in two of our cases (Figs. 4 and 5). In both these patients the palpatory findings were positively misleading.

Gynaecography is not, of course, a substitute for other radiological investigations, such as hysterosalpingography, which must be carried out to determine patency of the Fallopian tubes in cases of infertility. But the results of the present study indicate the value of gynaecography as a more routine procedure in patients with infertility or menstrual irregularity. The safety and simplicity of the procedure are of particular value in the young unmarried or nulliparous patient. In many instances the gynaecographic results taken in conjunction with the clinical and laboratory findings are sufficient basis on which to plan subsequent management and the indications for hormonal or operative intervention.

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Preliminary Communications

Obstruction of Bowel due to Lesion in the Myenteric Plexus

[WITH SPECIAL PLATE FACING PAGE 671]

Brit. med. J., 1969, **1**, 686-689

Summary: Two patients are described who were found to have a destructive lesion of the myenteric plexus. In one the upper small intestine was involved and the clinical picture was of recurrent pseudo-obstruction; in the other the lesion was in the distal colon and resulted in intractable constipation. The lesion cannot be demonstrated by conventional histological techniques, being seen only in silver preparations cut parallel to the bowel wall, and may have been overlooked in the past. The aetiology is unknown.

INTRODUCTION

Involvement of the autonomic nervous system is often invoked as a cause of disturbance of bowel function but is rarely confirmed by histological examination. We report two cases with a hitherto undescribed lesion of the myenteric plexus, which was demonstrated by examination of sections cut parallel to the bowel wall (Smith, 1967a). In the first case the lesion mainly involved the upper small intestine and resulted in recurrent unexplained intestinal pseudo-obstruction. The second patient had colonic involvement and presented with intractable constipation.

CASE 1

This patient gave a 19-year history of episodic unexplained intestinal obstruction. Symptoms had started at the age of 48, but there were no abnormal physical signs, though slight dilatation of the upper small intestine was shown by a barium meal. Symp-

toms had been severe enough to warrant laparotomy at the ages of 56, 60, and 65. Each time the stomach, duodenum, and upper jejunum were dilated, but no mechanical cause of obstruction could be found and the ileum was normal. During the third laparotomy hypertrophy of the wall of the bowel was described for the first time. Gastrojejunostomy and vagotomy were performed. After the third operation the abdominal symptoms failed to settle and he was found to have steatorrhoea (16-35 g. of fat excreted per 24 hours) and hypoalbuminaemia (2.8-3.2 g./100 ml.). Treatment with a gluten-free diet, steroids, and a wide variety of nutritional supplements failed to improve his condition. Five months later he developed loss of sensation in his right thigh, which was followed by progressively increasing weakness of both legs so that he had been virtually bedridden for seven months when he was transferred to this hospital. He was also losing weight progressively and suffering from bouts of abdominal pain, distension, and vomiting, which occurred two to three times a week.

Physical examination showed a wasted man with the skin changes of protein deficiency and moderate ankle oedema. The abdomen was distended and small-intestinal peristalsis was visible. Bowel sounds were loud and hyperactive.

Investigations showed a slight iron-deficiency anaemia (haemoglobin 10.2 g./100 ml.), hypoalbuminaemia (2.1-2.9 g./100 ml.), hyponatraemia (119-130 mEq/100 ml.), and hypokalaemia (2.9-3.5 mEq/100 ml.). Barium meal and follow-through examination showed gross stasis of barium in a markedly dilated upper gastrointestinal tract. Some barium had reached the lower jejunum at seven hours (Special Plate, Fig. 1), but most of it was still retained in the stomach and proximal loop at 24 hours. This stasis presumably accounted for the heavy contamination of the jejunal fluid by aerobic and anaerobic organisms, which had caused complete hydrolysis of conjugated bile salts. Serum antibodies could not be detected against *Toxoplasma gondii*, and virus was not cultured from the faeces.

In view of his disability a further laparotomy was performed on 12 September 1967. The stomach was hypertrophied and enlarged. There was gross dilatation of the duodenum and jejunum, and the bowel wall was thickened. At first the involved bowel was flaccid, then segmentation movements, but not progressive contraction waves, appeared after a brief period. There was a fairly sharp transition to macroscopically normal bowel in the lower jejunum. The ileum and colon appeared normal. The third and fourth parts of the

duodenum together with the dilated 100 cm. of jejunum were resected. An antrectomy was also performed, removing the pylorus and part of the antrum together with the old gastroenterostomy stoma. The stomach remnant was anastomosed to the normal jejunum and the second part of the duodenum to the jejunum at a lower level by an end-to-side anastomosis.

