with others, believes that "destruction of the medulla is the keystone of this phenomenon." Case 3 supports this view, as C.N.S. changes were well evident, while myocarditis was minimal.

The clinical diagnosis of myocarditis in poliomyelitis leaves much to be desired, though tachycardia without obvious cause is a possible clue (Fox *et al.*, 1953). Geffer *et al.* (1947), in describing 11 fatal cases, with myocardial pathology in five out of six necropsies, stated that they showed little clinical evidence of cardiac disease. Standard works do not mention cardiac signs in their accounts of poliomyelitis. Sudden death in poliomyelitis may have an association with myocarditis, as it was found in all of 14 subjects dying suddenly in a total of 28 fatalities (Shutkin, 1951). Weinstein (1957) points out that it is difficult to distinguish the origin of cardiovascular dysfunction among the competing factors arising from possible involvement of myocardium, lungs, and medulla. Among the welter of neurological and ventilatory disturbance in our five cases it would have been hard to say that clinical evidence of myocarditis existed. In the absence of satisfactory clinical or E.C.G. evidence the diagnosis of myocarditis has usually been a posthumous histological discovery. As only the severest cases come to necropsy no generalization is possible regarding the myocardium in all cases (Spain *et al.*, 1950). It seems safe to say, however, that no evidence has appeared which gives myocarditis any clinical importance in non-life-threatening paralysis, but in the severe cases it may be an unsuspected contributor to death.

Summary

Five fatal cases of paralytic poliomyelitis are described. All showed histological evidence of myocarditis. The severity of the myocardial lesions varied greatly from case to case, but the pleomorphic nature of the inflammatory cellular infiltration is probably characteristic of poliomyelitis. The difficulties in clinical diagnosis of poliomyocarditis and assessment of its importance are discussed.

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ADULT PYLORIC OBSTRUCTION DUE TO A MUCOSAL DIAPHRAGM

REPORT ON TWO CASES

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Intrinsic diseases of the stomach can cause pyloric obstruction, and include neoplasms, ulcer, congenital and acquired hypertrophy of the pylorus, spasm, tubercle, syphilis, and foreign bodies. The commonest cause of pyloric stenosis is a duodenal ulcer, either in the active state with associated oedema or in the chronic stage with cicatrization.

Obstruction can also be due to a mucosal diaphragm, which may occur either proximal to or at the level of the pylorus, with no visible or palpable cause on external

FIG. 1.—Diagrammatic representation of a mucosal diaphragm at the pyloric ring, consisting of two layers of mucous membrane, with a central aperture.

examination of the stomach. This diaphragm consists of a double layer of mucous membrane, with a small aperture, usually central in position, there being only submucous tissue and muscularis mucosae between the layers (Fig. 1).

At the time of writing only four cases have been described in the world literature, but Rhind (1959) has since reported further cases.

We record two case histories of adult pyloric obstruction due to mucosal diaphragms and discuss the aetiology and differential diagnosis.

Case 1

A housewife aged 55 attended hospital on September 2, 1958, with an eight-months history of fullness after meals. During this time she had lost just over 2½ st. (15.9 kg.) in weight. Her appetite was variable, and sometimes she had eaten reasonably well. Before her symptoms started she had some mental worry, and she thought they might have been due to this cause. She made herself vomit at least once a day, and during the previous two months had been vomiting more often, but not large amounts. Bowels and micturition were normal. On examination she looked reasonably well, and did not appear to have lost weight

or to suggest malignant disease. Abdominal and rectal examinations were negative. She suffered from rheumatoid arthritis of the hands and feet.

Radiographic Examination.—On September 4 Professor A. S. Johnstone reported as follows: "Oesophagus was normal. Most of the barium still remained in the stomach, which contained a moderate amount of food. Peristalsis was quite active but somewhat inefficient. The pyloric

FIG. 2.—Case 1. Normal appearance of stomach and duodenum at operation.

sphincter appeared as a narrow ring through which the barium passed quite readily when lying on the right side. No organic lesion was found. The duodenum filled normally. Conclusion: there are features to suggest a neuromuscular disorder possibly reflex, and further investigation with drugs might be of considerable value." On September 23 the patient was admitted to hospital, and after a short course of intravenous and intramuscular propantheline a further barium-meal examination was reported on as follows: "On this occasion the stomach was larger and contained more fluid, and no medium passed through the pylorus. These findings after a short course of propantheline indicate the absence of spasm and would favour a fibrotic stricture. The change since the last examination indicates that there may be an active ulcer, but none was noted earlier." It was evident from this report that operative treatment was indicated.

