In 17 missed cases, group C, the patients suffered angina pectoris virtually confined to effort.

The diagnosis depends, above all, on a careful case history. Fever, perhaps the most useful, is the most neglected physical sign; by contrast, excessive reliance is placed on the presence or absence of pericardial friction.

There is evidence that disability was more prolonged in (even mild) missed cases than in those diagnosed and treated early.

The significance of and reasons for delayed diagnosis, and the cardiographic changes, are discussed.

It is a pleasure to record my thanks to the consultant physicians at the Royal Sussex County Hospital, who allowed me to use their case notes, and particularly to Dr. R. Kemball Price, who advised me and encouraged me to use the records in the cardiac department; also to Sir Lionel Whitby, Regius Professor of Physic, for permission to publish this abstract from a thesis submitted for the degree of M.D. of the University of Cambridge.

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The Hyderabad health museum, one of the best of its kind in the Eastern Hemisphere, was recently rehoused in a new building in Hyderabad City, where it attracts many visitors. The high illiteracy rate in India makes the visual method of presentation and instruction all the more important in public health education. The museum includes models, posters, photographs, charts, and other visual aids. Films and lantern slides are shown regularly, and a van equipped with projectors and printed literature makes tours throughout the State of Hyderabad. The resources of the museum are fully described and catalogued in the second edition of an attractive 48-page illustrated handbook which has just been published by the Bureau of Health Education, Public Health Department, Hyderabad.

FAMILIAL HYPERTROPHIC PYLORIC STENOSIS

BY

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Congenital pyloric stenosis in the adult, first reported by Maier in 1885, is not so very rare, and most surgeons will encounter a case from time to time. The congenital condition occurring in infants is relatively common, and the literature contains a number of reports of cases occurring in two or more siblings. Gailey (1948) reports four proved cases in one family and mentions another family with four affected members reported by Finkelshtein and quoted by Sauer (1924). There are occasional reports of cases occurring in successive generations, and Schrieber, quoted by Laubscher and Smith (1947), described a patient whose father and grandfather both suffered from pyloric stenosis in adult life. In the family reported here hypertrophic pyloric stenosis was discovered at operation in a man aged 39; he subsequently revealed that two of his four sons had had Ramstedt's operation performed upon them in infancy, and he had a nephew treated medically for the condition in infancy.

In addition his eldest brother had died in infancy from persistent vomiting. The accompanying genealogical table shows the incidence of pyloric stenosis in this family.

Case 1

A man aged 39 first attended hospital on January 21, 1952, complaining of recurrent attacks of epigastric pain and heaviness after meals for 10 years. He had been investigated in 1944, when x-ray examination was said to show a duodenal ulcer and medical treatment was advised. Records of that period during the war are unfortunately missing. He had had no other serious illnesses, no previous operations, and gave no history of gastric trouble in childhood. His pain usually came on soon after meals, but was sometimes delayed for two and a half to three hours. It was situated in the epigastrium, did not radiate, and lasted 20 minutes to one hour. Often it would "make him lie down exhausted." Attacks would occur over a period of a few weeks, once or twice a year, but without seasonal incidence. Heaviness after meals was usual, and he was always a small eater. He often suffered from acid regurgitation, but vomited only occasionally. When he did vomit his pain and heaviness were always relieved. There had been no haematemesis or melaena, and weight loss had amounted to 7 lb. (3.2 kg.). His last attack had been more severe than any before and he had been off work for two weeks. He had lost an average of one to two weeks' work a year during previous attacks.

On examination he appeared to be a stable type of patient, rather thin but in fair general condition. In the abdomen there was only tenderness in the epigastrium; no tumour was palpable and succussion splash was not obtained. Barium-meal examination showed a large stomach with good tone, but it was empty in four hours. The duodenal cap was tender and irritable, and there was a suspicion of an ulcer crater in the right fornix. At this time the pyloric condition was not considered, and the x-ray appearance of the pylorus

was not recognized. A fractional test meal revealed acid on the high side of normal, especially in the resting juice. Blood was not present in any specimen.

