(Russian. Tumor in the Left Thalamus, with Loss of the Movements for Facial Expression in the Opposite Side of the Face.)
Eisenlohr. Deutsche Zeitschr. f. Nervenhk., 1893, iii, 4, 5.

*Crura Cerebri.*

Brissaud. Dégénerations secondaires dans le Pédoncule cérébral. Publications du Progrès méd., 1879.
Tomaszewski. Zur Pathologie des Grosshirnschenkels. Inaug.-Dissert., Breslau, 1881.
Schrader. Ein Grosshirnschenkelherd mit secundären Degenerationen der Pyramide und Haube. Inaug.-Dissert., Halle, 1884.
Leube. Deutsches Arch. f. klin. Med., 1887, xl, 2, p. 270.
Buschke. Zur Casuistik der Herderkrankungen des Hirnschenkels. Inaug.-Dissert., Berlin, 1892.
Bannister. Journ. of Nerv. and Ment. Diseases, 1890, xv, 9.

*Pons.*

Senator. Zur Diagnostik der Herderkrankungen in der Brücke und dem verlängerten Marke. Arch. f. Psych. u. Nervenhk., 1883, xiv, 3, pp. 643 *et seq.*
Bleuler. Zur Casuistik der Herderkrankungen der Brücke. Inaug.-Dissert., Leipzig, 1885.
Meyer. Beitrag zur Lehre der Degenerationen der Schleife. Arch. f. Psych. u. Nervenhk., 1886, xvii, 2, pp. 439 *et seq.*
Markowski. Arch. f. Psych. u. Nervenhk., 1891, xxiii, 2.
Delbanco. Beiträge zur Symptomatologie und Diagnostik der Geschwülste des Pons Varolii. Inaug.-Dissert., Berlin, 1891.
Porter. Brit. Med. Journ., April 18, 1891.
Brasch. Zur Casuistik der pontilen Herderkrankungen. Neurol. Centralbl., 1892, 2.
Diller (Pittsburg). Amer. Journ. of the Med. Sciences, November, 1892.
Moeli und Marinesco. Arch. f. Psych., 1892, xxiv, 3.
Kolisch. Wiener klin. Wochenschr., 1893, 14.
Starr. New York Med. Record, February 6, 1893, xliii.

*Cerebellum.*

Krauss. Ueber Tuberkelknoten des Cerebellum. Inaug.-Dissert., Berlin, 1888.
Bressler, Frank C. Abscess of the Cerebellum following Suppurative Otitis Media. Philadelphia Med. Times, August 1, 1888.
Gowers. Lancet, 1890, i, 18, p. 955. (Functions of the Cerebellum.)
Böhm. Ueber cerebellare Ataxie nebst einem casuistischen Beitrage zur Lehre von den Kleinhirngeschwülsten. Inaug.-Dissert., Strassburg, 1891.
Cramer. Beiträge zur pathologischen Anatomie und allgemeinen Pathologie. Jena, 1891, xi, 1. (Unilateral Atrophy of the Cerebellum.)
Luciani. Das Kleinhirn. Leipzig, Besold, 1893.

*Medulla Oblongata.*

Spitzka. Deutsche med. Wochenschr., 1887, 8, p. 157. (Focal Lesions at the Level of the Transition from the Pons to the Medulla Oblongata.)
Goldberg, Ludwig. Tumoren der Oblongata. Inaug.-Dissert., Jena, 1889.



## II. The Study of Cerebral Lesions with Reference to their Pathological Nature.

**Pathological Diagnosis.**—We have before pointed out that the question as to the nature of a brain disease is not only of interest to the physician, but of the greatest importance to the patient, as on this the prognosis as well as the mode of treatment turns. An error in the topical diagnosis may deserve the censure of scientific criticism, but does not necessarily entail damage to the patient. If, on the other hand, we mistake the nature of the lesion in a given case—if, for instance, a disease of the vessels is taken for a new growth, if the tuberculous or syphilitic nature of the affection is overlooked, or, again, a severe alcoholic intoxication is diagnosticated where in reality an apoplexy exists—when such errors have influenced the treatment, not only opportunities may be lost for the patient which may never present themselves again, but an unfavorable event of the disease may actually be brought about or at least precipitated. On these grounds we ought to be particularly careful and conscientious in forming this part of our diagnosis, and no symptom, however small it may seem, should be overlooked, as we never know but that it may later perhaps become of diagnostic value.

