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Literature (Modern).

General.

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Ibid.: Various previous articles; also Gaz. médic., 1877, No. 4.
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Wilder: American Neurological Association, 1885. Reported in Journal of Mental and Nervous Diseases, July, 1885, pp. 348, 349.
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Duchaine, F.: Des ruptures de l'artère méningée moyenne, Paris, 1890, p. 115.
Adamkiewicz, A.: Die Arterien des verlängerten Markes vom Uebergang bis zur Brücke, Vienna, 1890. [Reprint.]
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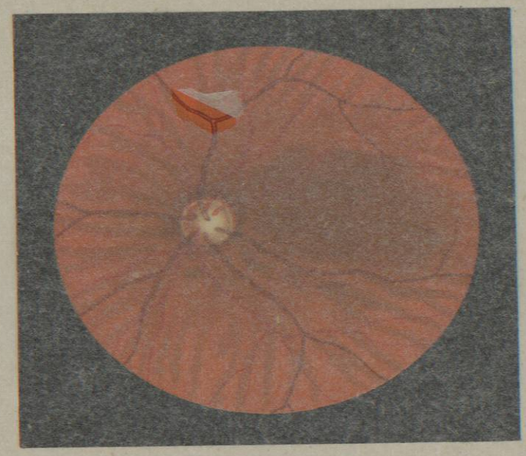

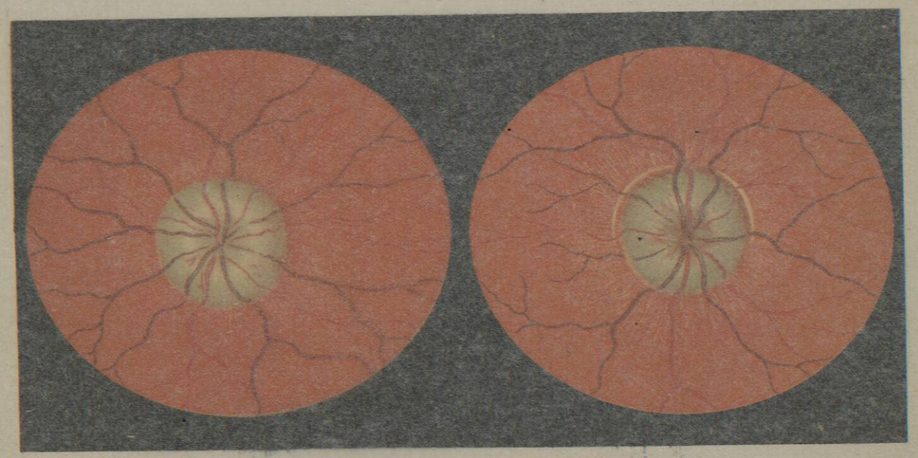

Representation (in the two lower pictures) of the condition known as "Choked Disk," as seen with the ophthalmoscope. For purposes of comparison, the appearance of the normal fundus oculi is shown in the upper picture.—*(From Haab—Atlas of Ophthalmoscopy.)*

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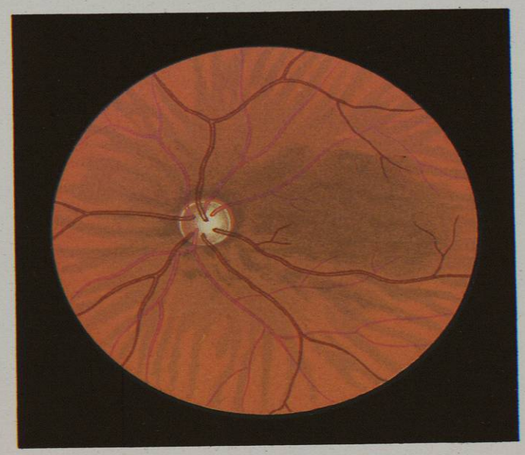

