Lymphatics.

Many articles by Arnold (1838), Hyrtl (1860), His (1865), Robin, Schwalbe, Obersteiner (1870), Riedel, Arndt, Krause, etc.
Key and Retzius: Studien in der Anat. des Nervensystems, etc., 1876.
Rossbach and Sehrwald: Uber die Lymphwege des Gehirns. Centbl. f. med. Wiss., 1888, Nos. 25 and 26.

BRAIN, COMPRESSION OF.—Notwithstanding the unhappy use of a word that does not describe the subtile condition it is meant to express, the term compression is a convenient one that seems justified by the necessities of analysis and study. It is generally used in a pathological sense to describe a complexity of symptoms arising from a particular state of the brain characterized mainly by disorder of the circulation within the cranium, and manifested by more or less derangement of the three great faculties of the nervous system.

As a principle used to account for the phenomena of brain disease, and as a complication that is often present after various lesions of the brain and its membranes, the special and differential diagnosis of compression is of grave importance, and includes a wide range of cerebral pathology. The prevailing opinions of writers on this subject are somewhat misleading, since compression, in its true sense of occupation of less space, is a condition of the brain which in most cases seems to be assumed rather than demonstrated to exist, and it is rare to find a case of compression in which there are not present the symptoms both of concussion and laceration of the brain.

Etiology.—The squeezing of its constituent parts or the diminution of volume that is supposed to interfere with the functions of the brain may be effected by pressure from without the encephalon, or by tension within its proper substance. The condition arises from a great variety of causes, most common of which are cerebral tumor, hemorrhage, and inflammatory foci. Hypertrophy of the brain, simple congestion, hydrocephalus, and effusion from various causes may result in compression. The effusion may take place quickly or slowly. Some experiments, however, point to the fact that the symptoms of compression are not to be attributed to augmentation of tension of the cephalo-rachidian liquid.

Other causes are foreign bodies, loose splinters, or larger portions of depressed bone, and, in fact, any injury of the head, whether simple contusion, scalp wound, or fracture of the skull. Whatever be the cause of compression, it should be borne in mind that the most characteristic symptoms are brought about by *sudden* action, for compression established slowly upon the brains of animals by the injection of a liquid causes no appreciable symptom, unless the quantity injected be unusually large; and it should be further remembered that, in the human subject, appreciable symptoms of compression are not always induced by such causes as sanguineous extravasation into the cranium and the cerebral ventricles, nor by the pressure of a foreign body, a tumor, or a fracture.

Symptoms.—Loss of consciousness and paralysis, which vary according to the seat and extent of the compression, are symptomatic of the condition, no matter whether the compression be sudden, like that following a wound or some mechanical injury of the skull and its contents, or whether it be slow, as that following enlargement of the brain from extravasation of blood, lymph, pus, serum, or tumors. Paralysis, excepting that of one or the other eye, is always on the side opposite to that which is the seat of injury. The symptoms may come on almost immediately after any injury of the brain that disorganizes its substance; but there are other cases in which compression takes place slowly and after a certain lapse of time. The initial symptoms that characterize this period are mainly subjective and those of congestion, as vertigo, headache, confusion of ideas, nausea, and, on rare occasions, vomiting. Then follows a lethargic sleep and more or less paralysis, and the patient cannot be aroused by any stimulus. The face is suffused and dusky, it wears an expression of well-pronounced stupor, and the eyelids are usually closed and immovable; respiration is slow, labored, and stertorous, something like the act of snoring, and the peculiar blowing movements of the lips in expiration have been compared to the act of smoking a pipe. Deglutition is impossible, and the tendon reflexes are abolished. As the intracranial trouble increases the pulse becomes slow and labored. It may be hard and frequent, or small and intermittent. Sometimes it is very irregular, and the symptoms may resemble those of anæmia of the brain and medulla, brought about by experimental means. Paralysis of the sphincters is generally present, with involuntary evacuations; or the patient may have torpidity of the bowels and obstinate constipation. Retention of urine, often present from paralysis of the bladder, is followed by incontinence as the result of overflow from distention. The skin may be cool or it may be hot and perspiring, and the temperature, though generally normal, may reach as high as 106° F. One or both of the pupils may be contracted or dilated, or they may rest immobile and unresponsive to the action of light. The symptoms of optic neuritis may also be present, and sometimes nystagmus is noted. Paralysis both of motion and of sensation, in one or both extremities, may exist in case the compression is exercised on the hemispheres, and convulsive movements and twitching of the limbs may occur on the paralyzed side or on the opposite side. Death occurs from arrest of respiration. (See *Coma* and *Asphyxia*.)

