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ON THE NERVOUS CONTROL OF ABDOMINAL BLOOD VESSELS AND VISCERA *

BY

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The problem dealt with in this communication is that concerning the role of parasympathetic and sympathetic systems in the regulation of muscular movements and of vascular conditions in the abdominal viscera. The method adopted for experimental investigation has been already described (*British Medical Journal*, 1933, i, 956). A statement made in that article was to the effect that cardio-inhibitor fibres may be found arising at a low thoracic level in the dog. Cannon apparently recognized this possibility, to judge from his description of the procedure of cardiac denervation. In man all cardiac branches are said to arise above the root of the lung. The present writer has twice unwittingly stimulated these fibres below the root of the lung in the dog, once with a fatal result.

VAGAL INTESTINAL CONTROL

When either vagus nerve is excited by a fairly strong current a balloon in most parts of the small intestine shows some degree of external pressure upon it, as seen by a single prolonged up-stroke of the tambour lever. This contraction may take place as a response to stimulation of one nerve only, or it may fail to occur on stimulating either nerve. There are portions of the intestine where one can predict to some extent which nerve is going to show the greater influence. Jacobi said that in the rabbit the intestinal motor effects of the vagus were confined to one side. In the dog one gets no indication of such a distribution. In this animal I have found that in the upper reaches of the gut the action of the left vagus is somewhat more pronounced than that of the right, while lower down there seems to be little difference, at any rate in certain parts. The distribution of the nerves is not uniform, and there is no sharp definition of areas of right and left preponderance. Kuré and his associates say that the numbers of parasympathetic fibres supplied to the intestine vary greatly in different dogs.

If one now proceeds to cut the splanchnic nerves the intestine will often be observed to enter immediately into spontaneous rhythmic activity, but, if not, one has only to repeat the vagal stimulus to elicit rhythmic activity, which ceases with the stimulus and can again be evoked by repeating it. The splanchnic nerve inhibits particularly the rhythmic action induced by the vagus, but the antagonism is seen in quantitative as well as qualitative respect (Fig. 1). In this figure the arrows indicate the points of excitation and show the positions of corresponding points in the records. Note that there is a reversal of the blood pressure change and increase of the intestinal and gastric responses after splanchnic section. The observation points to the constant antagonism of splanchnic tone to vagal muscular impulses. Attention has recently been drawn by Jasper and Monnier and others to the rhythmic nature of the response to a single stimulus

of the crab's nerve. The well-known Ritter tetanus is a somewhat similar phenomenon. It is a feature which is masked in most simple responses. Here in the intestine is presented a good instance of masking and unmasking of the phenomenon. The fact that at a particular moment the quiescent intestine is induced by vagal excitation to enter into rhythmic activity, which ceases with or soon after the stimulation, suggests that the pneumogastric nerve is directly concerned with this form of activity, which is regarded by many observers as a purely myogenic process. Of course the evocative stimulus is iterative, but the serial contractions outlast it. From one point of view the phenomenon is not strictly comparable to that in the crab's nerve, which enables a single excitation to cause serial impulses.

Vagal contraction of the gut was annulled by atropine in two experiments before splanchnic section and in three experiments after section, and it was usually enhanced by injection of acetylcholine or pilocarpine, thus showing the true parasympathetic nature of the fibres concerned. Bayliss and Starling stated that large doses of atropine failed to paralyse the intestinal vagus, as they obtained contractions on stimulating the nerve after injection of large doses of the drug. Possibly the conditions in which those very accurate observers worked differed in some way from those of the present experiments, in which neither electrical excitation of the vagus nor acetylcholine produced any contraction after injection of atropine to the extent of 0.7 mg. per kilo. Possibly, too, we are at cross-purposes as between sympathetic and parasympathetic elements, the interrelations of which will be considered later.

In experiments performed with A. and B. Chauchard we determined the chronaxies of the gastric response and the vascular reaction (to be presently described) of the pneumogastric nerve. The gastric motor chronaxy is the greater,

FIG. 1.—Curves of stomach and intestinal contractions and blood pressure. Excitation of right vagus (r.v.) before splanchnic section in (A), and after section (s.c.) in (B). Reversal of effect on blood pressure. Time = 5 seconds in all records, and excitation period 15 to 20 seconds.

the capacity measuring 0.5 mF., which, with a resistance of 10,000 ohms, represents a chronaxy of 2.0σ as compared with 1.5σ for the vascular pressor response. Section of the splanchnic nerves determined a diminution of the gastric vagal chronaxy, which demonstrates the influence of a nerve on the property of excitability of its antagonist, the quantitative change being probably determined by some modification of the effector mechanism in the end-organ.