In looking over the several pathological processes which here concern us, we find that their number is comparatively limited. First of all, we shall devote our attention to diseases of the blood-vessels, which so frequently are the cause of cerebral lesions. We shall have to determine the nature of these diseases, and carefully distinguish the affections of the blood-vessels from the secondary changes produced by them. The clinical symptoms, the complaints of the patient, and the objective signs are a direct consequence of the latter only, and it is therefore not the disease of the blood-vessels which we have practically to deal with, but the changes in the brain substance which they entail. The clinical manifestations vary according to the seat of the diseased vessel and the portion of the brain supplied by it. The symptoms we shall describe in detail later; but first let us speak of the pathological nature of the diseases of the cerebral vessels.

### Affections of the Brain due to Disease of the Blood-vessels.

A. **Diseases of the Cerebral Vessels and their Consequences.**—The arteries of the brain are derived from the internal carotids and the basilar, which is formed by the two vertebrals.

The internal carotid gives off two terminal branches, the anterior cerebral (arter. corpor. callos.) and the middle cerebral (arter. foss. Sylv.). The basilar divides into the two posterior cerebrals (arter. profund. cerebri). These receive on each side a communicating branch from the internal carotid, the so-called posterior communicating artery, while the two anterior cerebrals are connected by an anterior communicating branch, so that a closed circle (or rather a heptagon, according to Hyrtl) of arteries is formed, known as the circle of Willis, an arrangement which is of the last importance for the distribution of the blood in the brain (cf. Fig. 68).

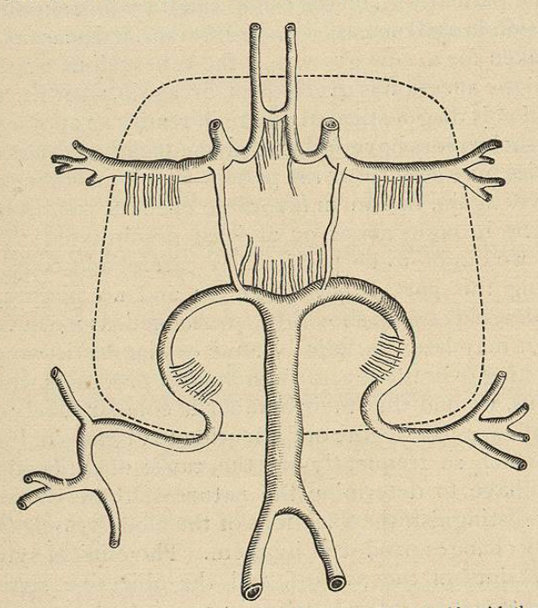

Fig. 68.—Diagram showing the Circle of Willis. The carotids with the anterior and middle cerebral arteries and the basilar with the posterior cerebrals are connected by communicating branches.

The fact that the left carotid comes off from the aorta nearly in a straight line with the blood-current in the arch, while the innominate, which gives off the right carotid, leaves the aorta almost at right angles, easily explains the greater frequency of embolism on the left side. A somewhat similar condition exists in the vertebrals, where the left, often the larger one, arises from the subclavian at its highest point. This is, however, of less moment for cerebral lesions, as the blood has first to pass the basilar before entering the brain substance.



Of the three before-mentioned arteries—the anterior, middle, and posterior cerebrals—each one supplies two sets of vessels totally distinct from each other—namely, first, the so-called cortical arteries; second, the arteries of the basal ganglia. The important difference between these two systems consists in the fact that the former, as Heubner and Duret have shown, possess anastomoses, while the latter are, as they have been called by Cohnheim, terminal arteries—that is, they do not communicate with each other, but pass directly into the capillaries. The significance of such an arrangement is apparent, and we shall not be surprised to find that occlusion of an artery of the second set almost always produces death of the parts supplied by it.

