PEPTIC ULCER

of the

OESOPHAGUS.

Ulceration of the oesophagus may be caused by various things, and can be divided into tuberculous, syphilitic, malignant, and simple. There are many causes for the simple type such as the action of corrosives, a foreign body such as a sharp bone, the pressure of an aortic aneurism, the acute exanthemata, a traction diverticulum and, in the upper part of the organ, there is the very typical so-called "decubitus ulcer" caused by the pressure of the cricoid cartilage in a very ill and weak patient. In our present study we are not dealing with any of these, but only with one particular type which has been called peptic ulcer of the oesophagus. This ulcer is situated in the lower part of the organ, just above the cardiac sphincter, and has no apparent reason for its presence, although as the name suggests it is considered that the gastric juice plays some part in its causation. An ulcer of the lower end of the oesophagus which extends into the stomach for any distance should only be included in this group after very careful consideration, for in most of such cases this will be found to be an ulcer of the fundus of the stomach which has secondarily involved the lower end of the gullet.

Peptic ulceration of the oesophagus may be divided into acute and chronic types. The acute type is really the same as antemortem digestion of the oesophagus, and, as I shall later suggest, many of the chronic cases probably start as antemortem digestions, or acute peptic ulcers which are prevented from healing by certain disturbing factors. As is the case in the stomach and duodenum, so in the oesophagus it is impossible to draw a clean-cut line

between acute and chronic peptic ulceration, for we consider that the one stage merges insensibly into the other. Bolton (1) and Ivy (2) in their work on experimental gastric ulcer showed that these, if not too large, are epithelialised within 3 weeks of the initiation of the lesion, but that complete restoration of the mucosa requires a week or two longer. Obviously larger lesions will require a correspondingly longer period of time so that the time factor is really no criterion of chronicity except in that the larger the ulcer at the beginning, the greater will the tendency be for it to become chronic. Stewart and Hurst (3) differentiate them in the stomach by (a) the depth to which they have penetrated the wall, and (b) the type of inflammatory reaction in the floor and margin of the ulcer. In the case of the oesophagus depth is no use in our differentiation for, whereas in the stomach the acute ulcer does not invade the muscular coat as a rule, in the oesophagus acute peptic ulcer may commonly destroy all the coats and actually cause rupture into the pleural cavities. The better age criterion in the case of the oesophagus is that depending upon the cellular and vascular changes in the floor and margin of the ulcer. In the acute lesion the cellular reaction is of the polymorphonuclear and later of the lymphocytic type, there being a much more abundant cellular reaction than in the case of the stomach, where it is often marked by its absence. In the chronic lesion the leucocytic reaction is confined to scattered mononuclear cells, whereas fibroblastic proliferation is marked and is found to be replacing the muscular

layers to a varying degree.

We shall now describe acute and chronic peptic ulcer of the oesophagus separately, but later attempt to correlate them in discussing the etiology of the chronic type.

A. ACUTE PEPTIC ULCER.

By acute peptic ulceration, or antemortem digestion of the oesophagus as the condition is more commonly known, we mean an acute ulcerative process in the lower part of the organ which has no obvious causation such as trauma or the action of swallowed corrosive, but which appears to have some relationship to the action of gastric juice. The condition was first referred to by Kaufmann (4), and later by Pringle and others (5). Cases were later described by Lyall (6), and Polson (7). It is to be noted also that many of the cases described by Tileston (8) in his article on peptic ulceration of the oesophagus are really cases of antemortem digestion, although he groups them with the cases of chronic ulceration as one group.

Pringle and his co-workers (5) described 18 cases of the condition which they had observed over a period of 7 years and they thus concluded that the lesion was not an extremely rare one. The acute ulcerative lesion affects the lower end of the oesophagus and usually ceases abruptly at the cardiac orifice, but fades off in an upward direction. As already mentioned, the name of the condition suggests an association with gastric juice, and in fact, it is thought to be caused by the action of very acid gastric juice on the affected area of oesophagus. These patients appear frequently to have a high acid value in their gastric juices, and may complain that their vomitus burns their mouths and lips.

The most important symptom is the vomiting of black, or dark-brown material in a patient who is extremely devitalised from illness, frequently after an operation. The colour of the material is due to changed blood, and in fact fresh red blood may at times be actually vomited, but this is rare. The material is brought up frequently, and in small quantities, and in some cases where the patient is too devitalised even to vomit, the typical material may be found in the stomach. Sometimes there is a complaint of pain low down between the shoulder blades, or beneath the lower part of the sternum. In the less severe cases this may only be complained of when food is being swallowed and is then described by the patient as being most uncomfortable and having a "tearing" quality and coming on just before the food reaches the stomach.

We have seen 20 cases of this interesting condition over the past 5 years. Pringle and his co-workers saw their 18 cases over a period of 7 years. In their series they found that 10 of the cases were surgical ones, 6 medical, and 2 were accidents. Of the 10 surgical cases 6 were those of acute appendicitis.

We have divided our series into operation and non-operation cases. They are set out in the following table:-

SURGICAL CASES (Operation performed).		No. of Cases
Perforated duodenal ulcer	..	3
Gangrenous appendicitis	..	2
Strangulated herniae	..	2
Acute mastoiditis	..	1
Suprapubic cystostomy	..	1

	No. of Cases
Bilateral acute suppurative parotitis ..	1

OTHER CASES

Gastric ulcer with inanition and terminal perforation ..	1
Chronic phthisis; Gastric ulcer ..	1
Carcinoma: (gall bladder - 1 (rectum - 1 ..	2
Abortus fever ..	1
Diabetes with carbuncle of kidney ..	1
Cerebral haemorrhage ..	1
Acute nephritis ..	1
Congestive cardiac failure ..	1
Tuberculous peritonitis ..	1

Of our series we found that 10 of them, i.e. 50 per cent., were cases where an operation had been recently performed. In 8 of the cases sepsis in some gross form was present, and in 5 of the cases there was either gastric or duodenal ulceration found; and in 3 of these the operation was performed for perforation of the ulcer, which was of the duodenal type. Thus, in our series the important etiological factors would appear to be the presence or effects of (a) Operation; (b) Sepsis; (c) Chronic ulceration of the stomach or duodenum, especially the latter with probably concomitant hyperchlorhydria.

We shall not attempt to describe in detail all the cases we

have seen, but shall describe certain of them in order to illustrate the various degrees of the process, and shall add one case which we feel indicates healing of the lesion and is, of course, not included in our series of "post-mortem" cases.

CASE I This case illustrates the very earliest stage of the process which we have yet seen, and resembles a first stage burn in that acute congestion with only superficial destruction of the epithelium was present.

D- W- aged 59, Clerk, was admitted to the medical wards suffering from congestive cardiac failure. The patient responded for some weeks to treatment, but later went steadily downhill and a few days before death became delirious, and died 3 months after admission. For the 3 days before death he was extremely restless and could only be fed with difficulty. On some occasions during this time he vomited some clear fluid material, but this was by no means a marked feature.

The post mortem showed the presence of marked enlargement of the heart due to chronic endocarditis of the aortic and mitral valves. Other organs showed evidence of chronic venous congestion.

The lower third of the oesophagus showed congestion which ceased abruptly at the lower end, but faded off in the upper part. Here and there the epithelium was thinner than normal, but no actual ulceration was visible.

Histological examination showed marked congestion of

the sub-mucous layer of the oesophagus (fig. 1), and at areas large thin-walled vessels lay immediately under the epithelium (fig. 2). The epithelium was thinned and beginning to break away at certain places (figs. 3 and 4). At these areas some degree of leucocytic infiltration was seen at times (figs. 5 and 6) At a few areas the epithelium had completely disappeared and abundant leucocytic infiltration was apparent here (fig. 7). The leucocytes were mainly lymphocytes and mononuclears with a smaller number of polymorphs.

CASE 2 This is the ordinary type of case in which the whole mucous membrane has been destroyed, but the necrotic process has not penetrated the muscularis mucosae.

Mrs. S- aged 48 years, was operated upon for a perforated duodenal ulcer 7 hours after the onset of symptoms. The perforation was found in the first part of the duodenum, and was sutured. The case progressed favourably for 3 days when the patient had a slight haematemesis. From then till her death, 2 days later, she was almost constantly sick and brought up small quantities of "coffee-ground" material. Just before death she vomited about one pint of black material.

At the post mortem "kissing" ulcers were found in the first part of the duodenum, and that on the anterior wall had perforated and been efficiently sutured. No bleeding vessel was found in the bases of either of them.

The lower 8" of the oesophagus showed a black discoloration which ceased in a straight line at the cardiac sphincter of the stomach, but faded off gradually in an upward direction where the colour became brown rather than black. The discoloured area was ulcerated and had the typical appearance of antemortem digestion, the dark appearance being due to the soaking of the necrotic tissue with haemolysed blood. A small quantity of "coffee-ground" material was present in the stomach. Sections from the affected area of oesophagus showed complete destruction of the mucous membrane which was replaced by polymorphonuclear leucocytes closely packed together, and many of them showing degenerative changes (fig. 8). Superficial to this, and giving the oesophagus its dark coloration naked eye was a thin layer of altered blood pigment (fig. 9). At places complete necrosis of the superficial layers of the oesophagus had taken place and leucocytic infiltration and thrombosis of vessels was observed here (fig. 10). The cellular infiltration extended down to the muscularis mucosae, but rapidly faded off deep to this. Many of the vessels contained large numbers of polymorphs which were emigrating into the tissues (figs. 11 and 12). Examination of the epithelium at the upper part of the oesophagus showed that the line of destruction here was very irregular and at its edge had a very frayed appearance (fig. 13). The muscular coats were normal.

CASES 3 and 4 These cases illustrated a very advanced degree of

the condition with complete destruction of the oesophageal wall and perforation of the friable necrotic organ into the pleural cavities.

Case 3 Mrs. B- aged, 21, Housewife, was admitted to hospital suffering from hyperemesis gravidarum. While under treatment with fluid diet she developed an acute suppurative parotitis on the right side, and in a few days the left side was also involved. Multiple punctures were made into the inflammatory areas. On the day following operation she became delirious, and died in a short time.

At post mortem multiple small pyaemic abscesses were found in the lungs, and the other organs showed the changes associated with severe sepsis.

The lower end of the oesophagus showed antemortem digestion of very advanced degree. The lower third was black and friable, and tore upon slight handling (fig. 14). At its left side the organ had ruptured into the pleural cavity and free gastric juice was present in the left pleura. The base of the lung which was bathed in the fluid was haemorrhagic and some fibrinous deposit lay on the pleural surface. In an upward direction the lesion faded off into a brownish discoloration of the oesophagus, and then finally in an irregular manner into intact mucous membrane. Whereas the upper limit of the ulceration was indefinite, the lower end lay at the cardiac sphincter and was very definite and clean cut.

Histologically, tissue from the lower, most advanced area of ulceration, showed simply necrosis with no cellular reaction. In the upper less affected areas, however, much polymorphonuclear infiltration was observed. Sections of the affected lung showed the presence of haemorrhage and pneumonia. The vessels were markedly congested and many had ruptured into the lung tissue (fig. 15). At other parts the bronchi and air sacs were filled with polymorphonuclear cells (fig. 16). Large numbers of phagocytes, some with dark pigment, were seen at areas mixed with desquamated endothelial cells and polymorphs and surrounded by congested vessels (fig. 17)

Case 4 N- S- aged 52, Labourer, was admitted to the surgical wards with a perforated duodenal ulcer, which was successfully sutured. The patient progressed very well for 7 days when he developed pain high up in the epigastrium and accompanied by vomiting which became dark brown in colour and contained altered blood. The vomiting of quantities of this dark, altered blood continued at frequent intervals, and the patient became progressively paler and more collapsed. Death took place 6 hours after the beginning of his "brown" sickness.

