rection—e. g., outward—with paralysis of the opposite internal rectus. This is known as secondary deviation. It depends upon the fact that, if two muscles are acting together, when one is weak and an effort is made to contract it, the increased effort—innervation—acts powerfully upon the other muscle, causing an increased contraction.

(*d*) *Erroneous Projection.*—"We judge of the relation of external objects to each other by the relation of their images on the retina; but we judge of their relation to our own body by the position of the eyeball as indicated to us by the innervation we give to the ocular muscles" (Gowers). With the eyes at rest in the mid-position, an object at which we are looking is directly opposite our face. Turning the eyes to one side, we recognize that object in the middle of the field or to the side of this former position. We estimate the degree by the amount of movement of the eyes, and when the object moves and we follow it we judge of its position by the amount of movement of the eyeballs. When one ocular muscle is weak, the increased innervation gives the impression of a greater movement of the eye than has really taken place. The mind, at the same time, receives the idea that the object is further on one side than it really is, and in an attempt to touch it the finger may go beyond it. As the equilibrium of the body is in a large part maintained by a knowledge of the relation of external objects to it obtained by the action of the eye muscles, this erroneous projection resulting from paralysis disturbs the harmony of these visual impressions and may lead to giddiness—ocular vertigo.

(*e*) *Double Vision.*—This is one of the most disturbing features of paralysis of the eye muscles. The visual axes do not correspond, so that there is a double image—diplopia. That seen by the sound eye is termed the true image; that by the paralyzed eye, the false. In simple or homonymous diplopia the false image is "on the same side of the other as the eye by which it is seen." In crossed diplopia it is on the other side. In convergent squint the diplopia is simple; in divergent it is crossed.

Ophthalmoplegia.—Under this term is described a chronic progressive paralysis of the ocular muscles. Two forms are recognized—ophthalmoplegia *externa* and ophthalmoplegia *interna*. The conditions may occur separately or together and are described by Gowers under nuclear ocular palsy.

Ophthalmoplegia Externa.—The condition is one of more or less complete palsy of the external muscles of the eyeball, due usually to a slow degeneration in the nuclei of the nerves, but sometimes to pressure of tumors or to basilar meningitis. It is often but not necessarily associated with ophthalmoplegia interna. Siemerling, in the recent monograph in which he has analyzed the material (eight cases) left by the late Prof. Westphal, states that sixty-two cases are on record. In only eleven of these could syphilis be positively determined. The levator muscles of the eyelids and the superior recti are first involved, and gradually the other

muscles, so that the eyeballs are fixed and the eyelids droop. There is sometimes slight protrusion of the eyeballs. The disease is essentially chronic and may last for many years. It is found particularly in association with general paralysis, locomotor ataxia, and in progressive muscular atrophy. Mental disorders were present in eleven of the sixty-two cases. With it may be associated atrophy of the optic nerve and affections of other cranial nerves. Occasionally, as noted by Bristowe, it may be functional.

Ophthalmoplegia Interna.—Jonathan Hutchinson applied this term to a progressive paralysis of the internal ocular muscles, causing loss of pupillary action and the power of accommodation. When the internal and external muscles are involved the affection is known as total ophthalmoplegia, and in a majority of the cases the two conditions are associated. In some instances the internal form may depend upon disease of the ciliary ganglion.

While, as a rule, ophthalmoplegia is a chronic process, there is an acute form associated with hæmorrhagic softening of the nuclei of the ocular muscles. There is usually marked cerebral disturbance. It was to this form that Wernicke gave the name polio-encephalitis superior.

Treatment of Ocular Palsies.—It is important to ascertain, if possible, the cause. The forms associated with locomotor ataxia are obstinate, and resist treatment. Occasionally, however, a palsy, complete or partial, may pass away spontaneously. The group of cases associated with chronic degenerative changes, as in progressive paresis and bulbar paralysis, is little affected by treatment. On the other hand, in syphilitic cases, mercury and iodide of potassium are indicated and are often beneficial. Arsenic and strychnia, the latter hypodermically, may be employed. In any case in which the onset is acute, with pain, hot fomentations and counter-irritation or leeches applied to the temple give relief. The direct treatment by electricity has been extensively employed, but probably without any special effect. The diplopia may be relieved by the use of prisms, or it may be necessary to cover the affected eye with an opaque glass.

IV. Fifth Nerve.

Paralysis may result from: (*a*) Disease of the pons, particularly hæmorrhage or patches of sclerosis. (*b*) Injury or disease at the base of the brain. Fracture rarely involves the nerve; on the other hand, meningitis, acute or chronic, and caries of the bone are not uncommon causes. (*c*) The branches may be affected as they pass out—the first division by tumors pressing on the cavernous sinus or by aneurism; the second and third divisions by growths which invade the spheno-maxillary fossa. (*d*) Primary neuritis, which is rare.

