When this is present we obtain no muscular twitching with the faradic current and none over the nerve with the galvanic; but *with the galvanic over the muscle a slow, worm-like contraction* occurs, and the response to the positive pole is as good as to the negative, or better, whereas normally there is far better response to the negative. This is the *complete* reaction of degeneration; in *partial* reactions of degeneration all the normal reactions may be present, but diminished in intensity.

Reaction of degeneration occurs in all diseases affecting the anterior motor horns of the cord or their prolongations downward in the peripheral nerves; for example, in anterior poliomyelitis, progressive muscular atrophy, transverse or pressure myelitis, and all severe forms of peripheral neuritis. In brain lesions this reaction rarely occurs.

In *prognosis* a reaction of degeneration persisting after six to twelve weeks is unfavorable for recovery of the use of the muscles in which it occurs. If reaction of degeneration is absent or partial from the start, prognosis is for relatively speedy recovery, weeks rather than months.

V. Speech and Handwriting.

Aphasia, the loss of the power to speak or understand speech, despite normal hearing and muscular powers, occurs in lesions affecting the third left frontal and first left temporal convolutions of the brain.[1]

The lesions producing aphasia may be permanent anatomical changes following hemorrhage or tumor, or they may be transitory, as in uræmia and migraine.

The power to write or read letters is lost (*agraphia*) when the angular and supramarginal convolutions are destroyed.

Degeneration of the handwriting, as compared with the standard of former years, is often a helpful bit of evidence in the diagnosis of *dementia paralytica*, but may occur temporarily in various fatigue states.

[1] In some left-handed persons the centres are on the right side of the brain.

VI. Trophic or Vasomotor Disorders.

Trophic lesions of the joints, muscles (atrophy), skin, and nails have already been exemplified (pages 497 and 52). They blend with and are by some explained as the results of vascular changes (*vasomotor*). *Herpes labialis* ("cold sore") and *herpes zoster* ("shingles") certainly seem to give every evidence of being due to nerve nutritive disorders and not to vascular changes. The *acute bedsores* which form in myelitis, the "*angioneurotic*" *local swellings* which appear here and there in certain persons, and the local syncope or asphyxia which sometimes lead to Raynaud's form of gangrene, seem to need both nerve and vessel changes to explain them.

In brain lesions these trophic and vasomotor changes are much rarer than in disease of the cord and peripheral nerves.

VII. The Examination of Psychic Functions.

The diagnosis of the mental factors of disease forms an important part of the study not only of neurology, but of all diseases wherever situated; but as it cannot be called physical diagnosis, it falls outside the scope of this book, except in so far as loss of consciousness, *coma*, may be considered under this heading.

Coma.

The causes of coma are identical with the causes of convulsions. Every disease which causes the one may cause the other; hence all that is here said on the diagnosis of coma applies equally well to the diagnosis of convulsions. Either or both may result from:

1. Apoplexy (including cerebral hemorrhage, embolism, and thrombosis).
2. Uræmia and hepatic toxæmia.
3. Diabetes.
4. Cerebral concussion (stun).
5. Cerebral compression.

6. Syncope (fainting).
7. Opium.
8. Alcohol.
9. Hysteria.
10. Epilepsy.
11. Gas poisoning.
12. Sunstroke.

Apoplexy is the probable diagnosis when an elderly person who has shown no previous signs of ill-health becomes suddenly and deeply comatose within a few seconds or minutes. If hemiplegia is present (with or without aphasia) and if we can exclude the other causes above mentioned, the probability of apoplexy is increased. To determine hemiplegia in a comatose patient, try the following tests:

(*a*) Lift the arm and then the leg, first on one side and then on the other, and let go. The supported member falls more limply on the paralyzed side.

(*b*) Pinch or prick the limbs alternately. The sound limb may be moved, while the other remains motionless. Pressure over the supraorbital notch may bring out a similar difference in the response of the two sides.

(*c*) Try the knee-jerks. On the paralyzed side the jerk may be increased.

(*d*) Try Babinski's reaction. It may be present on the paralyzed side.

