Diseases of the nervous system resulting from accident and injury.

Presented by Dr. Macdonald Critchley.
May, 1955.

A. C. Page

DISEASES OF THE NERVOUS SYSTEM

RESULTING FROM

ACCIDENT AND INJURY

BY

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ACCIDENT AND INJURY

THEIR RELATIONS TO DISEASES OF THE NERVOUS SYSTEM

TO

PROFESSOR STARR

IN GRATEFUL RECOLLECTION OF

MANY KINDNESSES

PREFACE

In "Accident and Injury in their Relations to the Nervous System," published several years ago, an attempt was made to furnish a systematic description of the nervous affections which result from injury and fright, generally known as the traumatic neuroses. These disorders exist independently of any demonstrated lesions in the nervous system, yet some of them bear close clinical resemblance to organic injuries. So close, indeed, may this resemblance be, that it was deemed advisable to embody in the book some statements relative to organic injuries and the means of differentiating them from their functional familiars. When the time came for a new edition of "Accident and Injury" it seemed to the author that, in addition to the incorporation of much new matter concerning the traumatic neuroses, the scope of the book should be extended, in the hope that it might win a place for itself as a treatise on all traumatic affections of the nervous system. To unite the mass of new matter made necessary by this change, with the old book, was found to be impossible. A complete recasting was necessary; and, in view of its enlarged purpose, the title was made more comprehensive.

Since injuries to the nervous system belong to both surgery and neurology, much pruning is necessary to keep the book which describes them in reasonable compass.

The present volume is written from the neurologist's standpoint. Those subjects most fully described in text-books

on surgery are dismissed with briefest mention. The late effects of brain injuries, for example, receive more notice than the acute symptoms, and the principles of treatment of purely surgical affections, while indicated, are not construed as to operative details.

The writer desires to express his obligations to Dr. Smith Ely Jelliffe for revision of the manuscript.

PEARCE BAILEY.

52 WEST FIFTY-THIRD STREET.

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INTRODUCTION

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No sharp line of demarcation can be drawn between the disorders of the nervous system which develop as a result of acutely occurring injuries and those which owe their existence to causes operating more slowly, or whose clinical manifestations, although they may appear suddenly, are the effects of subtle conditions that have been for a long time active. Yet, as every causal factor adds its special mark to the conditions it induces, so injury and shock put their stamps upon the nervous affections which are their results. It is the effects of such traumata on the nervous system which will be considered in the following pages. The word *traumatic* is permitted so many meanings that its limitations must be clearly understood. In its most restricted sense it applies only to such injuries as are the results of acutely acting physical violence, while in its widest meaning it includes any detriment to health which originates outside of the body. By the first meaning of the term, psychic influences are disregarded; by the latter interpretation, its scope would be practically unlimited, as it would include all morbid conditions which owe their existence to toxic or mechanical causes, such as lead-poisoning, alcoholism, or writer's cramp, as well as the crushing of nervous tissue from the

pressure of broken-down protecting structure, or the disturbance of equilibrium in cerebral processes brought about by mental shock or fright. For clinical purposes neither of these interpretations of the term is satisfactory. The first disregards the very important influence of nervous shock and fright as disease-inducing agents, and the second renders the word traumatic much more comprehensive than is desirable, or than is generally understood. In the following pages *traumatic* will be used as indicating *quickly acting physical violence or psychic shock which arises outside the body.*

The effects of trauma on the nervous system are manifold, and in many cases baffle satisfactory analysis. The immediate results of physical injuries can usually be interpreted at their true value. But the ultimate effects, as well as those of psychic traumata generally, which act through the emotions, are much more difficult of evaluation. The psychoses, certain neuroses, and hysteria, when they arise from fright and similar causes, open endless questions as to predisposition, to previous attacks, and to auxiliary causes. Physical and psychic injuries, acting either alone or singly, exert potent influences on the organism generally, notably on the circulation and on nutrition. Causes which disturb metabolism, and which bring about defective or toxic cellular action may some day explain the traumatic genesis of such elective diseases as paralysis agitans and general paresis. Again, injuries may so far change the mode of life of the injured person that he becomes a prey to alcoholism, to nicotinism, to morphinism, to introspection, and other disease-inducing influences which are the handmaids of idleness.

Traumata, especially in the aged, make preëxisting disease worse. What can be lightly thrown off by the young is a burden to the aged. Certain diseases, such as nephritis, which are the ordinary results of advancing years, occasionally

seem to appear first in sequence to injuries, though there is little reason to doubt that they preëxisted, though latent.