At post mortem, the duodenal ulcer which lay on the anterior wall of the first part of the duodenum was found to be efficiently sutured and no bleeding vessel lay in its base. The whole of the oesophagus, except the area immediately

beneath the cricoid cartilage, showed the presence of antemortem digestion. The condition was most marked in the lower end where it ceased abruptly in a straight line at the cardiac sphincter, and had destroyed the whole oesophageal wall so that perforation into both pleural cavities had occurred. In its upper limit the condition faded off as a series of black streaks into intact mucosa. Gastric juice lay in both pleural cavities and the posterior parts of the bases of the lungs showed black discoloration where they had been bathed in the acid juice. Incision into the lungs at these sites showed the presence of much haemorrhage, the appearance suggesting a haemorrhagic infarction.

Histologically the vessels of the affected area of lung showed marked congestion, and at parts they had ruptured, causing haemorrhage (fig. 18). A few polymorphs were present, but these were few in number.

The lung appearances in these 2 cases are interesting. They suggested that the gastric juice had caused an inflammatory reaction in the affected parts, and had also eroded the vessels, causing haemorrhage into the alveoli. In the latter case in which the oesophagus had probably ruptured a short time before death, haemorrhage and congestion were all that were found. In the former case, obviously of older standing, an inflammatory exudation of leucocytes was abundantly present.

CASES 5 and 6 These cases illustrate a healing stage of the condition. The whole mucosa over the affected area had not been destroyed, and the islets left were undergoing proliferation and becoming rounded off in appearance. If the patients had not died of the primary disease the condition would very likely have gone on to complete healing.

Case 5 Mrs. M- aged 28, was admitted to the medical wards complaining of faintness, giddiness, and sickness. This had come on the day before admission and been accompanied by vomiting. Upon admission she was found to be very anaemic and had a haemoglobin of 40%. She stated that the material vomited on the day before admission had been dark in colour, but after admission no further vomiting occurred. Melaena was present on admission, and later occult blood persisted for 12 days. After this the patient progressed rapidly, and in 3 weeks her haemoglobin was 60%. Five weeks after admission she developed sudden abdominal pain, became collapsed, and died the following day.

At the post mortem a perforated duodenal ulcer was found with an early acute diffuse peritonitis. No bleeding vessel was found in the base of the ulcer.

The lower third of the oesophagus was denuded of its epithelial lining and had a light-brown colour which appeared to be due to old blood staining. The affected area was covered by multiple small light-coloured islets of epithelium which

were rounded off at their edges and measured from 1 mm. in size upwards (figs. 19 and 20). At places many of these were fusing together, and elsewhere the areas were longer and rather linear in appearance, and lay in the sulci between the rugae at the lower end of the oesophagus. All of the patches were rounded, and had the appearance of proliferating epithelial rests.

Histologically the appearances were those of ulceration with no acute reaction present, but with some mononuclear infiltration persisting at places. Patches of epithelium remained in the ulcerated area and showed evidence of proliferation growing out at their edges into thin layers which were composed mainly of the basal layer of the stratum mucosum (fig. 21). At some parts where apposing edges of epithelium were growing together, the appearance of a healing wound of the skin was got (fig. 22).

Case 6 Mrs. P- aged 61, was admitted to hospital with persistent vomiting which had lasted for some days, and been accompanied by abdominal pain. While in hospital she vomited frequently, and each time brought up small quantities of material which had often a dark colour, and resembled changed blood. A laparotomy was performed, and a tuberculous peritonitis found to be present. Fluid in the peritoneum was allowed to escape, and the abdomen closed. Following the operation the vomiting became less, and ceased, and the patient

started to improve. Eleven days afterwards she suddenly collapsed, and died.

At the post mortem, tuberculous disease of the small bowel and tuberculous peritonitis, were found. Death was actually due to a large pulmonary embolism which had come from the right femoral vein. The lower third of the oesophagus showed loss of mucous membrane, with granulation tissue occupying the areas of denuded mucosa. The mucosa left consisted of rounded patches resembling blobs of white paint, and had the appearance of intact islets which were in a state of proliferation designed to cover the area which had been denuded (fig. 23).

Histologically the ulcerated areas showed the persistence at places of leucocytes which were mainly of the mononuclear class. The persisting patches of epithelium showed evidence of proliferation growing at their edges into thin wedges of cells which were one or two cells thick at the outermost parts, and in many areas composed chiefly of the basal layer of the stratum mucosum (figs. 24 and 25). Here and there mitotic figures were seen near the growing edges of the epithelial islets.

These 2 cases show a healing stage of the process, and suggest that the name "antemortem digestion" is not really correct as death may not occur; for in these cases recovery

would undoubtedly have taken place but for other more serious conditions which became superimposed - in one a perforation, and in the other a pulmonary embolism. Healing was obviously much more advanced in the first than in the second case, and this corresponds with the history, for in the first case the length of time after the acute phase was probably about 5 weeks, and in the second case about 12 days.

CASE 7 This is a case which was clinically an antemortem digestion of the oesophagus which went on to healing over a period of weeks.

J- L- aged 24 years, Postman, developed an acute tonsilitis for which Tinct. Ferri Perchlor. mixture was prescribed. The patient stated that his stomach was easily upset, and that the medicine caused him to be sick after he had swallowed it. After 4 days of illness a quinsy developed on the right side, and the patient became very miserable and ill. At this stage he vomited some watery material mixed with blood, and thereafterwards for some days, vomited at intervals small quantities of "coffee-ground" material. Antemortem digestion of the oesophagus was suspected, and the patient was given a half-teaspoonful of alkaline powder every hour, with small quantities of Liquid Paraffin in order to act as an emollient. The quinsy burst spontaneously, and the patient then commenced to improve rapidly. The vomiting ceased, but he then complained of

pain low down between the shoulder blades when he swallowed either liquid, or solid foods. The pain was not severe, but was extremely uncomfortable, so that the patient was almost afraid to take food. This slightly improved over the succeeding days, but for 6 weeks after his illness the patient continued to have discomfort and dysphagia on swallowing food. The condition cleared up, and the patient has been perfectly well for the past 3 years.

PATHOLOGY

The cases described illustrate very well the different stages of the process. Like burns of the skin, antemortem digestion of the oesophagus may be divided into stages.

Stage I This corresponds to a first degree burn of the skin where there is simply redness or congestion of the mucous membrane.

The vessels in the submucosa show marked dilatation. The superficial layers of the epithelium are becoming desquamated and leucocytes may be seen collecting under the thinned patches (figs. 1 - 7).

Stage II This corresponds to a third degree burn, as there is apparently no stage of blistering of the oesophagus in antemortem digestion to correspond to a second degree burn. At this stage the mucous membrane is destroyed over an area of the lower part of the gullet leaving, however, small undamaged islets of

epithelium from which regeneration may later take place.

Stage III The mucous membrane over the area has been completely destroyed leaving no islets of epithelium. The ulcerated area is dark in colour from haemolysed blood, and ceases abruptly in a straight line at the cardiac sphincter, but fades off in an upward direction.

Microscopical examination at this stage shows marked evidence of vital reaction in the form of polymorphonuclear emigration to the affected area, and the vessels of the area may be crammed with these cells (figs. 8 - 13).

Stage IV This corresponds to the last degree of a burn, where the whole part is charred. In the oesophagus the wall is completely destroyed, and rupture into one, or both pleural cavities has occurred. The lower third of the organ is black and friable, and tears on the slightest manipulation.

It is perhaps in these cases more than in any others that the absolute localisation of the process to the oesophagus and the escape of even the fundus of the stomach, impresses itself upon the pathologist. Histologically the only evidence of vital reaction in the affected part of the oesophagus may be extravasated blood, and leucocytic reaction is often absent till the upper reaches of the organ are examined, where the damage is much less intense.

Pringle and his co-workers (5) also observed this fact and suggested that the sudden and extreme nature of the

damage prevented any vital reaction occurring. The resemblance to a very severe burn is again apparent.

Stage V This is the stage of healing. The black, or dark-brown colour of the part becomes lighter, and a light-brown discoloration of the affected area appears to persist for some time. The polymorphs in the affected area largely disappear and are replaced by a few mononuclears and lymphocytes. Any intact islets of epithelium commence to proliferate to repair the breach, and that at the periphery begins to grow inwards.

Where the damage to the oesophagus has been that of the third stage and the organ completely denuded of mucosa over the affected area, regeneration of epithelium will require to take place from the periphery alone.

I have already mentioned that the process involves the lower part, usually the lower third of the oesophagus, and extends right down as far as the cardiac sphincter, completely involving the mucosa right down to the part where it undergoes a transition to gastric mucous membrane. Thus, in many cases regeneration will require to take place from above alone, and the obvious difficulty of this in an organ constantly being disturbed by the passage of food, and possibly the regurgitation of acid gastric juice, will be appreciated. I shall refer to this later when discussing the etiology of chronic peptic ulcer of the oesophagus.

At this stage it may be well to differentiate between antemortem and postmortem digestion of the oesophagus. Postmortem digestion has been long recognised, and the stomach is usually affected to a corresponding degree. It is, of course, due to the digestive action of the gastric juice on the tissues after death. The oesophagus in the condition has a pale appearance, and the epithelium is white, soft, and shreddy. The important naked-eye feature in differentiating it from antemortem digestion, is the absence of extravasated blood. Histologically the differentiation is easy. In postmortem digestion the surface of the organ is homogeneous with no cell outline visible. There is no evidence of congested vessels, extravasated blood, or leucocytic infiltration: in other words, there is complete absence of the features which we speak of in a cumulative way as "vital reaction" (fig. 26). In antemortem digestion all these features are present (fig. 27).

ETIOLOGY

It is fairly generally agreed that antemortem digestion has some relationship to the digestive effect of the gastric juice. Postmortem digestion of the oesophagus and stomach is well known, and has already been referred to.

Pringle and others (5) supposed that antemortem digestion arose through the vomiting of very acid gastric contents, and that the oesophagus failed to empty itself completely, so that some of the fluid accumulated in the lower

third, being kept there by spasm of the sphincter. This closure of the cardiac sphincter explained the sudden cessation of the process at the extreme lower end of the organ. I feel that the effect of the acid gastric juice in causing the lesion is undoubted, but I feel less certain about the so-called sphincter spasm. The lesion occurs usually in patients who are seriously ill, and in these the gastrointestinal sphincters are more likely to be relaxed than in spasm. The more important point I feel is not so much that the process ends at the cardiac sphincter, but rather that it ceases where the oesophageal epithelium changes to gastric mucous membrane.

The gastric mucosa has an inherent chemical and physical immunity against the corrosive and digestive action of the acid gastric juice during life. This is not so with the oesophagus, which is no more immune against the effect of acid juice than is the skin.

In our series we have noted 3 important features in the cases: the presence of sepsis, of operation, and in an appreciable number, the presence of peptic ulceration in the stomach or duodenum.

Moutier (9) has described 3 cases of acute oesophagitis in women following the administration of an anaesthetic - ether alone in two, and associated with nitrous oxide in the third. He considered that the lesion was produced during the initial stage when each violent inspiration was followed by the deglutition of the irritating vapour. The symptoms developed

after 24 hours when attempts were made to feed the patients.

In his cases the lesion appeared to be in the upper part of the oesophagus mainly, and he shows a possible way in which the vitality of the gullet may be lowered by anaesthesia. In advanced sepsis the cells of the parenchymatous organs particularly, but also those of other parts of the body to a lesser degree, show cloudy swelling which would very definitely lower the vitality and resistance to a nocuous agent such as very acid gastric juice.

