ON THE LOCAL REACTIONS OF THE ARTERIAL WALL TO CHANGES OF INTERNAL PRESSURE.

By W. M. BAYLISS. (Eleven Figures in Text.)

(*From the Physiological Laboratory, University College, London.*)

My attention was first directed to these phenomena by the occurrence of curves like that reproduced in Fig. 1[1].

In the course of experiments on vaso-dilator reflexes I sometimes observed what looked like a reflex of this kind, in a limb of which the nerves had been divided; the figure reproduced shows the effect of a fall of arterial pressure produced by exciting the central end of the depressor nerve on the volume of the hind leg of the rabbit, the sciatic and the nerves accompanying the femoral artery having been cut. It will be noticed that as long as the fall of blood-pressure lasts there is a passive diminution of volume in the limb, but that as soon as the previous height of blood-pressure is attained, on cessation of the excitation, there is a considerable expansion of the limb lasting for some time. Such a curve would usually be explained by stating that there was present all through the excitation a relaxation of the vessels, but that it was prevented from showing itself in an actual expansion of the limb by the simultaneous fall of blood-pressure; although it was

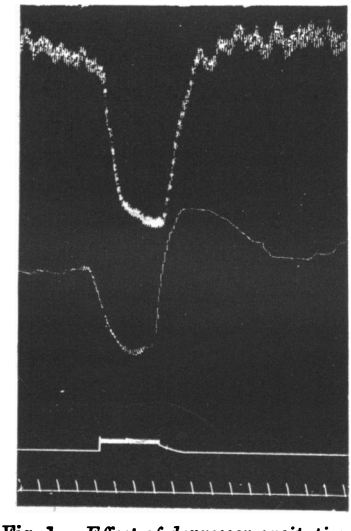

Fig. 1. *Effect of depressor excitation on volume of enervated leg.* Upper curve blood-pressure, next below it volume of leg, upper of two chronographs—period of excitation of depressor nerve, lower one—time in 10 sec. intervals.

[1] A preliminary account was published in the *Proceedings of the Physiological Soc.* for March 16th, 1901. (This *Journal*, xxvi. p. xxix.)

able to produce such an expansion when the blood-pressure rose again owing to the vascular dilatation in other parts, chiefly splanchnic area, lasting a shorter time than in the limb. Such an explanation, obviously, will not hold here, since the leg nerves were cut, and it is plain that some other explanation must be found.

Bearing in mind the well-known reaction of muscle in general to stretching, instances of which are, amongst others, the increased force of the heart-beat produced by increased intraventricular pressure, the effect of tension on the snail's heart[1], and the contraction of the body walls of the earthworm in response to a pull[2], it is natural to suppose that the muscular coat of arteries reacts in a similar way to increased intravascular pressure; and if so, being in a state of increased tone in response to the normal blood-pressure, a lowering of that pressure will be followed by an opposite reaction, viz. a relaxation.

This is, no doubt, the explanation of the curve reproduced in Fig. 1. During the fall of blood-pressure the arteries were, in all probability, relaxed in response to that fall, but not to a sufficient extent to produce an actual increase of volume of the limb. This relaxation shows itself at once, however, when the blood-pressure returns to its original level and lasts until the muscular wall of the arteries has responded to the rise of pressure by returning to its original state of tone.

Reaction to increase of tension.

Since this reaction is the more familiar one in other muscular tissues, I will first describe some experiments made to detect it in arterial muscle. So far as I am aware, indeed, the opposite form of reaction, viz. relaxation to diminished tension, has not, hitherto, been described. It must obviously occur, however, in a muscle in a state of tone, if that tone is present in response to a condition of tension, when the exciting cause of the tone, viz. the tension, is diminished.