Diagnosis.—These pathognomonic symptoms, which are chiefly owing to effusion under the dura or to fracture of the inner table of the skull with resulting secondary anæmia, are not always met with, nor do such injuries as those inflicted by nails and arrow-heads driven into the skull, and even by missiles lodged in the brain, always produce the symptoms of compression. Sometimes the brain may be compressed without any disturbance of its functions. Pressure from an abscess, causing a hollow in the brain as large as a man's fist, has been known to cause no symptom of compression. It is, moreover, doubtful whether depressed fracture be a frequent cause of compression, since the injury is always complicated by laceration, the symptoms of which are often mistaken for those of compression; and it is often difficult and always embarrassing to determine, after an injury of the head, whether we have to deal with a contusion of the brain, a simple concussion, or the more problematical symptoms of cerebral compression. One state so often merges into the other that the attempt to establish a clear basis for a sure diagnosis of the respective conditions seems hopelessly confused and intricate. Roughly speaking, the most characteristic symptom of concussion is somnolence and intensity of the evil from the outset; contusion manifests itself by agitation, delirium, convulsions, contractions, and the delay of febrile symptoms; while the most salient symptom of compression is paralysis, except in the case of effusion of blood into the convexity of the hemispheres or into the ventricles, when contracture or tonic muscular spasm of extended duration is the more prominent phenomenon. Without being a sure sign, paralysis constitutes at least a valuable element in the diagnosis of a condition that has no single confirmative sign. If there be an unequivocal sign of cerebral compression in the majority of cases following injuries of the head, it is, perhaps, that furnished by obstruction of the venous circulation, in consequence of which the blood of the eye is not returned into the cavernous sinus, when we find with the ophthalmoscope papillary or peripapillary congestion, a general or partial serous infiltration of the papilla, and a strong dilatation with tortuosity of the retinal veins. (These appearances are shown in the accompanying colored plate.) In concussion the fundus of the eye retains its normal state; in compression there is always serous peripapillary infiltration, dilatation, tortuosity, and sometimes thrombosis of the veins of the retina. The intraocular changes herewith portrayed are not always pathognomonic, but they lend to the diagnosis an additional source of correctness. As a rule, when the eye-grounds show arrest of the retino-choroidal circulation, we have an indication of the arrest of cardiac circulation and of the nervous functions.

PATHOLOGY.—According to many observers, the general symptoms of compression are owing to cortical anæmia, and the general impairment of cerebral function is owing to disturbance in the capillary circulation, which prevents the normal interchange between the blood and the tissues, and results in physiological death of the affected portion of the brain. In compression, the blood may be extravasated upon the surface of the brain, the effusion taking place between the skull and the dura, and in the cavity of the arachnoid, or in the intervening spaces that separate the membranes from the brain, or it may be effused into the ventricular cavities, or into the substance proper of the brain. The volume of an extravasation between the dura and the bone is sometimes very large; but, as a rule, effusions of this kind are always much larger at the vault of the cranium than at its base. Sanguinary effusions between the dura and the bone, according to the summarized opinions of writers on the subject, generally coagulate into a firm clot that may either be absorbed or undergo organization and become adherent to both the bone and the dura. This clot never becomes encysted as do clots in the cavity of the arachnoid, but may undergo ossification. It is also susceptible of other changes. Thus, for example, it may lead to necrosis of the neighboring bones of the skull, which may be perforated by an abscess having as its foyer the sanguineous effusion.

