7. Clinicians know that the pulse in the radial artery becomes more forcible when they begin to compress the arm. At the beginning of compression of the arm, the armlet, by obstructing the venous outflow and making tenser the arteries in diastole, improves the conduction of the systolic wave. The pulse in the radial artery, therefore, becomes reinforced. The dull sound and the reinforcement of the pulse are due to the same cause.

8. Evidence has been obtained then, by experiments on man, of the effect of increased tension of the arterial wall (lessened lability) on the conduction of the crest of the systolic wave.

The peripheral conditions affect the lability and the pressure readings.

The Measurement of Arterial Pressure in Man. II.—*A Schematic Investigation.*

By Martin Flack, Leonard Hill, F.R.S., and James McQueen.

(Received December 3, 1914.)

(From the Physiological Laboratory, London Hospital Medical College (London Hospital Research Fund), and the Pathological Laboratory, Aberdeen University.)

MacWilliam and Melvin* have demonstrated in the case of the excised artery—compressed in their schema—that a compressing force which was not sufficient to obliterate the pulse caused a great fall in the manometer, which they placed distally to the compression tube. To cite an example, the entering pressures in the proximal manometers were: systolic 178 mm. Hg, diastolic 118 mm. Hg. A compressing force of 140 mm. Hg caused a great fall in the distal manometers—systolic became 42 mm. Hg, diastolic 22 mm. Hg. We find that the artery, under these conditions, is flattened during diastole, and the inflow during systole is not of sufficient duration to maintain the distal pressure, supposing the resistance to outflow is unchanged. If the resistance to outflow is increased, no such distal fall of pressure occurs.

Their schema differs in essential points from the conditions which pertain to an artery embedded in living tissues and encircled by an armlet. The pressure within the armlet at first does not deform the artery, but expresses blood from, and increases the peripheral resistance in, the mass of tissue it

* 'Heart,' vol. 5, p. 153 (1914).

encloses, by compressing the capillaries and obstructing the peripheral exits. It thus converts the compressed area into a resonating mass; the pulse is not damped down in the labile arteries, but strikes the blood which fills to distension not only the main artery, but every patent arteriole throughout the mass, and causes the whole tense mass to vibrate.

Thus we find in the case of the living subject, if the systolic pressure be 115 mm. Hg and the armlet pressure be kept at 110 mm. Hg, the venous pressure in the limb rises distal to the armlet. If another armlet be put below the first, and the pressure raised within this, the pulse at the radial will not be obliterated until the pressure in this armlet reaches 115 mm. Hg. If the conditions found in the schema of MacWilliam and Melvin held good in the arm, a far lower pressure in this lower armlet would suffice to obliterate the pulse, for in their schema, under similar conditions, the distal manometers show a great diminution in pressure. In the case of the arm, as the pressure is raised in the upper armlet the venous outflow becomes obstructed, and the pulse then strikes a mass of blood congested within the vessels which permeate the tissues; no pressure less than systolic in the lower armlet can prevent the vibration of the mass reaching the radial artery. It is true that the pulse felt in the radial becomes feeble as the pressure is raised in the upper armlet to 110 mm., but the pressure in the radial does not sink, because the blood still flows in and cannot escape from the veins. The pulse in the radial is enfeebled by the resistance which arises from the deformation of the brachial artery brought about by a pressure in the upper armlet of 110 mm. Its force is partly spent in the labile artery above this armlet. The range of pulse pressure below the upper armlet is greatly diminished too, because the diastolic pressure is raised owing to the venous obstruction. There is in consequence a much smaller swing, but this swing cannot be stopped until the pressure in the lower armlet is raised to the full systolic pressure, 115 mm. Hg.

The facts we have detailed above show that the simple schema, in which an artery is compressed in a chamber full of water, does not represent the conditions which pertain in the arm.

We have attempted to imitate these conditions in the schema represented in fig. 1.

Two glass compression chambers are filled with water and connected with each other and to a compression bottle. In one is placed a piece of human carotid artery, in the other a schematic representation of the tissue vessels. This consists of a condom (thin-walled, wide rubber tube) filled to distension with chopped rubber sponge. The expansion of the condom is limited by