Operation.—On September 29 the abdomen was explored through an upper right paramedian incision. The stomach was found to be fairly large and its walls were thickened. There was no external evidence of an ulcer or any other lesion at the pylorus, either on inspection or on palpation (Fig. 2). Some small soft glands were present, mainly along the greater curvature. No other abnormality was noted in the

FIG. 3.—Case 1. Stomach has been opened along the lesser curvature. Arrow indicates position of diaphragm, opened through its central aperture.

abdomen. In view of the evident obstruction a mucosal valve was considered, and a partial gastrectomy was carried out by the retrocolic method with a Hofmeister valve. The fresh unopened specimen was examined, and a finger was inserted into the pylorus. Its passage was obstructed by a soft mucosal diaphragm at the level of the pyloric ring. The enclosing pyloric musculature was easily stretched over the finger. There was a central aperture in the diaphragm 1 mm. in diameter which admitted the tip of a Halsted artery forceps. At this stage the stomach and duodenal cuff were opened along the lesser curvature, but the diaphragm was not easy to demonstrate as in the unopened specimen (Fig. 3). There was no naked-eye evidence of an active or a healed ulcer.

Pathological Report.—"The specimen showed pronounced hypertrophy of the main muscle coat and of the muscularis mucosae. The gastric mucosa was hyperplastic in appearance and rich in goblet cells. There was no ulceration and no evidence of scarring in the region of the lesion."

The patient made a good recovery, and when seen later had had no further vomiting and was gaining weight.

Case 2

A coppersmith aged 52 was seen on October 7, 1958. Since 1953 he had suffered from an intermittent ache in the epigastrium, which had become worse during the previous six months. Upper abdominal pain and a feeling of fullness came on suddenly and was not related to food. He would vomit almost immediately and this would relieve the pain, which was also eased by taking alkalis. During the previous

FIG. 4.—Case 2. A finger has been inserted in the duodenum and the mucosal diaphragm has been pushed into the stomach, which was everted over the finger. Arrow points to central aperture.

three months he had vomited two to three times a week, mainly at night. He had lost 14 lb. (6.4 kg.) in weight during the previous six months, and his appetite was poor. In 1939 an x-ray examination suggested a peptic ulcer, and in 1941 a calculus had been removed from his left kidney. On examination he looked well and there were no physical signs in the abdomen or chest. Rectal examination was negative.

Radiographic examination on September 30 was reported on as follows: "The oesophagus outlined normally. There is a constant deformity of the lesser curve at the pyloric antrum, highly suspicious of a neoplasm. In addition there appeared to be irregularity of the adjacent part of the duodenal outline, possibly due to extrinsic pressure. Conclusion: gastric neoplasm."

Operation.—On October 16 the abdomen was explored through an upper left paramedian incision. There was no direct evidence of a pyloric carcinoma or ulcer, but the pylorus appeared slightly thickened on palpation. Both lobes of the liver contained deposits on their upper surface which suggested malignant disease, but no primary growth was found in the abdomen. A partial gastrectomy was

carried out by the retrocolic method with the formation of a valve. The fresh specimen was examined and a finger inserted into the pylorus. Its onward passage was obstructed by a soft mucosal diaphragm with a central aperture which measured 4 mm. in diameter. A finger was inserted through the duodenal cuff and the diaphragm pushed into the stomach, the latter being everted over the finger (Fig. 4). There was no naked-eye evidence of an active or a healed ulcer.

