and Guerry, 1939; Nygaard, 1939). It is held, therefore, that the reserve of prothrombin or of vitamin K is not adequate to maintain the prothrombin level in the newborn until the time when vitamin K is absorbed in sufficient quantities from the alimentary canal. In the adult intestine vitamin K is absorbed direct from the food in the presence of bile, and from the colon, where vitamin K may be synthesized by bacterial action.

Milk is a poor source of vitamin K, and in the newborn the contents of the intestine are sterile. Hence it has been assumed that enough vitamin K cannot be absorbed until bacterial invasion of the bowel occurs. Breast-feeding or the use of unsterilized breast milk on the first day should assist this synthesis by supplying numerous harmless bacteria, but does not afford any protection against the fall in plasma prothrombin. Other factors also may be involved. It is not known, for instance, whether the composition of bile in the newborn is suitable for optimal absorption of vitamin K. Jaundice and prothrombin deficiency do not necessarily coexist. The postulation, however, that icterus simplex neonatorum is due to excessive haemolysis and to functional immaturity of the liver (Rich, 1930; Ross *et al.*, 1937) inevitably suggests that deficient synthesis may also be a factor in neonatal hypoprothrombinemia.

There would seem to be no doubt that abnormal bleeding in the newborn coincides with excessive prothrombin deficiency. The association of an unduly low prothrombin index, generally where there has been maternal toxæmia or difficult labour, with the occurrence of symptoms of intracranial damage is shown in fourteen cases in this paper. Moreover, at the time when the prothrombin index is lowest and the clot formed *in vitro* most fluid there is often an increase in the signs of cerebral irritation. That a secondary oozing has occurred through this soft clot, possibly loosened from a birth injury by restlessness or a bout of crying, seems to be a reasonable conclusion. The rapidity with which symptoms were relieved after the use of vitamin K analogues and the infrequency of their occurrence when the mother has been treated suggest that the theoretical value of the naphthoquinones in suspected intracranial haemorrhage is worthy of further therapeutic trial.

It is evident that prevention of the prothrombin deficiency will decrease the incidence and severity of complications in the newborn. In most normal babies early breast-feeding will prevent abnormal or prolonged lowering of the plasma prothrombin. Adequate administration of vitamin K analogue to the mother less than twenty-four hours before delivery leads to its rapid absorption into the foetal circulation, and appears to supply a store of vitamin K sufficient not only to raise to normal limits the prothrombin content of the baby's blood at birth but also to maintain it within normal limits during the early days of life. The constancy with which a normal plasma prothrombin was found at birth and its importance in the formation of strong adherent clots in any case of difficult delivery need no emphasis. Indeed, they suggest that this is by so much the most rational method of treatment that the administration of naphthoquinone at this time may become as routine as the use of vitamins A and D earlier in pregnancy.

A similar effect after birth, with a similar absence of any untoward reactions, is obtained when the vitamin K analogue is given in the first few hours of life. Injection of the naphthoquinone gives a speed of action comparable to that of blood transfusion, and its effect seems to be more lasting.

All these facts suggest that the administration of a vitamin K analogue, either to the mother between twelve and four hours before delivery or to the newborn, would appear to be especially indicated (1) in cases of maternal toxæmia; (2) in premature labour; (3) in cases of difficult or instrumental delivery; (4) where breast-feeding is not possible; (5) when any cerebral symptoms develop during the first few days of life; (6) in cases of haemorrhagic diathesis, icterus gravis neonatorum, and anaemia; and (7) where an operation is necessary on the newborn.

We wish to acknowledge our thanks to Glaxo Laboratories Ltd. for the supply of vitamin K analogues, and especially to Dr. Patricia Fitzsimons and Mr. A. L. Bacharach for their interest and help; to Sisters Somerville, Anderson, and Nixon for their care in the administration of the drug; and to Professor R. W. Johnstone and Professor Charles McNeil for permission to use the drug in certain cases under their charge.

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PERFORATED JEJUNAL ULCER FOLLOWING PARTIAL GASTRECTOMY

BY

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Acute perforation following partial gastrectomy is a rare condition, only two such cases being found among the records of the London Hospital for the last ten years. During the same period twenty cases of perforation following gastro-jejunostomy were recorded. I was called upon to deal with one case of perforated jejunal ulcer following partial gastrectomy, a report of which is here given, together with the record of the previous case.

