their branches. Thrombosis of the internal carotid was found at necropsy in two of his four cases, but the clinical picture presented by his cases resembles that of periarteritis nodosa and is so different from those now under consideration that it is unlikely that the type of arteritis he described could be held responsible.

I have been unable to find in the literature a case similar to Case 3. Here there is no doubt that the thrombosis followed the rupture of a congenital aneurysm. It is possible that an aneurysm may be responsible in those cases with an apoplectic onset. The need for examination of the cerebrospinal fluid in every case is therefore clear.

From this summary it is obvious that the underlying cause is still obscure and further studies are necessary.

Treatment

Treatment for thrombosis of the internal carotid artery has hitherto been directed to the alleviation of spasm in the manner advocated by Leriche—by periarterial stripping, by cervical sympathectomy, or by excision of the thrombosed segment.

A study of the literature has shown no striking improvement after any of these methods. The course of the more chronic type of case indicates that recanalization of the thrombus may take place, and therefore excision of a segment of the artery appears to be unwarranted. The value of anticoagulants has not been estimated in this condition, but when early diagnosis can be made it would seem to be the most rational form of therapy.

I wish to acknowledge my thanks to Dr. F. Pygott for the radiographs, to Mr. H. Hashemian and Mr. Eric Turner for the arteriograms, and to Dr. R. J. Porter for his help and advice.

REFERENCES

Andrell, P. O. (1943). *Acta med. scand.*, **114**, 336.
Antoni, N. (1941). Ibid., **108**, 502.
Brissaud, E. (1898). *Rev. neurol.*, **6**, 579.
Chao, W. H., Kwan, S. T., Lyman, R. S., and Loucks, H. H. (1938). *Arch. Surg., Chicago*, **37**, 100.
Davis, L., and Perret, G. (1947). *Brit. J. Surg.*, **34**, 307.
Doerfler, J. (1935). *Arch. Psychiat. Nervenkr.*, **103**, 180.
Galdston, M., Govons, S., Wortis, S. B., Steele, J. M., and Taylor, H. K. (1941). *Arch. intern. Med.*, **67**, 1162.
Gilmour, J. R. (1941). *J. Path. Bact.*, **53**, 263.
Govons, S. R., and Grant, F. C. (1946). *Arch. Neurol. Psychiat., Chicago*, **55**, 600.
Hultquist, G. T. (1942). *Uber Thrombose und Embolie der Arteria Carotis*. Stockholm.
Hunt, J. R. (1914). *Amer. J. med. Sci.*, **147**, 704.
Löhr, W. (1936). *Zbl. Chir.*, **63**, 2593.
Moniz, E., Lima, A., and de Lacerda, R. (1937). *Pr. méd.*, **45**, 977.
Siegert, P. (1938). *v. Graefes Arch. Ophthal.*, **138**, 798.
Sorgo, W. (1939). *Zbl. Neurochir.*, **4**, 161.
Wolfe, H. R. I. (1948). *Lancet*, **2**, 567.

Dr. Darma Setiawan, former Minister of Health in the Republican Government of Indonesia and now Personal Adviser to the Republican Premier of Indonesia, is staying in England until the middle of December by invitation of the British Council in order to study public health and hygiene and the National Health Service. He is a member of the medical profession and has specialized in gynaecology. He has recently visited the Ministry of Health for discussions on the National Health Service, urban water supplies, purification of river water, the care of children, and the work and organization of international health conventions. On November 21 he visited the Horton Mental Hospital, Epsom, Surrey, to meet General Sir Gordon Covell, Malarial Adviser to the Ministry of Health, and to see the laboratories of the malarial control research unit at the hospital. He has now gone to Bristol to study the health and hygiene services.

OGILVIE'S SYNDROME OF FALSE COLONIC OBSTRUCTION

IS IT A NEW CLINICAL ENTITY?

