ANNOTATION

Ulcerative Colitis

The incidence of ulcerative colitis is greatest between the ages of 20 and 40, women being affected rather more often than men, and its natural history is notoriously variable. It is because of these unexplained variations that therapy is so difficult to assess. Rice-Oxley and Truelove¹ studied a series of 129 cases at Oxford and found that about 20 per cent. were of the acute fulminating type with considerable toxæmia while 14 per cent. had no more than a single episode of diarrhoea. Between these extremes 25 per cent. had chronic diarrhoea continuously and nearly 40 per cent. had repeated attacks over a number of years with long intervals of normal bowel function. Over 20 per cent. of the series were dead after one year and a third within five years. It is lamentable that the pathogenesis of this crippling condition is still unknown.

Infection is no longer thought to be the prime factor though it is not long since Hurst blamed the dysentery bacillus, Bagen implicated the organism eponymously named, and Dragstedt considered that the bacillus necrophorum might be responsible. Deficiency of vitamin A, an allergic response² and a lack of protective secretions in the ileum have been suggested without conclusive proof. The reasonable hypothesis that ileal enzymes when present in the colon in excess might digest the mucosa is not supported by surgical experience in human subjects or in experimental ileo-colic anastomoses. For many years the possibility of a psychosomatic basis has been acknowledged if for no other reason than the absence of other definite organic causes.

In 1930 Murray^{3,4} described the personality changes which are now associated with ulcerative colitis; Sullivan,⁵ Daniels⁶ and Wittkower,⁷ the last investigating Cullinan's patients in England, confirmed and amplified these views.

These and other investigators agree that about 60 per cent. of patients accord to one type of personality, immature over-dependant people who find it hard to break away from home and so to shoulder the burdens of everyday life. They give the impression of being agreeable friendly souls, though beneath this facade there is an undercurrent of resentment, frustration and indecision. Their aggression is often consciously bottled up and they are prepared to knuckle under rather than risk a quarrel or enjoy the luxury of overt anger. Can this queer trait be responsible for gross disease in the colon? Does the disease itself induce a passive helpless and despairing frame of mind because of its social and psychological stigmata? This problem is common to many of the psychosomatic disorders including peptic ulceration and bronchial asthma, diseases which are in some respects similar though the personality trends in each are different. Recent studies by Grace, Wolf and Wolff⁸ have drawn attention to changes in the human colon which accompany emotional disturbances and it seems safe to assume that they are relevant in clinical medicine. Mental trauma which might have no consequence in the normal individual, the inevitable turmoil of everyday life, may induce a state of hyperaemia and congestion in the colonic mucosa of the sensitive patient in whom frustration and indecision are prone to occur. Yet the true aetiological significance of such a mechanism is uncertain. Lium^{9,10} has said that chronic hyperfunction of the colon can be induced experimentally by mecholyl injections and that mucosal engorgement is followed by petechiae and ulceration. In such circumstances the lysozyme content of the colonic secretions is increased, as is said to be the case in human ulcerative colitis. The ability of lysozymes to dissolve the capsules of certain bacteria was re-

ported by Fleming¹¹ in 1922. They are present in tears and in gastric juice as well as in the large bowel and in pus. They seem to be basic polypeptides able to disrupt mucino-carbohydrates such as those found in bacterial capsules. It is probable that colonic lysozymes are increased in quantity in simple diarrhoea to a slight degree and by certain ganglion blocking agents and acetyl choline-like drugs. Such increases are not of the same extent as in ulcerative colitis. They are decreased by atropine, morphine and adrenaline. According to Meyer¹² whose viscometric method of estimation is often used, the levels are related to the severity of the disease. A tenfold increase is found in mild relapses, and in times of anger or frustration even during a remission of symptoms; during a severe attack 50 times the normal quantity may be found.

Unfortunately colonic lysozyme has no mucolytic power and only after contact for 24 hours does mucosal hyperaemia follow. Sammons¹³ was unable to show that lysozymes were of importance clinically but that another mucinase might be of significance.

