acid or the potash alkalies, as the case may be, will prove to be very beneficial. Each case must be studied by and of itself. Besides the merely medicinal and dietetic treatment, much good is accomplished by regulated active exercise in the open air.

DISEASES OF THE MEDULLA OBLONGATA.

HÆMORRHAGE.

Pathogeny.—It is a rare event to have hæmorrhage occur in the medulla or pons, but cases have been reported. The conditions causing the hæmorrhage are doubtless very much the same as those of the brain, miliary aneurisms and atheroma being the chief factors. The larger aneurisms of the basilar artery may by rupture cause a hæmorrhage affecting this as well as other organs. The medulla is compressed by hæmorrhages from above, breaking through on to the floor of the fourth ventricle. These conditions are not now under consideration, the inquiry being restricted to hæmorrhage into the pons or medulla. The vessel affected in any case is small, the resulting clot is small, but there are usually several clots at the same time. They vary in size from a pea to an olive, but those examples of hæmorrhage in which the pons is simultaneously affected, or which occur in the pons, are much larger. One case is reported in which the hæmorrhage filled the whole of the pons, burst through on the left side, and also filled the fourth ventricle.* Another, in which the pons and fourth ventricle were invaded, and into the right crus cerebri there was also an extravasation.†

Symptoms.—If the hæmorrhage is large, vomiting usually occurs, consciousness is lost, there is complete muscular resolution, abolition of all reflex acts takes place, the breathing is sighing and irregular, becoming rapidly shallower, or is stertorous and noisy, the pupils are apt to be irregular, one large and the other minutely contracted, or both minutely contracted, death occurring in an hour or two, or in a day or two, in a deeply comatose state. There is a fulminant form, in which, hæmorrhage taking place in the medulla at or about the spasm-center, the patient falls with a cry into general convulsions, becomes comatose, and dies in a few minutes, or in an hour or two. Not all pursue this rapidly fatal course. A small clot may form on one side of the medulla or pons, there occur the usual symptoms of apoplexy, and the patient

* Dr. T. S. Dowse, "Transactions of the Pathological Society," vol. xxvii, p. 7.
† Dr. J. W. Ogle, ibid., vol. xv, p. 9.

emerges from the condition of unconsciousness, after some hours or days, paralyzed as to motion and sensation on the opposite side (hemiplegia), or all of the extremities may be paralyzed more or less fully; or there may be a paraplegia, the arms escaping, but usually both upper and lower extremities are affected both as to motility and sensibility. There are usually paralyses of the cranial nerves—the third, fourth, fifth, the sixth, the seventh, etc.—and there may be paralysis of the body, on the opposite side of a unilateral lesion, while the cranial nerves are paralyzed on the same side. The breathing, owing to the proximity of the respiratory center, is irregular in rhythm, sighing, dyspnœic—often of the Cheyne-Stokes type. The action of the heart is not so much disturbed, but the pulse may be exceedingly rapid and irregular. Epileptiform convulsions are very usual and important from the diagnostic point of view, since Nothnagel's "spasm-center" is located in this organ, and hence clonic spasm would *a priori* be expected. Difficulty in swallowing (dysphagia) from paralysis of the palatal and pharyngeal muscles, and difficulty of speech from paralysis of the tongue (ataxic aphasia), and sometimes an obstinate singultus, are present in those cases emerging from the first coma. Albumen or sugar may be present in the urine.

Course, Duration, and Termination.—As the facts above given sufficiently indicate, the course of hæmorrhage into the pons or medulla is rapid. Death may occur in a few minutes, in a few hours, or after several days. Very few recover in the damaged way above described. If such partial recovery ensue, the usual changes of an atrophic kind take place in the motor tract below the site of the hæmorrhage. The paralyzed muscles, innervated by the cranial nerves, it is probable, lose their electro-contractility in a few days.

