tissue than are x rays, whilst its effect on the uterine muscle is greater.

Taussig states that "radium is less likely to produce vomiting, cramps, and catarrhal stools," and that it will induce amenorrhoea when x rays have failed. In the use of radium we have a better conception of dosage than when x rays are employed.

My own experience with the use of radium in the treatment of haemorrhage has been in chronic metritis, and the results have been so favourable that I shall be tempted to extend its application to the treatment of haemorrhage in cases of small uncomplicated uterine fibroids at the time of the climacteric—in other words, for this group of cases I regard it as legitimate to consider the use of radium as an alternative to hysterectomy.

In conclusion, if asked "In what cases do uterine fibroids still require operative removal?" my answer would be, In 55 per cent. of all fibroids no treatment whatever is needed. In the remaining 45 per cent., 35 per cent. require removal, leaving 10 per cent. to be dealt with by radium. This is equivalent to stating that the field for operation is from three to four times as great as that for radiotherapy.

In the treatment of uterine fibroids surgery and radiotherapy may be run tandem—but to radiotherapy we assign a very subsidiary status.

THE PATHOLOGY AND SYMPTOMS OF DUODENAL ULCER.*

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In contrast to gastric ulcer, increased activity of the stomach, both motor and secretory, is a characteristic feature in subjects of duodenal ulcer. Indeed, this increased activity (the duodenal type of stomach) may be regarded as the essential predisposing condition, and depends on the derangement of the normal neuro-muscular mechanism of the stomach.

Neuro-muscular Mechanism.

Though this is as yet imperfectly understood, clinical evidence is of the greatest assistance to a correct understanding. There are three points to be emphasized:

1. True peristalsis (that is, the combination of a deeply contracting segment immediately proximal to a synchronously inhibited segment) is seen neither in the oesophagus nor in the fundus of the stomach, but commences approximately at A-B (see figure), as proved both by radiography and direct inspection during laparotomy. True peristalsis is characteristic of those parts of the gastro-intestinal tract which are influenced by a dual antagonistic nerve control—(1) *motor* (that is, parasympathetic), acting through Auerbach's plexus; and (2) *inhibitory* (that is, sympathetic), acting perhaps through Meissner's plexus.¹ The inference is that the sympathetic only enters on its inhibitory functions at A-B. This receives support from a consideration of the second point.

2. In his study of Auerbach's plexus Keith² described a nodal point at A (see figure), and found that mechanical stimulation evoked a contraction on the greater curvature. That an ulcer in this situation produces the most marked spasmodic hour-glass stomach indicates that in both the stimulation of Auerbach's plexus at this point excites the greatest motor response, because there is here little or no counterbalancing sympathetic excitation to inhibit this phenomenon. The spasmodic hour-glass stomach opposite an ulcer at A may be obliterated by "pulling" during laparotomy, and will often repeatedly re-form (a feature progressively less pronounced as the duodenum is approached); but an incisure opposite an ulcer in the intestine is absolutely unknown, and manipulation only induces inhibition.

3. X-ray observations demonstrate a normal period of delay in the passage of food through the pylorus—a delay accentuated by over-excitation of the sympathetic (that is, by apprehension, anxiety, etc.), and associated with diminished secretory activity (the antithesis of "the duodenal stomach"). Perhaps I may be allowed to emphasize again³ that clinical evidence supports the

view that pyloric closure is controlled by the sympathetic, as Elliott⁴ has shown to be the case in the ileo-caecal valve. It is reasonable, then, to regard the fundus of the stomach, proximal to A-B, as a dilated pouch of the oesophagus controlled by the vagus alone. From this point onwards the sympathetic enters on its antagonistic inhibitory control with progressively increasing domination.

On these grounds we may assume that predisposition to a duodenal ulcer ("the duodenal stomach") is hyperactivity, due to a relative increase of normal vagus control, insufficiently balanced by normal sympathetic inhibition.

The pathology of duodenal ulcer is dependent, then, on two sets of factors—one predisposing and the other exciting.

PREDISPOSING FACTORS.

The predisposing factor is the relative increase of vagus excitation established by diminution of sympathetic control or by increased vagus stimulation.

Diminution of Sympathetic Control.

Now there is an outstanding example of an alkaloid which paralyzes sympathetic ganglion cells—that is, nicotine. On these grounds I have for some years prohibited smoking in cases of duodenal ulcer.

