

*Hepatic colic* has already been referred to, and *enteralgia*, *hepatalgia*, and *gastralgia*, *nephritic colic*, *lead poisoning*, &c., have been considered under the diagnosis of *enteralgia* (p. 182).

Cirrhosis, cancer, hydatids, abscess, acute yellow atrophy, and congestion, are diseases of the liver—the more important, or more striking diagnostic symptoms of which may usefully be contrasted. *Cirrhosis of the liver*.—There is a history of alcoholism, and frequent attacks of gastric catarrh, with much flatulence. The jaundice is very slight and is slowly developed. The liver is almost always diminished in size, and no globular tumour or nodules can be felt. The early symptoms are generally attributed to congestion, and later, the development of ascites—with the other symptoms associated with cirrhosis—confirms the diagnosis. The spleen is enlarged, and the urine is deficient in urea, but may contain leucin and tyrosin. In *cancer of the liver* the age, cachexia and emaciation, with enlargement of the liver and development of nodules, and absence or diminution of hydrochloric acid, are differential points. *Hydatid disease of the liver* produces enlargement, and sometimes a globular tumour can be felt. The development is slow, without fever, and it is generally painless. Palpation of the tumour reveals the characteristic, elastic fluid feeling, and possibly “thrill.” It is quite different from the hard nodules of cancer. It is more like an abscess; but in the latter case there will have been great fever and rigors. An enlargement of the gall-bladder may simulate a hydatid closely; but the seat, and the history, should suggest the diagnosis. An aneurism of the hepatic artery gives rise to a heaving expansile pulsation. A hydatid cyst pushing up the diaphragm simulates, very closely, a pleuritic effusion, but in such cases the use of the aspirator can at once clear up all doubt.

*Inflammation and abscess of the liver* is distinguished by the history of rigors, tenderness on pressure, and the presence of some causal affection—as dysentery or malaria. It may be confounded with *hydatid disease*, *enlargement of the gall-bladder*, *cancer*, *abscess of the abdominal wall*, and *purulent pleuritic effusions*. The first three have been already considered. An abscess of the abdominal wall does not disturb the liver functions. In empyema the history at the commencement of the disease is important. The use of the aspirator will clear up many doubtful cases. In *acute yellow atrophy* of the liver, the extreme wakefulness and headache, the sudden rise of the temperature, jaundice, and diminution of the size of the liver—are the striking features of the case. It occurs most frequently in pregnant women.

*Congestion of the liver* may be mistaken for catarrh of the bile ducts; but in the former disease the jaundice follows the liver symptoms, while in the latter it is usually preceded by the symptoms of gastro-duodenal catarrh. The passive forms of congestion due to heart and lung disease, have also to be noted.

## CHAPTER X.

## DISEASES OF THE NERVOUS SYSTEM.—Section I.

**Contents.**—Neuralgia—*Tic-douloureux*, *Sciatica*, *Herpes zoster*—**Neuritis**—**Raynaud's disease**—Peripheral paralysis—*Paræsthesiæ*—*Causalgia*, &c.—**Electricity**—*Ziemssen's motor points*—Paralysis of the cranial nerves—Facial paralysis—**Scheme of the brain and spinal cord**—Paraplegia, hemiparaplegia and hemianaesthesia—**Acute and chronic myelitis, and softening of the cord**—*Compression paraplegia*—*Secondary degenerations of the spinal cord*—*Spastic spinal paralysis*—*Combined scleroses*—*Alcoholic, syphilitic, hysterical, and reflex paraplegia*—*Spinal weakness, irritation, anæmia, and congestion*—Landry's acute ascending paralysis—Locomotor ataxia—Acute and chronic spinal meningitis—*Poliomyelitis anterior acuta* (infantile paralysis)—*Poliomyelitis anterior sub-acuta vel chronica*—*Progressive muscular atrophy*—(*Sclérose latérale amyotrophique* and *pachyméningite cervicale hypertrophique*)—*Pseudo-hypertrophic paralysis*—*Bulbar paralysis*—*Multiple sclerosis*—*Paralysis agitans*.

**Neuralgia.**—The changes which take place in the nerve trunks or within the nuclei of the cerebro-spinal centres, in cases of neuralgia, are still unknown. Pains of all kinds—as pleuritic and cardiac pains, renal and hepatic colic, peritonitis, &c.—are conveyed to the cerebral centres by the sensory nerves; but these are not considered as neuralgic pains, clinically, although some of them are really of that nature. Neuralgia is a pain, usually periodic, connected with a nerve, and which may arise from obscure changes within the spinal or cerebral centres, and be associated with a nerve trunk; but it is not easy to define it accurately, although a very common clinical entity. The pain may also arise from a peripheral irritation, and it is then a “reflex neuralgia”—e.g., caries of the teeth. Pressure upon a nerve trunk by growths, tumours, or bony excrescences, gives rise to severe pain—the pain being often referred to the peripheral extremities of the nerve. Neuralgia usually affects one side only. It is very rarely bilateral; and a characteristic symptom is the presence of “painful points” in the course of a nerve, aggravated by pressure, and generally found where the nerve issues from a bony canal, or pierces the fascia. A certain amount of anæsthesia of the skin supplied by the nerve affected, is almost invariably present in

cases of neuralgia. The constitutional condition is an important factor in the causation of neuralgia, and hence the toxic influences, or diathetic agents, in gout, diabetes, syphilis, rheumatism, lead poisoning, and malarial diseases, &c., must be considered in the treatment.

