on the ninth day after a one-stage prostatectomy (retropubic), with a blood urea of 300 mg. I am therefore not a convert to a universal one-stage operation although I practise it whenever it seems safe to do so.

In an uncomplicated case the convalescence after a retropubic prostatectomy (Millin, 1945), or a Harris operation with closure, or a Wilson Hey prostatectomy can be as uneventful as that after an interval appendicectomy, and the mortality should not exceed 5%. When there is chronic retention of urine or much renal damage or gross infection preliminary drainage will give the patient a better chance of survival. Drainage by the small suprapubic catheter has proved a safe and satisfactory method, and it does not interfere with the full exposure required for any of the modern methods of prostatectomy.

If endoscopic resection is to be done the presence of the suprapubic catheter makes the operation much simpler, as continuous lavage can be employed to keep the field clear of blood. Where intercurrent cardiac, pulmonary, or other disease makes a long period of drainage necessary it gives the greatest chance of getting the patient fit for operation. In cases of bladder paralysis it allows for adequate drainage during the critical period and does nothing to prevent the return of voluntary micturition or the establishment of automatic micturition. Its main advantages lie in the high insertion of the catheter, the oblique track, and the absence of leakage.

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OGILVIE'S SYNDROME OF FALSE COLONIC OBSTRUCTION

A CASE WITH POST-MORTEM FINDINGS

BY

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A syndrome of large-bowel colic thought to be due to sympathetic deprivation was recently described by Sir Heneage Ogilvie (1948), with a report of two cases, including laparotomy findings but without the possibility of full confirmation of the suggested anatomical diagnosis by post-mortem examination. The case now reported had been operated on before the first description of Ogilvie's syndrome, and its striking similarity to the original two cases (particularly to Case 2) was immediately apparent. When the disease had run its inevitable and very short course a full and careful post-mortem examination was performed. This third case may therefore be considered worth recording.

Case Report

The patient, a tall lean fitter aged 56, had long suffered from constipation, but had otherwise been in good health and free from pain until three months before his admission to hospital. Since that time he had noticed that his constipation was becoming more and more stubborn, and he suffered repeated attacks of colicky lower abdominal pain without diarrhoea or vomiting. These symptoms became increasingly severe and were attended by a progressive loss of weight, which he could ill afford, until, after four days' absolute constipation and a day's vomiting, on Sept. 8, 1948, he was sent into hospital as a case of chronic large-bowel obstruction due to carcinoma, becoming acute.

Examination on admission showed a very thin, ill-looking man with an unhealthy muddy complexion, in obvious spasmodic pain. The abdomen was not at all distended generally, and the wall relaxed normally. There was a tender fullness corresponding to a distended caecum and transverse colon, but no lumps could be made out through the abdominal or rectal walls. Peristaltic sounds were exaggerated. The only other significant positive finding was a small area of consolidation at the base of the left lung. The speech was slow, slurry, perhaps slightly dysarthric, but otherwise the nervous system seemed normal. This examination made it clear that any obstruction was not complete, and an enema produced a constipated result and flatus, with relief of pain. On the following days the symptoms recurred, and, although distension was never present and flatus was passed spontaneously, defaecation could be induced only by enemata.

Plain radiographs of the abdomen and chest showed some increase in normal colonic gas shadows not amounting to a picture of true obstruction, an area of collapse at the base of the left lung, and an enlarged lower mediastinal shadow, interpreted as being due to a primary or secondary neoplasm obstructing the left lower bronchus, with secondary mediastinal glands. After some days in hospital the patient complained of dysphagia, which he said had been present for a few weeks, but very mildly, and was now becoming more noticeable. A barium swallow and *x*-ray screening showed some hold-up in the lower thoracic oesophagus, again thought to be due to a mediastinal new growth.

