

**Woltman, H. W.** BRAIN CHANGES AND PERNICIOUS ANEMIA. [Arch. Int. Med. Jour., 1918, No. 6, J. A. M. A.]

Woltman examined the brains of seven persons who died of primary idiopathic pernicious anemia. The most salient features in the pathologic anatomy of these brains, were the following: 1. Not only do degenerated areas of the Lichtheim type, such as are typically found in the posterior and lateral funiculi of the spinal cord in pernicious anemia patients, occur in the medullary portions of the brains of these cases, but they occur with about the same frequency, though their demonstration may be rendered more difficult. 2. Patients who show degenerative changes in the spinal cord at necropsy, usually show the same type of lesion in the brain also. 3. In addition to these focal degenerative areas found in the white matter, which may or may not be associated with blood vessels, one also finds a diffuse degeneration, which, though it is, as a rule, somewhat more striking in the long association tracts, also occurs in the short commissural fibers passing from one gyrus to another, thus rendering the view untenable that it is the distance of these fibers from their trophic centers which is instrumental in causing the degeneration. 4. The gray matter is by no means immune from the destructive process. This is usually focal in character, and begins around the pyramidal cells of the marginal gray layer, the cells themselves being ultimately destroyed in the process, this, in turn, giving rise to a secondary and very diffuse degeneration of the medullated fibers in the white matter. 5. Though some degeneration was noted in the fibers of the internal capsule and in the long tracts passing through the pons, the degeneration at this level was less intense than that seen either in the cord or in the brain. 6. The appearance of these plaques, not only around the blood vessels but also around some of the larger pyramidal cells, seems additional evidence that lymph stasis is an important factor in the production of these foci. 7. Well marked psychoses, such as are occasionally associated with pernicious anemia, probably have little or nothing to do with these destroyed areas. 8. The milder mental manifestations such as somnolence, apathy, and terminal delirium, are probably in a measure dependent on these lesions, though the chief causative agent of these symptoms is probably the toxin itself.

**Thompson, Herbert E.** A CASE OF BRAIN TUMOR SHOWING EXTENSIVE DESTRUCTION, WITH BUT FEW DIAGNOSTIC SYMPTOMS. [Boston Med. and Surg. Journ., 1917, CLXXVII, p. 592.]

Patient was a man of seventy, who three years ago began to wander away from home at times. Quite early in the course of his illness he had loss of sphincter control. There was gradually increasing physical weakness and mental confusion. He became more irritable, and memory failed. He lay in bed a great deal, and often rose at night to look for burglars. He is said to have had a few attacks in which he became

pale, rigid, and confused; after these he could sleep for a long time, but no definite convulsions were noticed. He became very untidy in dress. Answers to questions were mostly relevant, but incorrect. Marked dermatographia. Blood serum negative Wassermann. No definite paralysis or aphasia: no history of headache. Necropsy: a glioma of the brain, which had caused extensive cerebral destruction. It involved the anterior portion of the corpus callosum, white matter of left frontal lobe, caudate and lenticular nuclei, and the internal capsule. The right frontal lobe was only very slightly involved. [Leonard J. Kidd (London, England).]

**Mairet, A., Piéron, H.** HEADACHE AFTER SHELL CONCUSSION. [Par. Méd., July 6, 1918.]

The authors report headache and tenderness along the course of the trigeminal nerve after a shell concussion. On pressure pain is felt upon the emergence of the nerve, especially above the orbit, pain is felt which spreads along the great occipital nerve to the back of the head. They believe that minute suffusions of blood probably cause the irritation.

**Chalier, J.** CRANIAL TRAUMA AND GREYING OF THE HAIR. [Progrès Méd., 1918, 210.]

The record of a case of a soldier aged twenty-four, who gave no hereditary or personal antecedents of importance, but who seven months after the receipt of a wound of the left parietal region, not involving the bone, developed canities of the left eyebrow and of the hair of the scalp and beard on the side of lesion. The canities was accompanied by almost complete loss of sensibility of the left cornea and disappearance of the corneal reflex on this side.

**Bayliss, W. M.** GUM INJECTIONS IN "SHOCK." [Jour. Phys., Apr., 1918.]

Bayliss finds that a low blood pressure brought about by various factors, hemorrhage, muscle injury, temporary local anemia, slight hemorrhage with exposure to cold or plus acid injection, diminished oxygen supply, can be permanently restored by intravenous injection of 6 per cent. gum acacia in 0.9 per cent. sodium chlorid. If the bulbar centers have already been paralyzed by lack of oxygen or there has been section of the spinal cord in the cervical region this is unavailing. So also is transfusion, which does not in any case show any advantage over the gum, unless hemorrhage has caused the loss of more than half the blood total. The gum must be administered slowly. When a progressive delayed fall of pressure has been produced upon injections of acid, neutralization by an alkaline produces no effect. It is concluded therefore that the effect is due to some other changes and not to the acid as such. Gum injections prevents the development of shock symptoms