totalled 4 litres. It was turbid, dark brown and faeculent, and its reaction was strongly alkaline. Here, for the same reason as in case 1, estimations of plasma electrolytes were not carried out.

Within 12 hours of admission a confident diagnosis of acute dilatation of the stomach was made. The quantity and character of the gastric fluid, the state of alkalosis and the minimal bowel sounds all pointed strongly in that direction. Radiography showed a dilated stomach and several dilated loops of small bowel with fluid levels (this in no way invalidated the diagnosis as it is known that the duodenum and jejunum are frequently involved with the stomach). Although the possibility of intestinal obstruction was borne in mind throughout, it was felt that acute potassium depletion had caused the condition and treatment was carried out on that assumption.

It was estimated from examination of the urine, the character

of the gastric fluid and the general condition of the patient, that he would require at least 7 litres of fluid during the next 24 hours, and about 12 g. of KCl intravenously. The proportion of dextrose solution to saline was to be controlled by the results of the Fantus test. A daily fluid-balance determined the fluid requirements for each ensuing period of 24 hours from 8 a.m. to 8 a.m.

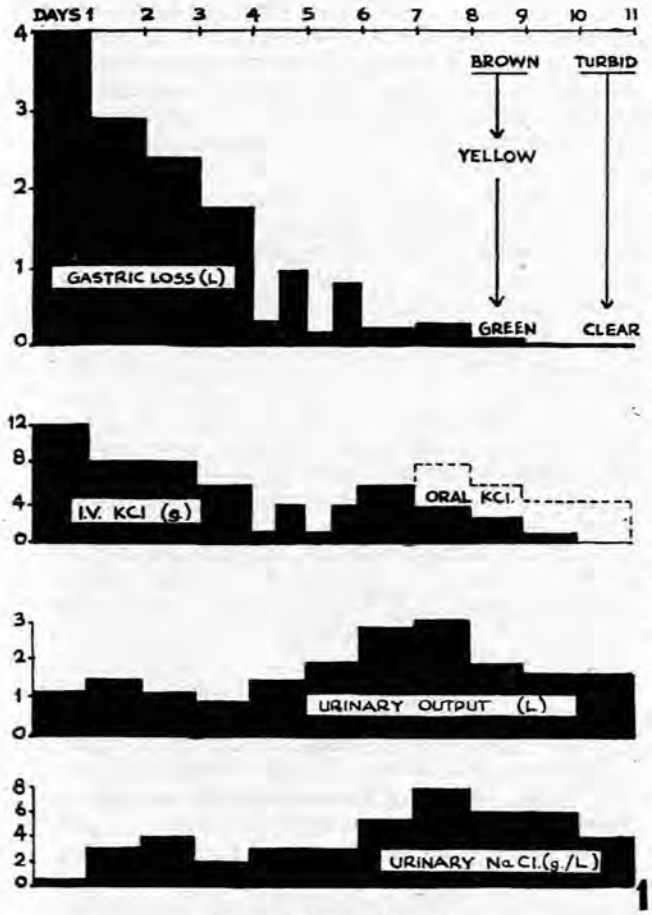

Fig. 1. Progress of case 2.

Fig. 1 shows the course during the period of treatment. Two points of particular interest appear. The first is the reaction of the stomach and bowel to KCl administration. The quantity and character of the gastric fluid rapidly returned to normal while KCl was being administered and by the 4th day of treatment less than 2 litres of clear green fluid was withdrawn in 24 hours. Bowel sounds were audible and a small soft stool was passed. At this stage it was decided to observe the response to variations in the KCl intake during the 5th and 6th days.

Starting at 8 a.m. on the 5th day the next 48 hours were divided into 4 12-hour periods during which the amount of KCl added to the intravenous fluids was to be varied. From 8 a.m. to 8 p.m. on the 5th day only 1 g. of KCl was given. During the next 12 hours 4 g. KCl were given. This was repeated on the 6th day.

The response was dramatic (Fig. 1). The sudden diminution in KCl intake was followed by an increase in gastric fluid to 1 litre in 12 hours, and a return to the brown, turbid, foul-smelling state. A rise in KCl intake during the second 12 hours was followed by an equally definite diminution in the gastric fluid to 200 ml. in 12 hours. This pattern was repeated on the next day.

By the 8th day, although the patient was now receiving most of his fluids, as well as KCl and NH_4Cl, orally, the intravenous KCl administration was still 4 g. per day, and only by the 10th day was the intravenous route abandoned.

