respect due to an arthritis that is likely to be progressive. Time alone will then solve all doubts of the diagnosis. In the meantime, they must be considered in a class by themselves "for observation."

SUMMARY

Fifty-six of the ninety-seven cases proved to be tuberculous.

Thirty-four were suffering from a transitory arthritis only, without any abnormality being shown by x rays at any time.

Seven had a transitory arthritis as a reaction to a localized bone infection near the joint, without true joint infection.

A transitory arthritis without x -ray changes is common in childhood, and is frequently diagnosed wrongly as tuberculous.

Sometimes it may be traumatic in origin, but more often it is infective, the infection being often secondary to a focus elsewhere in the body.

The prognosis of this transitory arthritis is excellent. A follow-up of twenty-two cases for an average period of three years has been given.

A transitory arthritis is the most difficult differential diagnosis of tuberculous arthritis of the hip in childhood. Its differentiation from tuberculous and other types of arthritis is discussed.

I am indebted to Mr. W. Rowley Bristow for permission to describe these cases, all of which have been under the care of the orthopaedic department of St. Thomas's Hospital.

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DYSPEPSIA AND ITS SURGICAL SIGNIFICANCE

BY

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It can scarcely be said that the present position of surgery in relation to the dyspepsias is either defined or satisfactory, nor is it likely to improve until the surgeon is given a clearer interpretation of the physiological eventualities which culminate in distortions of the metabolic processes. Symptomatic classifications, together with chemico-physiological abnormalities, provide the therapist with material to work on, but the surgical craftsman requires further information if he is to restore function to normal. He must know the changes that result in symptoms, and why these changes take place. When he has determined why, he is in a position to decide whether or not surgical intervention is of avail. It is not enough to correct the changes and trust that the reason for their being may disappear.

In this article I purpose to set out, shortly, my findings when I endeavoured to discover why changes in the mechanical metabolic processes take place, and where surgery may be applied as a corrective.

INVESTIGATION

To study the behaviour of the intestinal tract in chronic dyspeptics I adopted the simple method of x rays and opaque meals. In old-standing cases the changes seen proved very constant. In cases of short duration the changes were less marked, or only intermittently present. As I became familiar with these changes they were to be

seen not only in simple dyspepsias, but also in cases complicated by peptic ulceration.

The stomach varies from the hypersthenic type to the completely asthenic. Its rhythmic propulsive action remains constant, but changes from forceful in the former to tired and sluggish in the latter. The ultimate purpose of the stomach, that of evacuating its contents towards the duodenum, is to a varying extent thwarted by disturbed pyloric function. This difficulty is at first overcome by increased tonus in the stomach, but frequently this tonus later fails and evacuation is rendered slow. The chemical balance on either side of the pyloric sphincter clearly ceases to be its controlling factor. The duodenum may appear normal or become distended. There is delay in the passage of contents along the small intestine, and barium is held up in the lower ileum. Dilatation and often prolapse of the caecum is present, most marked in cases of long standing.

EXPLANATION OF FINDINGS

From evidence collected and given in the *British Journal of Surgery* (1932, p. 634), I think it is clear that these stomach and bowel changes seen in the chronic dyspeptic are due to abdominal sympathetic activation. Such activation results in junctional sphincteric spasm, or hypertonus, with relaxation and delay in the remainder of the bowel. These changes reduce bowel activity, and thereby protect any lesion within the peritoneal cavity.

If one realizes that in the majority of dyspeptics the departure from normal in bowel function is due to sympathetic excitation it then becomes imperative to elucidate the various causes of this excitation. In the aforementioned article I have shown that inflammatory lesions in relation to the abdominal peritoneum are causal agents, and experimentally it has been demonstrated by Alvarez, Mahoney, and others that chemical irritants to the peritoneum produce similar results. Further, I have noted that mechanical irritants such as intraperitoneal bands may result in a like syndrome. Lastly, it would appear true that chemical changes in the blood composition, septicaemias, and toxæmias may produce abdominal sympathetic excitation, not by any direct action on the nerve centres or trunks, but reflexly by their irritant and congestive effects on the abdominal peritoneum.

In investigating cases I have found the primary causes of sympathetic excitation in dyspepsia, in order of frequency, to be: (1) appendix disease; (2) disease of the uterus and appendages; (3) dental disease; (4) intra-abdominal bands and adhesions; (5) abdominal adenitis, with and without mesenteric contractions; (6) renal insufficiency; (7) nasopharyngeal sepsis, or other source of chronic sepsis. Finally, there is a small group of cases where I have failed to demonstrate any lesion, and I class these as being due to primary sympathetic instability or to some endocrine abnormality.

