GASTRIC ILEUS DUE TO POTASSIUM DEPLETION AFTER GASTRO-ENTEROSTOMY

BY

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Failure of the stoma to work is an unpleasant and common sequel of gastro-enterostomy done for obstruction at the pylorus or in the duodenum. The clinical features are familiar to all abdominal surgeons.

I wish to suggest that this condition is a gastric neuromuscular paralysis or ileus due to potassium depletion and that it may be treated successfully by giving potassium. Potassium is essential in neuromuscular activity. Paralysis of skeletal muscle due to potassium lack, and its cure by giving potassium therapeutically, have been known for many years. That potassium depletion may cause intestinal ileus has been suggested by Randall *et al.* (1949), Darrow (1950), and Streeten and Ward-McQuaid (1952).

Most of the obstructive situations in which gastroenterostomy is employed are likely to result in potassium depletion. The reasons are various: low intake; the vomiting or aspiration of potassium-containing gastric juice; the excess tissue breakdown of starvation, leading to freeing of intracellular potassium and its consequent excretion by the kidneys; the administration of sodium, displacing potassium from the cell, and its consequent excretion—all are important. Displacement of potassium out of the cell for any reason will result in its excretion in the urine, for, so it is generally believed, there is no renal defence of potassium. Glycogen formation following the administration of glucose will result in the withdrawal of potassium from the extracellular fluid, though not from the body, since in this case it enters liver and muscle cells. Furthermore, it seems that to correct hypochloræmia in such a patient may be difficult unless hypopotaemia be first

corrected; and, if the unwise attempt is made, the great quantities of sodium necessarily given with the chloride will further deplete potassium and cause oedema. In short, both the obstruction and the usual therapy are likely to deplete body potassium, and the depletion will become the greater as urine flow is restored. The subject is discussed exhaustively by Darrow (1950), and by Randall *et al.* (1949) and Hawkins *et al.* (1951).

Case Report

The patient had an obstruction of the second part of the duodenum due to a gall-bladder carcinoma. On admission she was dehydrated, hypotensive, uraemic, hypochloreaemic, alkalotic, and in tetany. At this stage the serum potassium was low, there was general weakness, and she had bradycardia and frequent extrasystoles. Hawkins *et al.* noted frequent extrasystoles in a similar situation. The data are shown in the Table.

After rehydration, restoration of serum chlorides, and restoration of urine flow a gastro-enterostomy was done on the seventh hospital day. There was not the expected flow of gastric contents into the jejunum. Aspiration volumes continued greater than oral intake. Parenteral water, sodium, and chloride were continued in order to restore blood pressure and chlorides from their post-operative fall. At this stage the serum potassium fell to 10.9 mg. per 100 ml., the tendon jerks disappeared, the patient was oedematous, and the stoma still was not working on the fifth post-operative day. Potassium administration up to this time had been in such cautious quantities as to be ineffective, and much of it was given orally. On this and the following day adequate potassium was given rectally and, in addition, on the evening of the fifth post-operative day 1.5 g. of potassium chloride was injected intravenously. Within a few hours gastric aspiration decreased to nil and oral nutrition was thenceforth satisfactory. Sudden let-through into the jejunum, occurring spontaneously, is the usual end of this syndrome. It is suggested that the gastric paresis was cured in this case before it would otherwise have happened and that it could have been prevented by early adequate parenteral potassium. Confirmation must obviously await further experience of the method.

The Materia Medica and Therapeutics of Potassium

For oral and rectal use iso-osmotic potassium chloride solution (1.15%) is suitable. For intravenous administra-

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tion quarter-strength iso-osmotic solution (0.28%) is suitable. The solutions are easily sterilized in the autoclave. For subcutaneous use Hawkins *et al.* recommend iso-osmotic solution with hyalase to facilitate absorption. Given subcutaneously, potassium chloride solution is painful, but with the usual clouding of consciousness these patients will not, at least initially, object. Given intravenously, potassium chloride rapidly produces a painful phlebitis. The vein usually remains patent, and such a phlebitis is not an indication for changing to another vein or stopping the infusion.

It is probably safe to give a potassium-depleted patient a pint (570 ml.) of quarter-strength iso-osmotic potassium chloride solution intravenously in two hours. It is also probably safe to give a pint (570 ml.) of iso-osmotic potassium chloride solution subcutaneously to a similar patient in four hours. 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. In such a case as is here described potassium therapy should be started early and not when a pre-existing deficiency has been made worse by sodium and water administration. Nevertheless, potassium must not be given till rehydration is under way and a flow of urine has begun; otherwise fatal high levels may be attained in the extracellular fluid. Very great caution is required if there is associated renal disease or adrenal insufficiency. Potassium should not be given in the first 24 hours after operation.

I am indebted to Mr. Ian Fraser, in whose care this patient was, and to Sister Harrison, of the Musgrave Clinic, Belfast, for accurate records of treatment and for the devoted nursing which sustained this patient through a long illness.