Case I

In 1926 the patient, a woman aged 45, was admitted to the London Hospital, under Dr. Rowlands, with a three-years history of severe colicky epigastric pain radiating to the hypogastrium, not related to meals. Vomiting occurred about once a week, and relieved the pain. The pain and vomiting were worse for the three months prior to admission, and the vomitus was alleged to contain occasional streaks of blood. The appetite was poor and there was loss of weight. There was a history of melaena and dyspnoea on exertion.

On examination the patient had a sallow appearance, with pallor of the mucous membranes. No abnormal physical signs were found. A test meal revealed free HCl 0.28 per cent. and total acidity 80. A barium-meal radiograph showed a low hyperperistaltic stomach, an active pars pylorica and first stage of duodenum, and a defect on the inner aspect of the duodenal cap. The blood count showed erythrocytes,

3,900,000 per c.mm.; haemoglobin, 40 per cent.; colour index, 0.57; leucocytes, 3,440 per c.mm. Anisocytosis and poikilocytosis were present.

First Operation.—In April, 1926, an operation was performed by Mr. E. C. Lindsay. A chronic duodenal ulcer was found on the anterior aspect of the first part of the duodenum. Posterior gastro-jejunostomy and appendicectomy were performed. The appendix showed slight submucous fibrosis. The patient made an uninterrupted recovery and was discharged to a convalescent home.

SECOND ADMISSION

The patient did well at first, but had a relapse in 1927 and was readmitted to hospital with the history of recurrence of lower abdominal pain one month after operation, with diffuse distribution and no definite radiation. It was worse on the right side than the left, and was not related to food. There was occasional nausea but no vomiting, and a history of constipation since operation but no melaena. The appetite was good, but she had lost 8 lb. in two months. Examination showed a thin poorly nourished woman with no abnormal physical signs. The test meal showed free HCl 0.10 per cent. and total acidity 40.

On radiographic examination the barium meal was held up in the cardiac end of the stomach for a considerable time, owing apparently to a constriction of the body above the anastomosis. The stoma appeared to work when the meal passed the constriction, but most of the meal passed by way of the pylorus. During observation in the ward the patient had further melaena and more pain, particularly three hours after food.

Second Operation.—In view of the severity of the symptoms a second operation was performed by Mr. Lindsay in February, 1927. A large ulcer was found at the site of the old anastomosis, which showed marked constriction. The old duodenal ulcer was healed. The anastomosis was resected, the jejunum closed, and an anterior Polya partial gastrectomy performed. The amount of stomach removed from the pylorus measured 7.5 cm. along the lesser curve and 17 cm. along the greater curve. The patient made an excellent recovery and was discharged to a convalescent home.

In March, 1929, there was a further recurrence of symptoms consisting of epigastric pain and nausea. An x-ray picture showed evidence of a jejunal ulcer. Treatment by diet and alkaline powder controlled the symptoms effectively. In 1936 there was a recurrence of pain and vomiting, with one haematemesis and one attack of melaena. A radiograph revealed a delay at the stoma with evidence of jejunal ulcer. Medical treatment again resulted in an improvement.

THIRD ADMISSION

In December, 1938, the patient was readmitted to the London Hospital as a surgical emergency. There was a history of four days' increase in generalized abdominal pains after food. For the last four hours there had been acute generalized abdominal pain with nausea, but no vomiting, no melaena, and no haematemesis. On examination the patient presented an ill appearance and was lying very still, with a definite pallor, a cold moist clammy skin, and dry furred tongue. The abdomen showed a marked restriction of movement, with generalized tenderness most pronounced in the epigastrium, and board-like rigidity, particularly above the umbilicus. Diminution of liver dullness was found on percussion. The diagnosis of perforated anastomotic ulcer was made.

Third Operation.—This was performed by me for Mr. Lindsay. A right paramedian incision was made. Gas and much free fluid were found in the peritoneal cavity and a large perforation a quarter of an inch in diameter was present in the jejunum just distal to the anastomosis. This was embedded and covered over with omentum, and one local drainage tube was inserted. During the operation 350 c.cm. of saline was administered intravenously, and twenty-four hours later a blood transfusion of 400 c.cm. was given. The patient made an excellent and uncomplicated recovery, and

on discharge to a convalescent home had no pain, nausea, or vomiting, and was enjoying a bland IV diet. Two months later in the follow-up of this case there had been a recurrence of vomiting.