**Neuralgia of the Fifth Nerve.**—*Trifacial neuralgia, Tic-douloureux.*—The severe form of this affection is somewhat rare, but the milder forms are common. The pain is paroxysmal, and attacks the patient with great suddenness, and it is extremely violent. The whole of the nerve is not always affected. The muscles of one side of the face are contorted, and the expression indicates the intense suffering. The paroxysms may only last a few seconds or a minute, but they recur from time to time. The attack may extend over a few hours, with remissions extending over a few days, or longer. In the chronic cases, the whiskers or beard, on one side, may be worn off by the constant friction to which the face is subjected during the painful seizures; and even the configuration of the bones may become flattened, and the expression altered, in the very protracted cases. The "tender points" are well marked in these cases, and pressure upon a tooth, or a cold draught, may excite an attack. The severe type of trifacial neuralgia is often associated with mental disease, and with migraine. It is believed never to arise from any reflex causes. It is sometimes due to "impaired nutrition of the neuron from an obliterating arteritis." The milder forms of neuralgia of the fifth nerve are frequently reflex. Chronic inflammation of a tooth pulp is the commonest cause; but other forms of dental disease give rise to it. The dentist should be consulted, and a thorough examination of the teeth should be made in all cases of facial neuralgia. The possibility of an early morbid growth pressing upon the nerve trunk has also to be remembered.

Neuralgia may affect other nerves; but these need not be described in detail. "Cervico-occipital" and "cervico-brachial" neuralgia may be noted. Neuralgic pains in the arm may be due to reflex causes; and dental disease is frequently a cause of these pains. Disease of the articular processes of the cervical vertebræ is also a possible cause, and so is aneurism of the subclavian or axillary arteries.

**Sciatica** is a very common neuralgia, differing from other forms inasmuch as the pain is more or less constant, with severe paroxysms of "lightning" pain. In severe cases the pain sometimes radiate into the sciatic nerve of the opposite side, and it is much increased by pressure. The "tender points" may be readily discovered, and the whole length of the nerve is unduly sensitive. In the chronic and severe cases there is wasting of the muscles, partial anæsthesia of the skin, and the knee and hip-joints become permanently flexed. The chief causes of sciatica are exposure to cold, and over-fatigue—besides the cases which may arise from pressure on the nerve by sacral and other growths. Fæcal accumulations, when excessive, may give rise to the milder forms, and certainly constipation aggravates the pain in all cases of sciatica, whatever the original cause.

In the *diagnosis* of sciatica, when the pain is in the calf, an examination for femoral thrombosis should be made. Hip-joint disease can be excluded by testing the joint by pressure. Very advanced cases of sciatica may be mistaken for spinal disease. The possibility of sacral growths and tumours pressing on the sacral plexus must be noted. Pathologically, sciatica is, sometimes, a *neuritis*.

The *duration* of neuralgia is variable, and it depends upon the amenability to treatment. *Tic-douloureux* is most obstinate, and may be incurable. The disease may last for years. In sciatica the improvement is often very slow. Some cases extend over a year with more or less pain at intervals. The mild cases may cure in a week or two; but some of the severe cases extend over a long period of time, and render the patient totally unfit for any employment. Neuralgia—however wearing out it may be—takes long to produce death by exhaustion.

