might have been due to maternal toxæmia. A case of true knot in the cord causing foetal distress during labour was described recently by Price (1947).

Incidence of Cord Complications

Cord complications are rare, but the foetal death rate due to them is very high. DeLee gives the foetal death rate in treated cases as 40–50% and in untreated cases as 80%. In this series the foetal death rate in treated cases was 40.5%. Because of this rarity it is difficult for any one person to get sufficient experience in the treatment of cord complications, and this is one reason why the foetal death rate will always remain high. Early diagnosis and adequate treatment are essential, and two necessities for treatment are an intelligent and well-trained nursing staff and a resident obstetrician.

Diagnosis and Treatment

The diagnosis of prolapsed cord may be made first by seeing the cord presenting at the vulva. More commonly the first indication is slowing of the foetal heart, the cord being found on routine vaginal examination. In a few cases the cord is nipped at the pelvic brim and cannot be felt with the examining finger.

The treatment of prolapsed cord depends on the presence or absence of foetal heart sounds and on the degree of dilatation of the cervix. In all cases except where the cord is macerated the patient is placed in the knee-chest or the high Trendelenburg position while preparations for further treatment are being made. Pressure on the cord can further be relieved by the operator inserting two fingers into the vagina and holding up the presenting part above the brim. Even when pulsation has ceased, relief of pressure on the cord sometimes restores the circulation, but the foetus cannot stand total obstruction of the cord for more than a very few minutes. The knee-chest or Trendelenburg position, though useful in an emergency, can be maintained for only about 10 to 15 minutes without great discomfort to the mother. When operative intervention is to be used the patient can be anaesthetized in the Trendelenburg position, and should be moved only when the operator is ready to begin. If the cervix is as much as three-quarters dilated, then immediate delivery is performed—by forceps in a vertex presentation or by manual delivery of a breech or transverse lie. In such circumstances there is no need to waste time trying to replace the cord. When the cervix is less than three-quarters dilated the difficulties are increased.

Reposition of the cord can be attempted, but this, easy in theory, is very difficult in practice, and the actual reposition may cause further foetal distress by pressure and kinking. If this method is tried the hand is the best repositor, but the cord is an awkward and slippery organ with a will of its own. The foetal heart must be counted at least every five minutes after reposition in case of further pressure as labour progresses. In one case it seemed to me to be a good idea to give an injection of nikethamide into the cord before reposition, and this was tried. The foetal heart improved in tone and frequency, but the cord continued to bleed at the site of injection.

Internal version to a breech presentation is not satisfactory. The idea is that the breech fits less closely into the brim and so leaves more room for the prolapsed cord. In practice this does not work very well, because the cord may become entangled round one leg or the child may sit down on it at the level of the brim.

Caesarean section is the method of choice when the cord is pulsating strongly, the cervix is less than three-quarters dilated, the operating theatre is available, and the operation

is quick. It is no good subjecting a mother to caesarean section for prolapsed cord if the infant is going to be still-born. She can have her stillborn infant without the added discomfort of an abdominal incision.

There were no maternal deaths in this series. Though the foetal results are poor, they can be improved by constant watchfulness on the part of the midwife and by quick and accurate diagnosis by the obstetrician, with minimum delay in treatment.

REFERENCES

- DeLee, J. B. (1943). *Principles and Practice of Obstetrics*, p. 654. Philadelphia.
Kerr, J. M. Munro (1937). *Operative Obstetrics*, p. 201. London.
Price, John (1947). *British Medical Journal*, 1, 928.

HODGKIN'S DISEASE CONFINED TO THE JEJUNUM

BY

G. L. ROBINSON, M.D.

Pathologist to the Seamen's Hospital Society

The subvariety of Hodgkin's disease affecting the alimentary tract, though by no means excessively rare, is better described in journals than in textbooks. A clear outline of its features, with references to date, is given by Hayden and Apfelbach (1927), who selected 26 previously reported cases for their study, adding three of their own: spleen and liver are not enlarged and nodules within them are uncommon; mesenteric but not other groups of lymph nodes are involved; lesions in the gut tend to be multiple and small, starting in the mucosa or submucosa, forming an ulcer with an enclosing "wall," and quickly penetrating muscular and serous coats; histologically, lymphocytes and endothelial cells predominate, while Sternberg-Reed cells are scanty; symptoms are intestinal and not of long duration; death often follows perforation or haemorrhage, or is cachectic.

