not necessarily disintegrate, as in hæmorrhage, but an equalization of the blood pressure can take place, which will cause the disappearance of all the symptoms.

Cerebral thrombosis rarely gives rise to a stroke, owing to the slowness with which the process takes place, and when an apoplectiform attack actually does occur, it must be due to the previous obstruction of other, neighboring, vessels. We had a considerable area dependent for its blood supply on a single vessel which before remained open, but has now gradually become so narrow that the pressure in it becomes too low to keep up the function (Wernicke).

The necrosis (softening, encephalomalacia) to which the obstruction of an artery, if lasting sufficiently long, is bound to give rise, manifests itself by certain focal symptoms, which may, just as in hæmorrhage, be divided into direct and indirect. Among the indirect the hemiplegia, often attended with hemianæsthesia, which closely resembles that described above, is the most important. Monoplegias also and hemianopia may set in without a definite stroke, and may be produced indirectly from the focus of softening, which lies in close proximity to the part the functions of which are interfered with. If an embolus obstruct an artery which can communicate by anastomoses with those of neighboring areas, and thus the damage can be compensated, we shall meet with transient focal symptoms (Wernicke), which at the most require eight days for complete recovery.

To determine the exact seat of the focus of softening, we must go to work with the same caution as in making a topical diagnosis of a cerebral hæmorrhage. Here, as there, we have to look for direct focal symptoms, and it is to these that most attention should be given in our examination; on the other hand, we must not forget that a focus of softening, even if it be of considerable extent, may pass through all its phases without a single symptom. No one region of the brain seems to be more exposed to softening than another. We found that the number of hæmorrhages at the base largely preponderated over those in the cortex; in embolism this is not the case. It is only because the surface covered by the cortex is much larger than that of the brain stem that we find in the latter numerically fewer cases of softening than in the cortex (Wernicke). The thalamus and pons are only rarely the seat of isolated softening, while hæmorrhages are found there much more frequently,

whereas the medulla oblongata is more commonly the seat of softening (cf. Berlin. klin. Wochenschr., 1891, 24). To diagnosticate hæmorrhage in the medulla oblongata during life is practically impossible, as in these cases death is almost instantaneous.

Prognosis.—The prognosis in embolism is, *cæteris paribus*, in general better than that of hæmorrhage. Not only is the outlook for complete recovery more favorable even if the attack has been severe and has lasted for a considerable time, but in most cases the danger to life is far less than in apoplexy.

Indirect action upon the medulla oblongata, in consequence of which the urine may contain albumin or sugar, is a rare occurrence. Even a softening of considerable extent may exist for a relatively long time without the manifestation of any grave general symptoms. Yet an unfavorable turn is not impossible, and this should always be feared if a sudden and marked elevation of temperature takes place.

Treatment.—The treatment is very limited; indeed, embolism as such, and the necrosis produced by it, are entirely out of its reach. It can only be directed against the attack or consist of the prophylactic measures by which we may hope to prevent the occurrence or repetition of the accident. The latter undoubtedly is the more important, and much can be accomplished by repeated local bleeding from the head (Laborde), a procedure which is also indicated in the treatment of the attack itself, as the cerebral circulation is possibly favorably influenced by it. That absolute rest is strongly indicated in cases where heart disease exists, needs hardly to be mentioned. Where there is a reasonable suspicion of syphilis, potassium iodide, 2.0 to 5.0 (grs. xxx–lxxv) *pro die*, ought to be exhibited.

Where there are multiple foci of softening the symptoms naturally depend on their seat. At the autopsy a number of such foci may be found which could not be diagnosticated during life because they were too small and were situated in so-called indifferent places. If several portions of the brain are affected, each of which gives rise to a focal symptom, there may be a complication of the most varied clinical manifestations.

Of great practical interest is the observation to which of late years attention has repeatedly been called, namely, that foci of softening may occur in that cerebral portion of the cortico-muscular tract which contains the fibres destined to

supply the muscles used in speaking and swallowing. These fibres pass from the lower third of the central convolutions, where the supposed centres for the hypoglossus and facial are situated, and end in the nuclear region of the medulla oblongata. Such foci have again and again been found, Sometimes they were bilateral and situated in the basal ganglia, especially the lenticular nucleus, sometimes on one side only—e. g., in the right corpus striatum—and it has been observed that they sometimes give rise to a complication of symptoms which simulate most closely those of Duchenne's bulbar paralysis. The fact, however, should be especially emphasized that the occurrence of such a focus on one side is sufficient by itself to produce all these symptoms (Lépine and Kirchhoff, cf. lit.).