(a) *Influence of Tobacco.*—The proportion of men to women is roughly four or five to one (in itself perhaps some indication of the influence of nicotine), and 96 per cent. of the males were smokers. I have endeavoured to divide the smokers into three groups: excessive, heavy or continuous (together comprising 60 per cent.), and mild smokers (40 per cent.) in whom it was not a fixed habit.

Of the smokers, 22 per cent. abandoned the habit, or nearly so, with the result that, independently of the character of the surgical treatment instituted, 90 per cent. were cured, 8 per cent. greatly relieved (98 per cent.), and 2 per cent. only failed. A mild reminder of the original symptoms was voluntarily attributed to tobacco in 5 per cent. of these. Where tobacco was unchecked, only 47 per cent. were cured, and 12 per cent. recurred. Recurrence is four times as frequent in non-abstainers. The comparison is still more striking in Table I.

TABLE I.

	Abstained.	Habit Unaltered.
Excessive smokers (29%)	Cured ... 90% Relieved ... 8% Recurred... 0 Failure ... 0	Cured ... 33% Relieved ... 15% Recurred...=33% Failure ...=13%
Heavy or continuous smokers (31%)	Cured ... 55% Relieved ... 45% Recurred... 0	Cured ... 44% Relieved ... 33% Recurred...=22%

In mild or occasional smokers the influence on the result is negligible, while 14 per cent. of those who did not vary their consumption volunteer that their "reminders," or occasional "stomach upsets," are attributable to tobacco. In one instance recurrence after six years' complete immunity following operation was definitely attributable to gross over-indulgence. The influence of tobacco in the development of duodenal ulcer will be further elaborated, but mention must be made here of a possible second factor in the diminution of sympathetic control.

(b) *Sex.*—I have long noted that symptoms of visceral disorders in women are often either accentuated or relieved by the onset of the climacteric. Now we know that the suprarenal medullary hormone is an adjuvant to sympathetic nerve stimulation; and, since the antagonistic vegetative nerve impulses normally balance each other, it is reasonable to infer that their chemical associates similarly contribute to the establishment of a physiological equilibrium. Falta (quoted by Higier)⁵ classifies the hormone of the sex glands with the adrenal medullary hormone; and it is here suggested that perhaps the loss of this ovarian inhibitory influence at the climacteric may contribute to

* A lecture delivered to the Nottingham Medical Society, February 20th, 1924.

the vagus domination characteristic of the "duodenal stomach." Thus, in females, tobacco can be excluded in all but a doubtful 12 per cent.; but, allowing for occasional mild dyspepsia, definite ulcer symptoms appeared at an average age of 45, as against 37 in men. In thin women mesenteric lesions (which will be next considered) could only account for ulcer predisposition in 22 per cent., while in 33 per cent. the onset coincided approximately with the menopause; therefore the establishment of the "duodenal stomach" may perhaps be accounted for in 55 per cent. of this class. In stout women (in whom the mesenteric factor is usually lacking) 57 per cent. date the onset of ulcer symptoms approximately at the menopause. The figures are curiously similar in the two groups. There is further evidence of abnormal marital relations (two cases); artificial menopause (one case); premature death of the husband (one case); all of which perhaps strengthen the hypothesis. It is interesting that in one instance symptoms disappeared during pregnancy (a small ulcer recently inflamed).

Increased Vagus Excitation.

The vagus is the motor nerve as far as the distal third of the transverse colon, the situation of one of Keith's nodes;² and it is reasonable to suppose that reflex vagus excitation is most readily elicited by stimuli (either inflammatory or mechanical) in the area of vagus supply. Tuberculous ileal glands, appendix adherent to the ileal mesentery, "Lane's kink," etc., are all examples of irritation of the sensitive mesentery. I have elsewhere pointed out that the resulting symptoms are but a mild mimicry of the severe manifestations of acute infective conditions when the mesentery is involved in the inflammatory process.¹ Prolapse of the right colon similarly leads to excessive excitation when the mesentery (with its contained nerves) assumes supporting functions. Usually general visceroptosis similarly excites a dominating sympathetic response; though, if sympathetic control be diminished as already suggested, it may contribute to vagus domination. Five years ago the reflex effects on the stomach of such morbid conditions were emphasized in detail;¹ the present analysis supports the view that these are definite predisposing factors in establishing vagus domination in the stomach.