BY

J. A. MACFARLANE, F.R.C.S.Ed.
Senior Registrar, Law Hospital

AND

S. K. KAY, F.R.C.S.Ed.
Registrar, Law Hospital

Cases of large-bowel obstruction of which none of the usual causes could be demonstrated at operation have recently been reported (Ogilvie, 1948; Dunlop, 1949; Handley, 1949). Instead there was malignant-tissue infiltration of the retroperitoneal tissues in the subdiaphragmatic area. These authors have considered this to be a new clinical entity.

We have recently had three cases of large-bowel obstruction which clinically resembled those reported above but which differed from them in their operative findings.

Case 1

A man aged 59 was admitted to the medical side of this hospital on September 6, 1948, with the following history. He was perfectly well until one year before admission, when he had a coronary thrombosis. He was treated for this in another hospital for six weeks. Following this he began to complain of increasing constipation, fullness of his abdomen, occasional cramp-like pains in the epigastrium, loss of appetite, and some vomiting. These symptoms increased in severity, and for the four days before admission his bowels had not moved, his abdomen had become more distended, and he was vomiting after every meal. During the next few days, in spite of a fair amount of flatus and faecal results from soap-and-water enemas, his abdomen remained distended and he continued to complain of cramp-like epigastric pains.

The electrocardiogram showed widespread myocardial degeneration but no evidence of infarct. A radiograph of the chest revealed basal opacities due to post-pulmonary infarcts. A straight radiograph of his abdomen showed gaseous distension of the large bowel, whilst a barium enema revealed no abnormality.

The patient was seen by us in consultation on September 19. His abdomen was greatly distended, and he was complaining of colicky abdominal pain. Increased peristaltic sounds were audible on auscultation. Rectal examination was negative. Because the clinical picture seemed to be one of large-bowel obstruction, probably carcinoma, a laparotomy was advised.

This was carried out on September 19 by one of us (J. A. M.) through a right paramedian incision. The terminal ileum, ascending colon, and transverse colon were greatly distended. The descending colon appeared to be of normal size. No tumour could be felt. As this type of case had not been met with before it was felt that somehow a ring stricture had been missed. Accordingly, a transverse colostomy was performed. The colostomy was opened the next day, and though gas escaped there was no faecal result for five days. Thereafter it worked normally. The patient's convalescence was marred by the development of deep venous thrombosis of his right leg, but this was successfully treated with heparin.

As it was still thought that this patient had a carcinoma in the region of the splenic flexure a second laparotomy was performed on November 25 by Mr. Brandon, surgeon-in-charge. The abdomen was thoroughly explored, but no abnormality could be found. His colostomy was closed on January 12, 1949. Following this the patient experienced occasional colicky abdominal pains, but his bowels moved regularly.

He was discharged on February 10. When last seen on April 28 he had no complaints.

Case 2

A married woman aged 45 was admitted on February 24, 1949. She had been well until September, 1948, when meningitis ensued, for which treatment had been given in another hospital. Since then she has had attacks of dizziness and vomiting with more recently increasing constipation. During the week before admission she complained of colicky lower abdominal pains, with increasing fullness of the abdomen and associated anorexia and nausea, but no vomiting. Though her bowels had moved slightly during the past week there had been no passage of flatus.

On examination the patient was seen to be well built and well nourished. Her abdomen was distended and diffusely tender, though no areas of rigidity were present. Percussion was tympanitic, with no evidence of ascites. On auscultation enhanced peristaltic sounds were heard. The cardiovascular and respiratory systems appeared normal.

A straight radiograph of her abdomen showed gaseous distension of the proximal colon. A soap-and-water enema gave no flatus or faecal result. A diagnosis of large-bowel obstruction was made and laparotomy was performed on February 25 (J. A. M.). The abdomen was opened through a right paramedian incision. The caecum, ascending colon, and transverse colon were moderately distended. The descending colon was collapsed. The distension reached as far as the splenic flexure. This area was thoroughly examined, but no abnormality could be detected between the distended transverse colon and the collapsed descending colon. The abdomen was closed.