Of the three cerebral arteries, the middle, the Sylvian artery, has by far the widest distribution and is the most important; for while the anterior supplies the corpus callosum, the gyrus rectus, the paracentral lobule, and the præcuneus; the posterior, the crus, the temporal, and the occipital lobe, and the cuneus, sending also a few

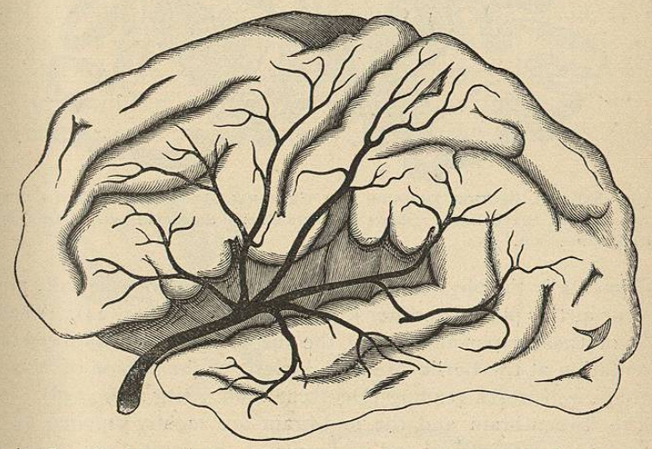

Fig. 69.—The Cortical Distribution of the Middle Cerebral Artery. (After Charcot.) From left to right the five branches are named as follows: The inferior frontal branch to Broca's convolution, the ascending frontal branch, the ascending parietal branch, the parieto-sphenoidal, and the sphenoidal branches.

branches to the optic thalamus (arter. optic. posterior.), it remains for the middle cerebral to supply the whole lenticular and the caudate nucleus, and, above all, the internal capsule. Moreover, the central and cortical motor region, the cortical areas concerned in the process of speech (on the left side), the cortical centre for hearing, probably also for vision, depend on this artery for their nutrition.

Its cortical distribution, its subdivision into the frontal, parietal, parieto-sphenoidal, and sphenoidal arteries, is made clear by Fig. 69.

Its distribution to the lenticular nucleus is illustrated in Fig. 70. The internal artery of the corpus striatum, also called the lenticular artery, goes to the first and second segment of the lenticular nucleus, while the external branches are the so-called lenticulo-striate and lenticulo-optic arteries. Among the former, the one which supplies the third segment of the lenticular nucleus, the upper portion of the internal capsule, and the caudate nucleus deserves special mention. It is so frequently the seat of hæmorrhage that Charcot has called it "*l'artère de l'hémorrhagie cérébrale.*" Mendel has attempted to show

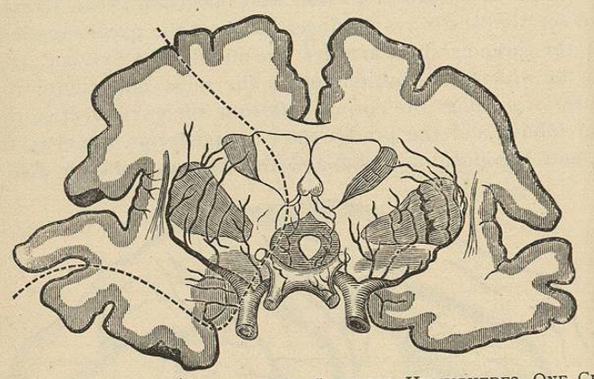

Fig. 70.—Frontal Section through the Cerebral Hemispheres, One Centimetre behind the Chiasm. Shows the distribution of the middle cerebral artery in the lenticular nucleus.

experimentally the physical reasons why ruptures are especially prone to occur at this place (Berliner klin. Wochenschr., 1891, 24). The account of these experiments and the discussion which followed their presentation at the Berlin Medical Society, in the session of May 27, 1897, are well worth reading (Deutsche Med.-Zeitg., 1891, 46).

