

by Sormani, who based his statements upon an extensive study of statistics (Riv. clin., ser. 2, i, 12 Dicembre, 1871). The same author is also inclined to attribute to the barometric pressure some influence on the mortality, as in his opinion sudden changes in the weather materially increase the mortality from apoplexy.

Symptoms and Course.—The rupture of a fair-sized cerebral vessel is always, no matter what part of the brain is affected by it, attended with more or less violent symptoms.

Only in exceptional cases is it preceded by premonitory indications (*præmonitorium apoplecticum* of Boerhaave). Occasionally there are temporary sensory disturbances in the extremities of one side, formication, numbness, a feeling of heaviness in the limbs, pain in the soles of the feet, certain choreiform movements in the face and arms (*hemichorea præhemiplegica*, Raymond), symptoms which indicate that things are not going in their usual order. The patient may also complain of headache and a feeling of fullness in the head, which makes itself manifest on the least provocation, on the slightest emotion, or after a small amount of wine has been taken. But rarely enough are such premonitions sufficiently appreciated by the patient, and only too often are they incorrectly interpreted by the physician. Usually they are overlooked, and are first remembered when the catastrophe is either imminent or has already taken place.

When the attack does come on, the patient gradually or suddenly loses consciousness, and remains in this condition for a few minutes, hours, or even for a day or two, according to the severity of the "stroke." The higher the blood pressure, and the greater the rapidity with which the blood escapes, the more pronounced and severe are the general symptoms, which collectively are called "apoplectic stroke" (the "insult" of the Germans). The way in which the disturbance of consciousness comes on varies very widely in different cases. Thus one patient may for some hours before the actual attack present a peculiar excitement, he is restless and bewildered, may even have forgotten the ins and outs of his own house, his speech is agitated, etc.; another patient may complain of headache and vertigo; a third of a feeling of heat in his head and of general prostration ("different forms of delayed stroke"). All these premonitory symptoms which we have described may, however, be absent, and a person apparently enjoying the best of

health may suddenly, as if "struck by lightning," sink to the ground and lie there unconscious (*apoplexie foudroyante*).

If we are called to such a case, the following conditions will present themselves to us on our first examination: The patient lies on his bed as if asleep; his respiration is either quiet and deep or loud and stertorous; he can not be aroused in any way, not even by strong irritation of the skin (pricking, tickling); his eyes are closed, and the pupils, usually of medium size, neither much dilated nor much contracted, have lost their power to react. With every expiration the cheeks are slightly puffed out, and it is often soon apparent that one corner of the mouth is lower than the other. The extremities are relaxed, and when raised drop loosely. The tendon reflexes are absent in severe cases, and neither the cremasteric nor the plantar reflex can be obtained. The pulse is full, somewhat slow; the temperature normal, perhaps slightly subnormal; the urine presents no changes, or may contain a trace of albumin, rarely of sugar.

This condition may, as we have said before, last several minutes, several hours, or even one or two days. It is modified gradually according as the hæmorrhage sooner or later comes to a stop or continues without interruption until a fatal result ensues. In the former case the patient gradually begins to react to strong stimuli, and may open his eyes for a short while, when called loudly or when water is thrown over him; he may give a loud yawn and show some voluntary motion of the extremities. Gradually consciousness returns, and the patient attempts to make himself understood by gestures and words, and in the most favorable instances, which are, however, unfortunately very rare, the physician can feel assured that everything has cleared up, that the patient is again in possession of perfect consciousness, of the power of speech, and of motion. In such cases the "general" symptoms have disappeared without leaving behind any of those belonging to the second class, namely, the so-called focal symptoms (Griesinger), and we speak of a "stroke without focal symptoms."