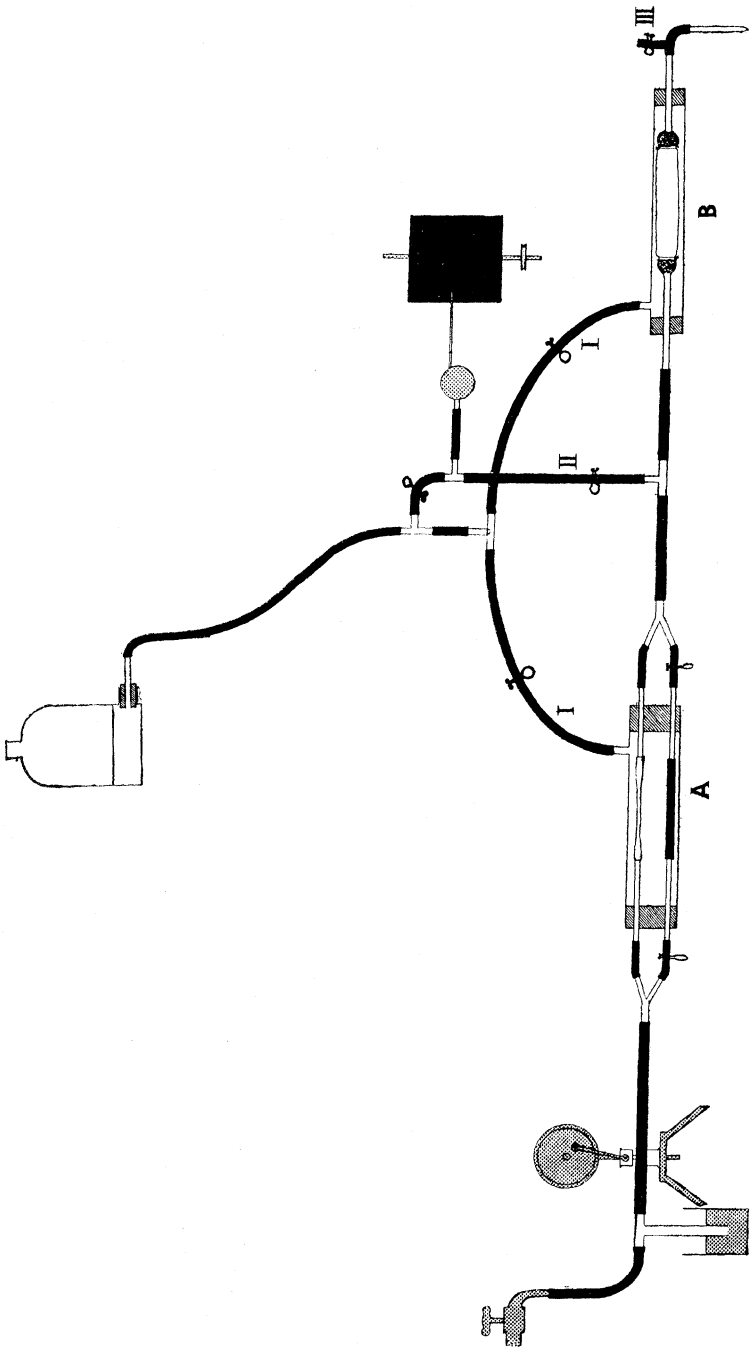


FIG. 1.

an external coat of muslin. The condom is tied at either end on to a thistle funnel; the tube of each funnel passes through a rubber cork. The corks close the ends of the compression chamber.

A pulsatile flow of water is maintained through (A) the artery, (B) the tissue schema. The water finally escapes through a glass nozzle into a pail. The resistance of the tissue schema is such as to give a continuous flow from the nozzle, marked at each systole by a slight pulsatile increase. The pulsatile flow is secured in this wise.

Water flows through the tap through a length of rubber tubing to the schema. Close to the tap a mercury valve is inserted so that the pressure in the tube is kept constant. The rubber tube is pulsed rhythmically between two wooden discs (cotton reels), one of which is fixed to the support, and the other to the piston rod itself, of Brodie's respiration pump. At each stroke of the pump the tube is compressed and the flow interrupted. The rate of the pulse can be varied. T-pieces are inserted in the schema so that the pulse and pressure can be recorded in turn from (i) one compression chamber or both chambers, (ii) the tube connecting artery and tissue schema, (iii) the outflow nozzle. An alternative pathway is arranged in the compression chamber which contains the artery, so that the flow can be directed either through the artery or through a piece of rubber tube, which acts as a rigid tube. Or two pieces of artery, one acting as artery and the other as vein, can be placed in this compression chamber; the flow is then made to pass through (1) the artery, (2) the tissue schema, (3) the vein, and so to the outlet nozzle. Or the flow can be made to go through

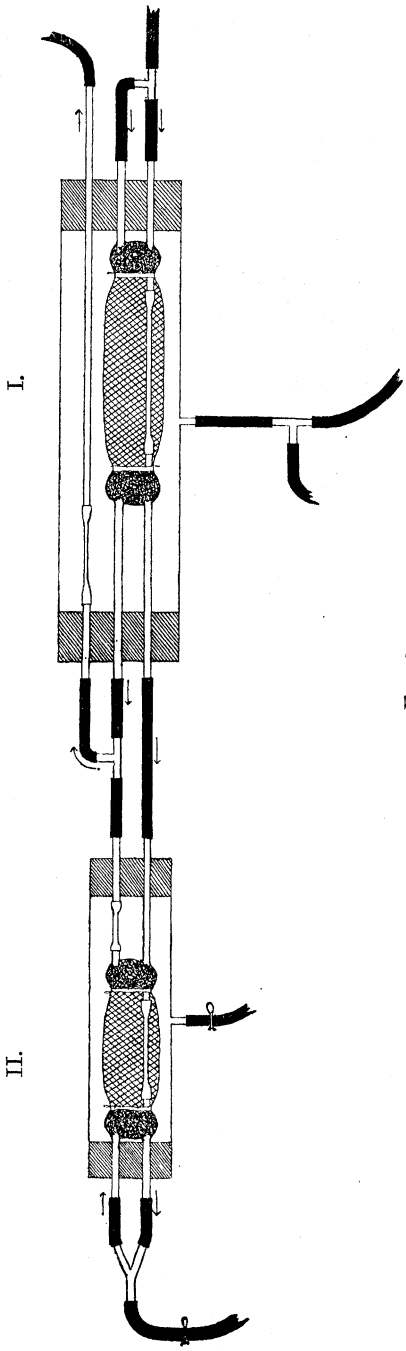


FIG. 2.

the artery and vein alone, excluding the tissue schema, or through the artery alone.

For the tissue schema we have substituted a human kidney in some of our experiments. We finally modified the above schema and made a still closer imitation of the conditions which pertain to the circulation in the arm. The artery passes through the tissue schema and is surrounded by it. The inflow tube branches and the water flows through both the tissue schema and the artery; the outflow tubes from artery and tissue schema join and pass to another length of artery placed in the same compression chamber; this acts as the vein.

We have used two such complete schemata joined in series in some of our experiments, one representing the upper arm, the other the forearm (fig. 2).

Experiment I.

We first observed the effect of circulating water through two lengths of artery—in place of one—both being placed in the same compression chamber. The water flowed through (1) the first length of artery, (2) a connecting length of rubber tubing, (3) the second length of artery, and so to the outlet (fig. 3).

When the compression chamber was connected with the recording spring

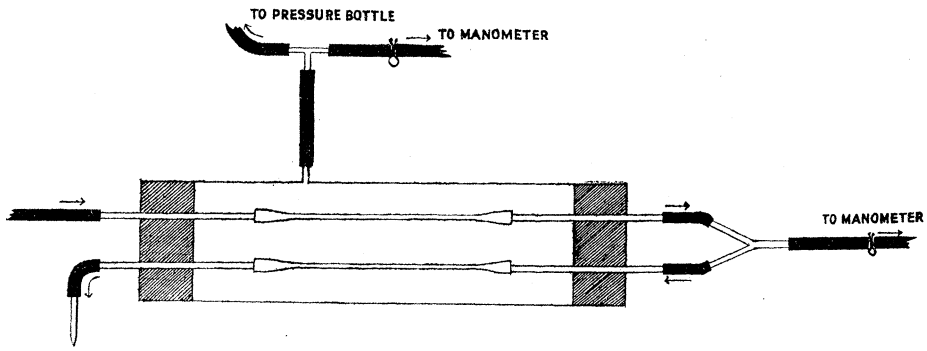
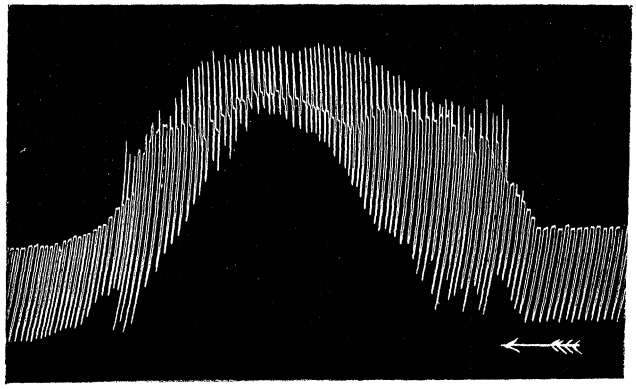