EFFECTS OF SPLANCHNIC EXCITATION

Stimulation of the cut end of the great splanchnic nerve produces inhibition of the gut, an effect which is presently increased by the influence of the adrenaline secreted. This is the generally recognized form of the splanchnic action both on stomach and on intestine, but an excitatory action is also in evidence, the converse picture of vagal inhibition, as is easily demonstrated by stimulation of separate bundles of splanchnic fibres. Two distinct types of activity are

* Read in opening a discussion in the Section of Physiology and Biochemistry at the Annual Meeting of the British Medical Association, Dublin, 1933.

evoked by these motor fibres—namely, rhythmic and tonic. The former response, an increase of the rhythmic contractions already in evidence, may be immediate or may succeed a brief inhibitory phase. The tonic or peristaltic type of response is more commonly observed in a quiescent gut. The existence of nematode worms, seen in some experiments, seems to favour this tonic response, but it occurs independently of these parasites. The circulatory or vascular conditions in the gut are supposed to have an important influence in determining activity or quiescence of the muscle. Anaemia, as determined by vaso-constriction, or bleeding, or aortic occlusion, is said to lead to inhibition and rest, while the converse proposition that congestion tends to cause contraction, is implied. The type of response must also depend to some extent on these conditions, but the effects of nervous excitation show a good deal of independence of such variations. Motor effects from splanchnic stimulation were also observed by Bechterew and Mislowsky.

EXCHANGE OF NERVOUS ELEMENTS

In a given reaction from excitation of one branch of the double system of nerve supply certain elements received by communication from the other branch may be responsible for the result. In the absence of precise anatomical dissection

FIG. 2.—Effect of atropine negligible. R.V. excitation causes typical reaction before atropine in (A), and after in (B). At C.R.V. cervical vagus excited to show full effect of atropine.

FIG. 3.—Effects of ergotinine citrate (5 cg. injected at ERG). Left (L.V.) and right (R.V.) vagal excitation. The drug caused a prolonged fall of blood pressure (folded out in the record). Renal volume on top.

and histological distinctions pharmacological tests provide a method of judging how far such communication comes into play. A discussion of the point has arisen concerning the type of vagal fibres supplying the stomach. From anatomical evidence these were said by Kiss to be sympathetic in kind, but McSwiney and Spurrell excited the intracranial vagus and got gastric motor effects. These workers could obtain no evidence physiologically of inhibitory and motor fibres being conveyed to the stomach by different elements. While they prove that the motor fibres to the stomach are parasympathetic—and I think that the action of atropine supports this view—these observers have not demonstrated that the inhibitory fibres are of the same kind. These fibres may be sympathetic. Occasionally in the atropined preparation inhibition replaces contraction of the stomach when the vagus is excited, but the evidence is not complete without the complementary test with ergot. Kuré and his co-workers are of the opinion that these inhibitory gastric fibres are of sympathetic type. The existence of sympathetic elements in the vagus trunk will presently be shown for the control of blood vessels. There is a close connexion between sympathetic and vagus in the dog.

We have already seen that the effect of excitation of the pneumogastric nerve is merely enhanced by splanchnic section, and therefore the main vagal supply to the intestine is independent, but this observation does not preclude the possibility of transference of some fibres from vagus to splanchnic. Parasympathetic fibres of spinal origin may find

their way into the splanchnic, as is suggested by the work of Hasama, Kuré, and others, who found widely distributed parasympathetic fibres coming from the dorsal spinal roots. The effect of atropine on the tonic splanchnic reaction of the gut was a diminution without abolition. Further tests with atropine and ergot are necessary to determine the nature of the nerve fibres producing the response. The mixing of elements of the two systems will be referred to again.

