Conclusion.

A case of severe rickets is described which has been under treatment during the greater part of the year 1925. From April to the beginning of September the child received cod-liver oil as an out-patient, but with only slight improvement; during the last three months of the year he was admitted into hospital and irradiated cholesterol was administered, with the result that the rickets was cured.

Whilst it is clearly impossible to generalize from one case, the results obtained suggest that this method of treatment is well worth a trial, either alone or in combination with cod-liver oil, in cases which do not respond quickly to cod-liver oil. It is not an expensive method of treatment; the amount of cholesterol used weekly represents a cost of about 2s. 6d.

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THE ETIOLOGY OF GASTRIC AND DUODENAL ULCERATION.*

BY

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THE etiology of chronic gastric and duodenal ulceration is one of the most actively debated pathological problems to-day. A great deal of experimental and clinical research is being done on the subject, but there are many unsettled questions. Among the most important are these: What is the role, if any, of infection? What is the actual part played by the gastric secretion? Is it the prime and sufficient cause of chronic ulceration? If not, what antecedent morbid conditions contribute to the appearance of ulcer? What, if any, is the part played by the nervous system?

INFECTION.

Important in its bearing on the possible part played by infection in the causation of gastric ulcer is the work of Bolton.[1] He employed a serum produced by repeated injections of the gastric cells of one animal into another. In the presence of gastric juice injection of this serum resulted in the appearance of gastric ulceration. If the gastric juice was put out of action by sodium bicarbonate ulceration failed to appear. The serum, therefore, did not produce necrosis, but rendered the gastric cells susceptible to attack by the gastric juice. He considered that other blood-circulating poisons could prepare the way for self-digestion, and that the frequency of gastric ulceration in general intoxication was due to attack by the gastric juice when the resistance of the gastric cells was lowered. It did not appear from his researches that hyperacidity alone was able to produce ulceration, but he found that if a series of animals were injected with serum, and hyperacidity produced in some and not in others, then those with hyperacidity showed much more marked gastric lesions than those without. He further showed that if, in the cat, delay in gastric emptying was produced by putting the animal on a meat diet, the formation of ulcer was more rapid than when the animal was on milk and there was no delay. When there was a definite delay in the emptying time of the stomach, then there was a definite delay in the healing of experimental ulcers.

Bolton's work bears, therefore, on the subject from two directions—first, on the possible influence of intoxications, specific or general, in preparing the ground for auto-digestion; and secondly, on the role played by the gastric secretions. He found that the injection of toxins other than gastrotoxin—for example, hepatotoxin, enterotoxin, prepared in a similar manner—led to the formation of gastric ulceration.

Reeves[2] drew attention to certain local circulatory peculiarities of the stomach and duodenum which appeared to predispose to stasis and thrombosis. He stated that along the lesser curvature of the stomach the circulation is carried on through parallel arterioles supplied at both ends by arterial majors, and this condition, he considered, gave an opportunity for some stasis. Further, that a triangular supply throughout the first two inches of the duodenum predisposed to thrombosis. He had in mind that this tendency to stasis and thrombosis might be made actual by infective processes.

As long ago as 1907 Türck stated that he had produced peptic ulcers of the stomach and duodenum of dogs in 100 per cent. by feeding the animals on bouillon cultures of *B. coli communis*. On cessation of the feeding the ulcers were found in varying stages of healing. These ulcers for the most part caused the death of the animals from perforation and haemorrhage.[3] Türck's experiments have not, so far as I know, been repeated and confirmed.

Dawson[4] in 1912 accepted the proposition that the primary causes of gastric ulceration were toxic and bacterial agencies which acted directly on the gastric cells. He considered that these produced in the first place what he called the mucous ulcer, and that the chronic ulcer was a further development from this. He stated that in a series of personal observations he had found the stomach sterile in cases of chronic ulcer; fourteen such cases were examined bacteriologically, and ten showed sterility.

