to 0.01 metre in diameter (⅛ to ½ inch), rather longer than broad, with a smooth but rather dull plane, and a very smooth and shining, convex surface. The former has an excentric white nucleus, from which a brown, right-handed, spiral line reaches the edge in about two turns; the outer portion of this surface is variegated with light-brown markings, and finely striated with numerous lines running in the opposite direction. The convex surface is slightly asymmetrical, the thickest point being nearly opposite the nucleus of the spiral just mentioned. It is brownish flesh-colored in the centre, shading to white at the margin. Many specimens are much worn by the action of the sea; these are white as coral, and the spiral structure is difficult to make out. Eye-stones are composed mostly of lime carbonate, and perform entertaining movements occasioned by the discharge of carbonic acid from the surface when placed with the flat side down, in a vessel containing dilute acids. They have long been a popular resource in treating foreign bodies in the eye, for which one is placed under the lid, where it is carried around by the movements of the eye. When it comes in contact with the mote, this is carried along with the "stone" by capillary attraction, and is finally expelled with it. They are a clumsy means of doing what a small amount of skill will accomplish with far more certainty, and are deservedly nearly obsolete. The belief that they are alive and move themselves about in the eye, until they "find" the mote, is a popular fallacy. The demand for them now is very limited, and confined to the ignorant. The supply is also small and uncertain, those of this port (Boston) coming mostly in little lots from sailors who bring them from the Bahamas and elsewhere, as curiosities or private ventures. "Crab's-Eyes," lenticular concretions found in the lining membrane of the stomach of the crawfish, look much like eye-stones, and are described for them by several authors. The description just given will easily distinguish them.

W. P. Bolles.

EYES, ARTIFICIAL. See *Artificial Eyes.*

FACIAL NEURALGIA. See *Neuralgia.*

FACIAL PARALYSIS (Bell's Palsy).—Etiology.—In a large proportion of cases, peripheral facial paralysis is the result of so-called rheumatic influences, such as exposure of one side of the face to a draught, working in a damp room, or sudden chilling of the body while in a state of perspiration.

The disease may also be produced by a large number of organic lesions, which may be situated in the cranial cavity, in the course of the nerve through the Fallopian canal, or after its exit from the mastoid foramen.

The lesions within the cranial cavity which may give rise to this disease include basilar meningitis, tumors or exostoses situated at the base of the brain, syphilitic lesions in this situation, and aneurisms of the vessels of the base of the brain.

Within the Fallopian canal the paralysis may be the result of caries of the petrous portion of the temporal bone, otitis media, tumors which extend into the canal from adjacent parts, fracture of the base of the skull extending through the temporal bone, syphilitic deposits within or near the nerve, or an accumulation of wax in the external auditory canal.

After the exit of the nerve from the mastoid foramen, the paralysis may be produced by direct violence to the nerve (a slap on the face, fall, etc.), pressure upon the nerve, for example, the pressure of the forceps during parturition (a few cases have also been reported in which facial paralysis occurred during difficult and tedious labor, although the forceps had not been used), extension of inflammation to the nerve from adjacent parts (tuberculous abscesses of the glands situated near the angle of the jaw, parotitis, incised wounds during operations in this region, etc.). A case has also been reported in which the disease was produced by leukæmic infiltration of the nerve.

In comparatively rare cases facial paralysis follows certain of the infectious diseases, such as diphtheria, smallpox, erysipelas, typhoid fever. Gowers and others report cases of facial paralysis after tonsillitis, and Halschek has seen a case of double facial paralysis after mumps. This symptom has also been observed as a part of multiple neuritis. Many writers regard the disease as usually infectious in its origin. Several cases have also been reported in which the disease occurred during the secondary stage of syphilis, and was attributed to the direct action of the syphilitic virus. von Burski reports two cases in which the paralysis developed ten weeks after syphilitic infection, during the roseola.

The disease is more frequent in males than in females. It occurs very rarely during childhood and infancy.

Some writers believe that there may be a neuropathic predisposition to the disease. Browning reports the following case which bears on this point: A girl had facial paralysis after a cold; a few years later an older sister had a similar attack. The younger sister had two daughters; one of these had facial palsy after a cold, the other had a similar attack during childbed, from sleeping at an open window.