Post-operatively the bowels moved spontaneously on the fourth day, and the distension rapidly subsided. The patient was discharged on March 12, and when last seen as an outpatient, on May 5, she had had no return of symptoms.

Case 3

A 60-year-old man was admitted on June 4, 1949. Three days previously he began to complain of colicky cramp-like pains across his lower abdomen. These pains had increased in severity and were present on admission. There was complete constipation, and during the previous 24 hours he had been vomiting. There was nothing relevant in his past history.

On examination his abdomen was greatly distended, and some tenderness was present in the right lower quadrant. Borborygmi could be heard during a spasm of pain. Rectal examination was negative. The diagnosis of large-bowel obstruction was made and laparotomy was performed on June 4 (J. A. M.).

Exploration through a right paramedian incision showed both large and small bowel to be greatly distended. In the region of the pelvic colon there seemed to be a ring of spasm, and below it the bowel was collapsed. The abdomen was thoroughly explored, especially the subdiaphragmatic region, in view of Sir Heneage Ogilvie's article. No abnormality could be detected. The pelvic colon was again examined. The area of spasm had disappeared. Because of the pronounced distension a transverse colostomy was thought advisable.

The colostomy was opened the following day and began working shortly afterwards. The distension, however, subsided only slowly. The patient improved and gave rise to no anxiety. On the seventh post-operative day, however, he suddenly collapsed, became pulseless and cyanotic, perspired freely, and died a few hours later. The physicians called in to see him thought that he had developed either a pulmonary embolus or a coronary thrombosis. Unfortunately a necropsy was refused.

Discussion

Ogilvie, Dunlop, and Handley have individually reported cases under the heading of "Large-intestine Colic due to Sympathetic Deprivation," "Ogilvie's Syndrome of False Colonic Obstruction," and "Chronic Ileus" respectively. The only cause of the obstruction seemed to be malignant invasion of the posterior abdominal wall in the region of the coeliac plexus. We have here presented three cases of large-bowel obstruction, for which at operation no cause could be found. While at first glance our cases seem to represent a condition distinct from that described by the previous authors, we feel that they have a common progenitor. We believe that the basic factor responsible for the obstruction in their cases and ours is the entity known as spastic ileus or enterospasm.

Zimmerman (1930) defines spastic ileus as an intestinal obstruction the origin of which depends solely on a persisting contraction of the intestinal musculature. Freeman (1918) describes the appearance of the intestine as being contracted to the limit, rendered thereby white, bloodless, and so firm that often it may be picked up by one end and held horizontally without bending. The intestine above the spastic area may be normal, but should the condition last long enough the bowel will dilate as in any other form of obstruction. The spasm often persists after the abdomen is opened, although it may disappear, and is sometimes found at necropsy. This description is substantially similar to the one given by Murphy (1896) as quoted by Wangensteen (1937). The reporting of that case first established spastic ileus as an entity. The spasm can occur over several inches or may be ring-like, and may affect either the small or the large bowel.

There are two features of spastic ileus that have a bearing on our hypothesis. The first is that the spasm usually disappears under anaesthesia, but may persist. In such cases the manipulation associated with abdominal exploration will cause the spastic area to relax. Ogilvie, in his second case, notes the finding of a temporary ring of spasm in the pelvic colon, adding that it is "an unusual sight under general anaesthesia." We found a similar condition in our third case.

The second aspect of spastic ileus we should like to emphasize is that distension of the proximal bowel can and does occur. Freeman and Murphy noted it. Wangensteen states quite categorically that the bowel dilates above the spastic area with gaseous fluid accumulations. Zimmerman, too, states that if the case is seen early the abdomen may be normal; if seen late, ballooned.