Follow-up of this barium seemed to indicate a complete hold-up in the pelvic colon, with gross colonic distension proximally. However, on further screening the radiologist reported that he did not think there was an organic obstruction at this level, and suggested a barium enema. Meanwhile the general condition and the morale of the patient were deteriorating, the severe pains continued, the bowels remained immovable, and there were repeated requests for any operation which offered a chance of relieving his symptoms. It was felt that even with a negative barium enema laparotomy would eventually be forced by these facts, although, in view of the chest condition, at best a very temporary palliation was all that could be hoped for.

Laparotomy was performed on Sept. 21 through a midline incision. Below the level of the gastro-hepatic omentum the whole abdomen was normal. The colon showed neither distension nor spasm. The liver was studded with secondary growths, and an apparently carcinomatous mass was palpable in the pre-aortic region about the level of the coeliac axis. A small piece of liver secondary was excised for microscopy and the abdomen closed. After operation the patient became more and more cachectic, a terminal chest infection appeared, and he died on Oct. 21, one month after operation.

Post-mortem Examination.—The primary lesion was found to be an extensive carcinoma of the left lower bronchus, with secondary glands in the mediastinum invading the oesophagus, a large secondary in the right lobe of the thyroid, multiple secondaries in the liver, and some secondaries in the pre-aortic region. The apex of the left and base of the right lung showed an acute tuberculous process, which presumably had hastened the end. Microscopically the growth was highly anaplastic, and consisted of sheets and cords of spherical cells with round pyknotic nuclei and very little cytoplasm. The splanchnic nerves were very carefully dissected throughout, and were found to be entirely free from any involvement in growth in their preganglionic course. On tracing down the connexions of the semilunar ganglion distally the mat of efferent nerve fibres leaving the left side of the ganglion passed direct into a carcinomatous mass, presumably deriving from an affected pre-aortic gland. After fixation this relation was even more striking and definite. The left vagus was probably involved by growth in the thorax, although visible as a normal nerve uninvolved by carcinoma below this level; the right was uninvolved throughout its course. The brain and meninges were normal.

Discussion

The similarity of this case to those in the original description needs no emphasis. After laparotomy in my case two explanations seemed possible: (1) colonic muscle imbalance caused by interruption of sympathetic pathways (stated thus vaguely, with none of Sir Heneage's precision), or (2) that

suggested by Cushing's demonstration of acute peptic ulcer due to spasmodic and incoordinated contractions of stomach muscle in the presence of vagal overaction and sympathetic paralysis caused by lesions in the hypothalamic region (Cushing, quoted by Bockus, 1944). In view of the suggestion of dysarthria, it was thought that cerebral secondaries might be present, one of which could be causing a similar disturbance of colonic innervation and function. This explanation disposes of the difficulty about sympathetic afferents mentioned by Ogilvie, and may be thought to remain a theoretical possibility. The lesion actually demonstrated bears out Ogilvie's speculations about causation very accurately, including his forecast of its post-ganglionic position, where the fibres are non-medullated and presumably more vulnerable. Clearly the syndrome can occur only where efferent sympathetics are affected and afferents spared. This fact perhaps accounts for its rarity, while malignant disease of coeliac and pre-aortic glands is all too common.

In the absence of post-mortem findings the original description suggested that the dysfunction lay in the left colon. The symptoms irresistibly suggest a lesion of the left colon *by analogy* with those of an obstructing cancer of that organ. But if the wasting, distension, colic, and constipation are not due to organic obstruction, they may as easily originate in the mid-gut as in the hind-gut. That in fact they do is suggested by the following points.

(1) The sympathetics demonstrably affected were the splanchnics; these supply the bowel nourished by the superior mesenteric artery. (2) A subdiaphragmatic lesion of the ganglionated sympathetic chain denervating the left colon should also denervate and cause vasodilatation in the left leg (Telford, 1948). This effect is not recorded in any of the extant cases. But none of the cases had any carcinoma sufficiently distal to interfere with the "lumbar splanchnics" or the inferior mesenteric plexus—i.e., the left colon's sympathetic supply after leaving the chain. Thus a sympathetic denervation of the left colon seems anatomically very unlikely. (3) In the present case a radiograph taken 48 hours after a barium swallow showed a colon dilated up to the splenic flexure (the lower limit of the mid-gut) and normal or spastic thereafter, and gross dilatation of a small-bowel loop in the position of the terminal ileum. The plain radiograph taken on admission shows substantially the same picture.