**Herpes Zoster. Zona. Shingles.**—This affection is often included among the skin diseases, owing to its being associated with a vesicular eruption; but it is believed to be due to morbid changes within the ganglia of the posterior nerve roots, and in adults, at least, it is almost always accompanied by severe neuralgic pain. The pain generally precedes the eruption, which consists of groups of papules, which very soon become vesicles, drying up in about a week. Sometimes the vesicles become purulent, and the dried-up pustules may leave small cicatrices. The early groups are those nearest the origin of the nerve, and these may often be seen healing up while the later groups, following the distribution of the nerve, are in the stage of maturation. The vesicles sometimes coalesce and leave large purulent and eczematous looking patches. The groups vary in number from one or two to eight or ten. Three clusters are a very common number, when an intercostal nerve is the seat of the disease. This is perhaps the commonest site; but the nerves of the arm or leg, and the cervical or facial nerves often suffer. Herpes zoster is almost invariably unilateral. The pain often continues for a long time after the disappearance of the eruption. The causes are unknown; but it has been observed to follow the use of arsenic, medicinally, in a few cases. In the *diagnosis* of herpes zoster the most important differentiation is that of erysipelas, when the nerve affected is the first division of the fifth. When seen early there is no difficulty, but when the upper eyelid is swollen and the redness is diffused, a mistake may be made. The history of a unilateral lesion, and the absence of a high temperature and constitutional disturbance, are the points to be noted. Ulceration of the cornea and iritis frequently attend this form of shingles, and the eyesight may be permanently damaged. In other cases—when seen late—the occurrence of purulent-looking patches, instead of groups of vesicles, may appear puzzling; but not when there are several of them in the course of a nerve. When an intercostal nerve is affected, simple pleurodynia, intercostal neuralgia, caries of the ribs, and pleurisy, have to be excluded—in the early stages, before the appearance of the eruption.

**The Treatment of Neuralgia.**—All reflex causes, as caries of the teeth, should be removed. Five grain doses of quinine—repeated twice within four hours, at the time when the attack is expected—will often ward off the paroxysm. The quinine has also a curative effect. Fifteen grains of antipyrin may be used in the same way. If a constitutional cause be suspected, iodide of potassium, in five or ten grain doses, should be prescribed. Gelsemium and aconite are also recommended. Exalgine has of late gained a great reputation in the treatment of neuralgia (B 54). The hypodermic injection of morphia (half a grain, if necessary) or of pure chloroform (five to ten minims) is highly beneficial—not only as a palliative, but, ultimately, as a curative measure. The hypodermic injections are often imperative, especially in cases of sciatica. Galvanism may also be tried in persistent neuralgia. Arsenic, iron, and cod-liver oil are often strongly indicated. In herpes zoster, the patches of vesicles should be treated with zinc ointment, and covered with cotton wool and collodion. In sciatica, constipation should be actively treated; and surgical measures may be necessary (blistering, Corrigan's button, nerve stretching, sciatica needles, &c.).

Neuritis, or inflammation of a nerve trunk, may result from wounds or injuries, and sometimes from direct exposure to cold. Often it is caused by syphilitic deposits—especially in the cranial nerves—pressing directly upon the nerve fibres. The latter form is generally chronic in character and it leads to atrophy and degeneration of the nerve trunks; but the acute forms begin with hyperæmia and pass on to suppuration and softening. Recovery may take place even after extensive inflammation.

The *symptoms* begin with chilliness and headache, with severe pain in the line of the nerve shooting to the peripheral distribution. There is often cramp of the muscles supplied by the nerve; anæsthetic areas arise from destructive changes within the nerve trunk, in the later stages. When chronic changes ensue, the muscles waste, and certain "trophic" changes appear—as loss of hair or nails, herpes, or "glossy skin," &c. The electrical reactions become abnormal. The commonest form of *multiple neuritis* is the *paralytic*, due to toxic agents—alcohol being the most frequent. In alcoholic neuritis the disease may manifest itself suddenly, but it runs a chronic course. Sometimes there are prodromata, as cramps, numbness, slight pains, and weakness, with other subjective symptoms. The muscles are excessively tender to pressure. The skin of the fingers, &c., may become red or œdematous. The knee and elbow jerk are lost. The most marked sign of the paralysis is the foot and wrist drop—the feet being slightly œdematous, and the muscles atrophied. There is tactile anæsthesia, and often hyperalgesia. Temperature and pain sense are lessened. The muscles give the reaction of degeneration. The mental condition is sluggish, with confusion of thought, and sometimes slight delirium. There may be retinal hyperæmia, or even, sometimes, optic neuritis. The sphincters are not permanently involved. Contractures of the limbs may take place in severe and advanced cases. In the *sensory* or *pseudotabetic*

form of neuritis there is less paralysis and more pain. The paresis, muscular wasting, trophic changes, and electrical reactions distinguish it from loco-motor ataxia. This form is more likely to be caused by diabetes, infectious disease, or metallic poisoning—as arsenic and lead. An endemic and epidemic form exists (Beriberi), in which there is more œdema, serous effusions, and gastro-intestinal disturbance. A fourth form is the *acute pernicious*, which is very rapid and fatal. A septic cause is believed to exist. Landry's acute ascending paralysis is of this nature (see p. 228). Raynaud's disease (symmetrical angio-neurotic gangrene) may be discussed here, as a neuritis—believed, however, to be secondary—has been found. There is spasm of the vessels of the extremities, coldness, waxiness of toes or fingers, or blueness or swelling followed by dry gangrene. The disease comes on suddenly. Senile gangrene, frostbite, &c., must be remembered in the diagnosis. The prognosis is favourable. The treatment consists of galvanism, warmth, and tonics.