The literature supports our view that spastic ileus can produce the complete picture of mechanical ileus. There is the complaint of spasmodic crampy abdominal pains, with vomiting and constipation. Whether the abdomen be scaphoid, normal, or distended depends entirely on the size and extent of the contracted segment and its duration.

The causes of spastic ileus are many, and numerous classifications have been devised. Essentially, the basic cause must be a sympathetic-parasympathetic imbalance. The nervous control of the bowel is still incompletely understood. The smooth muscle of the bowel has inherent motility. Within the bowel wall are the two intrinsic autonomic plexuses of Auerbach and Meissner. Synapsing with these are the extrinsic autonomic nerves, the vagus and pelvic splanchnics, and the sympathetic plexuses; and superimposed upon these is the controlling influence of the autonomic ganglia in the central nervous system. The precise part played by the extrinsic nerves is still a moot point. It is generally agreed that they exert a controlling and modifying influence on intestinal motility and tone, and, generally speaking, the sympathetic fibres are inhibitory, the parasympathetic ones motor, though their activity is not diametrically opposite.

While admitting, therefore, that spastic ileus represents an autonomic imbalance, the incomplete nature of our knowledge regarding the autonomic control of bowel activity precludes a comprehensive classification of the

causes of that condition. Zimmerman, however, has presented one that has the merit of locating the cause, where possible, to the three major divisions of the nerve supply to the bowel. Thus he divides the cases of spastic ileus into those due to (1) stimuli acting on the bowel wall directly, (2) stimuli reaching the bowel from distant lesions, and (3) stimuli reaching the bowel from the central nervous system. To these we would add a further group—(4) unknown. Of these four groups of cases, the second and fourth have a direct bearing on the cases we are reviewing.

The distant lesions, presumably acting through the extrinsic nerves, that may cause spastic ileus include lesions involving the coeliac plexus. Klett's (1923) case (quoted by Zimmerman, 1930) was due to carcinoma of the pancreas with retroperitoneal extension. Other reported cases have included acute pancreatitis and peptic ulcer penetrating posteriorly. In the 159 cases of spastic ileus collected by Zimmerman lesions involving the coeliac plexus were responsible for four. These lesions are very similar to those reported by Ogilvie, Dunlop, and Handley, in which malignant infiltration of the coeliac plexus was found. If it is accepted that such lesions can cause spastic ileus and that spastic ileus can produce the full clinical picture of mechanical ileus, then we feel that there are good grounds for believing that the cases reported by Ogilvie and others represent cases of spastic ileus.

Several cases of spastic ileus have been described in which no cause could be found. Zimmerman collected 39 cases. Aird (1949) also refers to this group. At operation an inexplicably dilated bowel is found, the spasm perhaps having been relieved by the anaesthesia. It is an obstruction without a cause. Our three cases, we believe, fit into this group, for they all presented the clinical picture of a mechanical ileus, whilst at operation there was distended bowel with no evidence of obstruction.

Summary

Evidence is offered suggesting that the cases of false colonic obstruction described recently in the literature are merely cases of spastic ileus.

Three cases of idiopathic spastic ileus producing a picture indistinguishable from mechanical obstruction are described.

We would like to express our thanks to Mr. Brandon for his helpful advice in treating these cases and in the writing of this paper.

REFERENCES

Aird, I. (1949). *Companion in Surgical Studies.* Livingstone, Edinburgh.
Dunlop, J. A. (1949). *British Medical Journal,* **1**, 890.
Freeman, L. (1918). *Ann. Surg.,* **68**, 196.
Handley, R. S. (1949). *British Medical Journal,* **1**, 891.
Klett (1923). *Zbl. Chir.,* **50**, 644.
Murphy, J. B. (1896). *J. Amer. med. Ass.,* **26**, 15.
Ogilvie, W. H. (1948). *British Medical Journal,* **2**, 671.
Wangensteen, O. H. (1937). *Therapeutic Problem in Bowel Obstructions.* Baillière, Tindall and Cox, London.
Zimmerman, L. M. (1930). *Surg. Gynec. Obstet.,* **50**, 721.

