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Taxis in Increased Intra-Ocular Tension.

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TAXIS IN INCREASED INTRA-OCULAR TENSION.

BY S. O. RICHEY, M.D.

The principal arteries of the globe enter it posteriorly and pierce the sclerotic directly, limiting as far as possible resistance to the access of nutrient blood. On the other hand, the veins pass very obliquely through the sclerotic in channels varying from 1.5 mm. to 5.5 mm. in length and contain no valves distal to their junction with the ophthalmic vein. The intra-ocular vessels have no vaso-motor constrictors, and the tension of the eyeball depends upon the pressure in the extra-ocular arterial current[1]; after death it is lowered. Given an abnormal propulsive force in the extra-ocular arterial current from any cause, and the increased influx of arterial blood raises intra-ocular pressure. So long as this influx does not exceed the ability of the venous system to remove it, the increased tension is within the limits of safety; but should there be any slowing of the outflow of venous blood, that is, a loss of balance between the influx and the efflux of blood, "a vicious circle" is at once formed, which is more or less able to break itself,

[1] Resistance of the eyeball to pressure is equal to 26 mm. Hg., and pathologically may reach 70 mm. (Norris and Oliver, System of Diseases of the Eye, Vol. i, p. 125.)

N. von Kries found arterial pressure in the capillaries of the hand when raised to be equal to 24 mm. Hg.; when the hand hung down, = 54 mm. (Physiology, Landois and Stirling, 2d Ed., p. 151.)

depending upon the less or greater violence and persistence of the general vascular excitement.

Unfortunately, from this point of view, the anatomic course through the sclerotic of the veins favors obstruction to the outflow of venous blood, they for a limited part of their length being parallel with the sclerotic and within its meshes; so that *any* increase of pressure within the eye must narrow their lumen and diminish the outflow. This in turn adds to the existing intra-ocular pressure, farther reducing the caliber of the veins, and resulting in edema of the surrounding tissues, conveying a false impression that glaucoma is due to a disturbance of intra-ocular secretions.

Venous stasis in chronic glaucoma, if not relieved, results in thrombosis and connective tissue hyperplasia, in addition to other recognized changes. The difficulties and uncertainties arising in the history and management of chronic glaucoma have their immediate cause in thrombosis of the choroidal veins, complete relief from which depends upon the length of time it has lasted, and the more or less permanency of the changes within the vessels from the presence of coagula; for organized tissue may have formed there, with final abolition of the vessels, of which no mitigation is possible.

Previous to organization of the coagula and obliteration of the vein, during stasis and even when thrombosis is recent, the flow of venous blood is favored, or the coagula may be broken up, passed into the larger vessels and re-dissolved by pressure upon the external surface of the eyeball in imitation of the normal interrupted pressure upon it of the eyelids, of the extrinsic muscles and their tendons, and that produced by the

contractions of the iris, which here, as elsewhere, promotes the movement outward of the current within the veins, and is antagonistic to stasis. This proceeding[2] I suggested and described at the meeting in 1896 of the American Ophthalmological Society, under the name of *taxis*, because the movement of the fingers is the same as in reducing hernia. It is an imitation of a normal function adjusted to a condition of normal tension, and is necessary because this function of the lids and muscles requires aid in a state of high intra-ocular tension, which offers increased resistance to pressure. It should be cautiously practiced when the coats of the vessels are weak, to guard against intra-ocular hemorrhage, the risk of which is small, except when hemorrhage is imminent or has previously occurred. It will accomplish what may be achieved by operation, with the advantage that it may be repeated with the recurrence of venous stasis and increased tension, which is a chronic tendency in chronic glaucoma.

That a myotic stimulates the iris to the production of this kind of pressure; that a mydriatic favors an attack of glaucoma by paralysis of the iris, thus allowing an advance of the vitreous body and lens, and consequent increased intra-ocular engorgement and obstruction of the anterior channel of filtration; that iridectomy, the force of the extra-ocular circulation being lessened, acts by temporarily relieving pressure upon the surface of the veins, permitting them to disgorge themselves; that after iridectomy, malignant

[2] A paper on "Massage of the Eyeball as a Therapeutic Measure in Glaucoma," by Dr. G. M. Gould, to be read at the meeting of the AMERICAN MEDICAL ASSOCIATION next month, is announced.

glaucoma, intra-ocular hemorrhage and failure of the anterior chamber to reform are caused by the persistence of undue *vis a tergo* in the extra-ocular arterial system, I am convinced.

This paper is an appeal to my colleagues to transfer their attention in glaucoma from the anterior channels of filtration to the fundus; from the relative amounts of secretion and excretion of the fluids of the eye to the influence of the vascular system, local and general; from a possible vicious circle in the anterior to a more rational vicious circle in the posterior section of the eyeball; satisfied that only thus will they find an acceptable solution of the many perplexing phenomena which have been observed in glaucoma.