The inference to be drawn from the presence of gastric and duodenal ulceration in many of the cases is obvious, and readily explains the very acid nature of the vomited material which scalds the throat and lips in these cases. Thus, we suggest that the lesion is caused in a patient limp from illness, with relaxed sphincters so that gastric juice may readily regurgitate into the oesophagus which has its mucosa devitalised by the giving of an anaesthetic or distant sepsis. Once the process is started and the protected mucosa has been desquamated, degree is simply a matter of time, and thus the final stage may even be reached where the excavation penetrates the whole oesophageal wall and causes rupture into the pleural cavity.

TREATMENT

The logical treatment would appear to be the giving of small doses of alkali at frequent intervals in order to

neutralise the very acid gastric juice which is present in such cases. Emollient substances such as Liquid Paraffin, Gum arabic solution, or Olive Oil, might be swallowed in an attempt to soothe the inflamed and ulcerated mucosa. Any spasm of the oesophagus might be allayed by Belladonna.

B. CHRONIC PEPTIC ULCERATION.

Chronic peptic ulcer of the oesophagus would appear from the literature to be an extremely rare lesion.

Stewart (10) stated that he had met only one case of this interesting condition in a series of 10,000 autopsies performed over a period of 18 years. Miller (11) saw only 2 cases of the disease during his 12 years' experience as Pathologist to large city hospitals. Tileston (8) described 3 cases of peptic ulceration of the oesophagus and collected other 41 cases from the literature, bringing the total up to 44. In this series, however, he has included both the acute and chronic lesion, and the report of one of his own cases is as follows, and is typically that of antemortem digestion, or acute ulceration of the gullet - "The lower end of the oesophagus for a distance of 11 cm. is denuded of mucous membrane, except for a few small islands, and is of a dirty, brown-yellow colour. The ulceration is very shallow, not invading the muscularis."

We agree with Stewart (10) that certainly no more than nine of his 44 cases can be definitely considered as cases of chronic ulceration.

Zenker (12) denied the existence of peptic ulcer of the oesophagus and claimed that these simple ulcers were due to some definite cause as a traction diverticulum, cancer, etc. Quincke (13) published an account of the condition with a report of 4 cases, and by a very careful histological

examination of the material, left no doubt about the simple nature of the lesion, and in fact, established the condition as a very definite entity. It is very interesting to note that Dr. Finlayson (14) in 1882 showed a specimen of a simple perforating ulcer of the oesophagus opening into the left bronchus and causing gangrene of the lung. He mentioned that the specimen was illustrative of the simple ulcer of the oesophagus exactly resembling that of the stomach. He also referred to a case reported by Professor Flower in the Medico Chirurgical Transactions for 1853, in which case the perforation took place into the aorta, and to cases reported by Part and Wilks. Interesting case accounts have also been given by Chiari (15), Debove (16), Ortmann (17), and Kraus (18).

More recently Chevalier Jackson (19) by the routine use of the oesophagoscope in all cases of slight dysphagia, claimed to have found 21 active ulcers and the scars of 67 in a series of 4,000 cases. His paper contains excellent reproductions of the oesophagoscopic appearances. His work has been criticised, very rightly, we think, by quite a few who have concluded that many of the ulcers described by him were really acute ulcers undergoing healing, and should not have been included in the category of the much rarer chronic ulceration.

Friedenwald, Feldman and Zinn (20) came to the same conclusion as Jackson about the relative frequency of the

condition, and published 13 cases from their own practice in which the condition was diagnosed by oesophagoscopy and X-ray examination. Eusterman, Moersch and Camp (21) reported 3 cases, the ulcer being situated at the junction of oesophagus and cardia in one, and at the lower end in the other two, causing partial stricture. Aurelius (22) gave a description of the condition, and Hurst (23) reported 2 cases.

Owing to the confusion caused by many writers failing to distinguish between the acute and chronic lesion, it is very difficult to arrive at a definite idea as to the number of cases of chronic peptic ulceration described up to date. Stewart and Hurst (3) in their handbook recorded a total of 19 cases. Adding the cases described since that time the total up to date must be no more than 35.

We have seen 13 cases of this rare and interesting condition over the past 8 years. We shall give an account of these, and then discuss their pathological appearances and etiology.

CASE REPORTS

Case I. - Mrs. O'H., aged 75 years, was admitted to hospital on Jan. 14, 1936, with signs of acute obstruction of the bowels. She was operated upon as an urgent case and a caecostomy performed. Cellulitis of the surrounding abdominal wall developed, and death took place from sepsis on Feb. 13, 1936. The patient stated in her history that she had had periodic attacks of indigestion for many years, but these had never been severe. There was no dysphagia.

Post-mortem Report. - The body was that of a woman of medium build and rather poorly nourished. A collection of pus was present between the layers of the abdominal wall in the region of the operation wound. The abdominal cavity showed only some adhesions binding the terminal ileum to the brim of the pelvis. On the anterior and posterior walls of the stomach, close to the middle of the lesser curvature, were chronic ulcers. That on the anterior wall had muscle in its base and showed marked fibrous induration around. That on the posterior wall was slightly less advanced. Each measured 1.5 cm. in diameter.

The oesophagus was opened from behind. It was normal in calibre, but at its lower end was an area of chronic ulceration extending completely round the tube. It measured 2.5 cm. in length and its lower edge coincided with the cardiac sphincter except at its left side, where it extended as a longitudinal ulcerated area measuring 2 by 1 cm. into the cardiac end of the stomach. The upper edge of

the oesophageal ulcer was very poorly defined and faded off irregularly. The lower edge was more sharply seen. In the base of the ulcer the circular muscle of the oesophagus was apparent as transverse markings. Some thickening of the tissues outside the oesophagus was apparent. Besides the chronic ulcer there was also present a late antemortem digestion of the oesophagus showing some evidence of attempts at healing. The whole of the lower two-thirds of the tube was completely denuded of epithelium, there being simply the submucosa remaining stained light-brown with changed blood. Between this and the upper normal third of the tube was an area containing small rounded islets of epithelium which were obviously growing in an attempt to cover the denuded area.

Histological examination of the ulcerated area showed the appearance of chronic ulceration with some irregular fibrosis extending into the muscular coat of the oesophagus. The upper part of the oesophagus showed antemortem digestion, and at parts a thin growing edge of epithelium was seen showing attempts at healing.

The post-mortem diagnosis was cellulitis of the abdominal wall following caecostomy; chronic ulceration of the oesophagus; antemortem digestion of the oesophagus.

Case 2. - Mrs. C., aged 51 years, was admitted to hospital on May 25, 1935, complaining of distension of the abdomen and marked breathlessness. Examination showed a very marked ascites, and

multiple tumour masses were felt in the abdomen. Paracentesis was frequently performed, but the patient slowly went downhill and death took place on Nov. 17, 1935. There was no history of indigestion, and while in hospital the patient was fed on light diet with no particular symptoms to suggest an oesophageal lesion.

Post-mortem Report. - The body was that of a rather spare woman. The left pleural cavity contained 500 c.c. of slightly blood-stained fluid; the right contained 600 c.c. of thin purulent material with a few flakes of fibrin. Both lungs were normal. The peritoneal cavity contained 9 litres of blood-stained fluid. The peritoneal surfaces were studded with nodules of tumour tissue which had spread from a carcinoma of the uterus. The stomach and intestines were normal.

The oesophagus was opened from behind. At its lower end was an ulcer extending almost completely round it. It started a fraction of a centimetre above the cardiac sphincter and extended upwards for a length of 5 cm. On the right posterior aspect, about the middle of the ulcerated patch, was a roughly circular area of much deeper ulceration, which extended completely through the muscular coats. At its upper edge the ulcer ceased in an irregular circinate manner, so that close observation was necessary to make out the line of demarcation. At its lower end it ceased in a very distinct, almost straight line. Some induration of the tissues around was felt. On separating the ulcer from the adjacent mediastinal tissues, the base gave way at its deep excavated part. No direct

communication was found between the ulcer and the right pleural cavity, but the septic condition of the tissues in the region of the deepest part of the ulcer suggested that the inflammatory process had spread from here through the mediastinal tissues to the right pleural cavity.

Histological examination showed the appearances almost exactly resembling those of chronic gastric ulcer. On the surface of the ulcer was a thin layer of necrotic debris mixed with fibrin; beneath this a layer containing some leucocytes; and below this a layer of fibrous tissue, the uppermost part of which contained some congested vessels. The deepest part of the base contained very mature fibrous tissue (fig. 28).

The post-mortem diagnosis was carcinomatosis of the peritoneum; chronic ulceration of the oesophagus; acute pleurisy.

Case 3. - Mrs. B., aged 55 years, was admitted to hospital on Feb. 1933, with a haematemesis. For four years the patient had been troubled with periodic attacks of indigestion. Each attack lasted for a period of from a few days to three or four weeks, and were separated by relatively free periods. The pain appeared immediately after the taking of food and was referred through to the back, being felt between the shoulder blades. Flatulence was troublesome during the attacks. No vomiting had occurred until a week before admission to hospital, when her appetite disappeared and she commenced to vomit greenish, mucoid material. High epigastric pain was present at this time and became much worse whe

she attempted to take food. On the morning of the day of admission she felt very faint and drank 2 drachms of brandy. She immediately felt very sick and vomited some dark blood-stained material. A short time later she vomited some fresh blood and was admitted to hospital. After admission she vomited about 5 oz. of clotted blood mixed with mucus. Examination showed nothing of note apart from slight tenderness in the epigastrium. In 1919 the patient had had oöphorectomy performed, and in 1929 had had an abscess in the lower abdomen drained. Her teeth had all been removed some time previously.

Three days after admission the patient developed a pneumonic consolidation of the right lung. Blood-culture showed the presence of Type II pneumococcus. Death took place on Feb. 18, 1933.

Post-mortem Report. - The body was that of an elderly, well-nourished woman. An old midline scar was present in the lower abdomen. The cardiac muscle showed cloudy swelling. On the posterior cusp of the mitral valve were some recent vegetations of the ulcerative type. The upper lobe of the right lung was in the condition of grey hepatization. The stomach showed a certain degree of hour-glass contraction which was due to a transverse scar 5 cm. in length lying about the middle of the lesser curvature. No active ulceration was present.

The oesophagus was opened from behind. At its lower end was a chronic ulcer which extended completely round the tube and measured 5 cm. in length. Its lower edge was fairly well defined and

corresponded to the cardiac sphincter. The upper edge was irregular and faded off into intact mucosa. Circular muscle showing areas of cicatrization was seen in the base. No bleeding vessel was found. The oesophagus above was of normal size.

Histologically the ulcer showed chronicity in the form of fibrous induration, extending for some distance outwards (figs. 29, 30). Some endarteritis of vessels was apparent. Section of the gastric scar showed a line of well-formed old fibrous tissue.

The post-mortem diagnosis was chronic ulceration of the oesophagus; haematemesis; lobar pneumonia.

Case 4. - Andrew J., aged 71 years, was admitted on April 16, 1936, complaining of pain in the abdomen of two months' duration. The pain was situated in the epigastric region and was of an aching character. It tended to appear especially in the evening and was relieved by alkaline powder. The pain had no definite relationship to food, but the patient's appetite had been very much poorer than normal since its onset. There was no vomiting at any time during the illness up to the time of admission. The bowels were somewhat irregular, and on one occasion a week or two before, the patient noted that the stool had a very black colour. Since the beginning of his illness the patient had lost over 2 st. in weight. His past health had always been very satisfactory and he did not remember having ever been previously confined to bed. Upon admission the patient showed evidence of having recently lost a good deal of weight. His jaws were edentulous and his tongue dry and furred.

