

The most common sites for synovectomy are the knee, the small joints of the hands, and the tendon sheaths. Additional indications for synovectomy in the knee are the presence of a Baker's cyst—which can cause pressure symptoms—or a history suggestive of a ruptured Baker's cyst. If there are no erosive changes at the time of operation 80% of patients will be free of pain and have a good range of movement after a year. The results are not so satisfactory when erosions already exist. Erosions may develop rapidly in the joints of the hand, and even using special radiological techniques they may be impossible to detect. As with the knee, synovectomy should result in loss of pain and return of good function, but the operation requires technical excellence—particularly in cases in which erosions have developed.² A trial of unilateral synovectomies which suggested that synovectomy has a prophylactic value had to be abandoned because of a rapid deterioration in function in the unoperated hand.³ Excision should be considered in dorsal sheath effusions, particularly along the extensor pollicis longus or on the ulnar aspect of the wrist, since invasion of the tendon may lead to rupture. Synovectomy of the flexor tendons can relieve pressure on the median nerve in the carpal tunnel, triggering of the fingers, and pain on gripping.

More recently the value of excision of cysts connected with the shoulder and of synovectomy in the elbow joint has been emphasized. For the elbow synovectomy with debridement may be valuable in a late stage of the disease. In all the 25 cases described by D. W. Wilson⁴ changes were seen on x-ray examination. In 13 cases the head of the radius had to be resected, yet 76% of all patients had complete relief of pain and maintenance of function. There was a similar satisfactory result in 25 out of 28 cases reported by A. E. Inglis and colleagues.⁵ Occasionally synovectomy may be considered as a palliative procedure in late disease in other joints. In the intermediate stage a painful ulnar styloid process may need excising or ruptured extensor tendons to either the thumb or fingers require repair.

Joint destruction in the late stage of the disease is treated by arthroplasty or arthrodesis. Total replacement of the hip⁶ is valuable in many cases, though the risk of infection is increased in patients with rheumatoid arthritis. Many procedures are used for the knee, ranging from fusion (if few other joints are affected) to osteotomy, the insertion of a Platt or MacIntosh prosthesis, or total joint replacement.⁷ No doubt better prostheses will be developed, but even now they are a practical proposition. Encouraging trials of elbow and shoulder prostheses are under way. Metacarpophalangeal arthroplasty,² either by simple excision or with the use of the new prostheses,⁸ is a satisfactory procedure. In the foot excellent pain relief is achieved by the excision of worn metatarsal heads. Fusion of an unstable atlanto-axial joint is sometimes necessary for pain in the neck radiating up to the occiput or for neurological lesions. Other sites where fusion is particularly useful are the wrist, the hindfoot, and the interphalangeal joint of the thumb.

Since rheumatoid arthritis often affects many joints, particularly in late disease, multiple procedures may be required. Medical supervision, physiotherapy, and occupational therapy are essential adjuncts to surgery.⁹ Medical considerations may make surgical intervention inadvisable, and the timing of any operation should be a combined decision between the surgeon and the physician.

- ² Bailey, B. N., and Desac, S. M., in *Modern Trends in Rheumatology*, 2, ed. A. G. S. Hill, p. 240. London, Butterworths, 1971.
³ Nalebuff, E. A., in *Early Synovectomy in Rheumatoid Arthritis*, ed. W. Hijmans, W. D. Paul, and H. Herschel, p. 48. Amsterdam, Excerpta Medica Foundation, 1969.
⁴ Wilson, D. W., *Proceedings of the Royal Society of Medicine*, 1971, 64, 264.
⁵ Inglis, A. E., Ranawat, C. S., and Straub, L. R., *Journal of Bone and Joint Surgery*, 1971, 53A, 652.
⁶ Cockin, J., and Duthie, R. B., in *Modern Trends in Rheumatology*, 2, ed. A. G. S. Hill, p. 267, London, Butterworths, 1971.
⁷ Taylor, A. R., in *Modern Trends in Rheumatology*, 2, ed. A. G. S. Hill, p. 292, London, Butterworths, 1971.
⁸ Calnan, J. S., *British Journal of Hospital Medicine*, 1971, 5, 487.
⁹ Conalty, J. P., and Nickel, V. L., *Journal of Bone and Joint Surgery*, 1971, 53A, 642.