Clinical History.—Facial paralysis is almost always confined to one side of the face. In very rare cases it is bilateral (diplegia facialis), and in such cases the paralysis sometimes appears on the two sides at different times. Sometimes a similar cause operates upon both facial nerves (usually the so-called rheumatic influences); sometimes one lesion acts upon one nerve, an entirely different lesion upon the other nerve. Diplegia facialis is also a symptom of certain bulbar affections, but the consideration of the symptoms which distinguish the peripheral from the central variety will be reserved for the section on diagnosis.

Ordinary peripheral facial paralysis may begin suddenly, or it may be preceded for some time by a feeling of fulness and puffiness in the face, and by peculiar gustatory sensations on the affected half of the tongue. Rare prodromes consist of tinnitus aurium, dizziness, and pain in the face. Gowers has observed fever and temporary albuminuria. In Hoffman's case the paralysis was preceded by severe pains in the right side of the neck and face, and was attended by unconsciousness. Hence this writer believed that the attack was constitutional in origin. The paralysis may occur suddenly and completely in all branches (in rheumatic cases from a couple of hours to one or two days from the action of the exciting cause), or it may spread from one group of muscles to another (when it is the result of compression by a slowly growing external tumor or other lesion which acts in a similar manner). In the majority of cases all the muscles are affected in an approximately equal degree.

The appearance of the face is well shown in Fig. 2078. The patient, whose photograph is here shown, was directed to contract all the muscles of the face, but, as is evident from the illustration, power over the left facial muscles is entirely lost. The left eyebrow is slightly elevated, the palpebral fissure is enlarged, the naso-labial fold is effaced, the angle of the mouth droops slightly and is drawn nearer to the median line (this is observed even when the face is in repose), the mouth is kept a little open on this side, and the tip of the nose deviates slightly to the healthy side.

A patient suffering from complete facial paralysis is unable to wrinkle or corrugate the brow, on account of the paralysis of the frontalis and corrugator supercilii muscles. On making a vigorous effort to close the eyelid the globe is rolled upward and inward (or rarely outward), so that often only the sclera remains visible. But the upper lid descends a little, even when the nerve appears to be completely paralyzed. The descent of the eyelid has been attributed to a partial relaxation of the levator labii superioris, but it must be confessed that this explanation is not very satisfactory. On account of the paralysis of the orbicularis palpebrarum the puncta lachrymalia are no longer kept applied to the globe, and consequently the tears cannot make their way into the

lachrymal canal and nasal cavity. Hence, the patients suffer from overflow of tears. The paralysis of the orbicularis also prevents winking and the closure of the eye during sleep. The consequent irritation of the eyeball may give rise to conjunctivitis and keratitis, though this does not happen very often. The lower lid may become everted.

The ala nasi on the paralyzed side cannot be distended so vigorously as on the healthy side, and hence smell is somewhat impaired. This impairment of smell is increased still further owing to the fact that diversion of the tears from the nasal cavity causes dryness of the Schneiderian mucous membrane.

The angle of the mouth droops a little, and is drawn slightly toward the median line. This phenomenon is increased upon bringing into play the muscles which are inserted into the opposite angle of the mouth, as in the act of laughing. The lips cannot be closed on the affected side, and hence whistling is rendered impossible. The pronunciation of labials may be interfered with, but in many cases articulation is undisturbed. The cheek on the paralyzed side flaps loosely when the patient attempts to puff it out. Mastication is also interfered with to a certain extent, on account of the paralysis of the buccinator muscle. The food is apt to accumulate between the cheek and teeth, and must be dislodged frequently by the patient's finger. In a considerable proportion, perhaps the majority, of cases taste is impaired upon the anterior third of the tongue on the paralyzed side, and sometimes the patient complains of peculiar subjective sensations in this locality. This is owing to the fact that the chorda tympani nerve, which supplies the anterior third of the tongue with gustatory fibres, joins with the seventh nerve during its course through the Fallopian canal. Some doubt still attaches, however, to the anatomical and physiological relations of this nerve.

Fig. 2078.—Well-marked Facial Paralysis of the Left Side. (From a photograph taken at the moment when the patient was told to contract all the muscles of his face.)