The work of Rosenow requires examination in some detail. In a paper published in 1913[5] he stated that ulcer of the stomach or duodenum, or both, was produced by intravenous injection of streptococci in eighteen rabbits, six dogs, and one monkey. In most of the rabbits and "some" of the dogs there was also arthritis, in some myositis and the "picture of an ascending nephritis." Ulcers occurred when streptococci of a middle grade of virulence were injected. These streptococci were derived from cases of rheumatism. Areas of haemorrhage were found in the mucous membrane within twenty-four hours of injection, and ulceration in forty-eight hours. Some of these ulcers became chronic, but no details as to persistence are given in the paper. In 1916[6] he reported the experimental production of ulceration and haemorrhage (the percentage of ulcer incidence is not stated) in 60 per cent. of 103 animals injected with eighteen strains of streptococcus derived from human gastric ulcers. The ulcerations were often single and deep, and showed a marked tendency to perforation.

In 1921[7] he published a further series of observations. Sixty-five animals were injected with streptococci derived from patients suffering from ulcer of the stomach or duodenum. Thirty-two of these were injected with cultures from the tonsils, six with organisms in the pus from the tonsils, seven with streptococci from three excised ulcers, and twenty-three with cultures from infected teeth. The incidence of lesions (that is, ulceration or haemorrhage) was about equally high in each group—that is to say, 80 per cent. The numbers in which actual ulceration developed is again not given. The animals were killed two to three days after the last injection. From actual human ulcers thirty-seven strains of streptococcus were isolated and injected into 168 animals; of these, 68 (45 per cent.) showed "lesions" in the stomach and duodenum; the type of lesion is not stated, nor the comparative frequency of gastric compared with duodenal lesions. Rosenow published a further paper in 1923[8] giving a parallel series of observations in the hog, cow, sheep, calf, and dog. He stated that streptococci were isolated in pure culture or in predominance from a series of gastric ulcers in these animals. Ulcer, or haemorrhage, or infiltration of the stomach was produced in 86 per cent. of rabbits and dogs injected with freshly isolated cultures. Similar results were obtained with cultures of streptococcus from infected tonsils in the cow and the dog which had ulcer. Rosenow maintained that results such as these had not been obtained with streptococci of similar morphology from sources other than ulcer.

* A paper read before the Pathological Section, Liverpool Medical Institution.

There is so far no confirmation of Rosenow's experimental results. Wilkie has stated that he had been unable to confirm them in certain respects, but I am not aware that he has published any personal investigation. Their bearing on the etiology of the chronic ulcer of the stomach and the chronic ulcer of the duodenum in man is not clear. He appears to have shown two things: that streptococci can be isolated from the majority of chronic gastric and duodenal ulcers; and that streptococci from these sources, and also from many other sources, produce in a large percentage of animals when intravenously injected certain acute lesions in the gastric mucous membrane, sometimes without obvious lesions elsewhere. There is a want of detail in the published experimental results which makes it difficult to judge their significance. Durante (a colleague of Rosenow in the Mayo Clinic) says: "If the results obtained in experimental work do not exclude the possibility of ulcers forming in consequence of bacterial infection, the fact remains none the less that bacteria are found in a limited number of cases only." He appears to incline to the view, in the face of Rosenow's work, that the presence of organisms is the result rather than the cause of ulceration.

Gibson in 1921 described the lesions produced in a monkey by injection of a streptothrix obtained from a case of acholuric jaundice. These included an inflammation of the spleen with thrombo-phlebitis giving rise to infective emboli which in the stomach resulted in ulceration, there being several circular ulcers with sharply defined edges not confined to any area. This, no doubt, was an example of gastric lesions produced by retrograde venous thrombosis, a condition known to surgeons and recognized as the explanation of some cases of gastric haemorrhage in certain abdominal infections.

In supplement to the experimental investigations above noted there is a general clinical view that gastric disorder, chronic gastritis, and also gastric ulcer, are frequently associated with infections elsewhere—dental, nasal, tonsillar, and in the gall bladder and appendix. At any rate it is a clinical experience that must be noted that gastric pain, and also the other signs and symptoms which are usually associated with ulcer, are often ameliorated and not infrequently disappear after the elimination of infection in one or other of these situations.

Rosenow has drawn the conclusion from his work that ulcer—that is, chronic gastric ulcer and chronic duodenal ulcer found in the human subject—is a lesion produced by a specific streptococcus which attacks the gastric wall, reaching it by way of the blood stream, and that this streptococcus is the effectual cause of the disease. Bolton, on the other hand, had an equal experimental success with a variety of toxin inoculations, and considered that attack by the gastric juice was an essential part of the process of ulcer production.

