nerves affected, the disease is unrecognizable, since identical symptoms may be produced by intense engorgement of the meninges. In typhoid fever, in which meningitis is very rare, the twitchings, spasms, and retractions of the neck are almost invariably associated with cerebro-spinal congestion, not with meningitis.

A knowledge of the etiology gives a very important clew. Thus, in middle-ear disease the development of high fever, delirium, vomiting, convulsions, and retraction of the head and neck would be extremely suggestive of meningitis or abscess. Headache, which may be severe and continuous, is the most common symptom. In the fevers, particularly in pneumonia, there may be no complaint of headache. Delirium is frequently early, and is most marked when the fever is high. Convulsions are less common in simple than in tuberculous meningitis. They were not present in a single instance in the cases which I have seen in pneumonia, ulcerative endocarditis, or septicæmia. In the simple meningitis of children they may occur. Rigidity and spasm or twitchings of the muscles are more common. Stiffness and retraction of the muscles of the neck are important symptoms; but they are by no means constant, and are most frequent when the inflammation extends to the meninges of the cervical cord. Vomiting is a common symptom in the early stages, particularly in basilar meningitis. Constipation is usually present. Optic neuritis is rare in the meningitis of the cortex, but is not uncommon when the base is involved.

Important symptoms are due to lesions of the nerves at the base. Strabismus or ptosis may occur. The facial nerve may be involved, producing slight paralysis, or there may be damage to the fifth nerve, producing anæsthesia and, if the Gasserian ganglion is affected, trophic changes in the cornea. The pupils are at first contracted, subsequently dilated, and perhaps unequal.

Fever is present, moderate in grade, rarely rising above 103°. In the non-tuberculous leptomeningitis of debilitated children and in Bright's disease there may be little or no fever. The pulse may be increased in frequency at first and subsequently is slow and irregular.

**Treatment.**—There are no remedies which in any way control the course of acute meningitis. An ice-bag should be applied to the head and, if the subject is young and full-blooded, general or local depletion may be practised. Absolute rest and quiet should be enjoined. When disease of the ear is present, a surgeon should be early called in consultation, and if there are symptoms of meningo-encephalitis which can in any way be localized trephining should be practised. An occasional saline purge will do more to relieve the congestion than blisters and local depletion. I have no belief whatever in the efficacy of counter-irritation to the back of the neck, and to apply a blister to a patient suffering with agonizing headache in meningitis is needlessly to add to the suffering. If counter-irritation is deemed essential, the thermo-cautery, lightly applied, is more satisfactory, because the pain inflicted is transient.



The gastro-intestinal symptoms should receive appropriate treatment. Gowers states that in two instances of septic meningitis which recovered the good effects seemed to be due to large doses of the perchloride of iron. Iodide of potassium and mercury are recommended by some authors.

The application of an ice-cap, attention to the bowels and stomach, and keeping the fever at a moderate height by sponging, are the necessary measures in a disease recognized as almost invariably fatal, and in which the cases of recovery are extremely doubtful.

(*b*) *Chronic Leptomeningitis.*—This is rarely seen apart from syphilis or tuberculosis, in which the meningitis is associated with the growth of the granulomata in the meninges and about the vessels. The symptoms in such cases are extremely variable, depending entirely upon the situation of the growth. They may closely resemble those of tumor and be associated with localized convulsions. The leptomeningitis infantum may be chronic. In the cases reported by Gee and Barlow the duration in some instances extended even to a year and a half. The involvement of the posterior part of the meninges and of the ventricles may lead to dilatation and hydrocephalus. The symptoms upon which these authors lay stress are convulsions, and retraction of the head, which is particularly marked when the child is made to sit up. There may be rigidity of the limbs and epileptiform convulsions.

## II. AFFECTIONS OF THE BLOOD-VESSELS.

### Hyperæmia.

Congestion of the brain has played an important part in cerebral pathology. Undoubtedly there are great variations in the amount of blood in the cerebral vessels; this is universally conceded, but how far these changes are associated with a definite group of symptoms is not quite so clear. The hyperæmia may be either active or passive.