But the bleeding may continue, although only under low pressure, and only cease very gradually; then the symptoms abate but slowly and the recovery is only partial; the patient lies for days in a state of somnolence, and repeated examinations show that one corner of the mouth is distinctly lower than the other, and that the saliva dribbles from it involuntarily. If we can, by strong stimuli, evoke spontaneous move-

ments, it becomes evident that only one side is moved, that only one arm or one leg is raised, while the other side remains perfectly motionless, and after consciousness is fully restored the certainty is forced upon us that one side of the body is deprived of its power, or, as we say, is paralyzed. This we call a "stroke with focal symptoms." At this stage, however, we are unable to decide whether the focal symptoms are the result of a destruction of nerve paths or centres—in other words, whether they are direct and therefore incurable—or whether they depend upon indirect action, so that the loss of function is only temporary. If the latter be the case, we speak of "indirect" focal symptoms.

Again, the hæmorrhage may not cease at all, but continue with increasing blood pressure; then the patient remains unconscious, the breathing becomes irregular and more rapid, and assumes the so-called Cheyne-Stokes type, the pulse becomes more rapid and small in volume, the face grows pale and haggard, the saliva getting into the trachea produces the well-known tracheal rattling, the temperature rises gradually but noticeably, and the patient dies without having come to himself, and after a period of unconsciousness which may have lasted many hours or even several days and nights.

If, in the course of a "delayed stroke," the breathing, until now quiet and regular, suddenly gives place to a rapid, irregular, stertorous respiration, and if at the same time the partial unconsciousness deepens into a profound coma, the reflexes become lost, and tetanic convulsions of the whole body and hemicontracture of the paralyzed side make their appearance, then we can assume that the hæmorrhage has burst through into a ventricle, and give an absolutely unfavorable prognosis, because in a few hours, more rarely in one or two days, death almost invariably follows. The hæmorrhage itself in such cases is, as can be demonstrated at the autopsy, generally by no means copious, but the fact that it is found, even if the ependyma of the ventricles be thickened and hardened, speaks most clearly for the high arterial pressure under which the blood escapes (Wernicke). The bursting of the blood into the fourth ventricle is the most rapidly fatal, and it is in these cases that we sometimes observe nystagmus.

The disturbance of consciousness in its many gradations, from the slight vertigo to the deep coma, is the most characteristic, or at least the most important, symptom of an apoplectic

attack produced by hæmorrhage, and it ought not to be underrated, even if it does not become fully developed, but only amounts to a transient slight speech disturbance, accompanied by a feeling of faintness and weakness. There are patients in whom such disturbances occur several times before the onset of a real attack. Such patients complain of transient vertigo, slight weakness, and heaviness in one or the other hand or foot; they can at times not find the right word, the correct expression, or lose speech entirely for a short while. All these indications are premonitions, not direct forerunners of the attack, but symptoms which warn us, indicating that the brain is subject to alterations in the blood pressure, a condition which may lead to serious consequences if the arterial walls are diseased ("apoplectic equivalents").

Complete absence of all disturbance of consciousness is a rare exception, and can only be found when the blood escapes quite slowly, so that the increased pressure rises only very gradually, and to no great degree. The patient then is seized with a sudden weakness, purely physical; he sinks into a chair, and after a few moments, during which time there is not the slightest disturbance of consciousness, he becomes aware of a sort of difficulty in moving the extremities of one side, which, in the most unfavorable instances, in a short time passes into a genuine paralysis of that side (focal symptoms without stroke). Here may also be mentioned the cases observed by Römberg, Graves, Andral, Senator, and others, in which after a hemiplegia no trace of hæmorrhage was found at the autopsy, but only a diffuse hyperæmia of the brain could be demonstrated—"pseudo-apoplexy."

On the other hand, it is not a very unusual occurrence that a patient awakening in the morning after a quiet night's rest finds himself paralyzed on one side; in such cases we are, of course, not able to decide how much his consciousness would have been impaired had he been awake.