His postoperative progress was satisfactory; the episodes of intestinal obstruction ceased, the abdominal distension diminished, and barium follow-through showed no obstruction in the small intestine. Over the next five months he gained 28 lb. (12.7 kg.) in weight, his appetite and strength improved, and the skin changes and peripheral oedema resolved. He became mobile. His abdomen remained moderately distended and bowel sounds were loud and hyperactive. Laboratory investigations showed that his nutritional deficits had been corrected, but the features of the stagnant loop syndrome were largely unchanged. Ten months after the operation he again began to suffer from symptoms of subacute intestinal obstruction, but these have been mild and infrequent to date.

Pathology.—Macroscopically the smooth-muscle coats were thickened, the mucosal ridges were widely spread, and there was no evidence of ulceration or obstruction. Paraffin sections, cut transversely, taken at intervals through the jejunum confirmed the smooth-muscle hypertrophy, which appeared to affect the two coats equally and also involved the muscularis mucosae. The mucosa and submucosa were normal. The myenteric plexus showed some fibrosis; neurones were scanty, and those that were present were rather large. In one or two places, including the duodenum, macrophages were seen within the ganglia. Silver preparations were made of sections cut parallel to the bowel wall, and changes were shown which were more marked in the distal jejunum. There was a reduction in the number of neurones, and those that remained were very abnormal. They were enlarged with thick, irregular, and clubbed processes (Special Plate, Figs. 2 and 3). There was an increase in the number of Schwann cells in the nerve trunks and a reduction in the number of axons. Frequently a single axon remained in a mass of Schwann cells. Some of the remaining axons were beaded and fragmented, and a few pursued an irregular course outside the anatomical plexus and sometimes turned back on themselves. This damage to the myenteric plexus extended up to the distal end of the specimen.

CASE 2

This patient had suffered from constipation since the age of 10, and was often given aperients as a child. Constipation became more severe at the age of 26, when bowel actions occurred only every three to six days and resulted in firm, pellet-like motions. When aged 28 she was investigated at another hospital: no abnormal physical signs were noted and sigmoidoscopy was normal. Barium enema showed no abnormality, but she was unable to evacuate the barium. Barium meal and follow-through examination was normal.

She was first seen at this hospital at the age of 31, when bowel actions occurred about every fourth day, and the faecal weight varied from 80 to 265 g. Her entire colon was loaded with faeces, but abdominal distension was not obvious and bowel sounds were normal. Motility studies were performed by Dr. G. Misiewicz, who reported: "The colon showed a normal resting record, but the rectum was more active than the colon. There was some suggestion of a failure of propulsion, since the amplitude of the waves in the sigmoid was more diminished at 25 cm. than at 20 cm. from the anus. The responses to both neostigmine and a meal were abnormal, as inhibition was recorded in all leads instead of the usual stimulation." Studies with balloons in the anal canal were performed by Mr. N. Porter and showed that the pressure was normal at rest but that there was very little power of voluntary contraction. Electromyography of the anal sphincter indicated *normal resting activity*. Inflation of the balloon showed gross *blunting of conscious rectal sensation* and an enormous rectal capacity. There was no desire to defaecate until the volume was 650 ml., and *inhibition of the voluntary sphincters did not occur until 700 ml.* was reached.

Stretching of the anal sphincter failed to relieve her symptoms, and she was readmitted for surgery at the age of 32 (June 1966). A transverse colostomy was performed; this functioned well, and a Duhamel operation was therefore carried out. The abdominal organs were all normal. The rectal wall was hypertrophied, but the colon was collapsed beyond the colostomy, and there was no

evidence of megacolon. However, when the colostomy was subsequently closed the constipation persisted. Over the next six months she was readmitted on two occasions with faecal impaction, and sphincterotomy produced only transient improvement. Consequently a sigmoid colostomy was fashioned, but this never functioned satisfactorily. Therefore in March 1968 (aged 33) 46 cm. of the remaining distal colon was excised and an anastomosis made between the colon proximal to the site of the previous transverse colostomy, which had functioned well, and the rectum. It was hoped that effective emptying of the transverse colon into the restricted rectal reservoir would create a high enough intrarectal pressure to allow inhibition of the sphincter. Since this operation her constipation has dramatically improved.