To produce a rise of arterial pressure in these experiments, in order to provoke the response, one can either excite the peripheral ends of the splanchnic nerves, or produce a reflex rise by exciting the central end of a pressor sensory nerve, or by producing asphyxia in a curarized animal. In the two latter cases, naturally, the vaso-motor nerves of the organ must be cut, as also in the case of splanchnic excitation, when a viscus supplied by that nerve is under observation. As regards the

[1] Biedermann. *Sitzungsber. Wiener Akad.* 1884, p. 24.
[2] Straub. *Pflüger's Archiv*, LXXIX. p. 387. 1900.

limbs I have not noticed that there is any difference in the reaction whether the nerves are cut or not. The reaction is, therefore, of peripheral origin, and, as I believe, myogenic in nature; but this point will be discussed later.

The animals used, dogs, cats, and rabbits, were anæsthetized with morphia (30 to 130 mgrms. subcutaneously) and kept under A.C.E. In most cases curare was also given.

The curves of Fig. 2 show the effect of exciting the peripheral end

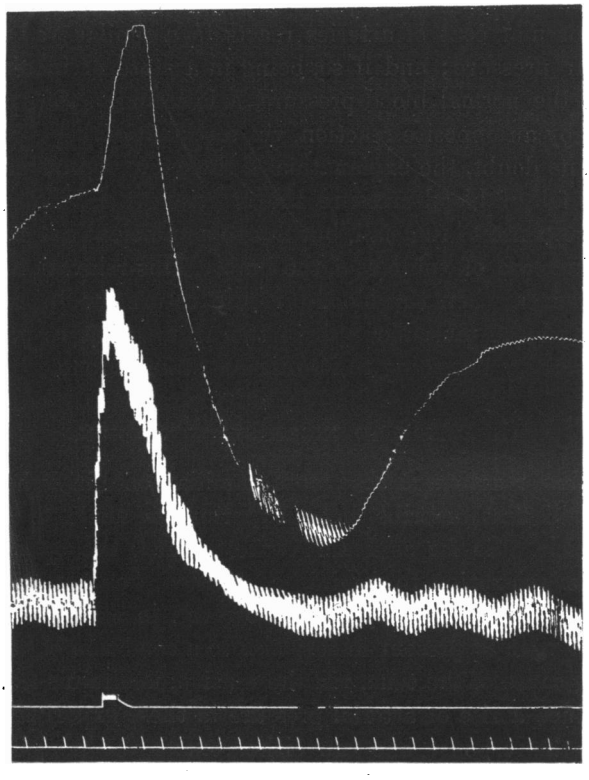

Fig. 2. *Effect of splanchnic excitation on enervated limb of dog.* Upper curve—volume of limb. Lower curve—blood-pressure, zero being 45 mm. below excitation marker, which is the upper of the two bottom lines. Time in 10″.

of the cut splanchnic nerve on the volume of the enervated hind-leg of the dog. As the arterial pressure rises the limb is distended passively, but instead of merely returning to its original volume when the blood-

pressure has come down again, it constricts much below its previous level and only gradually returns[1].

Fig. 3 shows a similar effect in the cat.

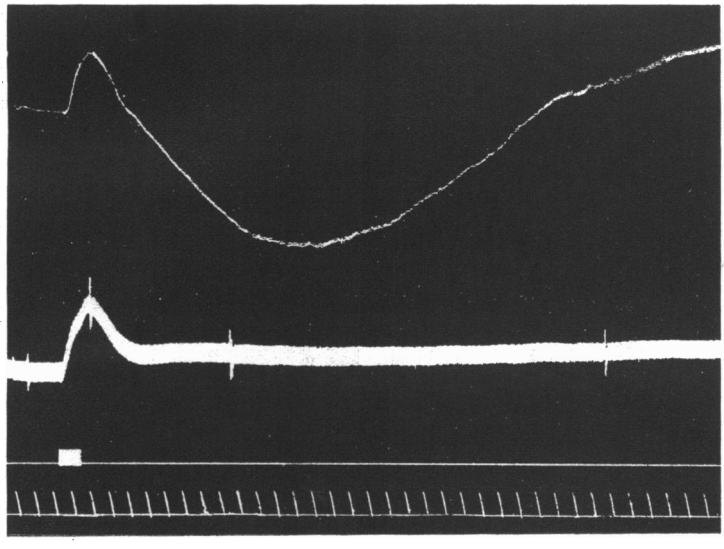

Fig. 3. *Effect of splanchnic excitation on the enervated hind-leg of cat.* Explanation of Fig. 2 applies also to this.