FOURTH ADMISSION

The patient was readmitted for investigation in March, 1939, nearly four months after the perforation, with a history of repeated small vomits, streaked with blood on one occasion, together with periodic vague abdominal discomfort. The appetite was fair and there had been no loss in weight. Examination disclosed a thin woman with slight epigastric tenderness. A test meal showed free HCl 0.09 per cent., with total acidity 60. In forty-five minutes the free HCl was *nil* and total acidity 10; following which the HCl steadily rose to 0.2 per cent. in two and a half hours, with a total acidity of 84. A barium-meal radiograph showed narrowing and fixation of the efferent loop, causing delay in emptying, and a large residue of barium, in all probability due to jejunal ulcer.

A gastroscopic examination by Mr. Hermon Taylor showed the amount of stomach to be about half normal, with three inches of lesser curve. A considerable area of secreting mucosa was still present, with marked hyperplastic gastritis. The stoma was very greatly distorted and stenosed, tense oedema of the mucosa probably screening an ulcer beyond. The conclusion reached was that there was still an excess of gastric hyperplastic mucosa, with indirect evidence in favour of a persisting jejunal ulcer. The patient was discharged home on medical treatment—and the possibility of carrying out a further gastrectomy is under consideration.

Case II

The patient, a male aged 30, was admitted in March, 1935, to the London Hospital under Mr. Alan Perry, with the diagnosis of chronic duodenal ulcer. There was an eight-years history of periodic epigastric pain radiating to the back and keeping him awake at night, with no relation to meals. Occasional vomiting relieved the pain, which was also relieved by bismuth powder. There was no loss of weight and the appetite was good. Examination showed that the patient was a well-nourished man with no abnormal physical signs. A test meal gave free HCl 0.30 per cent., with total acidity 110, and a radiograph showed a "cup and spill" type of stomach which was very hypertonic, and a deformed duodenum. At an operation performed by Mr. Alan Perry multiple scars in the duodenum were found. An anterior Polya partial gastrectomy was done. The microscopical report on the stomach stated that there was focal chronic inflammatory infiltration, and the amount of stomach removed from the pylorus measured 5 cm. along the lesser curve and 9 cm. along the greater curve. The patient's recovery was uninterrupted, apart from prolapsed piles, and he was free from symptoms when seen on subsequent occasions in the follow-up department.

READMISSION TO HOSPITAL

In November, 1937, the patient was readmitted as a surgical emergency with a twelve-hours history of sudden onset of severe epigastric pain and sensation of faintness, the pain radiating to the left shoulder. On examination he was lying very still and presented an ill appearance. The temperature was 100.4°, pulse 120, and respirations 30. There was tenderness and rigidity in the upper abdomen, more pronounced on the left of the midline. Liver dullness was diminished on percussion. At operation (Mr. C. L. Heanley for Mr. Perry) free gas and fluid were found in the peritoneal cavity and there was a small perforation in the anterior wall of the jejunum half an inch distal to the line of the previous anastomosis. The perforation was sutured and suprapubic drainage carried out. An uninterrupted recovery was made, the post-operative test meal showing free HCl to be 0.18 per cent. and total acidity 63.

Discussion

Both of these were cases of chronic duodenal ulcer in the first place, with high acidity. In Case I gastro-enterostomy resulted in a considerable lowering of free HCl

from 0.28 per cent. to 0.10 per cent. and the total acidity from 80 to 40; but, nevertheless, jejunal ulceration ensued, even after partial gastrectomy, and in spite of symptomatic relief from medical treatment ran a progressive course to final perforation. In Case II, despite a partial gastrectomy, the final level of acidity was still high, the free HCl being 0.18 per cent. and the total acidity 63 (these being figures after the perforation had been successfully treated). The perforation occurred in a similar site in both instances—namely, in the jejunum just distal to the line of the anastomosis and on the anterior surface, therefore being truly jejunal.