Uræmia.—The diagnosis between apoplexy and uræmia is sometimes impossible, since uræmia may produce hemiplegia and the urine in the two conditions (as obtained by catheter) may be identical. Usually, however, the uræmic patient has previously shown obvious signs of nephritis—œdema, headache and vomiting, long-standing oliguria, or polyuria with albuminuria. Convulsions more often precede or follow the coma of uræmia than that of apoplexy. Retinal hemorrhages or albuminuric retinitis, if recognized by ophthalmoscopic examination, point strongly to uræmia.

The hepatic toxæmia in which many cases of cirrhosis die is distinguishable from uræmia only if the previous history of the case is known to us and the signs of liver disease (ascites, jaundice, caput Medusæ) are evident.

Diabetic coma is usually recognized with ease, because the evidences of advancing diabetes lead gradually up to it. The emaciation of the patient, the sweetish odor of the breath, the presence of sugar, and especially the evidences of acetone and diacetic acid in the catheter-urine, are the essential factors in diagnosis. Dyspnœa ("air hunger") precedes the coma in about one-third of the cases.

Concussion (or stun) after a blow usually clears up in a few minutes and so presents no difficulty in diagnosis. If the coma lasts on for hours or days (as it sometimes does) the suspicion arises that we are dealing with

Compression. For this the evidences are: Focal symptoms, convulsions, slowing of the pulse, and signs of depressed fracture. To determine the latter fact may be impossible without trephining, since the inner table of the skull may be broken, while the outer is intact. The focal signs to be looked for are paralyses (ocular or peripheral).

Syncope (or fainting) is usually over in a few minutes and so betrays its nature, but it must not be forgotten that a slight convulsion may occur just as the patient comes out of coma. No suspicions of epilepsy need be aroused thereby, but if there have previously been signs of hysteria we may be in doubt whether the fainting fit is not of hysterical origin. The history of the case, the circumstances at the onset of the attack, and the presence or absence of hysterical behavior during it usually guide us aright.

Opium poisoning produces a coma from which the patient can usually be more or less aroused. Contracted pupils and slow respiration are the most characteristic signs. A laudanum bottle or a subcutaneous syringe found near the patient often assist the diagnosis.

Alcoholic coma is rarely complete. The patient can be aroused. The circumstances under which he is found, the odor of alcohol on the breath, the absence of paralysis, fever, small pupils, or urinary abnormalities are the main supports in diagnosis. There is no char-

acteristic pulse and the pupils show no constant changes, though in many cases they are dilated.

Hysterical coma usually occurs in young women who have previously shown signs of hysteria. In falling they never hurt themselves. The eyelids are contracted, often tremulous, and when forcibly pulled open often expose eyeballs rolled up so that the whites alone are seen. The hands are apt to make grasping motions, and there are irregular, semipurposive movements of various parts of the body. A startling word may arouse the patient, but anæsthesia to pain (over one-half or all the body) is often complete.

Postepileptic coma is usually recognized with ease, because of the convulsions which precede it and which are usually known to have occurred at intervals before. The scars of previous falls may be found on the head.

Gas poisoning rarely presents any diagnostic difficulties, because the circumstances under which the patient is found make clear the cause of his condition. An odor of gas may hang about his breath for some hours.

Sunstroke is recognized by the state of the weather and the presence of a very high temperature (106°, 110°, 115° F., or even more). There is no other characteristic sign.

APPENDICES.

APPENDIX A.

DISEASES OF THE MEDIASTINUM

I. Mediastinal Tumors.

New growths of the mediastinal glands[1] usually manifest their presence by the following symptoms and signs:

(1) Cachexia and substernal pain.

(2) Evidence of pressure against:—

- (*a*) The *gullet*.
- (*b*) The *windpipe* or primary *bronchi*.
- (*c*) The large *venous trunks*.
- (*d*) *Nerves* which pass through the mediastinum.
- (*e*) The subclavian arteries.
- (*f*) The heart.
- (*g*) The ribs, clavicle, or sternum.

(3) Secondary deposits in the cervical or axillary glands.

(*a*) By pressure on the gullet swallowing may be rendered difficult or impossible (dysphagia).

(*b*) By pressure on the windpipe may be produced displacement of the latter to one side, or fixation so that it cannot be moved in any direction. The larynx may be drawn down into a noticeably low position, and the laryngoscope may demonstrate that the tracheal wall is bulged inward by the pressure of the new growth upon it.

Dyspnœa, either inspiratory or expiratory, or both, and often

[1] Tuberculous glands not being here included.