In asphyxia one occasionally meets with curves like Fig. 4. In this case the leg nerves were cut, and at the point A the artificial respiration was stopped. At first the leg is passively distended, but, while the blood-pressure is still at its height, the limb begins to diminish in volume, attaining its original size while the blood-pressure is considerably above its previous height. Of course the effect in this case is possibly complicated by the admixture of the direct action of asphyxial blood on the vessels, but there is reason to believe that the latter is rather of the nature of a dilatation than constriction, although the point is still uncertain.

In the case of the kidney vessels this reaction to increased tension is very marked, as shown in Fig. 5. All the nerves in the hilus that could be found were torn through, the vessels and ureter thoroughly

[1] The large waves at the bottom of the limb-curve in the figure are accidental, due to the lever of the piston-recorder coming into contact with that of the mercurial manometer at this point.

painted with concentrated phenol, which I have found a very useful means of destroying the conductivity of nerve-fibres. The vessels and

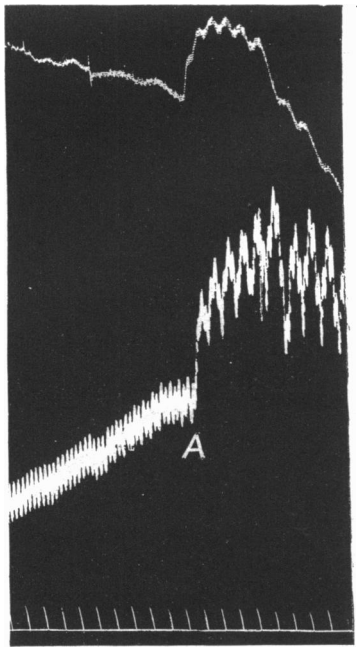

Fig. 4.

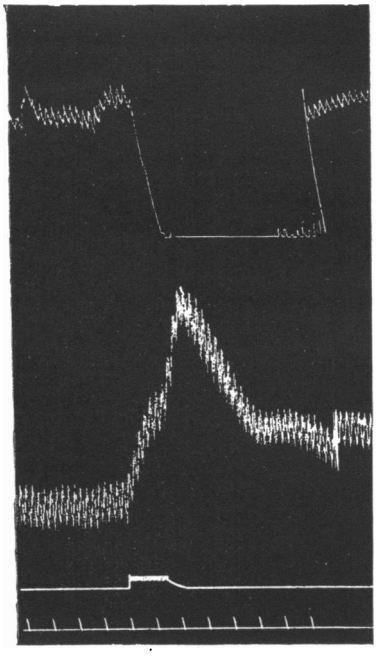

Fig. 5.

Fig. 4. *Effect of asphyxial rise of blood-pressure on volume of enervated leg.* Same explanation as previous figures. Blood-pressure zero—13 mm. below time tracing. At A artificial respiration stopped, the animal being under curare.

Fig. 5. *Effect of splanchnic excitation on volume of kidney of dog.* Upper curve—volume of kidney. Lower curve—blood-pressure. Excitation and time markers as previous figures. Blood-pressure zero—55 mm. below excitation marker.

ureter, moreover, were tightly pinched with forceps, at the risk of causing the blood to clot, which fortunately, however, did not take place until the experiment had been performed. The kidney was enclosed in a Roy's oncometer, filled with warm physiological saline instead of oil, the fluid only reaching to the beginning of the tube leading to the piston-recorder, which contained air. The splanchnic nerve of the same side was exposed in the abdominal cavity and on Ludwig electrodes. The rise of blood-pressure produced by exciting the nerve is seen to cause at first a slight passive distension of the kidney, but even when the pressure is still rising the kidney begins to

react by constriction which carries the piston of the recorder down to the limit of its excursion. The drum was stopped for a short time to allow the lever to return to its original position. The only source of fallacy that I can see in these experiments is the possible escape of current from the electrodes on the splanchnic nerve to the kidney vessels directly. In order to exclude this I made another experiment in which the lower end of the divided spinal cord was excited, the cord was cut at the level of the 4th thoracic nerve, and the lower end excited in the spinal canal (Fig. 6).