VASCULAR EFFECTS

The vascular effects obtained on stimulating the peripheral vagus have been previously described (*loc. cit.*). The hyper-hypotensive reaction is the characteristic one for fairly strong stimuli on both sides. In a few cases the pressor phase was more marked than the depressor one. With a galvanic current or a weak induced current the pressor effect alone is common, and occasionally no response occurs with such a current. The latent period of the hypotensive phase depends on the duration of the first phase, but when the former occurs alone the delay is about seven to ten seconds. Atropine does not annul the reaction, although the depressive effect is sometimes slightly diminished by large doses of this drug (Fig. 2). This vascular response was not intensified by injection of vagotrope sub-

FIG. 4.—Effects of ergotoxine (0.5 mg. and 1.5 mg. at intervals). The vascular reaction is gradually annulled.

FIG. 5.—Effect of yohimbine (12.5 mg.) at second arrow mark; depressor action of vagus R.V.1, very feeble at R.V.2, and lost at R.V.3. Renal volume on top.

stances like pilocarpine and acetylcholine, but it was definitely annulled in several tests by injection of sympathetic paralytics, ergotinine citrate (Fig. 3), ergotoxine (Fig. 4), and yohimbine (Fig. 5). These drugs had a much more marked effect in abolishing hypotension than hypertension. This observation shows a certain degree of dissociation between the two phases as possible.

EFFECT OF SPLANCHNIC SECTION

After section of the splanchnic nerves the vascular reaction to vagal excitation disappears completely or is considerably modified. In some seven out of ten experiments in which the characteristic rise and fall of pressure occurred on stimulating the nerve, splanchnic section caused abolition of the depressive phase (Fig. 1). In experiments performed in the laboratories of the Sorbonne, where the galvanic stimulus was mainly employed, we obtained most often the pressor phase only, which disappeared in about half the cases when the splanchnic nerves were severed. In a few instances we were able to measure the chronaxy of the reaction before and after section. In my own separate experiments this pressor phase persisted in a majority of the tests on splanchnic section, whereas in practically all of them the hypotensive effect disappeared. In two experiments in which the vagal response was almost purely hypotensive before section it was reversed after it (Fig. 1). The significance of these observations will be referred to again. For the time being I conclude that there are hypertensive and hypotensive fibres

in the vagus nerve, and shall proceed to give an account of the attempts made to ascertain their seat of action. Of course, an objection may be raised to this form of investigation on the ground that strong faradic stimulation is abnormal, but the same must hold for any form of artificial excitation. So long as we demonstrate by any means the presence of elements with a certain form of activity, it is not unreasonable to infer that they have a purpose in the economy which can be evoked by some natural agency.

ONCOMETRIC RECORDS

The volume of the spleen showed no change other than a passive one, corresponding to variations of blood pressure. Masuda observed similar passive changes in this organ after vagal stimulation in the neck. He observed no change of volume when the blood pressure change was compensated. Intestinal volume in two records underwent slight increase as a result of vagal excitation (right)—it was in a loop of jejunum—and a passive diminution with left vagal hypotension. Other parts of the intestine showed mostly a passive diminution. In all these experiments the hypotensive vascular response was in evidence, and judging by these results it seemed that intestinal vaso-dilatation was not enough to account for it, but we have to consider the possibilities of limited areas being affected and of our missing these in a particular experiment. Vaso-dilatation seems to occur in certain portions of the intestine, which are not the same for each nerve. Bunch stated that no vaso-dilator action of the vagus was observed in the intestine of rabbits and cats. In dogs, he said, peripheral vagus stimulation "may, in certain circumstances, give rise to vaso-dilatation of intestinal vessels."

FIG. 6.—Effects of acetylcholine. A before and B after injection of atropine at interval. Readjusted levels in B. Renal volume above.

A somewhat intriguing feature of vagal activity is the alteration which occurs in renal volume. This showed increase after excitation of the pneumogastric in a few experiments. In some others it showed a well-marked decrease which was independent of pressure change. In some pronounced instances of shrinkage due to excitation of the nerve it was evoked several times when the hypotension was fully compensated. Béco and Plumier showed that excitation of the vagus caused diminution of renal volume. Tournade and his associates repeated their experiments and observed similar results; Rose Bradford had previously demonstrated the existence of both vaso-dilator and vaso-constrictor fibres in the renal splanchnic nerves. Hamet observed that section of the vagus led to diminution of renal volume, a change which he ascribed to the cutting off of afferent regulating impulses running up in the trunk of the nerve.