The researches of Ehrnzooth have demonstrated that local injuries to the nervous system create a *locus minoris resistentiæ* which facilitates the action of toxic principles. In his experiments two series of animals were injected with cultures of various pathogenic bacteria. The animals of one series had previously been subjected to slight injuries to the head. The injured animals showed a much lessened degree of resistance, both local and general, to the bacteria, than the uninjured ones.

It is possible that certain infectious diseases, such as tuberculosis and cerebro-spinal meningitis, which sometimes appear to be connected with a preceding trauma, without external wound, are explainable on such an hypothesis; it is possible, also, that new growths, appearing after injuries, such as tumors of the nervous system, or multiple sclerosis, may find some such ætiological explanation.

With causes so obscure and so manifold, and with so complex a structure as the nervous system acted upon, it is not surprising that wide differences of opinion are met with in individual cases.

In many cases of traumatic nervous disease the clinical picture is so plain that a cursory examination suffices for an immediate and correct appreciation of the cause, the nature, and the probable outcome of the injury. The ambulance surgeon may instantly recognize a fracture of the spine, and fractures of the skull are of so common occurrence in emergency hospitals that experienced surgeons recognize the condition by the general appearance of the patient alone. Yet from the organic lesions of the nervous system whose nature is readily apparent, to those which require the closest study for their diagnosis, is not so very long a step. In presumable

brain injuries, when the patient is comatose or delirious, examination for localizing signs is often unsatisfactory, and the history of the accident may be unobtainable, or if told by witnesses unreliable, so that the diagnosis of concussion or laceration of the brain, as opposed to some of the idiopathic forms of coma, is very difficult or at times even impossible.

In the diagnosis of the puzzling forms of contusions of the back—as a result of which, without evidences of lesion to the vertebræ, there are selective symptoms of injury to the spinal cord—considerable knowledge of the anatomy and pathology of the nervous system is often necessary. This fact becomes evident upon the perusal of the works of Erichsen, the pioneer in traumatic neurology, who failed to recognize the true condition in many cases of back injury. Affections so common as traumatic peripheral nerve palsies, especially those which affect the nerves around the shoulder joint, can in certain cases be recognized only by careful electrical examination. The diagnosis of the disorders which must still be known as functional are diagnoses by exclusion, to make which presupposes a considerable knowledge of the symptoms of organic nervous injuries.

At the outset, the student should have clearly in mind the meaning and the limitations of the terms *organic* and *functional* as applied to nervous diseases. An organic disease is one in which there is visible destruction or alteration in the nervous system. Fibers and cells are lacerated, or degenerated, hemorrhages take place, new tissue grows; and all this in a way to be recognizable by the naked eye or by means of the microscope. The symptoms from such lesions are usually pronounced and often focal. In functional disease no visible changes occur which can be recognized as characteristic by any means at our disposal. Even under the microscope the nervous system appears normal, and the symptoms, which are wide-

spread and changeable, do not point to any actual destruction of nervous tissue. We can but believe that with increasing delicacy in the methods of investigation some diseases now classed as functional will be shown to be organic; and already there is a difference of opinion among authors as to the functional or organic character of some of them. As a general rule, the gravity, both of immediate symptoms and of prognosis for ultimate recovery, is much greater in the organic than in the functional disorders. At the same time, organic lesions, if small and in unimportant regions, permit practically complete restoration to health, while some functional affections are permanent.

Although the inherent difficulties of diagnosis due to the intricacies of the anatomy and physiology of the nervous system were already great enough, the commercial spirit of the times has added others. The bringing of claims for damages for personal injuries has reached so exaggerated a degree that it is no rare occurrence for an injured person to see an attorney before he sees a doctor; and the story which this latter hears often speaks more eloquently for the medical knowledge of certain members of the legal profession than for the veracity of the patient.

When the injuries become the basis for a claim for damages, there are many complicating factors. Under these circumstances, while the account of the resulting symptoms is to be listened to with attention and respect, it should be accepted with reserve, and only in so far as it may seem to be justified by the attendant circumstances. Such conservatism cannot be construed as a reflection upon the honesty of the patient or of his lawyer. As a rule of clinical medicine it is universally observed, and is necessary for the avoidance of the deception caused by the misstatements of ignorance as well as those of intention. The question of liability is one with which the

physician has little to do. It concerns him only in so far as he may be able to show that the physical or mental condition of the claimant at the time of the accident was of a character to render him unable to take proper precautions against danger.

