Broadly speaking, the choice is between interpretations 2 and 3, although it must be recognized that these are by no means mutually exclusive. Since 2 is concerned with a specific selective process and 3 with a hypothetical type of induced mutation, the two could well be combined.

Alternative 2, the immunological theory of cancer, has the great advantage of being susceptible to experimental attack along the lines discussed by Green (1954). I have not been able to find any account of experiments in which the action of standard carcinogens was tested in mice treated with *x* rays and cortisone to eliminate or greatly diminish the antibody response. A strong anticarcinogenic effect under these circumstances might speak strongly for the correctness of Green's hypothesis.

Alternative 3 is virtually what has been suggested by Haddow and others largely on the basis of the work by the Millers, Weiler, etc., on the production of hepatomas in the rat. It is wholly speculative at the present time, the difficulty of establishing it being the familiar biological one of differentiating between spontaneous mutations with selective survival and a direct environmental effect. Past experience should make us lean toward a Darwinian rather than a Lamarckian interpretation, but, on the other hand, we cannot ignore the possible analogies from the process of differentiation.

Differentiation can be legitimately regarded as a process by which the potentialities of a cell are progressively reduced with its taking on of a specialized function. Tissue culture studies indicate that to a considerable extent the differentiated cell is so in virtue of its inheritance. Even in the absence of normal body controls, its descendants differ from the descendants of other types of specialized cells. If this is so the differentiation must also involve the genetic mechanism. Various aspects of this must be "closed down" either by some form of inhibition or by actual irreversible loss of the genetic unit in question. This closing down cannot be of the nature of a random mutation; it is causally determined in the process of development. The sequence of cause and effect is unknown, but speculation would follow the lines adopted by Pollock (1953) in his interesting discussion of the way enzymes may be developed in the process of biochemical ontogeny. An enzyme is built when a substrate for it appears: a gene will be inhibited, destroyed, or lost when its final product ceases to be of functional significance. To convert that teleological statement into mechanism is a task for the future. At the very least it seems to demand that there is a "feed-back" of information from the cytoplasm to every functionally active component of the genetic mechanism. Most cytologists nowadays would probably be willing to allow this.

There may therefore be some justification for going beyond random somatic mutation to account for the high intensity of carcinogenic action of certain chemical agents.

[*Parts III and IV, with a list of references, will appear in our next issue.*]

The Casualties Union has published a booklet describing its history and work. In 1942 Mr. E. C. CLAXTON, then commandant of the Surrey County Civil Defence Rescue School at Leatherhead, founded the Casualties Union to provide skilled "casualties" for use in Civil Defence training schemes. So that they could play their part with realism these "casualties" were given a medical briefing and a theatrical make-up. The scheme proved so successful that by 1944 the School was turned over wholly to training members of the Allied Armies. When in 1945 Civil Defence was stood down the Casualties Union decided to continue its work, and still provides skilled "casualties" for use in rescue training of all types, first-aid lectures, and rescue competitions. The Union, with a membership of about 1,500, is run on voluntary subscription, and has extended overseas. Inquiries about the Union will be welcomed at its headquarters at 316, Vauxhall Bridge Road, London, S.W.1.

# CLINICAL AND SOCIAL PROBLEMS OF PEPTIC ULCER*

BY

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## Diagnosis

Establishment of the diagnosis of peptic ulceration can be difficult. The clinical picture may be atypical and there are fallacies about the various investigations.

### Clinical Picture

Typically there is epigastric pain associated with food, eased by alkalis and coming on in attacks, but there are many variations. The pain may occur high up in the chest over the sternum, or lower down around the umbilicus or even in the iliac fossae. Sometimes the pain is predominantly in the loin under the costal margin, spreading into the back, simulating a renal pain, especially on the right side. It may be felt only in the back and thought to be muscular in origin—"dyspeptic backache." Apart from the position being unusual, the pain may range from a minimal discomfort to one of considerable severity. It is not uncommon for ulcer pain to come on unrelated to meals, particularly towards the end of the day; relief with alkalis, although the rule, may be lost, particularly with chronic perforation of the ulcer from the stomach or duodenum into the surrounding viscera. Sometimes no pain at all is felt and the ulcer reveals itself by bleeding or perforating. Heartburn, anorexia, periodic nausea, bouts of vomiting, waterbrash, all without pain, may be the only symptoms.

These unusual clinical pictures cause serious difficulties, particularly with patients in whom the radiologist has difficulty in demonstrating an ulcer. This occurs especially in ulcers in the roof of the pyloric antrum and with post-bulbar duodenal ulcer; in such cases a series of negative reports may be recorded before the presence of the ulcer is finally proved, perhaps by clinical complications, gastroscopy, or laparotomy. These patients tend to be diagnosed as having an abdominal neurosis, chronic pancreatitis, or lumbago. They may well develop a secondary neurosis from the uncertainty of their lives and from the failure of their doctors to establish a physical diagnosis. In practice these patients have a consistent story, clear-cut and unembellished by the flights of fancy so common with the true neurotic. The ulcer subject may have pain "like knives sticking in me," which is true, but he will not say he is being "burned alive with wind."

Why do some patients have atypical symptoms? The ulcer with a thick slough may itself be insensitive to pain while pylorospasm is producing oesophageal reflux and heartburn. Enterospasm may occur and the patient may complain of flatulence, trying to bring up wind unsuccessfully, as it may be locked in the splenic flexure of the colon. The ulcer may penetrate surrounding structures, giving rise to parietal pain in the back. A stomal ulcer after partial gastrectomy may become adherent to the diaphragm and chest wall, causing severe

*The second of two Lumleian Lectures delivered to the Royal College of Physicians of London on April 10 and 12, 1956. The first lecture was printed in last week's issue (p. 719).

intractable pain which is possibly diagnosed as intercostal neuritis. Similar pain may be felt in the back, with posterior perforation, and may be diagnosed as rheumatism. Even without parietal extension, pain reference may be bizarre. As the foregut is embryonically a midline organ, the pain reference may be to either side, and left-sided pain may be felt with a duodenal ulcer even when a gastric lesion has been fully excluded. Individuals vary greatly in their threshold to pain—some are very insensitive to parietal pain, others extremely sensitive—and this can be measured with an algometer (Keele, 1954). Lack of pain from viscera may correlate with pain insensitivity as measured in this way, but this is inconsistent and more work is needed on this point.