The case here presented conforms to the above description and is very similar to that published (without necropsy) by Badia (1943) of a woman aged 29, with a six-months history, who died seven weeks after a loop of small intestine had been resected and diagnosed histologically as Hodgkin's sarcoma of the jejunum.

Case Report

A ship's chief wireless operator, aged 51, was admitted to the Dreadnought Seamen's Hospital on June 20, 1947, with a three-months history of burning epigastric pain two hours after meals, loss of 1 st. 7 lb. (9.5 kg.) in weight, loss of appetite, and weakness of the legs when climbing ships' ladders. Vomiting had not occurred and the bowels were regular. Except for pneumonia eleven months before (Long Island College Hospital), his health had been good. His mother had died at 84 (heart) and father at 79 (cancer of stomach); two brothers and a sister were alive and well. Physical examination revealed wasting, very bad teeth, and no palpable glands. Weight was 6 st. 12 lb. (43.5 kg.), blood pressure 110/70 mm. Hg., pulse 100 (and higher thereafter), temperature 96.2° F. (35.7° C.), but between 99 and 102° F. (37.2 and 38.9° C.) thereafter.

The following investigations were carried out. Radiograph of chest: chronic bronchitic changes. Barium meal: stomach and duodenum appear normal. Barium enema: there is a filling defect in the sigmoid. Urine: no chemical or microscopic abnormality. Wassermann and Kahn: negative. Alcohol-histamine meal: no abnormality. Blood count: haemoglobin 53% (Haden); R.B.C. 3.2 millions and W.B.C. 6,200 per c.mm.; colour index 0.83. Sedimentation rate (Westergren): 24 mm. at one, 49 mm. at two hours. Blood urea: 30 mg. per 100 ml. Plasma protein: 5.25% (albumin 3% / globulin 2.25%

= 1.3). Occult blood : strongly positive benzidine test, negative amidopyrine test. Sputum : four times negative for tubercle.

Carcinoma of the colon was diagnosed, and laparotomy was performed (July 8) after transfusion had raised the haemoglobin level from 47 to 61% (Haden). An adhesion between sigmoid and small intestine was freed and the resulting hole in the colon closed. An abscess cavity was found in the mesentery, to which small intestine was bound. The loop and mesentery

Photomicrograph showing the disease process in the mesentery adjacent to the surgically excised loop of jejunum ($\times 190$).

were resected, and from them a histological diagnosis of Hodgkin's disease was made (see Fig.). The patient died on the fifteenth post-operative day.

The specimen removed at operation consisted of a piece of jejunum 24 cm. long with attached mesentery up to 5.6 cm. wide, incised about midway, where the gut was thickened, haemorrhagic, and bunched by fibrosis, to show a brawny bile-stained excavation, defined by scar tissue, extending 3 cm. from the incised gut into the mesentery. A ragged gaping hole, occupying most of the free aspect of the thickened jejunum opposite this excavation, was caused by surgical dissection. There was thus evidence of a swollen, fibrosing, penetrative lesion encircling the middle 5 cm. of the loop of gut resected and rather abruptly demarcated from the remaining gut, which was of normal appearance. The lymph nodes in the attached mesentery were enlarged up to 1.3 by 0.8 by 0.7 cm. and showed uniform yellow glistening cut surfaces.

The necropsy showed general purulent peritonitis, with a number of tough fibrous adhesions in addition to the fibrin binding loops of the small gut. These adhesions were found to occur on the serosal aspect of ulcers in the upper jejunum, where roughening, slight puckering, and fibrous tags but no tubercles were seen. There was no gross abnormality of stomach or duodenum. The uppermost lesion was a shallow ulcer, 1 by 0.6 cm., situated a few centimetres beyond the duodeno-jejunal flexure, with its axis transverse to that of the gut, with its floor short of the muscularis, and with its edges sheer. This was the only ulcer without accompanying fibrous adhesions on the serosa. The next lesion, 80 cm. below the flexure, was the anastomosis representing the site of the surgically excised loop, with considerable sepsis in the adjacent mesentery. Below this, at a point 140 cm. (pre-operatively 164 cm.) from the flexure, came a group of four small ulcers (largest 0.8 cm. diameter) of punched-out appearance, with sloughing floors piercing the muscularis. An area, about 2.5 cm. in diameter, of faint white stippling could be seen infiltrating the mucosa throughout this ulcer-bearing zone when it was brightly illuminated; there were also a few slit-like erosions about 1 mm. long. Below this group, 170 (pre-operatively 194) cm. from the flexure, was a single girdle-type ulcer measuring 2 by 0.8 cm., with its floor penetrating the muscularis and with its edge raised, swollen, and mainly rounded, smooth, and tense-looking, though eroded in places. Another similar