Mr. C. Price Thomas, thoracic surgeon to the Brompton and Westminster Hospitals, and Dr. Robert Machray, his anaesthetist, are visiting Portugal under the auspices of the British Council, and two Portuguese nurses have returned after studying the care of Mr. Price Thomas's patients at the Brompton Hospital. The nurses will supervise the nursing of his patients at the Instituto de Oncologia, in Lisbon, where Mr. Price Thomas is demonstrating operations with Dr. Machray as anaesthetist. Mr. Price Thomas is lecturing on the surgical treatment of pulmonary tuberculosis and of carcinoma, and Dr. Machray on recent advances in anaesthesia.

KARTAGENER'S SYNDROME

BY

C. T. ANDREWS, M.D., F.R.C.P.

Physician, West Cornwall Group of Hospitals

The association of complete transposition of the viscera with bronchiectasis was first noted by Siewert (1904) in the case of a 20-year-old male patient who had had a productive cough from the age of 1 month and at the age of 20 was found to have signs of cavitation at the left apex. Apart from brief case reports by Oeri (1909) and Guenther (1923), there is no further mention of the condition in the literature till Kartagener's (1933a) report of four cases, followed later by a further seven cases (Kartagener, 1933b; Kartagener and Horlacher, 1935). About the same time cases were also reported by Nüssel and Helbach (1934), Behrmann (1935), and Kautzky (1936).

Adams and Churchill (1937), surveying 23 cases of situs inversus totalis admitted to the Massachusetts General Hospital from 1886 to 1937, found that five of these had bronchiectasis. All five also had disease of the upper respiratory air passages. The incidence of bronchiectasis in this series of 23 was thus 21.7%, the incidence in all cases (232,113) admitted to the hospital during the same period being 0.306%. The most extensive survey is that of Olsen (1943), who examined the records of 85 cases of dextrocardia admitted to the Mayo Clinic during a period of 27 years and found evidence of bronchiectasis in 14 (16.5%). Ten of these had upper respiratory infection.

In some of the later cases, notably that of Richards (1944), mention is made of congenital absence of the frontal sinus, usually associated with ethmoid and maxillary infection. The case here presented showed congenital abnormality of the paranasal sinuses to an extent which has not hitherto been recorded.

Case Report

A man aged 44 was referred to the medical out-patient department on October 8, 1947, with a history of productive cough of many years' duration. He was an only child, full-term delivery, without history of cyanosis at birth. His father died about 40 years ago, cause unknown, his mother is alive at 65 and is normal. Several cousins have been traced and are also normal.

The patient gave a history of measles at 3, whooping-cough at 5, and several attacks of pneumonia of increasing severity at the ages of 4, 10, and 16. The left side was always most involved in these attacks. For 20 years he had complained of pain in the left upper chest aggravated by coughing, deep breathing, or turning in bed. In 1927 he had five months in a sanatorium after what appears to have been an attack of left-sided pleurisy. Repeated sputum examination was negative for the tubercle bacillus. No mention was made of transposition on this occasion. His general condition deteriorated after discharge and the amount of sputum increased, with occasional slight haemoptysis. Up to twelve months ago he had suffered from severe frontal headache, which had now ceased.

Examination showed him to be a man of small stature (5 ft. 4 in. = 1.62 m.) and sallow complexion, weight 7 st. 13 lb. (50.35 kg.). He was right-handed and showed moderate finger-clubbing. The right testicle hung lower than the left. Situs inversus totalis was demonstrated by finding heart sounds and cardiac dullness to the right of the sternum, palpable descending colon in the right iliac fossa, and liver dullness on the left side. Confirmation was obtained by barium meal and E.C.G. examination.

Plain radiographs of the chest revealed a condition suggestive of multiple cysts in the left upper lobe. A bronchogram showed that the lungs were transposed, that the cysts seen in