Alterations in Secretion.

That auto-digestion plays a part in ulcer is a view which dates from before John Hunter, who explained the fact that man did not invariably digest his own stomach wall on the ground that the gastric juice could not attack living tissue. Various writers have ascribed ulcer formation to alterations in the chemistry of the gastric secretion, more particularly to hyperacidity and hyperchlorhydria. Until the method of the fractional test meal examination was introduced hyperchlorhydria was credited by many with the chief role in the production of the chronic ulcer. Investigations by this method have led to the abandonment of this view.

According to Moynihan[9] hyperchlorhydria was demonstrated in 72.7 per cent. of a series of 71 cases of duodenal ulcer, and in 20 per cent. only of a series of 39 cases of gastric ulcer. These figures are approximately the same as those of other investigators. The combined figures of Bell and Hunter, as given by Ryle,[10] are:

In gastric ulcer:	
Normal acidity	57 per cent.
Hyperchlorhydria	33 ,,
Achlorhydria	10 ,,
In duodenal ulcer:	
Hyperchlorhydria	70 per cent.
Normal acidity	30 ,,
Achlorhydria	0 ,,

In duodenal ulcer in particular the fasting juice shows a high acid curve up to 80 and 100 c.cm., compared with a normal of 20 c.cm.

Eusterman[11] recorded the findings in two series of gastro-jejunal ulcers. In one series of 47 cases hyperacidity was present in 60 per cent., in a second series of 36 hyperacidity was present in 73 per cent.

It is not necessary to review the numerous observations on the gastric secretions in ulceration which have been recorded by workers using the fractional test meal method. It is generally agreed that in duodenal ulcer the acidity of the resting juice is usually high and that there is generally a climbing curve, after the initial fall which follows the meal, up to about the end of the second hour, and that in the absence of stenosis the curve then usually falls, to rise later to the high fasting level. Further, that in pyloric stenosis there is a climbing acidity curve which usually rises steadily during the third hour and often later. Thirdly, that in gastric ulcer without stenosis there is no acidity curve that can be called characteristic. The figures given by Ryle show, in fact, that the percentage of hyperchlorhydria curves in gastric ulcer cases may not reach the percentage in 100 normal individuals.[12]

	Hyperchlorhydria.	Normal.	Hypochlorhydria.
Duodenal ulcer (50) ...	68	28	4
Gastric ulcer (16) ...	13	75	12
Normal (100) ...	15	80	5

I do not know that the proposition has ever been put forward that hyperacidity and a presumed consequent hyperactivity is solely responsible for the initiation and development of gastric and duodenal ulcers. At any rate it is perfectly clear that such a proposition has no basis to rest on, for the following sufficient reasons:

First (I quote Carlson as the authority here). Normal gastric juice is equal in total acidity to the maximum acidity reported by clinical observers for so-called hyperacidity in man. He says: "So far as I am acquainted with the literature there is no evidence that the gastric glands under any pathological conditions are able to or do secrete a juice of higher than normal acidity. Moreover, the presence in the stomach of gastric juice of full acid strength leads by itself and immediately to no untoward results."[13]

Secondly. It has been pointed out by Boldyreff and others that the acidity curve is not a secretory curve, and that its variations depend in the main on the presence or failure of that process of neutralization by regurgitation which appears to have as its object the production of an optimum digestive activity varying with the stages of digestion.[14]

Thirdly. The examination of normal individuals has shown that curves varying from high acidity through normal and down to complete anacidity are recorded without any corresponding variations from apparent health. Carlson says:[15] "There is no disease known capable of inducing true gastric hyperacidity, the pathological variations in acid and pepsin concentrations are invariably in the direction of a decrease." Actual hyperacidity in the sense of a gastric juice of greater than normal acidity has not been demonstrated in any disease and probably does not exist; actual hypersecretion does, however, of course occur.