ACTION OF DRUGS

Atropine had little or no effect on the renal constrictor response to vagal excitation. The action of acetylcholine in this connexion seems to me to be of much interest. In doses up to 0.2 mg. per kilo acetylcholine caused, independently of electrical excitation, the usual hypotensive effect, and this was accompanied by immediate renal shrinkage (Fig. 6), which was followed by a phase of enlargement, and on this shrinkage occurred again. Atropine was then injected, and in a few minutes the previous dose of acetylcholine was repeated. The vascular action of this drug was

now reversed; it became markedly hypertensive, while the shrinkage of renal volume took place as before. Feldberg and Minz* obtained a pronounced rise of blood pressure in the pithed atropined cat from injection of acetylcholine, due, they say, to its action on splanchnic blood vessels. No special renal constriction is referred to. Hirose injected acetylcholine into intestinal, renal, and other arteries, and concluded that vaso-constriction occurred, as a rise of general blood pressure soon followed. That acetylcholine is a strong stimulant of sympathetic fibres is at once evident from its use after atropine. In strong dose it has both a direct and an indirect action, the latter arising apparently from the production of adrenaline. It has a double action on the kidney; vaso-constriction is evident in the early phase, then vaso-dilatation succeeds, and alternating periods of expansion and contraction occasionally last some time afterwards. The stimulating action of acetylcholine on sympathetic fibres was also observed by McMichael. He said it was of slow onset. Once the vagus is annulled, however, it is quickly in evidence; the sympathetic is at first masked by the parasympathetic action.

DISCUSSION OF RESULTS

The disappearance of vagal hypotension after splanchnic section suggests at first sight that the vagal depressor fibres pass into the splanchnic system, but there are other ways by which the effect might be explained. The influence of the vagus in causing hypotension may be due to the counteraction of splanchnic vascular tone. When this is annulled by section of the nerves and a fall of pressure occurs, then if the mechanism be as suggested no further fall of pressure should arise with vagal stimulation. Section, however, does not always lead to a fall of pressure, or the fall, if it takes place, is quickly compensated. In such a case vascular tone is still in evidence, but vagal excitation does not affect it. The vagal hypotensive reaction shows a tendency to recover after a time, though it is never so pronounced as it was before interference with the splanchnics. It may be that the rupture of the splanchnic nerves disturbs vagal responses in some way other than by a break in the anatomical continuity, but the sudden and complete disappearance of the hypotensive reaction after this rupture is suggestive of continuity. Blocking and unblocking of splanchnic conductivity by a galvanic current caused some variations of the hypotension. Generally it was diminished but not quite abolished by the blocking current. Only a few such observations were made.

The question of the existence of sympathetic fibres in the gastric vagus, already referred to, opens up that of another with regard to the presence in the vagus trunk of sympathetic vascular fibres. The views of the Hungarian anatomist Kiss are of interest in this connexion. From histological and anatomical study he concludes that the relations between pneumogastric and sympathetic are the same as those between spinal nerves and sympathetic. The intracranial vagus, he says, contains large and small myelinated fibres, of which the latter are the more numerous. There are no true sympathetic or non-myelinated fibres in this part, but between vagal and sympathetic ganglia free communication occurs, the vagus containing pre-ganglionic and post-ganglionic (non-myelinated) fibres of sympathetic type. He says that all para- and pre-vertebral ganglia belong to the sympathetic exclusively. There are no grounds for the idea that there is a parasympathetic system in the abdominal cavity, and all so-called parasympathetic phenomena are negative sympathetic phenomena. Kiss apparently takes no account of pharmacological distinctions, nor does he consider differences of physiological effects. He distinguishes and classifies nerve units by their histological characters.

The pressor-depressor response to stimulation of the peripheral vagus already described was, as stated,