Diagnosis.—It is often extremely difficult to distinguish between the coma and insensibility of hæmorrhage into the pons and the narcosis induced by opium or alcohol. There is no symptom produced by one which may not also accompany the other, but the antecedent history, taken with the group of symptoms as a whole, ought to conduct to right conclusions. The deviation of the head and eyes to the side of the intra-cranial disease, and from the paralyzed side, is a symptom of cerebral hæmorrhage, and not of opium or alcohol poisoning. Convulsions are uncommon in opium and alcohol poisoning, very common in hæmorrhage of the medulla. The pupils are often contracted in hæmorrhage, but never so minutely as in opium-poisoning. During the period of unconsciousness it may not be possible to diagnosticate between cerebral hæmorrhage and hæmorrhage of the pons and medulla, but the more frequent occurrence of convulsions, the vomiting, and the irregularity of respiration, may afford indications. Afterward the character of the paralysis, the manner in which the cranial nerves are affected, the paralysis of the palate, and difficulty of deglutition,

the singultus, and the urinary derangements, serve for a ready and definite decision.

Treatment.—The management of hæmorrhage into the medulla or pons is the same as for cerebral hæmorrhage, which has been fully discussed.

OCCLUSION OF THE VESSELS OF THE MEDULLA AND PONS VAROLII.

Pathogeny and Symptoms.—The vertebrals and the basilar are the arteries affected. The mode of occlusion is by thrombosis and embolism, and the pathological results are such as have been described. The immediate effect of occlusion of the vertebrals is a sudden and intense anæmia, with or without loss of consciousness. There are paralysis of the tongue, palate, pharyngeal and laryngeal muscles, and paresis of the facial. Sometimes the ocular muscles, innervated by the third, and the masseters are also paralyzed, and usually there are great irregularities in the respiratory and cardiac movements. Paralysis of the four extremities, more frequently hemiplegia, as the left vertebral is the one ordinarily closed, results, and there may be, although not the rule, lessened sensation in the same parts. Death may ensue at once; the affected area, receiving no blood, ceases to functionate. In other cases, the first shock of the accident passes off, the paretic extremities contract and become rigid, and may remain in this state for many years. The symptoms produced by obstruction of the basilar are bilateral, and, as the glosso-pharyngeal and par vagum are paralyzed, there occur at the same time severe laryngeal and respiratory symptoms, with intense dyspnœa, and rapid carbonic-acid poisoning, and, if the immediate effects are survived, paralysis of the four extremities. The treatment of this malady is the same as for the same condition affecting the cerebral vessels.

ACUTE INFLAMMATION OF THE MEDULLA—ACUTE BULBAR PARALYSIS.

Pathogeny.—The changes resulting from inflammation of the medulla oblongata are the same as those of encephalitis: hyperæmia; exudation of serum, with its albumen and fibrin; migration of white corpuscles and diapedesis of the red; disassociation of the nerve-elements; changes in the neuroglia (multiplication of its cells)—the ultimate result being a spot of softening.

Symptoms.—The inflammation makes rapid progress. The onset of symptoms is sudden: a violent headache; intense vertigo; nausea and vomiting; excessive hiccough; inability or great difficulty in swallowing; toneless voice, or speaking difficult—and these symptoms appear without apoplectic symptoms or convulsions. As the medulla contains so many important centers within a narrow area, it is obvious

that there may be much variety in the symptoms. If the pneumogastric nucleus is involved there will be embarrassed breathing, cyanosis, carbonic-acid poisoning, and the heart's action will be irregular, rapid, and weak. Paralysis usually invades the extremities, and varies much in extent: there may be hemiplegia, or all four extremities may be weak; sensation is not much affected. Neither tonic contractions of the muscles nor convulsions have been observed. The progress of the case is rapid. The difficulty of swallowing increases to absolute inability; the respiration is exceedingly irregular, and carbonic acid accumulates so that coma results, death occurring by failure of respiration.