The *duration* is short in the acute form, and recovery may be complete or partial. In the chronic forms, the electrical reactions are a guide to the course and prognosis. The *treatment* consists of rest, and the limbs may be painted with menthol, and enveloped in wadding. Morphia should be injected; and quinine or phenacetin may be given in large doses. In the chronic forms, galvanism should be used; and large doses of iodide of potassium should be administered.

**Peripheral Paralysis.**—Paralyses of the cranial nerves are of most interest to the physician; but any nerve, or group of nerves, may be affected, and the symptoms will vary according to the distribution of their branches to the muscles. A few examples of these may conveniently be introduced here.

Injuries to the brachial plexus, or simple pressure upon the nerves as in "crutch paralysis," may be followed by more or less loss of power and sensation in the arm. Stretching the nerves—as frequently happens in the young, from active gymnastic exercise—may produce a partial loss of power (paresis) with tingling sensations in the limb. Sleeping with the arm over a chair is another familiar instance, with similar symptoms. In more obscure cases, aneurism, bony growths, or tumours must be carefully searched for. Caries in a wisdom tooth has been known to cause severe pain in the arm, with partial paralysis. This is of the nature of a "reflex" paralysis, and it disappears with the removal of the cause.

Paralysis of the *serratus magnus* supplied by the *posterior* or *long thoracic nerve*, may be produced in certain trades by over-exertion, or by a blow upon the shoulder. The "wing-like" projection of the scapula, and the inability to raise the arm above the shoulder, are the prominent symptoms.

Paralysis of the muscles supplied by the *circumflex nerve*, is also common. The deltoid is the chief muscle affected, and the patient cannot lift his arm from the side. Disease of the shoulder joint should be noted in the diagnosis of this form.

Other examples might be given, but it will be obvious that only anatomical knowledge of the nervous distribution is necessary, to suggest the seat of the lesion and to properly interpret the symptoms. It should be noted that the unopposed contraction of the sound (antagonistic) muscles produces contractions and deformities; and that a lesion of one nerve may soon spread to other nerves. The electrical re-actions of the nerves and muscles are modified in paralysis (see p. 206).

**Anæsthesia** (loss of common sensation), **analgesia** (loss of painful sensation), **hyperæsthesia** (increased sensation), and **causalgia** (burning sensation) are morbid conditions, affecting the sensory fibres of the nerves, and they are often associated with paresis and paralysis of the nerve trunks. They are also, however, just as frequently symptoms of diseases of the brain and spinal cord. The anæsthesia may be measured by means of the æsthesiometer, with due regard to the degrees of sensibility of the different parts of the body. These abnormal sensations in affections of mixed sensory and motor nerves, arise from the same causes which produce the paralysis; but they are sometimes absent, and they often, when present, differ in degree—the paralysis being often great while the sensory fibres are only slightly affected.

**Paræsthesia** is the name given to a group of subjective symptoms (prickling, numbness, formication, tickling, burning) frequently presenting a clinical condition—as much a disease as neuralgia. There is less irritation in the nerve than in the latter affection. It occurs in *neurasthenia*, or it may be the result of toxic or diathetic agents.

**Causalgia** is believed to be a morbid condition of the peripheral end organs, and it is frequently associated with a "glossy" state of the skin. Cold applications relieve the pain. It is a functional disorder which, in many cases, is soon cured, or gradually passes away; but sometimes a severe type is met with, in which there is ulceration of the skin, painless whitlows, and shedding of the nails. (Morvan's disease: analgesic paralysis with whitlows.) Affections of the nerve trunks are sometimes—as in spinal disease—followed by painful swellings of the joints. In some cases they may end in ankylosis and deformity of the limbs.

The diagnosis of affections of the nerve trunks is generally obvious; but *progressive muscular atrophy* must be noted. All possible causes of compression of the nerve must be looked for—as tumours, &c. *Reflex* causes must also be excluded. In the lower limbs, a pelvic tumour may compress the sacral plexus and simulate a case of spinal disease.

The **prognosis** is favourable in the majority of the simple affections of the nerve trunks. The paralysis generally passes off in a few weeks provided the cause can be removed.

The **treatment** of the peripheral paralyses is suggested by the cause. Electricity, rest, counter irritation, and the removal of reflex causes are necessary. The electrical re-actions are important in estimating the probable rate of recovery.

The following figures (26, 27, 28, 29, 30) show the chief motor points and indicate the muscles supplied by them:—

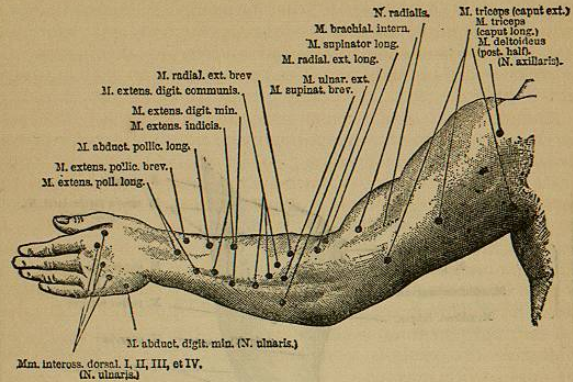


Fig. 26.—Motor points of the radial nerve and the muscles supplied by it; dorsal surface (from Landois and Stirling's *Physiology*).