With any recurrent pain from below the nipples to the iliac crest, in the loin or in the back, one cannot exclude an ulcer because the pain is unrelated to meals or unaffected by alkalis or vomiting, and further investigation is needed. Physical examination, too, may be misleading. Usually there is epigastric tenderness, but this can be entirely absent in the presence of an active ulcer. Further investigation may involve examination of stools for occult blood, barium meal, gastroscopy, and gastric secretory tests.

### Investigations

*Barium-meal Studies.*—With present-day radiology there are still practical difficulties in the demonstration of peptic ulcer, and there is need for the clinician to be more aware of the practical problems of radiology of the alimentary tract. Too often still the radiological report is regarded as final proof. The interpretation of various shadows, deformities, or lack of deformities, must be interpreted in relation to the clinical picture. Some radiologists readily interpret minor mucosal irregularities in terms of peptic ulcer, and others are not so misled. The interests of the patient are best served by maintaining a personal contact between radiologist and clinician who jointly discuss the films.

Considering the physical limitations of the method of investigation with its reliance on shadows and outlines, results are surprisingly accurate in expert hands. Nevertheless, both false-positive and false-negative reports can be made, but these diminish in direct proportion to the contact between clinician and radiologist, and their mutual experience of gastroscopy.

*Gastroscopy.*—With first-class radiological assistance, the scope for gastroscopy is limited, but it is of help in those cases where there is a discrepancy between the clinical and the radiological picture. Occasionally a chronic gastric ulcer missed by the radiologist may be discovered, and this occurs particularly with ulcers well away from the lesser curve or in the antrum. Sometimes rugal folds may simulate a gastric ulcer on *x*-ray examination, and the diagnosis can be disproved by gastroscopy. The diagnosis of a gastric diverticulum may be firmly established by direct inspection. The finding of gross mammillation and rugosity of the mucosa may lead to further radiography of the duodenal bulb for duodenal ulceration, or *p*H studies. Gastroscopy is especially useful in assisting in the diagnosis of bizarre *x*-ray pictures, when the question of malignancy arises. With present-day technique and adequate premedication, the procedure is extraordinarily well tolerated, and the risk of oesophageal trauma or other hazards is very small. The examination may be done under general anaesthesia if the patient is especially sensitive. Gastroscopy also has a place in defining the cause of bleeding after haematemesis and melaena, particularly in demonstrating acute gastric ulcers which heal quickly.

*Gastric Secretory Tests.*—Normally gastric secretory tests play a very limited part in the diagnosis of peptic ulcer. Their contribution has been very much overrated in the past, as their fallacies have not been sufficiently appreciated. As commonly done by the nursing staff, fractional test meals are an almost worthless investigation and do not justify the discomfort to the patient or the time spent by the laboratory staff. If any value is to be given to a report it is essential to know where the tube was in the stomach. In at least one in five tests the tube will curl up in the fundus, where the level of acidity is often substantially lower than in the body of the stomach. This fallacy can be avoided by using a radio-opaque tube and screening the patient at onset to make certain the end of the tube is to the left of the spine and not coiled in the stomach. No test has yet been devised which conclusively separates duodenal ulcer patients from those without ulcers, but duodenal ulcer tends to be associated with hypersecretion, and its demonstration may contribute to the diagnosis in some cases. Alternatively, the finding of achlorhydria would be against the diagnosis. The stimulus of food may be better than histamine. Watkinson and James (1951) found that, by taking the *p*H of gastric juice aspirated at hourly intervals over the 24 hours, they could demonstrate the presence of acid often for hours in "achlorhydric" patients with gastric ulcer; during this time patients were eating their usual diet.

*Stool Examination for Occult Blood.*—Examination of the stools for occult blood provides the clinician with an important screening test in the investigation of dyspepsia, a positive reaction needing further investigation and a negative test being reviewed in the light of the patient's history. It is important to realize that negative tests may occur sometimes in the presence of active peptic ulcers. The Gregersen slide technique with benzidine is a simple and reliable test, and, provided the patient is not eating liver or dishes containing animal blood, no dietary restriction is needed.

## Special Diagnostic Problems

There are certain recurring problems in diagnosis, and these particularly concern post-bulbar duodenal ulcer, giant gastric or duodenal ulcers, and the distinction between simple and malignant gastric ulcers.

### Post-bulbar Duodenal Ulcer

These cases tend to be especially difficult to diagnose, as the duodenal bulb may fill well and distract attention from an ulcer crater which is difficult to see without careful study. Even at laparotomy the ulcer may be missed unless the duodenum is opened. Certain clinical features are to be noted. Pain tends to be in the back, occurs particularly towards the end of the day and at night, and is difficult to relieve. Bleeding is quite common, and episodes of intermittent obstruction with sudden pain and vomiting may occur.

A man aged 52 gave a three-year history of intermittent retrosternal pain going through to the back "like knives." It would occur at any time of the day and night, but particularly at night, sometimes awaking him. He would often vomit, but without relief. The pain would be severe for 20–30 minutes and then ease spontaneously. Alkalis gave little relief, but "liver salts" seemed to help. Two previous *x*-ray examinations had been reported as normal and a diagnosis of pancreatitis was suspected. Stools were negative for occult blood. When *x*-rayed again at Central Middlesex Hospital no ulcer was found: the duodenal cap was large and filled well. The examination was repeated and the diagnosis of post-bulbar ulcer was clearly established and confirmed at operation.

A man aged 38 had had a melaena four years previously and later developed severe attacks of vomiting and back pain which lasted several days, recurring six to seven times a year, with complete freedom between them. He had three negative *x*-ray examinations at different hospitals and was diagnosed as neurotic on two occasions, and as having intestinal obstruction on the third occasion. An emergency laparotomy was performed, but nothing abnormal was found. When he attended Central Middlesex Hospital, a barium-meal examination showed a duodenal ulcer in the second part of the duodenum, so he was admitted for partial gastrectomy. At operation no ulcer was apparent from the external appearances, but a crater was felt in the second part of the duodenum on the posterior wall.