Mesenteric Lesions.—Amongst those mentioned, prominence must be given to prolapse of the right colon, commonly seen in thin subjects, less often in stout subjects with well supported viscera. Now, the sympathetic effects of such conditions on the intestine are inhibitory, and they will be manifested by constipation; this symptom, then, may broadly be taken as evidence of the existence of a mesenteric lesion. Again, all types of subjects do not run a similar clinical course. Accordingly I have divided the cases into three groups: a fat type (constipation noted in 29 per cent.); a medium (athletic) type (constipation noted in 36 per cent.); and a thin type (constipation noted in 46 per cent.). Constipation is noted in 58 per cent. of the total; and since tobacco exerts little influence in this respect (rather the contrary), it is significant that 60 per cent. of men were heavy or continuous smokers. Now, such lesions in the vagus area also stimulate the sympathetic, which, I have shown, usually dominates.¹ The effect of nicotine should be to reverse this effect; and, in addition to evidence already adduced, the following facts are significant:

Of the three types the percentage of cures is approximately the same (that is, 60 to 65 per cent.); but abstinence from tobacco after operation is definitely associated with the cure in 63 per cent. of stout subjects, 40 per cent. of medium build, and only 6 per cent. in the thin type. If we classify together those cured and greatly relieved, and consider stout and medium types together, 83 per cent. were cured, and tobacco influenced the result in 53 per cent. In thin subjects 80 per cent. were also cured, but in only 33 per cent. could any influence of tobacco be traced.

It may be inferred that tobacco is the most prominent predisposing factor in stout types with well fixed viscera; and in thin subjects with visceroptosis mesenteric lesions figure most prominently in the etiology. In the latter tobacco is less harmful, because sympathetic domination is normally marked.

The analysis of various operative procedures supports these deductions:

Gastro-enterostomy Alone.—Excluding perforation, an equal proportion were cured or greatly relieved. In stout or sturdy

subjects about 50 per cent. were cured or nearly so—a result influenced by tobacco in 30 per cent. Thin subjects present a marked contrast (Table II).

TABLE II.

	Cured.	Cured and Much Relieved.
Stout and medium.	46% { Tobacco influence 80% Short history ... 20%	72% { Tobacco influence 50% Short history ... 9%
Thin.	60% { Tobacco influence 0% Short history ... 66%	100% { Tobacco influence 0% Short history ... 40%

It is clear that gastro-enterostomy alone, together with subsequent abstinence from tobacco, will usually prove successful in stout subjects; this is especially so when vomiting is a prominent symptom (70 per cent.), but in the thin type a search for, and remedy of, a remote mesenteric lesion is indicated, as well as a reduction of tobacco. This is supported by the fact that of the total recurrences and failures nothing beyond the short circuit was done in 50 per cent.; while the 40 per cent. partial successes in thin subjects are attributable to the failure to remedy associated mesenteric abnormalities.

Gastro-enterostomy with invagination of the ulcer (60 per cent.) should be practised wherever possible. Thus 64 per cent. were completely cured in a total of 85 per cent. satisfactory results. There were no recurrences. Of the total partial successes, in 33 per cent. the ulcer was not buried. Of the 60 per cent. cures in stout subjects, in 40 per cent. the ulcer was buried; in thin subjects 82 per cent. were cured, and the ulcer was buried in 87 per cent.

Gastro-enterostomy together with Associated Mesenteric Procedures.—Excluding cases of obstruction, Table III confirms the view that caecopexy, ascending colopexy, plastic release of Lane's kink (and its analogue, the so-called "controlling appendix"), etc., by removing any cause of vagus over-excitation, contribute to the completeness of the result of gastro-enterostomy with invagination of the ulcer, particularly in thin subjects. Further, of the total complete cures, caecopexy or ascending colopexy (according to findings) was performed in 26 per cent., and in 23 per cent. of those greatly relieved; while the only recurrence when gastro-enterostomy and colopexy were combined was due to tobacco beyond all question.

TABLE III.—Cured and Relieved.

	Cured.		Relieved.	
	Stout and Medium (60%).	Thin (70%).	Stout and Medium (26%).	Thin (21%).
Ascending colopexy...	11%	45%	25%	20%
Other mesenteric procedure in the vagus area	18% } 29%	6% } 51%	— } 25%	40% } 60%
Appendicectomy only, when no gross lesion is reported	30%	—	—	—
Gastro-enterostomy alone	41%	19%	50%	—

When the influence of tobacco in stout and medium types is recalled, it is significant that when mesenteric procedures were combined (29 per cent.) 60 per cent. were cured, while in thin subjects this combination in 51 per cent. gave 70 per cent. cures. It is also significant that gastro-enterostomy alone cured 41 per cent. of sturdy and only 19 per cent. of thin subjects.