Nevertheless some such substance may connect the disordered personality with the diseased viscus and would, by relegating the infective element to secondary importance, explain the absence of a specific organism. We may speculate that in a patient with an abnormally dependant personality a state of chronic hyperfunction of the colon is induced by excessive activity of the autonomic nervous system. These impulses probably reach the colon through the lumbo-sacral parasympathetic rather than via the vagus, and in this way a sustained elevation of lysozymes may follow. They, in turn, by disturbing the mucus covering lead to damage to the mucosa and predispose to non-specific bacterial infection. This is a long chain hypothesis and many of the links are weak, if not missing, yet the way is pointed to therapy not primarily directed to the colon. Although the personality cannot be altered it is possible that psychotherapy might help to avoid emotional upset which often precedes a relapse, or its harmful effect limited.

Advice concerning marriage, engagement or a new job may be of value, and a knowledgeable

general practitioner can be as successful as the psychiatrist (Paulley).¹⁴

The parasympathetic impulses may be blocked at a lower level by new atropine analogues or surgically by excision of the lumbo-sacral fibres. This operation is at present under investigation by Hinton and Sharihoff¹⁵ and its position appears to be as debatable as posterior pulmonary plexus dissection in asthma. Among the many treatments directed at the colonic mucosa, surface adsorbents and antibiotics seem the most promising though the search for new remedies has progressed little. Aureomycin seems to be the most effective antibiotic and the bowel is rapidly sterilized, but the reduction in diarrhoea and toxæmia is not reflected in the sigmoidoscopic picture. Dessicated ileum as recommended by Morton-Gill¹⁶ is no panacea, and ACTH has little to offer. Kirsner and Palmer¹⁷ treated 40 patients of whom 27 derived benefit though in none did the mucous membrane return to normal and over half subsequently relapsed within a few months. On the other hand the indications for surgical treatment have recently been clearly defined and summarized by Hardy and Brooke.¹⁸ The prohibitive mortality of 20 per cent. associated with ileostomy in the first cases has fallen to under 5 per cent. now that the importance of pre-operative preparation is realized and the operation performed earlier in the course of the illness. The modern type of Rutzen or Torbot bag has made the problem of ileostomy control less awkward and helped considerably to restore the ileostomy subject to a normal life.

But ileostomy alone is not curative and the normal anatomy can rarely be restored without recrudescence of the colitis, as Lahey¹⁹ has pointed out. He finds that ileo-rectal anastomosis is rarely feasible and that colectomy is to be advised in many cases. The persistence after ileostomy of rectal discharge, fistulae, toxæmia, haemorrhage and such remote complications as arthritis, skin sepsis and liver disturbances amongst which cirrhosis and bacterial hepatitis are to be included, are the main indications for colectomy. Some, including Lahey and Sloan,²⁰ would cite a 5 per cent. risk of carcinomatous change as an

additional hazard after the disease has been present for ten years.

It is a radical concept which would connect the personality disorder with the malignant change, but such speculation, though idle, is of interest.

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THE TREATMENT OF LUMBAR DISC-LESIONS

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The discovery that the common cause of back-ache and sciatica is a disc-lesion has caught the medical profession largely unprepared. For centuries these symptoms had been considered a reason for instituting treatment by heat, massage and exercises. These methods have now lost their last shred of theoretical justification but are being replaced only very slowly by acceptable alternatives. Nevertheless, a number of simple measures exist, none a panacea, each with its due proportion of successes. Few patients cannot be relieved if conservative treatment is intelligently used, and it is for only a few of this remainder that surgery need be considered.

Prophylaxis

The direction in which a damaged disc tends to move depends on the forces acting on it. During lordosis the inclination of the joint surfaces (Fig. 1) is such that the pressure of the body weight on the joint pushes the disc anteriorly. During kyphosis (Fig. 2) the tilt becomes reversed and the disc is pushed backwards. Hence it is only postures involving flexion that are apt to result in posterior displacement. The dura mater and the nerve roots lie posteriorly and are the sensitive structures that make the patient aware of disc-protrusion, the lumbar joints being in themselves all but insensitive. The important movement to avoid is stooping, especially when

weight-lifting increases the compression force on the joint. Industrial medical officers should teach workers how to lift, using their knees rather than their backs (Fig. 3), should see to it that heavy objects are presented at a suitable height and

FIG. 1.—Joint space in extension. Tracing of X-ray photograph of the fourth lumbar intervertebral joint in a normal subject, bending backwards. Note that the front of the joint space is $\frac{1}{2}$ in. (1.25 cm.) wide, but the back only $\frac{3}{16}$ in. (0.5 cm.). In this position backward movement of the intervertebral disc is virtually impossible.