Pathology.—Paraffin sections showed some thickening of the inner muscular coat. In the first (distal) resection specimen a few macrophages and lymphocytes were seen in scattered areas, especially at the proximal end. Neurones were scanty. In the second specimen only a few ganglia contained macrophages, and neurones were more frequent, though some were enlarged. Silver preparations of the first specimen showed a marked reduction in axons and neurones, the majority of which were shrunken and misshapen, though a few were enlarged and globular, with swollen processes. Collections of inflammatory cells were present in the ganglia. Loss of axons and neurones was less severe in the second specimen, and proximally some were normal. Other neurones were enlarged with a bizarre shape and swollen processes. Considerable increase in Schwann cells occurred in both specimens.

DISCUSSION

PATHOLOGY

The main macroscopic feature was thickening of the bowel wall, which was due to smooth-muscle hypertrophy. This hypertrophy occurs in the absence of normal innervation and is in marked contrast to the atrophy that develops in striated muscle under similar circumstances. It may be produced in the experimental animal by procedures that damage innervation (Ishikawa, 1923; Keeler *et al.*, 1966), but the mechanism is unknown. The condition is irreversible and should be distinguished from reversible conditions in which the obstructed bowel is dilated but thin-walled. Bowel wall hypertrophy may therefore reflect chronic damage to the myenteric plexus.

Examination of the bowel of these two patients by usual histological techniques showed only minimal and debatable abnormalities in the region of the myenteric plexus, until special techniques were applied. The histological changes were similar in both patients, though different segments of bowel were involved. These were hypertrophy of smooth muscle, especially the inner circular coat, loss of neurones and axons with a resultant increase in Schwann cells, and abnormalities in the residual neurones. Enlargement of the perikaryon, which sometimes assumed a bizarre shape, could be seen in Case 1 and in the proximal area of Case 2. Many cells had swollen irregular processes. In the distal colon of Case 2 neurones were very scanty, and those that remained were shrunken and dark. A few macrophages could be seen in some ganglia in both cases, and in Case 2 two or three ganglia were full of chronic inflammatory cells.

A few conditions have been described in which there is damage to the myenteric plexus in man, but they differ in one respect or other from the lesion described here. In Chagas's disease, in which neuronal damage is due to *Trypanosoma cruzi*, the histological picture is of loss of neurones, but abnormal forms are rarely seen except for axonal swelling in relation to the residual inflammatory reaction (Smith, 1967b). Hirschsprung's disease also shows absence of ganglion cells in the contracted segment, and though abnormal perikarya are seen, particularly at the junction zone, they usually consist of strongly argyrophil masses without any processes. Moreover, there is a striking abnormality of the plexus layout, which was not seen in our two cases. Plexus damage has been described in a single

case of purgative addiction (Smith, 1968). The histological picture was of simple disappearance of neurones with an increase in Schwann cells. The neurones that remained were abnormal but different from those in our two cases, and there was no inflammatory reaction. In achalasia of the cardia myenteric neurones are lost, and chronic inflammation of an oesophageal ganglion has been reported (Hurst and Rake, 1929-30). This may be a similar lesion to those in our two cases, but examination of early cases by our technique has not been carried out.

AETIOLOGY

Although the significant pathological changes in these two cases are similar, it cannot necessarily be implied that the aetiology is identical. Histological differentiation from conditions known to damage the myenteric plexus in man has already been described. Infections caused by bacteria or *Toxoplasma gondii* and damage due to some neurotoxic drugs (Smith, 1967c; Smith, unpublished observations) also produce a different histological picture. Another toxic or metabolic agent could be responsible but would be unlikely to affect a localized segment of the small intestine as in Case 1. A viral cause is more likely on histological grounds, but the aetiology must remain speculative until more cases are studied and the virus is isolated.

SMALL-INTESTINAL SYNDROME

The true incidence of this syndrome cannot be assessed, as causes of intestinal obstruction without evidence of mechanical blockage are legion (Cantor, 1967), and cases may have been reported under a variety of names. For instance, the syndrome of spastic ileus (Zimmerman, 1930) may include various obscure cases of intestinal obstruction, which may occasionally show hypertrophy of the bowel wall (Steigmann and Singer, 1935). The syndrome of pseudo-obstruction of the intestine (Dudley *et al.*, 1957; Stephens, 1966) includes cases in which the clinical picture suggests mechanical obstruction, though no lesion can be found, but they differ from Case 1 in that steatorrhoea and bowel wall hypertrophy are not reported. However, the absence of these features may be due to investigation earlier in the course of the disease, as they were not confirmed in Case 1 until the third laparotomy 17 years after the onset of symptoms. Certain cases of duodenal ileus and megaduodenum may be of a similar nature (Fischer, 1960; Erskine, 1963).