This means that prolapse of caecum and ascending colon may lead to vagus over-excitation by mechanical trauma of the mesentery,¹ and that inflammatory lesions produce a similar effect. While these are prominent predisposing factors in thin subjects, tobacco is the most prominent factor in the sturdy, though it is important in both.

Incidentally the figures afford an independent testimony to the value of various "fixation operations," when designed to protect the mesentery from mechanical trauma.^{1 6}

The conclusion to be drawn from the foregoing is that predisposition to a duodenal ulcer consists in a relative increase of vagus domination, leading to excessive motility, excessive secretion, and incompetence of the pyloric sphincter.

EXCITING FACTORS.

The exciting factors may be enumerated as follows: Acid, distension, trauma, infection.

Acid, in the absence of pyloric control, has free and continuous access to the first part of the duodenum, in a persistent degree defying normal neutralization. The effect of acid on injured tissues has been emphasized by Sherren,⁷ Patterson,⁸ and others, while the numerous and exact experimental and clinical observations of Bolton⁹ have established the effect of acid as a primary exciting factor in gastric ulcer. The effect of acid on healed ulcers is common clinical experience—they are always likely to break down and lead to secondary ulceration. Bolton¹⁰ has shown that the high acidity in duodenal ulcer is largely due to defective regurgitation. He assumes that this phenomenon is due to pyloric closure, but I shall endeavour to show that it is prevented by the closure of the duodenum at the site of the ulcer by the incisura. Obstruction here further exposes the ulcer to continued irritation by acid, trauma, etc., and favours persistence. Pyloric closure would protect the ulcer and tend to assist healing—clinically this is not the case.

Distension, resulting from an incompetent pylorus, is constantly seen during operation; it probably interferes with normal circulation.

Trauma.—The rapid rush of insufficiently digested food through an incompetent pylorus invites trauma of the duodenum; just as, with the closed pylorus, trauma is a factor in exciting pyloric ulcer.

Infection may take effect in three ways: (a) Direct. Normally a short stay in the acid contents of the stomach does much to negative direct infection; in the "duodenal stomach" organisms are passed rapidly into the duodenum; I believe this route of infection to be commonest. (b) By the blood stream. This is parallel to acute blood infections of the appendix, gall bladder, and intestine (for example, Henoch's purpura, etc.).¹¹ Wilkie¹² suggests retrograde venous embolism as a cause. The embolic origin of perforating ulcers is, in my belief, common, and constitutes a different type of ulcer. Thus I estimate that only 13 per cent. of perforations give a long-standing history of definite ulceration; while some (unlike the long-standing ulcer) show a permanent recovery with invagination alone. In this respect the length of history related to the type and tobacco habit is instructive. Excluding perforation, about 25 per cent. of sturdy subjects had symptoms for less than six months (also under one year); in 67 per cent. there was over two years' history. In thin subjects the figures were reversed—that is, under six months, 44 per cent. (total under a year, 66 per cent.); over two years, 33 per cent. Table IV (length of history related to tobacco) shows curiously similar figures.

TABLE IV.

	Excessive or Heavy Smokers.	Stout and Medium Subjects.	Moderate or Light Smokers.	Thin Subjects.
	Per cent.	Per cent.	Per cent.	Per cent.
History of six months and under	23	25	45	44
History of two years and over	67	67	40	33

From this it may be inferred that the massive and extensive "tobacco ulcer" characteristic of stout subjects produces its effects slowly; and such ulcers run a chronic infiltrating course and seldom lead to acute perforation. Conversely the small puckered ulcer, less influenced by nicotine, and commonly found in medium and thin subjects, runs a more rapid course and is more liable to acute perforation. (c) The possibility of lymphatic infection has been emphasized by Braithwaite.¹³ The difficulty experienced in demonstrating this route would seem to indicate its infrequency. The frequency of infection from the appendix has, I believe, been exaggerated.

Although alcohol undoubtedly exaggerates the symptoms, I have been unable to get positive evidence that it is an exciting cause.

SYMPTOMS.

A study of the commoner symptoms in relation to type, character of lesion, habits, and result of treatment, is instructive.

