In slight cases the disturbance is confined to the extremities and concerns more the sensibility to touch than the sensibility to pain. The patient feels the prick of a pin, but is unable to direct his fingers properly if the eyes are closed; he makes mistakes in recognizing objects which are given him to feel; he is unable to fasten small buttons, etc. Changes in the muscular sense also may exist for a considerable time, the patient being unable with his eyes closed to give any information about the position into which his hand has been brought.

In examining sensation in hemiplegics, Oppenheim (cf. lit.) has noticed that at times bilateral impressions are appreciated only on one side; that, for instance, if a patient is pricked simultaneously in the right and left thigh, he only perceives one prick—namely, that on the well side.

One of the rarest of sensory disturbances is the persistent hyperæsthesia of the paralyzed side, described by M. H. Fischer (Arch. de phys. norm. et path., February 15, 1887, ix, p. 185).

The psychical condition is not always the same. In certain cases the patients seem to have regained all their former faculties satisfactorily, so that a careful examination brings to light nothing more than a slight loss of will power and of the capacity for grasping ideas; but in other instances the patient becomes mentally weaker and at the same time irritable. He is easily made to cry and is liable to sudden changes of temper. Such patients are, however, notwithstanding their apparent obstinacy, very manageable and easily guided. Again, there are cases in which the mental weakness becomes very apparent. The patient forgets the commonest things, the number and the names of his children, confuses things and places, does not know what day of the week and what season of the year it is, etc.; at the same time he may have different delusions and hallucinations. Some cases finally go on to complete dementia, which takes a course not unlike that of general paralysis. Legrand du Saulle has published an interesting study of such disturbances among the apoplectics of the Salpêtrière (Gaz. des hôp., 68–71, 1881).

In the further course of severe hemiplegias where regeneration is impossible to any great extent, motor disturbances which we have designated as posthemiplegic (cf. lit. under treatise of Greidenberg) may follow. One of these is the so-called hemichorea, consisting of involuntary irregular move-

ments in the paralyzed limbs, which become aggravated by every mental emotion and voluntary movement and which entirely cease during sleep. These movements, which are best studied on the upper extremity, occur more frequently after cerebral infantile hemiplegia than in any other affection. The "hemiataxia" described by Grasset (cf. lit.) is closely related to hemichorea, and ought to be regarded as a variety of it. According to Charcot, the seat of the lesion in these cases is in the posterior portion of the internal capsule, the posterior part of the optic thalamus, and in the foot of the corona radiata. The so-called hemiathetosis will be considered in the chapter on the cerebral palsies of children.

A second class of motor disorders is made up of those peculiar involuntary movements which have been described as "associated movements." They are observed in the paralyzed extremity when the patient moves the corresponding, unaffected, one; thus, for instance, if a patient uses his right, well arm, the paralyzed arm makes similar movements, of course being restricted to a lesser or greater extent by any contractures which may be present. These movements have nothing in common with the so-called reflex movements which are found to occur in the paralyzed limb on stimulation of the sound one by the prick of a pin, the faradic current, etc. A peculiar instance of "associated movements" in an old hemiplegia I had the opportunity of observing for months. It was as follows: Every time the patient yawned the left arm was raised involuntarily at the shoulder-joint, and was kept up while the yawning continued; as soon as it ceased the arm dropped down helplessly. Sometimes one sees the sound limbs make involuntary movements if the patient attempts to use the affected ones, and again and again I have seen patients, straining to bend the paralyzed leg, become greatly astonished at the flexion which took place in the well leg without any such intention on their part. That in intended movements of certain muscle groups the antagonists begin to make involuntary movements—that, for instance, if an extension of the flexed fingers be attempted, the flexion at first becomes more forcible before extension begins (Hitzig)—is, according to our experience, very exceptional.

There are other associated movements which occur in the paralyzed half of the face when the sound side is moved; thus, for instance, in laughing, the muscles of the paralyzed side are

seen to contract equally, or even more strongly, than those of the well side.

Various theories have been proposed to explain associated movements (Westphal, Benedikt, Broadbent, Ross), but none of them can be taken as entirely explaining the facts. It is by no means impossible that all such motor disturbances are reflex in nature (Charcot and Brissaud; cf. also Senator, Ueber Mit- und Ersatzbewegungen bei Gelähmten, Berliner, klin. Wochenschr., 1892, 1).