Blood extravasated between the layers of the arachnoid, especially on that part which covers the cerebrum, is of common occurrence in the severest head injuries, and forms at the convexity of the hemisphere a thin, evenly spread layer. It clots rapidly, loses its coloring matter, undergoes organization, assuming the form of a false membrane or of a membranous cyst, and may be absorbed and take on a secreting action like other cysts.

Effusions between the visceral layer of the arachnoid and the pia are less common than those in the cavity of the arachnoid, and are generally associated with some injury of the brain itself. They spread extensively into the spaces usually occupied by the cerebro-spinal fluid, and do not become encysted as in other situations.

Traumatic effusions into the brain substance may occur in any situation. They are generally associated with laceration or other severe injury of the brain, and are for the most part fatal. Should recovery take place, the blood clot undergoes changes similar to those observed in the organization of an ordinary clot from cerebral hemorrhage.

All effusions of blood between the dura and the brain are susceptible of being reabsorbed, of being encysted in a false membrane, and of giving place to encephalitis, meningitis, and other grave symptoms that come eminently within the province of surgery; in fact, it is chiefly from this point of view that compression has been treated by the great masters.

Clinical and experimental facts show that in compression from injury of the occipital region death results not from failure of the heart, as often surmised, but from failure of the respiration. The symptoms are not mechanical but depend on trouble of circulation in the bulb, and have their point of departure in the cortex of the hemispheres including the vaso-motor centre.

In place of attributing many of the troubles grouped under the subtile heading of compression to pressure exerted upon the brain, it would be better to attribute them to congestion, to contusion, to laceration of the cerebral substance, and to interference with the function of the respiratory centre, which puts us in a position to doubt whether the effects of compression upon the brain are not susceptible of further and more convincing proof.

PROGNOSIS.—The integrity of the brain is not compromised by mere compression itself, nor is the condition a very fatal one. Its gravity depends on consecutive inflammation, secondary anæmia, and the effect on the respiratory centre. Sometimes the symptoms disappear spontaneously and gradually without interference.

The TREATMENT of compression raises many questions of operative interference, which are discussed elsewhere under their respective headings. The whole end of treatment is to restore interrupted respiration and prevent cerebral inflammation. In endeavoring to do this, artificial respiration and heat to the head by irrigation appear most commendable. *Irving C. Rosse.*

BRAIN, CONCUSSION OF THE.—(Concussion of the cord is analogous to concussion of the brain.) The term "concussion of the brain" comes down to us from the earliest ages; it was used by Hippocrates, Galen, and Celsus; its modern significance was given it by Boirel, but it was Littré who first studied the subject post mortem, in 1705.

The subject of concussion of the brain, or commotio cerebri, has received much attention from various workers, especially in the last fifty years. The theories concerning it have varied widely, even to the extent of contradiction. The general cause of this condition—traumatism—and the characteristic symptoms of the same—sudden unconsciousness, feebleness of heart and respiration—are well known; but what occurs in the cells affected is as yet undiscovered.

Verneuil, the eminent French surgeon and writer, after much observation and experience on this subject, formulates the following definition: "It is a series of phenomena occurring more or less suddenly which result from a mechanical shaking (jarring movement) of the anatomical cells, tissues, and organs, characterized by temporary excitation or depression of the properties, offices, or uses of the parts which are shaken; and as a result there are caused anatomical changes similar to those which are normally seen in the successive phases of functional activity and functional repose."

According to Pick, this term was first used because there was thought to be a shock and molecular disturbance to the cerebral tissues with no visible lesions or lacerations. This cannot hold, since the autopsy almost invariably discloses macroscopic lesions. However, these lesions are not necessarily the cause of the insensibility. It has been shown by William Savory that the state of unconsciousness and insensibility passes away, while the lesions remain. In many cases the extent of the lesion is not sufficient to produce the existing symptoms, and, again, the lesions may be present when there is no insensibility.

