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.

My thanks are due to Mr. H. O. Blauvelt for permission to publish this case, to Dr. Doel for the radiological opinions, and to Dr. Prendiville for the post-mortem and pathological findings.

REFERENCES

Bockus, H. L. (1944). *Gastro-enterology*, vol. 1, p. 334. Saunders, Philadelphia.
Ogilvie, H. (1948). *British Medical Journal*, **2**, 671.
Telford (1948). Quoted in Harold Edwards's *Recent Advances in Surgery*, p. 330. Churchill, London.

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

evidence of enlargement of some of the coils of the small gut, probably involving only the ileum. The appearances are consistent with partial obstruction such as could be produced by adhesions. No definite filling defect was seen in the lumen of the small gut."

Operation (anaesthetist, Dr. O. Carden Sibley; gas, oxygen, ether).—At laparotomy the small intestine was seen to be moderately distended but no obstruction was found. Hard glands were palpable in the upper part of the mesentery of the small intestine and a growth was found in the body of the pancreas, spanning the aorta, fixed to the posterior abdominal wall and quite irremovable. The large intestine was of normal size. A gland was taken for section and the abdomen was closed.

The patient became progressively distended after the operation despite gastric suction and intravenous glucose and saline, partly burst his wound, and died on the fourth post-operative day. No necropsy was obtained, but the gland removed at operation showed replacement by masses of undifferentiated carcinoma.

Discussion

A somewhat cursory search of the literature shortly after the patient had died, and discussion with colleagues, failed to throw any light on the matter. Ogilvie, who likewise failed to find any reference to the subject in the literature, appears to be the first to publish a description of this unusual syndrome. The manner of its production is of great but academic interest, since the symptoms are produced only after inextricable invasion by carcinoma of a relatively inaccessible region of the body. Ogilvie considers three methods by which the symptoms might be produced: (1) by parasympathetic stimulation from invasion by the tumour; (2) by secretion of a cholinergic substance by the tumour cells; or (3) by sympathetic paresis from invasion by the tumour. He believes, despite some contrary points, that the last is the probable explanation.

A fourth explanation might be considered in my case—that interference with the blood supply by pressure of the tumour on the superior mesenteric vessels might have evoked the symptoms. Mesenteric thrombosis will cause colic, borborygmi, and distension, though the picture is more acute. Against this theory is the fact that my patient's intestine showed no cyanosis. In Ogilvie's first case there was evidence that the pelvic colon was greatly affected, though the tumour cannot have caused interference with the inferior mesenteric artery. It seems, therefore, that the vascular hypothesis must fall down.

A carefully detailed post-mortem examination of a similar case might clear up the mystery, and it is to be hoped that it will be forthcoming.

REFERENCE

Ogilvie, W. H. (1948). *British Medical Journal*, **2**, 671.

A six-months training course for hospital records officers is being jointly run until about the end of July by the Association of Medical Records Officers, the King Edward's Hospital Fund for London, and the Board of Governors of the Middlesex Hospital. It is hoped that similar courses will be organized later. The Ministry of Health states that the Minister welcomes the growing interest of hospitals in the proper maintenance and organization of patients' records and the decision of many of them to overhaul their records systems and appoint designated records officers. If this development is not to go astray only adequately trained or experienced officers should be appointed; and boards and committees thinking of making such appointments should defer doing so, unless there are exceptional reasons for immediate action, until there are enough really suitable candidates. Some boards and committees may be considering buying more or less elaborate equipment, such as punched card machinery, for their records departments. The Minister appreciates the value of such equipment where the volume of work makes it an economic proposition, but he is advised that the circumstances of a single hospital, or even in many instances of a management committee group, do not normally justify its purchase. No steps of this kind should therefore be taken by management committees without consultation with the regional hospital board.

THIOURACIL IN THYROTOXICOSIS

BY

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In this department 62 patients with thyrotoxicosis have been under treatment with thiouracil or methyl thiouracil for periods between 6 months and 4½ years. The series includes all cases of thyrotoxicosis admitted to the ward except a few so mild that phenobarbitone alone was used in treatment. The general results of treatment are here briefly recorded, and certain aspects of management in relation to auricular fibrillation, pregnancy, and granulopenia are discussed in the light of the experience gained.

Particulars of a Series of Cases

The distribution of cases by age, sex, and the presence or absence of exophthalmos is shown in Table I. Ten of the patients were males and 52 were females; their ages varied from 16 to 66 years. Exophthalmos was more common among the younger patients. No attempt was made to differentiate between primary and secondary thyrotoxicosis, and in only one case was there a definite history of an enlargement of the thyroid preceding the thyrotoxicosis by several years.

TABLE I

Age:	10–19		20–29		30–39		40–49		50–59		60–69	
Sex:	M	F	M	F	M	F	M	F	M	F	M	F
Eyes ..	E	O	E	O	E	O	E	O	E	O	E	O
No. ..	1	2	1	2	6	1	2	8	3	1	2	10	6	1	1	4	3	1	7

E = Exophthalmos present. O = No exophthalmos present.

All cases were admitted to the ward for treatment, and the effect of rest in bed and 1 gr. (65 mg.) of phenobarbitone three times a day was observed for one to three weeks. When the sleeping pulse had settled to a constant level for several nights, and the patient's symptoms showed no tendency to abate further on this treatment, the basal metabolic rate was estimated with the Benedict–Roth apparatus on two successive mornings, and treatment with thiouracil or methyl thiouracil was begun. The dose of thiouracil used at first was 0.6 g. daily, and this proved effective in all cases. Later 0.2 g. was tried, but, while this would control thyrotoxicosis of mild or moderate degree, it failed to do so in two severe cases. It was found, however, that 0.4 g. of thiouracil daily was as effective an initial dose as 0.6 g. Methyl thiouracil proved equally satisfactory in doses of 0.2 g. daily, and is now used for all cases. Each patient's progress was followed clinically and assessed every ten days, when the basal metabolic rate was estimated. After three weeks on thiouracil the patient was usually discharged on a maintenance dose of 0.2 g. of thiouracil or 0.1 g. of methyl thiouracil daily, and was seen thereafter at intervals of six weeks. White cell counts were done twice a week in hospital, and at each out-patient attendance, although it was realized that they were of no value as indicators of impending agranulocytosis. They served to remind the patients of the possible dangers of the treatment, which had been explained to them in the ward, and revealed several cases of granulopenia.

Most cases were adequately controlled by 0.1 g. of methyl thiouracil daily or less; some required 0.15 g. The dose was decided on a clinical estimate of the patients' condition when they reported to the follow-up clinic. No single observation was found to be completely trustworthy, but a reasonably accurate idea of the state of the patients was obtained by asking them how they felt, how much