In the study of traumatic nervous disease so many factors enter that it has seemed more advisable to entitle this introductory chapter—which essays to outline some of the simpler methods of investigation—a consideration of the case rather than an examination of the patient. As the most successful physicians are those who treat patients rather than diseases, so he can give the most valuable opinion in regard to an injury of the nervous system who has considered it from every point of view.

The necessity for a painstaking examination of cases which are in themselves obscure, or of those in which for forensic reasons accuracy of diagnosis is especially essential, needs no emphasis. It is always advisable to put the results of examination immediately in writing, and in medico-legal cases it is well to have the assistance of a stenographer, in order that the procedure may not be unnecessarily delayed and that as full notes as possible may be taken.

The examination may best be divided into four stages, viz.:

I. Previous history of the patient (ancestral; personal).

II. History of the accident.

III. Physical evidences of predisposition to nervous disease.

IV. Examination for the actual injury.

I. Previous History of the Patient

In nervous disease originating as the result of injury the influence of hereditary predisposition is usually not very evi-

dent, and is always difficult to prove. Few nervous diseases are directly transmitted from parent to child; heredity seems rather to act by giving to the offspring a nervous system whose powers of resistance are diminished, but in which the development of disease depends upon the specific action of various exciting causes which vary with environment. Among the morbid conditions which render their victims particularly prone to transmit unstable nervous systems to descendants may be mentioned alcoholism, insanity, epilepsy, syphilis, hysteria, and tuberculosis. If any of them have been present in both parents, or have been generally disseminated throughout a family, the chances are all the greater that the offspring will be endowed with an imperfectly developed nervous system, or with one which will easily succumb to injurious influences.

The object sought for in traumatic cases in inquiring for a history of the patient's personal life is to establish whether he were or were not a healthy man before the accident. The patient is asked the diseases he has had, the amount of work he has been capable of, and the kind of life he has been able to lead. More valuable than his own replies as to his physical condition are facts concerning his surroundings and mode of life. Age, sex, nationality, and race are considerations of paramount importance, which are described in succeeding pages. Certain occupations, notably those which expose the workman to chronic poisonings, are important predisponents to nervous disease. On account of real or assumed ignorance, reliable information concerning the family or personal history is often difficult to obtain, and the physician will do well to avoid putting too much credence in statements which seem at variance with the actual condition of the patient.

II. History of the Accident

Definite and reliable information concerning the accident is often unobtainable. In general accidents, such as railway collisions, where a large number of persons are involved, there are usually no spectators. The circumstances which surround such catastrophes make each individual a participator rather than a witness; and the suddenness of the accident and the terror which it causes usually render the victim unable to give an intelligent account of the way his own injuries were received. Even if he were not rendered unconscious, he is rarely able to describe just what befell him. When, however, statements are obtainable from witnesses, such testimony is often more valuable than that of the victim himself. The general character of accidents determines, to a certain extent, the nature of the resulting injuries. In the consideration of individual cases it is, of course, impossible to rely too largely on generalizations. Although it may be known that certain causes are usually followed by similar results, a variation of conditions may bring about exceptions. In most cases, however, the character of the accident is an index of the severity and of the nature of the resulting injuries.

In serious general accidents the body may be subjected to every degree of violence, which may induce immediate death or cause mutilations of all degrees. The fate of passengers or employees in any single given collision or derailment cannot, of course, be foretold. On the other hand, it can be stated positively that the sudden stopping or starting of a vehicle, by which the passenger may be jarred but receives no external wound, rarely if ever exerts sufficient force to cause irreparable injury to the nervous system. In many other forms of mishap we are justified in inferring that the violence was inadequate to cause structural injury. Thus, a light pasteboard box falls a

few feet and strikes a young girl on the head without inflicting any serious wound. She immediately goes into convulsions, and, on regaining consciousness, it is found that she is insensible on the left side of the body. In such a case it is just to infer at once that the injury could not have caused any lesion of the brain which might explain the symptoms, but rather that they were due to the disordered mental state of the patient, and were the immediate results of fright.

In addition to the facts concerning the accident itself, the condition of the injured person immediately afterwards should be ascertained. Was shock or fright prominent? Were there external wounds? Was the patient conscious? Could he walk? Did he vomit? Did he complain of pain, or dizziness, or double vision? Was he catheterized? If paralyzed, how soon did the paralysis ensue? What method of treatment was employed? In short, varying with the region of the nervous system supposed to be injured, all possible information should be obtained regarding the mode of development of the symptoms.