*Active hyperæmia* is associated with febrile conditions, with increased action of the heart, chilling of the surface, contraction of the superficial vessels, and with the suppression of certain customary discharges. Among other recognized causes are plethora, functional irritation, such as is associated with excessive brain work, and the action of certain substances, such as alcohol and nitrite of amyl.

*Passive hyperæmia* results from obstruction in the cerebral sinuses and veins, engorgement in the lesser circulation, as in mitral stenosis, emphysema, from pressure on the superior cava by aneurisms and tumors, and in the venous engorgement which takes place in prolonged straining

efforts. In its most intense form it is seen in the compression of the superior cava by tumors and in death from strangulation.

The anatomical changes in congestion of the brain are by no means striking. Active hyperæmia is never visible post mortem. The veins of the cortex are distended, the gray matter has a deeper color, and its vessels are full. The arteries at the base and in the Sylvian fissures contain blood. Nothing, however, can be more uncertain or indefinite than the post-mortem appearances of hyperæmia of the brain. The most intense distention of the vessels is seen in early death during the specific fevers, or in the secondary passive congestion due to obstruction in the superior cava or in the lesser circulation.

**Symptoms.**—There are no characteristic or constant features of cerebral hyperæmia. It may exist in the most extreme grade without the slightest disturbance of the cerebral functions, as is witnessed frequently in the pressure of tumors on the superior vena cava. How far the headache and delirium of the early stage of the infectious fevers is to be assigned to hyperæmia of the blood-vessels of the brain it is not easy to determine. The headache, dizziness, and unpleasant sensations in aortic insufficiency and in some instances of hypertrophy of the heart may be due to the cerebral congestion.

As a separate clinical entity, congestion of the brain rarely comes under observation. I have no knowledge of instances associated with delirium, fever, insomnia, and convulsions, or of the so-called apoplectiform variety described by some writers. Very plethoric persons are subject to attacks of headache with flushing of the face and irritability of temper, attacks which may recur frequently and are sometimes relieved by bleeding at the nose. These are usually attributed to congestion of the brain. When passive hyperæmia reaches a high grade, there may be torpor, dulness of the intellect, and ultimately deep coma.

### Anæmia.

This may be induced by loss of blood, either quickly, as in hæmorrhage, or gradually, as in the severe primary and secondary anæmias. The anæmia may be local and due to causes which interfere with the blood supply to the brain, as narrowing of the vessels by endarteritis, pressure, narrowing of the aortic orifice, or it may follow an unequal distribution of the blood in consequence of dilatation of certain vascular territories. Thus, rapid distention of the intestinal vessels, such as occurs after the removal of ascitic fluid, may cause sudden death from cerebral anæmia. The commonest illustration of this is the fainting fit from emotion, in which the blood supply to the brain is insufficient on account of the diminished arterial pressure. Anæmia of the cerebral vessels may be caused by pressure of fluid in the ventricles. The partial anæmia results from obliteration of branches of the circle of Willis by embolism or thrombosis. Ligature of one carotid sometimes causes a transient marked anæmia and disturbance of function on one side of the brain.



The anatomical condition of the brain in anæmia is very striking. The membranes are pale, only the large veins are full, the small vessels over the gyri are empty, and an unusual amount of cerebro-spinal fluid is present. On section both the gray and white matter look extremely pale and the cut surface is moist. Very few *puncta vasculosa* are seen.