There is, therefore, at present no evidence that a high acidity of the gastric juice is a primary factor in the causation of ulcers, gastric or duodenal. We must not, however, lose sight of the remarkable difference between the average duodenal ulcer acid curve and the average gastric ulcer acid curve; in the explanation of this difference there may be a clue to ulcer formation. The work of Boldyreff and Carlson brings us definitely to the conclusion that the climbing curve of duodenal ulcer and of pyloric stenosis is due in both instances to a failure in neutralization by regurgitation, and this leads up to the study of stomach motility and its disturbances.

Disturbances of Tonus and Rhythm.

Rogers and Hardt,[16] studying the stomach movements by the rubber balloon and *x* ray, and comparing their results with the contributions of Cannon, Carlson, and Forsell,

conclude that the normal stomach exhibits the following types of muscular activity:

1. A tonic grasp of the upper stomach musculature upon the food. This tonic condition exhibits slow rhythmical variations.
2. Peristaltic contractions of the antrum pylori.
3. Peristaltic contractions (hunger contractions) of the entire stomach.

During normal digestion the peristaltic waves sweep over the lower part of the stomach. In the meantime there are slow rhythmical tonus variations of the upper part of the stomach. As the stomach empties itself the peristaltic waves arise from points higher and higher toward the cardiac end, run over the entire stomach, and culminate in more or less tetanic contractions of the antrum. Both tonus rhythm and peristalsis may be inhibited by introducing food or liquid into the stomach.

Carlson, Orr, and McGrath, studying the hunger contractions of the Pavlov pouch, conclude that these contractions are controlled by a gastric automatism and not by motor impulses through the vagus. It has, however, been demonstrated that section of the splanchnic nerves increases gastric tonus and augments hunger contractions.

What are the variations from normal gastric motility which occur in disease?

It is, I presume, agreed that gastric pain is the pain of gastric muscular spasm or tetanic contraction, and that it is frequently present in the absence of any ulcer lesion in either stomach or duodenum. It is the tetanic contractions of the hunger period which wake the infant: the infant does not cry because he thinks of food, but because his gastric musculature is causing him a discomfort. As in the case of the small intestine, where contents of a certain kind set up the painful contractions of colic, which contractions cease on expulsion of the said irritating contents, so also in the stomach contents of certain kinds may set up abnormal contractions which may be felt as discomfort or pain. It seems probable also that psychic influences (as shown by Rogers and Hardt) are capable of interfering with the normal course of gastric rhythm. There is less actual clinical evidence that intoxications are capable of interfering in the same way, but nicotine at any rate does so, and it may be the case also in bacterial intoxications. Further, there is much clinical evidence that visceral disease elsewhere—in the gall bladder and appendix in particular—may be associated with abnormalities in the gastric muscular mechanisms. Among actual observations we have the figures given by Ryle showing hypertonus to be nearly twice as common in cases of appendicitis and cholecystitis as in the normal individual.

In putting forward a thesis that alterations in gastric rhythm, produced by one cause or another, are themselves the primary disorders in the process which ends in chronic ulcer, I propose to consider the duodenal ulcer first.

Duodenal Ulcer.

Clinical and radiological observations show that in individuals with actual duodenal ulcer there are the following variations from normal gastric motility:

1. A marked early hypertonus leading to an exceptionally early evacuation.
2. A relaxation of the pylorus.
3. A late exaggeration of hunger contractions which are sensible as pain—that is to say, a late spasm of pars pylorica and pylorus which is often shown radiographically by an unduly long retention of the last portion of the opaque meal.

Hurst has shown that this composite picture may be regarded as an extreme exaggeration, a vicious exaggeration of a rhythm type commonest in the male, which shows in radiograph the steer-horn stomach outline, in contrast with the J-shaped stomach, commonest in the female.

Certain characteristic alterations in the chemistry of the gastric contents are the consequence of, not the cause of, these alterations in gastric rhythm. As far as the chemistry of the stomach is concerned, in a typical case the gastric juice after the first quarter of an hour shows an increasing acidity owing to absence of neutralization from the duodenum; the failure in regurgitation is no doubt due to the increased tonus on the gastric side of the pylorus, sufficient to prevent any reflux of duodenal secretions into the stomach. As far as the duodenum is concerned, the effect of the morbid rhythm is the early forcible discharge into this segment of gut of an unneutralized gastric juice.