Pathological Report.—"Several sections were examined, including one through the central aperture of the diaphragm (Fig. 5). The report showed an intact mucosa in the pyloric region with evidence of superficial gastritis. The muscularis was prominent and slightly hypertrophic. The mural musculature was normal except for some thickening at the pylorus. Within the mucosa and in the submucosa there were numerous follicular formations of giant and epithelioid cells with a surrounding rim of lymphocytes. Some of the follicles appeared to have arisen within foci of lymphoid tissue in the submucosa. They were most numerous in sections from the body, but a few were present at the pylorus. The giant cells were of both Langhans and foreign-body type. No anisotropic material was seen with polaroid lenses. Caseation was not seen and prolonged search showed no evidence of tubercle bacilli. The possibility that these follicles were sarcoid must be considered, especially in view of the lesions seen in the liver at operation. Tuberculosis or, less likely, foreign-body reaction should also be included in the differential diagnosis. No evidence of malignancy was seen."

Further Investigations.—X-ray examination of the chest revealed nothing abnormal. The Wassermann reaction was negative, and the Mantoux test (1 in 1,000) positive. X-ray examination of the hands and feet showed no bony changes.

The patient made a good recovery and was discharged from hospital 12 days after operation.

Discussion

A mucosal diaphragm as described in these two cases must be distinguished from an abnormal gastric mucosal prolapse into the pylorus. There are many reports of this condition, and Hermann *et al.* (1955) described it in a woman aged 39 in whom redundant gastric mucosa prolapsed intermittently through the pylorus into the duodenum. The barium-meal examination showed no abnormality.

Idiopathic hypertrophy of the pylorus in an adult is another entity which should not be confused with a mucosal diaphragm. In this condition the pylorus is obviously thickened and hypertrophied both on inspection and on palpation, as in a congenital pyloric stenosis.

The cause of adult hypertrophy in the absence of any other disease, as well as the congenital type, has never been fully explained. Kleitsch (1952) thought that the congenital type would persist into adult life unless operated upon. He reported four cases, and believed the condition probably occurred more often than was generally appreciated. Armitage and Rhind (1951) did not believe that there was any connexion between the condition in adults and the congenital type. The circular muscle of the pylorus is greatly hypertrophied and may be as much as 1.5 cm. thick, as in the case reported by Vorhaus (1946), who suggested that continued pylorospasm may lead to hypertrophy. Barrie and Anderson (1948) described a case of even concentric hypertrophy associated with massive eosinophilic infiltration, and peculiar periarterial giant-cell follicles. The patient, a young woman aged 27, also had a constant blood eosinophilia. They suggested that both the congenital and the adult "idiopathic" hypertrophy may be examples of a true organic intestinal allergic reaction.

One other pyloric condition which should be mentioned in discussing the differential diagnosis of a mucosal diaphragm is a chronic hypertrophic antral gastritis. Arendt (1945) fully described this disease, where there was a visible hypertrophy of the pyloric muscle.

Among the four reported cases of a definite diaphragm, Touroff and Sussman (1940) found a congenital, prepyloric, membranous obstruction in a premature child, who survived after multiple incisions of the septum and a pyloroplasty. They reviewed the literature on congenital obstructions of the stomach, which, except for hypertrophic stenosis, appear to be extremely rare. They made the interesting remark that in hypertrophic stenosis a redundant fold of mucosa is commonly encountered within the pyloric lumen.

FIG. 5.—Case 2. Photomicrograph of mucosal diaphragm ($\times 11$) through central aperture, which is on the right. Gastric mucosa is on left and duodenum on right. Arrows indicate position of sarcoid lesions.

Rota (1953) described a diaphragm in a woman aged 73, the case being very similar to our cases. No ulcer was found in the stomach or on the diaphragm, and there was a complete absence of scarring. A radiograph taken prior to operation had shown a large stomach with no obvious cause of obstruction. Nine years previously a pyloric ulcer with obstruction had been demonstrated on x-ray examination. In discussing the aetiology, he favoured a congenital origin, despite the late onset of symptoms and the advanced age of the patient.

