

## THROMBOTIC CORTICAL AMAUROSIS \*

*Report of a Case of Bilateral Calcarine Softening*

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Unilateral destruction (1) of the visual cortex following thrombosis of branches of the posterior cerebral artery is not an unusual condition, but very few bilateral lesions have been reported; Wilbrand and Saenger (2) collected eighteen such cases from the literature.

The following case is considered significant not only because of the diagnostic features involved but also because it appears to controvert, in part at least, the precept of John Hunter (3) on the distribution of arteries. He held that they were distributed without relation to function.

*Case:* E. O. M. Long Island Hospital, Boston. Age, fifty-five. White. Admitted July 26, 1920. Occupation, paper hanger. Chief complaint, blind. Family history and past history essentially negative. The following statement accompanied the patient from another hospital: "Admitted because of poor vision and loss of memory. Had taken two drinks of whiskey, immediately after which he had severe gastroenteritis and his vision began to fail. Examination at that time was negative except for blindness and loss of memory. It was thought that the supposed whiskey contained wood alcohol. Ophthalmologists feel that there is no hope of his regaining his sight. Diagnosis: Toxic amblyopia."

Examination on admission July 26, 1920: Can see something move when hand is passed before eyes, but cannot recognize objects or count fingers. B. P. 220/120. Otherwise the examination is negative. Diagnosis: Toxic amblyopia.

November 15, 1920: Referred to Neurological Service. Patient is totally blind, fundi normal, neurological examination negative, spinal fluid normal. Opinion—Cause of blindness not determined.

February 28, 1921: Developed bronchopneumonia.

March 3, 1921: Died. Autopsy: Cause of death—bronchopneumonia.

Brain: The pial vessels are slightly tortuous and are evidently sclerosed, most profusely in the large and medium-sized branches which are flecked with prominent gray and yellow areas. The sclerosis is most marked in the Circle of Willis. Shrinkage of the tips and inferior surfaces of the occipital lobes is quite pronounced. (Figure 1.)

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The tissues are soft and there is very slight resistance to light pressure. On horizontal section of the occipital lobes the left presents a large "cyst of softening," extending from the cortex of

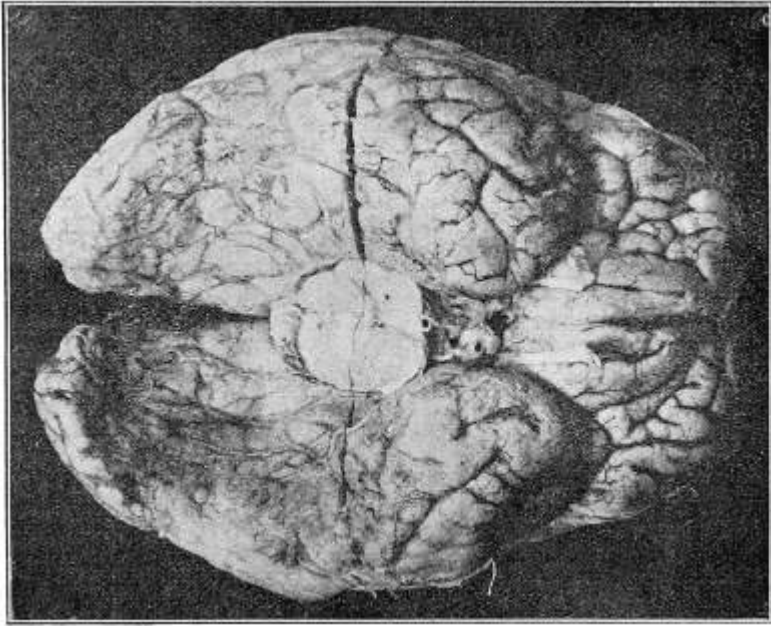


Fig. 1: Photograph of base of the brain with pia attached. The dark portions of the occipital lobes mark the areas of softening.