Two reports of intestinal pseudo-obstruction and steatorrhoea have been published. Naish *et al.* (1960) described recurrent intestinal obstruction in a man aged 36 who later developed a malabsorption syndrome. Operation revealed a disturbance of peristalsis but no organic obstruction. Full-thickness jejunal biopsy indicated that there was gross hypertrophy of the inner muscle coat, but the mucosa, lamina propria, muscularis mucosae, ganglion cells, and nerve fibres of the myenteric plexus were reported as normal. McClelland *et al.* (1962) described the case of a woman aged 55 who had suffered from diarrhoea for four years and had then developed intestinal obstruction and steatorrhoea, which resulted in her death seven months later. The duodenum and jejunum were dilated and thickened, and microscopy showed hypertrophy of the muscle coat with a myenteric plexus that was reported as normal. Naish *et al.* (1960) also searched the literature for further examples, and collected eight cases in which some of the features of the syndrome occurred, but the complete picture was present in only one subject. These cases are probably similar to Case 1.

The apparently normal myenteric plexuses do not exclude the diagnosis, since conventional sections of bowel wall may not

reveal the lesion. The presence of disordered peristalsis and muscular hypertrophy may both be explained by a lesion of the myenteric plexus. The clinical signs of distension, visible peristalsis, and hyperactive bowel sounds may also be attributed to this cause. The association of steatorrhoea and malnutrition should be attributed to the stagnant loop syndrome and the direct effects of mechanical obstruction on appetite and delivery of chyme to the small-intestinal absorptive surface. All these factors were present in our case.

Jejunal diverticulosis may sometimes present as chronic recurrent intestinal pseudo-obstruction, and in these cases hypertrophy of the diverticulum-bearing area of the bowel has been noted (Phillips, 1952-3). However, true mechanical obstruction of the bowel in association with jejunal diverticulosis appears to be very rare (Pinto, 1968). It has been suggested that a localized neuromuscular disturbance results in disordered peristalsis and the formation of pulsion diverticula (Edwards, 1936), and the presence of muscular hypertrophy adds weight to this theory. The stagnant loop effect in such cases may therefore be due to faulty peristalsis as well as stasis in the diverticula, and cases such as ours may differ only in their resistance to the formation of diverticula. However, many subjects with jejunal diverticula do not show hypertrophy of the bowel wall and do not present with intestinal obstruction.

The syndrome must be differentiated from primary malabsorption states complicated by pseudo-obstruction by the use of intestinal biopsy, but this may prove impossible to perform if stasis is severe. The mucosa is abnormal in primary malabsorption, obstructive episodes are reversible, the bowel wall is thin, and motility is diminished. Systemic sclerosis may produce localized adynamic areas in the bowel, which cause intestinal obstruction and a malabsorption syndrome; though peripheral signs of scleroderma may be absent, Raynaud's phenomenon is often present.

Treatment.—These cases present with a history of repeated negative laparotomies for intestinal obstruction. Under such circumstances a further laparotomy may not be considered justified, but experience with our patient suggests that once the diseased segment has been excised relief of pain and malnutrition may follow. It is important that the surgeon ascertains that the dilated hypertrophied segment is the site of disease and that it has not developed in response to a more distal obstruction. If there is an adequate amount of normal bowel remaining distally the diseased area should always be totally excised, as normal ileum can take over most of the functions of the jejunum (Dowling, 1967). A major clinical problem is the identification of the extent of the lesion. Radiography overestimates the area involved. Appearances at operation are unreliable, as the distal end of our resection specimen contained microscopic lesions, though it appeared macroscopically normal. Furthermore, frozen section biopsies may fail to show the lesion.

COLONIC SYNDROME

This condition does not appear to have been delineated before and has probably been grouped with other varieties of idiopathic megacolon (Todd, 1968). Hence the true incidence is impossible to assess. The syndrome cannot be outlined on the basis of one case, but the important points appear to be the late onset (at age 10), which suggests an acquired lesion, and the progressive course with failure to relieve the constipation by successive operations. Motility studies may be useful in showing a failure of propulsion in the colon with normal resting activity of the anal sphincter but changes in the inhibitory sphincteric reflexes on electromyography.

