the great toe, the heel raised from the floor. If now the top of the knee be struck a smart blow with the edge of the hand, and some voluntary motion be given to the limb in this position, a rhythmical movement, clonic spasm—the ankle clonus—will then go on independently. This movement is due to clonic contractions of the gastrocnemius, resulting in alternate elevation and depression of the knee and heel. In certain diseases, as lateral sclerosis, the ankle clonus is produced without preparation. If the heel is held in the operator's left hand, while the right puts the foot into the position of dorsal flexion by pressure on the ball of the great toe, the ankle clonus will then appear on tapping smartly the top of the knee. The clonus consists in rhythmical contractions and relaxation of the muscle, and consequent elevation and depression of the toes.

ACUTE AND CHRONIC NERVOUS DISEASES.

CEREBRAL HYPERÆMIA.

Definition.—*Cerebral hyperæmia*, or cerebral congestion, is a malady characterized by an increase in the amount of blood in the brain. The hyperæmia may be arterial, or *active;* venous, or *passive.*

Causes.—Any condition diminishing the amount of arterial blood in other parts will divert a larger quantity to the cranial cavity: compression of the abdominal aorta, ligation of an important artery, are examples. The suppression of an habitual discharge of blood—as that of hæmorrhoids, for illustration—is alleged to produce the same effect. Cerebral congestion occurs in the cold stage of an ague, and is also produced by the application of cold to the surface of the body. Prolonged intellectual effort, insolation, or sunstroke, protracted wakefulness, over-indulgence in alcoholic beverages, and the use of such narcotics as belladonna, are supposed to induce congestion of the brain. Hypertrophy of the heart, fullness of the general vascular system, and general plethora, are also alleged to have this effect, but grave doubts may well exist on this point. Passive congestion is produced when there is an obstacle to the return of blood from the cranial cavity, as when the superior *vena cava* and the jugular are compressed by intra-thoracic or cervical tumors, or when the venous system is overfilled by mitral or tricuspid disease. Venous stasis is also caused by atheromatous degeneration of the arterial tunics, feebleness of the cardiac contractions, and lowered vascular tonus.

Pathological Anatomy.—There are no structural changes beyond

an increase in the amount of blood, the displacement of a corresponding amount of cerebro-spinal fluid, and mechanical compression of the cerebral matter. The veins of the dura mater are distended, but still more those of the pia mater and choroid plexus. The sinuses are also overfilled. The convolutions are somewhat flattened, and the perivascular lymph-spaces are closed by the approximation of their walls. On section, more blood than normal flows out of the divided vessels, and the *puncta vasculosa* are more numerous. If the hyperæmia is of long standing, or if repeated attacks have occurred, the changes are more pronounced. The veins enlarge and become varicose, and small arteries previously invisible come into permanent view, and aneurismal dilatations form on the arterioles. There may be minute extravasations and capillary hæmorrhages, the evidence of which is afforded in old cases by pigment deposits and blood-crystals in the lymph-spaces. Transudations of serum may occur in the subarachnoid spaces and in the ventricles, and also in the perivascular sheaths, whence it follows, in old cases, that permanent dilatation of these spaces may have occurred, producing the *état criblé.*

Symptoms.—There are three well-marked forms of cerebral hyperæmia—the *light*, the *severe*, and the *apoplectic* (Jaccoud). In the light form the onset is gradual, and among the first symptoms is headache, which is soon followed by characteristic signs: the headache is dull and heavy, with occasional sharp, lancinating pains, increased by motion or sudden shocks, or by light and sound; there is inaptitude for any mental effort, and the attempt to exercise the mind causes a sense of cerebral exhaustion; there is singing in the ears, with other subjective noises; the conjunctivæ are injected, the retina is sensitive to light, and there are flashes of light and moving objects before the eyes; the sleep is fitful and unrefreshing, and disturbed by dreams of a terrifying kind; vertigo occurs, and the muscular movements are uncertain and fatiguing; the sensations are disordered, and numbness and tingling are felt in the extremities; the stomach is uncertain, and nausea is often experienced; and the heart is exceedingly irritable, the pulse rising considerably with the least mental or physical effort or emotional excitement.* The *severe form* may develop out of the light, or it may come on without any prodromic symptoms. As compared with the light form, we find the headache is more intense; the special senses are more irritable and intolerant of light and sound; the mind more disturbed, ideation more confused, illusions and hallucinations occurring; the wakefulness more obstinate and complete; the motor functions more excited, the movements more irregular and uncertain, jactitations appearing; the sensory functions are more perverted; besides the headache, are neuralgic pains, especially in the

