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The Action of Epinephrin upon the Muscle Tissue of the Vein

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THE ACTION OF EPINEPHRIN UPON THE MUSCLE TISSUE OF THE VEIN.

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The physiology of the venous circulation has, in comparison to that of the arterial, received very little consideration. It has been looked upon as a circulation carried on in a system of inert, elastic tubes, carrying the blood back to the heart from the periphery and without much, if any, physiological action on the part of the venous wall in the process. We are in the habit of looking to the heart, the arterial tension and the skeletal muscle action as being the forces driving the blood through the venous system.

The venous part of the circulation is of equal importance to the arterial, for under the conditions as they exist the one cannot be without the other and it is hardly to be expected that in so important a process the tissue would be left with only the physical force of elasticity upon which to depend for meeting the variable conditions to which it must be subjected. The attention now being given to the study of the venous circulation and the physiological activities of the venous wall has already given us a better understanding of phenomena connected with the general circulation and has changed our explanations of some of them.

One of the most important of the problems connected with the general circulation is that of surgical shock, a conditions which has been commonly described as being primarily the result of an alteration in the arterial portion of the circulation but the better understanding of the venous circulation leads us to place the phenomena as of venous origin and not arterial. It was in carrying out some experiments as to the true nature of shock that the experiments here reported were undertaken.

In shock the condition which has been most commonly accepted is one of extreme low arterial tension resulting from a more or less complete fatigue of the vaso-constrictor centres. The natural constricting impulses to the arteries then fail to pass through the centres, and without these impulses the arteries dilate and a low blood pressure results.

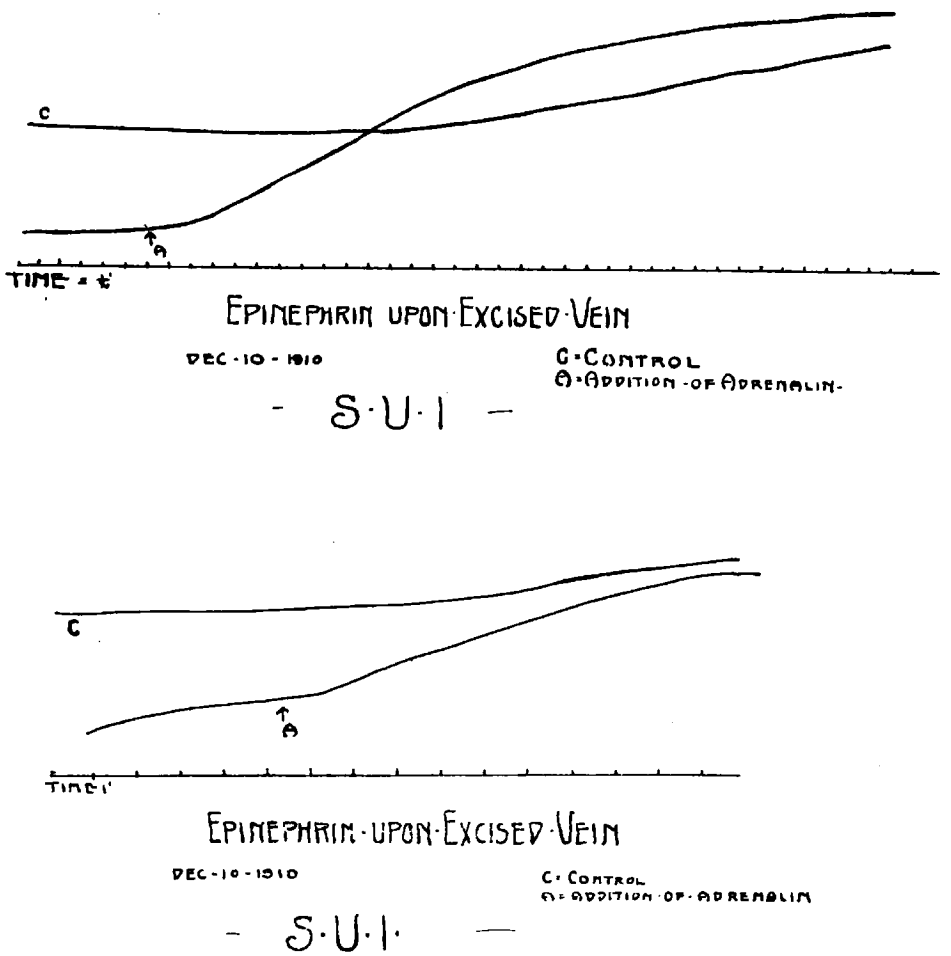
The experiments of Henderson, Porter, Seelig and others show that the condition of the artery in shock is one of extreme constriction and the low blood pressure is the result of a venous stasis brought about by venous dilatation from a loss of tonus in the venous wall.

