complete muscular resolution. On emerging from such an attack there may be hemiplegia; if right hemiplegia, associated with more or less disability of speech, possibly with aphasia. In other cases, with equal suddenness, but without any apoplectic seizure, there may occur a hemiplegia, or the paralysis may be limited to the arm, or to the leg, or to the face; it may be complete or partial (paresis), and with weakness there may be contractions and rigidity. The paralysis may disappear quickly, and after an uncertain period may occur again, or be succeeded by rigidity and contraction. The disappearance of a paralysis under these circumstances means the reopening of the obstructed area to the circulation by collateral channels or anastomoses—a condition of things only possible in the cortex. An autochthonous thrombus may form in a vessel of the basal system. The final occlusion of the vessel may be preceded by various prodromata—by headaches, vertiginous sensations, numbness, tingling, formication, coldness, muscular cramps, etc. Paralysis may develop slowly, as the thrombus slowly forms, or suddenly, with the usual phenomena of the apoplectic stroke; the paralysis is strictly localized and does not change, for, the vessels being of the terminal kind, collateral hyperæmia and œdema result, and the affected tissue goes through the process of necrobiosis. When occlusion occurs in this way, the subsequent phenomena are the same as those of embolism. As the embolus causing the cerebral mischief comes from some distant point in the vascular system, it is obvious that there can be no intra-cranial disorders produced by it ere it effects a lodgment in the brain. It is evident that there must be very considerable variation in the severity of the symptoms, according to the importance and the situation of the vessel occluded. In a majority of cases the attack is apoplectic—there may be for an instant intense headache and dizziness, sudden flush or pallor of the face, or the patient may utter a wild cry—he falls immediately into unconsciousness, with complete muscular resolution, or there may be a distinct epileptiform seizure. Instead of unconsciousness, the stroke may be nothing more than a severe vertigo, with confusion of mind, muscular twitchings on the affected side, and vomiting. Vomiting may also occur in the apoplectic form, just as the mental confusion is coming on. On recovering from the stroke or shock—which is doubtless due to the suddenly produced partial anæmia, effecting at the same moment an immense change in the intra-cranial blood-pressure—a hemiplegia is found to exist, and it is most frequently of the right side, owing to the arrangement of the vessels on the left side of the brain. Although right hemiplegia is usual, it is not invariable: there may be left hemiplegia, or bilateral paralysis, or paralysis of the different cranial nerves. Embolism may also affect the central artery of the retina, and amaurosis result from the occlusion. Double optic neuritis arises during the course of all "coarse

organic lesions" of the brain, and hence ophthalmoscopic examination is a necessary duty in such cases. The mental functions are variously affected. In the slow form of occlusion—thrombosis from chronic endarteritis—there is gradual mental failure, beginning in loss of memory, and thence the spectacle of senile dementia. In embolism the mental faculties are, during the period of coma, entirely suspended; if the patient emerge from this with hemiplegia, the mind is always enfeebled to a greater or less extent, the language faculty is variously impaired, the emotional nature is highly excited, and the reason and judgment are clouded. With right hemiplegia from embolism there is usually associated *aphasia*, or loss or impairment of the faculty of communicating ideas by words or by signs. The hemiplegia involves the tongue and the corresponding side of the face. The reflex movements are readily excited in the paralyzed parts. When there is embolic obstruction of the basilar artery, the symptoms differ somewhat from the description above given. The hemispheres are not involved, nor the important parts supplied by the Sylvian artery; there is no apoplectic seizure, nor loss of consciousness, nor troubles of the intellectual faculties. There are disorders in vocal expression, due to paralysis or ataxia of the muscles of the tongue (ataxic aphasia), but vertigo and vomiting are usual symptoms.