FIG. 3.

manometer the record showed that the maximal pulsation occurred at a lower level when the flow was through two lengths of artery (fig. 4) than when it was through one length (fig. 5).

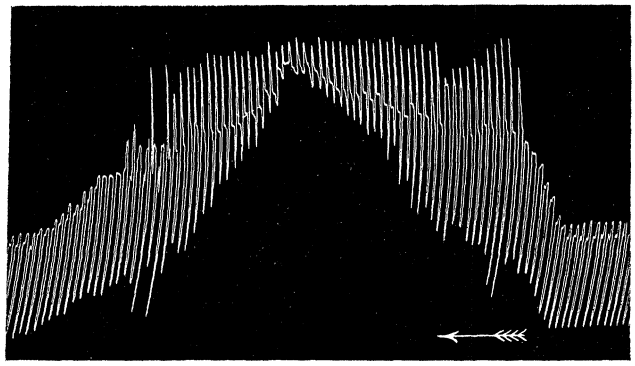
Owing to the frictional resistance in the length of tube through which the water flowed, the systolic pressure was partly spent in distending the labile first length of artery and in overcoming the frictional resistance during diastole; the second piece of artery, in consequence, had the lower diastolic

pressure, and was thus the first to be deformed and give its maximal swing. The first length of artery became taut, owing to the rise of diastolic pressure,



Off. On.

FIG. 4.



Off. On.

FIG. 5.

as the second length of artery was flattened. Finally, the first piece of artery was deformed by the increase of compression and gave its maximal swing. The excursion of this maximal swing, owing to the increased diastolic pressure, was smaller, and the pressure at which it occurred higher, than was the case when this length of artery was compressed by itself.

In one such experiment the following were recorded:—

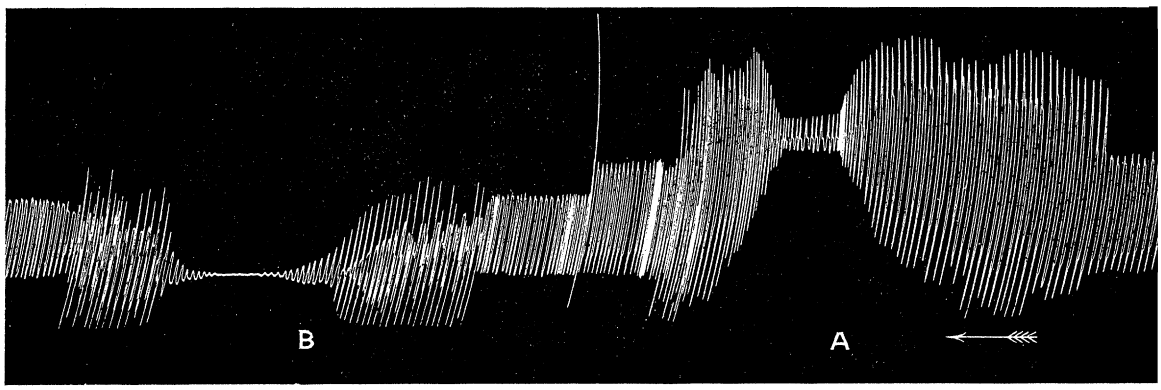
Compression.	Deformation.	Outflow per minute.
		c.c.
One length of artery—		
0 cm. H_2O	Nil	210
15 ,,	Nil	
45 ,,	Begins to flatten visibly in diastole	160
85 ,,	Flat in systole	Nil
Two lengths of artery—		
0 cm. H_2O	Nil	182
15 ,,	Second length of artery begins to deform	186
45 ,,	Second length flat in diastole	106
67 ,,	First length of artery flat in diastole	Drops
85 ,,	First length of artery flat in systole	Nil

In Experiment I the conditions, of course, are not the same as those which pertain in the arm, for in the arm there is the capillary field with its resistance which precludes the pulse from reaching the veins. However, the experiment shows that the behaviour of the artery is notably influenced by the compression of a vessel placed distally to it, and therefore that the study of the compression of a length of artery placed in a simple schema does not suffice to elucidate the compression of the brachial artery in the arm.

This conclusion is confirmed by Experiment II.

Experiment II.

We repeated Experiment I, but recorded the pressure in the tube which joined the first and second length of artery. On raising the compression to 5 cm. H_2O the second length of artery began to flatten in diastole, the first length became distended in diastole, and the record then showed a rise in pressure. A maximal pulse developed as the compression increased.



Off. On. Off. On.

FIG. 6.

Finally the first length of artery flattened (fig. 6, A). On repeating the experiment upon the first length of artery by itself we found it began to flatten and give a maximal pulse at 38 cm. H_2O; the pressure then fell in the manometer and reached zero as the compression was increased (fig. 6, B).

Experiment III.

