tion of the optic fibres is total. In dogs, cats, monkeys, and man, in whom, to a less or greater degree, the eyes are directed in parallel lines, the decussation is partial. In a rabbit or horse hemianopsia is impossible. In the other animals named it is observed.

Unilateral hemianopsia is produced by lesions affecting the optic chiasm only. If the lesion lies on the outer side of the chiasm it produces nasal hemianopsia on the side of the lesion. Thus a tumor pressing upon point *N* in Fig. 2599 would produce blindness in the temporal half of the left retina, and hence nasal hemianopsia in the left visual field. If the tumor lies in front of or behind the chiasm, or presses upon it from above or below in such a manner as to involve the decussating strand of fibres either before or after they have crossed to the opposite side, it will cause a temporal hemianopsia (*T* in Fig. 2599). As it is impossible to determine whether the fibres are affected before or after decussation, no statement as to the side upon which the lesion lies can be made.

Bilateral hemianopsia is far more common than unilateral. It may be of several varieties. Bilateral nasal hemianopsia implies a destruction of the direct optic fibres on both sides, the decussating fibres being unaffected. Such a case has been observed by Knapp, in which a tumor surrounded the chiasm, pressing upon its sides, but not affecting its centre. Bilateral temporal hemianopsia is a rare condition, and is produced only by a lesion which divides the chiasm through its antero-posterior diameter without affecting its lateral halves, thus destroying both decussating strands. It has been observed in a few cases of tumor of the hypophysis.

Bilateral homonymous hemianopsia is a not uncommon condition, and may be produced by many causes, and by lesions situated in many various parts. This will be better understood after the course of the optic tracts has been followed to their terminations in the visual area of the cerebral cortex (Fig. 2599), since a lesion at any point in this course will produce the symptom named. Each optic tract, after leaving the chiasm, passes around the crus cerebri, lying directly upon the fibres which pass through the foot of the crus (pes pedunculi), and ends on the level of the tegmentum of the crus in the external geniculate body, in the pulvinar of the optic thalamus (*i.e.*, the eminence forming its posterior surface), and in the corpora quadrigemina anteriora, which latter it reaches by the brachium conjunctivum. The last-named fibres of the optic tract have probably nothing to do with conscious vision, and may therefore be excluded from consideration in studying hemianopsia. They form the sensory part of a reflex arc, whose motor part is made up of the motor nerves to the eyeballs. The functions of this reflex mechanism are to direct the motion of the eyeballs, and to regulate the process of accommodation and the size of the pupil. The primary visual centres are, therefore, the external geniculate body and optic thalamus. The fibres of the optic tract end in the cells of these ganglia, and from these cells new fibres arise which collect in a large tract and issue from the posterior external angle of the optic thalamus into the posterior third of the internal capsule. This visual tract turns upward and backward in the internal capsule, radiates into the centrum semiovale of the occipital lobe, and, passing around the outer border of the posterior horn of the lateral ventricle, terminates in the convolutions of the occipital cortex, including the cuneus. At no point in this course is any decussation found. The only decussation of fibres between the eye and the cortex is in the optic chiasm.[1]

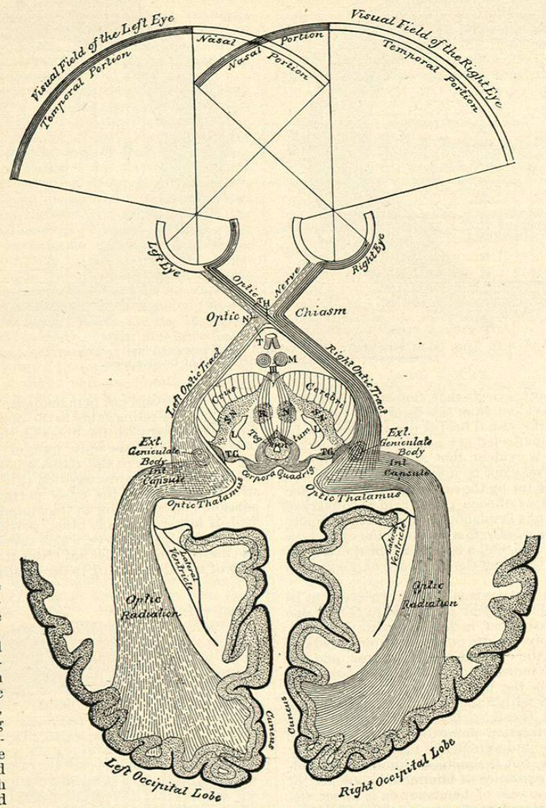

FIG. 2599.—The Visual Tract. The result of a lesion anywhere between the chiasm and cuneus is to cause homonymous hemianopsia. III. Third nerve; S. N., substantia nigra; R. N., red nucleus of tegmentum; L., lemniscus; T. G., fibres from optic tract to corpora quadrigemina.