In every hemiplegia that occurs in the course and as a consequence of cerebral hæmorrhage there is a possibility of regeneration to a greater or less extent; but whether this regeneration will take place, and when, and, moreover, whether it will be complete or not, are questions that can not at once be decided. They all depend on the condition of the cortico-muscular tract, as we have pointed out before—upon whether this

be actually interrupted, whether its fibres in places, for instance at the internal capsule, be completely destroyed, or whether their function be only temporarily impaired in consequence of the increased blood pressure, so that after the cessation of the hæmorrhage a *restitutio in integrum* of the nerve tissue can follow. In the latter case the paralysis disappears after a few hours or days, while after an actual interruption of the cortico-muscular tract the hemiplegia is incurable, and the patient is deprived of the free use of the affected limbs, and, even though he may regain after a long time some power of motion, his movements will always remain awkward and restricted.

Sometimes, and this is not very rare, a patient may have an apoplectic stroke after which the paralysis disappears quickly and entirely, but which is in a few days, on some slight provocation, followed by a second attack, accompanied by a severe permanent hemiplegia, which under certain circumstances can cause death. Such a possibility should always be thought of, and we would here say that the prognosis, no matter how slight and favorable the apoplexy may seem, should always be very guarded.

Among the "concomitant symptoms" which only exceptionally persist for any length of time, and ought therefore to be regarded as indirect focal symptoms, may be mentioned a peculiar deviation of the eyes and the head—the "*déviatio conjugée*" of Prévost—generally toward the side of the lesion, so that the eyes "look toward the disease-focus." This has been thought to be associated with a lesion in the upper parietal lobule. Prévost and Landouzy gave this rule: "Le malade regarde son hémisphère altéré s'il y a paralysie—il regarde ses membres convulsés s'il y a excitation" (the patient looks toward the damaged hemisphere if he have a paralysis; if there be irritation he looks toward the convulsed limbs). This is seen, for instance, in the so-called cortical epilepsy, which we have spoken of on page 187. The head seems forcibly turned to one side, and the eyes are turned so far over to the canthus that we are scarcely able to test the condition of the pupil; along with this symptom there is found almost always a more or less marked dullness of the sensorium. Why this condition is generally transient is explained by the fact that the muscles of the eyes and neck can be innervated from both hemispheres, so that even if one side becomes incapable of working, the other can act vicariously for it. Only in bilateral hæmor-

rhages which produce a permanent paralysis of the eye muscles is conjugate deviation found to persist.

Unilateral oculo-motor paralysis on the side of the hemiplegia is very rare; it is supposed to be associated with lesions of the lower parietal lobule.

After a severe attack there may be a transient polyuria lasting for one or two days; the specific gravity of the urine, which is then faintly acid, may be 1.003 or 1.002; at times, but not always, albumin or a trace of sugar can be demonstrated (Loeb, Prager med. Wochenschr., 1892, 50). This some authors, among them Ollivier, were inclined to attribute to an action on the centres situated in the floor of the fourth ventricle, the existence of which Claude Bernard had already demonstrated. This polyuria after an apoplexy does not persist, while this may be the case in tumors of the posterior fossa, in focal lesions of the pons or the medulla oblongata, where it has to be looked upon as a focal symptom (Kahler, Zeitschr. f. Heilk., vii, 2, 3, 1886).

In proceeding to the examination of a fresh hemiplegia—that is to say, one of a few days' or weeks' duration—the following points must be borne in mind:

The facial and the hypoglossal nerve deserve the most attention (cf. also Koenig, Deutsche Med.-Zeitg., 1892, 25, p. 298). The former is injured in its central course, and shows a paralysis or only a paresis in its lower branches, while the upper branch is intact; the patient is unable to inflate the paralyzed cheek, and can not whistle, while wrinkling of the forehead on the paralyzed side presents no difficulty. Careful examination shows distinctly that the disturbance on the paralyzed side of the face is much more marked on attempting voluntary movements of one side alone, whereas those of expression—for instance, laughing, crying—are at least passably executed. This, again, may be explained by the fact that muscles used involuntarily are innervated from both hemispheres. The duration of the facial paralysis varies; sometimes the difference between the two sides of the face disappears almost completely in a few days, while in other instances it may be noticeable for weeks or, in rare exceptions, even during the whole life. In this point it resembles the speech disturbance caused by a lesion of the hypoglossus, a disturbance consisting essentially in faulty articulation, which is noticed by the patient more than by those who converse with him. It may disappear

in a few hours, but may persist for months, even years, when improvement in the affected side has gone on for a long while, and gratifying progress has already been made. A paralysis of the same nerve, or rather of the genioglossus muscle supplied by it, is also responsible if the patient is unable to protrude the tongue straight; it is deviated to the paralyzed side because the well genioglossus is stronger than the diseased one, and consequently pushes the tongue over toward the side of the latter.