The second point of interest is the marked and sudden diuresis accompanied by an increase in the urinary chlorides, which occurred on the 7th day, when intravenous fluids were being decreased. This event heralded recovery and by the 12th day the patient was on a normal diet again. Its significance is discussed later.

Soon after recovering the patient was sent to Cape Town, where thorough clinical, radiographic and laboratory examinations were carried out. Nothing abnormal was found. At present, after 4 months, he is still well.

ETIOLOGY

As long as 30 years ago electrolyte deficiencies were already thought to be responsible for some cases of paralytic ileus, but attention centred mainly on the sodium and chloride ions. It was not until 1943 that Ariel and his colleagues[3] reported 5 cases of post-operative ileus associated with a sodium-chloride-refractory hypochloraemia, in which recovery took place after parenteral and oral administration of protein; others had noticed an equally favourable response in cases receiving serum intravenously.[1] This led to work on other electrolytes and in 1949 Randall and others[4] suggested potassium deficiency as a cause of post-operative ileus and started using potassium in their treatment. Since then numerous reports on its value in paralytic ileus and gastric dilatation have appeared.[5–9]

At the same time experimental work has supported the clinical findings.[10–12] Of particular interest are the experiments of Streeten and Williams on NaCl-depleted dogs. Intestinal paralysis was induced only when intracellular potassium was lost as a result of adreno-cortical activity due to chloride deficiency. *In vitro* experiments on intestinal segments immersed in different electrolyte media showed that a NaCl-deficient medium could abolish intestinal movements, but a potassium-deficient medium failed to do so. In the former case a low potassium concentration was demonstrated in the bowel wall.[6]

These observations indicate that intracellular rather than extracellular potassium deficiency is responsible for intestinal paralysis, and this is not surprising, considering that nerve conduction, acetylcholine formation and cell irritability are all dependent on the potassium level.[1, 13]

The maintenance of a constantly high intracellular potassium concentration is therefore of great importance. Although cell membrane is usually permeable to them, potassium ions (K^+) do not occur in osmotic equilibrium on either side of the membrane, the intracellular K^+ concentration (144 mEq./litre of cell fluid) being far greater than the extracellular K^+ concentration (5 mEq./litre). This gradient can only be maintained by active cation transfer, the small amount of energy required being supplied by the metabolizing of glucose.[13] The tendency for sodium ions (Na^+) to enter the cell by osmosis is overcome by the same process.

The conditions under which K^+ is lost from the cell are still incompletely understood. Experimental work has shown that the cell, far from being impermeable to Na^+ ions, will under certain circumstances attract them at the expense of K^+.[13–15] For instance, in a state of K^+ deficiency a small amount of Na^+ will enter the cell. If a NaCl infusion, potassium-free, is now given even more

Na^+ enters the cell, displacing K^+. If K^+ is infused a sodium diuresis occurs.

Deoxycortone acetate, apart from its well-known action of increasing K^+ excretion in the urine, has been shown to produce a similar exchange of Na^+ for cellular K^+ in experiments on rats.[16] ACTH and cortisone also cause an increase in urinary K^+ and, clinically, adrenocortical hyperactivity has the same effect.[6, 14, 17]

Furthermore K^+ conservation in states of depletion is notoriously poor. Even during starvation the kidneys continue to excrete K^+ which is lost from the cell during protein katabolism.

These mechanisms are seen at work in the 2 cases described. In case 1 an intravenous infusion poor in potassium and containing NaCl resulted in a rapid deterioration, with salt and water retention ending in pulmonary oedema. In case 2 adequate potassium administration resulted in a return to normality with a chloride, and presumably a sodium, diuresis on the 7th day.

Turning now to the role of K^+ in the digestive system, it becomes evident that it is important not only in cell metabolism but also in maintaining the composition and reaction of the digestive juices, and the activity of their enzymes.[1] This is effected by variations in the ratio of the bulk ions Na^+, K^+ and Cl^- in the different parts of the gastro-intestinal tract. Thus K^+ and Cl^- occur maximally in the proximal or 'secretory' portion, while Na^+ (and HCO_3^-) occur maximally in the distal or 'absorptive' portion, where the K^+ level is very low. K^+ and Cl^- are presumably essential for the maintenance of function in the proximal gut, where they occur in concentrations far in excess of the plasma values (11·2 mEq. K^+ and 116·2 mEq. Cl^- per litre are average figures for 'recent ileostomy fluid'[4]).