Course, Duration, and Termination.—The course of symptoms referable to the changes preceding and resulting in thrombosis is essentially chronic. Months and years may be occupied in reaching the point of coagulation, and other months, even years, may be passed in the paralytic state. When the lesions are of the basal system they are permanent. Although there may be some improvement, which, however, does not continue, the members paralyzed remain in the condition at which they had arrived after several months. In thromboses the most sudden and considerable improvement takes place in paralysis of members, defects of speech, and disorders of sensations, due to disease of the vessels of the cortex; but the probability of the return of these lesions, or of the appearance of other lesions, should not be forgotten. While the prospect of great immediate improvement is good in such cases, the future must be regarded with apprehension. On the other hand, in embolic occlusion, the immediate results are more severe. Death may be the result of the occlusion of a large vessel within two or three days, or longer, the patient never emerging from the coma. In other cases the patient arouses from the coma, hemiplegia exists with aphasia, the temperature rises a little as the collateral hyperæmia and œdema come on, but falls again in a few days, and the case then pursues the usual course of localized softening from any cause. Right hemiplegia and aphasia, from blocking of the left middle cerebral, may occur in youth, early manhood, at any period in fact, and are associated with valvular disease, usually of rheumatic origin. These lesions

may also be associated with aneurism, with syphiloma, or with ulcerative endocarditis.

Diagnosis.—The diagnosis of thrombosis rests on the evidence of chronic arteritis—the simultaneous presence of the changes in the radial, the color of the hair, the condition of the skin, an arcus senilis; on the variability and diffusion of the prodromal signs, and those of the established lesions. Embolism is known by the age of the subject (often so at least), by the history of rheumatism, the existence of valvular lesions, by the suddenness of onset without prodromes.

Treatment.—The author has had remarkable results from the following plan of treatment in thrombosis: Carbonate and iodide of ammonium (ten grains of the former and five grains of the latter) are given three times a day in a suitable vehicle, for several months, usually, the object being dual—to increase the action of the heart and arteries, and to effect a solution of thrombi forming by maintaining the alkalinity of the blood. To postpone and possibly arrest the atheromatous degeneration of the vessels, cod-liver oil and the sirup of the lactophosphate of lime are regularly exhibited (a teaspoonful of each) three times a day, immediately after meals. The ammonia solution is administered before meals. At the same time these remedies are being given, a daily dose (at 10 A. M.) of quinine (five to ten grains) is also prescribed, should there be a condition of depression and languor of the intracranial circulation requiring it, but the carbonate of ammonia is usually sufficient. With this plan is conjoined a suitable regimen—a simple but nutritious diet, moderate exercise, and careful supervision of the various excreta. As soon as possible after an embolic obstruction has occurred, carbonate of ammonia should be given—very usefully in the liquor ammonii acetatis—and should be kept up for weeks. The most absolute rest should be maintained, and the diet should be light and unstimulating. In a month or two a very light galvanic current (from two cups) may be passed through the brain in both directions. Quinine is most useful, especially if there be any elevation of temperature; but in all cases it has seemed to the author highly useful after some weeks' administration of ammonium carbonate.

OBLITERATION OF THE CEREBRAL CAPILLARIES.

Pathogeny.—The capillaries of the brain are occluded by the finer particles which readily pass through the larger vessels. In the severer forms of acute malarial poisoning small particles of pigment are formed, and, entering the cerebral capillaries, lodge, and are known as "*pigment embolisms*." Violent delirium, terminating in coma, and sometimes convulsions, may result from the occlusions formed in this way. The white-blood corpuscles, under conditions not now understood, aggregate in masses and form emboli. These are probably examples of

pyæmic change, for such emboli have been formed in connection with pyæmia, erysipelas of the face, etc. Emboli, consisting of particles of cancerous, septic, or decomposing material—*infective emboli*—may also be minute enough to pass the larger vessels and occlude the cerebral capillaries. In very rare cases the capillaries are blocked by lime salts, taken up at some point where disintegration of bone is going on—*lime-salts emboli*. Again, emboli consist of fat-globules which enter the blood from the marrow of fractured bones—*fat emboli*. The capillaries of the lungs may arrest them entirely, and hence the most serious symptoms are referable to these organs; but the finest globules may pass through the lungs and block some of the cerebral capillaries. As the anastomoses between the capillaries are very abundant, it is obvious that if the obstructions are but few in number they will be compensated for. When numerous, there will be produced anæmia, followed by the usual changes of necrobiosis, ending in softening.