Irritable Bowel Syndrome

The irritable bowel syndrome is a symptom-complex which is frequently misdiagnosed and poorly understood.^{1 2} It has many synonyms such as irritable colon, colonic dysfunction, spastic colon, functional bowel disorder, muco-membranous colic, nervous diarrhoea, colon neurosis, and dyssynergia of the colon. Names such as mucous colitis are particularly misleading in that they suggest inflammatory disease, which may confuse the doctor and alarm the patient. Moreover emphasis on colonic disturbance is unwarranted, since there is evidence of abnormal motility throughout the intestine.³⁻⁶

The syndrome is often divided into two types on the basis of symptoms.⁷ In the commonest variety (spastic colon) abdominal pain is associated with an alteration of bowel habit which results in constipation, diarrhoea, or alternating periods of both; an uncommon variant consists of pain on its own. In the second main variety (painless diarrhoea) the patient has intermittent or continuous diarrhoea. Other abdominal symptoms may be present and include distension after meals, heartburn, flatulence, anorexia, nausea, and vomiting. In addition the patient may complain of fatigue, depression, anxiety, insomnia, fear of cancer, and other nervous symptoms.

This is a common condition and may account for up to 70% of the patients referred to a gastrointestinal clinic for diagnosis.⁸ It is especially common in women between the ages of 20 and 60 years and may occur in children,^{9 10} but it should not be diagnosed when symptoms occur for the first time in an elderly patient. The aetiology is unknown, but heredity, dysentery, and emotional stress may all play a part. Symptoms may persist long after the initiating factor has disappeared. The association with lesions elsewhere in the abdomen such as duodenal ulcer, gallstones, gynaecological disease, and the results of vagotomy is obscure. Symptoms may be erroneously attributed to these lesions, and the scarred abdomens of many patients attest to the failure of surgery to cure the irritable bowel. Continuing symptoms may even be blamed on adhesions or nebulous conditions such as the post-cholecystectomy syndrome. However, an association between the irritable bowel and diverticulosis coli is established.¹¹

Since there is disturbance of function without a structural defect, the diagnosis is usually made by exclusion of more serious diseases. However, the diagnosis need not be accepted reluctantly, for certain symptoms indicate exaggerated motility of the bowel and the risk of missing malignant disease is slight, at least in patients with diarrhoea.¹² Though constant or colicky pain may occur at any site in the abdomen, it is aggravated by eating or purgation and decreased by defaecation or passage of flatus; the pain is poorly

¹ Eyring, E. J., Longert, A., and Bass, J., *Journal of Bone and Joint Surgery*, 1971, 53A, 638.

localized by the patient (negative pointing sign). The stools often consist of pellets like rabbits' droppings, and the diagnosis is further suggested by the passage of large quantities of mucus; blood is absent. Diarrhoea is often limited to the early morning. The first motion may be normal, but subsequent bowel actions produce small quantities of stool or mucus, so that records of stool weight and volume are more useful than those of bowel frequency. Though there may be urgency of defaecation, incontinence does not occur; nocturnal diarrhoea suggests an organic disturbance. Another feature is constipation associated with pain which steadily increases until relieved by a bout of diarrhoea. Physical examination usually shows a fit, well-nourished patient—but not always. Borborygmi may be audible, and tenderness will be elicited over various segments of the colon and may diminish if palpation is continued.

Simple screening investigations should include measurement of haemoglobin, white cell count, estimate of erythrocyte sedimentation rate, and examination of the stool for occult blood, pathogenic bacteria, and parasites. Sigmoidoscopy may produce a typical attack of pain either on insertion of the instrument or after insufflation of air. Spasm of the bowel may throw the mucosa into rugose folds, which, though hyperaemic, do not bleed on wiping. Rectal biopsy is normal; if inflammatory changes are found, organic disease should be sought in both the large and small intestine.¹³ Barium enema provides evidence of irritability in about three-quarters of patients.¹⁴ Recordings of intraluminal pressure from the sigmoid colon or more proximal segments are not available as a routine procedure but are useful in relating attacks of pain to colonic dysfunction, whether the pain is spontaneous, post-prandial, or induced by prostigmine.^{5 15-17} Though it has been shown that patients with spastic colons tend to have increased motor activity whereas those with painless diarrhoea have flat tracings, the differences are not always clear-cut.^{18 19} Furthermore, pressure studies do not measure movement of the bowel wall or the transport of contents.