It is precisely these differences between the proximal and the distal gut which, according to Starr, justify the recognition of acute dilatation of the stomach as an entity apart from distal paralytic ileus, though related to it. K^+ and Cl^- deficiencies are liable to affect the proximal gut sooner than the distal gut. Conversely recovery from the deficiency state occurs earlier in the distal than in the proximal gut, because of its lower K^+ and Cl^- requirements, and gastric paralysis with hypersecretion may be maximal while bowel sounds and even bowel actions occur. Furthermore NaCl administration alone may effect recovery in cases of distal ileus, but will only aggravate a proximal ileus.

That the 2 conditions will overlap is to be expected and in all probability that is what occurs in the majority of cases. Nevertheless the predominant element should be recognized and treatment guided accordingly.

In both of the cases described the stomach was predominantly involved and in case 2 intestinal peristalsis was not abolished at any time.

PATHOGENESIS

When acute dilatation of the stomach is encountered it is almost always after an operation, the maximum incidence being on the second post-operative day.

About 2½ litres of gastric juice, containing 9—116 mEq. Na^+, 0·5—32·5 mEq. K^+, and 7·8—154·5 mEq. Cl^- per litre, are normally secreted per day, and totally reabsorbed in the intestines.[4, 18] A quick calculation will show the almost unbelievable fluid and electrolyte depletion which may follow non-reabsorption of this juice over a period of 3 or 4 days. Therefore, where an operation has been preceded by diarrhoea or vomiting, and where the post-operative period has been associated with further losses of fluids and electrolytes, particularly due to prolonged gastric suction, a potassium deficiency may be expected. When, in addition, treatment consists of saline infusion without potassium replacement an even greater, rapidly progressive, hypokalaemia is inevitable.

Acute post-operative dilatation of the stomach, however, does not occur only after abdominal operations, or in association with starvation, vomiting and gastric suction. It may occur after the most trivial surgical procedure on any part of the body. The mechanisms whereby K^+ depletion arises in these cases have already been mentioned. The role of urinary K^+ loss deserves further attention.

The post-operative changes in water and electrolyte metabolism are today well known. They occur even after minor operations, where hydration is adequate and intravenous infusions unnecessary. The important features are a retention of Na^+ and water, accompanied by a marked increase in K^+ excretion in the urine lasting for 48—72 hours.[14, 17, 19] This K^+ loss may amount to 'the equivalent of 10 g. KCl' in the first 3 post-operative days,[14] resulting in a negative K balance, at least as long as food is not taken.

It has been suggested that these changes are the result of adrenocortical hyperactivity initiated by surgical trauma. This view is based on the observation that ACTH and cortisone can cause all the changes seen post-operatively, including an eosinopenia as well as increased K^+ and nitrogen excretion.

It is probably not quite as simple as this, however, and Le Quesne[17] suggests that the Na^+ and water retention, at any rate, depends on at least 3 factors: (*a*) the secretion of an antidiuretic hormone by the posterior pituitary, causing primary water retention, (2) the secretion of salt-retaining corticoids by the adrenal cortex, and (3) renal haemodynamic factors. Whatever the exact mechanism, the result may again be a serious K^+ depletion in the presence of a raised extracellular Na^+ concentration.

So much for post-operative ileus. Of equal interest are those conditions associated with hypokalaemia where no operation has been performed. These include starvation,[7] prolonged diarrhoea or vomiting, whatever the cause (particularly in pyloric stenosis, infantile diarrhoea,[20] ulcerative colitis and steatorrhoea), diabetic ketosis, and chronic nephritis. It is surprising that paralytic ileus and acute dilatation of the stomach are so seldom reported in the literature on these conditions, but the fact that they do occur[7] is important, for here one has the opportunity of studying the condition uncomplicated by metabolic changes due to operation. Thus it may be found that the simple administration of one or two electrolytes brings about a recovery, and in the case described here it was actually demonstrated that improve-

ment and deterioration in the condition closely followed the variation in the amount of KCl administered.

Even in this case, however, adrenocortical hyperactivity cannot be entirely excluded as a contributory factor. Firstly, the amount of potassium lost at the onset of diarrhoea and vomiting alone could not have been very great, and secondly, although the chloride (and sodium) diuresis on the seventh day *may* have been due to the replacement of intracellular K^+, as suggested earlier, it may equally well have been due to the cessation of adrenocortical hyperactivity. Estimations of the K^+ lost in the urine would have been of tremendous interest.

That potassium plays an important, if not the most important, role in gastro-intestinal paralysis is no longer doubted. The exact degree to which adrenocortical activity contributes is far more uncertain.