As a third posthemiplegic phenomenon we have the tremor. It is not rare, and that form especially which occurs on voluntary movements of the affected side is rather frequently met with; on the other hand, we shall very rarely have the opportunity of observing this tremor while the extremities are at perfect rest. Relatively, the largest number of cases who presented tremor, in my experience, showed sensory changes, which consisted of paroxysms of pain in the affected extremities. On a cursory examination this tremor may be mistaken for unilateral paralysis agitans (hemiparalysis agitans), especially as the number of oscillations is about the same in both affections, $4\frac{3}{4}$ to $5\frac{1}{4}$ in a second. Pronounced intentional tremor, which we look upon as a pathognomonic symptom in multiple sclerosis, I never have observed in hemiplegia. Probably the cause of this posthemiplegic tremor has to be sought in the general increase of reflex activity, which, as we may remark here by the way, is observed besides only in a very few cases of tremor of a different nature. Here it seems to play the most important *rôle*.

Of great interest, as well as, at times, of no small practical importance, is the fact that in cases of incurable hemiplegia the non-paralyzed side, that is, the apparently well extremities, undergo certain changes which we are compelled to regard as pathological. Thus, Pitres has found that the well arm loses somewhat in strength, and that this is often more marked in the beginning of the hemiplegia than later on. On an average the loss amounted to about 38 or 40 per cent, while no increase in the tendon reflexes could be demonstrated at the same time. The well leg becomes weaker, and indeed in a more marked degree than the arm, the strength being reduced in some cases even by one half. The patient, though able to move that leg with perfect ease while in bed, finds it almost useless to attempt to stand or walk. Pitres was also the first

to notice that the patellar reflex of the sound side, as well, is abnormally active, an observation which is daily confirmed. The presence of the ankle clonus is noted by Westphal and Dejerine. All authors, however (Hallopeau, Brissaud, Féré), agree that it is extremely unusual to find the later contractures on the non-paralyzed side. On the whole, these changes, which occur on the so-called unaffected side, are more marked and of greater significance to the patient than we should be led to suppose from a superficial examination.

LITERATURE.

Maeltzer. Ueber Pseudo-Apoplexien im Gehirn. Inaug.-Dissert., Breslau, 1881.
Loewenfeld. Studien über Aetiologie und Pathogenese der spontanen Hirnblutungen. Wiesbaden, 1886.
Hochhaus. Berliner klin. Wochenschr., 1887, 1. (Meningitic Hemiplegia.)
Loewenfeld. Zur Lehre von den Miliaraneurismen des Hirns. Wiener med. Wochenschr., 1887, 47.

Greidenberg. Arch. f. Psych., 1886, xvii, 1. (Post-hemiplegic Motor Disturbances.)
Stephan. Revue de méd., 1887, 3. (Pre- and Post-hemiplegic Tremor.)
Brissaud et Marie. Progrès méd., 1887, 5, 7. (The Condition of the Facial in Hysterical Hemiplegia.)
Wernicke. Berliner klin. Wochenschr., 1889, 45.
Schaffer. Beitrag zur Lehre von der secundären und multiplen Degeneration. Virch. Arch., 1890, cxxii, 1, 2.
Scheiber. Arch. f. Psych., 1890, xxii, 1, p. 221. ("Athetosis spastica.")
Erben. Versuch einer Erklärung für das verschiedene Verhalten der Sehnenreflexe nach Hirnblutungen. Wiener med. Wochenschrift, 1890, 45.
Strauss. Zweihundert und fünfzig Fälle von rechts- und links-seitiger Hemiplegie; ein Beitrag zur Frage nach der functionellen Verschiedenheit beider Hemisphären. Inaug.-Dissert., Berlin, 1890.
Vizioli. Gaz. degli ospedali, 1892.