The tongue is protruded in a straight line, but, on account of the displacement of the oral fissure toward the healthy side, there is an apparent deviation of the organ toward the paralyzed side.

In certain cases (*vide* the section on diagnosis) the palate and uvula are found to be paralyzed. Upon inspection the paralyzed velum palati is seen to hang lower than on the opposite side, and the uvula may be deflected to one or the other side (the latter phenomenon is sometimes observed in healthy individuals). These parts also remain almost motionless during phonation, or when reflex action is excited by irritation of the fauces. Deglutition is also interfered with to a certain extent, so that the patients often swallow the wrong way. Some writers deny the occurrence of an affection of the velum during facial paralysis. Lermoyez comes to the conclusion that the velum is innervated almost entirely by the pneumogastric.

As a rule, the sensibility of the integument remains unaffected, but in a few cases the patients complain of a feeling of numbness of the skin. In these cases, however, we have never been able to detect any objective evidences of disturbed sensation. The symptom in question is probably the result of an implication of some of the recurrent fibres of the trigeminus in their course along the facial nerve.

Hearing may be affected, sometimes as the result of the lesion which gave rise to the paralysis, sometimes as the result of the paralysis itself. In the latter event the patient suffers from hyperacusis (also called oxyokoia), *i.e.*, increased sensitiveness to auditory impressions. This has been explained by the paralysis of the stapedius muscle (which is supplied by the facial nerve), and the consequent predominance of the action of the tensor tympani (supplied by the fifth nerve). Herpes zoster is sometimes seen. Klippel distinguishes febrile zoster (infectious disease) combined with facial paralysis from the accidental herpes eruptions, which, like the accompanying facial paralysis, are due to definite lesions of central parts or of the peripheral nerve. In the former event, the eruption may be present only on the paralyzed side of the face or also in other parts, or it may not be located in the face at all. Remak reported a case, complicated with zoster of the anterior two-thirds of the tongue on the paralyzed side. The zoster recovered during the first week of the paralysis.

In some cases the muscular and nerve irritability remains normal throughout the course of the disease. Such cases usually recover spontaneously and with great rapidity.

Many cases present various stages of transition from normal electrical excitability to the complete form of degeneration reaction. Indeed, we find not infrequently that one case presents various conditions of electrical excitability in different muscles and branches of the nerve.

If the patient does not recover from the disease, the electrical irritability of the muscles gradually diminishes, and is finally abolished.

Experimental investigations have shown that the degeneration reaction is the result of certain lesions of the paralyzed nerve and muscles. When the degeneration reaction is at its height, it is found that the axis cylinder has disappeared, the medullary substance has undergone fatty degeneration, and the nuclei of the sheath of Schwann are increased in number; the interstitial connective tissue and nuclei undergo proliferation. At the same time the muscular fibrillæ are diminished in size and their nuclei increased in number; the transverse striæ are less distinct or absent, and the interstitial connective tissue is increased in amount. If recovery ensues all these changes gradually disappear.

Flatau reports a case of left facial paralysis due to chronic tuberculous otitis media, with total degeneration reaction (D.R.). Death occurred within a year from tuberculosis. The central portion of the nerve was found degenerated in its intramedullary and basal course; the cells of the left facial nucleus were swollen, misshapen, etc. The degeneration extended to the intranuclear facial fibres, the left ascending root, the efferent root, etc. These findings show that Waller's law of degeneration is no longer tenable as first laid down. The peripheral motor nerve is dependent on the cell, but is only part of a coherent, indivisible entity (the neuron), and the destruction of any part of this unit leads to changes in the entire neuron.

In another case of paralysis from middle-ear disease, Darkschewitsch and Tichonow found, on autopsy, the neurilemma unaffected; the lesions were those of purely parenchymatous neuritis.

In a few cases recovery is not complete, and spasms and contracture of the muscles are left over as sequelæ. These conditions may exist separately, but they are usually associated with one another. The spasms consist of short, quick contractions of the muscles, occurring irregularly, and very much resembling facial tic. These spasmodic movements are unattended with pain and are often unnoticed by the patient.