CLASSIFICATION OF DYSPEPSIAS

With this interpretation it now becomes possible to separate dyspepsias into two main groups—primary and secondary. The primary dyspepsias are medical, the secondary are entirely reflex, and, apart from certain ones that are renal in origin, are therefore surgical. The secondary dyspepsias, for diagnostic purposes, I subdivide into: (1) reflex septicaemic dyspepsia, and (2) reflex abdominal dyspepsia.

Reflex Septicaemic Dyspepsia.—I use this term merely because it suggests that the reflex sympathetic upset has resulted from a general condition. The principal reasons for the presence of this general toxæmia or septicaemia appear most often to be dental and nasopharyngeal sepsis, renal inefficiency or insufficiency, and any other source of chronic sepsis. The method by which the abdominal

sympathetics are disturbed by such lesions is somewhat obscure. A recent case of profound haemolytic streptococcal septicaemia from a finger wound which came under my care demonstrated the violence with which the abdominal sympathetics may be disturbed. Extreme gastric discomfort with vomiting was present; marked duodenal soreness was followed by the real pain of ulceration, then haematemesis, and melaena. The abdomen became still and almost rigid. It is significant that a general peritoneal congestion with effusion is present in such a case. It may be, therefore, that a congestive irritability of the peritoneum is readily induced by milder degrees of septicaemia and toxæmia, and reflexly a sympathetic excitation is induced. My attention has been drawn to an article by Beattie in the *Journal of the Canadian Medical Association* (March, 1932), in which he traces an afferent connexion between the olfactory tract and the supra-optic nucleus of the diencephalon. Certain cells in the latter when stimulated produce hyperaemic areas at the pyloric end of the stomach. It may be that nasal lesions produce dyspepsia in this reflex way. Again, it appears to be the effect on the peritoneum of changes in the blood chemistry which result from insufficiency in urinary excretion that produces sympathetic excitation in such cases. When renal insufficiency is present it is well to remember that varying degrees of this condition may be produced by, as well as produce, a sympathetic activation. Though the exact method by which toxic and chemical blood changes result in dyspepsia may yet be uncertain, the recognition of the fact that they do so greatly assists in the investigation and relief of such a case.

Reflex Abdominal Dyspepsia.—Under this heading I include all dyspepsias secondary to an abdominal lesion. The absence from my list of lesions of the gall-bladder may appear somewhat conspicuous. I omit this group because the present study has led me to the belief that this organ becomes affected secondarily to sympathetic disturbances. Its innervation tends to confirm this belief. It remains apparent, however, that when gall-bladder disease has extended so as to involve its peritoneal covering the organ requires to be dealt with.

THE APPLICATION OF SURGERY

This suggested classification simplifies the sorting of dyspepsias for treatment. Those amenable to surgery should first be eliminated. If the case is of the reflex septicaemic type I have seen no harm in dealing immediately with the source of trouble, whether it be a tooth or a prostate. In dealing with the reflex abdominal type, where the cause of reflex excitation is symptomatically clear, there seems no reason why it should not be immediately eradicated. Where no reflex exciting agent is discoverable it is neither justifiable to classify the case as a primary one, nor is it good surgery to perform immediate laparotomy in the hope of finding a possible cause. Dealing with such cases has brought me to this latter belief.

I have found that just as peptone or other shock may nullify sympathetic activity for a period of two weeks to two years, so also will a surgical operation. If, however, the operation has failed to eliminate the exciting cause, or if the case proves to be other than reflex abdominal, symptoms, more distressing than ever previously, will sooner or later return. It is evident also that the most constant dyspepsias are those where sympathetic excitation is always present, and its cause, in consequence, most perfectly protected and free from localizing symptoms. In such a case, where the condition is long-standing, one is driven to the belief that a state of sympathetic activity has become the normal, and no surgical intervention can be of avail until a reversion to the original state of

quiescence has been secured. In every case of dyspepsia with no clear reflex exciting cause I approach treatment by, as far as possible, restoring bowel function to normal. This means undoing the protective sympathetic reflex, exposing the exciting cause, and rendering it the chief source of symptoms.