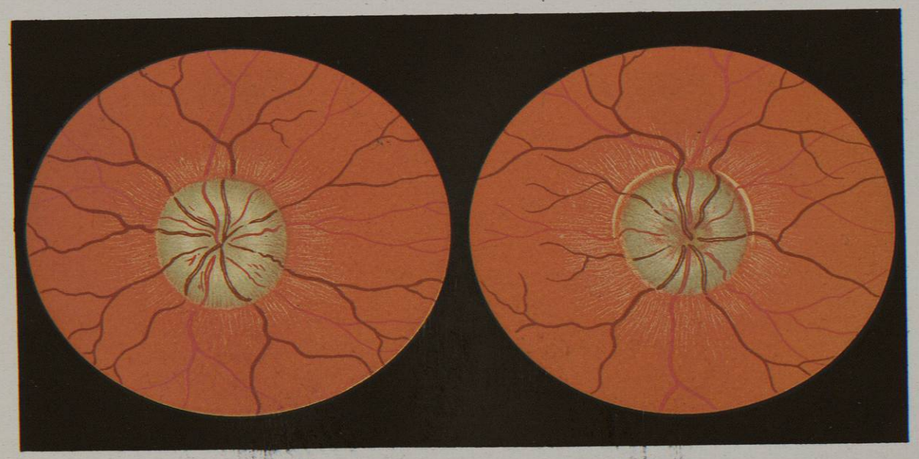

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Lymphatics.

Many articles by Arnold (1838), Hyrtl (1860), His (1865), Robin, Schwalbe, Obersteiner (1870), Riedel, Arndt, Krause, etc.
Key and Retzius: Studien in der Anat. des Nervensystems, etc., 1876.
Rossbach and Sehrwald: Uber die Lymphwege des Gehirns. Centbl. f. med. Wiss., 1888, Nos. 25 and 26.

BRAIN, COMPRESSION OF.—Notwithstanding the unhappy use of a word that does not describe the subtile condition it is meant to express, the term compression is a convenient one that seems justified by the necessities of analysis and study. It is generally used in a pathological sense to describe a complexity of symptoms arising from a particular state of the brain characterized mainly by disorder of the circulation within the cranium, and manifested by more or less derangement of the three great faculties of the nervous system.

As a principle used to account for the phenomena of brain disease, and as a complication that is often present after various lesions of the brain and its membranes, the special and differential diagnosis of compression is of grave importance, and includes a wide range of cerebral pathology. The prevailing opinions of writers on this subject are somewhat misleading, since compression, in its true sense of occupation of less space, is a condition of the brain which in most cases seems to be assumed rather than demonstrated to exist, and it is rare to find a case of compression in which there are not present the symptoms both of concussion and laceration of the brain.

Etiology.—The squeezing of its constituent parts or the diminution of volume that is supposed to interfere with the functions of the brain may be effected by pressure from without the encephalon, or by tension within its proper substance. The condition arises from a great variety of causes, most common of which are cerebral tumor, hemorrhage, and inflammatory foci. Hypertrophy of the brain, simple congestion, hydrocephalus, and effusion from various causes may result in compression. The effusion may take place quickly or slowly. Some experiments, however, point to the fact that the symptoms of compression are not to be attributed to augmentation of tension of the cephalo-rachidian liquid.

Other causes are foreign bodies, loose splinters, or larger portions of depressed bone, and, in fact, any injury of the head, whether simple contusion, scalp wound, or fracture of the skull. Whatever be the cause of compression, it should be borne in mind that the most characteristic symptoms are brought about by *sudden* action, for compression established slowly upon the brains of animals by the injection of a liquid causes no appreciable symptom, unless the quantity injected be unusually large; and it should be further remembered that, in the human subject, appreciable symptoms of compression are not always induced by such causes as sanguineous extravasation into the cranium and the cerebral ventricles, nor by the pressure of a foreign body, a tumor, or a fracture.