Contracture is observed particularly in the levator palpebræ superioris alæque nasi and zygomatici muscles, and causes retraction of the angle of the mouth upward and outward. It thus tends to overcome the original deformity produced by the paralysis. When the contracture is very marked, the deepening of the naso-labial fold to which it gives rise may create a deceptive appearance of paralysis upon the opposite side of the face. An error in diagnosis may be obviated by directing the patient to laugh, whereupon it becomes evident that the contractured muscles remain motionless, while those on the healthy side contract normally.

Gowers thinks it probable that contractures and clonic spasms are due to changes in the facial nucleus, caused by continued interruption in the nerve tracts and constant irritation of the centre during attempts to move the facial muscles. The cell resistance is diminished and hence the cells react with abnormal facility.

If complete paralysis of the muscles remains permanent, the muscles will undergo atrophy, so that the affected half of the face looks smaller than the other side.

Oppenheim has described a rare combination of peripheral facial paralysis with hysterical symptoms, viz., hysterical hemianæsthesia on the same side. The paralysis persisted but the hemianæsthesia disappeared in a short time.

In diplegia facialis the face presents a remarkable appearance inasmuch as it is absolutely devoid of expression, and even the most violent emotions are experienced by the patient without the slightest change of countenance. The lower lip droops, and saliva is constantly flowing from the mouth. Articulation and deglutition are interfered with to a very marked degree.

Diagnosis.—The differentiation of peripheral from central facial paralysis is usually quite easy. In the latter affection the muscles of the forehead and eyelid are very slightly involved. As a rule, however, on directing the patient to close only the eye on the paralyzed side, it becomes evident that this is done with less vigor and promptitude than on the healthy side. The affected muscles present no changes of electrical excitability, however profound the paralysis may be. It will also be noticed that the voluntary and the reflex contractility of the paralyzed muscles are often in marked contrast to one another, the latter being much greater than the former. Furthermore, the clinical history of the two affections is usually decisive. In the cerebral variety the facial paralysis is almost always associated with paralysis in some other part of the body, usually of the arm and leg upon the same side. In addition, cerebral facial paralysis generally develops after an apoplectic seizure, which is usually accompanied by unconsciousness.

In a certain proportion of cases the disturbance of gustation on the anterior portion of the tongue, the paralysis of the velum palati and uvula, and the history of a previous disease which may have produced a lesion of the seventh nerve, will aid us in clearing up any possible doubt in diagnosis.

In rare cases, however, facial paralysis produced by lesions of the pons Varolii presents most of the characteristics of peripheral facial paralysis. If the lesion is situated in the lower half of the pons, the facial paralysis is associated, as a rule, with hemiplegia of the opposite side of the body. When the lesion is situated in the upper half of the pons (before the decussation of the seventh nerves), the face and limbs are paralyzed on the same side of the body. The frontalis and orbicularis palpebrarum may be entirely paralyzed, as in ordinary peripheral paralysis, and, in addition, there may be marked changes in the electrical excitability of the facial nerve and muscles. But a mistake in diagnosis is usually obviated by the presence of other symptoms of a pons lesion, such as contraction of the pupils, marked difficulty in swallowing and articulation, and paralysis of various cerebral nerves (trigeminus, abducens, hypoglossus), etc.

After the diagnosis of the peripheral character of the disease has been made, we should also endeavor to determine its location more accurately. As a rule, this can be done with great certainty on account of the peculiar anatomical relations of the nerve.

If the lesion is situated above 5 (Fig. 2079), the patient will suffer from paralysis of all the facial muscles, of the uvula and velum palati, and from disturbances of hearing, but gustation will be unimpaired. This is owing to the fact that the chorda tympani nerve enters the facial nerve, in all probability, at the ganglion geniculatum