The kidney nerves had been cut, painted with phenol and tightly pinched with forceps. The reaction is not so striking as in Fig. 5, but there is an obvious constriction of the organ, while the blood-pressure was at its height. The circulation through the kidney was not so good in this case as in that of the previous figure, since the vessels had been pinched a longer time before the experiment was made, and had probably become partially obstructed with clot.

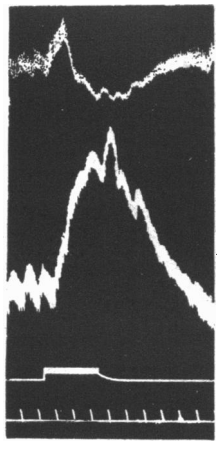

Fig. 6. *Effect of exciting spinal cord on volume of enervated kidney.* Explanation as previous figures. Blood-pressure zero 23 mm. below excitation marker.

Reaction to diminished tension.

We may now consider the opposite form of reaction, viz. the relaxation produced by diminution of blood-pressure.

Fig. 7 is an example of the effect produced on the volume of the hind-limb by aortic obstruction. The cannula recording the arterial pressure was in the femoral artery of the opposite side, so that its excursions record the actual pressure in the artery of the limb under observation. This dog had been used for experiments on vascular reflexes, so that the sciatic and anterior crural nerves were not cut, but it was cut off from the vaso-motor centre by extirpation of the abdominal sympathetics on both sides and by section of the cord at the 2nd lumbar vertebra. There is seen to be a large dilatation of the limb, following the rapid diminution of volume, produced by compression of the abdominal aorta by means of a thread on a ligature-staff. There is also

seen here a constriction following the dilatation. This is still better shown in Fig. 8, and is no doubt to be explained by the sudden inrush of blood at high pressure into the relaxed arterioles causing a con-

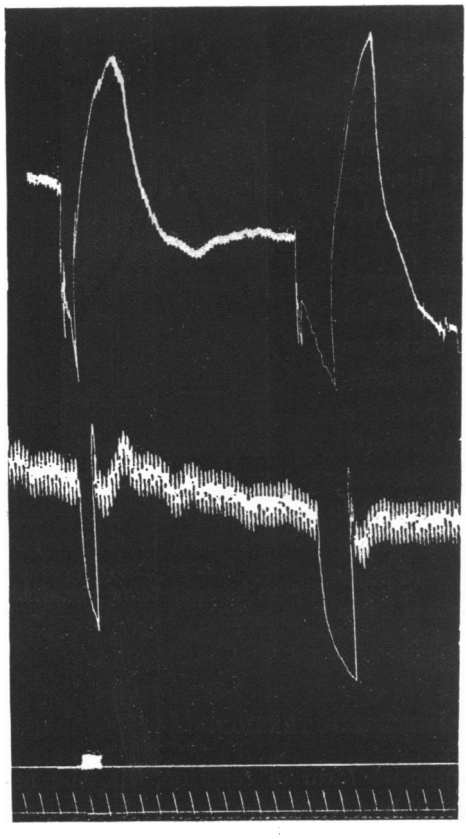

Fig. 7.

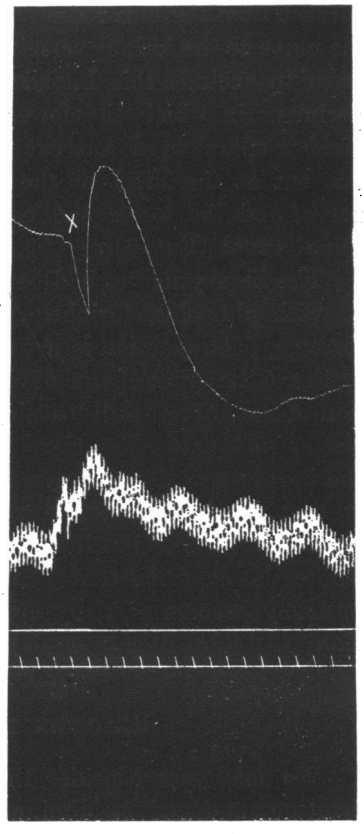

Fig. 8.