No epigastric tenderness was present. A blood examination showed the presence of a marked secondary anaemia, the cell-count being red blood-corpuscles 2,220,000, white blood-corpuscles 5,400, the Hb 32 per cent. and the colour index 0.72. After admission the patient periodically vomited small quantities of brown blood-stained material. On April 29, 1936, he vomited 500 c.c. of altered blood and soon afterwards collapsed and died.

Post-mortem Report. - The body was that of a male subject with a fairly large frame but showing much emaciation. The skin was very pale. The heart showed slight hypertrophy of the left ventricle. Both lungs showed marked oedema posteriorly. The prostate showed marked enlargement, due to adenomatous hyperplasia, and the urinary bladder showed hypertrophy and dilatation. The stomach contained 300 c.c. of dark-red blood-clot. No ulceration was present in the stomach or duodenum and the blood had obviously come from the oesophageal ulcer.

In the lower 8 cm. of the oesophagus was an area of ulceration extending completely round the organ. At its upper end the ulcer extended upwards as two rounded areas with a tongue-shaped process of mucous membrane running downwards for a short distance between them. On its right side the ulceration was fairly superficial, and circular muscle could be seen in its base at places. On this side the ulcer faded off above into intact mucosa, the exact line of demarcation being very indefinite. On its left side the ulcer was much deeper over an area measuring 5 cm. in length and 2.5 cm. in

breadth. The edges of this deeper area of ulceration were very steep in their inferior parts, but showed some terracing in the upper part. Near the lower edge of this part was an oval, very deeply ulcerated patch measuring 0.8 by 0.3 cm. which had as its base the intima of the aorta; and on holding the specimen up to the light a series of small cracks could be seen through which oozing of blood had taken place (figs. 31, 32, 33).

Histological examination showed the appearances of chronic ulceration with much fibrous induration.

The post-mortem diagnosis was chronic ulcer of the oesophagus eroding the aorta.

Case 5. - Bernard McA., aged 53 years, was admitted to hospital on Dec. 29, 1932, suffering from haematemesis of four days' duration. For thirty years the patient had suffered from indigestion. The attacks lasted for weeks or months at a time and were separated by free intervals, which had lately been becoming shorter. Epigastric pain appeared from an hour to an hour and a half after the ingestion of food and lasted for a variable period, being frequently relieved by the taking of more food. Vomiting at first had been infrequent, but had been becoming more frequent during the preceding four years. Upon admission the patient was found to be an emaciated man who looked older than his years. He continued for some days to vomit "coffee-ground" material and to pass changed blood per rectum. As he was going downhill rapidly it was decided to cauterize the bleeding ulcer. On Jan. 11, 1933, a large blood

transfusion was given and, thereafter, the abdomen opened under local anaesthesia. "Kissing" ulcers were found on the anterior and posterior walls of the first part of the duodenum. That on the posterior wall was bleeding and was cauterized. A posterior gastro-enterostomy was then performed. Death took place a few hours after operation.

Post-mortem Report. - The body was that of an emaciated, elderly looking man. On the anterior and posterior aspects of the first part of the duodenum were chronic ulcers measuring 1.5 cm. in diameter. The floor of the posterior one was formed by pancreatic tissue. Slight pyloric stenosis was present and the stomach was a little hypertrophied and dilated. At its lower end the oesophagus was adherent to the tissues outside. It was opened from behind. In its lower end towards the right side was an area of chronic ulceration. This was roughly circular, measuring 3.5 x 4 cm., with edges which were rather serpiginous except the lower one, which was clean cut and ran in an almost straight line transversely across the lower end of the oesophagus. At its right side this edge lay just above the cardiac sphincter, but towards its left sloped slightly upwards from it. Growing from this edge was a small sprouting mass of what looked like granulation material. Compared with this lower edge the upper and lateral ones were indistinct and faded off into intact mucosa. The ulceration was comparatively superficial in its upper part and circular muscle was exposed in its base. In the central and

lower parts, however, the process had penetrated much deeper, cicatricial fibrous tissue lying in the base. The oesophagus above the ulcer appeared slightly dilated and hypertrophied, but showed no other abnormality (fig. 34).

Histological examination of the ulcer showed the appearances of chronic ulceration, there being much fibrous tissue formation with small collections of round cells. Endarteritis of vessels was seen near the surface.

The post-mortem diagnosis was chronic duodenal ulcers; haematemesis; chronic ulcer of the oesophagus.

Case 6. - John K., aged 69 years, was admitted on Feb. 7, 1935, complaining of pain in the upper abdomen and vomiting of six weeks' duration. For many years the patient had been troubled with indigestion, which was marked by pain coming on at a variable period after food, flatulence, and water-brash. Six weeks before admission his condition became very much more urgent. The pain and especially the vomiting became very severe, the former having no definite relationship to food, and becoming, if anything, worse after it was taken. The patient had had pneumonia in 1916 and acute cholecystitis in 1933. He was a moderate smoker and used to drink a large quantity of alcohol. This had, however, ceased some months before admission. The abdomen was slightly scaphoid and the skin dry and loose, suggesting fairly recent loss of weight. The tongue was moist with a slight white fur. All the teeth had previously been extracted except two which appeared healthy. Slight

tenderness was present over the whole abdomen. There was some anaemia present of the secondary type, the red blood-cells being 3,030,000 and the white cells 5,800 per c.c. The Hb was 60 per cent. A gastric analysis showed complete absence of hydrochloric acid, with a total acidity slightly above normal. X-ray examination after a barium meal showed a moderate amount of dilatation of the stomach, but no evidence of ulceration. During the patient's residence in hospital melaena was almost constantly present. He improved slightly under treatment, but on Feb. 28, 1935, he developed signs of internal haemorrhage from which he died on the same day.

Post-mortem Report. - The body was that of a poorly nourished looking man. The stomach was very greatly dilated, with hypertrophied wall. Immediately beyond the pylorus was a deep chronic ulcer on the posterior wall of the duodenum 2 cm. in diameter and with over-hanging steep edges. The base was formed by pancreatic tissue, and the ends of several small arteries were visible. Much fibrosis extended from the ulcer into the pancreas and lesser omentum. Black altered blood was present in the ileum and a small quantity was also found in the colon. The sigmoid loop showed long-standing diverticulitis. The gall-bladder contained twelve faceted gall-stones of the mixed type.

The oesophagus was opened from behind. Its mucous membrane was very pale in appearance. There was an area of ulceration involving the lower 5 cm. of the organ and running completely round it. The

lower edge of the area lay just above the cardiac sphincter and was fairly well defined. The upper edges faded off in a series of ill-defined arcades. In this superficial ulceration were three longitudinal areas of much deeper excavation, each measuring about 1.5 cm. in breadth and showing cross striation in its base due to exposure of circular muscle fibres. At their lower edges, i.e. near the cardiac sphincter, these areas were specially deep but became much shallower above. It was the upper edges of these areas which caused the arcade-like appearance already mentioned. The wall of the oesophagus in the region of the ulceration was much thickened, fibrosed, and adherent to the structures around, so that separation was effected only with difficulty. Above the ulcer the oesophagus showed marked hypertrophy and dilatation (figs. 35, 36).

Histological examination of the ulcer showed it to have the characters of a chronic ulcer involving the circular muscle coat at most places, which showed varying degrees of fibrosis with collections of round-cells here and there.

The post-mortem diagnosis was chronic ulcer of the duodenum with haemorrhage; chronic oesophageal ulcer.

Case 7. - John M., aged 71 years, was first admitted to hospital on Aug. 18, 1926, complaining of loss of appetite, sickness, and vomiting of six weeks' duration. Except for occasional slight attacks of indigestion the patient had had good health previous to this time. For the first few weeks of his illness the vomiting had

occurred about one and a half hours after food, but had gradually been getting much worse, and just before admission, was occurring ten minutes after each meal. It was present even when he was on light diet. Abdominal pain was absent except for a slight feeling of discomfort in the upper abdomen. He had lost 1½ st. in weight during the six weeks of his illness. The patient admitted that he had previously taken a fairly large quantity of alcohol every week. He smoked 3 oz. of tobacco per week. Upon admission the patient was found to be a rather thin elderly man. There was evidence of recent loss of weight. A neoplasm of the stomach was suspected, but X-ray examination of the gastro-intestinal tract was negative. He was put on light diet, his sickness ceased, and he commenced to put on weight. He was discharged on Sept. 16, 1926, feeling very well and with no return of symptoms.

On Sept. 1, 1931, the man was readmitted to hospital. He stated that shortly after his discharge five years before his stomach had recommenced to trouble him. This took the form of periodic attacks of epigastric pain and vomiting, the pain appearing at a variable period after food, often coming on soon after its ingestion. He found that heavy foods such as potatoes and meat aggravated his symptoms and he had been avoiding these. For the five days preceding admission the symptoms had become very urgent, the pain in the epigastrium being very severe, and hiccup was almost constantly present. The day before admission he vomited a quantity of "coffee-ground" material. Examination showed the patient to be

a rather spare, elderly man. Constant hiccup was present. The patient had no teeth. The breath was foul and the tongue thickly coated with a dirty, brownish fur. Deep pressure in the epigastrium elicited some tenderness but no mass was palpable. The lungs and urinary and nervous systems were normal. The patient appeared a little confused and gave only a very indefinite account of the symptoms. He improved under very light diet, and on Sept. 8 the sickness had ceased and the hiccup was only occasionally present. He was being given 3 pints of milk and a ½ pint of Benger's food daily. On admission the stools contained obvious melaena, but on Sept. 12 it was only found on careful chemical examination. Examination of vomited material on Sept. 13 showed free hydrochloric acid (titrated with N/10 NaOH) 50; total acidity, 78. On Oct. 19, 1931, the improvement had persisted with the result that patient's mental condition was much clearer. He complained that his food stuck near the lower end of his sternum after he had swallowed it. An X-ray examination after a barium meal was carried out and the report was "there is obstruction of the oesophagus, the appearance being highly suggestive of neoplasm". An oesophagoscopic examination was carried out on Dec. 2, 1931. No carcinoma of the oesophagus or fundus of stomach was seen. The walls of the oesophagus appeared rather closer together than normal, suggesting an extrinsic pressure on it, but no decisive finding was obtained. The patient was now put on 5 min. of tincture of belladonna three times daily.

This improved his oesophageal condition and he was able to swallow his food normally after this. On Dec. 7, 1931, he was discharged from hospital feeling very well.

On Aug. 30, 1932, the patient was admitted to hospital for the third time. He complained of haematemesis and epigastric pain of one day's duration. He said that he had been keeping fairly well since his discharge from hospital less than a year previously. Four days before his present admission he developed epigastric pain accompanied by marked nausea. Alkaline treatment only helped him slightly. On the day preceding admission the pain was very much more severe and the patient vomited brownish material which contained blood. Examination showed him to be a moderately well nourished elderly man. His complexion was florid and his mucous membrane of good colour. He complained of vague high abdominal pain but no rigidity was present. Soon after his admission he had a copious motion which contained altered blood. While in hospital he had various attacks of vomiting, the vomitus consisting of partly digested food. On Sept. 11 he had two attacks of haematemesis which weakened him considerably. He also complained of pain high up in the epigastrium shooting through to the back. On Sept. 27 it was noted that he continued to vomit about once daily and that the vomited material always contained some altered blood. This vomiting appeared to be unaffected by any medicinal treatment. His general condition became poorer, he became confused mentally, and died on Nov. 5, 1932.