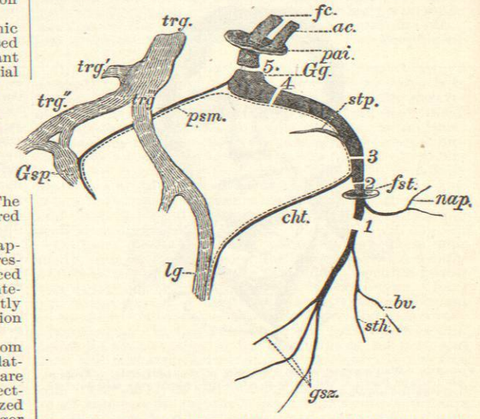

Fig. 2079.—Schematic Representation of the Ramifications of the Facial Nerve. (After Eichhorst.) *fc*, Trunk of facial nerve; *ac*, trunk of acoustic nerve; *pai*, internal auditory foramen; *gg*, ganglion geniculatum; *psm*, nervus petrosus superficialis major; *stp*, nervus stapedius; *cht*, chorda tympani; *fst*, styloid foramen; *nap*, posterior auricular nerve; *bv* and *sth*, nerves to the digastric and stylo-hyoid muscles; *trg*, *trg'*, *trg''*, *trg'''*, trunk and branches of the trigeminus; *gsp*, spheno-palatine ganglion; *lg*, lingual nerve.

through the petrosus superficialis major nerve, *psm* (from the trigeminus). The chorda tympani leaves the facial nerve between 2 and 3, *cht*, and then joins the lingual branch of the trigeminus.

If the lesion is situated between the ganglion geniculatum and the point at which the nerve to the stapedius muscle, *stp*, is given off, the symptoms will consist of paralysis of all the facial muscles, disturbances of hearing, and impairment of taste on the anterior third of the tongue. The velum palati is unaffected in this case, probably because its motor fibres are given off at the ganglion geniculatum, *Gg*. If the lesion is situated between the origin of the stapedius nerve and the point at which the chorda tympani, *cht*, leaves the facial nerve, the symptoms just mentioned will be present, with the exception of the disturbances of hearing. A lesion below the point at which the chorda tympani is given off will simply produce paralysis of the superficial muscles.

It must be remembered, however, that these remarks hold good only in those cases in which the lesion is of such character as entirely to destroy the conductibility of the nerve at the affected part.

Prognosis.—The prognosis depends chiefly on the

character of the lesion which has given rise to the paralysis, and therefore varies according to that of the primary disease. When the nerve is involved directly, as in cases of rheumatic origin, or those due to infectious diseases, etc., the prognosis may be determined, in great measure, by the changes in electrical excitability.

When the excitability of the nerve and muscles is unchanged, the disease will usually recover spontaneously in from two to four weeks. If complete degeneration reaction is present, recovery cannot be looked for in less than from three to six or nine months. But the case should not be regarded as absolutely hopeless, even if the electrical excitability is abolished for a short period.

The development of muscular spasms and contracture is a very unfortunate event. In no case of this kind which has come under our observation has recovery occurred. The contracture usually continues to increase in severity, and after a while the muscles undergo a certain amount of atrophy. These two factors may give rise to considerable disfigurement of the face.

As a rule, the disease runs a longer course in those cases in which the lesion is situated within the Fallopian canal.

Treatment.—The causal treatment varies with the nature of the primary disease (affections of the middle ear, syphilitic lesions, tumors, etc.). The use of leeches, blisters, and the administration of strychnine, which are strenuously recommended by some writers, have been attended with such unsatisfactory results in our hands that we now rely exclusively upon electrical treatment.

In those cases in which the electrical excitability of the paralyzed nerve and muscles is unchanged, treatment is unnecessary, since recovery occurs spontaneously.

When complete degeneration reaction is present, electricity should be applied to both the nerve and the muscles, and in the case of the nerve the galvanic current alone should be employed. A small electrode, the anode, is placed immediately over the mastoid foramen (between the mastoid process and the lobe of the ear) on the affected side, the cathode (with a similar electrode) over the opposite mastoid foramen. The current will therefore pass through the petrous portion of the temporal bone, and act upon the nerve in its passage through the Fallopian canal. It should not be strong enough to produce a feeling of pain or vertigo, no interruptions should be made, and the sittings should be held for three or four minutes every other day. With this method we may combine labile galvanization of the muscles. One medium-sized electrode (usually the cathode) is placed upon the back of the neck, the other small electrode is slowly passed over the affected muscles. The frontalis and corrugator supercilii muscles are brought into play by passing the electrode horizontally across the forehead, a little above the eyebrow. The levator labii superioris alæque nasi, zygomatici, and buccinator are brought into play by passing the electrode along the side of the nose (beginning near the inner angle of the eye), and then outward across the cheek, immediately below the malar bone. Labile applications may also be made directly to the orbicularis oris and chin muscles. In applications to the orbicularis palpebrarum, we are in the habit of placing a very small electrode upon the muscle, at the outer angle of the eye, and then interrupting the current (by means of an interruptor in the handle of the electrode).