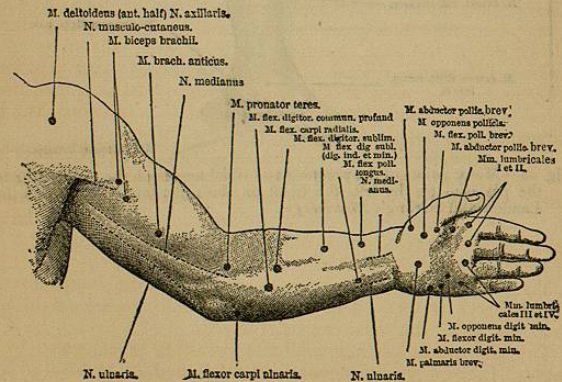


Fig. 27.—Motor points of the median and ulnar nerves, with the muscles supplied by them (from Landois and Stirling's *Physiology*).

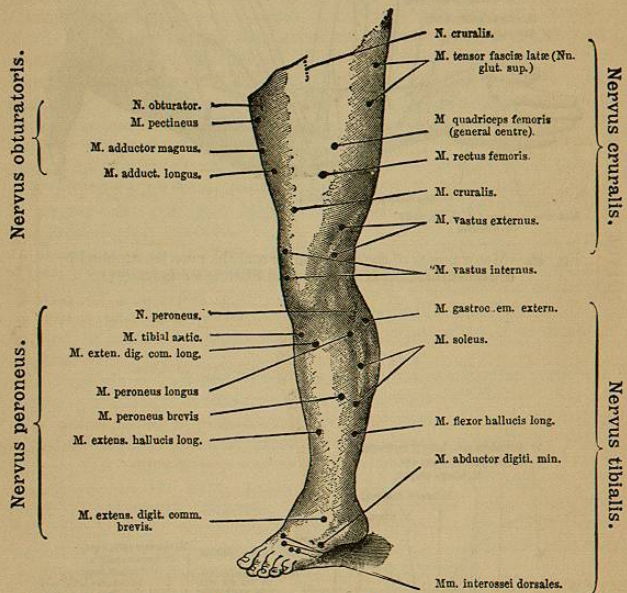


Fig. 28.—Motor points of the peroneal and tibial nerves on the front of the leg; the peroneal on the left, the tibial on the right (after *Eichhorst*) (from Landois and Stirling's *Physiology*).

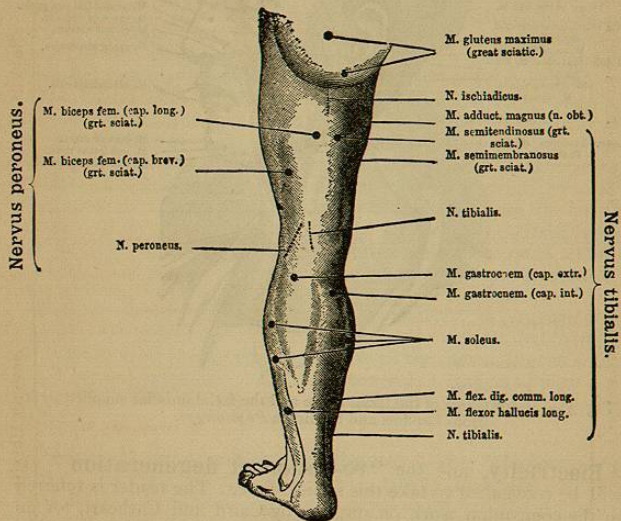


Fig. 29.—Motor points of the sciatic nerve and its branches; the peroneal and tibial nerves (from Landois and Stirling's *Physiology*).

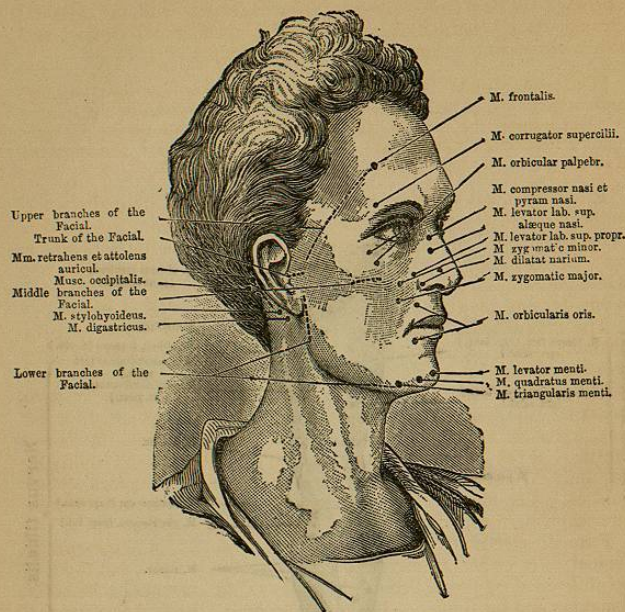


Fig. 30.—Motor points of the facial nerve and the facial muscles supplied by it (from Landois and Stirling's *Physiology*).