The effect of unneutralized gastric juice on the wall of the duodenum has been studied experimentally. The most important experimental study is that of Mann.[17] He describes the production of peptic ulcer in the dog by the following method. The pylorus was cut and the distal end closed. The first portion of the jejunum was cut and the distal part anastomosed to the pylorus. The proximal jejunal end was then anastomosed to the ileum. In this way the stomach is made to empty itself into the jejunum and the duodenal secretions into the ileum at least 50 cm. from the pylorus. Following the operation a typical chronic peptic ulcer developed in 90 per cent. of the dogs. Mann satisfied himself that operative trauma was not responsible for the ulcers. They did not begin to heal until after the anastomosis had healed; they did not involve the suture line primarily. In certain other experiments the duodenal secretions were carried to the jejunal loop close to the pyloric anastomosis; ulcer did not form under these conditions. When, however, in the same animals, the duodenal secretions were led away to the lower ileum ulcers developed at the usual site and in the usual time. In these cases the site of the anastomosis was untouched at the second operation and the ulcers could not therefore be ascribed to operative trauma. The same type of ulcer lesion was produced in the duodenum following transplantation of the common bile duct and the pancreatic duct into the terminal ileum. None of the ulcers healed spontaneously, but if the duodenum was reanastomosed to the jejunum close to the pylorus the ulcers healed, and this also occurred when the ulcer area was protected from the impingement of the gastric contents. Mann interprets his results as follows: (1) The operative procedure of draining the alkaline secretions of the duodenum away from the area of emergence of the gastric contents exposes the jejunal mucosa to an acid medium longer than normal. (2) The ulcer develops where the gastric contents primarily impinge on the jejunal wall. He thinks, however, that there must be some underlying primary change in the mucosa, such as infection, though why he thinks so he does not say.

Another less elaborate series of experiments was reported by Brancati.[18] He states that peptic ulcer developed in five out of ten dogs after resection of the pylorus and the pyloric part of the stomach. His experiments appeared to demonstrate that peptic ulcers follow removal of the pyloric part of the stomach and consequent early flooding of the duodenum with gastric secretions.

These experiments lend strong support to the view that, in duodenal ulcer at any rate, the gastric juice, when discharged rapidly into the first part of the duodenum and unneutralized by the duodenal secretions, is capable of initiating and developing ulceration, the ulcer forming where from impact the damage of the mucous membrane is maximal. In support of this view is the fact that surgeons are acquainted with a class of case in which all the symptoms of duodenal ulcer are present along with characteristic radiographic and test meal findings, which prove on operation to be examples not of developed ulcer but of a duodenitis, the wall of the gut showing hyperaemia and organized lymph on the serous coat without actual underlying ulceration.

It is also true that in almost all cases of actual duodenal ulcer there is also duodenitis; it is a common experience to find an ulcer on the posterior wall associated with definite signs of inflammatory reaction on the anterior wall, the serous coat being covered with a vascular organized lymph coating. In other cases, again, there is widespread duodenitis without ulcer. Recently I operated upon a case presenting a complete clinical picture of duodenal ulceration with corresponding radiographic findings and fractional test meal result. I found no ulcer, but the whole of the duodenum was hyperaemic and the first part was overlaid with a vascular lymph coating; the hyperaemic blush extended to the first part of the jejunum. Further, I was able to demonstrate a remarkably patulous pylorus; without any difficulty three fingers were invaginated

through it from the duodenal side into the stomach. With such a condition, given a stomach of high tonus, the gastric contents will early flood the duodenum.

The counterpart of this condition is the "blushing" stomach wall which is commonly associated with pyloric spasm and unduly long retention of gastric contents. This condition I have often observed, in the absence of ulceration, in cases of pyloric spasm and gastric delay associated with such conditions as chronic cholecystitis. The blush may be a vasomotor phenomenon, but I look upon it as probably caused by the irritation of retained gastric secretions; in cases of pyloric spasm it is most marked in the antrum pylori. Recently I operated upon a case of chronic cholecystitis, the chief symptom being recurring attacks of violent vomiting; the radiograph showed a considerable six-hour remainder; the fractional test meal showed a rapidly climbing acid level after the first hour; the pylorus was a tight ring with a lumen of pencil size, and over the antrum pylori was an intense blush sharply defined at the pylorus. Whether this blush is in fact a reaction to irritation, whether it is indicative of a gastritis, cannot, perhaps, be definitely stated, but this is, I think, the view generally held by surgeons.