though slightly smaller ulcer occurred 200 (pre-operatively 224) cm. below the flexure. Next below this, 230 (pre-operatively 254) cm. from the flexure, came the largest of the lesions, which consisted of an ulcer, 6.2 by 2.2 cm., encircling the gut for about two-thirds of its circumference. In contrast with the other lesions the ulcer floor was in this case hypertrophied, so that it stood up as a plateau 0.3 cm. above the mucous membrane; it was coarsely nodular, fissured, and stained slate-colour by altered blood. A smooth, tubular, and slightly undermined edge, itself ulcerated in places, surrounded it like a rail. Two more adjacent punched-out ulcers (the larger measuring 1 cm. in its longer axis, transverse to the gut) were found beyond this at a distance of 240 (pre-operatively 264) cm. below the flexure, and here the lesions ceased. A short Meckel's diverticulum was present. The colon was distended by pale sprue-like faeces. Mesenteric lymph nodes were moderately enlarged up to 1.8 by 1.5 by 1.2 cm. and showed uniform yellow cut surfaces. There was no enlargement of other groups. The spleen was firm and below normal size.

Comment

The histology of Hodgkin's disease is present only in sections of the gut lesions, the affected piece of mesentery (where the picture is most typical), and the larger of the mesenteric lymph nodes. In most sites the changes appear to be early and rather atypical. In some nodes large Hodgkin-like cells are scattered diffusely and the cells of the sinuses are proliferating without destruction of normal architecture. Fibrosis in the nodes is only beginning, and typical giant cells have to be searched for. Sections of the ulcers show necrosis of the base with replacement of all coats by granulation tissue containing a variety of chronic inflammatory cells together with small hyperchromatic giant cells, and affected by pyogenic infection to varying degrees. In one cut, at the periphery of an ulcer, the muscularis mucosae is seen breaking up beneath a mucous membrane which is still intact. In all regions there is a prominence of cells of reticulo-endothelial type, as noted by Hayden and Apfelbach (1927) and also mentioned in the report of a case of gastric Hodgkin's disease by Jungmann (1943). These cells, tending to be diffusely scattered, to show distinctly eosinophil cytoplasm and rather well-defined outlines, sometimes look at first glance like the expected infiltration of eosinophil polymorphs, which is not a noticeable feature in these sections. Intravascular plugs of granulation tissue, sometimes mixed with thrombus, are common and often contain small hyperchromatic giant cells. It seems possible that this invasiveness for blood vessels may by infarction play a part in the production of the ulcers. Vascular penetration has been photomicrographed both by Donati and Bragaglia (1942) in an intestinal case and by Bini and Parvis (1940) in a gastric case. Histological malignancy is stressed in both these cases. In the present case the short history, the small hyperchromatic giant cells, and the cellular rather than fibrotic reaction are held to indicate simply an early and acute form of Hodgkin's disease.

REFERENCES

- Badia, P. D. (1943). *Amer. J. Surg.*, **59**, 577.
 Bini, G., and Parvis, A. (1940). *Pathologica*, **32**, 89.
 Donati, A., and Bragaglia, R. (1942). *Folia clin. biol.*, **14**, 103.
 Hayden, H. C., and Apfelbach, C. W. (1927). *Arch. Path. Lab. Med.*, **4**, 743.
 Jungmann, H. (1943). *Brit. J. Radiol.*, **16**, 386.

The Third Annual Report of the Liverpool Cancer Control Organization has some interesting figures relating to cases of cancer referred for treatment during 1944. Among 2,668 cases the disease was so advanced as to render treatment impracticable in 993. Of the 1,675 cases in which treatment could be undertaken there were only 971 in which the condition was at a sufficiently early stage to warrant radical curative treatment being attempted. Of the 971 cases so treated 72 subsequently died of intercurrent disease or were lost sight of. There remained for review 899 patients, of whom 617 were alive three years after the start of treatment.