Post-mortem Report. - The body was that of an adult man of rather

poor nutrition. The heart and lungs showed no abnormality apart from senile degenerative changes. The stomach showed slight dilatation, but no ulceration, recent or old, was present. No other abnormality was found in the abdomen.

Externally the oesophagus felt greatly thickened in its lower half and was firmly adherent to the tissues outside, so that it could not be completely separated from them. It was opened from behind. The lower two-thirds of the organ showed very marked thickening of its wall due to replacement by firm fibrous tissue, and measured 7 mm. in thickness at parts. The mucous membrane was completely absent in the lower half. The ulcerated area ceased abruptly in a straight line at the cardiac sphincter, but at its upper part it faded gradually off into intact mucosa, there being a 'transitional' area where islets of epithelium started to appear, showing some attempt at healing. The base of the ulcer consisted mainly of cicatricial fibrous tissue, but at parts the cross striation of the circular muscle coat was apparent. Two small perforations into vessels were found in the middle of the ulcerated patch. Blood exuded from the lower one on slight pressure. Just outside the oesophagus, on the left side 3 cm. above the cardiac sphincter, was an enlarged gland the size of a bean containing much anthracotic pigment (figs. 37, 38).

Microscopical examination of a piece of tissue taken from the middle of the ulcer was carried out. The remains of longitudinal muscle were present at one end of the section. It showed a

well-marked increase of fibrous tissue between its fibres. The fibrous tissue was dense and hyaline at parts but became more cellular and of more recent origin nearer the surface. Many of the vessels showed marked endarteritis obliterans (fig. 39). There was no evidence of malignant or syphilitic change. In the fibrous tissue deep to the muscular coats numerous lymphoid follicles with prominent germ centres were seen.

The post-mortem diagnosis was chronic ulcer of the oesophagus with haemorrhage.

Case 8. - Patrick C., aged 58 years, was admitted on June 8, 1935. For years his stomach had troubled him, there being an indefinite history of pain after food accompanied by occasional vomiting. An X-ray photograph was taken, the suspected diagnosis being peptic ulcer; but no abnormality of the stomach or duodenum was seen. For some months before admission the patient's disability had become extreme. He was afraid to take food because of the pain, had become very neurasthenic, and had taken to alcohol as a means of relieving the pain. There was the occasional vomiting of "coffee-ground" material. Anaemia had become very marked, and he was admitted to hospital with a condition of spasm of the vessels of the fingers resembling Raynaud's disease. Upon admission the patient was very weak and breathless. He admitted having drunk half a bottle of whisky an hour or so before admission. He died fairly suddenly

four hours later.

Post-mortem Report. - The body was that of a middle-aged male in the last stages of emaciation. In the posterior aspect of the lowest lobe of the right lung was an area of red hepatization. A less advanced degree of pneumonia was also present in the middle lobe. All the organs of the body showed evidence of inanition. The marrow of the femur showed a well-marked erythroblastic reaction.

Marked thickening was present in the tissues around the lower end of the oesophagus, so that carcinoma was suspected on external examination. The organ was opened from behind. At its lower end, 1 cm. above the cardiac sphincter, was a deep oval ulcer, measuring 5.5 by 4 cm. almost completely encircling the circumference of the organ and leaving only a small patch of intact mucosa 1.5 to 2 cm. in breadth. The edges of the ulcer were very steep and overhanging at parts. The base was firm and infiltrated, and at its left side was composed of the pleural surface of the left lung, which showed some fibrosis at this part. Gentle manipulation broke down the adhesions binding the ulcer to the pleura, causing a direct perforation into the left pleural cavity, showing how the patient had been living on the verge of a precipice for some time. Closer examination of the mucous membrane in the region of the ulcer showed the presence of a remarkable state of affairs. The intact mucosa separating the lateral edges of the ulcer was found to be heterotopic gastric mucosa which extended as a tongue-shaped

process of well-preserved tissue upwards from that of the fundus of the stomach (fig. 40). The two important features were the close resemblance of the ulcer to the typical chronic ulcer of the pyloric region of stomach or first part of duodenum and the presence of the heterotopic mucosa.

Microscopical examination showed marked fibrosis spreading from the base of the ulcer, with complete destruction of the muscle coats in the neighbourhood. On the surface of the ulcer was a thin layer of necrotic material infiltrated with polymorphs and fibrin. Deep to this was a fairly cellular granulation tissue with many fibroblasts. Beneath, this fibrous tissue became more dense and avascular (fig. 41). The heterotopic gastric mucosa bore a resemblance to that found normally towards the pyloric end of the stomach, the glands being fairly short and wide. Oxyntic cells were present, but were comparatively few in number.

The post-mortem diagnosis was chronic peptic ulcer of the oesophagus; advanced inanition: lobar pneumonia.

Case 9. - T- B-, male, aged 51, Unemployed Labourer, was admitted to the medical wards with haematemesis. For many years he had suffered from indigestion, which had been specially severe for the preceding year, and characterised by pain coming on about 1½ hours after food. The pain was localised to the epigastrium, but shot through to the back between the shoulder blades. The haematemesis started on the morning of the day of admission, when he vomited about one pint of blood. After admission the patient

had smaller haematemeses for some days afterwards, although melaena of fairly marked degree persisted while the patient was in hospital, and he became more anaemic and weakly in spite of treatment. Suddenly, 4 weeks after admission, he developed a specially severe attack of abdominal pain, and died in a few hours.

At the post-mortem examination a chronic ulcer measuring 3 by 2 cm. was found at the pyloric end of the stomach. It lay on the anterior wall, and in its base was adherent gall bladder. At one part perforation into the general peritoneal cavity had occurred, and through this gastric contents were escaping. Two eroded vessels from which blood could be expressed lay in the floor. The stomach contained a large quantity of thick, altered blood, and somewhat similar but darker material was found in the small and large intestines. The mediastinal tissues around the lower end of the oesophagus were infiltrated and adherent.

On opening the oesophagus the lower 5 cm. were found to show a diffuse ulceration which ceased very abruptly at the cardiac orifice, but faded off imperceptibly in an upward direction. One area measuring 3 x 2 cm. and situated at the extreme lower end of the ulcerated patch was very deeply excavated and had penetrated the whole muscular wall, and showed mediastinal tissue in its base.

Histological examination of the ulcer showed the presence of fibrous induration in the base replacing the superficial part of the

circular muscle in the greater area of the ulcerated patch, but extending deeply and going outwards into the mediastinal tissues in the deeper area. The ulcer was obviously of some standing.

The post-mortem diagnosis was chronic gastric ulcer; haematemesis; perforation; chronic peptic ulcer of the oesophagus.

Case 10. - A- A-, aged 57 years, Clerk, was admitted to the surgical wards complaining of acute pain in the abdomen which had been present for 12 hours. For the past 1½ years the patient had suffered from attacks of indigestion which were marked by the onset of epigastric pain at a variable period after food, and at times accompanied by vomiting. The pain was situated in the upper epigastrium and extremely severe at times. Relief was got by taking baking soda on occasions; but at other times this appeared to have no beneficial effect. For about 5 weeks before admission to hospital the pain had been almost constantly present after every meal, and been accompanied by the frequent vomiting of green material. Twelve hours preceding admission it had become extremely severe and had made the patient breathless and almost afraid to move in bed.

Upon examination marked rigidity and tenderness were found in his upper abdomen. The air intake of both lungs was limited, but this was thought to be secondary to the abdominal lesion. A perforated duodenal ulcer was diagnosed, and operation performed. At operation evidence of a chronic duodenal ulcer was found, but no perforation or other acute abdominal lesion. Death took place

on the following day.

At the post-mortem examination the body was found to be that of a well-nourished, male subject. In the first part of the duodenum "kissing" ulcers were found, each measuring about one cm. in diameter. Both showed evidence of marked chronicity, being surrounded by many adhesions. At the lower end of the oesophagus was a chronic peptic ulcer measuring 3 x 2 cm. in size (fig. 42). The edges were steep and rounded, and in the base lay the longitudinal muscular coat. At one part of the base a small perforation went through into the right pleural cavity, and associated with this there was an acute pleurisy with much fibrinous deposit and a small quantity of free pus in the pleural cavity.

Histological examination of the ulcer showed the presence of fibrous tissue formation in the base replacing the longitudinal muscular layer at areas. The circular layer of muscle was completely gone at the site of ulceration.

The diagnosis was chronic duodenal ulcer; chronic oesophageal ulcer; perforation into the right pleural cavity; acute pleurisy.

Case 11. - W. McA-, aged 32 years, Blacksmith, was admitted to the medical wards complaining of indigestion over a period of 5 years. During this time the patient had suffered from flatulence and heartburn after meals, with occasional attacks of vomiting. The pain came on at a variable period after food, and

frequently appeared soon after a meal. Relief was obtained by taking alkaline powder. The vomited material was usually food he had taken, but about 7 months before his admission he had vomited blood-stained material. About 6 months before admission the patient had started to suffer from intermittent dysphagia, and an X-ray examination soon afterwards disclosed an obstruction near the lower end of the oesophagus, the appearances suggesting carcinoma. Distension of the abdomen developed soon after this, and when the patient was admitted gross ascites was present. The abdomen was periodically tapped and clear fluid obtained. Dysphagia and vomiting continued after admission in spite of treatment. The vomited material contained large quantities of changed blood. Anaemia and weakness slowly increased, and death took place 6 months after he had first complained of dysphagia.

At the post-mortem examination the body was found to be that of a fairly well nourished, young male subject. In the lower part of the oesophagus was an area of chronic ulceration measuring about 8 cm. in its longitudinal length. At the lower end it terminated very sharply at the cardiac orifice of the stomach, but at its upper part had faded off irregularly into normal mucosa (figs. 43 and 44). In the lower part of the ulcer longitudinal muscle was seen and in its middle area circular muscle was exposed. A few bleeding points were seen in the base. Some hypertrophy of the oesophagus above the area of ulceration

was noted. On the right side of the affected part of the organ a gland the size of a marble was found, and incision into it showed the presence of necrotic tumour tissue.

Microscopical examination of the ulcer showed the presence of chronicity with fibrous induration of the base and muscular layers. It was found that malignant disease had become superadded, and had spread to the enlarged gland already mentioned. The malignant condition was a carcinoma of the spheroidal celled type, and was composed of clumps of cells of varying size (figs. 45, 46, 47), which at places had spaces in their centres giving the appearance of adeno-carcinoma (fig. 48). The fibrous tissue of the base of the ulcer was being involved at places (fig. 48). Mitotic figures were abundant. The tumour had the appearance of that typically got in the stomach and bowel, but only rarely found in the oesophagus, and appeared to be of fairly high malignancy.

The diagnosis was chronic peptic ulcer of the oesophagus with carcinoma superimposed; haemorrhage from the ulcer.

Case 12. - D- D-, aged 22 years, Forester, was admitted complaining of abdominal pain and vomiting which had been present for about one year. A large quantity of pus was found in the urine, and pyelography showed the presence of a calculus in the pelvis of the left kidney. This organ was exposed, and found to be hopelessly destroyed by suppuration, and was excised.

Pathological examination showed the presence of advanced

suppuration due to the presence of a calculus. Two weeks after operation the patient developed pain in the right side of the chest accompanied by a cough, and brought up large quantities of foul-smelling sputum, which was found to be due to an abscess in the upper lobe of the right lung. The patient became weaker, and 2 weeks after the onset of the complication he coughed up a quantity of blood, and died in a few hours.