The first problem in children is to differentiate the psychogenic megacolon which follows refusal to defaecate. This condition is less refractory to treatment, and there is no

motility disturbance in the colon and minimal changes on electromyography.

Other acquired lesions of the colon should be considered in older patients. Chagas's disease is differentiated by its geographical distribution and characteristic histology. Pseudo-obstruction, the so-called colonic ileus, presents a much more acute picture and is reversible (Stephens, 1966; Bryk and Soong, 1967). Megacolon syndrome may also be diagnosed in older patients who are in mental institutions or who suffer from diabetes mellitus, Parkinson's disease, or traumatic paraplegia (Berenyi and Schwarz, 1967). In these patients disturbance of the autonomic nervous system is suspected, but conventional histological studies are normal. Differentiation from Hirschsprung's disease is helped by the age of onset (though cases presenting in adult life have been recorded), the extent of the lesion, and histological and radiological differences. Patients in whom there is a long segment of agangliosis do not survive the first few years of life without surgical treatment.

Treatment.—Among the many adult patients who complain of chronic constipation there will be a few who have adult megacolon, and in some of these the myenteric plexus may be damaged. The role of surgery in their management is still controversial and often disappointing. Resection may appear drastic but is the treatment of choice, since local procedures on the sphincters may not be of permanent benefit, and bypass operations may not prove adequate owing to a failure of progressive movements in the intestine. The extent of the lesion is difficult to assess and cannot always be determined by biopsy, as in Hirschsprung's disease, and so the initial colostomy may fail to function properly if it is affected by the disease, and a further more proximal stoma may be required.

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Medical Memoranda

Cervical Lymphadenopathy Caused by *Aspergillus terreus*

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The known pathogenicity of *Aspergillus terreus* to man has hitherto been limited to superficial infections of the skin, nails, and external ear (Austwick, 1965). That author noted its ability to cause mycotic abortion in cattle, but systemic infections in man are not known. In the following case *A. terreus* was repeatedly isolated in pure culture from enlarged cervical lymph nodes. Fungal elements were also seen in histological sections of the nodes. Precipitating antibodies to the fungus were found in the patient's serum.

CASE REPORT

A 40-year-old sailor on a Nile steamer was admitted to Khartoum Civil Hospital at the beginning of September 1967 complaining of headache for one year, epileptic fits and left-sided weakness for six months, and swelling of the neck and left hand for two months. He had always been well except for an attack of pyelonephritis, which was treated in 1963. He began to complain of repeated attacks of headache a year before admission to hospital. These were not accompanied by vomiting or visual disturbances. Six months later he suddenly had a grand mal epileptic fit with loss of consciousness, after which he developed left-sided hemiplegia with left facial palsy. A few weeks later he began to have right-sided Jacksonian fits, mainly of the leg.

During the last two months swellings developed, first on the right side of the neck and then on both sides. These swellings gradually

increased in size and were accompanied by swelling of the left arm and hand.

On examination he was found to be mentally dull. Large, discrete, hard, and painless glandular swellings were noticed on both sides of the neck. There were no sinuses. Pitting oedema was present in the left arm. Heart and chest were normal. The liver was palpable about two fingerbreadths below the costal margin, and firm but not tender. The spleen was not enlarged. The central nervous system showed gross papilloedema, left-sided facial palsy, and left-sided hemiplegia, but there were no sensory disturbances.

Investigations.—The urine was normal except for phosphates, but there was no sugar. No parasites were seen in the stools. Sputa were examined seven times; the Ziehl-Neelsen stain showed no acid-alcohol-fast bacilli and culture remained sterile. Blood culture and Widal and Kahn tests were negative. Haemoglobin, packed cell volumes, mean corpuscular haemoglobin concentration, blood urea, serum bilirubin, and thymol turbidity were normal. Erythrocyte sedimentation rate was raised, and in the plasma the albumin-to-globulin ratio was reversed. There was marked eosinophilia. These results are shown in the Table.

Nothing abnormal was found on x-ray examination of the skull, sella turcica, and cervical spine, but chest films showed a large rounded opacity at the upper half of the anterior mediastinum which might have been glands.

Phenobarbitone and later phenytoin helped to control his fits. While in hospital he had fever for three weeks, but investigations for malaria, typhoid, brucellosis, and leishmaniasis were negative.

The patient suddenly discharged himself, but was traced to his home two weeks later. He looked anaemic and had a dry cough. He was started on potassium iodide, but his condition continued to deteriorate, and he died in March 1968. Necropsy was not carried out.