

its wall to have thickened, its endothelium displaying hyaline degeneration and proliferation, the adventitia disclosing hyperplasia of connective tissue.

In such cases thrombosis of the central retinal vein is followed by glaucoma, its main branch being obliterated.

Glaucoma forms in such cases by accumulation of pathological products of altered metabolism (carbonic acid, lactic acid, acetic acid, citric acid, etc.), the vitreous body swelling gradually by their influence. Under normal physiological conditions, hydration corresponds to 98.5 mm. of water, and under the influence of lactic acid augments to 166.0 mm.

X-ray irradiative treatment of haemorrhagic glaucoma is indicated.

SIGNIFICANCE OF VENOUS PULSATION OF THE EYEGROUND*

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PULSATION of the eyeground has been known since 1853 (van Tricht, Coccius), but the origin of this phenomenon is still much discussed. There can be no doubt as to the pathological nature of arterial pulsation. Under normal conditions diastolic pressure of the central retinal artery surpasses ocular tension, and in consequence, circulatory flow is not impaired, but if ocular tension increases, as for instance with glaucoma, and surpasses diastolic pressure of the central retinal artery, which diastolic pressure is about half of the value of that of the brachial, being about 30 mm. of mercury, the flow being obstructed, arterial pulsation starts. The same is the case if the ocular tension being normal, the diastolic pressure decreases considerably; then the normal ocular tension becomes relatively higher than the diastolic pressure of the central retinal artery, and pulsation ensues as in cases *e.g.* of insufficiency of the aorta, collapse or grave anaemia.

In opposition to the pathological nature of arterial pulsation of the eyeground, venous pulsation is regarded dogmatically as being physiological. Two theories prevail concerning its origin. According to one of them, spontaneous venous pulsation is caused

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by the wave of arterial pulsation transpiring through the capillaries, into the vein and thus inducing it to pulsate. The other theory, generally accepted, is represented by the final analysis of Serr (*Arch. f. Ophthalm.*, 1937, Vol. CXXXVII, p. 487). According to this view, the pressure of the central retinal vein is 1-2 mm. of mercury (0.039-0.078 inches) higher than the tension of the eye, a fact easily proved by very gently pressing a finger against the eye, and thereby causing an increase of pressure equal to 1-2 mm. of mercury. In this case the central retinal vein pulsates. But if the difference of 1-2 mm. of mercury between ocular tension and venous pressure is absent, an increased amount of blood enters the eye at every systole, ocular tension increases accordingly, and surpassing the venous pressure, causes the vein to pulsate, circulation being obstructed.

Consequently, the venous pulsation of the eyeground is chiefly dependent on ocular tension, evident also by the fact, that under the influence of mydriatics, the spontaneous venous pulsation increases. Baurmann reported (1925) observations according to which the pressure of the central retinal vein is furthermore dependent on intracranial pressure (i.c.p.), inasmuch as spontaneous venous pulsation disappears simultaneously with the increase of i.c.p. But he, as well as Kukan, report cases in which despite the augmentation of i.c.p., venous pulsation was present.

To elucidate this question has been the chief aim of the present investigation. As, according to my previous investigations, the blood-pressure of the eyeground is generally diminished in pregnant women, and according to Professor Imre and Stephen de Grósz, ocular tension is likewise decreased in them, my investigations started by examining them, and it was found out that in 38 per cent. of the women sent by the obstetrical department of our Polyclinic, spontaneous venous pulsation was apparent. The age of these women varied between 18-38 years. Blood-pressure of the individuals displaying venous pulsation varied between 95-150 mm. of mercury of systolic pressure, the average was 123 mm. (3.53-1.468 inches, average 4.84 inches) of mercury. Systolic pressure of that group, members whereof did not display venous pulsation, ranged between 110-118 mm., 130 mm. of mercury average (4.330-5.116 inches, average 7.081 inches). Material of the internal department examined, revealed that venous pulsation is more commonly present in individuals, who have a disposition to hypotonia, complaining about headache and giddiness. Consequently I came to the assumption that spontaneous venous pulsation of similar cases may be due to the circumstance, that in accordance with arterial hypotonia, venous pressure in general has diminished likewise. Venous pressure determined by Groak's method at the cubital vein of such persons,

determinations having been conducted in co-operation with George Reisner, M.D., rendered normal values between 5-11 c.c. of water (2-4½ inches) in everyone of the cases investigated, and ocular pressure was found to be likewise normal, rendering values between 20-26 mm. of mercury (0.786-1.021 inches).

Subsequently the i.c.p. was investigated. According to Baurmann and Hippel, as has been mentioned above, the pressure of the central retinal vein increases correspondingly to the increase of the i.c.p. A case is quoted proving this. Cysternal puncture was followed by collapse, and while it lasted, spontaneous venous pulsation became apparent, which had not been present previously, and disappeared again concomitantly with recovery. Another case displaying spontaneous venous pulsation got seriously unwell after a cysternal puncture.

Convincing proof for the dependence of the pressure of the central retinal vein and spontaneous venous pulsation of the fundus of the eye, on i.c.p. was rendered by a series of experiments, in the course of which persons displaying spontaneous venous pulsation were put into a prostrate position and their head bent backwards, thereby increasing cranial pressure, the spontaneous venous pulsation stopping instantly. The same was the case if Queckenstedt's sign was elicited by pressing the finger against the carotid, while the patient was sitting, i.c.p. being increased thereby also.

But spontaneous venous pulsation did not disappear despite prostration with another type of cases, suffering from Basedow's disease and hypertonia, cases where with the amplitude of the pulse, *i.e.*, the difference of systolic and diastolic pressure, was large. Venous pulsation in such cases did not develop with pressure of the vein decreasing in connection to the undulations of ocular pressure, concomitant with the revolutions of the heart, as Serr believes, but exclusively by transpiring of the wave of capillary pulse towards the vein.

According to my researches spontaneous venous pulsation of the eyeground therefore originates by two mechanisms. One of them consists of a transpiring of the arterial capillary pulse-wave at high blood-pressure, towards the vein. The other works by the i.c.p. being eventually diminished, the pressure within the central retinal vein being reduced to the level of ocular tension, and thus inducing pulsation. There can be of course no discussion about the validity of Pines' opinion published in the August number of the *Brit. Jl. Ophthalm.*, 1938, according to which local anatomical conditions, namely, an angular bend of the central retinal vein within the physiological excavation of the papilla influences pulsation, the less so, as in a given case the one, or other, or both eyes display venous pulsation.