At the post-mortem examination the body was found to be that of a rather spare young man. On the posterior wall of the first part of the duodenum, 3 cm. from the pylorus, was the scar of an old ulcer. The remaining right kidney showed some hypertrophy, but was otherwise normal. The lower part of the oesophagus showed a diffuse ulceration which ended sharply at the cardiac sphere, but faded off irregularly in the upper part. Circular muscle was exposed in the base at most parts, but deeper areas of ulceration were visible, and one at the lower area of the ulcerated patch measured 3 x 3 cm. and showed longitudinal muscle exposed in its base. Fibrous thickening was present around the ulcerated part.

Histological examination showed the presence of chronic ulceration with fibrosis, but at parts areas of polymorphonuclear infiltration were visible suggesting a more acute ulcerative process on top of an older one. In the upper lobe of the right lung was an abscess cavity occupying most of the lobe and with very irregular edges. Associated with this the right pleural

cavity contained a large quantity of thick pus, and much fibrinous deposit was present on the pleural surfaces.

The post-mortem diagnosis was pyelonephritis; lung abscess; empyema; chronic peptic ulcer of the oesophagus.

Case 13. - R- McF-, aged 68 years, was admitted to the surgical wards complaining of acute abdominal pain. For some time before the patient had suffered from indigestion with pain coming on about half an hour to an hour after food, accompanied at times by flatulence and heartburn. Four hours before admission to hospital he was seized with sudden upper abdominal pain of great severity. When admitted he was found to have marked tenderness and rigidity in the epigastric region, the appearances resembling a perforated gastric or duodenal ulcer. Operation was performed, but no abnormality was got in the abdominal cavity. Death took place 5 hours later.

At the post mortem the body was found to be that of a well-nourished, elderly male subject. A moderate degree of cardiac hypertrophy with recent dilatation was present. Marked hypertrophy of the median lobe of the prostate was observed, and associated with this the bladder showed hypertrophy with trabeculation, and both kidneys showed moderate hydronephrosis. At the lower end of the oesophagus, just above the cardiac orifice, was an elongated ulcer with its long axis lying vertically, and measuring 1 x 2.5 cm. The edges were slightly thickened, and in its base lay longitudinal muscle fibres. At one area the

ulcer opened into an abscess cavity the size of a walnut which lay in the mediastinum (fig. 50). This cavity in turn had burst into both pleural cavities, which showed acute inflammatory change. In both there was a quantity of thin, slightly blood-stained, purulent fluid containing numerous fibrinous threads.

Histologically the ulcer showed fibrous tissue formation in its base replacing the muscle layers and suggesting chronicity.

Some recent acute inflammatory reaction with polymorphs spreading between fibrous tissue layers was also found, and the appearances suggested chronic ulceration with recent acute spread.

The diagnosis was chronic peptic ulcer of the oesophagus; mediastinal abscess; perforation into both pleural cavities; double empyema.

The following Table shows the main features of the cases described.

Sex	Age	Symptoms	Oesophagus	Gastric or Duodenal Ulceration	Cause of Death
Female	75	Periodic attacks of indigestion.	Chronic ulcer with antemortem digestion.	Chronic gastric ulcers.	Cellulitis of abdominal wall after caecostomy.
Female	51	No history of indigestion.	Chronic ulcer more acute at one part.	None	Carcinomatosis of peritoneum, with terminal mediastinitis and empyema due to oesophageal ulceration.
Female	55	Indigestion for 4 years: haematemesis on admission.	Chronic ulcer.	Scar of old ulcer across lesser curvature.	Lobar pneumonia following haematemesis from oesophageal ulcer.
Male	71	Indigestion for 2 months.	Chronic ulcer of rounded type.	None	Haemorrhage from eroded aorta.
Male	53	Indigestion for 30 years.	Chronic ulcer of rounded type.	Chronic ulcers of duodenum.	Death following operation on duodenal ulcers.
Male	69	Indigestion for many years.	Chronic ulcer.	Chronic ulcer of duodenum.	Death due to haemorrhage from duodenal ulcer.

Sex	Age	Symptoms	Oesophagus	Gastric or Duodenal Ulceration.	Cause of Death
Male	76	At first like gastric condition. Later dysphagia.	Very chronic ulceration with marked fibrosis.	None	Death from constant bleeding from oesophageal ulcer.
Male	58	Indigestion for many years, much pain latterly. Very neurasthenic and alcoholic because of pain.	Typical peptic ulcer of oesophagus with heterotopic gastric mucosa.	None	Pneumonia following advanced inanition from haemorrhage and starvation due to ulcer.
Male	51	Indigestion for some years.	Chronic diffuse ulceration.	Chronic gastric ulcer.	Perforated gastric ulcer.
Male	57	Indigestion for 1½ years.	Chronic localised ulceration.	"Kissing" ulcers of duodenum.	Empyema from perforated oesophageal ulcer.
Male	32	Indigestion for 5 years; intermittent dysphagia for 6 months before death.	Chronic diffuse ulceration with carcinoma superimposed.	Nil.	Haemorrhage from oesophageal ulcer.
Male	22	None recorded.	Chronic diffuse ulceration.	Scar of duodenal ulcer.	Abscess of lung: empyema.
Male	68	Indigestion for some months.	Chronic localised ulcer.	Nil.	Empyema from perforation of oesophageal ulcer.

DISCUSSION OF OUR CASES.

In 8 of the 13 cases described, the oesophageal ulcer could be said to be an important factor in causing death. In 6 of the 8, death was directly due to the ulcer; and in the other 2 actually took place from pneumonia; but in both the ulcer was undoubtedly the predisposing factor, in one case being superimposed on the very advanced inanition which it had produced in the patient, and in the other following a haematemesis from the ulcer. In 3 of the cases there was an empyema due to spread of infection from the ulcer, in one case there being a direct perforation into the pleural cavity through the base of the ulcer, and in the other 2 there being a preceding acute mediastinitis with the formation of an abscess in one of them.

In 3 of the cases bleeding from the ulcer base was the cause of death. In one of these the bleeding was from erosion of the aorta; and in the other 2 very small vessels in the base of the ulcer. In one of the latter cases malignancy had become superadded to the simple ulceration, and this in a male of 32, at which age ordinary malignant disease of the gullet is rare. The unusual type of carcinoma in this case should be noted.

In 7 of our 13 cases, i.e. more than 50 per cent., there was evidence of ulceration of the stomach or duodenum, in 2 of them the ulcer having healed leaving only the scar apparent. In 5 of these 7 cases the ulceration was in the neighbourhood of the pylorus and was associated with some degree of narrowing here.

The average age of our subjects was 57 years. It should be noted that all of our patients, except 2, were over 50 years of age, and that these 2 were aged 22 and 32 years respectively.

Ten of our patients were males, and 3 were females, so that males appear to suffer from the lesion three times more commonly than females. This sex incidence tallies with the higher incidence of pyloric and duodenal ulceration in males, and it has been noted that these occurred in more than 50 per cent. of our cases.

Symptoms. - A remarkable feature of our cases was the absence of symptoms referable to the oesophagus except in 2 cases, and then only at a fairly late stage of the disease when, in the one case fibrosis was marked, and in the other when malignant disease had become superimposed. In 3 of our series no notable dyspeptic symptoms had been present. In the others, indigestion had been present for a variable period of time, but in 5 of these there was post-mortem evidence of active gastric or duodenal ulceration so that the symptoms might well have been due to this. In 4 of our series, however, no evidence of gastric or duodenal ulceration was present, and we therefore conclude that the dyspeptic symptoms were due to the oesophageal lesion.

It is interesting to note that the symptoms were referred not only by the patient, but also by the physician, to the stomach or duodenum, and even in one of our cases where the symptom of dysphagia in the later part of the illness pointed to

the oesophagus, there had been a period of 5 years preceding this when the patient had been repeatedly treated for a gastric lesion. This brings to mind the increasing difficulty which physicians have in clinically distinguishing gastric from duodenal ulceration, and makes one wonder if, in the future, the three types of peptic ulcer - oesophageal, gastric, and duodenal, - may not be simply included in the one group; for each type of ulcer as a rule gives a very similar general train of symptoms, with perhaps difference only in detail. One also wonders if the individuals of this class will not be distinguished only by special methods of examination such as barium meal and oesophagoscope; and if oesophageal ulcer will not be looked for in a routine manner along with gastric and duodenal ulcer in certain dyspeptic cases.

We shall now give a general account of the symptoms and complications of chronic peptic ulcer of the oesophagus as found in those from the literature and our own cases.

Symptoms. - The three cardinal symptoms of the condition are stated to be pain, dysphagia, and haematemesis. Let us consider each of these in turn.

(1) Pain. - This is a fairly prominent symptom in most of the cases, but occurs at a very variable time. In certain cases, a relatively small number, in fact, it occurs during the last part of the act of swallowing, or immediately afterwards. In certain cases it was shown

that the pain only occurred after hard food was swallowed (Barclay (24) Hurst (23)), and in these cases this fact was made use of in the barium meal examination and the pain was seen to be accompanied by spasm of the oesophagus. In most cases the pain appears at a variable period after food, and in some of them came on 2 or 3 hours after a meal and resembled that of gastric or duodenal ulceration. This later pain is usually helped by alkalies, and it would appear to have a relationship with the action of the acid gastric juice on the ulcer, unlike the early pain which we have already mentioned as being due to oesophageal spasm as the ingested food passes over the ulcerated area. This later type of pain is "burning" in character and unlike the early "choking" type of pain.

The point of maximum intensity of the pain lies in the upper part of the epigastrium, and quite typically just deep to the ziphisternum. From here it radiates in various directions - through to the back between the shoulder blades, to the left side of the chest, up to the left supraclavicular region, or downwards towards the umbilicus. The pain may come in attacks with periods of freedom between, just as is the case with gastric and duodenal ulceration. Like these conditions also, it tends to become more frequent and the attacks longer as time goes on.

Sometimes there may be no actual pain, but rather a

feeling of discomfort as in Christopherson's case (25). He felt as if there was something abnormal in his chest and he explained that he felt something present if he swallowed, more especially if he swallowed nothing, and also if he took a deep breath. He localised the uncomfortable spot as being deep behind the sternum. It should be noted that this patient died of a fatal haemorrhage from the aorta 7 days after consulting his doctor about this discomfort.

We must also remember in this connection the fairly large number of cases which make no complaint of pain at all, and in whom the oesophageal ulcer is simply found when a post mortem is being performed and death has occurred from some other cause.

(2) Dysphagia. - This symptom is not a very prominent one in my experience and, in fact, only two of my cases showed it and that at a late stage of their illnesses. It does not appear to have occurred very frequently in the other cases recorded, for it would at once point to the oesophagus as the site of the lesion. When it occurs in the early part of the illness it is due to reflex spasm of the organ just above the ulcer. The food is felt to stick, but passes on after a few seconds. At a later stage severe spasm occurs and may be associated with regurgitation of ingested material which may be of fairly large amount if dilatation of the

oesophagus has occurred. This regurgitated material is distinguished from gastric contents by its neutral or alkaline reaction associated with the absence of hydrochloric acid or ferments; and if there be milk present it is usually uncurdled. At a later stage the oesophageal narrowing is due to cicatricial contraction, and this may become absolute, as in one of Hurst's (23) cases.

When dysphagia is a feature of the cases it is frequently intermittent just as is the pain, and is probably associated with an acute exacerbation of the ulcerative process as is the case with peptic ulcer of the stomach or duodenum.

(3) Haematemesis. - The patient may vomit quantities of "coffee-ground" material due to the blood oozing from the ulcer into the stomach and lying there for some time before being expelled. More typically, however, the patient has a series of haematemeses. It is said that the oesophagus should be suspected as the source of bleeding when the vomiting is effortless in character. A sudden massive haematemesis due to erosion of the aorta is simply a complication of the disease - not a symptom - and shall be considered later.