Gastric Ulcer.

In considering whether this view of ulcer formation will explain the gastric ulcer, reference must be made to the views of Barclay.[19] He remarks that septic conditions in the mouth and from other sources, constipation, mucous colitis, and a variety of other conditions are capable of producing spasmodic contractions of various parts of the stomach. The spasm may be so severe that it produces a narrowing of the lumen which from time to time is of great functional importance, causing a definite obstruction to the passage of food. By some indiscretion of diet or want of mastication something too large to pass easily through the channel has to be forced through by peristalsis. This produces bruising or possibly an abrasion at the point where the lumen is narrowed and where there is constant irritation of food passing over it. It is probable also that the spasm interferes with the blood supply. A surface is exposed that is not structurally fitted to withstand the action of the gastric juice; in this way an ulcer is formed which perpetuates the original spasm that determined the site of the ulcer. Barclay stated that spasmodic hour-glass contraction of the stomach is quite common without ulcer; also that in fully half of the cases of stomach ulcer there is pyloric obstruction. It is not, perhaps, possible to accept the idea of direct mechanical injury by stomach contents, but the suggestion that the primary morbid condition is abnormal gastric spasmodic contractions is noteworthy as the opinion of an experienced radiologist.

Gastric ulcer occurs characteristically in the hypotonic J-shaped stomach. In this type the common alteration from the normal in motility is the appearance, at an early stage of digestion, of exaggerated peristalsis and contractions of the circular fibres of the pyloric region and antrum pylori. When this condition of exaggerated peristalsis is marked we have a type of "dyspepsia" in which early pain is the chief symptom. In ulcer of the lesser curvature the most constant abnormal muscular phenomenon is a spasmodic constriction about the mid-stomach; in ulcer about the pylorus the most constant muscular abnormality is spasmodic contraction of the pylorus itself.

In these spasmodic contractions, occurring in different situations, it is easy to see how the effect of impact in damaging mucous membrane, causing a local gastritis and eventual ulcer, may come into play. In persistent spasm of the stomach body the greater curvature is always drawn up to the fixed lesser curvature by the circular muscle fibres; under these conditions discharging gastric contents will impinge on the lesser curvature and may damage the mucous membrane sufficiently to pave the way for digestion by the gastric juice. The objection that the spasmodic contraction or incisura is the consequence and not the cause of ulcer formation cannot be maintained; radiographers and surgeons are agreed that a fixed spasm of the circular fibres, particularly at the pylorus or at the junction of the fundus and the antrum pylori, is a common finding in cases suspected of ulcer on account of gastric pain, but proved to be cases of gastric disorder from other causes. In such cases the point of impact of the gastric contents when the stomach contracts will be the lesser curvature near the pylorus or in the antrum pylori, and in these situations, of course, ulcer is most common.

The type of stomach in which gastric ulcer is most common has certain functional peculiarities. This stomach is usually hypotonic so far as peristaltic movements are concerned, but it exhibits an early pyloric spasm, which is clinically shown by early pain and radiographically by delayed emptying—that is to say, the stomach wall is subjected for an unduly long period to the presence of unneutralized gastric secretion. Its characteristics are the exact counterpart of those of the stomach in which duodenal ulcer tends to occur. They may be tabulated as follows:

In duodenal ulcer:	*In gastric ulcer:*
Hypertonus.	Hypotonus.
Patulous pylorus.	Spasm of pylorus or pars media.
Rapid emptying.	Slow emptying.