Fig. 7. *Effect of compression of the abdominal aorta on the volume of the hind-limb.* Explanation of curves as before. Blood-pressure zero at level of excitation marker. Two compressions, the second not marked by the signal.

Fig. 8. *A similar curve to Fig.* 7. Blood-pressure cannula in this case in carotid. Leg nerves cut. At x, obstruction of abdominal aorta. Blood-pressure zero—48 mm. below excitation marker.

strictor reaction, of the kind described in the earlier part of this paper, so that there is, so to speak, a kind of reverberation. This second reaction is also well shown in Fig. 9, an example from the cat.

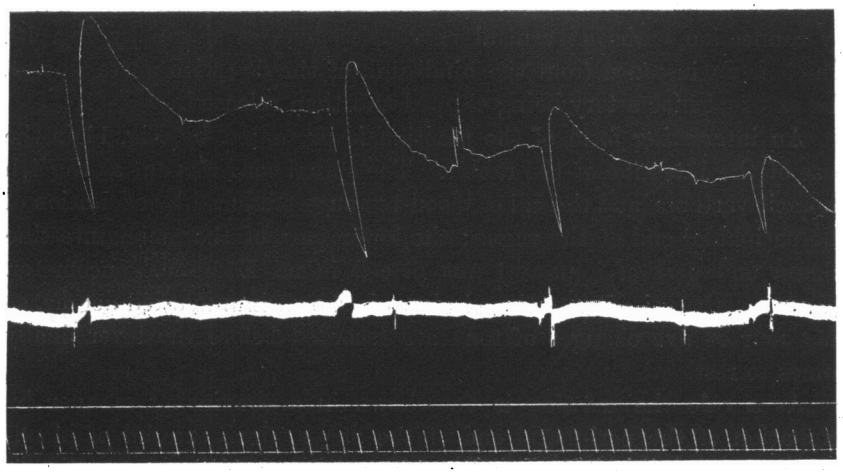

Fig. 9. *Four cases of compression of aorta producing double reaction in the enervated hind-limb of the cat.* Blood-pressure zero—45 mm. below excitation marker.

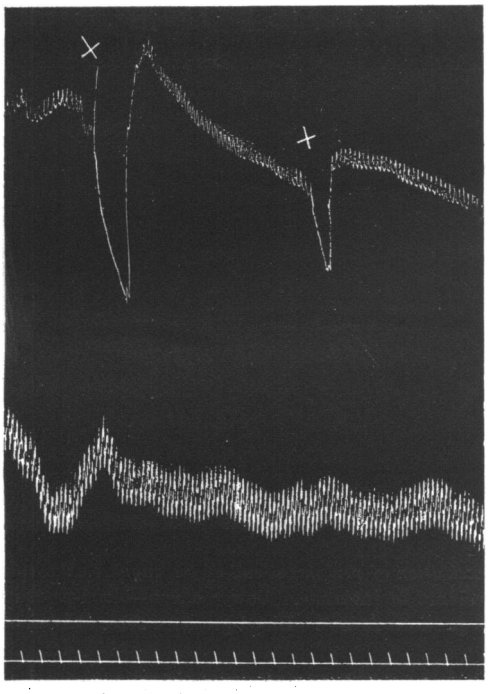

Fig. 10. *Effect of compression of iliac artery on volume of limb with nerves intact.* Explanation as previous figures. Artery clipped twice at x. Blood-pressure zero—50 mm. below excitation marker.

As remarked previously the reaction in question is not prevented by connection with the central nervous system, as shown by Fig. 10, which is an instance from the hind-limb of the dog with nerves intact, the external iliac artery being clipped and then released.

An interesting form of the reaction is presented by Fig. 11 in the case of the intestine of the rabbit. The spinal cord was cut at the 4th thoracic vertebra, and the fall of blood-pressure produced by excitation of the peripheral end of the vagus; the lower part of the small intestine (ileum and part of jejunum) was placed in an Edmunds' oncometer. The "reverberation" mentioned above is shown in a marked degree, so that four or five changes of tone are recorded before the lever finally comes to rest.