The use of epinephrin, or under the proprietary name adrenalin, has been widely recommended in conditions of shock because of its stimulating effect upon the circular muscle of the arterial wall, constriction of the vessel follows this stimulation and an elevation of the blood pressure is thus brought about.

Under the extreme condition of arterial constriction described by Prof. Henderson to exist in shock, the beneficial effects of epinephrin must be accounted for in some other way than by any action upon the already over constricted artery. In the discussions upon the effect of ephinepherin in causing a rise of blood pressure its action upon the arterial wall alone as has been considered and we have found no statements as to its effect upon the venous wall although we find smooth muscle here as in the artery but of course not to anything like the amount. To obtain some direct evidence as to the action of epinephrin upon the vein and thus possibly upon the blood pressure the following experiments were undertaken:

From an anesthetised dog rings $\frac{1}{8}$ inch wide were cut from the external jugular vein. Two such rings of the vein were used at a time, one for control and one to show the action of the drug. The rings were connected to light writing levers in such a way that any contraction as would produce a constriction of the vessel would cause an elevation of the writing point and this would be recorded upon the surface of a smoked drum revolving at a very slow speed. The time involved from the removal of the tissue until the proper connections to the writing lever had been made and the tissue immersed in the whipped blood of the same animal was not more than two or three minutes and the tissue was still irritable and free from post mortem changes.

After imersion a record of the position of the two levers was made lasting for from 2 to 8 minutes in different experiments. After thus establishing a normal, quiet condition in the two rings a few drops of a 1-1,000 solution of adrenalin was added to the blood containing one of the immersed rings of the vein. The concentration of the adrenalin in blood was about 1-100,000. The curves in figures I and II show a rather sudden elevation of the lever following a latent period of about one minute after the addition of the epinephrin and the contraction lasting for an indefinite period before relaxation took place. Upon direct inspection of the vein we found it contracted so as to have a



lumen of 1-2 to 1-3 that of the control while they were of the same size at the beginning of the experiment. This experiment was repeated a sufficient number of times to give us conclusive evidence that the muscle tissue of the vein is stimulated by the adrenalin and that it will cause in the excised external juglar vein a marked constriction of the lumen.

Theoretically this is what we should expect as the muscle tissue which is present in the vein is well supplied with sympathetic nerve fibres, and according to the work of Thompson, Langley and Bancroft, they have, when stimulated a true constricting influence upon the vein. It has been further shown that the action of epinephrin is similar to the effect of stimulating the sympathetic fibres supplying the muscle tissue. Thus in the circulatory system it is a constrictor influence while in the intestines it causes a relaxation of the muscle. If the analogy between sympathetic action and epinephrin is correct the contraction

of the vein should be expected and this is what we have found from our experiments.

The intravenous injection of epinephrin into the intact vein has not as yet shown any rise in the venous pressure as far as experimental evidence goes. Prof. Henderson in the American Journal of Physiology of November, 1910, reports that he has been unable to produce any change in venous pressure by the use of epinephrin, while he has been able to do so with the use of CO_2.

There are, however, many factors which must be taken into consideration before a final statement can be made. The fact that slight changes of increased pressure are easily compensated for by the dilation of the very extensive and elastic venous structure; in fact that in venous circulation we have practically at one end, the cardiac, an open channel and thus the easy escape of blood will prevent any marked change in the venous pressure unless the contraction of the vessels is maintained for some time, this epinephrin does not seem to do as its action only lasts for a comparatively short time. The arrangement of the muscle structure found in the veins is also of importance in considering possible changes in blood pressure which can be produced by the contraction of the venous wall. While we do find in most veins some circular muscle fibres the extent of such fibres is very small and they are much scattered over the vein. The arrangement of muscle is mostly in a longitudinal direction and thus the stimulation of the muscle while it may produce a slight contraction of the circular fibres has most effect in shortening the vessel and in the establishment of a more rigid wall rather than in the constriction of the lumen and the elevation of blood pressure.

Further experiments are needed before the true effect of the injection of epinephrin into the intact vein is determind. For while it may cause the constriction of the excised vein this effect as far as producing changes in blood pressure may be lost because of a greater and more effective change in some other direction when the intact veins are taken together as a whole.

In conclusion we may say:

1. Experiments are here reported that show that epinephrin will cause the constriction of the excised external jugular vein, similar to its effect upon the arterial wall.

2. That while intravenous injection may cause the constriction of some veins the lack of circular muscle in many veins prevents its uniform action throughout the venous system.

3. The beneficial effect of intravenous injection of epinephrin in conditions of low blood pressure with a constricted arterial system may be due to the establishment of a more rigid venous wall by the contraction of the longitudinally place muscle fibres in the veins.

4. More experimental evidence is needed along these lines to establish the true physiological action of the venous wall as a force in the circulation.