In an apparently incurable case of facial paralysis due to a gunshot wound, Faure and Furet stitched the peripheral extremity of the facial nerve to the adjacent branch of the spinal accessory. The results in this case were not very satisfactory, but in view of the experiments made by various observers it is probable that surgical interference will prove useful in a limited number of cases.

In some cases the excitability of the muscles will be found to have sunk to such a low ebb that it becomes necessary to employ the intrabuccal method of galvanization. One electrode is then placed upon the muscle which we desire to stimulate, the other directly opposite upon the mucous membrane of the cheek.

After the nerve has recovered its electrical excitability, the faradic current may be employed, either upon the muscles themselves, or by simply passing the electrode along a vertical line immediately in front of the ear, in order to stimulate the pes anserinus as it spreads out in this locality.

We may attempt to relieve contractures by stabile galvanization of the affected muscles, the negative pole being applied to the mastoid foramen, the positive pole to the contractured muscle. Massage of the muscles has also been employed for this purpose.

In all electrical applications in this disease, the electrodes should be very thoroughly moistened, and the current should merely possess sufficient strength to produce visible muscular contractions. *Leopold Putzel.*

FÆCES.—The term "fæces" is applied to the excrementitious substance that normally leaves the body through the anus. The material consists of the waste from the intestinal walls and the unabsorbed residues of the various secretions into the alimentary tract, together with an admixture, in varying amounts, of undigested and partially digested food, and at times such adventitious substances as may be taken into the alimentary canal *per os* and which are not absorbed. The common idea that the fæces in health consist chiefly of food residue is not sustained by the results of recent investigations.

After the intestinal contents have passed the ileo-cæcal valve, they rapidly take on the characteristic consistence, color, and odor of fæcal matter, being altered by the absorption of the liquids and soluble substances, as well as, to some extent, by the continuation of putrefactive and fermentative processes and by admixture with the strongly alkaline secretions of the large intestine. The extent of these changes is largely influenced by the rapidity with which the contents pass through the colon.

Physical Characters.—The *quantity* passed in twenty-four hours varies greatly, but is estimated to average 120–200 gm., in a healthy man on a mixed diet. It is dependent to some extent on the quantity and quality of food eaten, but is influenced as well by the condition of the digestive organs as regard secretion and absorption, and the frequency of evacuation. As a rule the quantity of fæces increases with the relative increase in the amount of vegetable food in the diet. It is probable that this is due largely to the greater resistance of vegetable food to digestive action and the subsequent stimulation of peristalsis by the undigested residues, with a resulting diminution of absorption. In an infant fed on cow's milk the quantity is regularly considerably greater, even up to ten times, than the amount passed by a breast-fed babe. This is due not alone to difference in digestibility, but to a considerable extent to the much larger quantity of milk given in artificial feeding, which both adds to the quantity of undigested food residue and increases the residue from intestinal secretion and waste. The quantity of fæces evacuated is occasionally increased by pathological products, including mucus, blood, pus, and serous fluid.

The *color* is normally derived from the intestinal secretions, notably from the bile, and under certain conditions is also very considerably influenced by the food, the stools of a mixed diet usually being a medium shade of brown, those of a meat diet dark to blackish-brown, and the evacuations following the exclusive ingestion of vegetable food being a light brown. The stools of an exclusive milk diet vary in color from orange to light yellow. The dark color is due to hæmatin, and to some extent to the compact, dry condition of the fæcal matter. In particular instances the color may be modified by special coloring matters ingested with the food, as, for example, chlorophyl, derived from large quantities of green vegetables. Adventitious ingesta as, *e.g.*, certain medicaments, may determine the color. Thus, calomel may cause a green coloration, probably by causing the presence of biliverdin, and bismuth or iron may each be reduced or may exceptionally appear as the black sulphide. Again, unusual changes in the intestinal contents may