In the subsequent follow-up and investigation of the first case the free HCl was 0.09 per cent. rising to 0.20 per cent., and the total acidity 60 rising to 84, and gastroscopy also showed hyperplastic gastritis with secreting mucosa in the remaining portion of the stomach. The actual perforation is of course only a complication of the persisting ulceration, but since partial gastrectomy was accepted as a treatment for duodenal ulcer cases with persistence of symptoms after it have been accumulating. The two cases reported suggest that if a partial gastrectomy must be done for a duodenal ulcer it should be a high one, and that a more radical or subtotal gastrectomy should be performed primarily in these cases of chronic duodenal ulcer with high acidity and hyperplastic gastritis rather than the less complete partial gastrectomy, the aetiological factor in ulcer production—namely, excess acid—being associated usually with a hyperplastic gastritis. Other aetiological factors, such as the ulcer diathesis with a fairly high family incidence, infection, poor surgical technique, and lack of medical care, undoubtedly may also play a part, but these as a rule are either not so prominent or can be more easily excluded. Little could be found in the literature with regard to perforation of anastomotic or jejunal ulcers into the peritoneal cavity following partial gastrectomy.

The incidence of gastro-jejunal ulcer following gastro-jejunostomy varies from 4 per cent. (Walton) to 24 per cent. (Strauss). After resection these figures fall considerably: 0.4 per cent. (Schwarz) and 10 per cent. (Gatewood). Gastro-jejunal ulcer usually occurs in the efferent loop near the anastomosis or actually at the anastomosis—jejunal 25 per cent. and marginal 75 per cent. (Starlinger). Penetration into neighbouring organs is more common than perforation, the incidence according to Starlinger being 5 to 6 per cent. in 2,500 cases of gastro-jejunostomy and 3 to 5 per cent. in 300 cases of resection.

Over the same ten-year period at the London Hospital the records showed five cases of gastro-colic fistula following partial gastrectomy, and two cases of peri-anastomotic abscess the result of slow perforation or leakage. Following gastro-jejunostomy twenty-four cases of gastro-colic fistula are recorded. Marshall and Kiefer state that post-operative jejunal ulcer can also occur after partial gastrectomy, but does so with much less frequency than after partial gastro-enterostomy. Symptoms are usually severe, and intractable to medical treatment; there is also a tendency to bleed and to perforate into the abdominal cavity or colon. It is the general experience of those authors that post-operative ulcer is associated with persistently high gastric acidity.

Burke in his discussion of the operative mortality and morbidity of partial gastrectomy for gastric and duodenal ulcer in 606 consecutive cases gives a mortality rate of 5.44 per cent., but no case of perforation of an anastomotic or jejunal ulcer following this operation.

Lake mentions one case of perforated jejunal ulcer in a series of 198 followed-up cases of gastric and duodenal

ulcer treated by partial gastrectomy. Such a complication, therefore, is an uncommon one, and its incidence in cases of chronic duodenal ulcer is likely to be kept low by the performance of partial gastrectomy at a level high enough to ensure definite diminution in the free HCl production and the associated area of hyperplastic gastritis in the stomach wall. Unfortunately this is associated with a higher primary operative mortality, and in some cases where there is a real ulcer diathesis ulcers tend to recur, whatever the treatment.

I am most grateful to Mr. E. C. Lindsay and Mr. A. C. Perry for permission to publish their cases and for their advice and suggestions; also to Sir James Walton for his help with the bibliography, and to Mr. Hermon Taylor for his gastroscopic report and criticism.

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USE OF THE THIERSCH SKIN GRAFT

BY

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The rapid healing of any raw surface is important in minimizing a patient's discomfort and invalidity, but above all in obtaining the earliest and maximum return of function. Most raw surface areas are produced by accident or surgery. Skin grafting is the most prompt available method of obtaining healing of such an area, and it is the one which produces the least subsequent distortion. The general surgeon does not use skin grafting to full advantage. This can only be explained by a widespread unfamiliarity not only with its technique but with the results to be expected from its application. The Thiersch graft is the one most universally applicable, but certain conditions must be laid down for its use. If these, together with details of technique, are respected the results are good; if not they are correspondingly bad.

This paper is based on a review of all the Thiersch grafts done by the plastic surgery unit of the London County Council.* Special consideration has been given to cases in which the grafts partially or completely failed. Attention to various points will help to make the procedure of skin grafting more certain in the hands of a general surgeon. It may then be used as a prime method of treatment rather than as a last resort. It is significant that many of the conditions treated could have been dealt with more effectively by grafting at a much earlier stage in their clinical history. Moreover, primary skin grafting would have altogether prevented many of the gross deformities and much of the malfunction which have been seen.

* In all, 629 grafts; these include 535 surface grafts and ninety-four inlay grafts. Thirty others were excluded because of inadequate records.