III. Physical Evidences of Predisposition to Nervous Disease

In severe injuries, such as fractures of the skull or of the vertebræ, the previous condition of the patient is of secondary importance. A strong man may, of course, survive a shock that might prove fatal to the patient whose recuperative powers had been enfeebled through previous bad health or excess. Yet such a difference can rarely be positively proved. The chief forensic importance of previous condition attaches to those disorders of which the exciting cause has not been immediately dangerous to life, but has been the alleged starting point of diseases which are more or less serious though not directly fatal. The possibility of error in ascribing a traumatic origin

to a disease which antedated the injury is considered in Part III. Here it is necessary to mention only such conditions as furnish a favorable soil for the development of nervous affections.

Stigmata of Degeneration.—There are certain anatomical evidences which are regarded as indicative of a congenitally defective nervous system, which is consequently less tolerant of injurious influences. These are the stigmata of degeneration, and, although it would be beyond the scope of these pages to describe them in detail, their general character may be seen in the following classification as suggested by Peterson:

Stigmata of Degeneration.

- Cranial anomalies.
  - Asymmetry.
  - Deformities.
- Facial asymmetry.
  - Prognathism.
  - Retrognathism.
- Deformities of the palate.
- Dental anomalies.
- Anomalies of the tongue and lips.
  - Harelip and cleft palate not certainly stigmata.
- Anomalies of the nose.
- Anomalies of the eye.
  - Flecks on the iris, strabismus, chromatic asymmetry of the iris, narrow palpebral fissures.
  - Albinism.
  - Congenital cataracts.
  - Microphthalmos.
  - Pigmentary retinitis.
  - Muscular insufficiency.
- Anomalies of the ear.
- Anomalies of the limbs.
  - Polydactyly.
  - Syndactyly.
  - Ectrodactyly.
  - Symmelus.
  - Ectromelus.
  - Phocomelus.
  - Excessive length of the arms.

Stigmata of Degeneration—(Continued)

Anomalies of the body in general.	Hernias. Malformation of the breasts, thorax. Dwarfishness. Giantism. Infantilism. Feminism. Masculinism. Spina bifida.
Anomalies of the genital organs.	Cryptorchismus. Microrchidia. Spurious hermaphroditism. Hypospadia. Epispadia. Atresia.
Anomalies of the skin.	Polysarcia. Hypertrichosis. Absence of hair. Premature grayness.

The practical value of the stigmata of degeneration is impaired by the fact that they occur chiefly in idiots, lunatics, and epileptics, about whose degeneracy there could be no question. Also, almost any one of them may occur in persons who never present any other structural or functional anomalies.

More useful from a diagnostic point of view than these physical telltales of ancestral faults are such evidences of acquired predisposition as are left by certain diseases and by certain poisons. In a general way it may be said that any disease which impairs the general health makes the development of nervous disorders more probable. There are a few conditions, however, which stand so preëminently in a causal relation to neural diseases that it is very necessary to determine their existence before attributing too high a value to any exciting cause. Of these, the most important are alcoholism, syphilis, and arterio-sclerosis.

Alcoholism.—It is a matter of common remark that drunken persons who are in serious accidents often escape without any severe injury, or indeed any subsequent ill effects whatsoever. While it cannot be denied that the muscular relaxation and mental stupor of alcoholic intoxication not infrequently furnish a means of escape from physical injury and psychic shock, this is a very minor consideration when compared with the number of persons who are killed or injured in accidents which would not have occurred to an individual who was sober. Alcoholism stands in a very important causal relation to traumatic diseases of all kinds, but especially to traumatic diseases of the nervous system. It is frequently accountable for the accident and the injury, and always is prejudicial to recovery. After very slight blows on the head, or after trivial injuries to other parts of the body, alcoholic persons not uncommonly develop delirium tremens, pneumonia, or heart failure, or show in other ways that their recuperative powers have become impaired by alcohol; and in all serious injuries the prognosis as to life is greatly invalidated in alcoholic subjects.