In the first case the result of the gastric functional disorder is that the duodenum is early flooded with unneutralized gastric secretions. In the second case the result is a retention of the stomach secretions, unneutralized, in the stomach itself. Bolton found that when there was a definite delay in the emptying of the stomach there was a definite delay in the healing of experimental ulcers. In either case it seems inevitable to conclude that the gastric juice plays a very definite role. That it does so is not only strongly supported by the experimental evidence of Bolton and of Mann, but also by the clinical experience of jejunal ulceration after gastro-enterostomy. Ryle has pointed out that it is particularly in the case of the sthenic or hypertonic stomach that jejunal ulcer is liable to occur, and the explanation appears to be that the ulceration develops in consequence of the flooding of the jejunum by gastric secretions at so early a stage that the alkaline duodenal secretion has not fully developed. The influence of impact* in determining the site of the ulceration is also well illustrated; jejunal ulcers develop either at the edge of the anastomotic ring or on the jejunal wall opposite to the anastomosis.

Trophic Influences.

If it be true that morbid alterations in gastric rhythm play the role in the production of gastric and duodenal ulceration that is suggested, it seems likely that these motor disturbances are associated with some trophic changes, and that such trophic changes may also assist in paving the way to ulcer formation. The work of Durante is interesting in this relation.

Durante[20] produced gastric ulceration in dogs by resection or ligation of the median splanchnic nerve. Two types of lesion occurred—a haemorrhagic lesion and a necrotic lesion. The haemorrhagic lesions healed, but the necrotic lesions were traced to ulceration which tended to become chronic. He ascribed these lesions to the action of adrenaline produced in excess by the operative stimulation of the sympathetic. He says: "ulcer may be produced by any agent capable of damaging the sympathetic nervous system, as it is on the integrity of this system, which controls circulation, secretion, and profound sensibility in the stomach, that the very life of the gastric cell may be said to depend."

The ulcers produced by Durante's operative procedure on the sympathetic appear to have been definite chronic ulcers resembling anatomically the chronic ulcer in the human subject.

Summary.

The conclusions to be drawn from this review are as follows:

1. There is no satisfactory evidence that chronic gastric and duodenal ulceration is primarily due to direct invasion of the stomach or duodenal wall by specific organisms.
2. Chronic gastric and duodenal ulceration is not caused by alterations in the chemistry of the gastric secretions.
3. The evidence available points to alteration in gastric rhythm being the primary morbid condition, which when persistent leads to the development of ulceration.

* Professor Glynn has suggested "wear and tear" as an alternative to "impact." It is certainly a better descriptive term.

4. The site of the ulceration is the site of maximum wear and tear by gastric contents whose normal neutralization has been interfered with by the altered gastric rhythm.

5. The gastric secretions, innocuous to the stomach wall under normal conditions, cause irritation and eventual ulceration, when the gastric rhythm is altered; trophic changes may also prepare the way for this action.

6. The fundamental condition on which these morbid changes in gastric motility and rhythm depend is doubtless a disorder of gastric innervation, which may have its origin in toxic influences, or psychic influences, or in reflex influences from disease elsewhere. The innervation of the stomach is not, however, so completely analysed as to make it possible to say whether these influences bear on an autonomic gastric system or whether they affect the stomach by way of the vagus or sympathetic. There is, however, some evidence that in the gastric type, which is usually associated with duodenal ulceration, there is some inhibition of impulses by the splanchnic path, and that in the gastric type, which is usually associated with gastric ulceration, it is the vagus impulse which is depressed.

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THE SURGICAL TREATMENT OF DANGLE-FOOT.*

BY

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IN dealing surgically with the residual paralysis of anterior poliomyelitis our endeavour must be to utilize to its best possible advantage the power which remains to us and to make the limb as efficient as possible.

In the leg, where weight has to be borne, we must attempt to provide stability and independence of extraneous supports. In drop-foot many operative procedures have attempted to suspend the foot by slings of silk, tendon fixations, and fascial bands, but the results have on the whole been disappointing, as the constant drag of the pendulous foot in time produces elongation of the artificial ligaments. Many such cases seem to be brilliant successes for a few months, but, seen a year or two later, have to be written off as ineffective.

The commission appointed by the American Orthopedic Association, which inquired into the stabilizing operations on the foot, reported that as regards drop-foot "none of the standard operative measures considered can be recommended as a standard procedure."

Two years ago I began to try a method devised by W. C. Campbell of Memphis, U.S.A., by which the interference with the drop-foot was not provided on the anterior aspect of the ankle in the form of a sling, but at the back of the joint by a "bone stop." In a large percentage of paralytic feet it is necessary to remove bone to correct deformity or to stabilize the mid-tarsal region and subastragaloid joint. The bone thus removed is utilized to build up a bone stop at the back of the ankle.