Bilateral homonymous hemianopsia may be caused by a destructive lesion lying anywhere in the course of these fibres between the optic chiasm and the occipital cortex, or by a lesion in the cortex which destroys the perceptive centres in which the fibres end. Wherever the lesion, the character of the symptom will be the same. From the accompanying symptoms, it is in some cases possible to locate the lesion causing the hemianopsia. Thus, if the optic tract is involved as it curves around the crus cerebri, the same lesion which causes the hemianopsia will be likely to affect the motor tract in its passage through the pes, or to involve the third nerve in its exit from the pes. In this case hemianopsia and hemiplegia of one side, with oculo-motor paralysis of the other side, will be combined. Or, if the termination of the optic tract in the geniculate body and thalamus is involved the lesion, unless extremely limited, will affect the sensory tract from the opposite side of the body in its passage through the tegmentum of the crus, or in the posterior portion of the internal capsule; and in this case hemianæsthesia and hemiataxia will be associated with hemianopsia. Or, if the visual tract in its passage through the internal capsule is involved by disease in the basal ganglia or in the capsule, the proximity of both sensory and motor tracts in the capsule will render a combination of hemianopsia, hemianæsthesia, and hemiplegia quite probable. This is the seat of the lesion, and this is the combination of symptoms most frequently observed (see *Brain Diseases: Diagnosis of Local Lesions*).

Lesions in the centrum ovale involving the optic radiation may not produce any other symptom than that under discussion. If it is the left hemisphere, however, in which the disease is present, a condition of word-blindness (see *Aphasia*) is not infrequently associated with it; and in all the cases of word-blindness hitherto reported hemianopsia has been found. This is probably due to the destruction of association fibres between the occipital and temporal lobes which lie side by side with the visual tract.

Lesions in the cortex of the occipital lobe produce no other symptom than hemianopsia. It is impossible to limit the visual area of the brain to any one part of the lobe, since destruction of a portion of the convexity has produced the same result as destruction of its median surface.[2] Nor is it possible to project upon the cortex the retinal expansion, as has been attempted by Munk, since lesions in all parts of the occipital convolutions produce the same effect.

Lesions in the angular gyrus may produce hemianopsia, and it is well known that Ferrier has located in this convolution the visual centres. It is probable that such lesions have caused the symptom by affecting the visual tract as it passes beneath this gyrus in the centrum ovale. There is no reliable evidence that a unilateral lesion in the cortex can produce blindness of one eye alone.

Hemianopsia is, therefore, a local symptom of brain disease of great value in determining the situation of a lesion, when considered in connection with other symptoms. The symptom alone affords little evidence of the nature of the disease producing it, since it may be caused by any of the various forms of brain lesion (see *Brain*). It has been observed in cases of basilar meningitis, in tumors of the occipital lobe and basal ganglia, in hemorrhage and softening involving the internal capsule, and in embolism of the terminal branches of the posterior cerebral artery, and of the trunk and posterior branch of the middle cerebral artery, as well as in other rarer conditions.

The diagnosis of the symptom may be made by an examination such as has been described. The prognosis and treatment of it will depend entirely upon the nature of the disease producing it. *M. Allen Starr.*

[1] Von Monakow: Arch. f. Psychiatrie, xiv., 698–750; xvi., 151–200. Wernicke: Lehrbuch der Gehirnkrankheiten, Bd. I., S. 79–84.

[2] Compare cases of Haab with those of Fritsch and Westphal cited by Starr: Visual Area of the Brain, American Journal of the Medical Sciences, January, 1884; and these with Seguin's case, Journal of Mental and Nervous Disease, January, 1886. Full bibliographies are to be found in these articles.

HEMICRANIN is a mixture of phenacetin five parts, and one part each of caffeine and citric acid.
W. A. Bastedo.