Diagnosis.—It is probable that many cases diagnosticated hydrophobia were really examples of this disease. The distinction between inflammation, thrombosis, and embolism of the medulla, can not at present be made with certainty. While they all agree in symptoms of derangement of the important centers and nerves belonging to the medulla, myelitis of this part is not accompanied by apoplectic symptoms or convulsions, which belong to occlusion of the vessels.

Treatment.—The treatment is the same as that suggested for encephalitis.

CHRONIC INFLAMMATION OF THE MEDULLA—CHRONIC PROGRESSIVE BULBAR PARALYSIS.

Definition.—This disease is probably better known by the designation given it by Trousseau* — *glosso-labio-laryngeal paralysis.* This term was intended to express the main points in its symptomatology. Other names proposed are: *progressive muscular paralysis of the tongue, soft palate, and lips* (Duchenne†), and *progressive atrophic bulbar paralysis* (Leyden‡). *Chronic progressive bulbar paralysis*, the term proposed by Wachsmuth, and adopted by Erb, well expresses the seat and nature of the disease.

Causes.—The origin of the disease is very obscure. It occurs much more frequently in men than in women, and is a disease of advanced life, rarely occurring before forty. It has been referred to cold, to shocks, a blow on the neck, to rheumatism, to tertiary syphilis, to deep chagrin (Duchenne). It often coexists with progressive muscular atrophy (Friedreich§).

Pathological Anatomy.—Macroscopic examination may furnish only negative results. There may be changes of color and a dullness of appearance on section, and the medulla as a whole may appear to be shrunken,‖ or harder or softer than natural, in places, but definite

* "Clinique Médicale," vol. ii, p. 274.
† "D'Électrisation localisée," second edition, p. 641.
‡ Quoted by Erb, Ziemssen's "Cyclopædia," vol. xiii.
§ "Ueber, progressive Muskelatrophie," Berlin, 1873, cap. ix, s. 322.
‖ Lockhart Clarke, "Medico-Chirurgical Transactions," vol. lvi, p. 103.

results are obtained only by microscopic examination. While the lesions in the medulla are so obscure to the naked eye, the nerves coming from this organ are changed in the most obvious way, especially the hypoglossal and facial. The important alteration, in regard to which observers are generally agreed, is an atrophy and degeneration of the multipolar ganglion-cells of the anterior cornua. The vessels are dilated, leaving vacuoles, there are numerous corpora amylacea, the cells (nuclei of hypoglossus, etc.) are crowded with pigment, the neuroglia overgrown (hyperplasia). Subsequently the cells disintegrate and disappear, whence the marked decrease in size. The nerve-roots and the nerve-trunks are also much changed, the nerve-fibers having undergone fatty degeneration, the neurilemma sclerosed, and the axis cylinder wasted till it is barely visible, and only a mass of connective tissue left. The most advanced changes are found in the hypoglossal nucleus ; next, the spinal accessory and the par vagum are attacked, and the facial and glossopharyngeal are more or less damaged, and, according to Clarke, the nucleus of the fifth is invaded to some extent. Similar lesions occur in the brain and spinal cord—throughout the whole extent of the cord, in a case described by Lockhart Clarke, which, however, was accompanied by progressive muscular atrophy.