* Hammond, "Cerebral Hyperæmia," p. 48.

fifth, numbness and tingling being felt in the extremities; the vertigo is more decided, the upright position being maintained with difficulty, and all coördinated and combined acts being executed with difficulty; the action of the heart is more excited, the pulsations irregular and rapid, and the least effort sending the beat up many times; the head is more decidedly warm, the eyes more suffused, more deeply injected, the eyelids more swollen; the stomach is more disordered, and nausea and vomiting are excited by effort of the mind, or by attempt at close attention. The symptoms indicate the approach of acute maniacal excitement, or acute inflammation; but the mind, although occupied by illusions and hallucinations, is still able to correct them or reason correctly about them, and the febrile condition does not yet exist. The symptoms may subside in a day or two, and health be restored in a few days, or, the case unrelieved may then pass into the stage of depression; torpor succeeding to exalted activity, drowsiness to wakefulness, coma to delirium. In adults, convulsions rarely occur in the course of the severe form, but are usual in children.

In the apoplectiform variety of cerebral congestion, the patient may suddenly pass into unconsciousness, with the usual phenomena attending the apoplectic attack; there is complete muscular relaxation, involuntary evacuations may occur, but the reflex movements are not in abeyance, and in some minutes or hours the patient returns to consciousness, somewhat confused, however, and does not entirely recover for some days. Without losing consciousness, he may suffer confusion of mind, extreme vertigo, have defects of speech, or an entire loss of memory for words, numbness, tingling, and paresis of the members, nausea and vomiting, etc., also coming on suddenly, and disappearing after some hours and days without permanent disability. The symptoms belonging to the venous or passive form of hyperæmia are much less pronounced, although in some respects similar. There is headache, but a sensation of heaviness and dullness rather than acute pain; the eyelids are swollen and puffy, but the conjunctivæ are not injected; the superficial veins are full, but the scalp is cool; singing in the ears and impaired hearing are noted; vision is dull, and floating objects are seen before the eyes; the mental operations are dull and confused; somnolence passing into stupor, without continuous normal sleep, dreams, illusions, and sudden startings in the sleep, occur from time to time. On ophthalmoscopic examination, there are ascertained to be an enlargement of the retinal veins, more or less swelling of the optic disk, and vessels before invisible come into view. When the congestion of the brain is of the passive variety, the retinal veins are unduly enlarged and tortuous. Observations on the drum membrane disclose increased vascularity of this organ, which has intimate connection with the intra-cranial circulation. The superficial temperature of the head is elevated in active hyperæmia, but is not affected in the passive form. Surface thermometers and Lombard's thermo-electric pile are employed to ascertain the temperature of the scalp. In any case there will be but slight rise of the thermometer; hence, any considerable elevation should awaken suspicion of inflammatory action.

Course, Duration, and Termination.—The light form may terminate in a few hours or days, under appropriate treatment, to recur from time to time, it may be; or it may continue with fluctuations in the severity of the symptoms for months and years. A cure readily results, if the causes cease to operate and the right management is instituted. If the hyperæmia continue, other morbid conditions will arise out of it. The severe form has a variable duration. A cure may be effected if right treatment is instituted early enough, but structural alterations will not be long delayed, and mental derangement will occur at an early period, or a cerebral hæmorrhage may take place. The apoplectiform variety may terminate in health or in cerebral hæmorrhage, according to the method pursued and the nature of the causes. Attacks of this nature may precede cerebral hæmorrhage, as the author has several times witnessed, but they are not often repeated until the hæmorrhage takes place. The passive form pursues the fortunes of the lesions causing it, and hence the duration is very variable and the course protracted.