The disturbances which go to make up the clinical picture are at times exclusively, always chiefly, referable to speech and deglutition. They resemble at first sight so much those of bulbar paralysis that the name pseudo-bulbar paralysis, or paralysis glosso-labio-pharyngea cerebralis, seems justifiable. Still, there are some points which should help us to avoid mistakes. Thus, while the beginning of the true bulbar paralysis is slow and gradual, the cerebral form often sets in quite suddenly with apoplectiform symptoms; in the pseudo-bulbar paralysis there is a manifestation of other cerebral disturbances which do not occur in Duchenne's disease. Again, the latter runs an uninterrupted progressive course, while in the cerebral paralysis long remissions are frequently met with. A certain asymmetry of the paralysis, which is especially noticeable in the orbicularis oris (Berger), favors the diagnosis of the cerebral as opposed to the bulbar affection. Far more important than all these points is the condition of the paralyzed muscles, which show no atrophy (Lereche, cf. lit.), and of the tongue, which also does not become atrophied in the pseudo-bulbar paralysis, and hence does not assume the appearance so eminently characteristic of the true bulbar form. Consequently there are no changes to be made out in the electrical excitability, whereas in Duchenne's disease reaction of degeneration is the rule. If, finally, we add that in the cerebral form the laryngeal muscles seem to be not at all or only slightly affected, we have sufficient data to solve the question of differential diagnosis in most cases satisfactorily (cf. the excellent article by Oppenheim and Siemerling).

The prognosis with regard to life is just as unfavorable in

the one as in the other form, only this should be borne in mind, that in the pseudo-bulbar paralysis remissions may occur; that we therefore can with a clear conscience give the patient good hopes of improvement. The duration of the disease may be much longer than is ever the case in the genuine bulbar paralysis.

The treatment is not so hopeless as in Duchenne's disease. The galvanic current intelligently applied, and careful galvanization of the brain and peripheral faradization of the paretic muscles, frequent excitation of the muscles of deglutition, as was described on page 149, all may be tried with the justifiable expectation of effecting at least a transient, sometimes indeed a quite gratifying, improvement.

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3. *Endarteriitis* (*Syphilitica*).

This process, first accurately described by Heubner in 1874, affects more especially the vessels at the base of the brain. The walls become opaque, show grayish translucent or whitish thickenings, and the vessels may finally be converted into firm, grayish-white cords. The new tissue which encroaches upon the lumen of the vessel either originates in the intima by an increase of the endothelial cells, which become transformed into connective tissue (Heubner), or is derived from the nutrient vessels of the media and adventitia, and consists therefore of emigrated cells (Baumgarten). On account of this tendency to thickening and consequent obliteration of the vessels, C. Friedländer has proposed for the process the name endarteriitis obliterans. While not denying that Heubner, who has studied the question most carefully, has arrived at important results, we must at the same time affirm that the arterial disease, which he describes as specific in nature, is not peculiar to syphilis, but that we find the same changes wherever we have a chronic inflammatory process with the formation of granulation tissue, as, for instance, as a consequence of alcoholism (C. Friedländer). This one fact remains of the greatest practical importance, that in the course of syphilis the cerebral arteries are very frequently diseased, and that as the outcome of this diseased state the most diverse cerebral symptoms may arise. Chorioretinitis, for example, has been observed by Oswald (Deutsche Med.-Ztg., 1888, 86). That under certain circumstances a hemianopia can be the result of such disease is proved by the interesting case reported by Treitel and Baumgarten (Virch. Arch., Bd. cxi, Heft 2, 1888), where, as a consequence of gummatous arteriitis obliterans of the arteria corporis callosi dextra, although the optic nerves were intact, a unilateral temporal hemianopia had developed. Furthermore, it is to be remembered that often enough an autochthonous thrombosis due to this arterial disease gives rise to an attack which can not be distinguished from the above-described true apoplectic stroke with consequent hemiplegia. If recovery takes place in these cases the same thing may be repeated several times, and it is especially in syphilitic diseases of the arteries that this is relatively frequent. The patient suffers from intense paroxysmal headaches, occasionally loses his consciousness, and presents a transient hemiplegia, but again recovers

fairly well, until finally he succumbs to a graver stroke. This, then, is the usual course which the disease takes. It can, of course, only be diagnosticated where the history of syphilis is clear.