Symptoms.—The approach of the disease is very insidious. Headache felt in the occiput, some giddiness, a feeling of choking in attempting to swallow, a sudden inability to speak (Cheadle), are the symptoms first observed. The voice is not lost, but it has a nasal tone from the paralysis of the palate, and there is great indistinctness in speech because of the loss of power in the tongue and lips, the labial consonants not being pronounced. The tongue can not be protruded, and it wastes, becoming soon distinctly smaller. The food collects about the teeth and the cheek, so that the fingers are needed to dislodge it. The saliva dribbles from the mouth, the lips hanging limp and immovable. The taste is much less distinct or entirely wanting. It is a matter of great difficulty for the patient to get the alimentary bolus back into the pharynx. The efforts at swallowing excite coughing and suffocative attacks, and liquids are forced back through the nose. The palate and pharynx are so little sensitive that no reflex movements are caused by irritating them. The soft palate hangs limp and motionless in the fauces. When the disease reaches this point the appearance of the patient is eminently characteristic : the paralyzed lips and muscles of the face below the eye, their fibrillary trembling, and their motionless state in laughing, the flow of the saliva, the fatuous expression, the nasal speech, the inability to sound the labials, the choking in swallowing, the return of liquids through the nose, form a striking picture which no one can fail to comprehend. It is the sad fate of these patients to preserve their mental faculties, except that they become somewhat more emotional than formerly, and to continue conscious of their condition. The disease is truly *progressive*—the symptoms already described grow worse in every way—speech becomes less and less intelligible, swallowing more and more embarrassing and difficult, and the saliva increases in viscidity and quantity, the patient requiring a handkerchief constantly to absorb it. Other and more formidable symptoms now come on. The extension of the disease to the pneumogastric nucleus causes a paralysis of the muscles of the larynx, the voice is lost after preliminary weakness and huskiness, the respiratory muscles get weak and the lungs can not be expanded, and presently there are experienced oppression, heaviness of the chest, and constant dyspnœa, with paroxysms of a suffocative character, excited by the presence of mucus in the throat, by attempts of sneezing, coughing, or swallowing, or by the lodgment of some particle of food in the larynx. At the same time the action of the heart becomes irregular and weak, and attacks of præcordial oppression with a sense of impending dissolution occur. The condition of the patient is now truly pitiable. The mind is clear. The impossibility of swallowing leads to a rapid failure of strength, and, the digestive organs remaining unimpaired, an intolerable sense of hunger is felt. The termination may now be in a sudden failure of the heart, in an attack of pneumonia from lodgment of a foreign body, or by the slower process of starvation. The sensibility is unimpaired. The faradic contractility is at first diminished, but the muscles soon present the phenomena entitled by Erb the "reaction of degeneration." If the muscles are far advanced in atrophy, the electro-contractility may be lost. The disease in the medulla is often associated with the same degeneration in the spinal cord, when will be exhibited the phenomena of progressive muscular atrophy. Paralyses of muscles of the trunk and extremities, with contractions and without atrophy, have been observed, but these are probably complications.

Course, Duration, and Termination.—The course of the disease is progressive ; from small beginnings it grows into a formidable malady. Sometimes a stay in the progress has been noted, but only for a brief period, the course being resumed with the former intensity. The termination is fatal in from one to five years, in the mode above mentioned. An intercurrent malady may fortunately take life earlier ; pneumonia is the most usual. The frequent complication of progressive muscular atrophy, the identity of the muscular condition, and of the morbid process in the spinal cord, have led to the view, now generally accepted, that the diseases are the same, though differing as to the locality in the spinal cord affected.

Diagnosis.—Diseases of the bulb can hardly be confounded with those of other localities, because of the peculiar functional disturbances which indicate at once the seat of the mischief. Differentiation is to be made between progressive bulbar paralysis and occlusion

of the vessels, acute inflammation, and tumor. Occlusion of the vessels and inflammation occur suddenly with very severe symptoms, often apoplectic, and terminate in a few days. Such is not the behavior of progressive bulbar paralysis. Tumor of the medulla and pons comes on slowly: there are, at first, symptoms of irritation, followed by depression; in progressive paralysis, the onset is slow and obscure, but there are no symptoms of irritation, those of depression occurring at once. In the case of tumor, pressure on the cavernous sinus is exhibited in swelling of the retinal veins and "choked disks," in puffiness of the eyelids and distention of the facial vein—symptoms which do not occur in bulbar paralysis.