The 'tween-brain and the mid-brain are mostly supplied by the posterior communicating and its branches, the cerebellum by several so-called cerebellar branches (arter. cerebell. super. et infer.) coming from the vertebrals; the pons and medulla oblongata also by branches of the vertebrals, which are the so-called rami ad pontem and rami ad medullam oblongatam.

The internal carotid and the basilar measure 4 mm. in diameter; the vertebrals, 3.5 mm. (Luschka). The blood pressure in the carotid is generally taken to correspond to from 140 to 160 mm. Hg. How guarded, however, we ought to be in accepting such statements has been shown by Loewenfeld, who drew attention to the variations in the development of the cerebral arteries; and it seems at least possible that this is of considerable ætiological significance for different cerebral affections.



LITERATURE.

Capelli e Brugia. Arch. di psichiatria, science penale, etc., 1886, Fasc. 1. (On the Action of Certain Drugs upon the Cerebral Circulation.)
Loewenfeld. Arch. f. Psych. u. Nervenkrankh., 1887, xviii, 5,
Geigel. Virchow's Arch., 1890, cxix, Heft 1.
Geigel. Die Mechanik der Blutversorgung des Hirns, Stuttgart, Enke, 1890.
Lewy. Die Regulirung der Blutbewegung im Hirn. Virchow's Arch., 1890, cxxii, 1, 2.
Mendel. Neurolog. Centralbl., 1891, 24.
Kolisko. Ibid., 1892, 16. (On the Blood Supply of the Brain.)
Grashey. Experimentelle Beiträge zur Lehre von der Blutcirculation in der Schädel-Rückgratshöhle. Festschrift, München, 1892.

### 1. *Cerebral Hæmorrhage, Hæmorrhagia Cerebri (Periarteriitis Cerebralis, Miliary Aneurisms of the Cerebral Arteries).*

Pathological Anatomy and Ætiology.

Of all cerebral affections, hæmorrhage, the result of the rupture of a vessel, is by far the most important and the most frequent. As we should expect, hæmorrhages of various kinds may be produced by traumatism (injury to the skull, with or without fracture). They may occur between the inner side of

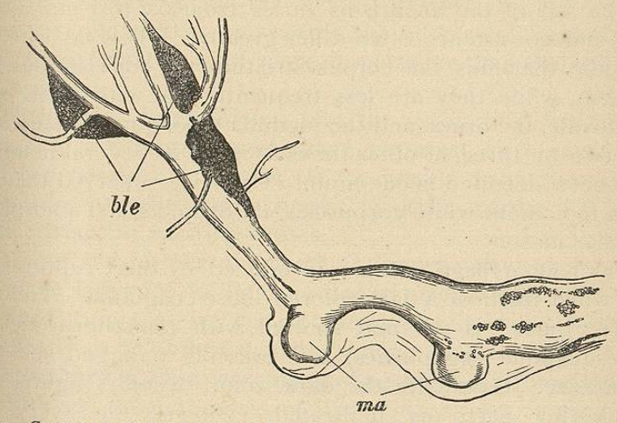


Fig. 71.—Cerebral Artery from an Apoplectic Focus. *ma*, miliary aneurism. *ble*, extravasations of blood into the adventitial lymph space. (After Cornil and Ranvier.)

the skull and the loosened dura mater, or in the sac of the dura or that of the pia (submeningeal hæmorrhage); but, disregarding these, there is one affection especially which gives rise to cerebral hæmorrhage—namely, a diffuse periarteriitis—which

was first described by Charcot and Bouchard in 1868. In this process a thickening of the lymph-sheaths and subsequent changes in the muscularis take place, by which the formation of miliary aneurisms is favored (cf. Figs. 71 and 72.) Rupture of these aneurisms then gives rise to hæmorrhages, and so frequently is this the case that the authors before mentioned found this condition in every one of seventy-seven consecutive cases which came under their observation.