Those who have essayed the reversion of sympathetic excitement to quiescence by therapeutic measures know how extremely difficult and delicate the task may be. I have confined my own attempts to the use of atropine, MacLean's alkaline method, and, finally, to the bromides. Atropine I abandoned because the dosage required to obtain results always produced the disturbing general symptoms of that drug and could not be continued. MacLean's alkaline treatment is often unsatisfactory in that it will only produce results so long as the reflex exciting cause of the dyspepsia remains quiescent. It acts by falsifying the normal physiological state of the stomach contents so that stomach function is quietened, and, the acid-alkaline balance reflex being destroyed, the physiological necessity for the protective reflex to the pyloric sphincter is hidden. With no direct action on the sympathetic reflex arcs, very careful dosage, diet, and rest are usually all required over a prolonged period. I sought a simpler and more efficacious method through the medium of the bromides. Massive doses over short periods, as suggested by some workers, did not fulfil the purpose I wanted, as time was obviously necessary to bring up the offending lesion and to stabilize the abdominal sympathetics in a state of quiescence. I found, however, that small doses given with an almost chloride-free diet acted, and could be given over prolonged periods with no general symptoms. To the bromide I added the heavy carbonate of magnesium mildly to re-establish the normal intrinsic forward peristaltic action of the bowel; and, later, to ensure calcium to reduce the irritability of the autonomic system and to act also as an alkali in the stomach, I introduced calcium carbonate. Much more uniform results were then obtained, and finally a tablet was produced which rendered treatment simple and satisfactory. The tablet contains 3 grains of sodium bromide, 7 grains of mag. carb. pond., and 5 grains of calc. carb.

This is given with or without diet, depending upon the ease or difficulty that is experienced in restoring function to normal. I have frequently found it wise to eliminate from the diet strong forward peristaltics such as red meat. Function is restored to normal when the patient is clinically free from symptoms and the bowel is acting at least once a day. The tablet is so arranged that this result is obtained by giving two of them three times daily. I have not had to exceed three. By this method the reflex exciting cause of the dyspepsia may become apparent without a renewal of dyspeptic symptoms.

SURGICAL INTERVENTION

When localizing abdominal symptoms make it reasonably apparent why a dyspepsia had been present I do not deem it wise immediately to operate. The more obstinate and long-standing the dyspepsia has been the longer I prefer to delay operation, and, in consequence, the better the final result seems to be. It has not been my experience that even where the appendix is the offending lesion it is in immediate danger of an acute flare-up. I have, however, removed a gangrenous appendix as early as four weeks from the institution of treatment and as late as fourteen months, in old-standing dyspeptics, where, prior to treatment, there had been no sign of appendix trouble.

To clear up old-standing cases entirely not only must the exciting cause of the sympathetic activity be removed, but the remote effects may also require attention. Organic pyloric stenosis demands a gastro-enterostomy, stomach

contractions from healed ulcers may require remedial operative measures, and duodenal dilatation may require improved drainage. Evacuation of the advanced asthenic stomach in the erect position is greatly facilitated by the application of a suitable abdominal support and pad.

CONCLUSION

I have attempted in this investigation to obtain a better understanding of dyspepsia, and from my findings to evolve for the surgeon a useful classification. I have then tried to indicate how and when surgery is best applicable as a means of remedy.

I am greatly indebted to all those who have assisted me in this work, and in particular to Dr. G. D. Roche of Scalloway, whose careful treatment and observation of cases was of great help. I am also grateful to Parke, Davis and Co., who undertook the tedious work of repeatedly producing the tablet till a satisfactory one was obtained.

VASO-DILATOR EFFECTS OF THE PNEUMOGASTRIC NERVE

BY

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The subject of the nervous control of the abdominal and pelvic viscera is one upon which many different opinions prevail. Of the various problems connected with the question that concerning the vascular changes determined by the vagus nerves is one which, at first sight, may seem to be mainly of physiological interest. In reality it is of much importance from a clinical standpoint, and for this reason a clinical journal is now chosen for a brief discussion of the subject with a view to developing the theme further in a subsequent paper. The direct method of approach to a solution of the question of vagal function—that is, by stimulation of the trunk—has been freely employed in determining vagal influence on the digestive mechanisms, its effect on blood vessels being for many investigators a more or less secondary consideration. Hallion and François-Franck, Bunch, Masuda, and a few others have observed the changes in blood vessels due to direct vagal stimulation, but very little definite information about them is to hand. Kiss, McSwiney, and other workers have been concerned with the question of vagal influence on gastric and intestinal muscle, while Gayet and Guillaumie, and Tournade with his co-workers, are recent investigators of the pancreatic and renal functions of the nerves.