The experience of this group of cases emphasizes the need to look for a post-bulbar duodenal ulcer when there are recurrent persistent symptoms of upper abdominal or back pain, and to be cautious in accepting a negative *x*-ray report.

### Giant Gastric and Duodenal Ulcers

Large simple gastric and duodenal ulcers are not rare; with large gastric ulcers having a diameter of more than 3 cm. radiologically, the probability is much in favour of a simple rather than a malignant lesion. In the admirable series recorded by Jennings and Richardson (1954) only 1 out of 17 proved to be neoplastic. The clinical picture with large ulcers is invariably atypical, and the patient may be thought to have carcinoma of the stomach or of the pancreas. There may be a considerable degree of cachexia, and a depressive state may add to the difficulties of diagnosis. Atypical pain may lead to an initial diagnosis of renal colic, fibrositis, or angina until it is realized that the alimentary tract may be at fault and a barium-meal examination is arranged. A large gastric ulcer with a short history may heal completely and a medical trial is worth while, but the prognosis is poor if there are other associated illnesses.

Large duodenal ulcers also cause diagnostic difficulties, as the size of the ulcer may superficially simulate a normal duodenal cap until close study demonstrates the loss of mucosal pattern and its consistency of size and shape in different films. Oedema around such large duodenal ulcers may cause distortion of the antrum and lead to an initial diagnosis of carcinoma of the stomach.

### Distinction Between Simple and Malignant Ulcers

A vital problem in the management of gastric ulcer is the distinction between simple and malignant ulcers. It has been stated frequently, particularly in the United States, that 10% of gastric ulcers are malignant and therefore all must be treated surgically. This bald statement of incidence is probably correct, but makes no allowance for the full use of radiology and gastroscopy in arriving at a diagnosis of a simple or a malignant ulcer. The gastric ulcer which projects clearly from the lesser curve, between the angulus and the cardia, is virtually never malignant. A follow-up study (Avery Jones, 1955a) showed that the chance of missing a malignant ulcer was under 1%.

A small point, but one of considerable interest, is the infrequency of aortic calcification in carcinoma of the stomach as compared with simple gastric ulcer. This was first reported by Elkeles (1949) and confirmed by our observations, although with lesser differences between the two conditions. It is not yet clear whether calcification is less common with carcinoma of the stomach or more common with gastric ulcer.

The distinction between simple and malignant ulcer at laparotomy can be difficult, and errors both ways are not uncommon. It would seem that *x*-ray films and gastroscopy are much more accurate aids to a correct diagnosis in the early case than inspection and palpation at laparotomy. The mistaking of malignant ulcers for simple ones at the time of acute perforation is referred to below. A wrong diagnosis of malignant ulcer may be made at laparotomy in the presence of a large simple ulcer associated with much inflammatory reaction and lymphatic glandular enlargement. A number of such cases have been seen where the surgeon was so confident of the diagnosis that no biopsy was taken, yet the follow-up has established the diagnosis of a simple ulcer.

There has been much controversy in the past about the risk of malignant degeneration of simple gastric ulcers. The experience at Central Middlesex Hospital would suggest that secondary malignant degeneration is extremely rare in a simple ulcer. With malignant ulcers the lesion has usually been malignant from the onset, but has undergone secondary peptic digestion. Alternatively, a carcinoma may have arisen independently, but adjacent to a simple ulcer. There is evidence that patients who have had undoubted simple gastric ulcers are, in fact, more liable to develop gastric cancer subsequently.

## Treatment

Does dietetic treatment influence the natural history of gastric and duodenal ulcer? The evidence is regrettably slender that real benefit is derived from the traditional dietary regime, and overtreatment can undoubtedly be positively harmful. There is, fortunately, a strong natural tendency to recovery, for which the patient tends to give the credit to his treatment. Martin and Lewis (1949) reviewed a series of 365 patients with gastric or duodenal ulcers 10 years after in-patient treatment and concluded: "It is undeniable that medical treatment relieves the discomfort of most relapses; but from our present evidence we cannot believe that it is any protection against further trouble if this is destined by the ulcer. . . . Those cases which were active persevered with their regimen and also suffered complications, while those who were inactive abandoned treatment and had no trouble." Rae and Allison (1953) reached a similar conclusion from a follow-up group of 63 men with proved peptic ulcer, kept on a careful dietetic regime for 12 months and then reviewed after five years.

Doll, Friedlander, and Pygott (1956) compared an almost normal (fried-free) diet with the standard ulcer regime in patients followed up for a year. In their series there were 64 in-patients with gastric ulcers, 80 out-patients with gastric ulcers, and 50 out-patients with duodenal ulcers. The out-patients were either advised to continue for a year on the standard ulcer diet with which they had previously been treated or were advised to revert to a wholly normal diet. At the end of the year the proportion who had remained free from pain and in whom the ulcer was radiologically healed was practically the same in both groups.

Do other measures besides ordinary dietetic care facilitate the healing of ulcers? In studying the effect of alkalis and milk drip, careful measurements concerning the rate of healing of gastric ulcer have been made with the addition of alkalis to a milk drip in sufficient quantity for the acidity of the gastric contents to be kept above *p*H 4 throughout the 24 hours. This trial (Doll, Price, Pygott, and Sanderson, 1955) demonstrated, however, that the milk drip produced a greater gain in weight—7 lb. (3.2 kg.) compared with 3 lb. 8 oz. (1.6 kg.)—and the impression was obtained that pain was relieved more rapidly with milk drip than without it, and that therefore it is a useful adjunct to the standard treatment in patients whose pain persists after rest in bed. Similar in-patient therapeutic trials have been made with phenobarbitone, ascorbic acid, and bed rest (Doll and Pygott, 1952), and with "robaden" and cabbage juice (Doll and Pygott, 1954). Two factors only have so far been demonstrated as having a beneficial influence—admission to hospital, which implies bed rest, and stopping smoking.

