routes, have come to the same conclusion—that vagotomy and Billroth I resection should be tried further. Vagotomy and limited partial gastrectomy has been very successful in the hands of Johnson and Orr (1953).

Summary

For duodenal ulcer, excluding cases of acute bleeding but including a few perforated cases which required more than simple closure, the operation of abdominal vagotomy combined with a low gastro-jejunostomy is a very safe procedure and appears to produce good results lasting four to six years in 88%.

The failures (7%) were submitted to a further operation without mortality: all except one case have been relieved.

A Billroth I conversion was the most satisfactory secondary procedure; also, vagotomy combined with a Billroth I partial gastrectomy may prove to be the best primary procedure.

Vagotomy should be included in the treatment of duodenal ulcer because only 50% of permanent good results can be expected from gastro-enterostomy alone.

We now regard vagotomy combined with gastro-jejunostomy to be at least the best second-choice operation.

Most of the follow-up work was done by S. O'D., but we are indebted to Mr. Harral Thompson for following up the cases at the Royal Southern Hospital during his tenure of the post of surgical registrar

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EFFECTS OF DORSO-LUMBAR SYMPATHECTOMY ON PEPTIC ULCERATION

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Extensive dorso-lumbar ganglionectomy and splanchnicectomy as carried out from the treatment of hypertension interrupts the sympathetic nerve supply to the stomach (Bingham *et al.*, 1950); by masking symptoms it may have a harmful effect on the course of peptic ulcer. Frequently it is only by complications such as pyloric obstruction, perforation, or haemorrhage that the ulcer announces its presence. The following case illustrates these features.

Case Report

A male shipping clerk aged 38 was admitted in April, 1949, suffering from angina of effort. This had started six months previously, when he had breathlessness and pain

down the inner side of the left arm on exertion; a week later the pain became substernal and compelled him to stop when walking. During the six weeks before admission pain had come on at rest and had lasted about five minutes. He gave a previous history of an appendicectomy for vague dyspeptic symptoms in 1930; the operation had given him relief, but at intervals he had continued to have mild epigastric discomfort. There was no history of nephritis or other renal lesion. The family history was indefinite; his mother had died in childbirth and his father died suddenly at age 40, cause unknown.

He was a moderately well nourished man whose blood pressure remained between 230/130 and 220/130 during his stay in the medical ward. The fundi showed early vascular changes but no haemorrhages, exudates, or papilloedema. Clinical examination of the heart, lungs, abdomen, and central nervous system was negative and there was no albuminuria. A cardiogram taken in March, when he was an out-patient, had shown evidence of ischaemia in the shape of terminal inversion of the T waves in lead V₄; but on admission the cardiogram at rest was normal. Radiologically the heart shadow was normal and the lung fields were clear. Intravenous pyelography showed normal concentration and excretion of the dye, and no abnormality was demonstrated in either kidney. Urea clearance was 74%, maximum urea concentration 2.3 g. per 100 ml., and blood urea 36 mg. per 100 ml.

In May, 1949, a left dorso-lumbar ganglionectomy and splanchnicectomy was performed by the transpleural route, the ganglia from D 6 to L 1 being removed. After this he had complete relief from angina of effort. His blood pressure remained in the region of 155/110 until the beginning of October, when it began to rise again. During this period, however, he complained of vague epigastric discomfort and distension with occasional slight nausea.

He was readmitted in November, 1949, with a blood pressure of 198/118, and a right dorso-lumbar ganglionectomy and splanchnicectomy was performed, the ganglia from D 6 to L 1 being removed. After this operation he remained completely free from angina, and his own doctor never found his systolic blood pressure higher than 180. However, he began to have attacks of epigastric distension and vomiting. At first these attacks occurred at intervals of several months, but later the intervals shortened to two or three weeks. During the attacks vomiting was profuse and continued at 24 hours or more until the stomach was completely emptied; food taken the previous day was sometimes recognized. His appetite failed, he lost weight, and he experienced increasing constipation. Pain was absent throughout. He was seen in consultation on July 25, 1951, when a diagnosis of pyloric stenosis was made; at that time his blood pressure was ranging between 160/100 and 140/90. A barium-meal examination showed a large resting content in the stomach and a marked degree of pyloric stenosis, which was not lessened after treatment with atropine. He was admitted once more in August, and laparotomy was performed in September. There was a large active chronic ulcer of the anterior duodenal wall, covered by omental adhesions; the stomach was dilated and its muscular wall was much hypertrophied. A posterior (no-loop antiperistaltic) gastro-jejunostomy was performed.

He remained well for eight months, then began to have a sense of fullness after meals with anorexia but no pain. On August 24, 1952, he was awakened with sudden severe pain radiating over the whole abdomen and accompanied by tenderness and rigidity. A laparotomy was carried out (Mr. J. Hinton Robertson); after evacuation of a considerable quantity of gas and fluid, a perforated jejunal ulcer was located diametrically opposite the stoma of the gastro-jejunostomy. This was closed by suture. During convalescence a fractional test meal showed total acidity of 48 units and free HCl 22 units. Two months later the gastro-jejunostomy was undone and a partial gastrectomy was

carried out, four-fifths of the stomach being removed; continuity was restored by an anterior Polya with Hofmeister valve anastomosis.