We varied Experiment I by arranging the outlet in a **U**-tube containing mercury, so that the resistance to outflow and diastolic pressure increased *pari passu* with the compression (fig. 7). The diastolic pressure being thus

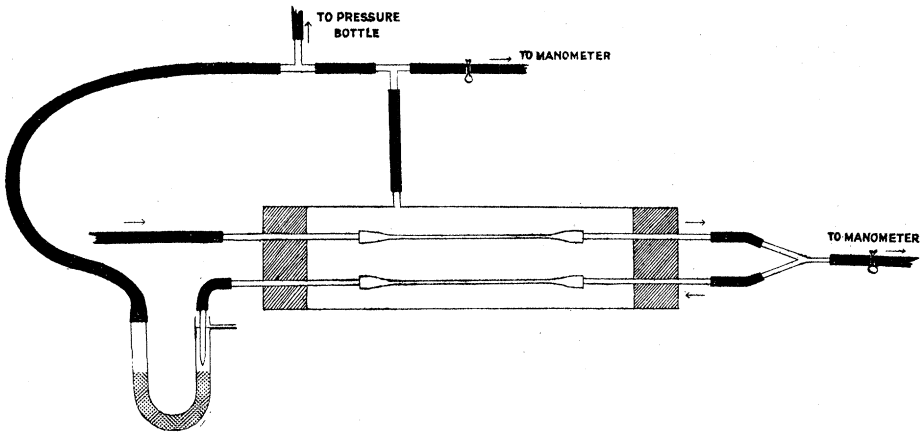


FIG. 7.

raised *pari passu* with the compression, the maximal pulsation appeared just before the point at which the artery was flattened in systole. The result under these conditions was, of course, the same whether one length or two lengths of artery (or artery, tissue schema, and vein) were used. Their walls were equally stretched and made more and more rigid by the rising diastolic pressure. There was thus little loss of systolic force as the pulse passed along the tubes. On the other hand, the pulse transmitted to the compression chamber and recorded by the manometer became less as the arterial wall became more rigid. When the diastolic pressure which pertained in the schema was over-topped, the length or lengths of artery began to flatten, and a maximal pulse resulted.

MacWilliam and Melvin* conclude from their study of the simple schema that the diastolic pressure and maximal pulse do not always coincide. By our experiments we bring into play the effect on the artery of obstructed venous outflow, and find the diastolic pressure and maximal pulse in agreement.

* 'Heart,' vol. 5, p. 153 (1914).

For let us consider the arm when it is compressed. The veins and capillaries under the armlet are first flattened and some are emptied, the pressure rises *pari passu* with the compression in all the remaining patent blood-vessels enclosed by the armlet. This must be so, for their outlet is obstructed. In the forearm, peripheral to the armlet, the venous pressure will steadily rise, and the veins become more and more swollen and tense if the compression is maintained just below the arterial pressure. Under these conditions the arterial blood can flow into the forearm, so long as capillary fields, hitherto empty, open out and the veins swell, the forearm becoming more and more congested.

If a second armlet be put just below the first, and the pressure raised equally in the two armlets, the conditions are made the same as in Experiment II, in so far as the peripheral resistance in the arm is concerned. But, be it noted, as the blood has other arterial pathways open to it in the rest of the body, the diastolic pressure in the arteries enclosed by the upper armlet is not raised nearly up to the systolic pressure. To make the conditions in that the same as in Experiment II, the resistance to outflow in all the arteries would have to increase *pari passu* with the compression of the arm.

In the case of the vessels enclosed by the lower armlet under these conditions the diastolic pressure is raised nearly up to the systolic pressure. Now, we have determined experimentally that the reading of systolic pressure taken with an armlet round the calf is raised by placing a second armlet round the thigh and raising the pressure therein to, and keeping it at, say, 50 mm. Hg. This correspondingly increases the diastolic pressure in the veins and arteries of the leg, and the arteries, being made more rigid thereby, conserve better the crest of the systolic wave in its passage from thigh to calf. Similarly, the pulse in the radial artery increases in amplitude at first when the compression is raised in an armlet placed round the upper arm, because the compression by obstructing the outflow and making tenser the arteries aids the conduction of the systolic wave.

Experiment IV.

A single length of artery was compressed and the outflow measured. The compression chamber was connected with the manometer. When the compression reached 25 cm. H_2O the artery began to flatten. At 34 cm. H_2O the pulse became maximal, and the water then issued in strong pulses; 212 c.c. flowed out in one minute. At 47 cm. H_2O the water issued in shorter pulses, for the artery remained deformed for a longer period during each diastole; the outflow was reduced to 166 c.c. At 70 cm. H_2O the outflow was reduced to feeble spurts synchronous with the systoles; while at 77 cm.

H_2O the outflow ceased to pulse and was reduced to fast drops. The artery then appeared flattened all along its length, but its end proximal to the pump was slightly expanded by each systolic wave. At 87 cm. H_2O drops still escaped from the outflow nozzle.

Experiments on excised arteries have been undertaken* to test the correctness of the obliteration method of measuring the systolic blood pressure, and the complete cessation of outflow has been taken as the index of obliteration. Wrong conclusions have thus been drawn as to the power of the arterial wall to resist compression. The disappearance of the pulse at the distal end must be taken as the index of obliteration, not the absolute cessation of outflow.

Experiment V.

The flow was through (1) a length of artery, (2) tissue schema, (3) a second length of artery acting as vein. All these were placed in the same compression chamber, and this connected to the recording manometer (fig. 8). The

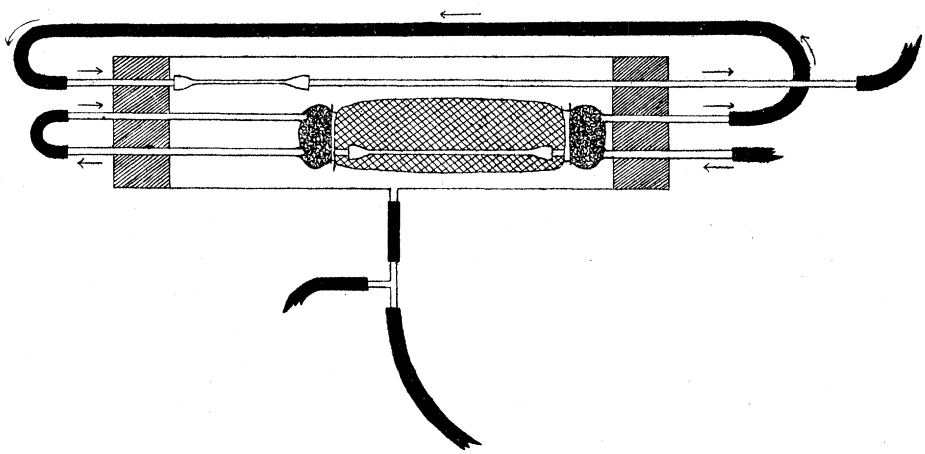


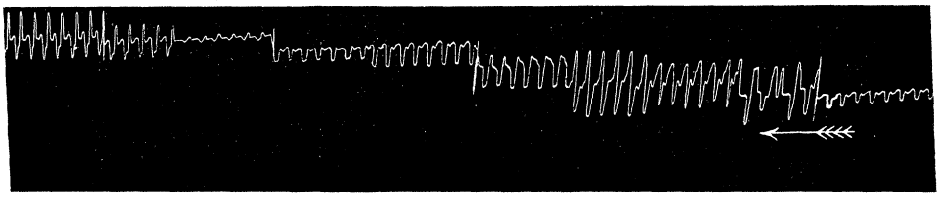
FIG. 8.

tissue schema was not tightly packed with chopped sponge, and the pulse travelled through it to the vein.