Our trials on out-patients with duodenal ulcer have not, unfortunately, produced any conclusive evidence of benefit from this treatment. Friedlander (1954) studied five groups of 25 duodenal ulcer subjects on "ribena" syrup, a control syrup, oral hexamethonium, olive oil, and oral methantheline, and followed them up clinically and radiologically for 15 months. Methantheline may have been of use in some cases in the initial relief of pain, but if there was any such effect it did not persist beyond the first nine months. There was otherwise no difference in the symptoms, rate of healing, or frequency of recurrence between the groups great enough to suggest a beneficial effect from any of the treatments. Even if there is no good statistical evidence for the beneficial effect of diet on the healing of ulcers, it cannot be abandoned in the acute stage when the patient is undoubtedly more comfortable on a light diet. Whether the patient is ambulatory or in bed initially, small two-hourly feeds are undoubtedly better tolerated than ordinary-sized meals, and enable him to become symptom-free more quickly. Very soon a reasonably generous basic diet may be given which

will enable the patient to maintain or gain weight. Patients who put on weight usually do well. If a milk drip has been started, it may be continued with the diet for a time in order to increase the calorie intake.

Bed rest is a most valuable measure. It probably means that the position of a gastric ulcer is altered in relation to the gastric juice and perhaps the jet of fluid from the pylorus may no longer impinge on a duodenal ulcer. It must be admitted in individual cases that it may be better for the patient to carry on at work rather than impose the added financial hardship of loss of work. Simple measures should be tried at first, designed to reduce fatigue, to increase regularity of meals, and to reduce or, preferably, stop smoking. If there is troublesome pain, bed rest is, however, necessary.

The next essential is to tackle the anxiety factor, which may seriously aggravate the disease process and retard healing. It is important to establish a good *rapport* with the patient, and this requires time, privacy, and patience, and on the whole is more easily achieved with a patient having bed rest. Once the patient feels he has a sympathetic independent person who is anxious to help in the capacity of a friend and not as a judge, he may be prepared to unburden the difficulties which too often have caused pent-up emotional stress because there has been no one in whom he could confide. The relief of such emotional tension is in itself of great therapeutic value; so often the patient has got his difficulties out of perspective and they have become much more important than they really are. This is where the independent observer can give valuable help. This approach is equally important with any other chronic illness, such as pulmonary tuberculosis and skin disease.

If there is pain, alkalis should be prescribed between each feed, using first an insoluble alkali, but if this is insufficient a mixture containing sodium bicarbonate may be used temporarily. There is no need to continue frequent alkalis after the symptoms have subsided, and the dose should then be reduced and finally stopped. Initial sedation is often very helpful.

During the past few years there has been a great spate of anticholinergic drugs. Belladonna has had a time-honoured role, and these new preparations have sought to emulate its action but with fewer undesirable side-effects. In general, the therapeutic contribution of these new drugs has not been impressive.

### Over-treatment

Can over-treatment of peptic ulcer be harmful? Clinical practice provides many examples of the undesirable effect of overdosage of dietary regimes. A proportion of patients are obsessional in their strict adherence to a medical regime, and three examples are given.

A medical student with a duodenal ulcer was being treated on a very elaborate regime. The failure of the nursing staff to adhere completely to the instructions induced a state of nervous apprehension and annoyance, and his pain persisted. When transferred to another ward the elaborate regime was stopped and he quickly became free from pain.

A hospital doctor with a duodenal ulcer had his meal-times spoilt because his diet was not contrived as perfectly as he would have wished. His symptoms ceased when his diet was stopped and he joined his colleagues in eating the ordinary hospital food.

An out-patient came to the hospital asking for a partial gastrectomy. Inquiry revealed that he had not, in fact, had any pain for five years, but he was now engaged and wished to take his girl friend to a restaurant and have ordinary meals.

It is perhaps the same obsessional nature, coupled with too blind a faith in advisers, which leads to the more serious complications of alkalosis or the milk-alkali syndrome. The risk of excess of soluble alkalis, particularly in the presence of vomiting from pyloric stenosis, is very well known. It is, however, less well recognized that excess of calcium from taking too much milk and alkali over long periods can cause a calcinosis and with it an impairment of renal function (Burnett *et al.*, 1949).

### Milk-alkali Syndrome

The first manifestation of the syndrome is usually general malaise with weakness and lethargy. Mental disturbance such as impairment of memory and confusion are common, and psychosis and coma have been reported. All cases so far reported have followed prolonged medical treatment for presumed or proved peptic ulcers, and a not uncommon presentation is that of nausea and vomiting occurring in the *absence* of pyloric obstruction. Polyuria may be a feature, and generalized severe pruritus has been a major complaint in many cases. An antipathy to milk may develop. Hypercalcaemia is consistently found, usually but not always with an alkalosis, but Burnett *et al.* pointed out that hypophosphataemia is noticeably absent and increased urinary excretion of calcium does not occur. The serum alkaline phosphatase is normal, but a raised blood urea and albuminuria are always present. Metastatic calcification may occur and nephrocalcinosis and nephrolithiasis are common findings. In advanced cases with irreversible renal damage death may occur from uraemia.

It would appear that the syndrome is precipitated by the absorption of base from the ingested milk and alkalis in excess of renal excretion with accumulation of calcium and possible magnesium in the body. Whether this can occur in the presence of previously normal renal function is still undecided. Wilkinson and Jordan (1934) suggested that antecedent renal damage is present in most cases, but Kirsner and Palmer (1942) disagree. It is known that a metabolic alkalosis can lead to renal damage, but not all the reported cases have shown an alkalosis when examined. Hypercalcaemia itself may lead to impairment of renal function, but it is a feature of the 30 cases so far reported that at no stage has an excessive renal excretion of calcium been noted. The importance of the condition lies in the fact that it is iatrogenic and reversible, the latter feature being unfortunately all too rare in chronic renal disease. A reappraisal of patients with peptic ulceration and so-called "chronic nephritis" may perhaps reveal more cases of the milk-alkali syndrome.

### Surgical Treatment

Surgical treatment of uncomplicated peptic ulcer will not be discussed in detail. In approximately half the patients with gastric ulcer and a third with duodenal ulcer persistence of symptoms and failure of the ulcer to heal earn the patient surgical treatment. The results, although generally good, do not allow complaisance and must encourage all further medical avenues of research. There is no reason for believing that a simple medical treatment will not one day be found.