The usual incision for astragalectomy, and for most stabilizing operations, is used, commencing one inch above the ankle, internal to and parallel with the fibula, and passing down towards the external cuneiform. The deep fascia is divided; the extensor tendons are retracted inwards. The ligaments and periosteum are cut down to bone and the mid-tarsus exposed. The rounded articular surface of the head of the astragalus is removed by osteotome. A large part, or the whole, of the scaphoid is removed. I have performed this operation at the age of 8 years and upwards, and find that in younger subjects it is better to remove the whole scaphoid. The articular cartilage is now removed from the os calcis and cuboid with enough underlying bone to permit of good apposition of the mid-tarsus. The subastragaloid joints are next opened and the articular cartilages completely excised. In exposing these joints the pad of fat and connective tissue lying in the subastragaloid fossa should not be removed, but merely turned aside and later replaced so as to leave no "dead space." The removed portions of bone and cartilage are placed in saline, and, while the anterior incision is being closed, an assistant cuts away all cartilage and prepares the bony pieces for the construction of the "stop" behind the joint.

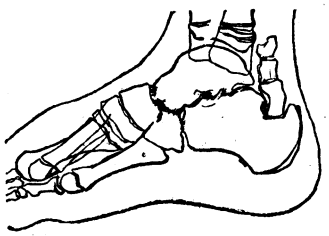

FIG. 1.—A lateral radiographic view of a foot a week after operation; the scaphoid, with its superimposed fragments, can be clearly identified.

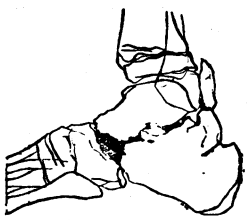

FIG. 2.—Shows the foot three months after operation, when the fusion of the various elements is seen to be complete.

The posterior incision is now made about six inches long directly over the tendo Achillis. The tendon is cleared and divided as for a Z-lengthening operation. The proximal and distal ends are turned up and down. The loose tissue lying between the tendo Achillis and the back of the tibia is now incised in a vertical direction and the back of the tibia and the upper surface of the os calcis exposed. A notch is cut in the os calcis large enough to receive the broader end of the trimmed scaphoid. This is placed in position and tapped down so as to get a hold. The smaller pieces of bone are now grouped together above the upper end of the scaphoid and the suturing of the tendo Achillis holds them nicely in place. The required amount of lengthening of the tendon is made so as to allow a right-angled position of the ankle. The skin is closed and a light plaster cast applied for six weeks. At the end of this time a brace is fitted so as to prevent any movement in the direction of plantar flexion for six months. I am in the habit of using a posterior iron for this purpose, as advocated by Campbell, and find it very effective.

I have now operated on nineteen cases in this manner. The operation which I am to perform at the Children's Hospital this afternoon will be the twentieth of the series. Two patients have had a secondary operation for repair of the bone stop, which had fractured at a point where the scaphoid joined the other fragments, a few months after use of the foot had been permitted. Repair was carried out by means of a small bone pin driven through the stop. If any later cases need repair I intend to cut a strip of bone from the back of the tibia for the purpose. I shall also show this afternoon eight patients upon whom I did this operation more than a year ago, so that you will be able to judge of the stability of the feet and of the effect of the "bone stop" in preventing drop-foot.

* Read before the meeting of the British Orthopaedic Association in October, 1925, when the operation was demonstrated and a series of results shown at the Royal Manchester Children's Hospital.

THE TREATMENT OF AMOEBIC DYSENTERY BY AUREMETINE.

BY

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AND

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IN a paper[1] read by us before the Royal Society of Tropical Medicine and Hygiene in May, 1923, the treatment and the results in a series of 503 cases of amoebic dysentery were detailed. It was shown that what might be termed the standard treatment in those days—consisting of 12 grains of emetine by intramuscular injection and 60 grains of emetine bismuthous iodide by the mouth, given concurrently in twenty days—was followed by a cure (as to the duration of which it was possible only to speculate) in less than one-third of the cases, and we