**Symptoms.**—The effects of anæmia of the brain are well illustrated by a fainting fit in which loss of consciousness follows the heart weakness. When the result of hæmorrhage, there are drowsiness, giddiness, inability to stand, flashes of light, and noises in the ear; the respiration becomes hurried; the skin is cool and covered with sweat; and gradually, if the bleeding continues, consciousness is lost and death may occur with convulsions. In ordinary syncope the loss of consciousness is usually transient and the recumbent posture alone may suffice to restore the patient to consciousness. In the more chronic forms of brain anæmia, such as result from the gradual impoverishment of the blood, as in protracted illness or in starvation, the condition known as irritable weakness results. Mental effort is difficult, the slightest irritation is followed by undue excitement, the patient complains of giddiness and noises in the ears, or there may be hallucinations or delirium. These symptoms are met with in an extreme grade as a result of prolonged starvation.

An interesting set of symptoms, to which the term *hydrocephaloid* was applied by Marshall Hall, occurs in the debility produced by prolonged diarrhœa in children. The child is in a semi-comatose condition with the eyes open, the pupils contracted, and the fontanelle depressed. In the earlier period there may be convulsions. The coma may gradually deepen, the pupils become dilated, and there may be strabismus and even retraction of the head, symptoms which closely simulate basilar meningitis.

### Œdema of the Brain.

In the pathology of brain lesions œdema formerly played a *rôle* almost equal in importance to congestion. It occurs under the following conditions: In general atrophy of the convolutions, in which case the œdema is represented by an increase in the cerebro-spinal fluid and in that of the meshes of the pia. In extreme hyperæmia from obstruction, as in mitral stenosis or in tumors, there may be a condition of congestive œdema, in which, in addition to great filling of the blood-vessels, the substance of the brain itself is unusually moist. The most acute œdema is a local process found around tumors and abscesses. An intense infiltration, local or general, may occur in Bright's disease, and to it, as Traube suggested, certain of the uræmic symptoms may be due.

The *anatomical changes* are not unlike those of anæmia. When a sequence of progressive atrophy, the fluid is chiefly within and beneath

the membranes. The brain substance is anæmic and moist, and has a wet, glistening appearance, which is very characteristic. In some instances the œdema is more intense and local and the brain substance may look infiltrated with fluid. The amount of fluid in the ventricles is usually increased.

The *symptoms* are in great part those of anæmia, and are not well defined. As just stated, some of the cerebral features of uræmia may depend upon it. Of late years cases have been reported by Raymond, Tenneson, and Dercum, in which unilateral convulsions or paralysis have occurred in connection with chronic Bright's disease, and in which the condition appeared to be associated with œdema of the brain. The older writers laid great stress upon an apoplexia serosa, which may really have been a general œdema of the brain.

## Cerebral Hæmorrhage.

The bleeding may come from branches of either of the two great groups of cerebral vessels—the *basal*, comprising the circle of Willis and the central arteries passing from it, or the *cortical group*, the anterior, middle, and the posterior cerebral vessels. In a majority of the cases the hæmorrhage is from the central branches, more particularly from those given off by the middle cerebral arteries in the anterior perforated spaces, and which supply the corpora striata and internal capsules. One of the largest of these branches which passes to the third division of the lenticular nucleus and to the hinder part of the internal capsule is so frequently involved in hæmorrhage that it has been called by Charcot *the artery of cerebral hæmorrhage*. The bleeding may be into the substance of the brain, to which alone the term cerebral apoplexy is applied, or into the membranes, in which case it is termed meningeal hæmorrhage; both, however, are usually included under the terms intracranial or cerebral hæmorrhage.

**Etiology.**—The conditions which produce lesions of the blood-vessels play a very important part; thus the natural tendency to degeneration of the vessels in advanced life makes apoplexy much more common after the fiftieth year. It may, however, occur in children under ten. On account of the greater liability to arterial disease (associated probably with muscular exertion and the abuse of alcohol), men are more subject to cerebral hæmorrhage than women. Heredity was formerly thought to be an important factor in this affection, and the apoplectic *habitus* or build is still referred to. By this is meant a stout, plethoric body of medium size, with a short neck. Heredity influences cerebral hæmorrhage entirely through the arteries, and there are families in which they degenerate early, usually in association with renal changes. The secondary hypertrophy of the heart brings with it serious dangers, which have already been discussed in the section upon arteries. The three special factors in



inducing arterio-sclerosis—the abuse of alcohol, syphilis, and prolonged muscular exertion—are found to be important antecedents in a large number of cases of cerebral hæmorrhage.