## Complications

### Pyloric Stenosis

Pyloric stenosis is an inaccurate terminology, as so often the obstruction is in the duodenum. Some degree of narrowing is a common occurrence from healing of juxtapyloric and duodenal ulcers. The presence of narrowing is not necessarily reflected by clinical symptoms of obstruction or radiological evidence of delayed emptying. There can be appreciable narrowing of the pylorus demonstrated at operation in patients in whom no clinical or radiological evidence of retention has been noted. The anatomical lesion may be fully compensated by muscular hypertrophy of the stomach. The development of clinical symptoms of pyloric obstruction is often due to an exacerbation of the ulcer with increased narrowing from oedema and spasm in the presence of an already narrowed lumen.

Failure of this compensation, either from chronic narrowing or from acute or chronic obstruction, leads to evidence of gastric stasis from gastric aspiration or by delayed emptying of the stomach. An evening aspiration of more than 300 ml. or an early morning volume of over 100 ml. suggests obstruction, and so does barium in the stomach six hours after a barium meal. It is necessary to distinguish between gastric stasis and pyloric obstruction.

Gastric stasis may occur apart from pyloric obstruction and be due to hypotonia from potassium depletion, or it may occur in association with migraine. The unexpected finding of a large slow-emptying stomach may sometimes be explained by an attack of migraine coinciding with the barium meal examination.

Pyloric obstruction is most commonly due to peptic ulceration or neoplasm, but acute small marginal erosions may be sufficient to cause distressing symptoms with pain and vomiting (Kinsella, 1951). Other causes include achalasia of the pylorus, hypertrophic pyloric stenosis in adults, a congenital duodenal diaphragm, a polyp of the stomach intermittently prolapsing through the pylorus, a cholecystoduodeno-colic ligament obstructing the first part of the duodenum, the delayed effect of corrosive poisoning, or ferrous sulphate poisoning, which has also caused pyloric stenosis in children.

Pyloric obstruction can be produced by adhesions to an inflamed gall-bladder, without duodenal ulceration being present. Although the symptomatology of pyloric stenosis is usually typical, the presenting clinical picture is sometimes unusual. A number of cases have been sent to hospital as haemorrhages because of the dirty vomitus. Diarrhoea is an occasional initial symptom with pyloric obstruction.

The important recent progress in the management of pyloric stenosis is related to a better understanding of the disturbances in electrolytes. It is well known that in pyloric stenosis there is a deficiency of serum chloride ions with an excess of bicarbonate. In the past this has been attributed to loss of hydrochloric acid by vomiting or gastric aspiration, and the dangers of alkali therapy have been stressed. More recently attention has been focused on potassium depletion as a cause of alkalaemia.

Black and Jepson (1954) have shown by retrospective balance studies that there may be a deficiency of up to 800 mEq of potassium in patients with pyloric stenosis. How this arises is not entirely clear. Some potassium is lost in the gastric juice, but the concentration is much lower than that of sodium and chloride, and vomiting by itself would hardly account for such severe potassium depletion. Deficient dietary intake and renal inability to conserve potassium under the metabolic conditions obtaining in pyloric stenosis are presumably additional important factors.

When potassium is lost from the cells it is replaced by sodium and hydrogen ion. giving an intracellular acidosis. If there is an adequate supply of sodium this process can be continued until one-fifth of the intracellular potassium has been lost without serious disturbance of function. This fraction has been called the muscle labile potassium. In potassium depletion the kidneys excrete a more acid urine than seems appropriate to the serum electrolyte levels, so that in pyloric stenosis with alkalaemia a "paradoxically" acid urine may be found. The paradox is explained by the intracellular acidosis, and overall there may be a greater deficiency of basic rather than acidic ions.

The response of patients with pyloric stenosis and alkalaemia to intravenous sodium therapy is explained in part by the ability of sodium to replace potassium in the cells. The potassium becomes available to the kidneys for excretion as alkali and the alkalaemia is decreased. In the untreated case there is rarely an excess of sodium available to replace the muscle labile potassium. In this way potassium deficiency can impair the renal ability to excrete alkali without gross depletion of the body reserves. Severe potassium depletion of the muscles can occur following prolonged sodium therapy.

Sodium deficiency may be gross in pyloric stenosis; in one of the cases reported by Black and Jepson it amounted to 1,600 mEq. Serum levels and clinical dehydration are probably reliable guides to saline therapy. Potassium depletion is more difficult to assess, and normal serum levels are the rule in the untreated case. It is probably safe to assume there is potassium deficiency whenever there is a raised serum bicarbonate level. In mild cases potassium by mouth will usually correct the electrolyte deficiency. In more severe cases intravenous normal saline should be given with potassium as soon as it is clear that there is adequate renal function.

### Acute Perforated Peptic Ulcer

The mortality from this complication has fallen very considerably since before the second world war. The changing mortality is seen in Table VI. With the introduction of antibiotics and relaxant anaesthesia the mortality in operative cases fell to 4% in 1952, but this level has not been maintained since and there is still a toll of delayed deaths in the third or later weeks from haemorrhage or sepsis. Forty per cent. of deaths occurred in patients not operated upon. A third of these patients were, in fact, not diagnosed in life, the figures being:

TABLE VI.—*Acute Perforated Peptic Ulcer*, 1939–55

| Years of Admission | Operated | | Not Operated | | Total | |
|---|---|---|---|---|---|---|
| | No. | Deaths | No. | Deaths | No. | Deaths |
| 1938–40 .. | 106 | 29 (27·4%) | 1 | 1 | 107 | 30 (28·0%) |
| 1941–3 .. | 115 | 20 (17·4%) | 8 | 7 | 123 | 27 (22·0%) |
| 1944–6 .. | 135 | 12 (8·9%) | 16 | 16 | 151 | 28 (18·5%) |
| 1947–9 .. | 168 | 4 (2·4%) | 14 | 12 | 182 | 16 (8·8%) |
| 1950–2 .. | 218 | 9 (4·1%) | 12 | 10 | 230 | 19 (8·3%) |
| 1953–5 .. | 168 | 13 (7·7%) | 15 | 13 | 183 | 26 (14·2%) |
| | 910 | 87 | 66 | 59 | 976 | 146 |

| | |
|---|---|
| Undiagnosed in life .. .. .. | 24 |
| Too ill for operation .. .. .. | 36 |
| Refused operation .. .. .. | 1 |
| Elective conservative treatment .. .. | 5 |