The constant oozing of blood from the ulcer which may never be large enough to cause a haematemesis but

be apparent as melaena, may cause a very marked fall in the blood haemoglobin. In one of Hurst(s (23) cases, it had fallen to 64 per cent., and in Zahn's (26) case the quality of blood became so poor that the diagnosis of pernicious anaemia was made.

Those are the symptoms of the typical case. Stewart and Hartfall (10) in their analysis of 19 cases found that there were three latent cases, and of the other 16 there was substernal or epigastric pain in 15; and dysphagia and haematemesis in 11 each. In three of the haematemesis cases he included the cases in which a very large vessel such as the aorta was eroded by the ulcer, and in which the haematemesis was terminal and really a complication. He found that the three cardinal symptoms - pain, dysphagia, and haematemesis, - were present in 7 out of the 16 cases. In spite of these figures, one cannot but note the marked variation in the symptoms of the published cases which frequently point to every other abdominal lesion except oesophageal ulcer. In Balfour's (27) case, the patient had had his gall bladder and appendix removed, and gastric or duodenal ulcer was also suspected. In one of Hurst's (23) cases, the patient had been repeatedly X-rayed after a Barium Meal, but no abnormality found. He was thought to be suffering from neurasthenia and treated by psychotherapy in America, on the Continent, and in England.

Stewart and Hartfall's (10) case was admitted to hospital and treated as a case of chronic gastric ulcer. In our own series of cases only two were diagnosed as oesophageal lesions before death (Nos. 7, 11). In those who had symptoms, gastric or duodenal ulcer was the provisional diagnosis made.

Before finishing our discussion of the symptoms of the condition, we must draw attention to the marked neurasthenia which may follow in severe cases of the condition, a neurasthenia which is only too apt to become progressively worse as the patient gets into the hands of more and more unsympathetic physicians who fail to demonstrate a lesion by the ordinary X-ray methods, and therefore assure the suffering patient that he is simply afflicted with "nerves". This was marked in one of our patients (Case 8), who was in the last stages of emaciation and worry. So terrified had he become of the pain that he was afraid to take food, and had finally taken to alcohol as a means of relieving it. In one of Hurst's (23) cases, the patient had become extremely neurasthenic because of the pain. "When a meal was brought into the room, his face became anxious and his hands shook. Several minutes elapsed before he could persuade himself to lift the fork in his trembling hand to his lips; he masticated much longer than was necessary in order to put off the moment of swallowing

as long as possible, at the same time shaking his head. He had lost much weight and strength and was extremely depressed".

To summarise the clinical aspect of chronic peptic ulcer of the oesophagus we may say that a number of cases are latent and give no symptom suggestive of oesophageal, or even gastric disease, until a complication of the condition, such as perforation into a pleura, appears; or the ulcer may be absolutely latent and be found only at post mortem where the patient has died of some other condition. There may be definite symptoms present over a period of a few weeks to many months. Typically, these symptoms consist of pain, dysphagia, and haematemesis. In some of the cases the symptoms suggest an oesophageal lesion, but in many, and in our experience in most of them, the symptoms suggest a gastric lesion. In a few cases such as one of ours (No. 7), and that of Reher (28), the symptoms are present for some years, being there for 6 years in ours, and 4 years in Reher's. In many of these, however, e.g. that of Miller (11), the symptoms were those of gastric disease, and the case described by the latter suffered from gastric pain after meals for 6 years and developed a severe haematemesis from the oesophageal lesion 2 days before death.

Complications. - As might be expected, the complications

of chronic ulcer of the oesophagus are due to the penetration or perforation of adjacent organs by the ulcer, and the onset of malignant disease.

(1) Acute Mediastinitis. - This is a not uncommon complication and is due to the spread of infection from the base of the ulcer into the adjacent mediastinal tissues. This occurred in 2 of our cases, in one an abscess developing, and in both a final spread to the adjacent pleural cavity occurring, causing the formation of an empyema. In Lindsay's (29) case, a mediastinal abscess developed, and then ruptured in the lung, causing death 37 hours after the onset of acute symptoms. In Miller's (11) case, a mediastinal abscess also developed and burst into the left pleural cavity, causing a pyopneumothorax. The course of the patient's last fatal illness was 9 days.

(2) Perforation into the pleura. - As has just been noted this is the usual termination of the cases where acute mediastinitis first developed. It may be the primary complication, however, as in one of our cases (No. 10), where a perforation opened directly into the right pleural cavity from the base of the ulcer. Perforation into the right pleural cavity occurred in Stewart and Hartfall's (10) case. In Lindemann's (30) case, perforation into both pleural cavities had occurred and given rise to a double pneumothorax. Perforation into the right pleural

cavity was a complication in one of Tileston's (8) cases. Christie (31) described a case where the ulcer perforated into the right pleural cavity. The case is interesting for the clinical picture suggested an acute peritonitis, although at post mortem there was a complete absence of peritoneal involvement. This feature was noted in two of our cases (Nos. 10 and 13). Both were thought to be perforated gastric or duodenal ulcers, but were actually cases of acute pleurisy from the rupture of the oesophageal ulcer into the pleura. Thus, even in its complications, oesophageal ulcer resembles its related gastric and duodenal lesions.

(3) Perforation into the left bronchus. - The oesophagus is related to the left bronchus at about the middle of its course, and thus a malignant ulcer of the middle of the oesophagus not infrequently opens into the bronchus. In chronic peptic ulcer this is an extremely rare complication because the ulcer in almost every case lies at the extreme lower end of the oesophagus. We have only traced one case in the literature where this occurred - that of Finlayson (14) which was shown to the Glasgow Pathological and Clinical Society in 1883. The patient was a young man aged 22 years who had been healthy up to 3 months before admission to hospital, when he started to vomit. A cough then developed and blood was expectorated at frequent intervals. He became extremely emaciated, and signs of

consolidation and excavation appeared on the left side of his chest. The patient was diagnosed as an advanced phthisis, but the combination of vomiting and coughing appearing immediately after the ingestion of food was puzzling, and thought possibly to be due to irritation of a diseased larynx during the process of swallowing the food. There was no hoarseness, however, which was against laryngeal disease.

At the post mortem a simple ulcer of the oesophagus was found to be opening into the left bronchus and causing gangrene of the lung.

Finlayson (14) who had searched the literature referred also to a case described by Part in the Eighth Volume of the Pathological Transactions where a simple ulcer of the oesophagus had penetrated the right bronchus and destroyed part of the associated lung.

(4) Perforation into the pericardium. - The lower part of the oesophagus is related anteriorly to the pericardium, and we should expect perforation into the pericardium to be a common complication. It is, in fact, a very rare one, and we have only found one case in which it actually occurred - that of Trotter (32). His patient was a woman, aged 25 years, who had been troubled with nausea and vomiting just after meals for 3 months, and dysphagia for 2 months, accompanied by an aching pain between her shoulders. Five weeks after admission she developed a

constant teasing cough, and the following day was found livid and pulseless.

Post Mortem. - A chronic ulcer of the oesophagus was found to have perforated into the pericardium which was filled with thin, yellow, sour-smelling, acid fluid.

(5) Perforation into the peritoneal cavity. - The lowest part of the oesophagus lies beneath the diaphragm in the abdominal cavity, and it is not surprising that an ulcer of its lower end may perforate into the abdominal cavity. This occurred in Eversmann's (33) case. An acute peritonitis occurred and was localised to the lesser sac of peritoneum.

(6) Erosion of the aorta. - This occurred in one of our cases (No. 4). The patient had had a two months' history of indigestion, accompanied by epigastric pain, and the final fatal bleeding was preceded by marked melaena and blood-stained vomitus for some days before.

The aorta was opened into by the ulcer in one of Eversmann's (33) cases. In this case dysphagia and substernal pain had been present for $3\frac{1}{2}$ months before. In Christopherson's (25) case the patient suffered from discomfort in the chest for only 7 days before death took place from perforation into the aorta. Finlayson (14) refers to a case described by Professor Flower in which perforation of a simple ulcer of the oesophagus, exactly

resembling that of the stomach, took place into the aorta.

It should be noted that death may occur from haematemesis when a relatively small vessel is eroded but continues to bleed. This occurred in two of our cases (Nos. 7 and 11), although in one of them (Case 11) malignancy had become superadded. Death from haematemesis, due to erosion of small vessels in the ulcer floor, also occurred in the cases described by Hödlmoser (34), and Zaleski (35).

(7) Development of carcinoma. - Ortmann (17) described the development of carcinoma in the scar of a peptic ulcer of the oesophagus. Tileston (8) stated that he had seen a similar specimen in Vienna. In one of our cases (No. 11), a male aged 32, malignancy had become superadded on a simple ulcerative process. This patient had suffered from indigestion for 5 years, and then developed intermittent dysphagia for some months before his death.

Diagnosis. - Where the clinical history shows the presence of substernal pain and dysphagia, an oesophageal lesion may be suspected, but in very many - if not most of the cases - these symptoms are not present and an oesophageal lesion is unsuspected.

Where the lesion is thought of, the two best methods of

confirming the diagnosis are by the barium meal and oesophagoscopy.

(a) The barium meal. - Barclay (24) described a case of peptic ulceration of the oesophagus in a girl aged 18 who complained of difficulty in swallowing from time to time. A barium meal showed no abnormality in the oesophagus or stomach, but when she was given some hard food such as toasted crumbs the pain appeared and she was at once given a barium meal which showed a complete obstruction of the oesophagus which persisted for 5 to 10 minutes, and was clearly due to spasm. An ulcer was suspected, and proved by oesophagoscopy.

Jackson (19) showed the presence of spasm in many of his cases. Friedenwald, Feldman and Zinn (20) actually succeeded in visualising the crater in one of their cases, and were the first to do so. In Hurst's (23) first case, a female aged 35 years, a solid meal mixed with barium was given, and pain was at once produced. A remarkable X-ray picture was then obtained. The cardiac sphincter of the oesophagus was wide open, and immediately above it was a round shadow which could only be the crater of a large ulcer at the extreme lower end of the oesophagus just above the sphincter. Immediately above the ulcer, the lumen of the oesophagus was narrowed as a result of constant spasm. The diagnosis was confirmed by

oesophagoscopy. His second case was that of a man aged 78. X-ray examination showed a huge gas bubble in the stomach which pushed up the left half of the diaphragm. Repeated X-rays showed no other abnormality however, but finally a deep chronic ulcer in the lower end of the oesophagus was demonstrated.

In our own series of cases, appearances of oesophageal disease were seen twice only (Cases Nos. 7 and 11). In both, the appearances suggested a carcinoma. In one there was actually neoplasm superimposed on a chronic ulcer; but in the other the appearances were due to fibrosis.

(b) Oesophagoscopic examination. - This is usually done to confirm the diagnosis after a barium meal examination has been carried out. It was by doing oesophagoscopic examinations in all cases of even slight dysphagia that Jackson (19) diagnosed the condition so frequently. Friedenwald and his co-workers (20) also used this as their chief means of diagnosis. The difficulty of making an accurate diagnosis by the oesophagoscope alone is well known, and thus, wherever convenient, a small piece of the edge of the ulcer may be excised for biopsy.

Pathology. - We have already discussed the points of difference between acute and chronic peptic ulceration of the oesophagus

and have suggested that fibrous tissue proliferation is the most important criterion of chronicity, and that size itself is really of little importance as acute ulceration may involve the whole of the lower third of the organ. Thus, in chronic peptic ulcer of the oesophagus there is evidence of fibrous overgrowth which more or less replaces the circular or longitudinal muscle layers, and spreads outwards into the mediastinal tissues around.