"Heartburn" and "waterbrash" is present in 23 per cent. of cases, of which 27 per cent. are smokers and 7 per cent. abstainers. The proportions do not differ materially in the three types. Tobacco is a prominent cause. After operation on the sturdy type these "reminders" could be controlled in 30 per cent. by a limitation of tobacco. This was only achieved in 10 per cent. of thin subjects. Further, of the 54 per cent. of cures in smokers 23 per cent. abstained

from the habit after operation; and it is probable that at least 25 per cent. of reminders are due to tobacco. The relationship of tobacco to hypersecretion has recently been emphasized by Moynihan. Heartburn should be regarded as indicating a predisposition to ulcer, but not as symptomatic of ulceration.

Pain.

Pain may be absent (10 per cent.), atypical (that is, not related to food), or conform to the typical "hunger pain." The variability of this symptom in relation to the constancy of the lesion indicates that the ulcer is itself insensitive, and this view is strengthened by the wealth of clinical evidence relating to the insensitiveness of the gastro-intestinal tract.¹⁴

Atypical pain may be present alone, or associated with (but distinct from) bouts of hunger pain in 72 per cent. of cases; of these, 40 per cent. were stout, 31 per cent. were medium, and only 18 per cent. were thin subjects. In this group the large tobacco ulcer (characteristic of large subjects) claims 54 per cent., the small puckered ulcer only 18 per cent. (as a contrast, note the percentages in hunger pain). In the large ulcer atypical pain is detailed as follows: violent or continuous epigastric pain 50 per cent., continuous or not related to food 35 per cent., ordinary "colic" and typical "gall-stone colic" types 7 per cent. of each. In the small ulcer the details are: acute epigastric pain 60 per cent., shortly after food 20 per cent., variable 20 per cent. Thus atypical pain seems dissimilar in these two types.

Remission of symptoms is characteristic of most varieties, but certain points may be emphasized. In the small ulcer remissions are often due to alternate healing and breaking down, as evidenced by the frequency with which the occult blood test may be negative in a quiescent period. In the large ulcer they suggest that a callous, insensitive ulcer (for example, varicose ulcer of the leg) is subject to recurrent inflammation; and the secondary hyperaemia of the adjacent sensitive mesentery and peritoneum is responsible for the symptom of pain, not necessarily related to food, during the periods of inflammation. Continuous pain in stout subjects (35 per cent.) is an example of this. In a small proportion pain is not related to the ulcer but to the factors contributing to its existence. It should be emphasized that, in contrast to the common type of ulcer, the large tobacco ulcer is not usually associated with hunger pain as a prominent symptom.

Hunger pain (60 to 65 per cent.) is the classical symptom of duodenal ulcer (Moynihan). It is not directly influenced by tobacco; nor is it due to any sensation in the ulcer. If the ulcer were responsible for the symptom, the larger ulcer should accentuate the hunger pain. The reverse is the case (Table V).

TABLE V.

Large Ulcer.		Small Ulcer.	
Hunger pain present = 40%.		Hunger pain present = 80%.	
(a) Prominent symptom ...	27%	(a) Prominent symptom ...	70%
(b) Occasional symptom ...	8%	(b) Occasional symptom ...	7%
(c) "Hungry feeling" ...	4%	(c) "Hungry feeling" ...	3%

In marked contrast to the large "tobacco ulcer" of stout subjects, hunger pain is characteristic of the small puckered ulcer of thin and medium subjects (in whom tobacco plays little part). This is supported by figures from an entirely different standpoint. Thus 68 per cent. of those complaining of hunger pain were completely cured; and this is practically identical with the proportion of cures in mild smokers who did not vary the habit after operation; of these only 23 per cent. were stout and 42 per cent. thin.

Pathology of Hunger Pain.

Increasing experience confirms the conviction that several factors are involved in the causation of this symptom, the significance of which depends on an appreciation of the mechanism of physiological hunger. Five years ago¹ I suggested that hunger is not, as hitherto believed, initiated by, but coincident with, the onset of gastric movements. Thus, as the fully oxygenated alveolus stimulates the afferent vagus to inhibit inspiration, so do the engorged gastric and pancreatic glands similarly excite the afferent vagus, while the efferent response initiates the movements coincident with hunger. The contraction of superficial blood vessels (and the consequent engorgement of the deeper ones) in cold weather was emphasized as contributing to an increased blood supply to, and a more rapid anabolism in, these glands; with the result that increased hunger is a feature of cold weather.