The condition of the soft palate is not the same in all cases. The velum may be considerably lower on the paralyzed than on the well side, but it may also occupy its normal position. The uvula is at times deviated to the well, at times to the paralyzed side, and again at other times its position may be unchanged. These changes do not give rise to any noticeable disturbance of function.

Examination of sensibility in the first few days reveals decided alterations. Sensibility to pain in most cases is dulled, and sensibility to touch and pressure is decreased, though to a less marked degree. The patient feels a pin prick either not at all on the affected side or, at any rate, with less acuteness.

Of the nerves of special sense, it is especially the optic which takes part in the disturbance. The apoplectic attack may be followed under certain circumstances by hemianopia of the corresponding side (Gowers), often, too, by amblyopia.

Smell and taste, as a rule, do not suffer to any great extent; but there is a decrease in hearing power, so that the patient is no longer able to understand words spoken in an ordinary tone at a distance of fifteen or twenty feet. Such a decrease is not rare, yet an absolute (unilateral) deafness never seems to follow as a result of an apoplectic attack.

With regard to mobility, examination shows that either the extremities of one side of the body are completely paralyzed (hemiplegia) or that the power of movement in them is impaired (hemiparesis). In the latter case the arm is usually more affected than the leg and the hand more than the arm. Indeed, the movements in the shoulders and elbow joint may be as good as normal, while those of the fingers are very awkward; in such cases the leg can generally be moved quite well. The muscles of mastication and those of respiration are, for the reasons above mentioned, almost intact, the muscles of the trunk are only slightly implicated, and, if at all, the change is

only apparent in the trapezius, so that the shoulder of the affected side is raised less energetically than its fellow.

The tendon and skin reflexes are, in the first few days after the attack, decreased or even lost on the affected side, a condition which, as we shall see shortly, soon becomes materially changed.

The sensorium usually clears up in from one to four days, especially in light cases. The patient again becomes conscious of his surroundings, and recollects quite well all incidents which happened nearly up to the time of the attack. Thence on, there is, of course, a blank in his mind. On awakening, at first he has no idea of what has happened to him. His frame of mind varies according to the degree of his bodily helplessness, but, as a rule, is better than we might expect, considering the damage which has been done. Sleep is for weeks much interfered with. The patients are extremely restless; they throw themselves about in bed, and are unable to remain in one position for any length of time.

The further course depends upon whether the hemiplegia proves to be an indirect or a direct focal symptom.

The slighter cases of indirect hemiplegia, when they have not completely passed off after several weeks, are at any rate generally improved. The one-sidedness of the face, seen at the beginning, has disappeared; the tongue is now protruded straight, speech is again normal, the leg can be moved almost as freely as ever, and the only thing which is left as a reminder of the dangers through which the patient has passed is a certain awkwardness in the movements of the affected hand.

The graver cases of indirect hemiplegia need from two to three months for complete recovery. For weeks after the attack the patient presents marked disturbances in motion as well as sensation, and only painfully and with the help of a stick can he hobble about his room, while the arm and hand are almost useless. Yet a constant progressive improvement of the paralyzed limbs enables us to recognize the favorable tendency of the case and to predict with certainty a complete recovery.

In cases of direct hemiplegia also the course of the disease may assume many varieties. All are characterized by the persistence of the focal symptoms. The attack, too, we should keep in mind, need not be particularly severe, nor need the initial general symptoms have been especially grave; only the conjugate deviation of the eyes and head is a symptom which

preferably occurs in grave hemiplegia. Its presence, therefore, permits *a priori* of an unfavorable prognosis with regard to complete recovery.