There are three grades: mild, severe, and fatal.

In explanation of the mildest grade the following has been offered: The cerebral cortex, owing to a momentary deprivation of nutrition, following lowered blood pressure and fall of temperature, ceases for the time being to function, and so follow loss of memory and unconsciousness.

In the more severe type the molecular disturbance is more violent and is naturally followed by graver symptoms. Mental functions are temporarily suspended, the condition of the patient somewhat resembles sleep, although the eyes are often in motion unnaturally with closed lids; or if at rest, they are not upturned and divergent as in normal individuals. This may be termed "the sleep of concussion." There is no snoring. Indeed, there may be disturbances of the cardio-pulmonary functions or paralyses of different parts of the body from localized lesions of the brain.

In the fatal form, death may follow at once or after a few hours.

ETIOLOGY AND PATHOLOGY.—The gross cause of cerebral concussion is, beyond doubt, some form of traumatism either applied directly to the head or indirectly transmitted, as in the case of a fall upon the feet—when the shock is transmitted through the spine,—or in that of a blow or a fall upon the chin. But the changes produced in the brain substance and the subsequent results have been the subjects of much argument.

Treves groups the theories of concussion under three heads:

1. Molecular disturbances.
2. Multiple hemorrhages.
3. Vascular disturbances.

In the molecular form of disturbances the brain substance is thrown into vibrations by the violence transmitted to it. The old idea that obtained, viz., that there were no macroscopical lesions, has been entirely overthrown by modern workers, for, as has been noted, in all fatal cases which come to autopsy macroscopical lesions consisting of hemorrhages or lacerations are almost invariably discovered. Not infrequently, however, these hemorrhages are too insignificant to be the cause of death or even of the symptoms of concussion. That cerebral anæmia exists is hardly disputed. The cause of this anæmia has offered a field for discussion.

Duret describes "the cone of depression and the cone of bulging," meaning by the first the point which receives the injury and by the second the area just opposite. The force of the injury imparts to the cerebro-spinal fluid an impetus which drives it from the lateral ventricles into the third ventricle through the aqueduct of Sylvius into the fourth, and the latter, receiving more fluid than it loses, becomes distended. This distention causes a stimulation of the restiform bodies resulting in cerebral anæmia. Duret also believes that the small hemorrhages are due to the blood-vessels losing the support of the cerebro-spinal fluid.

Anæmia may exist by displacement of blood following the indentation of the skull. According to Fischer the loss of vascular tone may result from the nervous shock of the injury and may cause permanent vascular degeneration. Emotion is probably followed by a loss of vascular tone. The results of shock are sometimes similar. Possibly this mechanical vibration causes both physical and chemical changes in the nerve cell.

Phelps believes that the stimulation of the restiform bodies resulting in efferent reflex action causes direct capillary contraction.

After the cortical centres have received a shock there is instituted an instability of cerebral nutrition due probably to increased sensitiveness of the vaso-motor centres and a liability to anæmia or hyperæmia from very slight causes. Oftentimes individuals after head injuries are unable to undergo severe labor or exposure to the sun or the effects of alcohol. These facts were clearly recognized in the late Civil War, for men who had suffered injuries of the head were relegated to the invalid corps.

SYMPTOMS.—The most characteristic symptoms of this condition are sudden unconsciousness, loss of memory for events just preceding or during the injury, muscular weakness, dulled sensibilities, general prostration, vomiting, and changes in the circulatory system, as illustrated by the respiration and the pulse.

These symptoms vary in their intensity and duration in accordance with the severity of the injury sustained. They may be very slight, consisting only of momentary unconsciousness, pallor, and mild interference with respiration; or they may be so severe as to be followed by death in a few moments. Treves notes three stages:

1. Stage of collapse.
2. Stage of reaction.
3. Stage of convalescence.

This is a convenient and comprehensive classification.