Now it is common knowledge that the gradual evolution of physiological hunger to the pain of starvation is only a question of degree; and it must first be premised that pain in the latter may often be an extreme exaggeration of the former. Three factors, then, may be emphasized as responsible: Mechanical, vascular, and neuro-muscular.

The Mechanical Factor.—There are many ways in which the movements coincident with "hunger" may irritate mechanically sensitive structures. If such stimuli arise in the vagus area they may, in addition, accentuate this symptom by the resulting anabolic effects of efferent vagus stimulation upon the glands mentioned. This mechanism is best illustrated by a case of some interest.

Some years ago a girl of 20 complained of very acute bouts of hunger pain (see figure). All investigations completely negated the existence of ulcer. Fortunately I explored her under spinal anaesthesia, for the only abnormality was the attachment (D) of a prolapsed caecum and ascending colon to the proximal transverse colon (a-b), which at a point opposite was supported by bands (c) attached to the sensitive peritoneum external to the duodenum (T). Three consecutive mass movements demonstrated that the caecum and ascending colon, during contraction, assumed the position of the dotted outline (E) with resulting tension on the proximal transverse colon, and therefrom on the point T.

FIG. 1.—a-b, inhibited area of transverse colon during mass movement. G-H and G'-H', altered position of inhibited area during contraction. T, the point where traction was manifest during contraction. C, bands attaching transverse colon to peritoneum near duodenum at T. D, attachment of caecum to proximal transverse colon. E, dotted lines showing altered position of caecum, and ascending colon during contraction.

The contraction stopped at a. A repetition of the phenomenon confirmed the observation. The third contraction again stopped at a, but on this occasion it "skipped" the segment a-b (which was at no time observed to contract) and recommenced at b. The inference was that in the segment a-b contraction was inhibited by mechanical traction. Such a mechanism, started with the movements of hunger, could well account for hunger pain, but in addition this was probably accentuated by the contraction driving the "inert segment" onwards (G-H, G'-H') and increasing the tension on T. On these grounds the caecum and ascending colon were freed from the transverse colon and ascending colopexy performed. A permanent cure resulted.

It need only be added that hunger discomfort, at times amounting to pain, is not uncommonly seen in thin subjects with peritoneal or mesenteric abnormalities when it is certain that no ulcer exists. Finally, such cases support the view that this symptom sometimes arises from the mesenteric factors contributing to the formation of the ulcer rather than from the ulcer itself.

The Vascular Factor.—If hyperaemia induced by cold weather, and, incidentally, the analogous artificial hyperaemia induced by alcohol (for example, "cocktails"), can accelerate glandular storage and accentuate hunger, it may be inferred that hyperaemia from inflammation can excite a similar effect. All surgeons are familiar with the free vascular connexions of the duodenum with the head of the pancreas (a feature not normally observed in relation to the body of the stomach), and there is little doubt that increased vascularity of the duodenum can be readily reflected in that of the head of the pancreas. Again, it is not very uncommon to find hunger pain associated with gastric ulcer. I have noted that in such cases the ulcer is high up and connected with the pancreas by loose vascular adhesions. It is significant that I have never known hunger pain associated with the hard callous ulcer infiltrating the pancreas, presumably because the surrounding ring of fibrous tissue effectively prevents any vascular connexion between the two. That the clinical accentuation of symptoms in winter is so common in cases of duodenal ulcer confirms the vascular hypothesis. Accordingly it is suggested that increased glandular storage

from hyperaemia, the result of inflammation in direct vascular connexion with the pancreas, is an accessory factor in the causation of hunger pain, particularly in duodenal ulcer.

The Neuro-muscular Factor.—An ulcer limited to the duodenum constitutes an inflammatory lesion of an insensitive structure and, as such, is devoid of sensation. But inflamed muscle does not contract. Such lesions, then, form an "inert area" which, I have elsewhere shown, "breaks into" the co-ordinated sequence of contraction and inhibition which together constitute the peristaltic wave. The intestine treats such "inert areas" (of which examples are numerous) as foreign bodies, and drives them down the intestinal tract. The attempted invagination is prevented by the anchorage of the sensitive mesentery; in the case of duodenal ulcer, by the sensitive peritoneum (analogous to the tension at T in the case described in the figure). If this conception is correct the small free ulcer, in which attempted invagination is easier of accomplishment, should be prominently associated with hunger pain. This is borne out by the fact that hunger pain is prominent in 80 per cent. of small and only 40 per cent. of large ulcers, while this symptom is infrequent in the large tobacco ulcer.