It seems certain, therefore, that in this case the dysfunction involved the lower ileum and proximal colon, and it is probable that this will be found to be a general rule.

If it is objected that sympathetic deprivation should cause spasm, not dilatation, it can be argued thus: (1) That, since all these cases were characterized by colic, spasm obviously occurs; the bowel is normally a rhythmic organ, and abnormal diastole may follow abnormal systole. (2) That the physiology of the human bowel's response to autonomic stimuli is still obscure, and its response to deprival of its sympathetic efferents while retaining sympathetic afferents much more so. (3) That, in this case, it is an experimental fact that a gross lesion of the sympathetic efferents to the mid-gut, in the presence of at least an intact right vagus, was followed by dilatation of the distal part of the mid-gut. (4) That more accurate study of further cases is needed before any final conclusion is possible.

Summary

A third case of Ogilvie's false large-bowel-obstruction syndrome is presented in which the primary lesion was a carcinoma of bronchus and there was post-mortem proof of carcinomatous infiltration of sympathetic post-ganglionic fibres below the semilunar ganglion. An alternative theory of causation of the syndrome is postulated, but it was proved incorrect in this case. Evidence suggests that the lesion affects the mid-gut rather than the hind-gut.

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CHRONIC ILEUS CAUSED BY MALIGNANT INVASION OF THE POSTERIOR ABDOMINAL WALL

BY

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Sir Heneage Ogilvie (1948) has described, under the title "Large-intestine Colic due to Sympathetic Deprivation: A New Clinical Syndrome," two cases in which invasion of the crura of the diaphragm, coeliac axis, and semilunar ganglia by malignant growth produced a functional paralysis of the bowel which mimicked obstruction. The following case history reports invasion of the same area by what was probably a carcinoma of the pancreas, producing symptoms which suggested obstruction of the small intestine.

Case Report

The patient, a man aged 56, had suffered from mild dyspepsia all his life. His appendix was removed in 1936. A barium meal in 1943 had shown a scarred duodenal cap. Apart from occasional discomfort after food and a tendency to vomit if he was mentally disturbed, he had been healthy until two months before he visited me. He had then begun to suffer from attacks of colicky pain centred around the umbilicus, with tenderness in the right iliac fossa. The attacks came every few days, lasted a few hours, and left a sensation of abdominal distension. These attacks had become more frequent during the past fortnight and he had vomited every other day. A barium meal had shown a normal stomach and duodenum, and a barium enema a normal colon. He had lost half a stone (3.18 kg.) in weight. There had been no constipation except during the past week. His own doctor had thought he could feel a vague mass to the right of the umbilicus. While the history was being taken borborygmi were audible.

Examination showed a right paramedian appendicectomy scar, tender in the centre, and slight abdominal distension; borborygmi were well heard without a stethoscope, but there was no visible peristalsis or other abnormality. The remainder of the physical examination was negative. A provisional diagnosis of subacute intestinal obstruction due to adhesion of a segment of terminal ileum to the back of the paramedian scar was made, and operation was advised.

The patient was admitted to a nursing-home and a small barium meal requested with the object of locating the point of obstruction in the small intestine. Dr. T. V. Crichlow reported on the screening examinations and films as follows: "At three hours there are some coils of dilated small gut in the left hypochondrium and left iliac fossa. The mucosa is swollen and the calibre of the gut enlarged. In the right iliac fossa there is one loop of gut which appears to be contracted. At five hours there is still some dilatation of the loop seen at three hours, but there is no adherent portion to be seen. At eight hours this loop is completely empty, but the terminal ileum in the region of the ileo-caecal valve and also in the region of the hepatic flexure shows a moderate degree of enlargement. There is a fair amount of delay in the passage of the barium through these loops. At twelve hours the barium is completely in the colon. *Conclusions.*—There is definite