HEMIDESMUS.—Hemidesmus Radix. *Indian Sarsaparilla.* "The dried root of *Hemidesmus indicus* R. Br." (B. P.) (Fam. *Asclepiadaceæ*). The plant is a slender, twining shrub, native of India. The description in the British Pharmacopœia, into which it is introduced apparently out of compliment to the Indian physicians, is as follows: "The root is long, rigid, nearly cylindrical, tortuous and longitudinally furrowed. It seldom exceeds ¼ of an inch (6 mm.) in thickness and is of a reddish-brown or dark-brown color. On one side of the root the cork is frequently separated from and raised above the cortex, and is transversely fissured. The transverse section exhibits numerous laticiferous cells in the cortex. The root has a fragrant odor and a somewhat sweet taste." A syrup, made from one ounce of hemidesmus to ten and one-half ounces of menstruum, is used in England.

The medicinal properties of hemidesmus are said to be those of sarsaparilla, in the stead of which it is used in British India; in Europe or in this country it is rarely used. Its composition has not been fairly studied, but it is safe to say that nothing physiologically very peculiar or active is contained in it. An odorous principle, perhaps cumarin, has been partially examined. The syrup in which it is prepared, like that of sarsaparilla, is scarcely more than a flavoring vehicle. *Henry H. Rusby.*

HEMIPLEGIA (ἡμισυς, "the half," and πλήσσω, "I strike"). Paralysis of one-half of the body.

Causes.—Hemiplegia, as ordinarily seen, is usually the final result of an apoplectic stroke, from cerebral hemorrhage, embolism, or thrombosis. The stroke is often attended by loss of consciousness and profound paralysis, but if the patient live the paralysis gradually lessens and leaves the final hemiplegic state. In some instances the paralysis is not ushered in in such stormy manner, but increases gradually for a day or two, either with or without loss of consciousness. The early symptoms are the expression of shock to a larger or smaller part of the brain, of pressure, and, sometimes, of inflammation of the tissues adjacent to the lesion, as well as of destruction of brain substance. The final symptoms are the expression of destruction and degeneration of brain tissue.

Hemiplegia may also come on slowly, caused by a brain tumor, brain abscess, chronic softening, meningitis, etc. In these instances the clinical picture may be complicated by a sudden paralysis from hemorrhage or thrombosis in the diseased area, or the like.

What has already been spoken of is organic hemiplegia, due to destruction or injury of the motor centres, or the cortico-muscular tract, that is, the tract of fibres carrying impulses from the motor centres to the muscles. But hemiplegia also occurs without palpable lesion. Notably this is true of hysterical hemiplegia.

Manifestations.—The paralytic manifestations are variable. Shortly after an apoplectic attack, if it be severe, most of the muscles on one side of the body are paralyzed. The arm and leg are entirely powerless. The muscles supplied by the lower branches of the seventh nerve, those of the cheek and mouth, are completely paralyzed. That side of the face is expressionless, and the mouth may be drawn toward the other side. But the muscles supplied by the upper branches of the seventh nerve—the orbicularis palpebrarum, occipito-frontalis, and corrugator supercilii—are as a rule but little affected. The tongue may remain motionless in the floor of the mouth. When protruded it deflects to the paralzyed side. The muscles of the chest are usually somewhat affected, and the breathing is less deep on the paralyzed side. The writer has found the latter symptom an aid to diagnosis of both the presence and the side of hemiplegia in cases in which the coma was so profound that it was impossible to elicit any sign of power on either side. On the other hand, some muscles almost invariably escape injury. These are the muscles supplied by the third, fourth, fifth, and sixth nerves—those of the eyeballs and of mastication—and the muscles concerned in swallowing and vocalization. The articulation is usually somewhat indistinct for a short period, or there is a loss of speech—aphasia. Aphasia occurs mostly with right hemiplegia (in right-handed individuals). But even indistinctness of speech, when there is no aphasia, is often more marked in right than in left hemiplegia.

The high degree of paralysis just described, though in

rare instances it remains permanently, is usually of short duration. The paralysis of the muscles of the trunk and of the tongue, and the indistinctness of articulation usually disappear at an early period. The facial paralysis also diminishes, sometimes disappears altogether. The paralysis of the arm is usually most profound, and slowest to improve.