Treatment.—Cheadle * reports a cure by the free administration of iodide of potassium, but this must have been a case of gummata. Iodide of potassium has never arrested the progress of, much less cured, a genuine case. Galvanism is the most promising remedy. Stabile applications, the electrodes on the mastoid processes, and in the opposite direction, galvanization of the sympathetic, and applications to the lips, tongue, and fauces, should be persistently used. The current should have sufficient tension to cause slight giddiness and faint flashes of light. The *séances* should be short but daily, and, if suspended occasionally, can be kept up for the necessary period. Hydrotherapy is, next to electricity, the most useful remedy. A wet pack can be worn about the neck every night, and a hot douche may be directed to the nucha for five minutes daily, but, better, a sponge dipped in hot water and kept in contact with the back of the neck for a few minutes. The good effects of the water applications are increased by the daily use of a mustard-plaster, in contact long enough to induce a little redness and nothing more. The internal medicines have not effected any improvement in the cases thus far treated. As, under analogous conditions, the chloride of gold and sodium has been of great service, it should be given a fair trial. Bichloride of mercury acts similarly. The utility of these agents probably consists in their power to check the overproduction of connective tissue. As lead and other metals, slowly introduced into the system, will produce analogous symptoms, and as syphilis has the same effect, it is good practice in every case of progressive bulbar paralysis to give iodide of potassium, freely at first—its subsequent administration being governed by the results of the first trial. From the beginning the utmost attention should be given to the diet, so as to postpone the period of decline. Soft solids are more easily swallowed, when the palate is paralyzed, than liquids. Rectal alimentation should be resorted to when the difficulty of swallowing becomes great. The injection of defibrinated blood may be employed with advantage.

* "St. George's Hospital Reports," vol. v, p. 123.

DISEASES OF THE SPINAL MENINGES AND CORD.

HYPERÆMIA.

Definition.—As the vascular supply to the meninges and cord is the same, and as hyperæmia occurs, necessarily in both simultaneously, the term *hyperæmia* must be understood to include the contents of the spinal canal. There may be an *active*, or arterial hyperæmia; and *passive*, or venous hyperæmia.

Causes.—Hyperæmia is the first stage in the inflammatory affections, and is a notable element in variola, typhoid, and intermittent fever. It is caused by over-stimulation of the cord in the performance of its functions: for example, protracted standing or walking, excesses in coitus, etc. Certain spinal poisons cause hyperæmia, as strychnia, picrotoxine, amyl nitrite, and alcoholic excess. The arrest of such an habitual discharge as from bleeding piles, the menses, etc., diverts an excessive quantity of blood to the cord. Probably the most frequent cause is exposure of the body while in a heated and perspiring state to cold and dampness. Congestion is produced by traumatism, concussion, etc. Workmen engaged at labor in compressed air suffer from hyperæmia, due to the solution and setting free of nitrogen in the blood of the spinal canal, as Bert has shown. Venous or passive hyperæmia is caused by obstructive disease of the heart and lungs, by cirrhosis of the liver, and by tumors of the abdomen.

Pathological Anatomy.—In active hyperæmia, vessels come into view that are invisible in health, and those of larger size are enlarged, giving to the meninges and cord a distinctly congested appearance. On section, there are more bloody points than in health; and numerous points of extravasation, due to the rupture of capillary vessels, are to be seen. The spinal fluid is increased in amount, and is more or less reddish from the admixture of blood. Passive congestion is much more distinct, owing to the large size and numerous anastomoses of the vessels, which are greatly distended, more or less tortuous, and cause a bluish discoloration by the increase in size of the numerous small veins. Ecchymoses may also form in passive congestion, and the spinal fluid is somewhat increased in quantity.

Symptoms.—The symptoms are of two kinds; those of irritation and those of depression. The onset is sudden in the active form, somewhat more slow in the passive form. Pain in the back, in the dorsal or lumbar region, or both, radiates downward through hips and thighs, and is increased by movements and by percussion of the skin. The pain