Trophic vaso-motor changes are not uncommon in the paralyzed limbs. While in the beginning of a hemiplegia the skin of the affected side is warmer and redder than that of the well side, it becomes cooler as the disease progresses, and frequently assumes a somewhat cyanotic color. The œdema often seen in the affected extremities is due to the absence of muscular movement and the consequent slowing of the blood and lymph current. In a patient who, two years before, had a pretty severe apoplectic attack with persistent speech disturbance, I have repeatedly observed slight repetitions of the hæmorrhage, during which the speech, which had consider-

ably improved, again became entirely unintelligible. Simultaneously there was developed on each such occasion over the whole body, and not merely over the paralyzed right side, an urticarial rash which persisted as long as the cerebral symptoms lasted. No doubt this was due to a disturbance in the vaso-motor innervation of the vessels of the skin, which reappeared with the transient increase in the intracranial pressure. Charcot describes an acute malignant bed-sore which appears two or three days after the onset of the hemiplegia in the gluteal region, beginning as a red spot and developing in a few days into a brown, dry eschar six to seven centimetres broad. It always ends fatally, and is, according to Charcot, a purely trophic disturbance, an alteration in the tissue, which we can attribute only to nervous influences.

The nutrition of the muscles which for years have been paralyzed usually suffers but little. We can easily understand that a slight degree of atrophy, due to inactivity, occasionally manifests itself, yet the excitability to both electrical currents remains normal. Only in exceptional cases is there pronounced muscular atrophy in the affected limbs when these, although their motion is impaired, can still be used to a certain extent. In such cases the atrophy can not be referred to inactivity, but we must rather assume a lesion in the trophic centres of the cortex, the seat of which is, however, still unknown. Since these conditions have received considerable attention of late, we add here some references.

LITERATURE.

Quincke. Deutsches Arch. f. klin. Med., 1888, xlii, 5.

Borgherini. Ueber die frühzeitige Muskelatrophie bei der cerebralen Lähmung. Deutsches Arch. f. klin. Med., November, 1889, xlv, 5, 6. (Atrophy on the Third Day after Hemiplegia; the lesion was thought to be in the thalamus.)

Eisenlohr. Muskelatrophie und electrische Erregbarkeitsveränderungen bei Hirnherden. Neur. Centralbl., 1890, 1.

Darkschewitsch. Zwei Fälle von frühzeitiger Muskelatrophie bei einem Hemiplegiker. Neurol. Centralbl., 1891, 20. (D. regards it as a cerebral amyotrophy.)

Mouratoff. Arch. de Neur., 1891, lxiii, p. 461.

Steiner. Ueber die Muskelatrophie bei der cerebralen Hemiplegie. Deutsche Zeitschr. f. Nervenhk., 1893, iii, 4, 5.

The simultaneous appearance of a hæmorrhage in each hemisphere is exceptional. It needs hardly to be stated that such an accident must necessarily give rise to the gravest symptoms: bilateral hemiplegia—that is, paralysis of all four extremities—bilateral facial and hypoglossal paralysis, amaurosis and total anæsthesia.

Diagnosis.—The diagnosis of cerebral hæmorrhage may give rise to considerable difficulties. It is easy only when a suddenly or gradually developing unconsciousness is followed by a paralysis or paresis of one side in a patient not suffering from any valvular disease of the heart. Under such circumstances the case is absolutely clear, and even the most cautious diagnostician, if he can exclude hysteria, may safely assume a cerebral hæmorrhage with consequent hemiplegia.

It is a different matter where we have to make a diagnosis at a time when we are unable to ascertain the presence or extent of the paralysis, but where we are restricted to an interpretation of the unconsciousness of the patient. Under these circumstances we have to be familiar with the conditions which, besides cerebral hæmorrhage, are capable of giving rise to unconsciousness, and be acquainted with the characteristic manifestations which each offers.

In the first place we may have to deal with a simple fainting fit. The concomitant symptoms—the wax-like pallor of the face, the small, frequent pulse, the cold sweat which covers face and body—are not likely to allow us to mistake the condition for one of apoplexy, especially as the gravest symptom—the loss of consciousness—as a rule, is not of long duration, but vanishes rapidly if the patient is laid down with the head low, the face sprinkled with cold water, or if ammonia or eau de Cologne, etc., be held to the nose. The success or non-success of these measures will help us to settle the differential diagnosis in a few minutes.