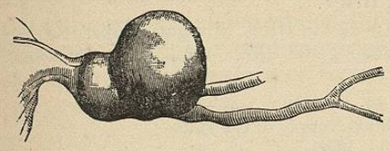

Fig. 72.—Miliary Aneurism of a Small Artery of the Lenticular Nucleus. (After Marchand.)

For the rupture of these aneurisms it is by no means always necessary to have any extraordinary exciting cause, such as an elevation of the blood pressure, which may temporarily be produced by bodily exertion, sneezing, coughing, vomiting, and the like, or which may permanently exist where the heart is hypertrophied, as in valvular disease or in cases of contracted kidney. In many of the instances nothing of the kind can be demonstrated.

The size of the aneurisms varies from 0.2 to 1 mm.; their color and consistence often differ greatly. Their favorite seat is in the thalamus, the corpus striatum, the convolutions, and the pons, while they are less frequently met with in the centrum ovale, the crura, and the medulla oblongata. Sometimes only two or three, at other times as many as several hundred, have been detected in one brain. On being squeezed they are found to contain white corpuscles, fat droplets, and amorphous granular masses.

The hæmorrhage which is produced by their rupture consists when fresh of a dark-colored loose coagulum. The wall of the "focus" is red and spotted with punctiform hæmorrhages (capillary apoplexies), and presents a ragged and torn appearance. Gradually the dark color becomes lighter, the neighboring parts are infiltrated, yellowish, and very soft (lemon-colored œdema). As a rule, the focus is later encapsuled by a layer of neuroglia, the fibrin masses become mixed with the *débris* of the nerve elements, and we get a smooth-walled cavity with liquid contents, the so-called apoplectic cyst, occupying a smaller space than the original hæmorrhage. If the walls approach each other before the



coagulum is transformed, a great increase of fibrillated connective tissue takes place and we get a so-called apoplectic scar.

The effect of the hæmorrhage varies according to its position, according to the calibre of the ruptured vessel, upon which depends its amount, and according to the rapidity or slowness with which the blood escapes. The favorite seat for hæmorrhages is in the large ganglia (Charcot, Andral, Rochoux); with decreasing frequency they are found (Nothnagel) in the remaining portions of the cerebral hemispheres, much more rarely in the pons and the cerebellum. The frequency is directly influenced by the size of the different cerebral vessels and by the blood pressure. The diameter of the vessels of the brain stem is considerably larger than that of those going to the cortex. The above-mentioned "artery of cerebral hæmorrhage" is of an especially large calibre (1½ mm.), and causes therefore when it bursts a particularly large and extensive hæmorrhage, because the bleeding is prolonged. "The traumatic effect of the hæmorrhage," as Wernicke calls it, is equal to the product of the mass of effused blood into the square of the rapidity with which it is poured out, which latter depends directly upon the blood pressure in the vessels. Hence it follows that, as regards the effect of a hæmorrhage, the blood pressure is of more importance than the calibre of the vessel.

**Ætiology.**—In examining into the ætiological factors concerned in a cerebral hæmorrhage, we must distinguish those which produce the disease of the vessels from those which directly cause the hæmorrhage; in other words, the predisposing from the exciting causes.

About the former not much is known; nevertheless, considerable influence in the causation of arterial disease must be ascribed to age, as we can not deny that it is decidedly less frequently to be observed in the young than in older persons, and that the smallest percentage of apoplexies is found between the fifth and thirtieth years of life. Still, to lay so very much stress upon the significance of age is not warranted by experience. The fact that cerebral hæmorrhage is by no means rare in people from twenty to thirty years old clearly shows that miliary aneurisms may occur even at a comparatively early period of life; nor are these cases by any means always those of persons laboring under hereditary disadvantages, since even