Recently I. G. Hislop²⁰ has reviewed 67 patients with particular emphasis on symptoms, psychiatric features, and response to antidepressive therapy. He noted a high incidence of pain in the back (51%), nocturnal pain (48%), and nocturnal diarrhoea, together with a delay in diagnosis which amounted to more than two years in 57% of the patients and to more than five years in one-third. Analysis of psychiatric factors showed that marital stress was more common

than in controls, but financial and childhood stresses were not greater, though psychiatric symptoms occurred with greatly increased frequency in all patients. Treatment with amitriptyline resulted in improvement or cure in 80% and was more effective than trifluoperazine. However, the trial was neither controlled nor double-blind, and the length of follow-up was not stated. Unfortunately there is little information on the prognosis of the irritable colon. S. C. Truelove¹ found that only 5% of his patients had intractable symptoms whereas S. L. Waller and J. J. Misiewicz⁶ found that 52% of their patients were unchanged or worse after 12 months.

The main object of treatment is to teach the patient to live with his symptoms by reassurance that there is no serious organic disease, simple explanation of disordered physiology, and sympathetic follow-up to reduce the effects of stress. Additional help may be derived from bulk-providing agents in the diet, antispasmodics, or remedies for the diarrhoea. It would seem wise to restrict the use of sedatives, tranquilizers, and antidepressive drugs to those patients who fail to respond to this simple regimen and to prescribe the drugs for a short period only. Dietary restriction should be instituted only if the symptoms can be certainly related to a specific item. Lactase deficiency is not of importance in the syndrome, and lactose-free diets are of doubtful benefit.²¹

¹ Truelove, S. C., "The Irritable Colon Syndrome," in *Recent Advances in Gastroenterology*, ed. J. Badenoch, and B. N. Brooke, p. 268. London, Churchill, 1965.

² Connell, A. M., *Postgraduate Medical Journal*, 1968, 44, 668.

³ Kalser, M. H., Zion, D. E., and Bockus, H. L., *Gastroenterology*, 1956, 31, 629.

⁴ Misiewicz, J. J., Connell, A. M., and Pontes, F. A., *Gut*, 1966, 7, 468.

⁵ Holdstock, D. J., Misiewicz, J. J., and Waller, S. L., *Gut*, 1969, 10, 19.

⁶ Waller, S. L., and Misiewicz, J. J., *Lancet*, 1969, 2, 753.

⁷ Chaudhary, N. A., and Truelove, S. C., *Quarterly Journal of Medicine*, 1962, 31, 307.

⁸ Kirsner, J. B., and Palmer, W. L., *Gastroenterology*, 1958, 34, 490.

⁹ Davidson, M., and Wasserman, R., *Journal of Paediatrics*, 1966, 69, 1027.

¹⁰ Stone, R. T., and Barbero, G. J., *Pediatrics*, 1970, 45, 732.

¹¹ Manousos, O. N., Truelove, S. C., and Lumsden, K., *British Medical Journal*, 1967, 3, 760.

¹² Hawkins, C. F., and Cockel, R., *Gut*, 1971, 12, 208.

¹³ Dyer, N. H., Stansfeld, A. G., and Dawson, A. M., *Scandinavian Journal of Gastroenterology*, 1970, 5, 491.

¹⁴ Lumsden, K., Chaudhary, N. A., and Truelove, S. C., *Clinical Radiology*, 1963, 14, 54.

¹⁵ Chaudhary, N. A., and Truelove, S. C., *Gastroenterology*, 1961, 40, 1.

¹⁶ Connell, A. M., Jones, F. A., and Rowlands, E. N., *Gut*, 1965, 6, 105.

¹⁷ Wangel, A. G., and Deller, D. J., *Gastroenterology*, 1965, 48, 69.

¹⁸ Chaudhary, N. A., and Truelove, S. C., *Gastroenterology*, 1968, 54, 777.

¹⁹ Ritchie, J. A., and Tuckey, M. S., *American Journal of Digestive Diseases*, 1969, 14, 96.

²⁰ Hislop, I. G., *Gut*, 1971, 12, 452.

²¹ Peña, A. S., Truelove, S. C., Lumsden, K., and Whitehead, R., *Gut*, 1969, 10, 1052.