Diagnosis.—The symptoms being due to disturbances of the intra-cranial circulation, the diagnosis rests on the absence of symptoms indicating structural lesions—notably the absence of fever, the widespread bilateral diffusion of the symptoms, and the fugitive character of the attacks. It may be confounded with delirium tremens, epilepsy, apoplexy, stomachal vertigo, etc. As respects delirium tremens, the distinction rests on the habits, the previous history, and the severity and persistence of the symptoms in this disease. The attack of epilepsy is preceded by a cry; then comé pallor of the face, stertorous breathing from tetanic fixation of the muscles of respiration, cyanosis, and general convulsions. Children with congestion of the brain may have such convulsions as a symptom, but the history preceding and succeeding is very different in the two maladies. The apoplectic form is distinguished from apoplexy by the persistence of the reflex movements, by the absence of conjugate deviation of the eyes, and by the early recovery without hemiplegia. Stomachal vertigo is preceded by attacks of indigestion, and is accompanied by the conditions of syncope and anæmia, instead of hyperæmia.

Treatment.—Causes of the hyperæmia should cease, if possible. If it be the active form, the head should be elevated and cold applied, the feet being immersed in hot mustard-water. To withdraw temporarily from the circulation some of the blood, a ligature should be applied around the thigh or thighs for a time, alternating the application of the ligature to prevent injury. Leeches may also be applied to the

mastoid process, or cups to the neck. In the apoplectiform variety venesection is advisable, as this is the most expeditious means of diminishing the intra-cranial blood-pressure. A brisk purgative is also an excellent expedient, relieving by acting as a derivative and by lessening vascular tension. The intra-cranial blood-pressure can also be lowered by the exhibition of veratrum viride, aconite, bromide of potassium, ergot, etc. These remedies are sufficient in the light form, but in the severe form a combination of the various means of treatment will be necessary. The treatment of the passive form is a part of the treatment required in the condition producing the hyperæmia, and need not now be discussed. The strictest attention must be paid to the diet and mode of life. An abstemious life—the diet consisting of fruit and vegetables chiefly—and early hours and the avoidance of all forms of excitement have prolonged life for many years, when an early demise was threatened by cerebral hyperæmia. Especially should alcoholic stimulants and the powerful emotions excited by speculations of all kinds be avoided. Such mild stimulants as tea and coffee even should be abandoned. In making these suggestions the author wishes his readers to note that he regards protracted rest to the mind as often injurious, and that light mental occupation is preferable to an entire disuse of the faculties.

CEREBRAL ANÆMIA.

Definition.—By *cerebral anæmia* is meant a lessened amount of blood in the brain. It may be *general* or *partial:* in the former the diminished supply of blood affects the whole organ; in the latter a particular district is deprived of its blood by the occlusion of a vessel. It is the general form of cerebral anæmia to be considered here.

Causes.—The most perfect type of cerebral anæmia is that produced by large loss of blood. Our knowledge of this condition has been rendered the more accurate by the experimental study of the subject in animals.* The effects of loss of blood on the functions of the brain are seen after severe hæmorrhage, as *post-partum* hæmorrhage, unavoidable hæmorrhage, menorrhagia, metrorrhagia, etc. Chronic wasting diseases, by the constant losses of nutrient material, induce cerebral anæmia. Phthisis, chronic dysentery, suppuration, and prolonged lactation, belong to this category. Maladies which impair the power to produce nutrient material, affecting the primary and secondary assimilation, will also cause anæmia of the brain. To this state as it occurs in infants was applied the term *hydrocephaloid* by Marshall Hall, who first demonstrated the important fact that a condition supposed to be due to inflammation was really the product of anæmia. Under the influence of shock, by powerful mental or moral emotions, a sudden

* Kussmaul and Tenner, "Sydenham Society's Translation."

contraction of the intra-cranial vessels occurs, and syncope, with loss of consciousness, ensues. Feebleness of the heart induces anæmia of the brain—a fact well exemplified in the sudden pallor and faintness experienced by convalescents on rising up after long decubitus; also in the case of those who suffer from weak heart, fatty heart, or obstruction at the aortic orifice, etc.