As a rule the reaction is not shown in a convincing manner by excitation of the peripheral end of the vagus, since the fall of blood-pressure so produced is usually followed by a rise, due partly to increased action of the heart, partly to slight asphyxial excitation of the vaso-motor centre, and this rise may be thought to be the cause of the expansion of the organ. In Fig. 11, however, it will be noticed that

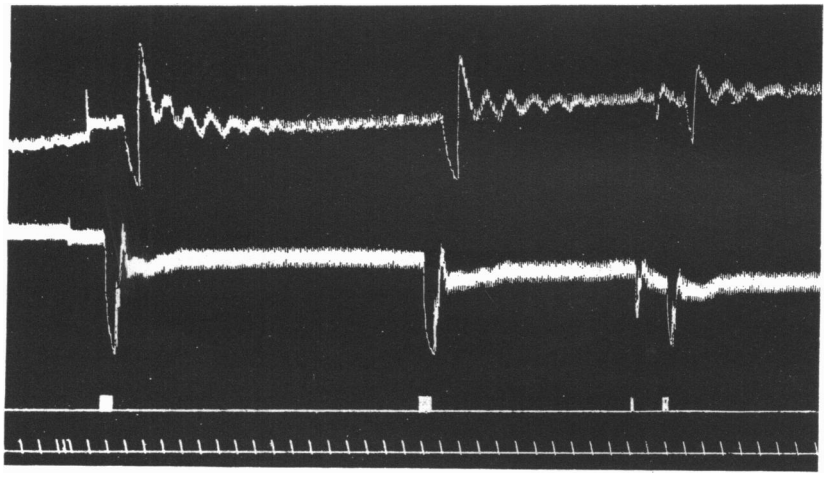

Fig. 11. *Effect of excitation of peripheral end of the vagus on the volume of the intestine.* Upper curve—volume of intestine. Lower curve—blood-pressure. Excitation and time-markers as previous figures. Blood-pressure zero, at level of excitation marker.

the rise of blood-pressure does not occur in the second excitation of the vagus, although the dilator reaction of the intestinal vessels is very marked.

It is necessary to meet a possible objection. It may be thought that the relaxation described is due to anæmia causing asphyxia of the arterial wall. That this is not so is shown by the fact that I have repeatedly seen it in a curarized animal, in which, owing to excessive artificial respiration, there was no rise of blood-pressure on stopping the pump until more than a minute had elapsed. The tissues were therefore copiously supplied with oxygen, and the obstruction of the artery produced the reaction if lasting only for a couple of seconds. In the case of the limb, moreover, it must be remembered that the muscle metabolism in a curarized animal is very small, and that a prolonged anæmia would be necessary to cause an appreciable degree of asphyxia. This objection is also answered by the experiments on excised arteries to be described below.

THE REACTIONS IN EXCISED ARTERIES.

It has been already pointed out above that these reactions are independent of the central nervous system, and are therefore of peripheral origin. Since no ganglia have been found in connection with the blood vessels it follows that the effects in question are of myogenic nature, as indeed one would expect from the known properties of smooth muscle. But, in view of MacWilliam's[1] researches on excised arteries, it seemed worth while to attempt to obtain reactions on them. Owing to the pressure of other work I have not been able to make many experiments of this kind. I have, however, obtained reactions, and in one case the effect was so marked as to be visible without the aid of any instrumental magnification. This case was that of a carotid artery removed from the body of a dog three hours after death by asphyxia, so that, if there were any ganglion-cells attached to the artery, they were presumably quite out of action. A cannula was inserted in one end of the artery, and the other end tied; the artery and cannula were filled with defibrinated blood and connected by india-rubber tubing to a mercury reservoir which could be raised and lowered. When now the pressure was raised inside the artery it was seen at first to swell, but immediately, and while the mercury was still kept at its height, a powerful contraction took place, in which the artery appeared to writhe like a worm. If the pressure was suddenly lowered again the artery did not return merely to the state corresponding to the lesser pressure, but underwent a considerable relaxation, which passed off just as in the vessels of the body as a whole. This reaction to

[1] *Proc. Roy. Soc.* 1902.

lowered pressure was better seen when the artery was inclosed in a small air oncometer, consisting of a wide glass tube connected with a piston recorder, by means of which an enlarged curve of the changes of volume could be obtained. I regret that the tracing was spoilt in varnishing, so that I am unable to reproduce it here.