The clinical picture of the mildest grade is illustrated by what is familiarly known as the state of being "stunned." In this condition the patient hears strange noises, there are visual disturbances, dizziness, general weakness, inability to stand or to use the arms; the eyes lose their natural expression, the eyelids close. The pulse is always weak; sometimes it is slow, at other times it is rapid—more generally the former; and it is probable that immediately after the injury it is always slow. Respiration is disturbed, irregular, and now and then of a sighing character. The paralysis which occurs is generally only temporary as well as functional, for these phenomena disappear with returning consciousness.

In cases of well-marked concussion the superficial reflexes and knee-jerks are not pronounced, neither are the cranial reflexes. In slight cases these are sluggish.

A common symptom is delirium. This may be violent or mild, the nocturnal form being most characteristic in cases of cerebral traumatism.

In the more severe cases the symptoms are more accentuated. The unconsciousness may amount to coma, the patient being incapable of being aroused; he cannot even swallow. There are successive attacks of vomiting; the pulse is irregular, small, and generally very slow. Respiration can scarcely be detected. The skin is cold. The pupils are equal and more or less dilated, their reaction to light varying with the extent of the injury. There is incontinence of rectum and bladder, for although the sphincters of these organs are relaxed the organs themselves are not paralyzed. The temperature is the same on both sides of the body; rectal temperature is invariably subnormal, even falling to 95° F.

In the stage of reaction there is a general improvement in the bodily functions. The temperature rises, the skin becomes warm, circulation improves, respiration is stronger, and the pulse gradually becomes normal. This stage is ushered in by vomiting. Consciousness comes back by degrees, and with returning consciousness headache often supervenes. The loss of memory noted in this stage may refer only to events just preceding the receipt of the injury, or it may also include those which occurred at the time of injury. The loss of memory may continue for some time after consciousness is fully regained. During this stage the patient may die with symptoms of encephalitis or of spreading œdema. Effusion of blood may often cause death in injury to the head with or without any marked external injury. Effusions may occur upon the surface of the brain after superficial lacerations of its substance.

The duration of the stage of convalescence may be from a few days to weeks or even months. When recovery is prolonged it is fair to assume that laceration has taken place. Such an hypothesis would also account for those cases in which complete recovery never takes place and in which mental disturbances persist.

DIAGNOSIS.—Concussion of the brain should be differentiated from opium poisoning, alcoholic coma, compression, and contusion. From opium poisoning it should be distinguished by the narrow pupils and stertorous breathing, which serve as diagnostic differences. It is often of medico-legal importance to diagnose this state from alcoholic coma. While in the latter the odor of alcohol might at first sight seem conclusive, it must not be forgotten that the brain lesion may also be present, having been received after the alcohol had been taken; also that alcohol in some form may have been given after the receipt of the injury. At all events, when a perfectly clear history pointing unmistakably to alcoholism cannot be obtained, give the patient the benefit of the doubt and treat with a view to the existence of the possible brain lesion, opium of course having been excluded.

Bourneville differentiates apoplexy and non-traumatic hemorrhages from concussion by the temperature, which is subnormal in the former, later becoming normal when recovery is to take place, but rising to an extreme degree in cases that terminate fatally; whereas in traumatic lesions the temperature rises at once while the results are uncertain.

The history will also throw much light on the diagnosis. Between concussion and compression the following table will be found to contain the points of differentiation:

Concussion.	*Compression.*
Onset of symptoms immediately after injury received.	Onset of symptoms some time after receipt of injury.
Onset sudden. Immediate unconsciousness.	Headache and drowsiness, gradually increasing to unconsciousness.
Muscular system generally relaxed, no definite paralysis.	Definite paralysis, local or general.
Pulse always weak, generally slow, sometimes rapid, and irregular.	Early slow, becoming rapid as the condition advances.
Respirations slow and shallow, may be sighing.	Early respiration is regular and slow, later becoming irregular, resembling Cheyne-Stokes.
There is incontinence of urine.	Retention and overflow of urine.
The pupils are equal, dilated, and react to light.	Pupils are irregular till the last stage, then are dilated and do not react to light.
The surface temperature is equal on the two sides.	Temperature is generally irregular.