Pathological Anatomy.—The morbid changes are very simple. The amount of blood is below the normal, and the vessels are less full. The appearance of the brain is pale and exsanguine, and on transverse section of the hemispheres there are no bloody points. The subarachnoid spaces and the ventricles contain a good deal of fluid, and the perivascular lymph-spaces are also well filled with fluid, for, as the vessels contain less blood, the cerebro-spinal fluid increases; while in hyperæmia the distention of the vessels forces the fluid out, closes the lymph-spaces, and flattens the convolutions. The opposite state obtains in anæmia: the brain is pale, white, and moist; the vessels small, the lymph-spaces large. In partial anæmia, other factors are concerned, and hence the local conditions differ.

Symptoms.—There are two distinct forms: *acute*, or sudden; *chronic*, or light. Venesection *ad deliquum animi* furnishes a complete picture of the first: the face grows deadly pale, the lips white, the pupils dilate, the action of the heart becomes very feeble, the pulse small, a cold sweat breaks out over the body, ringing noises sound in the ears, surrounding objects appear dim, and a mist gathers before the eyes; voices are heard in the distance, and the words are unintelligible, everything fades suddenly out of consciousness, and the patient falls as if lifeless, respiration having ceased, and the heat-beart scarcely continuing. There is complete muscular resolution, but in an instant the eyelids begin to tremble, the muscles of the lips and face twitch, and a general convulsion follows. The syncope, which is merely a fainting-fit, does not proceed any further than suspension of consciousness, and in a short time the respiration begins, the heart-beat grows stronger, the patient opens his eyes, looks around with a dazed expression, and asks what has happened; he tries to get up, and finds himself very weak, but in a short time the bodily vigor is entirely restored. The convulsions of cerebral anæmia are due to two factors: to an abnormal excitability of the "spasm-center"; to the circulation of black blood through this spasm-center. In the slow, habitual, or chronic anæmia, the condition is that of depression of function. The brain, inadequately supplied with nutrient material, functionates imperfectly; the special senses are both irritable and depressed—the sight is dull (amblyopia), and light is painful to the eyes; hearing is obtuse, there are subjective noises in the ears, *tinnitus*, etc., and loud sounds are distressing; the mental operations are slow and confused, and there may be illusions, hallucinations, maniacal excitement, etc. (puerperal

mania, insanity of lactation, etc.) ; muscular movements are excited, or depressed and feeble, tremulous or incoördinate ; the sensory functions are similarly affected—there may be excitement or depression, neuralgic pains, numbness, prickling, tingling, or anæsthesia ; vertigo is nearly always present, and consequent uncertainty of movements ; headache is also commonly present, and may be a sense of heaviness or oppression, or, more frequently, acute pain ; exertion causes great fatigue, and syncopal attacks are easily induced ; the action of the heart is weak, and rapid action is excited by the least movement ; and the sense of faintness is usually accompanied by nausea. In the form of cerebral anæmia, known as hydrocephaloid, the child is exhausted by a wasting malady ; its surface is cool, skin pale, the pulse quick and weak, the eyes are half closed, sunken, and surrounded by broad, dark areolæ, the fontanelle is concave, the head cool ; there is much fretfulness, although there is a somnolent state ; the stomach is irritable, the bowels relaxed.

Course, Duration, and Termination.—The acute form, so far as the immediate attack is concerned, lasts a few minutes only, but this is merely a symptom of a long-established anæmia of the brain. The chronic form has an indefinite duration, and pursues a varying course according to the management and the nature of the causes. The termination is usually in restoration to the normal state, if the treatment be suitable. So important are the changes in the vessel-walls in anæmia, that we should not overlook the gravity of any case that has continued a long time. Furthermore, as various intercurrent maladies may develop, prognostic opinions should be expressed with caution if the anæmia has persisted.