Fig. 2: Horizontal section of left occipital lobe showing the degree of "softening" in calcarine area and in the optic radiations.

the median surface inwards 1.5 cm., involving the area about the parieto-occipital fissure (Figure 2).

There are also signs of degeneration in the white matter in the optic radiation anterior to the parieto-occipital fissure. The right

occipital lobe presents a small "cyst of softening" between the parieto-occipital fissure and the occipital pole (Figure 3), with a partial destruction of the cortex about the calcarine fissure. There

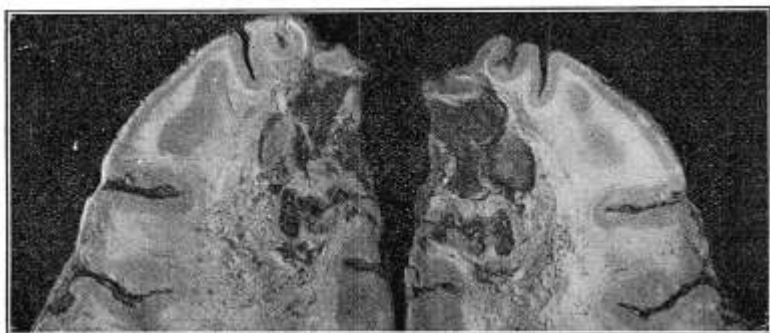


Fig. 3: Horizontal section of right occipital lobe showing the degree of "softening" in the cortex of the calcarine area and in the optic radiations.

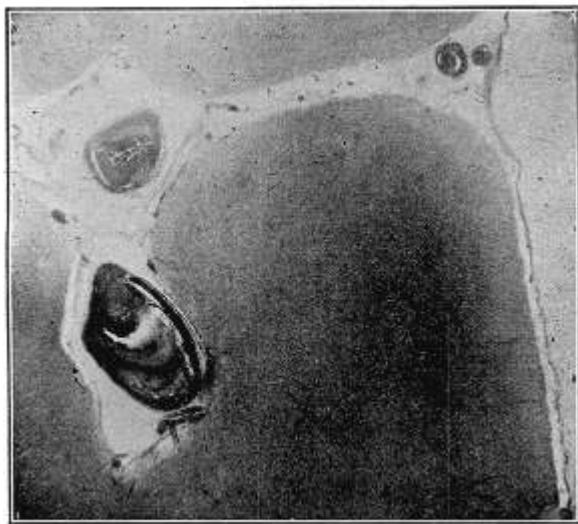


Fig. 4: Microphotograph at anterior border of section through "cyst of softening" in left occipital lobe. Here the degree of thickening and sclerosis of the elastica with narrowing of the lumen can be observed in the large artery.

is also a slight degeneration of the white matter in the optic radiation. Wherever sections are made in the occipital lobes the blood vessels stand out prominently and are characteristic of an advanced arteriosclerosis. Figure 4 is a microphotograph of a section of Figure 3

at the parieto-occipital fissure. It illustrates the thickening of media and the narrowing of the arterial lumen to such an extent that only a scant flow of blood can reach the parts supplied, resulting in degeneration of the calcarine area. The symmetrical position of the lesions makes the cause of blindness obvious. No lesions were encountered on examination of the optic tracts anterior to the occipital lobes.

Beyond the general arteriosclerosis, the brain, cerebellum, pons and medulla appeared normal.

*Conclusion:* This pathological picture proves the earlier diagnosis of toxic amblyopia to have been erroneous; furthermore it presents a condition which should be considered in all cases of amblyopia before making such vague diagnoses as "retrobulbar neuritis" and "toxic amblyopia" in the absence of fundus pathology.

The case tends to corroborate the views of Shellshear,(4) who believes that the arteries of the forebrain have a functional distribution and should not only be studied from the anatomical point of view, but in relation to their functional significance which is probably of paramount importance.

In the case here reported, one finds the complete loss of a single function from symmetrical lesions of the occipital arteries.

#### BIBLIOGRAPHY

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