On compression the vein first flattened and gave a maximal pulsation, while the artery became taut, then the tissue schema shrank. The outflow at this period was partly due to the water expelled from it. The recorded pulse now became very small as the whole system (artery, tissue schema, vein) was raised up to the diastolic pressure and approximated to a rigid system. Finally, the diastolic pressure was overtopped in the artery, and this gave a maximal pulse (fig. 9).

* Herringham and Womack, 'Brit. Med. Journ.,' 1908, B, p. 1614.

65 60 50 40 10 5 0 cm. H_2O.



Artery maximal pulse. Vein maximal pulse. On.

FIG. 9.

The following were the outflows at each stage:—

Compression.	Outflow.	Compression.
cm. H_2O.	c.c. per min.	
0	146	
5	71	Vein beginning to deform during each diastole.
10	63	Maximal pulse of vein.
40	33	Tissue schema shrinking. Manometer scarcely pulses at all.
60	11	Artery beginning to deform during each diastole.
65	2	Maximal pulsation of artery.

Experiment V elucidates the behaviour of the brain when compressed. When the brain is compressed by fluid forced into the subdural cavity the capillaries, venules, etc., similarly shrink, the pressure rises in these vessels, and the whole cerebral vascular system approximates to a rigid system and gives a small cerebral pulse. Similarly, when the armlet compresses the arm, part of the blood contained in the tissue vessels is expelled and the remaining patent vessels approximate to a rigid system, in which arterial pressure pertains and through which a diminished flow continues until these are emptied; the artery itself is then flattened; that is, when the systolic pressure is overtopped.

Experiment VI.

In Experiment VI the flow was arranged through the artery and the tissue schema placed in separate compression chambers. These chambers were connected with each other and the manometer (fig. 1).

A. On compression the tissue schema first shrank, then the artery began to flatten and the maximal pulse resulted. On decompression the maximal pulse was more ample and occurred at a lower level than on compression (fig. 10).

Like results were obtained when the tissue schema was replaced by the

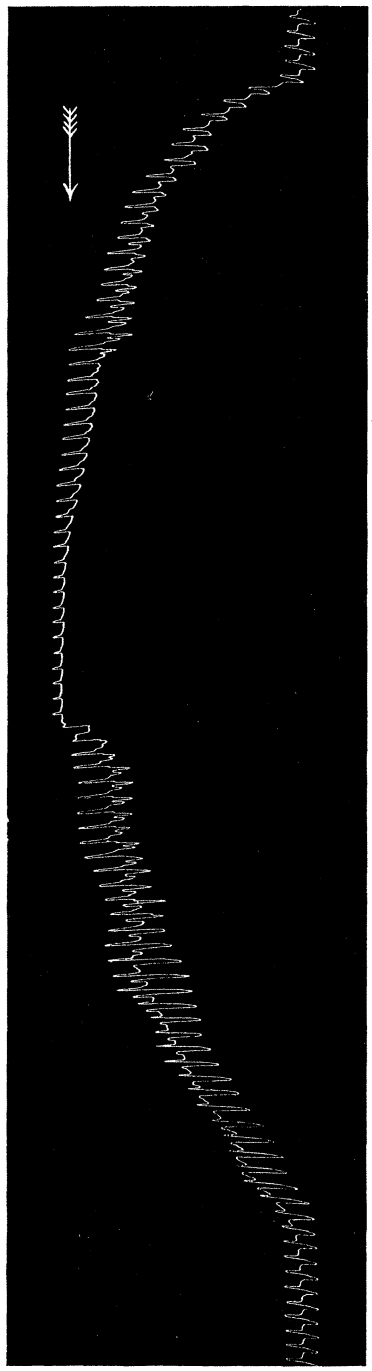


FIG. 10.

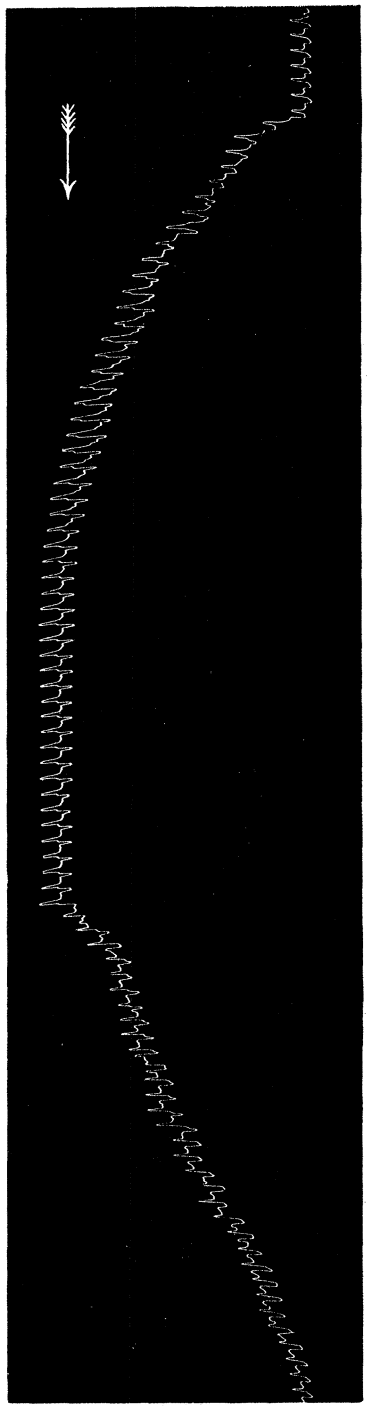


FIG. 11.

kidney, the length of artery being connected to the renal artery and the renal vein to the outflow.