**Electricity**, and the “re-action of degeneration” It will be convenient to take this subject here. The reader is referred to the companion work on surgery, by Caird and Cathcart, for an elementary outline of electricity. Such works as *De Watteville's*, and *Stevenson & Lewis's*, may also be consulted.

**Galvanism** consists of the use of the “primary, continuous, constant, or galvanic” current. When using it the skin should be made moist with salt and water. The one pole is placed upon the sternum or spine, while the other is pressed upon the motor point of the muscle it is desired to test.

At the beginning a very weak current should be used, and this should be gradually strengthened. The normal re-actions will be found to be as follow, viz. :—

A *weak* current produces a slight contraction at the *cathodal closure*, when *no* contraction occurs at the anodal opening or closing, or at the cathodal opening. A *strong* current produces a very strong contraction with the *cathodal closure*, and weaker contractions at the anodal opening and closing, and still *no contraction* with the cathodal

opening. A *very strong current* produces tetanus with the cathodal closing, strong contractions at the anodal opening and closing, and *now* a weak contraction at the cathodal opening.

[The terms “make” and “break” are sometimes used for “closing” and “opening.” The current is *made or closed*, the moment the sponge is placed upon the muscle (the other pole being always held firmly against the spine or sternum); and it is *broken or opened*, the moment it is lifted off.]

Erb's “re-action of degeneration” consists of a “qualitative” change in the galvanic re-actions. *Instead* of the cathodal closing contraction being the first to appear, it is the *anodal closing* contraction, which may be as strong as, or even greater than, the cathodal closing; while the *cathodal opening* contractions (which are the last to appear normally) *now* may appear *before* the anodal opening contractions.

The contractions themselves are also *slower* and more *tonic* in character than the normal, and sometimes only *half the number of cells* are required to produce them. Powerful *faraadic currents* often fail to produce contraction when the abnormal galvanic re-actions are present. Erb believes that the abnormal re-action depends, in some way, upon the separation of the affected structures from the nerve centres. If the nerve should not become restored, the excitability to galvanic currents is diminished, and ultimately it becomes extinguished. The muscles, in many cases, regain their normal re-action after showing for many weeks the degenerative re-action.

**Faraadic electricity** is the “secondary, induced, or interrupted” current. It excites clonic contractions in the muscles, but there is no difference in the action of the poles as the current is constantly “making and breaking.” The usual method of using is to place the two poles, a few inches apart, over the nerves or muscles to be tested.

**Paralysis of the Cranial Nerves.**—The olfactory (1st) nerve. Paralysis of the first nerve results in a loss of the sense of smell (anosmia). It is sometimes unilateral, and generally unnoticed then by the patient. This condition is of frequent occurrence in cases of hemiplegia. When both sides are affected the loss of smell is complete. Perfumes or bad-smelling odours are the same to him; but *pungent* or *irritating* vapours stimulate the terminal filaments of the fifth nerve, and the impressions thus reach the brain, and often cause him to sneeze. The gustatory sense is also, at the same time, impaired. He can still distinguish bitter from sweet, sour and salt tastes, but his appreciation of delicate flavours is entirely lost. Blows upon the head, either injuring the olfactory bulbs, or the temporo-sphenoidal lobes—in which, according to Ferrier, are the centres of smell—are causes of *anosmia*. Closure of the posterior nares, or chronic catarrh affecting the Schneiderian membrane, are also causes of loss of smell, independent of lesions of the olfactory nerves.