Diagnosis.—As cerebral hyperæmia presents many symptoms in common with cerebral anæmia, the diagnosis of these affections may be confused, but attention to a few points ought to conduct to right conclusions. The history of the causes, the appearances of anæmia, and the depression of the circulation, will indicate the nature of the case. The use of the surface thermometer, or thermo-electric pile, to ascertain the temperature of the scalp, is necessary, for in anæmia the temperature is rather below than above normal, but in hyperæmia the opposite condition obtains. Ophthalmoscopic inspection of the retina and otoscopic inspection of the drum membrane should be made, to ascertain the character of the circulation : in hyperæmia the retinal vessels are abnormally full and the drum is red and injected, whereas in anæmia the retina and drum membrane are pale and comparatively bloodless.

Treatment.—The recumbent posture and stimulation of the nares with ammonia are the only measures necessary in the treatment of syncope. When alarming depression is due to hæmorrhage, besides the measures necessary to stop the loss of blood, anæmia of the brain

is to be overcome by depression of the head and elevation of the limbs, by the administration of alcoholic stimulants, by the subcutaneous injection of stimulants, by the intravenous injection of ammonia, and by transfusion. The chronic form of cerebral anæmia is to be arrested by stopping the sources of waste, by the use of iron and the phosphates, and by judicious alimentation. The best results are obtained by the administration of a stimulant to the cerobro-spinal axis (strychnine) and a chalybeate tonic. Arsenic is often highly serviceable in cerebral anæmia, in combination with iron. For the maniacal delirium of cerebral anæmia, the hypodermatic injection of morphine is of the greatest value. When there is associated with this delirium a high degree of motor excitement, atropine or duboisine should be combined with the morphine.

OCCLUSION OF THE CEREBRAL VESSELS.

Definition.—Under this term are included all lesions which occlude or block the vessels, thus causing anæmia of some part or parts of the brain. The occlusion may form in a cerebral vessel, or may be produced by an embolism conveyed thither from any part of the vascular system. Under this term must be comprised the remote as well as the immediate results of occlusion.

Causes.—The factors chiefly concerned in the occlusion of intracranial vessels are thrombosis and embolism. Chronic endarteritis and slowing with weakening of the blood-current are the causes of thrombosis. The changes in the arterial tunics consist in atheromatous and calcareous degeneration ; the lumen of the vessel is gradually narrowed by the deposition of new material, and the intima is roughened. The propulsion of the blood is hindered by weakness of the heart's action, and by diminished elasticity of the walls of the arteries, due to the atheromatous changes in the tunics. When the disease in the walls of a cerebral vessel reaches a certain point, coagulation of the blood takes place and an occlusion (*autochthonous thrombosis*) is thus effected. The formation of a thrombus is also favored by the condition of the blood itself. In chronic wasting diseases, the relative proportion of fibrin in the blood being much increased, coagulation is promoted accordingly. An autochthonous thrombus may form in a vessel whose lumen had been obstructed by the pressure of a tumor.

Emboli consist of bits of fibrin, exudations, or concretions, which, formed at some distant point and carried into the circulation, are deposited in the brain. The most usual source of emboli is endocarditis, either of the ulcerative variety or of the chronic form with its polyp-like excrescences, or fibrin vegetations. According to the observations of Bertin, the emboli come from the left auricle, four times ; from the left ventricle, twelve times ; from the aortic valves, ten

times; from the mitral, twenty-four times. These figures agree with the usual experience on this point. Cardiac emboli are also produced in the following way: clots form, especially in the auricle, when the heart is weakened by myocarditis, fatty degeneration, uncompensated valvular lesions, and such chronic wasting diseases as cancer and tuberculosis. Such clots, subsequently pulverized by the cardiac movements, are carried into the circulation. Emboli may also be derived from aortic aneurism, from syphiloma of the great vessels, etc.