The evils of the drinking habit are so widely disseminated in all classes of society that it is very essential at the beginning of an examination of any accident case to determine the presence or absence of chronic alcoholic poisoning as a complicating factor. Old and steady drinkers present at all times such unmistakable physical stigmata of their vice that there is no difficulty in doing this. But many persons who have regarded themselves as moderate drinkers only, and who present none of the more evident signs of chronic alcoholic poisoning, after slight injuries exhibit symptoms which show that their use of alcohol has been too free. In such cases the history of excessive drinking is not necessary for diagnosis, because there is so great a variation in the individual tolerance to the poison

that a daily quantity which in one person could be taken without evil effects of any kind might be sufficient in some one else to cause acute symptoms, or to render the organism incapable of resisting what a healthy man could easily resist. Accordingly, more reliance is to be placed upon the evidences of alcoholism which the physician may discover himself, than upon the patient's history. They are usually abundantly and characteristically present, and are both physical and mental.

The physical symptoms consist of tremor, generalized or most prominent in the face, lips, and tongue, and in the fingers; the skin may be normal, or congested, or flabby and pale, with a peculiar appearance of œdema; gastric irritability is common; the heart is usually rapid, and there may be an impairment of the purity of the first sound at the apex; the functions of the special senses are often vitiated, and disturbances of general cutaneous sensation, both objective and subjective, are particularly frequent. The mental symptoms consist of coma, or stupor, or delirium, and their variations. The delirium is usually characterized by hallucinations of sight, less frequently of the other senses, which almost always have reference to living objects, such as animals, or crawling things, or strange persons. A common condition is one of forgetfulness and confusion; the memory may be lost for considerable periods of time, or the patient may be unable to collect himself sufficiently to tell where he is or how he came there. Two patients whom I recently saw at the Manhattan Hospital thought they went out every night, when in reality they both had been in bed for several weeks. Systematized delusions are not common, although they sometimes occur in the chronic form of poisoning, and consist, with particular frequency, of false beliefs in regard to marital infidelity.

The fact that the symptoms of chronic alcoholic poisoning resemble in many respects those of Oppenheim's "trau-

matic neurosis " has led Saenger to suggest the possibility that many of those cases are in reality cases of chronic alcoholism which have been made worse by injury. The tremor, amblyopia, impairment of cutaneous sensibility, and rapid pulse, which are prominent in the " traumatic neurosis," are all found in ethylism. Whether some of these cases are in reality nothing but " traumatic alcoholism," or whether the chronic toxæmia has supplied the foundation upon which has developed a neurosis or psychosis, we are at present unable to decide definitely. It is certain, however, that in many of the cases which will be described later alcoholism has played a very important rôle.

Syphilis.—The typical history of syphilis is obtainable in a certain number of the cases of nervous disease. In a large number there is the admission of a sore on the penis only, but of no subsequent syphilitic manifestations; in many instances the patients deny every symptom which might have been of specific character. In the absence of a syphilitic history, there are often discoverable certain physical defects which indicate that the syphilitic poison had at one time been active. Of these the most important are scars on the penis, scars on the legs, adhesions of the iris, and similar results of syphilitic inflammation. While these evidences are oftentimes characteristic, it is rarely possible to assert that they are the results of syphilis and could be the results of nothing else, although in the clinic they are accepted, usually correctly, as satisfactory evidences of preceding infection. But any one of them could owe their existence to other causes, and it is consequently almost impossible to be certain that syphilis has existed when all active syphilitic processes have subsided.

The part played by syphilis in the ætiology of nervous disease is of great importance. The venereal disease has an unquestioned and intimate connection with the causation of general paresis and of locomotor ataxia. Syphilis, like alco-

holism, is an active factor in the induction of premature senility. By attacking the blood vessels it causes serious disturbances of the circulation, which lead to degenerative changes in various organs, and which render the organism less resistant to deleterious influences.

It is also a significant factor in the genesis of such functional disorders as neurasthenia, hysteria, neuralgia, and epilepsy.

Very rarely after acute injuries latent syphilitic processes become active and give the symptoms of focal lesions.

Arterio-sclerosis.—The importance of the relationship between disease of the arteries and certain symptoms of disturbances of nervous function can hardly be overestimated. By attacking the coronary arteries, arterio-sclerosis causes a degeneration in the muscular mechanism of the heart with a consequent deficiency of propulsive power in that organ; by diminishing the caliber of terminal arteries, it also offers serious impediments to circulation. The process, in whichever way it acts, is a direct obstacle to blood supply and a menace to nutrition. Its effects are widespread, but particularly disastrous to the nervous system. It is a well-known fact that arterial degeneration is a common accompaniment of the retrogressive periods of life, but it is too little recognized that it is not rare in youth and middle age. In young subjects, especially, the effects of alcohol and syphilis are prominent in its causation. In the autopsies on the bodies of prisoners who die in the workhouse on Blackwells Island, arterio-sclerosis, either generalized or chiefly restricted to the cerebral arteries, is frequently encountered in subjects who are under forty years of age, but who have led a life of exposure and excess.