Paralysis of the trifacial or fifth nerve, causes anæsthesia of

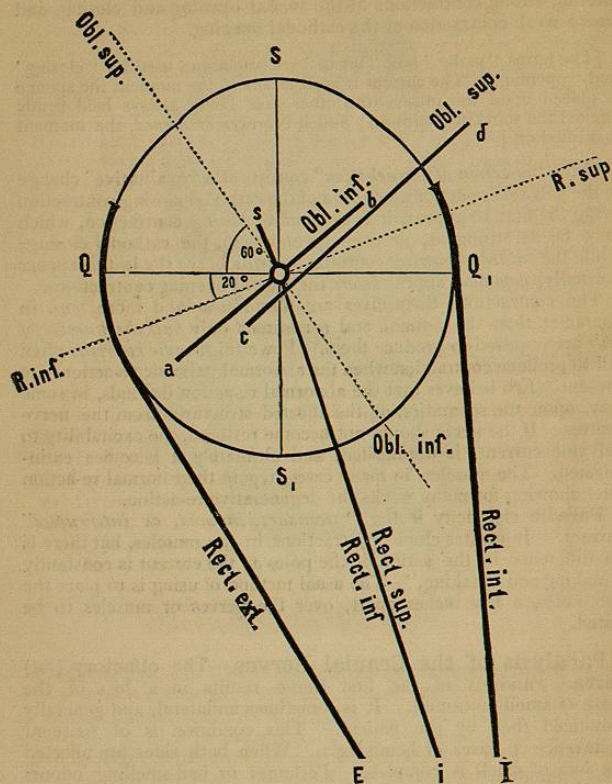


Fig. 31.—Scheme of the action of the Ocular Muscles— $S S_1$ , Visual axis;  $Q, Q_1$ , horizontal axis. 1. The rectus internus (I) and externus (E) rotate the eye almost exactly inwards and outwards. The plane of traction lies in the plane of the paper;  $Q, E$ , is the direction of the traction of the external rectus;  $Q_1, I$ , that of the internal. The axis of rotation is in the point of rotation,  $O$ , at right angles to the plane of the paper, so that it coincides with the vertical axis of the eyeball. 2. The axis of rotation of the R. superior and inferior (the dotted line, R. sup., R. inf.) lies in the horizontal line of separation of the eye, but it forms an angle of about  $20^\circ$  with the transverse axis  $Q, Q_1$ ; the direction of the traction for both muscles is, indicated by the line,  $si$ . By the action of these muscles, the cornea is turned upwards and slightly inwards, or downwards and slightly inwards. 3. The axis of rotation of both oblique muscles (the dotted lines, Obl. sup., and Obl. inf.) also lies in the horizontal plane of separation of the eye-

the face, and the nasal mucous membrane is insensible to pungent vapours—as ammonia and acetic acid; but the sense of smell is not impaired. The sense of taste in the anterior part of the tongue is lost, as in facial paralysis—the explanation being that the chorda tympani is the true gustatory nerve, and it is closely connected with both the fifth and the seventh nerves. Subjective sensations (numbness and

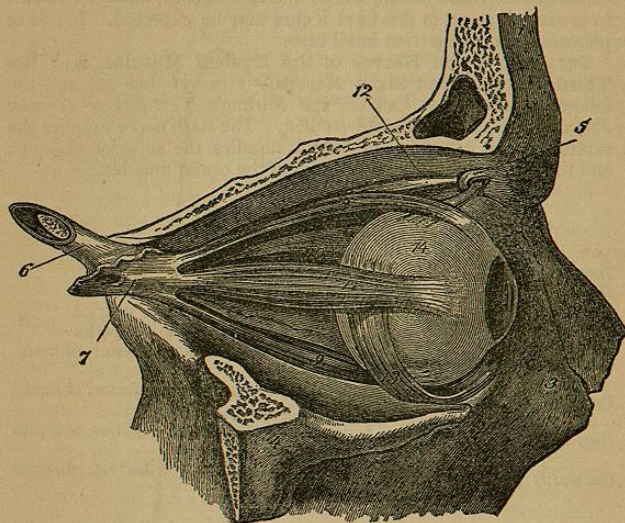


Fig. 32.—Lateral view of the muscles of the eyeball.—5, Trochlea or pulley of the superior oblique muscle, 12; 6, optic nerve; 8, superior, 9, inferior, and 22, external rectus; 13, inferior oblique. (Landois and Stirling's *Physiology*

tingling, &c.) are complained of, and a characteristic sensation is the feeling only half of a cup or glass when it is put to the lips. It appears to him to be broken. The conjunctiva is insensible. Mastication is imperfect. In some cases the gums become spongy and bleed, the circulation is interfered with and the affected cheek becomes livid, and often ulceration and sloughing of the cornea follow. These changes are due to the trophic centres being cut off,

ball, and it forms an angle of  $60^\circ$  with the transverse axis. The direction of the traction of the inferior oblique gives the line  $ab$ ; that of the superior, the line  $cd$ . The action of these muscles, therefore, is in the one case to rotate the cornea outwards and upwards, and in the other outwards and downwards. These actions, of course, only obtain when the eyes are in the primary position—in every other position the axis of rotation of each muscle changes. (Landois and Stirling's *Human Physiology*, 4th Ed., p. 965.)

it being now established that the fibres concerned in these changes lie in the inner portion of the trunk of the fifth nerve.