The hemiplegia of a later period is, as a rule, to be found only in the face and extremities, though sometimes the tongue continues, when protruded, to deflect toward the paralyzed side. If the facial paralysis be slight, it will be observed that the nasolabial fold is less marked than on the sound side, that the upper lip is less arched, and the angle of the mouth droops somewhat on the affected side. A slight paralysis becomes more marked when the muscles are actively exercised, as in smiling, exposing the teeth, etc. Not rarely the effort to expose the teeth will reveal a decided facial paralysis, while laughing will show none whatsoever—probably a psychic reflex act on the part of the thalamus. The opposite, the appearance of a more decided paralysis with laughter than with voluntary movements of the face, is observed in rare instances, probably indicating lesion of the thalamus. In the extremities usually certain groups of muscles are most likely to be paralyzed, or most deeply affected, *e.g.*, extensors of the fingers, supinators, trapezius muscles, anterior tibial, and peroneal group of muscles, and flexors of the knees. As a rule the hands and feet are more affected than the higher parts of the extremities. The skilled movements of the fingers are most affected. In walking a dragging of the toes on the paralyzed side is often observed.

In cases of permanent hemiplegia, especially if the paralysis be considerable, after a few weeks contracture of the paralyzed muscles usually occurs, a condition termed late rigidity. In a well-marked case we shall find the arm drawn toward the chest, the forearm flexed on the arm and pronated, and the fingers flexed; in the inferior extremity all the joints extended, and the foot in the position of talipes equino-varus. The rigidity is usually greater in the upper than in the lower extremity. In rare instances we find extension of the joints of the arm instead of flexion, or flexion in the lower extremity instead of extension. Sometimes the muscles of the face are also affected; then the naso-labial fold becomes deeper, and the angle of the mouth elevated on the affected side. In extreme cases the rigidity is more or less constant; but usually it is much less than in the instance above described, and it is then increased by voluntary efforts to move the parts, or by emotional excitement, while it is diminished or absent during sleep. In some cases the rigidity improves very much with time, so that it is only observable during acts requiring special skill.

Hemiplegics often succeed in walking with the aid of a cane, even though the leg be completely paralyzed, especially if the limb be at the same time rigidly extended. In this case the pelvis and hip of the paralyzed side are elevated by the contraction of the abductor muscles of the thigh on the sound side, and the foot is then propelled forward by the action of the inward rotators of the healthy limb, the toes usually scraping the floor during the forward movement. The body now rests partly upon the foot of the paralyzed limb, partly on the cane, held in the hand of the sound side, the centre of gravity being between them while the healthy limb is being brought forward.

In permanent hemiplegia the deep reflexes are usually very much increased. The knee jerk, elbow jerk, wrist jerk, etc., are exaggerated and the ankle clonus can be elicited, which is almost never found in healthy individuals. The deep reflexes may even be exaggerated, though to a less degree, on the non-paralyzed side. But it is also true that the latter side may be weaker than before the paralysis set in. There is often some alteration in the condition of the superficial reflexes. Only one of the latter has much diagnostic import, the toe phenomenon, recently described by Babinsky. The usual plantar reflex is flexion of the toes. Babinsky observed that when there is disease of the pyramidal tracts stroking the sole of the foot produces extension of the big toe. Care is usually necessary to get the proper response. The patient's mind should be distracted if possible, and then a stroke slowly made from the heel toward the toes. A not pointed pencil answers very well for making the stroke. This sign has not the full significance of the ankle clonus, because not so commonly found, yet it may have a greater diagnostic value, because it may be found very soon after the paralysis sets in, while one or several weeks elapse before the ankle clonus can be elicited. In several instances the writer has found the toe phenomenon within one, or at least a few, hours after the onset of an apoplectic stroke.

Other motor phenomena are observed more or less frequently, the probable results of irritation of, or lessened inhibition in, the motor areas. Often there is a movement of the paralyzed arm—hand lifted to the head or the like—in yawning, etc. A slighter movement in the paralyzed extremity is frequently observed at the same time with corresponding movements of the sound limb. The opposite is also observed, viz., contractions of corresponding muscles on the sound side with attempted movements of the paralyzed side. The kind of motor phenomena just described are usually spoken of as accessory movements. More rarely tremor, or choreic movements, or athetoid movements (slow continuous movements of fingers and toes) are found in the paralyzed extremities. The choreic and athetoid movements have been supposed to occur especially with lesion of the thalamus. If that be true, it is probable that it is rather because the posterior part of the internal capsule is affected than because of injury to the thalamus itself.