Symptoms.—(*a*) *Sensory Portion.*—Paralysis of the fifth nerve causes loss of sensation in the parts supplied, including the half of the

face, the corresponding side of the head, the conjunctiva, the mucosa of the lips, tongue, hard and soft palate, and of the nose of the same side. The anæsthesia may be preceded by tingling or pain. The muscles of the face are also insensible and the movements may be slower. The sense of smell is interfered with. There is loss of the sense of taste. There are, in addition, *trophic* changes; the salivary, lachrymal, and buccal secretions may be lessened, abrasions of the mucous membranes heal slowly, and the teeth may become loose. The eye inflames, the corneæ become cloudy and may ulcerate. These latter symptoms occur only when the Gasserian ganglion is affected, as the nerve itself may be involved for years without producing ophthalmia. Herpes may develop in the region supplied by the nerve and is usually associated with much pain. It is most common in the upper branch of the nerve. The pain which follows the herpes may be peculiarly enduring, lasting for months or years (Gowers).

(*b*) *Motor Portion.*—The inability to use the muscles of mastication on the affected side is the distinguishing feature of paralysis of this portion of the nerve. It is recognized by placing the finger on the masseter and temporal muscles, and, when the patient closes the jaw, the feebleness of their contraction is noted. If paralyzed, the external pterygoid cannot move the jaw toward the unaffected side; and when depressed, the jaw deviates to the paralyzed side. The motor paralysis of the fifth nerve is almost invariably a result of involvement of the nerve after it has left the nucleus. Cases, however, have been associated with cortical lesions. Hirt concludes, from his case, that the motor centre for the trigeminus is in the neighborhood of the lower third of the ascending frontal convolution.

Spasm of the Muscles of Mastication.—Trismus, the masticatory spasm of Romberg, may be tonic or clonic, and is either an associated phenomenon in general convulsions or, more rarely, an independent affection. In the tonic form the jaws are kept close together—lock-jaw—or can be separated only for a short space. The muscles of mastication can be seen in contraction and felt to be hard and the spasm is often painful. This tonic contraction is an early symptom in tetanus, and is sometimes seen in tetany. A form of this tonic spasm occurs in hysteria. Occasionally trismus follows exposure to cold, and is said to be due to reflex irritation from the teeth, the mouth, or caries of the jaw. It may also be a symptom of organic disease due to irritation near the motor nucleus of the fifth nerve.

Clonic spasm of the muscles supplied by the fifth occurs in the form of rapidly repeated contractions, as in "chattering teeth." This is rare apart from general conditions, though cases are on record, usually in women late in life, in whom this isolated clonic spasm of the muscles of the jaw has been found. In another form of clonic spasm sometimes seen in chorea, there are forcible single contractions. Gowers mentions an instance of its occurrence as an isolated affection.

(*c*) *Gustatory.*—Loss of the sense of taste in the anterior two thirds of the tongue, as a rule, follows paralysis of the fifth nerve. The gustatory fibres pass from the chorda tympani to the lingual branch of the fifth. Disease of the fifth nerve is, however, not always associated with loss of taste in the anterior part of the tongue, in which case either the taste fibres escape, or the disease is within the pons where these fibres are separate from those of sensation.

The *diagnosis* of disease of the trifacial nerve is rarely difficult. It must be remembered that the preliminary pain and hyperæsthesia are sometimes mistaken for neuralgia. The loss of sensation and the palsy of the muscles of mastication are readily determined.

Treatment.—When the pain is severe morphia may be required and local applications are useful. If there is a suspicion of syphilis, appropriate treatment should be given. Faradization is sometimes beneficial.

V. Facial Nerve.

Paralysis (*Bell's Palsy*).—The *portio dura* of the seventh pair may be paralyzed by (1) lesions of the cortex—supranuclear palsy; (2) lesions of the nucleus itself; or (3) involvement of the nerve trunk in its tortuous course within the pons and through the wall of the skull.

I. *Supranuclear Paralysis*, due to lesion of the cortex or of the facial fibres in the corona radiata or internal capsule, is, as a rule, associated with hemiplegia. It may be caused by tumors, abscess, chronic inflammation, or softening in the region of the internal capsule. It is distinguished from the peripheral form by two well-marked characters—the persistence of the normal electrical excitability of both nerves and muscles and the absence of involvement of the upper branches of the nerve, so that the orbicularis palpebrarum and frontalis muscle are spared. A third difference is that in this form the voluntary movements are more impaired than the emotional. There are instances of cortical facial paralysis—monoplegia facialis—associated with lesions in the centre for the face muscles in the lower Rolandic region. Isolated paralysis, due to involvement of the nerve fibres in their path to the nucleus, is uncommon. In the great majority of cases supranuclear facial paralysis is part of a hemiplegia. Paralysis is on the same side as that of the arm and leg because the facial muscles bear precisely the same relation to the cortex as the spinal muscles. The nuclei of origin on either side of the middle line in the medulla are united by decussating fibres with the cortical centre on the opposite side (see Fig. 3).

II. The *nuclear paralysis* caused by lesions of the nerve centre in the medulla is not common alone; but is seen occasionally in tumors, chronic softening, and hæmorrhage. In rare instances of anterior polio-myelitis the facial nucleus is affected. In diphtheria this centre may also be involved. The symptoms are practically similar to those of an affection of the nerve fibre itself—infranuclear paralysis.