The patient desired operative treatment, and this was agreed upon in view of the long history, previous failure of medical treatment, and recurrent loss of work owing to incapacity.

On March 13 the abdomen was opened through a midline epigastric incision. He had a large mobile stomach and duodenum, and very marked hypertrophy of the pylorus was at once apparent. There was firm thickening of the pyloric antrum spreading up from the pylorus, similar to the infantile condition. No active ulcer could be found in the stomach or duodenum, but on opening the lesser sac a small scar was found near the lesser curve just above the area of pyloric thickening. It did not seem that this could possibly have been active during his recent severe attack of pain, and there appeared to be no doubt that the hypertrophic pylorus was in fact the cause of his symptoms. Billroth I type of partial gastrectomy was therefore decided upon, and this was technically easy, the stomach being large and mobile, and the duodenum mobile and of wide diameter. After the operation recovery was entirely satisfactory; he took fluids by mouth next day, proceeded to a normal diet on the tenth day, and was discharged home, soundly healed, on the twelfth post-operative day. At twelve months, though it was too early to assess the final result, he stated that he was delighted with his operation, was entirely free of symptoms, and all heaviness after meals was relieved.

Section of the stomach had been made on the lesser curvature about 3 cm. below the cardia and on the greater curvature just below the second branches from the left gastro-epiploic vessels, the portion including a little more than half the stomach and a short cuff of duodenum. Hypertrophy of the muscularis of the pyloric antrum was considerable, and affected mainly the circular layer. Below, it stopped short of the level of the sphincter, the duodenal wall being of normal thickness; above, it extended up for a distance of about 4 cm., tapering off as the body of the stomach was reached. On the mucosal surface there was nothing to correspond with the small scar noted after opening the lesser sac. The pathologist reported as follows: "There is no evidence of ulceration, but sections through the pylorus show hypertrophy of the pyloric sphincter and of the muscle coats of the pyloric antrum; the maximum thickness of the muscle coats after fixation is 7.5 mm. There is in addition a moderate degree of chronic atrophic pyloric gastritis."

When the barium-meal films were reviewed in the light of the operative findings the characteristic x-ray features of adult hypertrophic pyloric stenosis were unmistakable. The pyloric canal was narrowed and elongated, being approximately 2.5 cm. long and 0.5 cm. in diameter. Marked concave indentation of the duodenal bulb was present, but the prepyloric cleft could not be made out.

Other Cases

Case 2.—The eldest son of Case 1 was admitted on January 10, 1942, when aged 4 weeks. His case notes, which are very scanty owing to the war, merely record that Ramstedt's operation was performed for congenital pyloric stenosis. He was discharged on February 2, and remained well and symptom-free.

Case 3.—The fourth son of Case 1 was admitted on April 26, 1950, at the age of 13 days. He had been breast-fed from birth and there had been projectile vomiting of all feeds for one day. On admission the child was listless and ill. His weight was 8 lb. 6 oz. (3.8 kg.), having dropped from 9 lb. (4.08 kg.) at birth. He was put on oral feeds of 2½ oz. (60 ml.) of 5% glucose in 1/5 normal saline two-hourly. Vomiting, however, continued after nearly every feed, and surgical treatment was decided upon on April 28. Ramstedt's operation was performed that day, a typical pyloric tumour being found. Recovery was satisfactory; he gained weight to 8 lb. 15 oz. (4.05 kg.), and was discharged on May 15. He has since remained well.

Case 4.—A nephew of Case 1, aged 7 weeks, the second child of his third sister, was admitted to hospital on September 16, 1945. Unfortunately his hospital notes have been lost, but reliable details of his case have been obtained from his private doctor and the ward report books. He was admitted with a firm diagnosis of congenital pyloric stenosis, a pyloric tumour being palpable. In hospital he responded to oral feeds of glucose-water and "eumydrin." Vomiting ceased altogether after a week or two, and he was discharged on October 28. He remained well up to the time of writing.