Symptoms.—In the case of pigment embolisms occurring during a malarial fever, the onset of this malady is announced by intense headache, vertigo, delirium, sometimes convulsions, and the febrile phenomena are greatly intensified. If, during the course of facial erysipelas, similar symptoms arise, they may be due to white-corpuscle embolisms, or, if occurring after a fracture of a bone, may be due to fat-embolisms. When the embolisms are not very numerous the symptoms may be less pronounced: there may be dizziness, loss of memory, and other mental defects, persistent headache, etc. In any case the diagnosis can hardly be more than a fortunate guess. The treatment may be conducted on the same basis as that of occlusion of the arteries.

OCCLUSION OF THE CEREBRAL SINUSES.

Pathogeny.—Thrombosis is the mode of occlusion of the cerebral sinuses, and it may result from venous stasis or from phlebitis. In the former case the propelling power of the heart is much reduced, and the fibrin of the blood increased (hyperinosis). This condition of affairs occurs chiefly in children exhausted by long-standing illness; in the cases observed by the author, there had existed an ileo-colitis of several weeks. The phlebitis is secondary to some morbid process in the neighborhood, most frequently to caries of the petrous portion of the temporal bone, and the petrosal or transverse sinus only may be attacked, but the purulent phlebitis extends occasionally to the cavernous sinus and the circular sinus. Next to caries of the bones, the most frequent cause of this form of thrombus is erysipelas of the head and face, carbuncle of the upper lip or nose, and malignant pustule of the lip. The position of the thrombus is determined by the nature of the cause: if caries, the thrombus is found in the transverse or petrosal or cavernous sinus; if erysipelas, or malignant carbuncle, in the ptery-

goid plexus and cavernous sinus ; if stasis from cardiac feebleness and hyperinosis, in the longitudinal sinus. The thrombus and the subsequent changes taking place in it are the same as those already described. The vessels entering the sinus, the seat of occlusion, are turgid, tortuous, and their tunics weakened, so that they yield to the increased pressure, and hæmorrhages occur at various points, on the hemispheres, especially in the cortex. Softening occurs to a small extent about the hæmorrhagic extravasations, and meningitis may arise as a complication.

Symptoms.—As the cases of thrombosis of the sinuses occur in the subjects of wasting maladies, or of cardiac feebleness, the symptoms produced by the thrombus are superadded to those of the original malady. The signs by which such an occurrence may be recognized are all the more obscure, since the anæmia of the brain may be accompanied by many of them. There have been observed the following: rigidity of the cervical muscles, the occiput being buried in the pillow, and sometimes general muscular rigidity ; ptosis, strabismus, nystagmus, and paresis of facial muscles ; hebetude of mind, stupor passing into coma, sometimes delirium ; headache, vertigo, nausea and vomiting ; delirium, ending in coma ; contractures, or paresis, local tremor, clonic convulsions ; paralysis may be crossed with contractures and rigidity. Indeed, so various and diffused are the symptoms that the diagnosis must always be in the nature of a guess. More importance is to be attached to circulatory disturbances affecting external vessels. The facial vein communicates with the pterygoid plexus of veins and the cavernous sinus ; the nasal veins communicate through the foramen cæcum with the longitudinal sinus, and the occipital veins communicate with the transverse sinus by the *emissaria mastoidea*.* Hence, bleeding at the nose, puffiness of the eyelids, swelling of the facial vein, and of the occipital veins, accompany thrombosis of the sinuses. From the same cause there will be prominence of the eyeballs, injection of the conjunctivæ, and a swollen and tortuous condition of the retinal veins, cloudy swelling of the optic disk (choked disks), etc. In the case of thrombus of the cavernous sinus, there may be irritation by pressure of the fifth nerve, and consequent neuralgia—of the fourth, and internal strabismus ; of the oculo-motor, and contracted pupil and external strabismus, etc. These symptoms have a high degree of importance if present ; but their absence does not negative the existence of thrombosis. During the course of chronic otorrhœa and caries of the petrous bone, cerebral symptoms may supervene, and a fever of septicæmic character develop. When delirium tending to coma accompanied with typhoid symptoms appears during erysipelas or phlegmon of the upper lip, there may be suspected, as in the former case, that the new symptoms may be due to thrombosis of a sinus. The diagnosis must always be largely conjectural.

* Henle, "Gefässlehre," p. 341.