DIAGNOSIS

Prompt and efficient treatment depends on early recognition. Diagnosis involves not only the discovery of adynamic dilatation of the stomach, but an assessment of the entire state of the fluids and electrolytes.

Copious vomiting in the early post-operative period will draw immediate attention. The more insidious case will continue to escape early diagnosis as long as surgeons refuse to use their stethoscopes. Undue delay in the onset of propulsive bowel sounds should at all times arouse suspicion, and the routine use of purgatives or enemas on the second or third post-operative day is not only unnecessary but may be positively harmful where the bowel is still in an adynamic state, or where bowel movement is just starting.[21] In case 1 diagnosis and treatment were delayed for 4 days, whereas in case 2 treatment was begun even before a definite diagnosis had been reached.

The established case presents with persistent, copious, effortless vomiting, the amount of gastric fluid being far in excess of 2 litres per day. The patient complains of thirst, the tongue is dry and the breath foul-smelling. There may be abdominal distension and some tenderness. Auscultation may reveal faint intestinal sounds and small amounts of flatus and even faeces may be passed, but advanced cases have an ominously silent abdomen associated with paralytic ileus.

Radiography of the abdomen will show the enormously dilated stomach. Often dilated loops of small intestine are seen as well and, since in advanced cases the condition merges into distal ileus, the entire gastro-intestinal tract may be involved.

Paralytic ileus due to peritonitis, typhoid fever, diabetic ketosis and uraemia must always be excluded since they require specific treatment apart from fluid and electrolyte replacement. There are also a number of conditions which may cause a so-called 'reflex ileus' but it is unlikely that they will be confused with acute gastric dilatation.[22] Finally it must not be forgotten that mechanical intestinal obstruction may terminate in paralytic ileus.

Symptoms and signs of hypokalaemia, apart from gastro-intestinal paralysis, are rarely encountered, and indeed it is still doubtful whether they can be attributed directly to the lack of potassium.[23, 24] Apathy and listlessness, muscular weakness and pains in the legs, are those most frequently reported and were present in both of the cases described here.

Examination of the urine is always profitable, and for the country practitioner indispensable. The volume, specific gravity and chloride content can all be measured clinically and provide information about the degree of salt and water depletion. When employing Fantus' test for urinary chlorides it must be remembered that in the presence of adrenocortical activity the values may be abnormally low owing to NaCl retention. There is no easy test for urinary potassium, but in the established case of post-operative ileus 1·5—3 g. of potassium may be lost each day.[6]

The electrocardiographic changes attributed to intracellular K^+ deficiency are not as reliable as formerly believed and may be absent in cases with undoubted K^+ depletion.[25] Those most often seen are prolongation of the Q-T interval, flattening or inversion of the T wave, and depression of the ST segment.

Estimations of the plasma K^+ concentration are useful when available but by no means indispensable, nor always reliable, as an indication of K^+ deficiency. It is quite possible to find a normal plasma K^+ concentration in the presence of a marked intra-cellular deficiency. Estimations of cellular K^+ have been made[6, 14] and in all probability the technique will eventually be perfected.

Of far greater practical importance is the frequent, if not invariable, occurrence of hypochloraemic alkalosis in acute gastric dilatation, because it is always accompanied by K^+ deficiency. Biochemical estimations of the degree of alkalosis have been found to be less reliable in many cases than clinical observations on the gastric juice.[1] Starr has drawn attention to the changes that occur; they are of the utmost importance: (1) The quantity is increased and exceeds $2\frac{1}{2}$ litres per day. (2) The colour changes from green to yellow and then to brown, the fluid becoming increasingly turbid and foul-smelling. It is vitally important to appreciate that faeculent vomiting in these cases is not due to regurgitation of intestinal contents into the stomach as a result of a low mechanical obstruction, and certainly not to a gastrocolic fistula. This point is stressed as it has been found that these are precisely the conditions commonly linked to the term 'faeculent vomiting'.

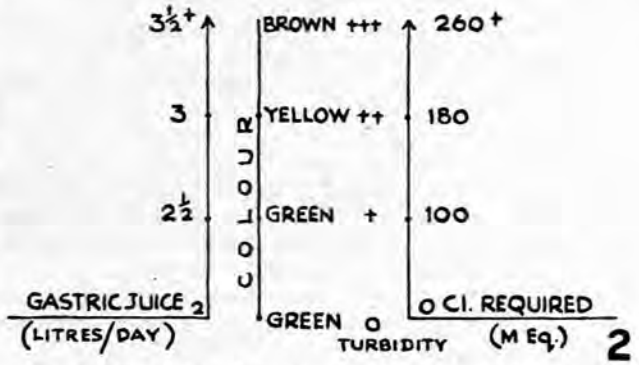

Fig. 2. Correlation between gastric juice and chloride requirements.