Secondly, we may have before us an epileptiform attack without convulsions or the coma which so often follows epileptic fits. Here the loss of consciousness is also complete, and the diagnosis can only be made if we can obtain a history of previous epileptic convulsions, or if we are able to assume this from scars on the tongue. In the absence of such evidence the color of the face may sometimes be of value to us; in some epileptics this is very pale, in cerebral hæmorrhage of a purplish color, yet this rule by no means always holds good, and should therefore be accepted *cum grano salis*.

The unconsciousness so often occurring in the course of a meningitis may be recognized from the temperature and the pulse, the peculiar drawing in of the abdomen (scaphoid abdomen), the jactitations, the rigidity of the neck, and possibly from the existence of choked disks. The possibility of an internal pachymeningitis hæmorrhagica must be thought of when the development of the condition has been characterized by sudden exacerbations and remissions, and when at the same time a history of alcoholism can be obtained.

In the beginning and in the course of progressive paralysis of the insane (dementia paralytica) apoplectiform attacks occur which resemble those produced by cerebral hæmorrhage very closely indeed, and which can be recognized as belonging to the former disease only from the previous history of the patient (and later from the results of the autopsy). If we can get no information from the history the differential diagnosis is impossible.

Intoxication with chloroform and alcohol may be attended by complete loss of consciousness. An individual in the unconsciousness of alcoholic intoxication is just as hard to arouse as one in apoplectic or epileptic coma, and the diagnosis may present some difficulties under certain circumstances—when, for instance, nothing can be learned about the cause, or what has immediately preceded the loss of consciousness. Usually, however, it is easy enough. Sometimes the smell of the ingested substance puts us on the right track, sometimes prompt reaction to energetic stimuli applied to the skin may make our diagnosis clear. As long as we are not sure of our ground, we ought to abstain from all therapeutic measures. Of opium or of morphine poisoning we need only think when the pupils of the patient are conspicuously small. A degree of myosis as high as we find in opium poisoning has only its parallel, and then but rarely, in hæmorrhages into the pons, which are rapidly fatal.

Uræmic coma can easily be excluded, if we are able to examine the urine, and can detect neither albumin nor tube casts; besides this, with the history, the examination of the heart for a possible hypertrophy should not be forgotten.

Diabetic coma, finally, is characterized by a peculiar fruity odor which comes from the mouth of the patient. It, of course, only enters into the question if sugar can be demonstrated (or has previously been repeatedly detected) in the urine.

It is not common to encounter any difficulty in deciding which side is paralyzed; nevertheless I have seen instances in which this was the case. Thus it occasionally happens that, owing to the deep coma in which the patient lies, the limbs of both sides fall equally flaccidly when allowed to drop, while no difference can be discovered in the two sides of the face. In such cases it is well to throw some ice-water over the patient, upon which it will be observed that he will make movements of defense only with the non-paralyzed side, and the facial muscles will contract only on that side.

The anatomical nature of the hemiplegia may remain entirely obscure, and only in certain cases are we able to give a decided opinion about it.

Whether hemiplegia following a stroke is due to hæmorrhage or embolism can only be determined by accompanying circumstances. The existence of valvular lesions and of atheroma speaks for embolism; nephritis, heart hypertrophy, albuminuria, for hæmorrhage; yet this rule has many exceptions, and we may assume that in about half the cases a correct diagnosis is impossible (cf. Dana, Med. Record, 1891, p. 30).

The meningitic hemiplegia has these points in common with the hæmorrhagic—namely, the paralysis on one side and the "conjugate deviation"; but, as we have before pointed out, in meningitis we generally have the characteristic rigidity of the neck and the scaphoid abdomen; where these latter symptoms are not even suggested, a differential diagnosis, or rather the recognition of a hemiplegia as of meningitic origin, is impossible.

The hysterical hemiplegia, finally, if it persist for a long time, and if other hysterical symptoms, as anæsthesias or contractures, are wanting, can never with any certainty be differentiated from that depending upon cerebral hæmorrhage. Both may present the same peculiarities, and a decision as to which condition we are dealing with may be beyond the powers even of the practiced diagnostician. We are indebted to Charcot for a new symptom, to which he has drawn attention, and which is said to be characteristic of hysterical hemiplegia—namely, a paroxysmal spasm of the muscles of the cheek of one side, associated with an excessive deviation of the tongue to the same side. This "glosso-labial hemispasm" never exists in organic lesions of the pyramidal tract, and is therefore pathognomonic for hysterical hemiplegia (Brissaud and Marie, cf. lit.).