The endocarditis of rheumatism and other fevers may indirectly lead to apoplexy by causing embolism and aneurism of the vessels of the brain. Cerebral hæmorrhage occurs occasionally in the specific fevers and in profound alterations of the blood, as in leukæmia and pernicious anæmia. The actual exciting cause of the hæmorrhage is not evident in the majority of cases. The attack may be sudden and without any preliminary symptoms. In other instances violent exertion, particularly straining efforts or, the excited action of the heart in emotion may cause a rupture.

**Morbid Anatomy.**—The lesions causing apoplexy are almost invariably in the cerebral arteries, in which the following changes may lead directly to it:

(*a*) Periarteritis with the production of miliary aneurisms, rupture of which is the most common cause of cerebral hæmorrhage. They occur most frequently on the central arteries, but also on the smaller branches of the cortical vessels. On section of the brain substance they may be seen as localized, small dark bodies about the size of a pin's head. Sometimes they are seen in numbers upon the arteries carefully withdrawn from the anterior perforated spaces. According to Charcot and Bouchard, who have described them, they are most frequent in the central ganglia. In apoplexy after the fortieth year if sought for they are rarely missed.

(*b*) Aneurism of the branches of the circle of Willis. These are by no means uncommon, and will be considered subsequently.

(*c*) Endarteritis and periarteritis in the cerebral vessels most commonly lead to apoplexy by the production of aneurisms, either miliary or coarse. There are instances in which the most careful search fails to reveal anything but diffuse degeneration of the cerebral vessels, particularly of the smaller branches; so that we must conclude that spontaneous rupture may occur without the previous formation of aneurism.

The hæmorrhage may be meningeal, cerebral, or intraventricular.

*Meningeal Hæmorrhage* may be outside the dura, between this membrane and the bone, or between the dura and arachnoid, or between the arachnoid and the pia mater. The following are the chief causes of this form of hæmorrhage: Fracture of the skull, in which case the blood usually comes from the lacerated meningeal vessels, sometimes from the torn sinuses. In these cases the blood is usually outside the dura or between it and the arachnoid. The next most frequent cause is rupture of aneurisms on the larger cerebral vessels. The blood is usually subarachnoid. An intracerebral hæmorrhage may burst into the meninges. A special form of meningeal hæmorrhage is found in the new-born, associated with injury during birth. And lastly, meningeal hæmorrhage may occur in the constitutional diseases and fevers. The blood may be in a large quantity at

the base; in cases of ruptured aneurism, particularly, it may extend into the cord or upon the cortex. Owing to the greater frequency of the aneurisms in the middle cerebral vessels, the Sylvian fissures are often distended with blood.

*Intracerebral hæmorrhage* is most frequent in the neighborhood of the corpus striatum, particularly toward the outer section of the lenticular nucleus. The hæmorrhage may be small and limited to the lenticular body and the internal capsule, or it may break the centrum ovale, or burst into the lateral ventricle, or extend to the insula. Hæmorrhages confined to the white matter—the centrum ovale—are rare. Localized bleeding may occur in the crura or in the pons. Hæmorrhage into the cerebellum is not uncommon, and usually comes from the superior cerebellar artery. The extravasation may be limited to the substance or rupture into the fourth ventricle. Twice I have known sudden death in girls under twenty-five to be due to cerebellar hæmorrhage.

*Ventricular Hæmorrhage.*—This rarely comes from the vessels of the plexuses or of the walls. It is not infrequent in early life and may occur during birth. Of 94 cases collected by Edward Sanders, 7 occurred during the first year, and 14 under the twentieth year. In the cases which I have seen in adults it has almost always been caused by rupture of a hæmorrhage in the neighborhood of the caudate nucleus. The blood may be found in one ventricle only, but more commonly it is in both lateral ventricles, and may pass into the third ventricle and through the aqueduct of Sylvius into the fourth ventricle, forming a complete mould in blood of the ventricular system.