Pathological Anatomy.—Owing to its position at or near the summit of the arch of the aorta, the blood-current from the aortic orifice is directed to the left common carotid, so that an embolus loosened from the heart naturally enters this vessel, and its prolongation within the cranium, the Sylvian artery. It necessarily follows from this that the left side is usually obstructed. It rarely happens that an embolus enters the vertebral arteries. Sometimes the embolisms are multiple, and enter the vessels on both sides, or are lodged in different places on the left side. As certain vessels are usually occluded, it is important to have a clear understanding of the parts supplied by them. The left Sylvian artery sends branches to the second and third frontal convolutions, the anterior and superior portions of the three temporal convolutions, the island of Reil, the parietal convolutions, part of the external and all of the internal capsule, the lenticular nucleus, and most of the corpus striatum. It is important to note, further, that the vessels of this part of the brain have the arrangement of Cohnheim's terminal arteries—arteries without anastomoses—while the vessels of the gray matter of the hemispheres, or the cortex, communicate freely with each other.* When an artery of the "basal system" is obstructed, either by a thrombus or embolism, an anæmia of the territory supplied by the vessel at once ensues—either a simple anæmia and white softening, or anæmia followed by collateral hyperæmia and œdema. The simple anæmia and white or yellowish-white softening occur when the blood in the whole extent of the occluded vessel coagulating, prevents the backward flow of blood through the capillaries, and consequently the collateral hyperæmia and œdema. The anæmic tissue dies or undergoes *necrobiosis* in consequence of the loss of its entire nutritive supply. The nerve-tissue elements become disassociated, break up into a diffluent granular mass, and are crowded with fat-cells, whence the color of the softened tissues assumes a somewhat yellowish aspect. Yellow softening is also a stage of the next form. When a terminal artery is occluded, and all parts of the vessel beyond the seat of obstruction remain pervious, blood flows back through the capillaries from the nearest artery and vein, until the previously anæmic and bloodless district is deeply engorged. Changes now occur in the walls

* The reader should peruse in this connection the articles on "Arteritis" and on "Thrombosis and Embolism."

of the vessels, permitting diapedesis of the red blood-globules. As, in the process of softening and disintegration which now ensues, the tissues are colored by the red corpuscles, the appearances are entitled "red softening." Minute extravasations occur here and there, from rupture of capillaries, and hence, in the midst of a uniform red there will be seen the dark points of "capillary apoplexy." These extravasations may be so numerous as to present the appearance of a cerebral hæmorrhage. In from two to four weeks the red softening becomes yellow softening in consequence of the transformation of the hæmoglobulin and the fatty degeneration of the nerve-elements. The softening proceeding to another stage becomes "white softening," when there is a milky, or rather creamy fluid, containing, mixed with it, masses or particles of broken-down nerve-elements. There is no abrupt line of demarkation, but the diseased part shades off into the surrounding healthy part by a fine gradation.

Symptoms.—There are two well-defined modes of onset: the gradual, which occurs to thrombosis; the sudden, or apoplectic, due to embolism. The first form, or thrombosis, is a malady of the old; the second form, or embolism, may occur at any period, frequently in the young. As, when chronic arteritis of the cerebral vessels exists, a number of them may be diseased at the same time, the resulting symptoms must necessarily be widely diffused, and, as the disease has proceeded to different stages at different points, there may be present, at the same time, the symptoms of excitation and depression of function. Headache, more or less persistent, and of variable intensity, is the earliest symptom; next, alterations of character become evident—the individual grows irritable, morose, and despondent, his mind is easily fatigued, and memory is impaired; at first names, then some unusual word, ultimately most words, are forgotten. Occasionally the only mental defect observed is loss of the memory for words—amnesia of verbal language—which may occur slowly or suddenly, with or without something of a stroke. After the headache, vertigo comes on, and may be occasional and caused by a change of posture, or it may be constant when sitting up and when recumbent. Difficulty of locomotion is experienced, in consequence partly of the vertigo, but chiefly because of weakness of a group of muscles or of a member; more or less of senile trembling may be present, or the trembling of muscular weakness; and the movements of the tongue may be imperfect and speech hesitating and mumbling. There are two causes for the symptoms just detailed—gradual encroachment on the lumen of diseased vessels, whence the blood-stream is lessened, and interference with the nutrition of the brain by reason of calcareous degeneration of the capillaries. The next point in the morbid complexus is the occurrence of a sudden attack, which may or may not be apoplectic. If apoplectic, the patient falls suddenly into a condition of insensibility, with