If the question of the anatomical seat of the hæmorrhage is to be considered in our diagnosis, we must in the first place not forget that the mere existence of a hemiplegia is not sufficient to give us an answer, for as long as we do not know whether to regard it as a direct or indirect symptom, we can say nothing positive. If we further add that even an indirect hemiplegia may persist for years, we can easily see with what difficulties we meet in attempting a topical diagnosis. It may be quite true that in a great many cases where an apoplectic attack is followed by hemiplegia, the lesion is situated in the internal capsule, and we have become accustomed to associate in our minds a certain typical clinical picture—that is, hemiplegia with more or less marked sensory changes—with a lesion in the internal capsule. We must, however, in making a diagnosis of that kind, always keep in mind that an indirect hemiplegia may be produced by lesions in any part of the brain, by lesions in the frontal, in the parietal, the occipital lobe, of the thalamus, of the lenticular nucleus, of the external capsule, and that, as we have also said, the duration of such indirect hemiplegias is by no means always restricted to a period either of a few days or a few weeks. Hence a certain reservation must ever be observed by a prudent diagnostician, and he should speak with some certainty only when he has some other direct focal symptom to guide him. Among these, we have, for instance, sensory aphasia for the (left) temporal lobe; for the occipital lobe, hemianopia; for the optic thalamus (with a high degree of probability), posthemiplegic chorea; for the crura, alternating oculo-motor paralysis; for the pons, alternating facial paralysis. According to Dürck, it is possible at autopsy to determine approximately the age of the hæmorrhage from the condition of the red corpuscles (whether they are normal, discolored, swollen, shrunken, etc.), and from the anatomical and chemical condition of the blood pigment. If these points are taken into consideration, its age within a period of from one to seventy-two days may be estimated (cf. Virch. Arch., 1892, cxxx, Heft 1, p. 89).

Prognosis.—After all that has been said, we hardly need to add anything about the prognosis. Any cerebral hæmorrhage is a grave event, which puts the life of the patient in danger, or rather it is a symptom which denotes that a grave arterial disease, without which a hæmorrhage never occurs, has reached a state dangerous to life. If once a hæmorrhage has occurred

we are not sure but that it may be repeated at any moment, since the condition which favored it, the brittleness of the arterial walls, means a lasting incurable predisposition to a fresh hæmorrhage.

In the presence of a recent apoplectic attack, it is impossible for us to give a certain prognosis, or to predict what will follow. The severity of the disturbance of consciousness is in a way indicative, and we may say that the severer this is found to be—in other words, the greater the traumatic effect of the hæmorrhage—the less favorable is, *cæteris paribus*, the outlook with regard to life, as well as with regard to recovery. Yet exceptions occur, and even a very severe coma which has persisted for hours does not only not always produce death, but need not necessarily leave behind it focal symptoms, as hemiplegia or the like, and such patients may then be well for years afterward. Unfortunately, so favorable a result is rarely met with. As a rule, a hæmorrhage of any considerable size is either fatal or is followed by a hemiplegia.

As to the difference in the prognosis for the individual, indirect as well as direct focal symptoms, most that deserves mention has already been spoken of. The indirect symptoms, as a rule, disappear after a certain time, and a *restitutio in integrum* is not impossible; the direct ones are only curable when vicarious innervation takes place from the unaffected hemisphere which assumes the function of the damaged one. This can be the case, for instance, in unilateral facial and hypoglossal paralysis, and in the lateral deviation of the eyes (lesion of the lower parietal lobule); it may also occur in motor aphasia if the patient is still capable of learning to speak with his right hemisphere (lesion of the region of Broca). On the other hand, it does not occur in cases of direct hemiplegia due to a lesion of the internal capsule; then the paralysis is incurable, and the improvement which may take place is always very imperfect, although a properly conducted treatment may effect some amelioration, and thus conduce much to the well-being of the patient.

Treatment.—The primary affection, the disease of the arteries to which cerebral hæmorrhage is due, is beyond the reach of therapeutics. We possess no remedy which can cause the miliary aneurisms to disappear, and our efforts are confined to combating those symptoms which accompany and those which follow the hæmorrhage. Thus we have to deal