The fact of the occurrence of the reaction in an excised and asphyxiated artery is of importance for three reasons:—

1. It disposes of the objection that the phenomena in question are due to any other cause than the change of tension.

2. It affords a proof of their myogenic nature.

3. In the particular experiment described above it shows that the contraction in response to increased pressure may show itself while the pressure continues, so that the form of curve given by the kidney, which I for some time looked upon with distrust, is quite a possible one, even in the complete absence of any excitation of vaso-constrictor nerve-fibres.

The meaning of the reactions.

It remains to indicate briefly how the behaviour of the blood vessels described in the preceding article may be of service in the normal organism.

The peripheral powers of reaction possessed by the arteries is of such a nature as to provide as far as possible for the maintenance of a constant flow of blood through the tissues supplied by them, whatever may be the height of the general blood-pressure, except in so far as they are directly overruled by impulses from the central nervous system.

As examples let us take first the case in which, say for cardiac reasons, a considerable general fall of blood-pressure is required. This, as we know, is produced by universal vascular dilatation (except in the brain); but, owing to the preponderance of the splanchnic area, the limbs, the relaxation of whose vessels is usually insufficient to produce an actual increase of their sectional area under the low arterial pressure prevailing, would be deprived of their due supply of blood when perhaps it might be particularly needed, were it not that their arterioles automatically relax to a still greater degree, and so make up for the lowered pressure by an increase of sectional area.

Take again the case in which as great as possible a rise, or fall, of arterial pressure is required; if the limb-vessels allowed themselves to be passively distended, or drained, as the case may be, the result would be to partially annul the effect of constriction, or dilatation, in the

splanchnic area, but, since they react in the way described, they assist in the production of the desired end.

An especially interesting case is that of the cerebral circulation. There is, up to the present, no satisfactory evidence of the presence of a vaso-motor supply to the brain; so that when an increased blood-supply is required in that organ it is provided by constriction of vessels throughout the rest of the body, which is, in this respect, so to speak the slave of the brain[1]. Now Hill has shown[2] that the tendency of increased arterial distension in the brain is to produce cerebral anæmia, owing to the skull being a rigid box, so that, although the arteries may increase in size, it is at the expense of the capillaries and veins; this swelling of the arteries is prevented by the automatic reaction of their muscular coat, as described above, so that they do not allow themselves to be distended, but become more or less rigid, and so permit the increased velocity of flow, due to increased pressure, to produce its desired effect in providing a more abundant blood-supply to the active cerebral tissue.

On the other hand it is evident that the effects of a rise of general blood-pressure of a moderate degree, produced, say, by constriction of the splanchnic area, would be to cause automatic contraction of the arteries of all other parts of the body, and thus raise still further the general blood-pressure, unless dilator impulses were sent to the vessels from the central nervous system. Thus, without the controlling government of the latter, every rise of pressure would automatically cause a further rise, and every fall, from whatever cause, a further fall of blood-pressure from peripheral vascular dilatation; *i.e.* a vicious circle would be established in the absence of the regulating functions of the vaso-motor centres.

Summary.

1. The muscular coat of the arteries reacts, like smooth muscle in other situations, to a stretching force by contraction.

2. It also reacts to a diminution of tension by relaxation, shown, of course, only when in a state of tone.

3. These reactions are independent of the central nervous system, and are of myogenic nature.

4. They are to be obtained both in the case of vessels in their normal condition in the body, and in excised arteries some hours after death.

[1] Bayliss and Hill. This *Journal*, XVIII. p. 358. 1895.

[2] *The Central Circulation*, London, 1896, p. 135.