Treatment.—The treatment consists in the free use of carbonate of ammonia and quinine, given with the objects in view indicated under the head of occlusion of the cerebral vessels. Unfortunately, when this accident occurs, there is little chance of accomplishing any good. Whenever a phlegmon of the upper lip appears, the probability of this accident should be kept in view. Free administration of quinine is undoubtedly serviceable in preventing this complication.

CEREBRAL HÆMORRHAGE.

Definition.—By this term is meant, the giving way of a vessel and the escape of blood into the cerebral tissues. *Apoplexy* is sometimes used synonymously with cerebral hemorrhage, but incorrectly, since it is a symptom merely, and not a disease.

Causes.—The principal cause of cerebral hemorrhage is disease of the vessels—aneurismal dilatations seated on the arterioles and varying in size from a pin's-head to bodies too minute for the unaided sight to recognize. It is rare for these bodies to form before forty, but they occur with increasing frequency with the advance in life. The change is a periarteritis and begins in the perivascular lymph-sheaths, thence extends to the adventitia, the muscular layer dilates, and the aneurism is formed.* Atheromatous degeneration of the tunics of the vessels may be an indirect cause, by leading to the formation of the miliary aneurism. Increase in the blood-pressure is said to have an influence in causing hæmorrhage, but not directly. When disease has weakened the vessels, an increase in the blood-pressure will cause them to yield, but, without such change in the walls of the vessels, mere variations of pressure will not suffice. The principal source of increased blood-pressure is hypertrophy of the left ventricle—that form associated with hypertrophy of the muscular layer of the arterioles and contracted or fibroid kidney. Besides the constantly exalted pressure, the intra-cranial vessels may be exposed to sudden increased strain by a variety of causes : by stimulants, as alcohol, opium, coffee, tea, etc. ; by a cold or hot bath, by a full meal, and by moral emotion. Cerebral hæmorrhage is notably increased by the cold weather of autumn. Venous hyperæmia may lead to cerebral hæmorrhage, as coughing, straining at stool, coitus, etc., but disease of the vessel-walls must predispose to the accident. The arterial disease on which hæmorrhage depends is probably transmissible, for it is a matter of common observation that the tendency to cerebral hæmorrhage is inherited.

Pathological Anatomy. — Certain parts of the brain seem particularly liable to cerebral hæmorrhage : the corpus striatum, the lenticular nucleus, the thalamus opticus. When these parts are affected, the damage is not always confined to them, but the neighboring parts of the

* Eichler, "Deutsch. Archiv für klinische Med.," xxi, 1, 32.

hemisphere are damaged simultaneously, and the lobes of the hemispheres are often separately attacked, the anterior and middle more frequently than the posterior lobe. Next in point of frequency, but much less often, the cerebellum is involved, and lastly, although rarely, the pons and medulla. The blood is not necessarily confined to the point whence it escaped : it may break through to the surface or into the ventricles and pass by the *iter* from the third to the fourth ventricle. When the amount is large, the dura mater may be put on the stretch, the convolutions compressed, the sulci lessened in depth. The blood may be collected in a mass or focus, or it may be spread out into a more or less thin layer. When in a focus, as is most usual, the collection is somewhat circular and varies in size from a pea to an English walnut, or larger. There may be one or several foci, and they may occur in symmetrical parts—as a focus in each corpus striatum, for example. Besides a recent there may remain the evidences of former hæmorrhages. Immediately after it has occurred there is a blood-clot, dark in color and homogeneous in its constituents, which are those of blood merely, although around it is broken-down cerebral matter, mixed with blood-clot, and in the mass somewhere will be found, if carefully traced out in water, the affected vessel and its ruptured miliary aneurism. Soon after the clot has formed, separation begins, and the fibrin collects in the center of the mass or at the periphery, while the corpuscles adhere in a group, and the serum pressed out saturates the adjacent broken-up cerebral matter. The next step, if death does not occur, is the retrograde change in the blood-clot, which becomes first of a dark chocolate-color, but the hæmatin disappears, the watery part is absorbed, and a yellow, puriform-looking material only remains. A limiting inflammation may occur in the adjacent cerebral matter, a connective-tissue membrane of a spongy structure forms, and the remains of the clot will be inclosed in this. Besides the yellowish, puriform fluid or a whitish, whey-like fluid, there are contained crystals of pigment in the meshes of the cyst-walls. The clot and the surrounding brain-substance do not always undergo this favorable disposition. An inflammation may be lighted up in the brain-tissue, around the clot, in a few days after it has formed, producing extensive softening and œdema. The cysts formed may continue indefinitely without further change, or they may ultimately disappear, leaving only a cicatrix of considerable area, but thin, and composed of either dense connective tissue, or of a spongy material containing pigment. The changes due to cerebral hæmorrhage are not limited to the site of the original injury. Some months afterward an atrophic degeneration has taken place in the nerve-fibers of the pyramidal tracts. These degenerative changes do not follow all cases of cerebral hæmorrhage. They occur after hæmorrhage into the internal capsule, the corpus striatum, the gray matter of the motor zone, and the subjacent white substance, and