An important feature of the problem is the extent to which sympathetic and parasympathetic systems communicate with each other. Simple dissection is not adequate for the purpose of disclosing fine and intricate connexions; evidence of these is often best revealed by physiological and pharmacological tests. I have made a study of the vascular reactions and, incidentally, of the intestinal and gastric effects resulting from electrical excitation of the subcardiac vagus. The blood pressure was recorded from the carotid artery, and the gastric and intestinal contractions by a balloon-tambour system. The vagi were reached in chloralosed dogs through the diaphragm from below, with the use of artificial respiration; in a few instances the chest was opened through the sternum. The nerves were severed on the lower end of the oesophagus, each peripheral part being placed on shielded electrodes and excited by galvanic and faradic currents of different strengths. Some of the salient features of the results obtained are given below, but the records to illustrate them are being kept, owing to restriction of space, for a later publication.

The effects of stimulation with a fairly strong current were in about two-thirds of the experiments (thirty in all) an initial phase of hypertension lasting ten to fifteen seconds, and a subsequent phase of hypotension lasting twice as long and measuring 20 to 40 mm. Hg; in some cases the reaction was purely hypotensive. Generally speaking this response was much the same for both nerves. Some care was necessary to avoid stimulation of cardio-inhibitor fibres, which seem to leave the main trunk sometimes at a very low level; a strong stimulus applied to these fibres may cause fibrillation of the ventricles. The vascular reaction was not annulled by atropine in doses which completely abolished the response of the cardio-inhibitor vagus in the neck. Atropine is the classical paralyser of parasympathetic elements, and its failure to modify hypotensive elements in the vagus trunk suggests that these are not of parasympathetic nature, an inference which is further supported by the failure of parasympathetic stimulants, such as acetyl-choline and pilocarpine, to intensify the reaction. The intestinal and gastric contractions, on the other hand—that is, increased contractions on stimulating the vagi—were abolished by atropine, thus showing the true parasympathetic nature of the fibres concerned; this finds support in the augmentor effect on them of acetyl-choline and pilocarpine. The dose of atropine employed was 0.7 mg. per kilo, which gave a positive result in several experiments. Bayliss and Starling seemed to think that atropine did not paralyse the motor intestinal fibres of the vagus, but the observations made in the present experiments indicate abolition of the effect with strong doses.

What is the type of fibre in the vagus trunk subserving the vascular function referred to? The answer suggested is that it belongs to the sympathetic system. To test this I have used sympathetic paralytics—namely, ergotinine citrate, ergotoxine, and yohimbine. Each of these injected intravenously had the effect of abolishing the hypotensive reaction. Large doses were necessary—for example, several centigrams of ergotinine citrate, which is active because of the ergotoxine it contains: pure ergotinine is said to be inert. Ergotinine citrate in doses of 1 cg. or more causes a rise of blood pressure; in large doses it causes a fall, and this must be produced to ensure abolition of the vagal response. The dose of yohimbine which definitely annulled the reaction amounted to more than 1 mg. per kilo. It is therefore justifiable to conclude that fibres of sympathetic nature, whatever be their anatomical connexion, exist in the vagus trunk for the control of the calibre of certain blood vessels. Non-medullated fibres of sympathetic type have been described in the vagus by various observers, who have ascribed different functions to them. Kiss and his co-workers attribute gastric motor functions to them, but the result obtained by administration of atropine, which annuls this influence, is opposed to that view.

An important feature of the pneumogastric reaction of hypotension is the modification produced in it by section of the splanchnic nerves. These nerves were cleaned and stout threads placed under them, so that by tugging on the threads they were instantly ruptured. The depressor phase of the vagal reaction was in several experiments suppressed or greatly diminished when this was done, suggesting that the depressor fibres passed from vagus to splanchnics somewhere about the diaphragmatic level. But there are other possibilities which must be considered. The vagus is easily fatigued, and a stimulus tends to lose its effect by repetition. In a given case it might be said that the splanchnic section was a mere coincidence, the true cause of failure to react being fatigue. Due precautions were taken to eliminate this source of fallacy, and there is no doubt that fatigue must fail as an explanation in certain tests. Further, the mechanism of vagal hypotensive influence may be the neutralization of splanchnic tone. The effect of central vaso-constrictor tone is, of course, cut out with section of the nerves, and vagal impulses should, if this mechanism be the one played on,