unaffected by atropine, like chorda tympani vaso-dilatation in the submaxillary gland. This observation suggested that it was due to stimulation of sympathetic fibres. Sympathetic paralysants annulled the response, the hypertensive phase being the more resistant. The action of ergot preparations on the sympathetic is generally said to be a paralysis of motor or vaso-constrictor elements, leaving depressor or dilator fibres intact, but some observers take a different view. Rothlin, Hamet and Rothlin, and Hamet state that motor and inhibitor sympathetic elements are paralysed by ergotamine and yohimbine. In the present experiments the vaso-dilator part of the response was more definitely abolished than the pressor phase by these agents. The thoracic vagus trunk in the dog accordingly contains fibres which regulate vascular tone and which give the reactions of sympathetic elements. The effects of splanchnic section suggest that they are transferred to this system, and there are two possibilities as to how it may happen. It may be that we are stimulating pre-ganglionic fibres in the vagus and that these join up with the splanchnic through an intervening ganglion—thoracic or other ganglionic group. The second possibility is that post-ganglionic fibres in the vagus are merely borrowed from the sympathetic chain to be restored to sympathetic groups in the splanchnic nerve. Certainly the vagal reactions are greatly modified by splanchnic section, and it looks as if at least some of the fibres concerned are cut in the process. The result is not to be explained by injury to vagal fibres in dissecting the splanchnic, for the reaction was frequently observed to exist after this procedure and before rupture. Possibly rupture injures vagal fibres to the coeliac ganglion. The abolition was best marked in case of the right vagus.

Concerning the seat of the vaso-dilatation, the work of McMichael and of Griffith and Emery is of interest. The former is satisfied, from the results of injecting acetylcholine, that there are no vaso-dilator fibres of parasympathetic nature in the liver. He does not seem to have stimulated the vagus nerve electrically to observe if a fall of blood pressure ensues. It may be quite true to state that there are no parasympathetic vaso-dilator fibres in the liver, yet the left vagus nerve may bring vaso-dilator fibres to this organ, fibres of sympathetic origin. Only in case of pneumogastric excitation producing hypotension with no increase of liver volume should it be said that the liver is not the seat of vaso-dilatation. McMichael further concludes from the effects of ergot that there are no sympathetic fibres in the liver. Griffith and Emery also are opposed to the view of Hallion and François-Franck that vagal dilatation of hepatic vessels does occur. Griffith and Emery stimulated hepatic nerves on visceral arteries. By this means it may be supposed that antagonistic fibres were simultaneously stimulated. Records of portal venous pressure showed a fall of this when the vagus was stimulated, but it is not possible to state if this was due to diminished resistance from the hepatic dilatation or to other change. Carnot, Gayet, and Merklen observed a similar fall of portal pressure from excitation of the pneumogastric nerve. They say that acetylcholine is of uncertain action on this pressure.

With regard to the changes of renal volume a feature was their variability in different animals. Ergot preparations diminished the shrinkage of the kidney. This observation of the failure of atropine to affect the reaction points to sympathetic elements as producing it. Kuré says that parasympathetic fibres are vaso-constrictor to the pulmonary vessels and dilator to the kidney and intestine. Tournade described a vaso-constriction of the kidney from vagal excitation, which causes increased secretion of adrenaline and thus adds to the direct nervous constriction. In the present experiments there was no clear effect either on blood vessels or intestine which

could be ascribed to any marked secretion of adrenaline resulting from stimulation of the pneumogastric. But injection of acetylcholine when this nerve was paralysed by atropine had effects which could be best explained by increased secretion of adrenaline.

SUMMARY

The vagus nerves were stimulated peripherally below the origin of the cardio-inhibitor fibres. Motor effects on the intestine were obtained which were abolished by atropine and not affected by sympathetic paralysants. They are occasioned by parasympathetic fibres which really exist as myelinated units separate from the sympathetic. Inhibitory vagal fibres to stomach and intestine may be sympathetic in type. Vascular fibres exist in the vagus which on excitation yield hypertension and hypotension. These are unaffected by atropine, and are paralysed by ergot and yohimbine. They seem to be sympathetic elements. It is not possible to say if these occur in the vagus as pre- or post-ganglionic fibres. Stimulation of the vagus causes constriction of the kidney, but the reaction is well marked only in a certain proportion of animals. The nervous units responsible for this reaction behave like sympathetic elements.

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THE AETIOLOGY AND PREVENTION OF THE TOXAEMIAS OF PREGNANCY

BY

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There are distinct signs that individual observers are beginning to recognize that ante-natal clinics, as they are at present organized, have not served to lower the maternal mortality rate, and that even efficient ante-natal supervision cannot be substituted for sound midwifery. It is not, however, realized that the death rate per thousand live births from puerperal albuminuria and convulsions and the other toxæmias of pregnancy in England and Wales during 1931 was slightly higher than it was in 1921. The investigation into the causation of these toxæmias is therefore not merely a question of absorbing academic interest, but one of real importance, for until radical changes have been effected in our maternity services it seems probable that the only hope of