PROGNOSIS.—The prognosis must always be guarded. In slight cases recovery is generally to be expected. Unfavorable signs are coma, very slow pulse, convulsions, and paralysis. Signs of compression and of lacerations in the stage of reaction modify the prognosis.

TREATMENT.—The treatment must vary with the stage at which the case is seen. If an external wound exists it must be treated, as in ordinary cases, according to its character. General directions for the stage of collapse are to regulate the depression of the circulatory and respiratory systems by means of warmth applied to the body; stimulants may be administered by the mouth if the patient can swallow; per rectum, or hypodermatically. Alcohol is contraindicated unless stimulation is indicated. The stimulants that may be used are ether, musk, strychnine, atropine, sparteine, nitroglycerin, camphor; counter-irritants such as sinapisms may be applied over the præcordium and epigastrium or to the calves of the legs. If the patient continues to be unable to swallow he may be fed through a stomach tube or per rectum. Black coffee enemata are also useful.

In the stage of reaction stimulants should be withheld, the diet should be light, and the bowels should be kept open by purgatives and enemata. Darkness and quiet are essential. When recovery is delayed shaving the head and the application of cold by Leiter's bags are to be commended; calomel and salines are beneficial.

In the stage of convalescence the patient should not be permitted to work and should be shielded from all mental excitement. Fresh air, rest, and light diet are essential.

Chloral hydrate and potassium bromide are indicated in cases of irritation. Potassium bromide aids in preventing inflammation. There must be no constriction at the neck; the head must be kept low; the scalp should be shaved and examined with care, for in fractures of the skull there are often no symptoms of a concussion for days or even weeks.

"Railway spine" or spinal concussion, formerly used to describe the results of concussion, is a misnomer.

Traumatic neurasthenia may result; this is similar to the condition following injuries to the spine when health, on account of organic disease or of overstrained nerves, is at a low ebb at the time of the injury. Traumatic inflammation of the brain or of its membranes may result. Abscesses may also follow.

Cerebral irritation is a condition sometimes following concussion. Its symptoms are probably owing to bruising of the frontal lobes. The patient lies on the side with the limbs curled up and is unconscious. He is restless and very irritable; the eyelids are closed; the pulse is slow—from 40 to 50; there is an absence of heat in the regions of the head and spinal cord; and generally there is incontinence of fæces and urine.

This condition may continue for as long a time as three weeks. The patient then extends the extremities and assumes the supine position. The temperature becomes normal, the pulse more rapid, and recovery may supervene. On the other hand, the mind may be permanently affected. There is absence of memory during the illness.

The findings at the autopsy table have been substantiated by the animal experiments performed by Koch, Fiehlene, Wittkowski, Albert, Goltz, and others.

Emma E. Walker.

BRAIN, DEVELOPMENT OF THE.—The development of the brain is bound up with that of the nervous system at large. While in the lower animals the nerve cells appear scattered or grouped in various parts of the body, and such scattered ganglion cells are found in even the highest animals, yet it is characteristic of the central nervous system as found in vertebrates that the nervous structures are collected into a common aggregate which contains only such admixture of other than nervous tissue as may serve to protect, support, and insulate the latter. It is obvious that the ordinary vegetative functions of life and many adaptive processes may be satisfactorily performed without a nervous system as such, as may be seen in the higher plants. The development of a central system, then, is an indication of preparation for higher functions than those of nutrition and metabolism or even of complex adjustment to the environment.