These observations leave no doubt that the ulcer constitutes an insensitive lesion of an insensitive tract, and that the characteristic hunger pain is caused by a combination of the three factors discussed.

Vomiting.

Vomiting (67 per cent.) was twice as frequent in the stout and medium types as in the thin type. It has been indicated that the inflammatory lesion irritates locally both Auerbach's plexus and the sympathetic. When the latter influence is diminished (or absent) the cells of Auerbach stimulate the muscle to contract (the "ulcer incisura"). Therefore the more extensive the ulcer the more marked will be the incisura, and the more complete the spasmodic obstruction. Since gastro-enterostomy is most effective in the presence of obstruction, the success of this operation should afford proof that the obstruction is spasmodic and is caused by the incisura (not, as Bolton¹⁰ claims, by "pyloric spasm"). Accordingly it is instructive to find that when vomiting was a symptom 90 per cent. of stout subjects were cured and 10 per cent. greatly relieved (100 per cent. satisfactory results). It should be noted that the large ulcer is characteristic of stout subjects. As a contrast only 66 per cent. of thin subjects (small ulcer) were cured and 20 per cent. relieved. (Allowing for the good effect of associated mesenteric procedures in these, the latter figures may well be even smaller.) That the obstruction (and prevention of normal regurgitation) is at the site of the ulcer (whether spasmodic or organic), and not at the pylorus, is confirmed by considering haematemesis.

Haematemesis.

Haematemesis (17 per cent.) occurred in 30 per cent. of stout subjects (large ulcer), 18 per cent. of medium, and in only 7 per cent. of the thin type (small ulcer). If the pylorus were closed melaena and haematemesis would be equally common to all types of subjects, and would not vary with the character of the ulcer. The fact that haematemesis was over four times as frequent in the stout subjects with the large ulcer (which is more liable to haemorrhage) than in the thin subject with the small ulcer (nearly seven times as frequent in stout and medium) indicates that the pylorus is patent and the obstruction lies distal to it at the site of the ulcer.

TREATMENT.

Discussion of the different methods of treatment is not possible within available limits; but there are one or two points deserving of mention. The effect of acid on scar tissue has been referred to, and the liability of such scars to subsequent ulceration. It is probable that ulceration deep enough to invade Auerbach's plexus will rarely heal without some degree of scarring (even if it is microscopic in degree). The invasion of Auerbach's plexus will always result in distortion of the "duodenal cap," as demonstrated by expert radiography; and if these premises be accepted, it is probable that permanent cure cannot be expected with certainty by other than surgical measures.

If, as already suggested, acute perforation is more probable in the free small ulcer with the recent history, early operation is the sound advice; and this receives some support from a study of the results in relation to the

length of history of ulcer symptoms. Here the tobacco factor must be allowed for.

Thus, if those with a history of one year and under be compared with those who have suffered for over two years, there is some evidence that earlier treatment is likely to yield the best results. For in the first class 71 per cent. were cured (tobacco was a factor in only 20 per cent.); whereas in the second class 52 per cent. were cured (tobacco influenced the result in 38 per cent.). There were no recurrences in the first group and 10 per cent. in the second, in 5 per cent. of which tobacco may be blamed. The failures were 5 to 7 per cent.; and whereas tobacco could fairly be held responsible in all of the first group, no such influence could be traced in the second.

Gastro-enterostomy forms an essential part of the treatment of the ulcer in the majority of cases, as it permits regurgitation of bile and diminishes intragastric and duodenal tension, and the ulcer should be invaginated whenever possible. In the thin type the associated remedy of mesenteric and peritoneal abnormalities is indicated. In all types the reduction of or abstinence from tobacco is essential.

CONCLUSIONS.

The predisposition to duodenal ulcer consists in the relative increase of the normal vagus stimulation, which establishes the "duodenal stomach." Tobacco is the outstanding cause. Factors which may excite ulceration in such a type of stomach are: (1) acid, (2) distension, (3) trauma, (4) infection, and possibly (5) alcohol; the effect of all these in producing ulceration is accentuated by an incompetent pylorus. Any combination of these exciting factors may determine ulcer formation in such a type of stomach; but in the "sympathetic type" with the closed pylorus (the antithesis) a gastric ulcer is the more probable result.

I should like to express my gratitude to many colleagues who have allowed me free access to the notes of their cases; and to add that in this study percentages have only been adopted as the simplest means of comparison.