less so when the lesion is in the optic thalamus and centrum ovale, and not at all when the hæmorrhage is in the caudate nucleus.* The atrophy extends downward through the *crus*, the *pons*, and the pyramidal tracts, and consists in wasting of the nerve-elements and an increase of the connective tissue.

Symptoms.—Many cases of cerebral hæmorrhage are preceded by distinct prodromes. The most usual are those connected with chronic arteritis, which may lead to thrombosis, or less frequently those dependent on cerebral hyperæmia. Headache, vertigo, sudden attacks in which the mind is confused, the memory for words is lost, or mistakes in the use of words occur ; changes in the disposition, becoming morose, dejected, and irritable, weakness of a limb or of one side, numbness, tingling, or a feeling of coldness in a member or several members, double vision, weakness of the tongue, paresis of the facial muscles, etc. Sometimes, as the author has witnessed, the apoplectiform variety of cerebral congestion is followed in a few weeks by severe or fatal cerebral hæmorrhage. In many cases there are no "warnings," no prodromata, but the hæmorrhage occurs suddenly. The character of the seizure varies greatly. It may be apoplectic ; the patient utters a cry or a groan, and falls insensible. Usually some symptoms occur just previously to the loss of consciousness ; there is headache of a very intense kind, or giddiness with nausea and vomiting, or the tongue is paralyzed and speech impossible, or there is delirium or incoherent rambling, or there is gaping, a feeling of great desire for sleep, and increasing drowsiness, or there may be intense weakness of the limbs and a feeling of exhaustion, or one limb may be seized with intense numbness and tingling, or there may be spasm of the muscles soon to be paralyzed—in a great variety of ways the attack may be announced some hours or minutes before the blow falls. The patient passes into unconsciousness, with complete muscular relaxation, and the extinction of reflex movements, the action of the heart and the respiration continuing. In the less severe cases the unconsciousness is profound, but strong irritation may induce reflex movements, and swallowing is possible if the substance is placed in the pharynx, and a difference between the movements of the two sides is also apparent. The eyes—and the head, also, frequently—deviate toward the side affected in the brain and from the side paralyzed : this movement constitutes a means of diagnosis between cerebral hæmorrhage and other causes of profound unconsciousness. Convulsions of the epileptiform variety may occur, when the hæmorrhage causes unconsciousness, and usually signifies large hæmorrhage, or hæmorrhage into the pons or medulla. When the hæmorrhage occurs slowly, and the patient glides gradually into unconsciousness, there may be

* Flechsig, "Archiv für Heilkunde," 1877, and No. 53, 1878.