The causes of paralysis of the fifth nerve are generally serious organic affections—as caries and necrosis of the bones; syphilitic disease; cancerous or sarcomatous growths; aneurism of the internal carotid; and morbid changes within the Gasserian ganglion. Prolonged exposure of one side of the face to cold, has been known to produce it; and in this form a cure may be expected. Iodide of potassium should be tried in all cases.

Paralysis of the Nerves of the Eyeball Muscles, *viz.*: the Third, Fourth, and Sixth Nerves.—Figs. 31 and 32, and the following table from Landois and Stirling's *Text-book of Human Physiology* should be carefully studied. The sixth nerve supplies the external rectus muscle; the fourth supplies the superior oblique; and the third supplies the remainder of the ocular muscles.

#### OPHTHALMOPLEGIAS.

TABLE OF THE ACTION OF THE OCULAR MUSCLES.

Inwards,	. . . . .	Rectus internus.
Outwards,	. . . . .	Rectus externus.
Upwards,	. . . . .	Rectus superior, obliquus inferior.
Downwards,	. . . . .	Rectus inferior, obliquus superior.
Inwards and upwards,	. . . . .	Rectus internus, rectus superior, obliquus inferior.
Inwards and downwards,	. . . . .	Rectus internus, rectus inferior, obliquus superior.
Outwards and upwards,	. . . . .	Rectus externus, rectus superior, obliquus inferior.
Outwards and downwards,	. . . . .	Rectus externus, rectus inferior, obliquus superior.

The nerves to the eyeball muscles cannot be studied separately, because the two eyes being moved simultaneously, different nerves are concerned in the production of a given movement; *e.g.*, the *external rectus*, supplied by the sixth, moves with the *internal rectus* of the other eye, and this muscle is supplied by the third nerve; also, paralysis of a muscle of one eyeball, besides producing its own symptoms, gives rise to a *deviation* from the normal movement in the opposite eyeball.

For convenience in description the *left* eye will be supposed, all through, to be the one affected, with the understanding that the *converse* is true when the paralysis affects the right one.

In paralysis of the external rectus, supplied by the sixth nerve, the eye cannot be moved *outwards* when its fellow moves *inwards*. If the patient be directed to look at an object which is gradually moved, the further the object is carried to the left the greater is the difference between the direction of the two eyes. This squint is called the *primary deviation*, and it is very obvious when the paralysis is complete. When the paralysis is only partial, the affected eye may only lag behind its fellow, and there is no squint; but by covering the

sound (right) eye, and then removing the cover after he has fixed the object with the affected eye, the patient finds that he has not his right eye fixed upon the object, and so he is observed to move it quickly to the left.

When the paralysis is more or less complete, force is used to bring the eye round, but in doing so, some part of the force is thrown upon the associated muscle of the other eye—*viz.*: the *internal rectus*, and hence the sound (right) eye is pulled too far to the left. This movement of the sound eye is called the *secondary deviation*; and it is important in slight cases of paralysis, because it serves to distinguish a paralysis of the external rectus from an ordinary strabismus produced by a contraction or shortening of one, or both, of the internal recti. A lesion of the sixth *nucleus* produces paralysis of the conjugate deviation of both eyes—the eyes cannot be moved past the middle line towards the side of the lesion.

Diplopia, or double vision, is commonly present in paralysis of the external rectus, but only when the eyes are moved to the paralysed side; and the *false image* (*i.e.* the image seen by the affected eye, and which may be distinguished by the patient covering that eye with a piece of violet-coloured glass) is *outside* the true image, and the two images get wider apart as the object is moved further to the paralysed (left) side. This latter symptom distinguishes paralysis from “concomitant strabismus.” Each eye should be tested separately, as sometimes double images are formed upon the retina of a single eye. It should be noted also that a *sudden* development of diplopia does not exclude the possibility of it being the result of concomitant strabismus; as should there have existed a tendency previously, and the eyes (or general health) should have become weakened, fatigued, or impaired, this symptom may occur abruptly. In paralysis of the external rectus there is not always double vision, and sometimes the image is only “blurred.” Giddiness, and sometimes staggering, arises, when the patient uses the affected eye *alone*; and as the object appears to him to be farther out than it really is, if asked to strike at it, his hand goes to the outside (erroneous projection).

In paralysis of the (left) superior oblique, supplied by the fourth nerve—the inferior rectus having now (in part) lost its antagonistic muscle—the patient squints to the right and slightly upwards, when he attempts to look at his feet. The *secondary deviation* of the sound eye will be downwards and to the left. The double images are seen when the patient looks downwards, and the *false image* lies *below* and to the left of the true one, and it appears *tilted*. It also appears to be on a plane nearer to the eye than the true image. To prevent a sensation of giddiness the patient often carries his head down and to the right.

In paralysis of the third nerve, the four remaining muscles of the eyeball are affected, when the whole nerve is involved. As, however, a branch only may be paralysed, the muscles must first be considered separately:—

The (left) *internal rectus*; the *primary deviation* occurs when the