Those undiagnosed in life presented a variety of clinical pictures—heart failure from cor pulmonale; pneumonia with pleuritic pain; unusual tachycardia after admission for severe haemorrhage. In none had there been any real clinical evidence of an acute perforation such as a sudden episode of pain or collapse, but there was usually a degree of toxaemia out of proportion to the presumptive diagnosis. The therapeutic dividends in this group are small, but it is important to remember this possibility when dealing with a patient whose degree of toxaemia is out of proportion with the presumed illness. No rigidity or tenderness may be present, and an *x*-ray examination is essential to establish the diagnosis.

Those too ill for operation are patients with peripheral circulatory failure who fail to respond to resuscitation. These are mainly patients with long-delayed admission to hospital. Early diagnosis is unfortunately sometimes prevented by the amazing fortitude of patients.

Operation has been the treatment of choice and medical management used only for patients whose perforations had obviously sealed off or whose general condition for some other reason precluded operation. Following an earlier analysis (Avery Jones and Doll, 1953) emergency partial gastrectomy has been performed particularly when there has been a history of ulcer symptoms for more than two years, and especially for perforated gastric ulcers, as this analysis had revealed that 10% of the assumed simple gastric ulcers at laparotomy were in fact neoplastic. The results since 1949 are shown in Table VII.

TABLE VII

| | No. | Deaths | Mortality |
|---|---|---|---|
| Suture .. .. .. .. | 365 | 18 | 4·9% |
| Emergency partial gastrectomy | 87 | 4 | 4·6% |

Hermon Taylor and Warren (1956) have made the practical observation that perforated acute ulcers—that is, those without a history of previous pain—may be treated safely without operation, and report on 47 patients who have recovered without complications. They confirm the value of emergency partial gastrectomy for perforated chronic ulcers.

### Haematemesis and Melaena

In my Goulstonian Lectures (Avery Jones, 1947) a survey of 649 admissions for haematemesis and/or melaena was made, and this series has now grown to 2,011 (Table VIII). The frequency of acute ulcers causing haematemesis reported previously has been further confirmed. Of 559

TABLE VIII

| Admission for Bleeding from: | No. | | % | Deaths | |
|---|---|---|---|---|---|
| Proved or probable peptic ulcer | 1,764 | | | 135 (7·7%) | |
| Chronic gastric ulcer | 340 | | 17·8 | 54 | |
| Duodenal ulcer | 625 | | 32·7 | 51 | |
| Post-operative group (previous gastro-enterostomy or partial gastrectomy) | 118 | | 6·2 | 8 | |
| Acute lesion group | 559 | | 29·3 | 14 | |
| Hiatus hernia | 34 | | 1·8 | 0 | |
| Unclassified (investigations incomplete) | 88 | | 4·6 | 8 | |
| Carcinoma ventriculi | 39 | 146 | 2·2 | 14 | 36 (24·6%) |
| Portal hypertension | 52 | | 2·7 | 15 | |
| Other causes | 55 | | 2·7 | 7 | |
| | 1,910 | | 100·0% | 171 (8·9%) | |
| Bled after Admission for: | | | | | |
| Dyspepsia | 29 | 101 | | 4 | 43 |
| Acute perforation | 38 | | | 17 | |
| General medical or surgical diseases | 34 | | | 22 | |
| Series total | 2,011 | | | 214 | |

patients in the acute-lesion group—that is, those with negative *x*-ray films—305 have been gastroscoped and 111 acute gastric ulcers demonstrated. In addition some further acute ulcers were discovered at necropsy or from the presence of minimal radiological evidence without previous dyspepsia.

Within this acute lesion group there are other causes of bleeding, such as acute gastritis, drug reactions, and traumatic ulceration from vomiting. Traumatic tearing of the oesophago-cardia junction, the Mallory–Weiss syndrome, is an important recent advance in our knowledge of the cause of haematemesis. It is well known that violent vomiting can cause a rupture of the lower oesophagus, but it is less widely known that the same trauma may cause lesser degrees of longitudinal laceration causing haematemesis (Decker, Zamcheck, and Mallory, 1953).

In the earlier series the patients were mainly treated medically to evaluate the mortality with the help of the then recently introduced drip blood transfusions and the more liberal feeding regime introduced by Meulengracht and developed in this country by Witts. It was, however, clear from the experience with these earlier series that a proportion of patients died whom surgical intervention might have been expected to help. A change in policy was considered necessary and from 1946 operation has been performed, especially on patients with known chronic ulcer whose age was over 40 and who had brisk recurrent bleeding after admission. In addition, patients with a proved or presumed acute ulcer have been operated on if there has been recurrent bleeding on two or three occasions, particularly if there was shock. There was a bias towards operation for cases with persistent pain after admission and for those whose bleeding was obviously rapid and causing marked circulatory collapse. Other factors, such as a shortage of blood of a particular group, might also have favoured surgical treatment.

In considering the problem of surgery for haematemesis and melaena, one is not concerned with the mortality of surgical intervention, for figures for surgical mortality can be very misleading. Some of the patients operated on were obviously appalling risks whose only chance of survival might have come from surgical arrest of bleeding. The proper criterion for success or failure of a surgical policy is whether the overall mortality of all admissions is influenced and also how the patients fare subsequently.

Table IX shows that changing from a medical to a surgical policy has not been accompanied by a decrease in mortality —if anything, the overall mortality has increased. The extent of emergency surgical intervention is shown in Table X.

The series is not a homogeneous one. The scales have been weighed against the later years as the proportion of patients over 60 has increased (Table XI). There has not, however, been any significant change in the proportion of chronic ulcers. Table IX does not allow for differences in age distribution, but when this is calculated there is still clearly very little change in mortality (Table XII). The differences are not significant, but the mortality has in fact decreased in the last period.