Discussion

In this family the incidence of the disease is confined to the male members. In the present adult generation there were three male offspring and four females. The death in infancy of the eldest brother of Case 1, the first-born male, resulted from persistent vomiting about the year 1902; the mother of Case 1 maintains that in her opinion this illness was identical with that occurring in her grandchildren, and this case is at least suggestive. If the case is accepted, then the incidence in this generation is in the first and fourth offspring, both males, with one male and four females unaffected. In the rising generation there is a total of twelve children, eight being males and four females. Case 1 has four boys, and here again numbers 1 and 4 are proved cases. In Case 4 the patient is a male child, being second-born, and the trait was transmitted through a female.

In Case 1 it may be questioned whether the pyloric condition was in fact really causing much obstruction. Fullness after meals was a constant complaint, but vomiting was only occasional, and x-ray examination revealed no four-hour residue. The barium-meal test, however, was carried out at a time when he was relatively free from symptoms, and after a period when he had been off work and on strict medical treatment under his doctor at home. If he had been x-rayed during an acute attack a four-hour residue might well have been found. In other respects the x-ray appearances were typical, the large stomach with narrowed elongated pyloric canal being unmistakable once they were looked for. At operation the "pyloric tumour" was quite obvious, and the absence of other obvious disease left little doubt that the pyloric condition was responsible for his symptoms.

Bockus (1944) divided cases of adult hypertrophic stenosis into three groups: those with signs and symptoms from childhood; those with atypical ulcer symptoms of long duration, beginning in adult life; and those who quickly develop signs and symptoms of pyloric obstruction in middle or late life. Case 1 had a ten-year gastric history without any evidence of trouble in childhood. His pain was not really suggestive of gastric or duodenal ulceration, and he therefore seems to fall into Bockus's second group. McCann and Dean (1950), reporting three new cases in the adult, classify them as (a) idiopathic, without other gastro-intestinal pathology; or (b) concomitant, with an associated gastro-intestinal pathology, such as duodenal or gastric ulceration or chronic gastritis with superficial ulceration. It may be that the symptoms in Case 1 were due to periods of gastritis with superficial ulceration, the scar noted on the lesser-sac surface of the stomach representing a deeper lesion which at the time of operation was old and completely healed. It is unfortunate that this region was not specifically sectioned by the pathologist, but on the whole it seems that Case 1 should be placed in the concomitant group.

Aetiology and Treatment

The aetiology of congenital hypertrophic pyloric stenosis remains obscure. Illingworth and Dick (1949) state that it is an inherited abnormality of recessive type, sex and primogeniture being also determining factors. Cockayne and Penrose (1943) conclude that there is a definite though small familial incidence. It seems to be generally accepted that the condition in the adult and the condition in the child are one and the same, though doubtless there are factors that determine the age of onset. McCann and Dean regard their

idiopathic type as a subclinical variety of true congenital hypertrophy, and they consider that their concomitant type may also be. The two idiopathic cases they have reported are both females and their concomitant case a male, but in addition they reviewed a total of 160 cases and found that the sex incidence was 130 males to 30 females, an incidence corresponding closely with that of the congenital condition occurring in infancy. The occurrence of one adult and three infantile cases in this family supports the contention that the adult and infantile conditions are in fact one and the same and that a familial incidence does exist.

In the infant, Ramstedt's operation has stood the test of time and is now accepted as the standard procedure. In the adult a variety of procedures have been adopted by different operators, and these include Ramstedt's sphincterotomy, finger dilatation of the pylorus, plastic procedures on the pylorus, gastro-enterostomy, and gastrectomy, either partial or subtotal.

McCann and Dean discuss treatment at some length and favour a Billroth I pyloroplasty. In their second case they performed a pyloroplasty, but symptoms subsequently recurred and a gastro-enterostomy was later carried out at another hospital. In their first case a previous gastro-enterostomy gave relief for only five years, after which Billroth I pyloroplasty was performed by them with complete relief of symptoms. Mor (1947), reporting seven cases in adults, also mentions failure of a gastro-enterostomy after 20 years, when resection had to be done. From time to time difficulty has occurred in distinguishing at operation the pyloric tumour of congenital hypertrophy from a malignant tumour infiltrating the pyloric antrum, and McCann and Dean mention this as an objection to Ramstedt's sphincterotomy. This objection applies equally to pyloroplasty and gastro-enterostomy. Other objections to Ramstedt's sphincterotomy that they mention are the possibility of diverticulum formation and of cicatrix formation with later recurrence of obstruction. I have personal knowledge of two cases in which failure to resect has led to further trouble. The second of these two cases is worthy of mention.