The compression chambers being connected with each other, a part of the pulsatile force transmitted through the artery to its chamber is conveyed to the chamber of the tissue schema and helps to pulse fluid out of the tissue schema.

When decompression is begun the tissue schema is shrunken, and it takes time to fill out. The outflow is in drops while the expansion is going on. The pulsatile force transmitted through the artery to its chamber is now less spent on the shrunken schema, for this is a more rigid structure. On the other hand, the pulse transmitted directly along the artery to the tissue schema spends part of its force in expanding the shrunken tissue schema. So long as the tissue schema acts as a rigid structure and stores little of the systolic force, the diastolic pressure in the artery will fall to lower level, and, in consequence, the pulsatile swing will be bigger.

The recorded pulse is the summation of that from either chamber, the pulse of the artery, and of the tissue schema.

There is a certain degree of expansion of the tissue schema, which favours the development of a maximal pulse, the stage when the arterial pulse is spent least on, and reinforced most by, the tissue schema.

If the compression and decompression be done in stages, and time be given between each stage for the tissue schema to shrink or expand, then the maximal pulse occurs at the same height on decompression as on compression.

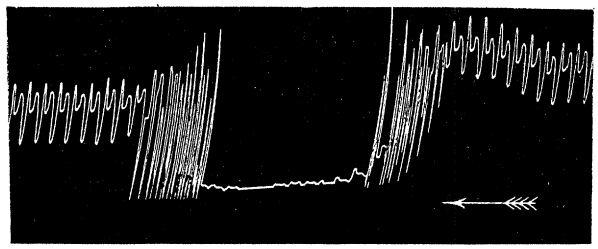
B. The share which the tissue schema takes in the phenomena is shown in fig. 11. The artery was replaced by a rubber tube (rigid). On compression the tissue schema shrank and gave a maximal pulse. On decompression the tissue expanded, and the recorded pulse in this case became smaller because the pulsatile force was spent largely on the expansion of the tissue schema.

Phenomena of the same order happen when the tissues of the arm are compressed by the armlet, and hence arise those differences between compression and decompression readings of systolic pressure which are so often recorded.

Experiment VII.

The flow was through a length of artery and the tissue schema placed in series (fig. 1). Each was in a separate chamber, and these were joined together and to the compression bottle. The tube connecting the artery and tissue schema was joined to the recording manometer.

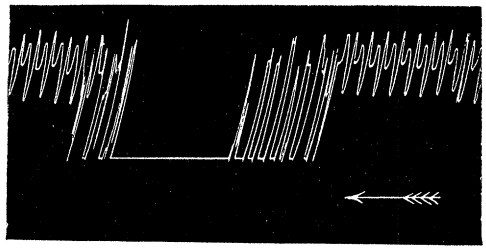
A. Compression of both artery and tissue schema (fig. 12). The pressure first rose and the pulse amplitude diminished owing to the shrinkage of the



Off. FIG. 12. On.

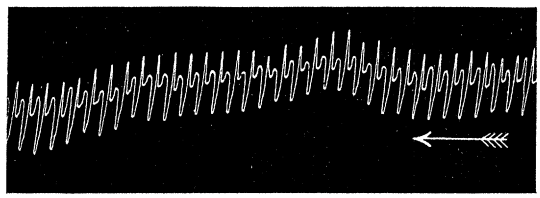
tissue schema, and the greater resistance thus developed in it, and higher diastolic pressure consequent in the artery. Then a fall of pressure accompanied by maximal pulses resulted owing to the artery flattening during diastole. Finally the artery shut up and the pulse ceased to reach the manometer. On taking off the compression the pressure and outflow did not return to their previous amounts until the shrunken tissue schema had expanded.

B. The compression tube leading to the tissue schema chamber was closed. On compressing the artery it began to flatten, and gave maximal swings, and then shut up. No rise of pressure occurred as in "A," because the tissue schema was not compressed (fig. 13).



Off. FIG. 13. On.

C. The compression tube leading to the artery chamber was closed (fig. 14). On compressing the tissue schema the pressure rose and the pulse



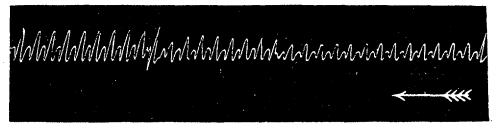
Off. On.
FIG. 14.

amplitude diminished; the tissue schema shrank and became more rigid as the resistance to flow was increased. The artery was not itself compressed in this experiment, but acted as a rigid tube in its closed chamber.

Experiment VIII.

The flow was through two lengths of artery joined in series. Each length was placed in a separate compression chamber. Tubes connected the two chambers with each other and with the manometer.

A. The tube leading from the compression chamber of the vein was closed. The artery alone affected the manometer.



A. C. B.

FIG. 15.

B. Both compression chambers were open, both artery and vein affected the manometer.

C. The compression chamber of the artery was closed. The vein alone affected the manometer.

When both chambers were open the pulsatile expansion of the artery was transmitted from the arterial to the venous compression chamber, and partly spent itself in pulsing fluid out of the vein. When the venous chamber was closed this no longer occurred, and the vein became a rigid tube and no longer stored the systolic force. The diastolic pressure therefore fell in the artery, and this gave a bigger swing, which acted with undivided force on the recording manometer. The like result was obtained when we replaced the second length of artery by a kidney, and connected the renal vessels, so that the flow went through (1) the length of artery, (2) renal artery, (3) renal capillaries, (4) renal vein. The effect was not obtained when we substituted a length of rubber tube (rigid) for the length of artery.

In the case of the brain or other encapsulated organ the arterial pulse transmitted through the substance of the organ helps to pulse blood out of the venous sinuses. In the case of the kidney urine is pumped out of the collecting tubules as well as blood out of the renal veins by each pulsatile expansion of the organ.

Experiment IX.

The following was an experiment in which the result is to be explained on the same lines.

The flow was through the artery and the tissue schema placed in series and each in a separate compression chamber. These chambers were connected with each other and with the pressure bottle.