The recognition may sometimes be difficult if other cerebral symptoms are present, such as speech disturbances, intention tremor, decrease in memory, and the like, when we are liable to think of multiple sclerosis, or progressive paralysis of the insane, and it may only be the amenability of the disease to specific treatment which will clear up all doubts. This consists in the use of bold doses of potassium iodide, 4.0–6.0 (℥j–℥jss.) a day in hot milk until sixteen ounces are taken, and an energetic course of inunctions—thirty to fifty inunctions of 2.0–2.5 (gr. xxx–xl) ung. hydrarg. It should be begun as soon as possible, as the patient is in no way injured by this procedure, while the benefit may be most conspicuous.

4. *Dilatation of the Arteries of the Brain.*

Aneurisms of the cerebral arteries may be of traumatic origin or, what is more common, may depend upon endarteriitis, and in this latter case syphilis again deserves special mention, as among fifty cases of brain syphilis there were found six instances with aneurisms (Heubner). Spillman reports fifteen cases in which following syphilis aneurisms of the basilar artery were found (Ann. de Dermat. et de Syph., 1886, vii, p. 641). Further, there is the embolic origin of aneurisms, which must not be forgotten (Ponfick).

Dilatations have been noted in the basilar artery, in the middle cerebral, and, though but rarely, in the vertebrals. Three cases of basilar aneurism have been reported by Nothnagel (Topische Diagnostik, p. 526); others by Watson (Lancet, October 13, 1888, p. 719). The symptoms presented nothing characteristic, but varied much, and even symptoms referable to the pons were not in all cases present. Vertebral aneurisms, as described by Cruveilhier, Lebert, and others, have occasionally been found to be attended with occipital neuralgia. Dilatation of the vertebrals produced by atheromatous degeneration may affect the surrounding parts and, as a consequence of structural changes produced in the neighborhood of the vagus, lead to attacks of twitching in the velum palati and to grave respiratory disturbances (Oppenheim and Siemerling).

Aneurisms of the ophthalmic or internal carotid in the

cavernous sinus may give rise to a pulsating exophthalmus, which can by appropriate manipulation be temporarily pressed back into the orbit. The pulsation of the eyeball, which may be propagated to the forehead and temple, is a source of great annoyance to the patient. In connection with multiple aneurisms, such as have been observed by Paulicki, for instance, existing simultaneously in the basilar, the anterior communicating, and the middle cerebral artery, epileptiform convulsions and psychoses have been noted. Definite pathognomonic signs do not, however, exist, and the diagnosis *intra vitam* is only exceptionally made with certainty. According to Gerhardt, there can at times be heard between the mastoid process and the thick cords of the muscles of the neck a murmur referable to the cerebral arteries; it is systolic or continuous, and is heard on one or both sides if the patient refrains from breathing or swallowing. Nevertheless, it is rather exceptional that a (small) aneurism of the cerebral arteries is diagnosticated correctly during life. In larger aneurisms, which produce characteristic focal symptoms, this will at times be easier, especially when ætiological data—e. g., traumatism—are present.

5. *The Neuroses of the Arteries of the Brain* (*Anæmia and Hyperæmia of the Brain*).

The vaso-motor nerves of the cerebral and meningeal arteries arise partly from the cervical sympathetic (Donders and Callenfels), partly from certain cranial nerves (Nothnagel). They may be excited or paralyzed idiopathically, or reflexly, especially from the stomach, and the resulting conditions, although as yet only imperfectly understood, are of great practical importance. Both stimulation and paralysis are, of course, usually only temporary, while in the intervals and in the normal state the vaso-motor nerves as well as their centres are in a state of moderate tonus. If the stimulation should from any cause be more than is necessary to maintain this normal tonus, a spasmodic contraction of the smaller arteries takes place, the absolute amount of blood in the brain becomes diminished, the patient gets pale, complains of dizziness, and loses consciousness—in other words, "faints" (acute nervous cerebral anæmia). At the same time the heart's action is weakened, the pulse is small, the face and body are covered with cold perspiration, and if this irritation is frequently repeated a

certain predisposition to slight changes in the blood pressure becomes gradually established, a condition of things which is favored by the mobility of the cerebro-spinal fluid. The attacks now occur on the slightest provocation, and in the intervals between them the patient complains of dull headache, vertigo, etc., the face at the same time usually being of a pale, wax-like color. Certain general diseases, especially chlorosis and pernicious anæmia, greatly predispose to these paroxysmal vascular spasms; in fact, cerebral anæmia is not infrequently one of the symptoms of general anæmia, as it is observed, for instance, after frequent and profuse bleeding from hæmorrhoids.