The chloride requirements have been correlated to these changes, as shown in Fig. 2.[1] Fantus' test can also be used to estimate the chloride loss in the gastric juice.

This hypochloraemic alkalosis is resistent to NaCl administration and will not be reversed unless the accompanying K^+ depletion is made up too.

TREATMENT

Once the condition has been recognized treatment must be prompt and bold. A delay of a few hours may result in death from oligaemic shock. Timid fluid and electrolyte replacement only delay the fatal outcome, and saline infusion without K^+ replacement increases the K^+ depletion, prolongs the state of ileus and results in circulatory failure or pulmonary oedema. Case 1 will always serve as a bitter reminder of this fundamental rule.

Serum and Dextran may be used to combat shock in the early stages but this must be followed as soon as possible by gastric aspiration and parenteral administration of fluid and electrolyte.

When calculating fluid requirements it must be remembered that an obviously dehydrated patient has a deficiency of at least 4 litres[22] and this must be made up in addition to the requirements for each day. Accurate fluid replacement is impossible without a meticulously kept chart recording the daily intake and output.

Correction of the Cl^- deficiency is guided by the changes in the gastric juice (Fig. 2) and is effected mainly by the use of KCl in order to correct the K^+ deficiency at the same time. If it is felt that this will provide too much K^+, part of it may be given in the form of NH_4Cl.

Describing the treatment of a case of post-operative gastric ileus Logan[9] states that 'rapid estimations of serum potassium are necessary, such as may be obtained with a flame photometer, and it is essential to check the serum levels daily or more often'. Fortunately for the country practitioner clinical observation and bedside tests can provide almost all the information he may need as long as he remembers that K^+ and Cl^- administration go hand in hand in the correction of the alkalosis. Parenteral administration of K^+ must be further guided by the following rules:

1. There must be a good output of urine.
2. K^+ replacement should not exceed 40 mEq. in 3 hours and the replacement solutions should contain not more than 40 mEq. K^+ per litre. (Note that 1 g. of KCl contains 13·4 mEq. K^+.) In the early stages it is best given in the form of KCl added to a solution of 5% dextrose in water, but once the initial deficiency has been corrected and the gastric juice regains its normal character all the bulk ions should be provided by giving a solution such as Darrow's, which contains 4 g. of NaCl, 2·7 g. of KCl, and 52 ml. of molar sodium lactate per litre. As a rule 1 litre of Darrow's solution per day will provide all the electrolyte requirements.
3. ACTH or cortisone should not be given.
4. Oral administration must replace parenteral administration as soon as possible. Here again Darrow's solution may be used.

The change-over to oral feeding is difficult and requires caution even when gastric motility has been re-established. Intravenous KCl administration must not be stopped until recovery is complete, but the amount is gradually decreased as the oral route is developed.

The clinical signs heralding recovery are easily recognized. The quantity and the character of the gastric juice return to normal; signs of dehydration disappear and the tongue stays moist; peristaltic bowel sounds return and flatus is passed regularly; the patient develops an appetite, becomes alert and active, and the days of hopeless despair are replaced by confidence and unbounded optimism.

SUMMARY

Acute dilatation of the stomach, though not often seen in general practice, is an important and serious condition. Two cases, encountered during the last year, are described. They illustrate most of the important features of the condition, as well as some of the problems in fluid and electrolyte therapy peculiar to a country practice.

Etiology, pathogenesis, diagnosis and treatment are discussed in the light of recent knowledge about the role of potassium in gastro-intestinal paralysis.

The character of the gastric juice is a reliable index of the degree of alkalosis accompanying the condition. This hypochloraemic alkalosis is resistent to sodium chloride administration and will not be reversed unless potassium is given too.

Although the emphasis throughout is on potassium deficiency, it must be realized that this is but one part of a generalized metabolic disturbance, every aspect of which should be considered during treatment.

I should like to express my thanks to Dr. A. Cohen for allowing me to attend his patients; to the Matron of the Gordonia Hospital for her willing cooperation during the treatment of these patients; and most of all to the nursing staff, whose careful attention under trying conditions dispelled most of the anxieties associated with prolonged intravenous-fluid therapy.

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