When last seen (November 15, 1954) he was working again and doing more than he had been able to do for many years, but had given up sport. He has remained completely free from angina of effort, despite the fact that his blood pressure has returned to the pre-operative level; recent readings have been 220/130 (October 30, 1953); 200/110 (September 3, 1954); 240/134 lying, 220/130 standing (November 15, 1954). Changes in the retinal arterioles have advanced, but the heart shadow remains normal radiologically, and the cardiogram was still physiological. During the past year he has from time to time had pain in the left upper abdomen, sometimes three hours after food, sometimes waking him at night. A barium-meal examination showed normal progress from the remaining portion of the stomach to the jejunum, and no evidence of ulcer was seen: progress through the small bowel was accelerated.

Discussion

Painful sensations arising in the abdominal viscera are transmitted by afferent fibres which accompany the sympathetic nerves and pass uninterruptedly through the sympathetic ganglia to enter the spinal cord by the posterior nerve roots. Dorso-lumbar ganglionectomy will abolish those forms of abdominal visceral pain which are normally transmitted through the sympathetic. When perforation occurs structures supplied by other afferents are involved, including the parietal peritoneum; pain from here reaches the spinal cord by way of the peripheral nerves and is not abolished by dorso-lumbar ganglionectomy.

Froehlich *et al.* (1942) reported four cases in which sympathectomy was performed as treatment for gastric ulcer. In all of them the distress was relieved and the ulcer craters showed radiographic evidence of healing. Sicard *et al.* (1945) carried out splanchnicectomy in eight cases of gastroduodenal ulcer. Although there was some relief of ulcer pain, cure was not complete. Five required gastric resection within 18 months.

Harkins and Willard (1950) speak of the relief of pain as "a Pyrrhic victory" in reporting a case of peptic ulcer in which sympathectomy had adverse effects. Weeks *et al.* (1946) describe the case of a man with duodenal ulcer and malignant hypertension. A transthoracic vagotomy was performed and at the same time the left thoracic-lumbar sympathetic chain was removed from D5 and L2. Two weeks later the second-stage sympathectomy was carried out on the right side from D5 and L2, after which the patient became extremely ill and died on the 15th post-operative day. Necropsy revealed a perforated duodenal ulcer with general peritonitis. An interesting feature was the absence of abdominal pain in the presence of a perforated ulcer and peritonitis. The authors state: "His absence of pain may have been due to the removal of the viscerosensory fibres associated with the sympathetic side of the autonomic nervous system." Blegen and Kintner (1947) describe a case with gastric ulcer which had three massive haemorrhages following extensive unilateral sympathectomy, and point out that the sympathectomy relieved the pain but the ulcer progressed.

Mason and Pollard (1949) found 13 instances of peptic ulcer in a group of 1,498 patients who underwent splanchnicectomy for hypertension during an 11-year period. They point out that frequent and severe complications developed in 12 because the normal epigastric pain sensation was either abolished or greatly reduced so that ulcers were permitted to advance without giving the usual warning.

The present case illustrates progression of a duodenal ulcer and development of pyloric stenosis after bilateral dorso-lumbar ganglionectomy and splanchnicectomy carried out for hypertension; a jejunal ulcer subsequently developed and later perforated. Pain was conspicuous by its absence while the original ulcer was progressing and while the jejunal

ulcer was developing, but severe pain occurred when the latter perforated. After dorso-lumbar ganglionectomy and splanchnicectomy the pain which characterizes exacerbations of a peptic ulcer is abolished; it is therefore suggested that in such cases more attention should be paid to vague abdominal symptoms which may take the form merely of slight epigastric discomfort or fullness.

It is well known that the lowering of blood pressure which results from sympathectomy has often been transient. Hypertension has recurred within 5 to 10 years in a high percentage of cases (Smithwick, 1948). In the present instance hypertension was abolished for three years but had returned by the end of the fifth year. The mechanism underlying recurrence of hypertension after sympathectomy is not fully understood. During the fifth year abdominal visceral pain returned; but it may well have arisen in the colon. The sympathetic nerve supply from the colon was not interrupted by the original operations.

Summary

A case is presented showing the progression of a duodenal ulcer following bilateral dorso-lumbar sympathectomy and splanchnicectomy, the subsequent development of a jejunal ulcer, and its ultimate perforation. The effect of sympathectomy on the pain of peptic ulcer is described and its mechanism is discussed.

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MALARIA AND THE SICKLING TRAIT

BY

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This paper reports confirmation of Allison's (1954) conclusion that the possession of the sickling trait confers some protection against *P. falciparum* malaria, but it presents somewhat different views on the nature and extent of this protection.

Of the two experiments performed by Allison, one determined the parasite rates in children under 5 years of age in the Kampala region, mostly (Allison, personal communication) in the out-patient department of Mulago Hospital. Forty-three sickling children showed a parasite rate of 27.9%, and 247 non-sicklers a rate of 45.7%. This difference seemed so striking that it was reasonable to wonder why it had not already been noticed, or whether some feature of the experiment had accentuated a difference that was real, but actually of lesser magnitude. Indeed, since *P. falciparum* clearly can establish itself in the peripheral blood of sicklers, and can even multiply in the erythrocytes of cases of sickle-cell anaemia (in which there is no normal adult haemoglobin), it seemed reasonable to suppose that the effect of the presence of sickle-cell haemoglobin could not be to prevent an infection, though it might well limit the extent to which an infection could develop. If this were confirmed, quite sufficient explanation would have been given of the way in which *P. falciparum* infection could act as a selective agent in preserving high sickle-cell gene frequencies in endemic malarial regions, because it is