nausea, vomiting, and pallor of the face, but in most cases of cerebral hæmorrhage the face is rather red and flushed. There is no constant rule as to the size of the pupils: a very minutely contracted pupil usually signifies hæmorrhage into the pons; and unequal pupils, one being largely dilated, indicate a large hæmorrhage breaking through into the lateral ventricle. The breathing has usually, but by no means invariably, the stertorous character, by which is meant the drawing in of the paralyzed cheek with inspiration and its puffing out with a sort of explosion in expiration. The pulse is small or full, slow or irregular, usually slow and full. There are apoplectic examples of cerebral hæmorrhage in which the unconsciousness is not profound—the patient may be roused, if he is loudly called, but lapses into a soporose state at once. There are many cases in which consciousness is not lost at all: there may be a temporary confusion, or some of the symptoms called prodromal, and then paralysis of one side occurs. Often it is sudden and complete; again it comes on slowly, and is not complete for some minutes. In the apoplectic form, death may occur during the unconsciousness—in from five minutes to three days. The fulminant cases, which terminate in a few minutes, are comparatively rare—sudden death being usually caused by heart-disease. If unconsciousness continues longer than twenty-four hours, death is the usual result. The temperature during the period of unconsciousness is low—below the normal, one or two degrees—but at the end of the first day a rise to normal or a little above takes place, and, if a fatal result, there is a great rise just before death. Pneumonia is apt to be the cause of death, especially when the cerebral lesion is somewhere in the right hemisphere, as Brown-Séquard has demonstrated. Consciousness may return in a few minutes, but usually in from half an hour to three hours. Again, the effects of the seizure may continue for days, there being stupor, confusion of mind, defects of speech. The return of consciousness is indicated by the revival of reflex excitability, by the effects of irritation, etc. The progress of restoration may be retarded by the onset of inflammatory symptoms at the expiration of two or three days; the temperature rises a degree or two; headache, confusion of mind, and delirium occur; tonic contractions ("early rigidity") ensue in the paralyzed muscles, and they become the seat of severe pain, which may persist for a month or more, while the other symptoms disappear in a few days.

When the disturbances due to the seizure subside, then may be clearly seen the extent of the paralysis. The shock of the attack suspends the functions of many parts of the cerebrum, which soon functionate again as these effects of the injury subside. Various paretic and paralytic symptoms, that appear at first, quickly cease, but the more permanent results are the more evident. The amount of paralysis varies from a hardly appreciable weakness to an absolute extinction

of motility. As there is usually but one focus of hæmorrhage, the resulting paralysis is *unilateral*, and on the side opposite the lesion, and involves the muscles of the face, of the tongue, of the body, and of the extremities—*right or left hemiplegia*—according to the cerebral hemisphere invaded. The muscles of the face paralyzed are those of expression, and are innervated by the seventh nerve. Those branches of the nerve distributed to the orbicularis palpebrarum, corrugator supercilii, and the frontalis are but slightly affected, the labio-nasal fold is flattened or obliterated, and the corner of the mouth is depressed. The tongue when protruded deviates toward the paralyzed side, and the palate may hang lower than normal and turned toward either side. In consequence of the paralysis of the expression muscles, many movements become awkward or impossible, as whistling, pursing up the mouth, laughing, etc. The muscles of the chest are paretic, and respiration somewhat hindered thereby (Nothnagel). The extensors seem to be more affected than the flexors, but this is only apparent, because of the greater power of the latter. Notwithstanding the immense preponderance of cases proving the crossing of the motor fibers, and consequently the occurrence of hemiplegia on the side opposite the seat of the lesions of the brain, there are opposing observations. Bilateral paralysis may be due to simultaneous lesions on both sides, and in this way bilateral hemiplegia may be produced. Paralyses are said to be "alternating" or "crossed" when the paralysis of the face is on one side and of the extremities on the other. This may occur in lesions of the pons, etc. Although the paralyzed parts may be motionless, they may execute "associated movements": thus, in coughing or sneezing the paralyzed member may give a jerk, or may imitate movements performed by the healthy side. The contractions which accompany the hæmorrhage, or which are excited by an inflammatory process about the site of the clot in a few days after the seizure (early rigidity), have already been referred to. The contraction which occurs later, after the paralysis has existed for a long time, is known as "late rigidity," but its intensity and persistence bear no constant relation to the character of the case, except its duration, and rigidity may not be present at all, although not often absent. Bouchard's explanation that the rigidity depends on the atrophic descending changes in the pyramidal tract continues to be the most generally accepted theory of this phenomenon, but it is not altogether satisfactory. Besides rigidity, long-paralyzed members may be affected by choreic movements, first described by Wier Mitchell and subsequently studied by Charcot, under the title "post-hemiplegic chorea," and now ascertained to be produced by changes in the motor centers on the opposite side. We have further to note that the paralyzed muscles preserve their electric excitability. Under some circumstances the