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Nervous and pharmacodynamic control of the retinal blood flow.

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The literature on the vasomotor control of the retinal blood vessels, especially the arterioles, is conflicting. Several authors report that the nervous control of the size of the blood vessels of the retina is in the cervical sympathetic, as is the case in the vessels of many of the other organs of the head. In the rabbit there is agreement, apparently, that the cervical sympathetic controls the size of the retinal vessels. In the higher mammals, however, especially in the carnivora, while some authors report similar control, other investigators obtained negative results. From a practical standpoint as well as from a theoretical one this is an important question; for in man we not infrequently see embolism of the central artery of the retina; and we are constantly seeking further light on the management of these cases.

Our work so far has been done on dogs. We found that stimulation with the faradic current of the central end of the vagus-sympathetic trunk, as a rule, produced narrowing of the retinal arteries and veins. The examination was always made by means of the electric ophthalmoscope. We made attempts to isolate the sympathetic nerve by following the nerves to their ganglia. The stimulation of the isolated sympathetic produced the same results as stimulation of the vagus sympathetic trunk. Section of the cervical sympathetic is followed by slight dilatation of the retinal arteries. In some animals, however, we were unable to see any effects following stimulation or cutting of the cervical sympathetic.

Stimulation of the internal carotid plexus produced marked reduction in the size of the retinal arteries of the same side. Stimulation of the central end of the sciatic nerve produced constriction of the retinal vessels, provided the cervical sympathetic nerves were intact.

During the course of our investigation we were able to confirm

an older observation that compression of the trachea (asphyxia) produces dilatation of the retinal veins.

Inhalation of amyl nitrite from a pearl broken into the mouth produced dilatation of the retinal arteries; in one case intermittent dilatation and constriction of the arteries was noted.

Chloroform anesthesia produced no noticeable change in the blood stream in the retina. Nor could we see any changes following intravenous administration of strychnine. Adrenalin produced a noticeable narrowing of the blood vessels in some cases. We failed to obtain a visible dilatation of the arteries as noted by an earlier observer. Adrenalin administration was followed by the same result as stimulation of the cervical sympathetic.

Late in several experiments, when the blood pressure was low, a moderate pressure on the eye, as in closing the eyelids and opening them, produced blanching of the retinal vessels, with rapid return of flow.

Different drugs were injected into the common carotid, usually on the same side as the eye investigated. The solutions were frequently stained with methylene blue, and the stain observed in the fundus.

Two to three minims of chloroform injected into the carotid caused slight dilation of the retinal vessels. *Strong contractions* of the retinal vessels were produced by 5 min. chloroform, by 2 cc. ether, 1 cc. quinine-urea-hydrochloride, and 3 cc. 40 per cent dextrose.

Four cc. 40 per cent alcohol produced at first a slight dilatation of the retinal arteries; this was followed almost at once by very marked constriction lasting about 10 to 15 minutes.

Nitroglycerine, 1/50 gr. produced a slight dilatation of the retinal arteries, and constriction of the pupil.

A number of other fluids, like warm (40-60° C.) or cold (15-25° C.) water, or calcium chloride solution, did not seem to have any effect on the size of the vessels.