*Subsequent Changes.*—The blood gradually changes in color, and ultimately the hæmoglobin is converted into the reddish-brown hæmatoidin. Inflammation occurs about the apoplectic area, limiting and confining it, and ultimately a definite wall may be produced, inclosing a cyst with fluid contents. In other instances a cyst is not formed, but the connective-tissue proliferates and leaves a pigmented scar. In meningeal hæmorrhage the effused blood may be gradually absorbed and leave only a staining of the membranes. In other cases, particularly in infants, when the effusion is cortical and abundant, there may be localized wasting of the convolutions and the production of a cyst in the meninges. Possibly certain of the cases of porencephaly are caused in this way.

Secondary degeneration follows when the motor cortex or motor path is involved. Thus, in persons dying some years after a cerebral apoplexy which has produced hemiplegia, the degeneration may be traced in the crus, in the anterior part of the pons, in the pyramidal fibres of the medulla, in the direct fibres of the cord of the same side, and in the crossed pyramidal fibres of the opposite side (Fig. 3).

**Symptoms.**—These may be divided into primary, or those connected with the onset, and secondary, or those which develop later after the early manifestations have passed away.



*Primary Symptoms.*—Premonitory indications are rare. As a rule, the patient is seized while in full health or about the performance of some every-day action, occasionally an action requiring strain or extra exertion. Now and then instances are found in which there are sensations of numbness or tingling or pains in the limbs, or even choreiform movements in the muscles of the opposite side, the so-called prehemiplegic chorea. The onset of the apoplexy, as cerebral hæmorrhage is usually called, varies greatly. There may be sudden loss of consciousness and complete relaxation of the extremities. In such instances the name *apoplectic stroke* is particularly appropriate. In other cases the onset is more gradual and the loss of consciousness may not occur for a few minutes after the patient has fallen, or after the paralysis of the limbs is manifest. In the apoplectic attack the condition is as follows: There is deep unconsciousness; the patient cannot be roused. The face is injected, sometimes cyanotic, or of an ashen-gray hue. The pupils vary; usually they are dilated and inactive. The respirations are slow, noisy, and accompanied with stertor. Sometimes the Cheyne-Stokes rhythm may be present. The pulse is usually full, slow, and of increased tension. The temperature may be normal, but is often found subnormal, and, as in a case reported by Bastian, may sink below 95°. In cases of basal hæmorrhage the temperature, on the other hand, may be high. The urine and fæces are usually passed involuntarily. Convulsions are not common. It may be difficult to decide whether the condition is apoplexy associated with hemiplegia or sudden coma from other causes. An indication of hemiplegia may be discovered in the difference in the tonus of the muscles on the two sides. If the arm or the leg is lifted, it drops "dead" on the affected side, while on the other it falls more slowly. Rigidity also may be present. In watching the movements of the facial muscles in the stertorous respiration it will be seen that on the paralyzed side the relaxation permits the cheek to be blown out in a more marked manner. The head and eyes may be turned strongly to one side—conjugate deviation.

In other cases, in which the onset is not so abrupt, the patient may not lose consciousness, but in the course of a few hours there is loss of power, unconsciousness gradually develops, and deepens into profound coma. This is sometimes termed ingravescent apoplexy. The attack may occur during sleep. The patient may be found unconscious, or wakes to find that the power is lost on one side. Small hæmorrhages may cause hemiplegia without loss of consciousness, more particularly when they are in the territory of the central arteries.

Usually within forty-eight hours after the onset of an attack there is febrile reaction, and more or less constitutional disturbance associated with inflammatory changes about the hæmorrhage. The patient may die in this reaction, or, if consciousness has been regained, there may be delirium or recurrence of the coma. At this period the so-called early rigidity may develop in the paralyzed limbs. Trophic changes may occur,