The tube connecting the artery with the tissue schema was connected with the manometer. The compression was raised until the artery flattened in diastole and the manometer gave maximal swings. On closing the tube leading to the compression chamber of the tissue schema the artery flattened to a greater extent and the pulse amplitude became diminished (fig. 16). The explanation is as follows :—

The tissue schema then became a rigid structure and no longer stored the

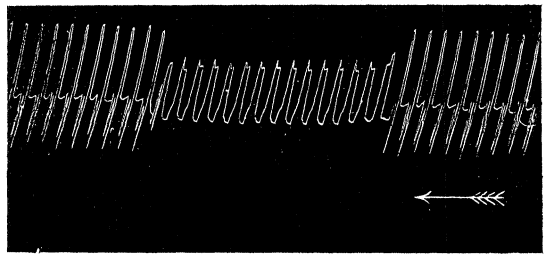


Fig. 16.

systolic force. Therefore the diastolic pressure in the artery diminished and the artery was more effectually closed by the compression during diastole. The systole in its turn was less effectual in driving fluid through the artery. The systolic force was spent more on the lability of the artery, *i.e.*, in opening it out.

Like results were obtained when we substituted the kidney for the tissue schema.

When the chamber containing the artery was closed in place of that

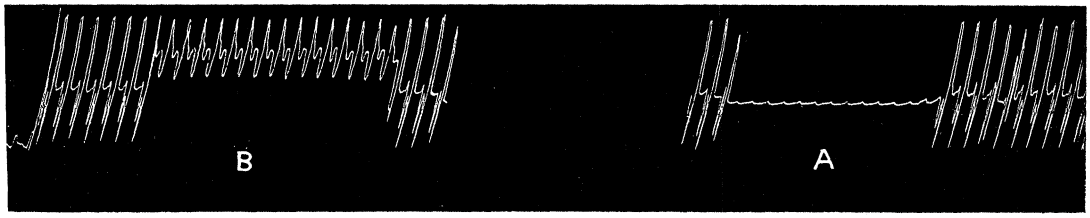


Fig. 17.

containing the tissue schema, the results were as follows, when the compression was arranged to produce a maximal pulsation. When the chamber was closed at the moment of diastole the artery became fixed in diastole and remained flat, so that the pulse scarcely came through (fig. 17, A). Closing the outlet now restored both pressure and pulse, for as the flow was thus completely stopped the artery distended. On the other hand, when the chamber was closed at the height of systole the artery was fixed fully open and the pulse came through, but with a diminished diastolic excursion (fig. 17, B).

Experiment X.

The artery and vein were in one compression chamber, the kidney in another separate chamber. The flow was from artery to kidney to vein. The compression chambers were connected with each other and to the manometer.

A. The compression chamber of the artery was closed. On compressing the kidney alone the kidney shrank, and the pulse of the renal arteries became at first more ample owing to lessened lability of the kidney vessels. Finally, the outflow almost stopped owing to the resistance in the kidney, and the renal artery, becoming distended, ceased to pulse. On now compressing the artery and vein in their chamber to a like amount the resistance increased in the vein, and the kidney expanded so that the pulse and flow began again. It took a much higher degree of compression applied simultaneously to artery, kidney, and vein to stop the pulse and flow.

B. The compression chamber of the kidney was closed. The artery and vein were compressed alone. The vein was flattened and the flow ceased, the artery becoming distended, and the systolic force spent in expanding the labile kidney. On now compressing the kidney, this organ shrank and became less labile (more rigid), and the pulse and flow began again.

Experiment XI.

The flow is through (1) artery, (2) kidney. Each is placed in a separate compression chamber. These chambers are connected together. The tube connecting artery and kidney is joined to the manometer.

A. The compression is increased in both chambers. The kidney shrinks, the pressure rises, and a maximal pulse of the arteries develops; as the pressure is made greater the flow and pulse cease. On decompression the maximal pulse occurs at a lower level than on compression (fig. 18).

B. The tube leading to the compression chamber of the artery is closed and the artery thus made rigid. The pulse transmitted to the manometer becomes ampler, because it is no longer damped down in its passage through

the labile artery (fig. 19). That this is so is shown by the fact that a like result is obtained when a piece of rubber tube (rigid) is substituted for the artery and the compression chamber is left open.

Off. On.

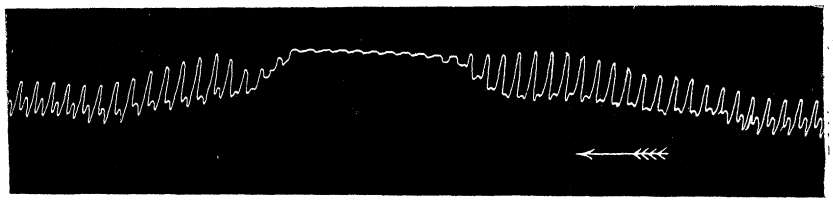
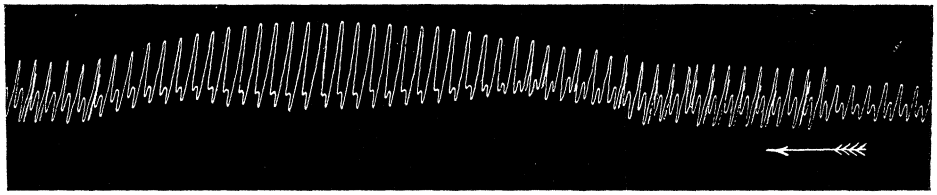


FIG. 18.



Off. On.

FIG. 19.

When the kidney is compressed the pressure rises and the pulse recorded by the manometer becomes increased still more. The explanation is as follows:—

The kidney shrinks and the resistance to the flow through its vessels increases. The renal vessels become rigid and no longer store up the systolic pressure. The diastolic pressure falls in consequence, and the full swing of the pulse is thrown upon the manometer.

Experiment XII.

The flow was through the two complete schemata placed in series, and each in a separate compression chamber (fig. 2). The compression chamber of schema II was connected to the manometer.

