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,

lose their seat of action. However, it is a common observation that arterial pressure, which drops by 25 or 30 mm. Hg at the moment of section, soon regains its former level, and vagal excitation is ineffective to lower it. Nevertheless, it may be that splanchnic tone is the point of vagal purchase, the secondary or subsidiary tone, which replaces it, being immune to vagal influence.

A more serious objection to accepting the view of transference of elements to the splanchnic trunk is that the vagal hypotensive reaction, abolished by splanchnic section, occasionally shows a tendency to restoration after a time, a fact which suggests fatigue as the cause of the phenomenon. But fatigue was almost certainly excluded in some cases where the phenomenon occurred. Another consideration is that trauma must be great when the splanchnics are cut. Slight traction or pressure on the intact nerves produces convulsive twitches, which indicate a very sensitive structure, and the shock of rupture may cause a depression of peripheral as well as central excitability. Whatever the explanation, splanchnic section, which gives rise to increase of the intestinal and gastric muscle contraction following pneumogastric excitation, causes, in a large proportion of experiments, a depression or even abolition of the vascular hypotensive reaction to this excitation. There are good reasons for the view that the pneumogastric nerve contains sympathetic fibres, and it must seem strange that it should receive these only to restore them to the sympathetic system for final distribution. While we cannot affirm that this is the case, we cannot assert that it is not so; further work is necessary to determine the point. The splanchnics were divided in the first instance with a view to demonstrating the possible transference to them of intestinal motor fibres from the vagus, since it had been found in previous work that excitation of certain bundles from the splanchnics caused increased intestinal activity. Splanchnic section, however, far from diminishing this activity, has an augmentor influence on it, eliciting spontaneous movements and increasing the effect of vagal stimulation. This observation points to the absence of motor communication.

It is important to ascertain the seat of the vaso-dilatation resulting from pneumogastric excitation, and efforts have been made in this direction by taking plethysmographic records of the kidney, intestine, spleen, and the limbs. Since the demonstration by Kuré, Hasama, and others of vaso-dilator nerves coming from different spinal segments it was thought well to test the limbs for enlargement, although it was unlikely that a vagal supply to them existed. The splenic and intestinal volume did not show any significant alterations; the variations in these parts were mostly passive in relation to blood pressure changes. The small portion of gut, however, which is introduced into the oncometer does not necessarily reveal what may be occurring in other parts. It is quite possible that a vagal control of mesenteric vessels may exist without a uniform distribution of vascular elements. The renal reaction showed some differences in individual animals. A marked vaso-constriction was observed after the vagal stimulation in two experiments. This was soon followed by enlargement of the viscus, but the renal spasm was synchronous with the fall in blood pressure, and still occurred when this was compensated. In two other experiments some degree of expansion of the kidney coincided with the depressor phase of tension, but a general feature of the renal change was alternation of expansion and contraction for some time after stimulation, more or less independently of the blood pressure. Tournade described a renal shrinkage as a result of pneumogastric excitation, which he ascribed to increased production of adrenaline. In the present experiments the latent period

of the early constrictor effect was too rapid to be accounted for in this way. The liver and stomach, of which the changes of volume were not recorded, remain as possible regions where the vascular change could occur.

It will suffice to state that the records obtained in the work and held over for publication provide clear evidence of the chief point in the contribution—namely, that there exist hypertensive and hypotensive fibres in the vagus trunk, the latter particularly being paralysed by ergot and yohimbine and unaffected by atropine. They are consequently to be considered of sympathetic type.

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THE SPHYGMOMANOMETER IN THE DIAGNOSIS OF CARDIAC IRREGULARITIES

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The clinical differentiation of cardiac arrhythmias has been much simplified as the result of electrocardiographic studies of the mechanisms involved, but there are always certain cases in which a definite diagnosis is impossible without the aid of some means of graphic registration. It is probable that no method except the graphic will ever be devised which will serve to distinguish with certainty between auricular fibrillation and those cases of frequent extrasystoles (more particularly those in which premature beats of both auricular and ventricular origin are present) which give rise to a complete disorganization of ventricular rhythm.

The fact that pulsus alternans may often be recognized during the routine taking of the blood pressure is fairly well known. It deserves, however, to be far more widely appreciated. The sphygmomanometer is an instrument which is now as much part of the everyday equipment of the medical man as the stethoscope; and pulsus alternans is a portent of such gravity that no method by which it can easily be recognized should be overlooked. It is not common for the alternating beats to be so marked that they can be distinguished by even the most sensitive finger. The sense of hearing is far more delicate than the sense of touch, and the auscultatory method of estimating blood pressure provides a means of detecting quite small differences in the strength of the ventricular contractions.

It is common knowledge that the systolic pressure is remarkably inconstant, even in the same individual. Not only does it vary from day to day but from minute to minute and even from beat to beat. Other factors remaining constant, the maximum arterial pressure achieved by any beat will depend upon the strength of the ventricular contraction. Where such contractions alternate in strength there will be alternation of maximum (systolic) pressure. This is, as a rule, quite easily recognized with the manometer. A variation of 5 mm. of mercury