Naked eye there appears to be two different types of ulcer found in the area, both of which may be classified under peptic ulcer of the oesophagus.

(a) In the first type, the ulceration over most of the area is relatively superficial and circular or longitudinal muscle is exposed in the floor. The ulceration involves the whole circumference of the oesophagus. The lower edge corresponds to the extreme lower end of the oesophagus, is clean cut, and is, as a rule, the deepest part of the ulcer. The upper edge is very irregular and indefinite. It has frequently a circinate appearance and fades off into intact mucosa. The appearances obviously suggest that attempts at healing are taking place at the upper edge, but that the ulcerative process is unhealing and indolent at the lower part. These appearances are well illustrated in Cases 1, 3, 6, 9, and 12.

A much deeper patch of ulceration may be present within the area of diffuse ulceration, and it is to be noted that when this occurs it lies at the lower end of the ulcer near the cardiac sphincter. This is well seen in Case 4.

(b) In the second type the ulcer is localised and is usually deep and penetrating with steep, often inverted edges, and resembles very markedly the chronic ulcer found in the pyloric region of the stomach or in the duodenum. The ulcer is rounded or oval, and lies at the lower end of the oesophagus, but has not the same strict relationship to the cardiac sphincter that the other type has. This type is well illustrated by certain of our cases - Nos. 8, 10, and 13.

In one of them gastric heterotopia was present, and shall be referred to later.

Histological examination of either of these types shows the appearances got in ordinary peptic ulcer of the oesophagus or duodenum, and the four layers described by Askanazy (36) may be seen. On the surface is the layer of fibrino-purulent exudate. This is most abundant near the margin of an ulcer, and may be absent in the centre of a large ulcer. Next is the zone of fibrinoid necrosis with pyknotic nuclear debris, and usually staining very intensely with acid dyes. This fades into the granulation tissue zone which contains fairly recently formed

blood vessels and leucocytes which are usually of the lymphocytic class. Imperceptibly this zone gives way to the deepest layer, the fibrous or cicatricial layer, which is composed of well-formed fibrous tissue replacing the breach in the muscular coats at the site of ulceration. The small arteries here show marked endarteritis obliterans.

Etiology. - The two types of ulceration described by us are so different in their appearances that we are tempted to look for two entirely different causes.

(1) The diffuse, more shallow type of ulceration affecting usually the lower part of the oesophagus, conforms in position and fairly widespread character to the acute form of peptic ulcer, or so-called antemortem digestion of the oesophagus. We feel that the name "antemortem digestion" is a misnomer as it clearly suggests that the lesion is a fatal one and occurs in individuals a short period before death. We have examined a large number of oesophaguses from all manner of cases coming to post mortem, and have concluded that, whereas the acute fulminating and fatal type of antemortem digestion is rather rare, the lesser degrees of the condition are no means uncommon, and may very likely be recovered from.

We have already likened antemortem digestion to

burns of the skin, and divided it into 4 degrees of severity. We have explained how complete healing of the damaged area might occur by proliferation of the epithelial islets left in the second degree of the process. Consider the third degree of antemortem digestion, where there is complete destruction of the mucous membrane of the lower part of the oesophagus. If such a case recovers from the primary illness, what will happen to the affected area? The necrotic material on the surface will separate, and attempts at healing begin, the mucous membrane at the periphery commencing to grow in, in order to cover the denuded area; but there are no epithelial buds left to fill in the central part, and if we carry our analogy with a burn of the skin still further, we can conceive how difficult it would be for such an area to become completely epithelialised; how in such a burn of the skin, healing usually takes place so far, and then ceases until skin grafts are applied. Still more difficult will it be for healing to take place in the case of the oesophagus whose mucosa, especially at the lower end, is being constantly irritated by food, and on occasions also by acid gastric juice, which, even in the normal oesophagus may irritate to the degree of sensitivity known as "heartburn". Thus a stage will be reached when the acute ulcer becomes replaced by a chronic ulcer of the diffuse and fairly shallow type, like many we

have described. The upper edge, which is farthest from the irritating gastric juice will show attempts at healing, and be irregular and indefinite, whereas the lower edge which is in close proximity to the gastric contents will be sharp and clean cut and show little or no healing. In some of our cases a deeper excavation was present at one part, always at an area adjoining the cardiac sphincter - an ulcer within an ulcer, as it were.

A comparison of the naked-eye appearances of antemortem digestion and chronic diffuse ulceration of the lower end of the oesophagus is more than suggestive. Both cease abruptly at the cardiac sphincter, but fade off in an upward direction; in fact, the only real difference, naked eye, is the dark haemorrhagic colour of the acute lesion.

The association of both the conditions with gastric or duodenal ulcer should also be noted. This was found in more than 50 per cent. of our chronic cases, and in 25 per cent. of the cases of antemortem digestion. It is probable that cases with gastric or duodenal ulcer and their accompanying hyperchlorhydria are more prone to develop the acute lesion which is then prevented from healing by the high acid content.

Lastly, in one of our cases (No. 1), both processes were present, an antemortem digestion being superimposed

on a chronic peptic ulcer of the oesophagus.

We feel that all the evidence points to this diffuse type of chronic peptic ulceration of the oesophagus as having its origin in a previous acute ulceration, the so-called antemortem digestion, which has become chronic due to a combination of size and abnormally acid gastric juice.

(2) The second type of ulcer differs markedly from the last type. It is very localised and is marked by depth rather than by diffuseness. A clue to its origin was given by the finding of a large patch of heterotopic gastric mucosa beside the ulcer in one of our cases (No. 8). Heterotopic gastric mucosa was found in Stewart and Hartfall's (10) case, in Fraenkel's (37) case, and Tileston's (8) case.

Small islets of glandular tissue resembling gastric glands are found in the oesophagus near its upper end. Schridde (38) found these in 70 per cent. of all cases at post mortem when the upper oesophagus was examined microscopically.

In a consecutive series of 900 post mortems Taylor (39) found six cases in which these heterotopias were distinguishable naked eye, varying in size from $\frac{1}{8}$" to $\frac{3}{4}$" in diameter. These cases apparently had no symptoms of the abnormality during life, for when the patches are

small and the amount of acid secreted is correspondingly little, it will be rapidly diluted by saliva and no harm result. If the patches are more extensive, however, and if some degree of spasm of the cardiac sphincter be superimposed, the accumulation of this acid secretion will set up an irritative, and later, ulcerative condition. That is the view usually held in cases of oesophageal ulcer associated with gastric heterotopia. If that were the correct explanation we should expect the ulceration to be rather of the diffuse type which we have already discussed, instead of the localised crateriform type. While not denying that such may be the correct explanation, especially where the gastric heterotopia occurs in the upper oesophagus and the ulcer in the lower part, from the appearances of our own case we would suggest that the ulcer may actually start in the heterotopic mucosa itself which, as is well known in the case of Meckel's diverticulum, shows a marked tendency to ulcerate when it finds itself in a foreign environment.

Treatment. - Various methods of treatment have been recommended and practiced for the condition. Barclay (24) used zinc ionisation, and his method was rather ingenious. A bobbin-shaped electrode attached to a wire and insulated with shellac (except at the neck of the bobbin) was swallowed by the patient till it reached the stomach. The patient was then given

breadcrumbs till he felt the pain of the oesophageal spasm come on. The bobbin was pulled up till it reached the spasm, and then by applying a little more traction its upper part slipped through the spasm and its neck was firmly gripped and lay in close proximity to the ulcer. A large electrode was placed on the arm and a current of 10 m.a. passed from the positive electrode in the oesophagus for 10 minutes. When the electrode was removed a black mark on one side of the neck of the bobbin showed the size and shape of the ulcer. The patient was well for 3 months, some faulty teeth then removed, and another ionic treatment given. The patient remained well for one year.

The author finishes by remarking that a letter had recently been received saying that a recurrence had taken place, and the procedure was repeated with a stronger current.

The difficulty of this ingenious method of treatment cannot be fully appreciated from its description. The author was going upon the assumption that the spasm always lay at the site of ulceration. In other cases in the literature the spasm lay above the ulcer. Moreover, the recurrence makes one wonder if the ulcer had actually healed and if the relief of symptoms was not merely due to the effect of the current in abolishing spasm of the muscle for a prolonged period of time.

Balfour and Eusterman (27) treated their case by the application of 20 per cent. silver nitrate followed by a bland diet and alkalies. In some weeks the stricture was dilated by a bougie. There was immediate improvement, but the symptoms returned in 2 years.

Hurst (23) treated both his patients with milk diet, alkalies, and atropine, and they rapidly lost the pain and dysphagia. The ulcers became smaller in both cases, but after many weeks, had not healed owing to the associated spasm in the one case, and the development of cicatricial contraction in the other. Gastrostomy was thus performed in both cases and resulted in complete healing with disappearance of occult blood.

In the case where spasm had been the feature preventing healing, the stoma was now allowed to close and the patient swallowed food without difficulty in spite of the development of a slight stricture at the site of the ulcer. In the other case, dilatation of the fibrous contraction by bougies was to have been carried out and the gastrostomy then allowed to close. The patient died, however, before this could be done. Hurst adds that he shall, in future, advise a temporary gastrostomy at once wherever the diagnosis has been made unless the patient is seen within a year of the development of symptoms. In such cases a fluid diet with alkalies and atropine might lead to a cure.

Stewart and Hurst (3) in their textbook advise starvation with the administration of saline and dextrose by the rectum. This should be followed by a period during which the patient is given the usual routine treatment for gastric or duodenal ulcer, and this be carried out till the symptoms have disappeared and there is no longer occult blood in the stools. They add that adrenaline is more likely to be of value where haemorrhage is taking place from an oesophageal ulcer than it is in a gastric or duodenal one.

Friedenwald and his co-workers (20) advised, and got good results from a fluid diet with alkalies and belladonna. Chevalier Jackson (19) advised the direct application of 10 per cent. silver nitrate by the oesophagoscope, once weekly.

Of the various methods of treatment advocated, that which gives rest to the ulcer combined with a reduction of the gastric acidity would appear to be the most logical. Rest to the ulcer can be more easily got in an oesophageal than a gastric or duodenal ulcer by the relatively simple procedure of doing a gastrostomy.

It should be remembered, as already pointed out, that gastric or duodenal ulcer may, and quite commonly does occur along with the oesophageal one, and in the case described by Hellmann (40) a gastrojejunostumy was also performed because of a co-existing pyloric obstruction.

Rectal feeding as advised by Stewart and Hurst (3) is not advisable for it can only in a very imperfect way supply the needs of the body, and we have been struck by the frequency with which complications - especially fatal haematemesis - have occurred in gastric ulcer cases being treated by a starvation diet. Because of this, we feel that wherever possible the patient should receive a diet containing all the accessory factors necessary to health. In the case of gastric or duodenal ulcer, the physician must try to strike the happy medium between a diet which may irritate the ulcer, and one which will supply the necessary accessory factors. In the case of the oesophagus, however, a full, nourishing diet may be given by a gastrostomy opening without touching the ulcer at all. Even the alkali may be given by this route, for the very act of swallowing is liable to set up spasm and it is this above all which prevents healing of a peptic ulcer.

As to the time when the gastrostomy should be performed there may be some difficulty. Hurst (23) suggests that a year or more of symptoms should be the indication for this. But we must remember those cases where the ulcer has been latent for a prolonged period and we suggest that the oesophagoscopic appearances should also be taken into account. If the ulcer be small and healing, medical treatment without gastrostomy should be carried out; whereas, if it be diffuse,

or is of the deep penetrating variety which is so apt to result in serious complications, gastrostomy should be performed forthwith so that the ulcerated area may be completely rested and healing encouraged.

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