Seat of Lesion.—Hemiplegia is most frequently caused by a lesion of the corpus striatum, or in its neighborhood, the paralysis being on the opposite side of the body. If the paralysis be permanent, it is because the internal capsule has been damaged, while the late rigidity is attendant on degeneration of the pyramidal tracts.

Hemiplegia, produced by lesions in other parts of the motor tracts, has in some instances special manifestations.

Cortex.—Lesions in the motor area of the cortex, the anterior and posterior central convolutions, more frequently produce monoplegia—paralysis of one limb, or of the face—than hemiplegia, but the latter may occur if the lesion be sufficiently extensive. Such lesions are often attended by convulsive movements, especially in case of a neoplasm. In such instances there occur usually periodic attacks of clonic spasms in one extremity or one side of the face; in the hand if the lesion chiefly affect its centre, etc. These spasms are likely to be at first quite limited and not accompanied by loss of consciousness. But in succeeding attacks the convulsions may extend to other parts of the body. They first travel over one side of the body. If the convulsion also seizes the other side, loss of consciousness is likely to supervene. There is often some blunting of the sensibility corresponding to the amount of paralysis, indicating that the cortical centres for motion have also direct relationship with the sensory functions. But such cases do not always give a like clinical picture. In a case of the writer's, in which there was a large tumor on the convexity, implicating chiefly the leg centre, there was paresis of the opposite arm and leg, but no appreciable impairment of sensation, and there had never been any convulsive movement.

Crus Cerebri.—Hemiplegia from lesion of the crus cerebri is often attended by paralysis of the third nerve on the side of the lesion. There is paralysis of the face and extremities on one side, and of the muscles of the eye on the other side.

Pons Varolii.—Lesions of the pons Varolii cause what has been termed alternate hemiplegia, that is, paralysis of the arm and leg on one side, and of the face on the other, the extremities being affected on the side opposite to, the face on the same side as, the lesion. The reason for this is the following: The central prolongation of the seventh nerve, like that of all other motor nerves, passes from one side of the brain to the other, the site of crossing being, in case of the seventh, in the pons. Lesions, then, in the latter locality may affect only the peripheral parts of the nerve—the nerve trunk itself, or its nucleus—and the facial paralysis be on the side of the lesion. The facial paralysis in such cases differs from that with ordinary hemiplegia, in that it has the stamp of peripheral paralysis. The paralysis is likely to be complete, all parts supplied by the nerve being equally affected, the eyelids and forehead as much as the lips and cheek. There will also be found changes in the electrical reactions, the so-called reaction of degeneration. The electrical reactions of the nerve and faradic contractility of the muscles are lost, while the galvanic contractility of the muscles may be heightened, but with a change in the normal formula of the reactions. Such changes in the electrical reaction are never found in central paralysis, only in paralysis of a peripheral type. If the degree of paralysis be great, some atrophy of the facial muscles may also take place.

Medulla Oblongata.—Hemiplegia from lesions of the medulla is likely to be attended by paralysis of other cranial nerves, while the seventh nerve escapes. There are likely to be a considerable paralysis of the tongue, indistinctness of articulation, and paralysis of the vocal cords from the involvement of the hypoglossal and pneumogastric nerves. The writer had under his observation a case of unusual interest, in which there was probably a lesion of the medulla and pons of traumatic origin. The injury was caused by an iron rod penetrating the right submaxillary space, and passing upward four inches in the direction of the foramen magnum. Immediately after the injury there was complete paralysis of the right arm and leg, of the cheeks, lips, tongue, and vocal cords. The patient was unable to swallow or to make any vocal sound. The upper part of the face was unaffected. He could move the eyes freely, open and close the lids, and was entirely conscious. There was also loss of sensation on the left side of the face. The condition of the patient rapidly improved, but there remained permanently a right-sided paralysis, considerable difficulty in articulation, and anæsthesia of the left side of the face.

Spinal Cord.—Hemiplegia from unilateral lesion of the cord is not attended by paralysis of any cranial nerve, but the arm and leg of the same side are paralyzed if the lesion be in the cervical region, and the leg only, if the lesion be at a lower level. Such a unilateral lesion produces a peculiar array of symptoms, spoken of as Brown-Séquard paralysis, because produced experimentally and first described by that distinguished physician. The symptoms are motor paralysis and loss of muscular sense, on the side of the lesion, in every part below it, and impaired or lost tactile, pain, and temperature sense on the opposite side. There is likely to be a narrow area, encircling the body, at the level of the lesion, in which there is hyperæsthesia. The explanation of the symptoms appears to be that the tracts for the motor fibres and muscular sense run up in the cord on the same side as are the corresponding nerve roots, and only cross to the other side in the medulla, while the tracts for tactile, pain, and temperature sense cross to the other side of the cord immediately after leaving the nerve roots.