In the third case, Sames (1949) described a diaphragm 1.5 cm. proximal to the pyloric ring in a woman aged 40. She was operated upon for a gastric ulcer which was found to be present on the lesser curvature. No evidence of an active or healed ulcer was found on or near the diaphragm. The enclosing pyloric musculature was hypertrophied proximal to the diaphragm, but was normal between it and the pyloric ring. In reviewing the x-ray film taken two years before, he was able to recognize the diaphragm radiologically. Sames favoured a congenital origin, but he also stated that "it might be argued that following sustained muscular contraction of the antrum, a permanent deformity of the mucosa may result, and give rise to an obstructing diaphragm."

Passalacqua and Romero (1955) reported a case of partial pyloric obstruction due to a mucosal diaphragm of congenital origin.

Rhind (1959) describes the condition and discusses its aetiology. He found that the narrowing was at the pyloric ring, except in one case where it was 2 cm. proximal to it; the apertures varied between 2 and 4 mm. in diameter, and were either central in position or nearer the lesser curve. He believes that the condition is the end-result of scarring of a superficial annular pyloric ulcer. In support of this he has seen small superficial ulcers at the edge of the aperture, and a superficial linear ulcer without a diaphragm.

Sarcoidosis can involve any tissue in the body, but has a predilection for lymphoid tissue. Intestinal lesions are rare (Macfarlane, 1955), and gastric sarcoid appears to be even rarer. Gore and McCarthy (1944) described a case in a soldier aged 26 in whom a diffuse indurated lesion of the pylorus, simulating a malignant mass, was resected. No significant mucosal changes were reported, but a large shallow ulcer was present on the greater curvature.

Desmond and Swynnerton (1957), in reporting 25 cases of adult hypertrophy, found mucosal changes in five of these. There was mucosal stenosis in three and a mucosal prolapse into the duodenum in the other two, but no diaphragm was described. They felt that the abnormal mucosal prolapse was due to the hypertrophied pyloric muscle preventing normal reduction, and that the mucosal stenosis was due to some degenerative change secondary to the prolapse. Alternatively, they suggested that the stenosis could be inflammatory in origin, possibly from an acute pyloric or duodenal ulcer. Sames (1949) drew attention to a paper by Bockus, who reported mucosal prolapse into the pyloric canal associated with adult hypertrophy. It is interesting to see that there are mucosal changes in adult hypertrophy and also in congenital pyloric stenosis.

It would seem that a mucosal diaphragm may be congenital, a view which has been taken by previous

writers, and that its onset in later life may be due to some added factor. The histological appearances, with no evidence of previous ulceration or scarring, seem to support this possibility. In this connexion the presence of a sarcoidosis in the second case is of interest, though we do not suggest anything more definite.

It has been suggested that the condition is secondary to a superficial linear ulcer at the pylorus, but we do not subscribe to this view, as there is no evidence of superficial scarring or of a deeper replacement of the muscularis mucosae and other tissues by fibrous tissue. These findings would be expected if there had been an ulcer which had healed, and should be present if there were changes sufficient to produce a permanent deformity.

A case could be made out for the hypothesis that both these cases are variations of an antral gastritis, perhaps associated with or due to pylorospasm, and complicated by some mucosal oedema and prolapse which might appear as a fairly well localized "diaphragm."

It may be that the condition is one that develops in adult life from some obscure cause, possibly allergic, or possibly from the local absorption of some irritating foreign body, medicinal or otherwise.

Summary

Two cases of adult pyloric obstruction due to a soft mucosal diaphragm are described, there being no visible or palpable cause found on external examination of the stomach and duodenum.

In order to recognize this condition it is essential to insert a finger into the pylorus, when its onward passage will be obstructed if a diaphragm is present. It is important, however, to stretch the enclosing pyloric muscular ring, which is usually contracted down over the examining finger. If the stomach is opened along one of the curvatures, even before fixation, the appearance and form of the diaphragm are difficult to demonstrate, and we found that this happened in our first case. It is really only possible to demonstrate a mucosal diaphragm in the fresh unopened gastric specimen.

The condition is of obscure origin, and no definite suggestion concerning its pathology can be made on these cases, or on the small number of cases reported in the literature.

It is a rare cause of pyloric stenosis, which should be considered in cases where there is no evidence of ulcer or neoplasm.

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