Editorial

SURGICAL TREATMENT FOR OBESITY

Intestinal bypass as a treatment for obesity was first introduced by Payne in 1956. This approach was based on the fact that obesity is a serious disease leading to multiple other disorders, and conservative therapy usually brings no success. The criteria for selection adopted by most clinicians is that the patients should be 100 pounds above their ideal weight and other methods of weight reduction should have failed after being given a trial period of five years. There should be no endocrinopathy leading to obesity such as Cushings syndrome or hypothyroidism. The presence of certain complications as Pickwickian syndrome, hypertension, diabetes mellitus, degenerative arthritis of the hips and knees, which may be alleviated by weight reduction, are an added indication. Patient co-operation with a consent to accept the risks of surgery are a necessity.

The pre-operative evaluation of the patient with special attention to the endocrine status should be carried out. The blood biochemistry including a liver biopsy and an upper gastrointestinal tract series with a follow through will reveal any abnormalities present and these should be duly corrected.

Two main procedures are used for bypass surgery. The Payne operation or end to side anastomosis (Payne and Dewind, 1969), involves transection of the jejunum with attachment of the distal end to the side of the terminal end of the ileum. In the Scott operation or end to side (Scott and Law, 1969), the jejunum and ileum are both transected and anastomosed. The defunctionalized bowel loop so formed is drained into the transverse colon.

The weight loss brought about by intestinal bypass surgery is due to a decreased food intake together with malabsorption of the ingested calories. A decreased absorption of fat, nitrogen, calcium, potassium, carbohydrates and vitamins has been observed (Benfield et al., 1976; Barry et al., 1977).

The calories loss in the stool rises (Crisp et al., 1977) by an increase in the bile acid excretion which contributes to steatorrea. Weight loss by this surgical procedure is permanent, may range between 14 to 100 kilogram and a stable body weight is usually achieved between 12 and 36 months.

Inspite of its advantages this surgery is not free of complications. Pulmonary embolism, wound infection, gastrointestinal haemorrhage, renal failure and pancreatitis may be encountered. Most of the patients develop diarrhoea, the number of stools ranging from 8 to 20 per day in the early phase. This gradually declines to 4 to 15 stools per day after 6 weeks and by six months it decreases to 2 to 6 daily. Malnutrition in the form of hypoproteinaemia, vitamin deficiencies and loss of electrolytes in variable degrees may be manifested (Moxcley et al., 1974). Potassium loss is the most significant leading to hypokalaemic symptoms.

Fat deposition in the liver has been observed within one to six months post-operatively (Marubbio et al., 1976), the cause of which has not been clearly defined.

Pseudo-obstructive megacolon due to overgrowth of anaerobic organisms in the small and large intestines, giving a picture of intestinal obstruction may occur one or more years later after the bypass. Effective antibiotics relieve the condition.

An increased calcium excretion combined with fatty acids leads to an excess of free oxalates which get absorbed and produce urinary calculi. The use of a low fat diet and administration of calcium and antacids prevents this complication.

Arthralgia and polyarthritis may be observed. This is caused by antigens against E. coli and B. fragilis, occasionally found in the circulation of the bypass patients (Stauffer 1977).

With the likely post-operative complications and the technical difficulties due to obesity, poor respiratory reserve and splinting of the diaphragm the patients selected for this surgical form of treatment should undergo a very rigid criterion so also the operation should be performed with the collaboration of an experienced team of internists, surgeons and psychiatrists and good laboratory and intensive care facilities.

References

Barry, R.E., Barisch, J., Bray, G.A., Sperling, M.A., Morin, R.J. and Benfield, J. (1977) Intestinal adaptation following Jejunoileal bypass in man. Am. J. Clin. Nutr., 30:32.

Benfield, J.R., Greenway, F.L., Bray, G.A., Barry, R.E., Lechago, J., Mena, I. and Schedewie, H. (1976) Experience with jejunoileal bypass for obesity. Surg. Gynecol. Obstet., 143:401.

Crisp, A.H., Kaluey, R.S., Pilkington, T.R.E. and Gazet, J.C. (1977) Some psychosocial consequences of ileojejunal bypass surgery. Am. J. Clin. Nutr., 30:109.

Marubbio, A.T. Jr., Buchwald, H., Schwartz, M.Z. and Varco, R. (1976) Hepatic lesions of central pericellular fibrosis in morbid obesity and after jejunoileal bypass. Am. J. Clin. Pathol., 66:684.

Moxeley, R.T., Pozefsky, T. and Lockwood, D.H. (1974) Protein nutrition and liver disease after jejunoileal bypass for morbid obesity. N. Engl. J. Med., 290:921.

Payne, J.H. and DeWind, L.T. (1969) Surgical treatment of obesity. Am. J. Surg., 118:141.

Scott, H.W. Jr., and Law, D.H. (1969) Clinical appraisal of jejunoileal shunt in patients with morbid obesity. Am. J. Surg., 117:246.

Stauffer, J.Q. (1977) Hyperoxaluria and calcium oxalate nephrolithiasis after jejunoileal bypass. Am. J. Clin. Nutr., 30:64.