Among the ætiological factors, certain occupations play an important *rôle*. Working in lead especially may give rise to a chronic vascular spasm, and thus to a cerebral anæmia, which is associated with almost constant headache (encephalopathia saturnina).

Tanquerel des Planches, the best modern authority on saturnine affections, has described this condition, and it has again and again been made the subject of the most careful inquiries. It would be beyond the scope of our present work to speak of these in detail; those interested in the subject will find references at the end of the chapter; suffice it only to say here that this saturnine anæmia, if the obnoxious action of the metal is continued and the disease is once established, may produce in the workers severe cerebral attacks, epileptiform convulsions, and the like.

The treatment of acute cerebral anæmia consists primarily in placing the patient in an appropriate position—that is, with the head low or at about the same level as the feet, so as to aid the blood flow to the brain; the use of stimulants (wine, brandy, coffee), occasionally a subcutaneous injection of ether, may be indicated. Those who are familiar with the procedure may inflate the Eustachian tubes, as Kessel recommends; this "air-douche" is said to be an excellent method of producing rapidly an increased flow of blood to the anæmic brain (Laker, Wien. med. Presse, 1891, 25).

For chronic cerebral anæmia galvanization of the brain or of the cerebral sympathetic may be tried. As a matter of course, attention must also be paid to a possible primary cause, and every pernicious ætiological factor removed (change of occupation, etc.).

The opposite condition, a paralysis of the vaso-motor nerves, produces a dilatation of the cerebral vessels, and thus an immediate overfilling of the same. This can be demonstrated by ophthalmoscopic examination. Often, but not always, the vessels of the face share in the disturbance; the countenance of the patient assumes a purplish-red color, he complains of throbbing in his temporals and carotids, of headache, of buzzing in the ears (acute nervous hyperæmia)—in general, of about the same symptoms as we have described in the vascular spasm, the only difference lying in the color of the face. It is observed in certain individuals regularly after the use of quite moderate quantities of alcoholic beverages (wine, beer), or, just as the anæmia, after emotions, strong bodily or mental exertions, too much study, etc.; the abuse of tobacco may also give rise to it.

On account of the very varied manifestations of the affection different forms of cerebral hyperæmia have been distinguished (Andral, Eichhorst). Thus, a cephalalgic, a psychical, a convulsive, and an apoplectic form have been described, according as either headache or psychical excitement, with insomnia or epileptiform attacks or periods of unconsciousness (which latter are not rarely followed by cerebral hemorrhage), are the most prominent symptoms. The transition between these "forms" is, however, so gradual, and so seldom are they sharply defined, that for practical purposes it does not seem worth while to make the distinction. We have repeatedly observed marked contraction of the pupils, while in anæmia they are more frequently dilated and react sluggishly. As we have pointed out above, simple cerebral hyperæmia may produce hemiplegia, which can easily be confounded with the apoplectic form (pseudo-apoplexy).

The treatment is rather unsatisfactory; it is true we may in acute attacks of cerebral hyperæmia give early relief to a patient by placing him in an appropriate—that is, nearly sitting—posture, by applying ice-bags to his head, or, finally, by free venesection; but these attacks are so frequently repeated in individuals predisposed to them that the question of such treatment is not of so much importance as of the adoption for months and years of a careful dietetic *régime*. Besides keeping the bowels well open—a thing which should never be omitted—the patient must be advised to take enough exercise, even practice gymnastics; he should be cautioned against indulgence in heavy, indigestible foods, and, above all, in alcoholic beverages. A yearly visit to places like Marienbad, followed by a stay in a pure mountain air, moderate but daily excursions on foot, the occasional use of Carlsbad water under the direction of the physician—all these may be prescribed with advantage. Much caution should, however, be used with the so-called cold-water treatment, which, like sea-baths, may only increase the hyperæmia. This applies equally to the massage treatment, which, unless carried out in accordance with certain indications and fixed rules, and under the supervision of a competent medical man, often is productive of more harm than good in this disease.

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B. **Diseases of the Cerebral Veins and Sinuses.**—The blood from the brain and meninges is carried back toward the heart by the internal jugular vein. This vessel emerges from the jugular foramen and after its junction with the external jugular becomes the common jugular, which, after it has in turn received the subclavian, is called the innominate vein. The two innominates together form the superior vena cava.

Between the two layers of the dura mater there exist spaces which convey venous blood but are without valves. These are called sinuses. The veins of the cortex empty themselves into the longitudinal sinus (sin. falc.f. maj.), which terminates behind in the