In the first three or four weeks things remain apparently about the same; the paralyzed side is flaccid and about five ninths to one degree centigrade warmer than its fellow, the slightest motion is impossible, speech remains impaired, and the face is one-sided. It is not until from three to six months have passed that we are able to notice a slight improvement in the power of motion, so that the patient (who is still confined to bed) is able to move with ease some of his toes, perhaps also the lower leg, while in the thigh motion is still incomplete, and in the arm and hand quite impossible. In such cases all the improvement that can be expected is but small and the damage which the stroke leaves very apparent. After from six to twelve months the patient again begins to be able to use the paralyzed leg, which in the meantime, in consequence of the flaccid condition of the ankle, has become longer. The walk is then very characteristic. Flexion in the hip being insufficient, the affected leg is brought forward by the aid of the pelvis, so that, trailing along the ground, it describes a half circle around the sound one. The centre of gravity of the body then is transferred to the paretic leg, the knee joint passively extended, and the leg thus used as a stilt (Wernicke). If improvement goes on, the movement of circumduction gradually disappears and the paretic leg is simply dragged behind. The gait is so characteristic that the diagnosis, especially when simultaneously there is a paretic condition of the upper extremity, can be made at a glance.

The upper arm is slightly abducted, the forearm flexed, the hand hangs down, the fingers, which are fixed in a somewhat flexed position, are completely useless, and the patient is unable to grasp large or small objects. The arm can hardly be raised at all, and the movements of the forearm on the upper arm are very limited. In the lower leg extensor are more frequently developed than flexor contractures, and it is remarkable that in the morning, when the patient awakens after a long sleep, how slight they are and how little they trouble him, whereas in the course of the day they are materially increased.

Contractures, which are in old hemiplegias hardly ever absent, are most likely to be attributed to a shortening of the muscles produced by disuse. This idea is supported by the

fact that by systematic passive exercise, begun as soon as possible, we are able to prevent contractures; and if they exist, a proper galvanic treatment, which takes the place of passive motion, perceptibly diminishes them. It is true it remains unexplained why contractures are not found in all cases, and why in some the paralyzed extremities remain for life flaccid. That anatomical changes, too, especially, as Charcot assumes, the secondary degeneration of the pyramidal tract, are not without influence, and that, at any rate, the contractures are more marked the farther this secondary degeneration advances, can not be denied.

A symptom which accompanies contractures, but which often occurs much earlier, is an increase in the tendon reflexes on the paralyzed side. Tapping of the triceps and biceps tendon of the arm, of the patellar tendon, and the tendo Achillis evokes lively muscular contractions. From the last named—the tendo Achillis—we can also obtain the so-called ankle clonus, of which phenomenon we shall speak later. Even tapping of bones is attended by jerkings, which are best seen in the leg when the tibia is struck (“periosteal reflex”). Here again we must leave the question open whether this increase in the reflexes is due to the degeneration in the pyramidal tracts or merely connected with the suspension of certain reflex-inhibiting influences in the brain. In favor of the latter hypothesis speaks the fact that this increase in the reflexes is occasionally observed as early as a few days after the stroke, at a time when there can be no question of degeneration in the spinal cord.

With the skin reflexes it is just the reverse; they are usually entirely lost on the paralyzed side or are at least decidedly diminished. This is especially the case for the abdominal and cremasteric reflexes, which can only in exceptional cases be obtained on the affected side.

Sensation either returns soon after the initial disturbance or is permanently lost. In the latter case—i. e., where besides the hemiplegia there exists also a hemianæsthesia—the lesion is to be located in the posterior portion of the posterior limb of the internal capsule. The hemianæsthesia takes in, in pronounced cases, the whole half of the body, including the mucous membranes, and extends as far as the median line. Face and trunk are equally affected; occasionally we may find that the trigeminus remains exempt.