Early Stages.—After the egg has been differentiated into an animal and a vegetative pole and after the vegetative cells (by a process of invagination or substitute for it) have acquired an internal position, the cells remaining at the periphery (ectoderm or epiblast layer) represent the rudiments from which are to spring all the cells and specific organs not only of the central nervous system but of the sensory apparatus which forms the avenue from the external world to the central system. The ectoderm also contains, of course, the forerunners of the cells of the epidermis at large.

In general, the rudiments of the central nervous system collect in a broad longitudinal band extending along the dorsum of the embryo, while the rudiments of the sense organs exhibit a tendency to be arranged in one or more series along either side of the central band or medullary plate (Fig. 822).* In lower, especially aquatic, forms this lateral sensory band is evident in late life as the series of lateral-line organs, and it is plain that some or all of the

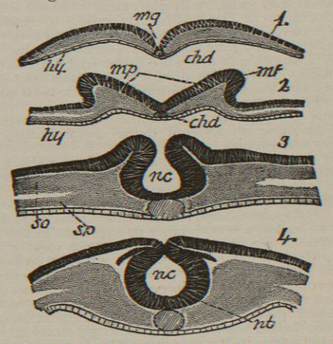

FIG. 822 (Nos. 1 to 4).—Transverse Sections through the Neural Plate and Neural Tube of an Embryo Bird. *chd*, Notochord; *hy*, entoderm; *mf*, neural folds; *mg*, neural groove; *mp*, neural plate; *nc*, neural canal; *nt*, neural tube; *so*, somatopleure; *sp*, splanchnopleure. (Mihalkovics and Balfour.) In No. 4 the rudiment of the spinal ganglia is represented but not lettered.

organs of special sense obey the same law of serial arrangement, the ear in particular betraying relationships to the lateral-line system. (Compare Vol. I., p. 627.)

The Neural Plate and Tube.—The neural or medullary plate is supported from below by a band of cells derived from the original entoderm which separates from the latter to form a solid rod (perhaps theoretically a tube at one stage), called the chorda dorsalis. The medullary plate grows more rapidly than the adjacent ectodermal tissue, and thus forms a raised border on either side with a groove in the median dorsal line. This medullary groove is continuous behind with the lip of the blastopore or opening left after the invagination of the entoderm. As the lateral margins of the medullary groove rise higher above the surface (both nervous and epidermal portions participating in the growth), the groove is transformed into a tube. The concrescence of the lips of the fold begins at a point corresponding with the site of the future midbrain. In the vicinity of the blastopore the groove may remain open for some time, and there is formed a direct communication with the primitive digestive tract through what is known as the canalis neurentericus, a

*These and several of the following cuts are introduced without change from the article by Professor H. F. Osborn in the former edition.

communication that is disturbed only by the final closing of the blastopore. At the cephalic extremity also the closing of the tube is long delayed, and there is left an opening called the neuropore communicating with the exterior at the front of the head.

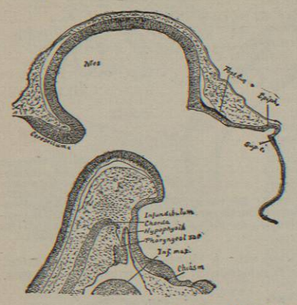

FIG. 823.—Median Section of Brain of Bird Embryo, to Show Hypophysis and Pharyngeal Sac.

In bony fishes and other vertebrates whose eggs have much yolk, the tube may be formed first as an apparently solid cord of cells, but the final result is the same.

At the cephalic extremity of the digestive tract, which at this stage ends in a blind sac separated from the pharyngeal invagination by the portion of the ectoderm known as the septum of Remak, is a region of fusion called the *area reuniens*. Here the cephalic end of the chorda, the base of the brain tube, and the angle of the enteric cavity tend to adhere temporarily. On the cephalic side of the septum the ectoderm gives rise to (an apparently single) median invagination which extends dorsally to meet a similar outgrowth of the infundibular region of the brain, combining with the latter to form the hypophysis or pituitary body. On the opposite side of the septum a somewhat similar outgrowth from the cephalic dorsal angle of the digestive tract forms the so-called Seessel's sac, which in some cases seems to connect with the cephalic end of the chorda. In birds at least this sac is distinct from the chorda which subse-

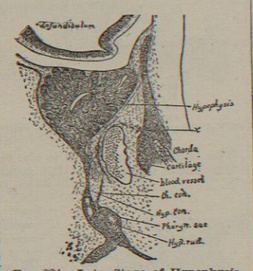

FIG. 824.—Later Stage of Hypophysis.