Symptoms.—Loss of consciousness and paralysis, which vary according to the seat and extent of the compression, are symptomatic of the condition, no matter whether the compression be sudden, like that following a wound or some mechanical injury of the skull and its contents, or whether it be slow, as that following enlargement of the brain from extravasation of blood, lymph, pus, serum, or tumors. Paralysis, excepting that of one or the other eye, is always on the side opposite to that which is the seat of injury. The symptoms may come on almost immediately after any injury of the brain that disorganizes its substance; but there are other cases in which compression takes place slowly and after a certain lapse of time. The initial symptoms that characterize this period are mainly subjective and those of congestion, as vertigo, headache, confusion of ideas, nausea, and, on rare occasions, vomiting. Then follows a lethargic sleep and more or less paralysis, and the patient cannot be aroused by any stimulus. The face is suffused and dusky, it wears an expression of well-pronounced stupor, and the eyelids are usually closed and immovable; respiration is slow, labored, and stertorous, something like the act of snoring, and the peculiar blowing movements of the lips in expiration have been compared to the act of smoking a pipe. Deglutition is impossible, and the tendon reflexes are abolished. As the intracranial trouble increases the pulse becomes slow and labored. It may be hard and frequent, or small and intermittent. Sometimes it is very irregular, and the symptoms may resemble those of anæmia of the brain and medulla, brought about by experimental means. Paralysis of the sphincters is generally present, with involuntary evacuations; or the patient may have torpidity of the bowels and obstinate constipation. Retention of urine, often present from paralysis of the bladder, is followed by incontinence as the result of overflow from distention. The skin may be cool or it may be hot and perspiring, and the temperature, though generally normal, may reach as high as 106° F. One or both of the pupils may be contracted or dilated, or they may rest immobile and unresponsive to the action of light. The symptoms of optic neuritis may also be present, and sometimes nystagmus is noted. Paralysis both of motion and of sensation, in one or both extremities, may exist in case the compression is exercised on the hemispheres, and convulsive movements and twitching of the limbs may occur on the paralyzed side or on the opposite side. Death occurs from arrest of respiration. (See *Coma* and *Asphyxia*.)

Diagnosis.—These pathognomonic symptoms, which are chiefly owing to effusion under the dura or to fracture of the inner table of the skull with resulting secondary anæmia, are not always met with, nor do such injuries as those inflicted by nails and arrow-heads driven into the skull, and even by missiles lodged in the brain, always produce the symptoms of compression. Sometimes the brain may be compressed without any disturbance of its functions. Pressure from an abscess, causing a hollow in the brain as large as a man's fist, has been known to cause no symptom of compression. It is, moreover, doubtful whether depressed fracture be a frequent cause of compression, since the injury is always complicated by laceration, the symptoms of which are often mistaken for those of compression; and it is often difficult and always embarrassing to determine, after an injury of the head, whether we have to deal with a contusion of the brain, a simple concussion, or the more problematical symptoms of cerebral compression. One state so often merges into the other that the attempt to establish a clear basis for a sure diagnosis of the respective conditions seems hopelessly confused and intricate. Roughly speaking, the most characteristic symptom of concussion is somnolence and intensity of the evil from the outset; contusion manifests itself by agitation, delirium, convulsions, contractions, and the delay of febrile symptoms; while the most salient symptom of compression is paralysis, except in the case of effusion of blood into the convexity of the hemispheres or into the ventricles, when contracture or tonic muscular spasm of extended duration is the more prominent phenomenon. Without being a sure sign, paralysis constitutes at least a valuable element in the diagnosis of a condition that has no single confirmative sign. If there be an unequivocal sign of cerebral compression in the majority of cases following injuries of the head, it is, perhaps, that furnished by obstruction of the venous circulation, in consequence of which the blood of the eye is not returned into the cavernous sinus, when we find with the ophthalmoscope papillary or peripapillary congestion, a general or partial serous infiltration of the papilla, and a strong dilatation with tortuosity of the retinal veins. (These appearances are shown in the accompanying colored plate.) In concussion the fundus of the eye retains its normal state; in compression there is always serous peripapillary infiltration, dilatation, tortuosity, and sometimes thrombosis of the veins of the retina. The intraocular changes herewith portrayed are not always pathognomonic, but they lend to the diagnosis an additional source of correctness. As a rule, when the eye-grounds show arrest of the retino-choroidal circulation, we have an indication of the arrest of cardiac circulation and of the nervous functions.