In Tables XIII and XIV the admissions during 1941–6 have been compared with those during 1951–4. In the former period extremely few were operated on; in the later period there was a firm surgical policy towards those with recurrent bleeding after admission, particularly those over 40 years and with probable chronic ulcers. Allowing for the effect of age, it will be seen that there is a definite, although numerically small, reduction in the mortality under

TABLE IX.—*Cases Admitted for Bleeding*, 1941–54; *Proved or Probable Cases of Peptic Ulcer*

| Year | No. of Cases | No. of Deaths | Mortality Percentage | |
|---|---|---|---|---|
| 1941 | 52 | 5 | 5·7 | Medical policy |
| 1942 | 71 | 2 | | |
| 1943 | 92 | 4 | 6·6 | |
| 1944 | 106 | 9 | | |
| 1945 | 124 | 8 | 7·9 | |
| 1946 | 118 | 11 | | |
| 1947 | 91 | 9 | 7·4 | Surgical policy |
| 1948 | 113 | 6 | | |
| 1949 | 136 | 9 | 9·5 | |
| 1950 | 117 | 15 | | |
| 1951 | 203 | 13 | 7·4 | |
| 1952 | 188 | 16 | | |
| 1953 | 179 | 16 | 7·9 | |
| 1954 | 174 | 12 | | |
| Total | 1,764 | 135 | | |

TABLE X.—*Extent of Emergency Surgical Intervention*, 1947–54

| Site | No. of Admissions | No. of Emergency Operations | Percentage of Admissions | No. of Partial Gastrectomies Performed | Deaths |
|---|---|---|---|---|---|
| Chronic gastric ulcer | 233 | 44 | 18·8 | 43 | 6 |
| Duodenal ulcer | 432 | 41 | 9·5 | 39 | 14 |
| "Post-operative" group | 80 | 6 | 7·5 | 4 | 3 |
| Acute lesions | 373 | 21 | 5·6 | 16 | 4 |

TABLE XI.—*Proportion of Patients Over 60 Years*, 1941–54

| Age | 1941–6 | 1947–51 | 1952–4 |
|---|---|---|---|
| Over 60 | 33% | 38% | 40% |

TABLE XII.—*Mortality Rate*, 1941–54

| | 1941–6 | 1947–50 | 1951–4 |
|---|---|---|---|
| Observed deaths | 39 | 39 | 57 |
| Expected ,, | 38·6 | 35·58 | 60·82 |

TABLE XIII.—*Mortality by Age and Period of all Male and Female Cases Admitted for Bleeding (Peptic Ulcer Group)*

| Age | 1941–6 | | | 1951–4 | | |
|---|---|---|---|---|---|---|
| | Admissions | Deaths | Mortality | Admissions | Deaths | Mortality |
| Under 60 | 379 | 13 | 3·4% | 453 | 9 | 2·0% |
| Over 60 | 184 | 26 | 14·1% | 291 | 48 | 16·5% |
| Total | 563 | 39 | | 744 | 57 | |

TABLE XIV.—*Mortality by Age and Period of Male and Female Cases Admitted for Bleeding (Peptic Ulcer Group)*

| Age | Site | 1941–6 | | | 1951–4 | | |
|---|---|---|---|---|---|---|---|
| | | Admissions | Deaths | Mortality % | Admissions | Deaths | Mortality % |
| Under 60 | Chronic gastric ulcer .. | 63 | 9 | 14·3 | 72 | 4 | 5·6 |
| | Duodenal ulcer | 141 | 3 | 2·1 | 192 | 2 | 1·0 |
| | Acute lesions | 121 | 0 | 0·0 | 151 | 1 | 0·7 |
| | Post-operative group .. | 28 | 1 | 3·6 | 20 | 2 | 10·0 |
| Over 60 | Chronic gastric ulcer .. | 44 | 11 | 25·0 | 66 | 14 | 21·2 |
| | Duodenal ulcer | 52 | 12 | 23·1 | 87 | 19 | 21·8 |
| | Acute lesions | 63 | 3 | 4·6 | 79 | 5 | 6·3 |
| | Post-operative group .. | 10 | 0 | 0·0 | 22 | 2 | 9·1 |

60, but over 60 the mortality was correspondingly increased. These results may be interpreted in two ways. It may be said that since the introduction of surgery in a proportion of the more severe cases with continued or recurrent bleeding the overall mortality has not changed significantly, and the same proportion of patients leave hospital as when a policy of energetic transfusion and other medical measures only were used. On the other hand, it may be said that certain groups have significantly benefited from a surgical policy, especially patients under 60 with recurrent bleeding and those patients with a chronic gastric ulcer. It may also be argued that there has, in fact, been some benefit to the group as a whole, as the later years have a higher age distribution and therefore should carry a higher mortality; and, again, the mortality from partial gastrectomy is already included in the overall figure. It must also be remembered that this analysis has covered only proved or probable cases of peptic ulcer and there are other possible causes, although rare ones, for severe gastro-intestinal haemorrhage which may require surgical intervention, as, for example, simple tumours of the stomach or intestine, diverticulitis, vascular lesions, etc.

The unchanged mortality cannot be accepted at once as proving that the surgical policy should be reversed and that one should return to general medical measures in nearly every case. Certain sub-groups of patients have a better prognosis with surgical rather than medical care. The situation has also to be reviewed with reference to the follow-up. There is a responsibility not only for ensuring that the maximum number of patients leave hospital alive, but also that the maximum possible number remain alive and well after a period of years. A follow-up analysis has demonstrated very poor late results in those treated medically as compared with those who had had emergency partial gastrectomy (Avery Jones, 1956). The situation has also to be assessed in relation to the state of the blood bank. During the second world war there were ample supplies of blood available in civilian hospitals, held in reserve against air-raid casualties, but to-day pressure is constantly brought to bear by the pathologists to reduce demands on blood. This naturally encourages a bias towards surgery. There are still some physicians who prefer not to seek surgical assistance, but the balance of evidence is in favour of selective emergency surgery.