Infantile Paralysis.—Some cases of hemiplegia of infancy, which are either congenital or acquired early in life, deserve special mention. The lesion is a varied one, from hemorrhage, inflammation, arrested development, etc., and is often of traumatic origin. The hemiplegia is usually attended by contractures of the paralyzed muscles, and by a considerable arrest of development. The leg and arm are smaller in circumference and shorter than those on the sound side, and the face is also frequently smaller than on the paralyzed side. The choreic, athetotic, and accessory movements already spoken of occur very frequently in these cases. Great impairment of intellect, even complete idiocy, is frequently found, and epileptic convulsions become established in many cases.

Hysterical Hemiplegia.—In hysterical hemiplegia the paralysis is mostly confined to the extremities, the face escaping. When there is an appearance of facial paralysis it is usually found that there is really spasm, not paralysis of the facial muscles. It is not so commonly true of hysterical as of organic paralysis that the arm is more profoundly paralyzed than the leg. Mostly the paralysis is not complete. There is paresis, rather than paralysis. In that case one frequently observes that the resistance of the patient to the efforts of the examiner to move the limb is done in a jerky manner. There is resistance for a moment, then none whatever, as though there were no effort on the part of the patient. More power may be manifested in emotional conditions, in gesticulation, etc. A degree of power may be revealed in an apparently paralyzed limb, by a sense of resistance on passively moving the limb, or by the limb remaining momentarily in the position in which it has been placed, a position requiring some muscular effort. The gait is ordinarily different from that of organic hemiplegia, the paralyzed foot rather dragging behind, than swinging forward in a circle.

The reflexes are commonly not affected, neither the superficial nor deep being altered in the way already described as occurring in organic hemiplegia. Not rarely contractures occur in the paralyzed limbs. As a rule they make the prognosis less favorable, at least in so far as the duration of the symptoms is likely to be much greater. Anæsthesia is commonly found with hysterical hemiplegia. The sensory symptoms are usually more extensive than is the paralysis, the face being affected as well as the extremities. There may be both superficial and deep anæsthesia and also of the special senses on the affected side. Not rarely there is partial anæsthesia, loss only of tactile, or only of pain sensation, or impairment of one more than of the other. The patient may be altogether unaware of the impaired sensation.

Hysterical hemiplegia affects mostly the left side. It has been said to occur three times as often on the left as on the right side. It may come on abruptly or gradually. Not rarely it follows a convulsive seizure. It may be caused by an injury, or any physical or mental shock. Very likely an emotional condition, or, at least, some mental impression underlies the hemiplegia in most instances, though it need not immediately follow upon the exciting cause.

The patient usually recovers from the paralysis, but it may take days or years. During this time the paralysis may remain unchanged, or it may vary greatly in its intensity. The disappearance of the paralysis, just as its inception, may be due to mental causes, shock, suggestion, or the like.

The characteristics of the paralysis just given may suggest or establish the diagnosis of hysteria. But it is well to remember the ofttime difficulties of diagnosis, not the less so that not rarely there is association of hysteria and organic disease. Where the question of hysteria has arisen mistakes have been made not infrequently even by the best men. Not only the peculiarities of the supposed hysterical symptoms should be considered, but also the personality of the patient, the apparent cause of the manifestations, as well as the history and the whole clinical picture.

Philip Zenner.

HEMORRHAGE.—From *αἷμα*, blood, and *ῥήγνυμι*, to break through. The blood may escape from the arteries, veins, or capillaries, and from its origin the hemorrhage is designated arterial, venous, or capillary.

In arterial hemorrhage the blood escapes from the arteries in jets synchronously with the contraction of the left ventricle, and flows continuously during diastole. The blood is then of a bright or pale red according to the quality.

In venous hemorrhage the blood escapes from the veins in a continuous stream, and is dark in color. Blood has been seen to escape from a hemorrhoidal vein in jets, but synchronously with the contraction of the abdominal muscles. In capillary hemorrhage the blood oozes from