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TREATMENT OF TUBERCULOSIS OF BONES AND JOINTS.*

BY

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TWENTY years ago tuberculous disease attacking the spine or the hip dealt hardly with the bodies of its victims; only too commonly the story was one of persistent pain, increasing deformity, sinuses profusely discharging, the dragging out of months, then lardaceous disease with its added miseries, and at the last end, death. But now there is little excuse for delay in diagnosis or prompt admission to a special hospital; and if admitted in good time the patient quickly loses all his pain, ceases to go downhill, and starts the slow process of recovery. This review must cover first the conception of the disease, then the general treatment of the patient, and finally the local treatment of bone or joint focus.

THE CHARACTER OF THE DISEASE.

Spinal caries—or any other bone or joint focus—is an obvious manifestation of a hidden deep-rooted disease.

Tuberculous disease of a joint or a bone is serious enough, but it is a weed with a very deep root; you may pluck the head, but the root will not come with it. A bone focus is a proof of established tuberculous infection of the body; but even this is not all, for the new focus is tangible evidence that the invasion of the body is advancing. For

months or years trench warfare has been carried on in the deep-seated lymph glands; from time to time there have been attacks, bacilli have been distributed in showers by the blood stream, but the tissue cells have known how to deal with them. But now things are going wrong.

The rival powers of man and tubercle bacillus seem very delicately balanced. The virulence of the tubercle bacillus in bone and joint disease is almost invariably constant.¹ Man is the variable factor. In good circumstances he has the upper hand; under sufficiently bad conditions he becomes its prey. But this is a fight in which we can join, and treatment has now become so effective that we can almost ensure victory. We can decide whether the patient will almost certainly recover or run very grave risk. This responsibility rests jointly upon those on whom it falls to authorize the admission to hospital and pay the maintenance, and upon those who carry out the treatment.

But success costs time and money. At present these necessities are often dealt out to us so grudgingly that we are driven to make a fine art of spending the smallest possible sum on the treatment of each patient. This makes our task not only very difficult, but often very unsatisfactory; for while it is comparatively easy to restore health in an open-air hospital, it is difficult to maintain that health after the patient goes home, and often impossible if he goes too soon. Similar causes produce similar results: a deep-seated tuberculous infection, and his home conditions, produced the focus for which our patient came into hospital. If he goes back with the infection inconclusively checked, and his home conditions are unimproved, his vitality and powers of resistance are likely to return to a low level. Further showers of bacilli may follow and lead to a new focus, or the old focus may be lit up again.

A cure in one hospital's register may be a very sick patient in another hospital's bed. This is tragical for the patients, misleads the surgeons, and falsifies statistics. But it is an experience that is disappearing where the hospital staff make certain of maintaining constant supervision of their cases after they go home.

THE TREATMENT OF THE DISEASE.

- I. First to raise the patient's general vitality as quickly and as fully as possible.
- II. Ultimately to restore the full health of the patient, who should be free from any sign of tuberculosis, focal or general, with his powers of resistance to the tubercle bacillus so raised as to enable him to repel any further attack.
- III. The prompt elimination of any factor, such as strain or movement, harmful to the diseased part, and the application, so far as possible, of rest, comfort, and stimulus to local circulation.
- IV. The avoidance of septic infection.
- V. The restoration of the strength and function of the affected part.

These points are best achieved by prompt admission of the patients to an open-air orthopaedic hospital. The development of perfect technical methods of treatment is the first duty of the hospital staff, the organization of team work to bring the patient promptly to that treatment is another, and for this they must rely on the help of the doctors of the district as well as of the tuberculosis officers. A little delay, then a little postponement, and we may find the disease has become, to quote the words of Percival Pott, "Most melancholy to see, most miserable to endure." We can neither afford delay nor to remit any established principle of treatment. The virtues of sunlight and moving air should not induce us to relinquish the accuracy of our splintage. Operations may be helpful, or even essential, to cure, but to consider them as short cuts is a sin which will be visited on our patients. Rest and time remain the essential ingredients of our prescription.²

General Treatment.

General treatment comprises the skilled use of four great natural remedies: *rest, good feeding, open air, and sufficient time*. Of the rest and the time, Hilton and Thomas have said all that is necessary—the one with charm, the other with emphasis.

Good Feeding.—Food must be adequate but not excessive, simple but well cooked and attractive, appropriate to the

* Founded on a paper read at the provincial meeting of the Tuberculosis Society at Cambridge, April 11th, 1924.