Our present policy is to continue a surgical bias knowing that the arrest of bleeding by partial gastrectomy in those patients with severe continued or recurrent bleeding can be undertaken without causing an increase in the overall mortality and that it offers a better outlook for the patient in the future. Our practice is as follows.

In patients with a known history of chronic gastric ulcer there is a strong bias towards a partial gastrectomy at some stage during the admission. In a few patients it may be decided to operate within the first twelve hours even if there is no further bleeding, and this applies particularly to poor-risk patients who might have difficulty in withstanding the strain of a further sharp bleed. If brisk bleeding occurs, or if there is obvious continued bleeding after admission, then an emergency partial gastrectomy is performed within a few hours and after further blood transfusions have been given. In the remaining cases, careful consideration will be given to the need for an elective operation before leaving the hospital. The general inclination is towards more surgery in this group and at any early phase of admission.

With a known history of duodenal ulcer, the tendency is towards less emergency surgery and more elective operations, but operating immediately if there is severe continued bleeding or severe recurrent bleeding with shock. The same policy holds for those who have had previous operations for duodenal ulcer.

Patients with a history suggestive of chronic peptic ulcer but in whom the site of the crater is uncertain are gastroscoped the same evening or the following morning. Aspirations of gastric juice may be made for *p*H estimations.

Patients with a short history only—less than two weeks' discomfort—or in whom there has been no previous dyspepsia, are gastroscoped, usually on the next regular gastroscopy session or after a recurrent haemorrhage. If there is bleeding and if shock recurs twice in patients thought to have an acute lesion, the stomach is opened and an attempt made to determine the bleeding-point by digital palpation. The usual procedure then is a partial gastrectomy in men and possibly a submucosal resection of the ulcer in women. If no cause for the bleeding is found, a partial gastrectomy is performed and a further search made of the fundus before the final anastomosis is made.

In patients with evidence of pyloric stenosis an elective operation is planned, if possible after one to two weeks, when the electrolytic balance has been restored.

Patients who have an acute perforation after admission for haemorrhage, or those with simultaneous bleeding and perforation, have an immediate laparotomy, when, if possible, a partial gastrectomy is performed.

Patients admitted with an acute perforated ulcer treated by simple suture who then bleed again after admission have a laparotomy with a view to an emergency partial gastrectomy.

## Conclusions

The increase in duodenal ulcer noted previously has continued, although the incidence of gastric ulcer seems to be stationary. Gastric and duodenal ulcers behave differently when analysed statistically in relation to many factors such as blood groups, age, incidence, social class, occupation, and heredity, and it seems probable that they represent independent disease processes.

The cause of gastric and duodenal ulcer remains unknown. Acute erosions and ulcers are common occurrences, and it seems likely that anti-healing factors exist which promote chronicity.

The diagnosis of peptic ulcer, although often simple, may at times prove difficult, and there are important fallacies in relation to radiological and other methods of diagnosis.

The evidence that dietetic treatment influences the healing of gastric and duodenal ulcer is unconvincing. Bed rest and cessation of smoking seem to be beneficial. There are dangers inherent in overtreatment, medical and dietetic.

Better appreciation of the role of potassium depletion has greatly improved the management of pyloric stenosis.

There is still scope for reducing the mortality from acute perforation. A proportion of cases have atypical clinical pictures and may escape detection. There is a role for immediate partial gastrectomy in patients with chronic ulceration, causing perforation.

The introduction of emergency partial gastrectomy for bleeding ulcer has not lowered the overall mortality, but nevertheless there are good reasons for undertaking it in selected cases.

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Three leprosy clinics have been opened in Sokoto Province, Northern Nigeria, by Dr. C. M. Ross, Senior Specialist in Leprosy for the Northern Region. Discharge certificates were recently presented to 23 people in the province who had been examined and found free from leprosy. These are the first discharges in the province, and are an indication of the amount of control which is now being gained over the disease. The Minister of Health for the region has recently announced plans for the building of a leprosy training school at Kaduna to continue the fight against the disease.

# RESULTS OF SURGERY IN TREATMENT OF CANCER OF THE LARGE INTESTINE

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In recent years the attitude of surgeons towards cancer of the large bowel has become increasingly more agressive. Formerly a large proportion of the patients suffering from this disease were regarded as inoperable, and if, as was often the case, the growth lay in the rectum or distal colon they were treated by colostomy alone. Experience has shown, however, that a simple colostomy of this kind seldom afforded any real palliation, and all too often its only effect was to add the inconveniences of an artificial anus to the patient's existing discomforts. Though it overcame any obstructive element it did little to allay the other distressing features of an inoperable growth—the incessant spurious diarrhoea with discharge of slime and blood, the occasional profuse and alarming haemorrhages, the severe sacral or sciatic pain, and the development of fistulae into other organs or externally. This is not surprising, for these symptoms all spring directly from the continued presence of the growth in the bowel and can only be obviated by its removal. It is therefore clearly desirable, where possible in treating cancer of the large bowel, to excise the primary growth for palliative purposes alone quite apart from any question of cure.

Fortunately, despite their well-known predilection to become fixed to adjacent organs or to the pelvic or abdominal walls, carcinomata of the rectum or colon can generally be excised by a really determined operation, comprising, if necessary, resection of implicated adjoining viscera or parietes. Pathological examination of the operating specimens thus secured shows that the binding adhesions often contain no actual growth but are purely inflammatory in character, so that the prognosis is frequently much better than was anticipated at the time of operation, and an excision which was undertaken essentially for palliation sometimes turns out to be curative. Even if the growth should eventually reassert itself the patient may often enjoy two or three years of comfortable and useful life before this occurs. There is thus everything to be said for excision if at all possible, and indeed with almost no other form of malignant disease does really extensive surgery pay such handsome dividends as it does with cancer of the large bowel.

In his pursuit of this more ruthless policy the surgeon is, of course, much assisted by the greatly enhanced safety of surgery since the end of the second world war, due to better blood-transfusion facilities, improved anaesthesia, a clearer understanding of the problem of fluid and electrolyte balance, and the advent of antibiotics for systemic and intestinal use. As a consequence there must now be few patients, even amongst the elderly frail individuals who represent such a large proportion of the sufferers from cancer of the large