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Effect of Shock on the Small Blood Vessels of the Ear of the Rabbit.

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Peripheral vasoconstriction has been thought to be one of the cardinal manifestations of developing shock if not the initiating factor, whether shock be produced by hemorrhage[1] or by trauma.[2,3] It has been demonstrated[4] that stimulation of the sympathetic nervous system produces a train of effects strikingly similar to those associated with traumatic shock. Also it has been found[1] that during the production of shock by repeated hemorrhage the peripheral blood flow is more greatly reduced in the intact animal than in the sympathectomized animal. It has been our object in this study to observe directly the differences in the responses of the peripheral blood vessels when the nerve supply is intact and when it is interrupted. Since the transparent chamber inserted into the ear of the rabbit has been shown recently by us[5] and previously by others,[6,7] to be suitable for physiologic studies, we have employed this method.

Method. For this study we used healthy young albino rabbits weighing between 2 and 4 kg. The chambers were fully vascularized in each instance. Observations were made on 7 animals. In 4 of them the innervation of the ear was intact; in the remaining 3 the ear had been denervated as described previously.[5] Shock was produced by intestinal manipulation with the animal under the influence of pentobarbital sodium as has been done in dogs.[8] Observations of the vessels were made with a compound microscope, a magnification of ×200 being used. Photographs of the vessels were taken at half hour intervals coinciding with the periods of intestinal manipulation.

Results. The average length of time required for these animals to go into shock was about 3 hours. Death usually ensued within about an hour after the onset of definite manifestations of shock or after the systolic blood pressure had declined to about 60 mm of mercury.

In the innervated ear the earliest change noted in the behavior of the peripheral vessels was a spasmodic constriction of the arterioles. As this continued, the period of relaxation of the arterioles subsequent to constriction became less and less. As the period of onset of shock was approached there were phases during which the blood flow in the chamber was completely shut off. As this train of events proceeded, the quantity of blood flowing in the vessels became less. Within a few minutes prior to death the arteriolar walls became relaxed and blood flow ceased.

In the denervated ear the blood vessels behaved in exactly the same fashion with one exception. At no time during the experiments did we observe maximal constriction with complete cessation of the blood flow in the denervated ear. In this group of animals there was rather a gradual progressive constriction of the arterioles until the final relaxation just prior to death.

In both groups of animals the functional capillaries diminished in number in proportion to the degree of constriction of the arterioles. At no time was there evidence of capillary dilatation. A striking feature of the peripheral

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circulation was the diminution of the amount of blood in the venules as the experiment progressed.

Comment From these experiments it has been shown that constriction of the arterioles in the ear of the rabbit during the production of shock by trauma can occur in the denervated as well as in the innervated ear. The question immediately arises whether there is a release of some vasoconstrictor substance during the production of shock. By administration of doses of ergotoxine sufficient to block the vasoconstrictor action of epinephrine it has been shown[9] that there was not a reduction of blood volume such as occurred when epinephrine was given alone.[10] It has also been shown[11] that an animal whose sympathetic nervous system has been paralyzed by ergotamine tartrate closely resembles a sympathectomized animal. Since the blood vessels of one group of our animals did not possess a sympathetic nerve supply it is logical to assume that the vasoconstriction observed was owing to the liberation into the blood stream of some vasoconstrictor substance. Whether this was epinephrine or some other substance is not known. The kidney, adrenals and pancreas have recently[12] been eliminated as factors in this peripheral constriction. The possibility that the vasoconstriction was the result or accommodation of the vascular bed to a decreased blood volume was considered. This was thought an inadequate explanation, since the vasoconstriction occurred early and much before a great change of blood volume was considered probable.

An increased number of impulses from the nerve of a traumatized limb has been demonstrated by Slome and O'Shaugnessy,[13] who expressed the belief that this barrage of nerve impulses is of fundamental importance in an understanding of the causation of surgical shock. In our experiments reflex production of peripheral vasoconstriction, we believe, has been eliminated by the complete interruption of the nerve supply to the vessels observed. Of course the possibility of a release of sympathin through nervous barrage from the site of trauma still exists as well as the remote effects of local capillary damage.[14] Others[2] have stated that it is very doubtful if nervous impulses play as important a role as has been ascribed[13] to them.

The observation of temporary cessation of flow in the innervated ear, as contrasted with the steady but progressive narrowing of the vessels in the denervated ear, is interesting. The apparently constant, although diminished, flow in the denervated ear may lend some support to the observations which have been made that the peripheral flow is maintained better in sympathectomized animals than in those that have intact nervous systems during the production of shock.[1,11]

Summary. A study has been presented of the reactions of the small blood vessels of the ear of the rabbit during the production of shock by intestinal manipulation. This study was made on four animals whose ears were innervated and on three whose ears were denervated.

Conclusions. In the rabbit marked peripheral vasoconstriction occurs during the production of shock but it is not entirely dependent on an intact nerve supply. However, the nature of the peripheral vasoconstriction is somewhat altered by denervation.

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