members of perfectly healthy families, while still young, may fall victims to a stroke of apoplexy. The influence of heredity as well as that of age has undoubtedly been overrated in this connection. It is true there are families in which apoplexy seems to be a natural occurrence, but such instances are exceptional, while on the other hand the arterial disease develops in an infinitely larger proportion of cases apparently without special hereditary cause. Sometimes the development of the disease seems to be favored by a peculiar "habitus" Thus, corpulent individuals of medium height, with short necks, broad thoraces, who on the least exertion or excitement become purple in the face, have usually been looked upon as particularly predisposed to apoplexy, and in many cases with justice; yet those who have in an extensive practice seen how often tall, spare individuals with narrow chests die from cerebral hæmorrhage, will readily give up the idea that an apoplectic habitus is a *conditio sine quâ non.*

The *rôle* which sex plays can not be denied. The disease is much more frequently observed in males than in females, while with embolism, as we shall see, the reverse is true. To explain this predisposition in males, other factors—namely, the mode of life—must, I think, be taken into account, and here it is, in the first place, the occupation, and, secondly, the abuse of alcohol, which must be considered. Notwithstanding the fact that we know very little about the influence of occupation on the formation of miliary aneurisms, our statistics of fatal cases of cerebral hæmorrhage in the different trades being somewhat unreliable, still we have some sure grounds, the correctness of which can scarcely be called in question. That, for instance, the working in certain poisons, especially in lead, predisposes to arterial disease, and consequently to apoplexy, is indisputable. In his thesis on encephalopathia and arthralgia saturnina, prepared under my auspices, Schulz (Breslau, 1885) points out the frequency of the so-called hemiplegia saturnina, and calls attention to the fact that Berger has made similar observations. In the second place, those who are exposed to radiating heat—workers at furnaces, puddlers—are in danger, especially if their work is connected with much bodily exertion, and this can hardly surprise us if we remember how much circulatory disturbances are favored by such circumstances. The same may be said of occupations which necessitate uncomfortable positions of the body, as, for instance, is the case in agate polishers,



who constantly have to lie on their abdomens, or in coal miners, who have to remain in a stooping position all the time.

In regard to the abuse of alcohol we refer not only to the confirmed drunkards, but much rather to that class of individuals who habitually consume more alcohol, especially beer, than is good for them. Such men rarely, if ever, get drunk, but they drink several times a day one or two glasses of beer, do not take enough exercise, and become fat and predisposed to fatty heart and arterial disease, especially arterio-sclerosis, which affection, we may say finally, is the real cause of the greater frequency with which apoplexy is met with in men than in women. The fatty heart may be present even without any marked obesity.

The important influence of syphilis in the origin of cerebral hæmorrhage is proved by many irrefutable observations, and, considering the part played by it in disease of the cerebral vessels, this can easily be explained. We shall mention it again in this chapter, and later dwell more particularly on the symptoms peculiar to the syphilitic hemiplegia. Exceptionally, hemiplegia occurs after diphtheria, sometimes in conjunction with a paralysis of the palate, sometimes independently. In a girl aged fifteen under my care, hemiplegia developed fourteen days after diphtheria without any simultaneous disturbance of consciousness, and only slight improvement was noticed after several years (cf. also Seifert, Neurolog. Centralblatt, 1893, 4). With equal rarity is this complicating sequela found after other acute diseases—for instance, scarlatina.

Sometimes no exciting cause can be demonstrated, but if such be observed, they are always associated with a sudden more or less marked increase of the blood pressure. People with diseased cerebral vessels are not rarely suddenly attacked by an apoplectic stroke after strong emotion, hard bodily exertion, during violent attacks of coughing, sometimes also in a cold bath and after a full meal. Christian (Arch. de Neurol., 1889, 53), and Bollinger in his monograph on late traumatic apoplexy (Festschrift für R. Virchow, 1891), have pointed out that traumatism may also lead to apoplexy.

How it comes about that the coldest months of the year yield the largest percentage of victims of apoplexy, and why it is that in the twenty-four hours there are two periods with a maximum and a minimum death-rate, if such be actually the case, can not be explained. Such has, however, been claimed