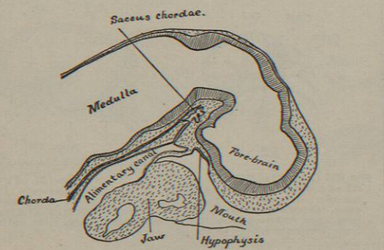

FIG. 825.—Illustration Showing the Relation between Chordal Sac, Alimentary Canal, and Brain Flexure in Opossum. (From Selenka.)

quently degenerates and leaves a convoluted thread-like vestige behind it that is closely connected with the site of origin of Seessel's sac. The latter, after the breaking through of the septum, comes, in birds, to lie in connection with the base of the hypophysis (Fig. 823).

This region seems to mark the morphological front of the head, and the medullary tube is often open at front, forming a neuropore (Fig. 826). Gradually the tube extends cephalically, and the base and roof unite along a line a part of which becomes that part of the base of the

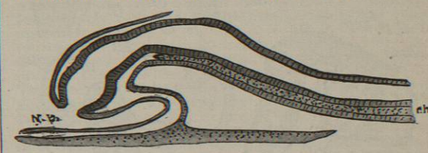

FIG. 826.—Brain of Torpedo, to Show the Neuropore, *n.p.*

brain occupied by the postoptic and preoptic recesses, the remaining part being the lamina terminalis (Fig. 826). The portion of the brain developed cephalad of the area reuniens, *i.e.*, in front of the cephalic end of the chorda, is called the prechordal as distinguished from the remaining, or epichordal part of the brain, and is morphologically different from the latter. In early stages of the development of the brain tube it appears segmented, and many attempts have been made to prove that these segments, or neuromeres, have a morphological significance and that they correspond with the segments discoverable in other and especially the mesodermal tissues. It has also been supposed that evidence of this primitive segmentation could be seen in the arrangement of the roots of the cranial nerves. On the whole, however, it must be admitted that, while it is not difficult to detect the segmental arrangement in the epichordal part of the brain, the prechordal portion either was not derived from

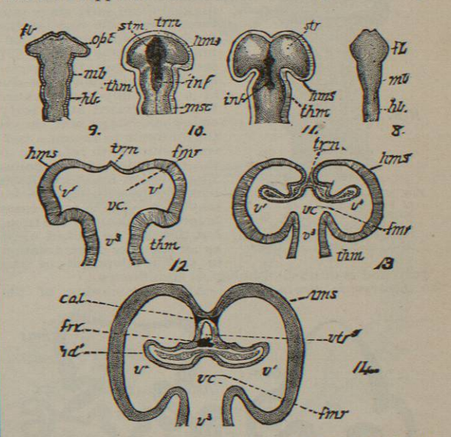

FIG. 827 (Nos. 8 to 14).—Horizontal Sections of the Forebrain (8 and 9, of a bird; 10 to 14, of a rabbit). (Kölliker, Mihalkovics, Löwe.) *cal*, Corpus callosum; *chd*, choroid plexus; *fmr*, foramen of Monro; *frx*, fornix; *hms*, cerebral vesicle; hemisphere; *inf*, infundibulum; *msc*, mesencephalon; *stm*, stem; *str*, corpora striata; *trm*, lamina terminalis; *thm*, optic thalami; *vc*, ventriculus communis; *vtr³*, fifth ventricle; *v'*, lateral ventricle; *v³*, third ventricle. Other letters as above.