BIBLIOGRAPHY

- Bellet, S. (1950). In *Postgraduate Gastroenterology*, edited by H. L. Bockus, p. 515. Saunders, Philadelphia.
 Darrow, D. C. (1950). *New Engl. J. Med.*, **242**, 978.
 Drabkin, D. L. (1950). In *Postgraduate Gastroenterology*, edited by H. L. Bockus, p. 498. Saunders, Philadelphia.
 Hawkins, C. F., Hardy, T. L., and Sampson, H. H. (1951). *Lancet*, **1**, 318.
 Randall, H. T., Habif, D. V., Lockwood, J. S., and Werner, S. C. (1949). *Surgery*, **26**, 341.
 Streeten, D. H. P., and Ward-McQuaid, J. N. (1952). *British Medical Journal*, **2**, 587.

PAROXYSMAL PARALYTIC MYOGLOBINURIA

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Paroxysmal paralytic myoglobinuria is a rare disease of unknown aetiology, affecting skeletal muscle and resulting in the liberation of myoglobin, which is excreted in the urine. The patient complains of painful muscle spasms and passes brownish urine.

Case Report

On December 13, 1946, a single woman aged 41 fell down "after a few gins" and sustained minor injuries. These were treated and she was sent home and advised to stay in bed. Three days later she suddenly developed cramps in both legs. At first the cramps were painless, but they soon became very painful and so severe that an hour later she was unable to stand. The pain started in the toes and spread upwards, first to the calves and then to the inner sides of the thighs.

On admission to hospital nothing relevant was found in her past medical or family history, and the temperature and pulse were normal. Physical signs were confined to the lower limbs, which were held extended with the feet in talipes equinus. All movements of the ankle-joints were limited by severe pain in the calf muscles, which were swollen, tender, and hard. Their "woody" consistency was similar to that felt in ischaemic contracture. The muscles on the medial side of the thighs were also tender, but their consistency was normal. All the signs and symptoms were more severe on the right side. The texture and colour of the skin were normal. The knee-jerks were normal, but the ankle-jerks were absent. The plantar reflexes were flexor, and there were no sensory changes. No pulsation could be felt in the popliteal, dorsalis pedis, and posterior tibial arteries on either side. Examination of the blood showed: red cells, 4,800,000; Hb, 96% (Haldane); white cells, 9,100 (polymorphs 80%, lymphocytes 15%, monocytes 4%, eosinophils 1%); platelets, normal; bleeding-time (Duke's) 2½ minutes; clotting-time 2½ minutes; prothrombin time 15 seconds. Wassermann reaction and Kahn tests were negative and the Donath-Landsteiner reaction was also negative.

Diagnosis was difficult. Thrombosis of the deep veins of the calves was considered most likely, and, in view of the absence of arterial pulsation and the possibility of permanent ischaemic changes occurring in the muscles, it was decided to perform a decompressing operation on the worst side. On December 16 a vertical incision 8 in. (20 cm.) long was made through the skin and deep fascia of the right calf. Contrary to expectation, the calf muscles did not bulge, and they looked paler than normal. The wound was left open, dressed with penicillin powder and petroleum jelly gauze, and supported on a back splint. Intravenous heparin treatment was started, and intramuscular penicillin was administered. Next day the legs were less painful, though the calves were still hard and tender. The patient said that the right calf felt a little easier after the operation. On this day it was first noticed that the urine was dark red. Clinically the specimen suggested severe haematuria, but microscopical examination revealed only an occasional leucocyte, a trace of albumin, and a heavy deposit of phosphates. There were no red cells or casts.

It was the investigation of this apparent paradox of haematuria without red cells which led to the diagnosis. The next day the urine was still red, but the following day it was normal in colour. During the ensuing days the symptoms and signs slowly subsided, the calves becoming softer and less tender. On December 24, eight days after the decompressing operation, secondary suture of the wound in the right calf was undertaken, and a piece of the right gastrocnemius muscle was excised for examination. Healing by first intention followed. The serum potassium level while at rest in bed was 15.5 mg. per 100 ml., and a day later after a brisk walk it was 17 mg. She was discharged from hospital free from signs and symptoms three weeks after admission. Unfortunately it has since been impossible to trace the patient.

Spectroscopic examination of the urine on December 17 showed an alpha band at 585 mμ. On substituting oxyhaemoglobin for the urine there was a clear-cut shift of the band. The position of the absorption band suggested myoglobin (Theorell, 1934; Millikan, 1939). A crude preparation of myoglobin obtained from human muscle gave a similar band at 584 mμ. The pigment was tentatively identified as oxymyoglobin, and this finding was confirmed by Dr. J. C. White.

Professor Crawford reported on the muscle biopsy: "The specimen consists of skeletal muscle which shows a scattered lesion involving individual fibres, shrunken and separated from their sheaths; they stain poorly and the appearances suggest that myoglobin has diffused out from the fibres. There is some increase in the nuclei of the sarcolemma and of the interstitial fibrous tissue. Special staining shows patchy loss of striation in the affected fibres.