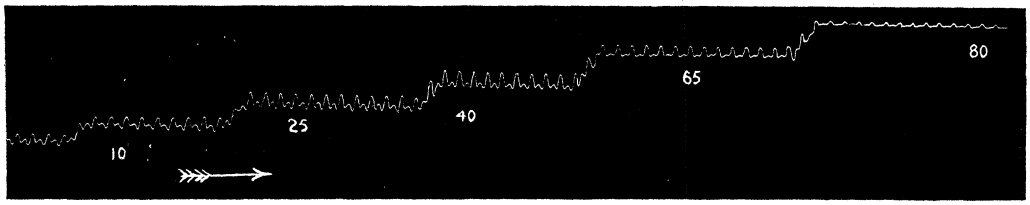


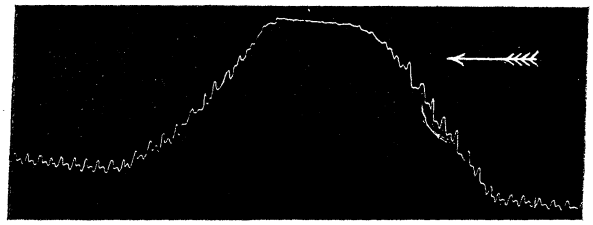
FIG. 20.

A. The first effect of compressing schema I was to increase the amplitude of the pulse in schema II (fig. 20). In this experiment the pulse in schema II was largest when the compression in schema I rose to 40 cm. H_2O.

On compressing the arm the pulse in the radial at first increases in amplitude—a fact well known to clinicians. The tracing in the previous paper shows this increase in amplitude as recorded in the radial artery. Our explanation of this phenomenon is as follows:—

The first effect of increasing the compression was flattening of the venous outlet which lay in the first schema. The resistance to outflow was thus increased and, in consequence, the diastolic pressure rose and the arteries and tissue schemata became more rigid, *i.e.* less labile. Therefore the pulse was less spent on the lability of the artery in the first schema and reached the second schema with greater force. On decompression of the first schema the pulse reappeared in the second schema at a lower degree of pressure than that at which it disappeared on compression of the first schema.

This was only the case, let it be noted, when the decompression was rapid (fig. 21). As we have said before, the same phenomena is often observed



Off. On.

Fig. 21.

when the systolic pressure is measured on man. The reappearance of the pulse generally gives a lower reading than the disappearance. On rapid decompression the tissue schema I was suddenly made easily extensile and, in consequence, the diastolic pressure of the artery fell and its lability increased. So, too, in the case of the arm when the pressure of the armlet is reduced. The full force of the pulse will not reach the second schema (or forearm) until the first schema (or upper arm) is filled and the artery rendered tenser. If the forearm is previously emptied of blood by elevation and bandaging before compression, it takes much longer for the pulse to return to its full force on decompression—the bandage being removed before decompression is made. This, too, was the case when the second schema was compressed before the first schema was compressed, and the compression of the second schema removed just before the decompression of the first schema.

Not only the tissue schema of the first but that of the second schema has then to be expanded and rendered tense before the pulse of the second schema becomes restored.

Our experiments demonstrate, then, the important influence which the tissue vessels have on the conservation of the pulse in the main arteries.

Let us now return for a moment to an experiment we published in a previous paper.* On fomenting with hot water the lower arm, and icing the upper arm, we found the radial pulse was obliterated by a pressure which was less when the armlet was applied to the upper arm than when it was applied to the lower arm. In the cooled upper arm the tissue vessels and veins were constricted and emptied. In the warm lower arm they were flushed and filled. The compression applied to the latter at once raised the diastolic pressure in the arteries, and by increasing their tension, *i.e.* lessening their lability, improved the conduction of the crest of the systolic wave. This was not so in the case of the cold upper arm. The pulse was not so well conserved by the action of the tissue vessels there, and the brachial artery was thus deformed at a lower pressure. We see then how potently the condition of the peripheral circulation may influence the reading of systolic pressure.

There is an experiment published by MacWilliam, Kesson, and Melvin,† which, it is claimed, refutes all the proofs we have brought forward as to the effect of the lability of arteries on the conduction of the pulse wave. These authors figure two lengths of tubing, one rigid tube, the other artery, connected by a T-piece to the aorta of a cat. They connect first one and then the other of these tubes to the manometer, and find the pulse records are the same. Therefore, say they, the artery is no more labile than the rigid tube.

We would point out that under their conditions the pulse, as recorded, is affected by the lability of the artery whichever tube is connected to the manometer. To make their experiment adequate they must clip off the artery while recording the pulse from the rigid tube. The lability of the artery comes no less into play when it is connected by one end to the rigid tube, as we showed in our previous communication.‡

Conclusion.

1. The simple schema, hitherto used for studying the compression of excised arteries, does not reproduce the conditions which pertain when the arm is compressed.

* 'Roy. Soc. Proc.,' B, vol. 87, p. 344 (1914).

† 'Heart,' vol. 4, p. 393, Experiment 7 (1913).

‡ 'Roy. Soc. Proc.,' B, vol. 86, p. 365 (1913).

2. A schema has been constructed by us in which artery, tissue vessels, and vein are represented.

3. This schema demonstrates some of the principles which govern the circulation in the brain or other encapsulated organ, and the effect of compression upon circulation in such organs.

4. Two such schemata arranged in series imitate the conditions which pertain respectively in the arm and forearm, and enable us to demonstrate the effects of compression, and in particular the conserving effect on the pulse of the tissue vessels.

5. The schemata demonstrate and elucidate these well-known clinical facts: (1) that the pulse may reappear on decompression at a lower pressure than it disappears on compression; (2) that the radial pulse is reinforced when the compressive force applied to the upper arm is below the diastolic pressure.

6. The schema demonstrates (1) that the maximal pulse occurs when the diastolic pressure is just overtopped by the compressive force, and is a good index of diastolic pressure; (2) that the diastolic pressure is raised towards the systolic pressure in proportion as the peripheral resistance is increased by compression or obstruction of venous outflow; (3) that this rise of pressure occurs throughout arteries, tissue vessels, and veins; (4) that the pulse is damped down by the lability of the vessel wall, and it is owing to lability that the pulse on decompression returns at a lower pressure level than it disappears on compression.

7. The explanation of the difference which pertains between the arm and leg systolic readings, taken in the horizontal posture, in cases of aortic regurgitation, is to be sought in the difference of the lability of the artery and the conditions of the peripheral circulation. The upper limb, as a whole, is more labile than the leg. In the normal person the difference of lability is brought out by exercise, which produces a big systolic wave. The leg is, so to speak, a tighter drum-head, and responds better to the bigger stroke.

It must always be borne in mind that the support of the column of blood in the artery is not only formed by the tissues of the arterial wall, but also by the surrounding tissues of the whole limb.