The patient, a man of 53, was first operated on early in 1949. He had a twelve-months gastric history, and carcinoma was suspected. At operation only a hypertrophic pylorus was found. The stomach was opened at that time and thoroughly explored, ulceration and new growth being positively excluded. A biopsy of the pylorus, which included all layers, failed on section to show ulceration or new growth. Subsequently symptoms recurred, and some two years later he was readmitted with haematemesis. While he was in hospital under treatment a prepyloric ulcer perforated, and this was repaired. Three days later further gastric haemorrhage occurred and an emergency gastrectomy was performed. The specimen removed included a pylorus hypertrophied as in the condition of congenital pyloric stenosis, and above it near the lesser curve was an ulcer, 2 in. (5 cm.) in diameter, which was apparently simple. On section, however, this ulcer was found to be malignant, the appearances being consistent with a diagnosis of carcinoma *ex ulcere*. There seems to be no doubt that a resection two years previously would have saved this patient, who died some three months later from his malignant disease.

The object of surgical treatment is to relieve the obstruction, and this is best done by removing it altogether. In Case 1, who had an acidity on the high side of normal, a Billroth I partial gastrectomy was performed, approximately half the stomach being removed. The result is so far satisfactory, and careful questioning has failed to reveal any of the usual symptoms of early "dumping." If the acidity is low Billroth I pyloroplasty as recommended by McCann and Dean would seem to be equally satisfactory in that the lesion is removed and a relatively large stomach remnant left.

Summary

A family with four members who have suffered from hypertrophic pyloric stenosis is reported. The incidence is confined to male members of two generations—one in adult life and three in infancy.

A possible fifth case in this family is mentioned, and the fact that the trait in one of the infants has been passed through a female member of the family is recorded.

Symptoms in the adult case are discussed.

Some aspects of aetiology are mentioned, and in this family the occurrence in adult and infants supports the contention that adult and infantile conditions are one and the same and that a familial incidence does exist.

Surgical treatment in the adult is discussed and a plea made for Billroth I gastrectomy or pyloroplasty.

I am indebted to Dr. J. H. Moseley for permission to publish details of Case 3; to Dr. C. N. Suter, of Gosport, for information on Case 4; to Drs. R. D. Clay and R. H. MacHardy for pathological and x-ray reports; and to Mr. Bernard L. Williams for helpful criticism.

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POST-PARTUM VENOUS THROMBOSIS AND LEG ULCERS

BY

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Of 557 women, in a series of 804 patients with leg ulcers all examined personally, there were 160 (28.7%) who had suffered from post-partum venous thrombosis of the lower limb. It is only by greater knowledge of its aetiology that we can hope to prevent this condition and the great disability it entails. Here it is intended to consider certain aetiological factors in relation to the present series of patients and to attempt to assess their importance.

When examined the patients were 28 to 80 years of age (the majority being 35 to 60); they were seen at varying periods after the confinements and the thromboses which followed. Information could be obtained only from the history and physical examination, a method more practicable but less accurate than that of following the course of a patient from time of thrombosis to that of ulceration of the leg. Nevertheless, details of the confinements, though not full, seemed essentially accurate. Women usually remember about their abnormal labours.

Obstetrical History

The number of confinements, from 1 to 13, of the women in this series are shown in Table I. Table II shows the number of the first confinement after which there was thrombosis. It occurred after more than one confinement in some women. In nine there were 12 further attacks of venous thrombosis after subsequent confinements.

In order to determine the incidence of venous thrombosis in relation to the number of confinements, the number of thromboses occurring after the first or later confinements is shown in Table III. Thus there are records of 157 first confinements after which venous thrombosis occurred on 78 occasions (50%), but after 123 second and after 96 third confinements the incidence of thrombosis dropped to about