Arterial degeneration, either disseminated or limited to the cerebral arteries, may exist for a long time without causing any subjective symptoms, or it may be the direct cause of dizziness,

failure of mental power, headache, epileptiform attacks, etc. When existent it is directly unfavorable to recovery from acute injuries, both by reason of its causing a diminution in recuperative power and because the patients are particularly liable to such complications as pneumonia and heart weakness. On both clinical and pathological grounds it is not to be doubted that the disturbances of cerebral circulation which it causes are often responsible for many nervous symptoms which are classed as functional.

In the examination of an accident case, accordingly, it is very essential to determine whether the injury has acted upon a perfectly healthy person or upon one whose arteries were diseased. This is not always easy to do. In advanced stages when there are objective signs, such as thickened peripheral arteries, a hypertrophied and overacting heart whose second aortic sound is intensified, urine with low specific gravity and which is, perhaps, slightly albuminous, the diagnosis is plain. But in the earlier stages one or all of these signs may be absent, and, although the previous life of the patient may have been such as to have caused degenerative vascular changes, there are no objective evidences that such conditions exist.

The sphygmomanometer (q. v.) may be useful in this connection. If repeated examinations show a persistent increase of arterial tension it is probable that arterio-sclerosis exists. Arterio-sclerosis may exist, however, without an increase in blood pressure.

Even in the absence of objective signs the physician should hesitate in attributing a purely functional character to such symptoms as dizziness or cardiac irregularity in persons whose previous life has been of a character to bring about degeneration in the circulatory organs.

Age.—Age is important, both in causation and prognosis. After fifty years there is less effort at repair, and injuries may

bring to light diseases hitherto dormant. Age is determined not only by years, but by the general condition of the patient. "A man is as old as his arteries." In the aged, the so-called "functional" symptoms are of more serious significance than in the young.

IV. The Examination for the Actual Injury

In addition to the search for nervous symptoms, it would seem superfluous to emphasize the necessity for a thorough general physical examination of the patient were it not for the fact that it becomes so frequently apparent, in both clinical and medico-legal cases, that the examination has been entirely too superficial to furnish sufficient grounds for a definite opinion. In cases of organic injury, although the nature of the case may be plain at the first glance, the more searchingly the physician pushes his inquiries the more capable will he be of giving an opinion of value. In functional nervous disease the diagnosis can only be made where it can be proved that organic disease is in all probability absent. How serious may be the errors incurred through superficiality of examination was illustrated by a case to which my attention has recently been called:

A woman sought advice for a peculiar nervous condition which ensued immediately after a blow on the head from a heavy bale of carpets. She asserted that previously to this she had been healthy in every respect, but that since the accident she had been nervous, emotional, and unable to concentrate her mind on her work. Immediately after the accident she developed huskiness of speech and an annoying tracheal cough, very similar to hysterical cough, without any expectoration. The patient was extremely suggestible, and an attempt at hypnotizing her was successful in inducing the first degree of hypnosis. The condition was diagnosticated by the physician as one of "traumatic neurosis." A subsequent examination of the chest and larynx, when the patient was admitted to the hospital, revealed

unequivocal evidences of an aneurism of the aorta. Thus, although the nervous symptoms were undoubtedly functional, the cough and laryngeal symptoms were the expressions of a fatal disease whose existence had been entirely overlooked.

The intimate structural and functional relationship of the nervous system to every organ in the body, and the frequent association of nervous disease with diseases of other organs or systems, render any examination of the nervous system incomplete which has not been preceded by an investigation of general somatic conditions. Such a procedure is always essential for purposes of prognosis, and in many cases is the one means of avoiding diagnostic errors. To determine the presence of eruptions, or cicatrices, or other evidences of present or past lesions of the skin, examination of the whole cutaneous surface is often necessary; no examination is satisfactory which leaves undetermined the condition of the heart and lungs; the urine must be tested for the presence of albumen or sugar—in fact, most of the important means of diagnosis must be resorted to before the physician can hope to obtain a clear comprehension of the case. It may seem almost elementary to lay stress upon such self-evident facts, but it is certain that the errors which are occasionally laid to the neurologist's door most frequently arise from the lack of thoroughness in this respect.

SYMPTOMS

Symptoms are subjective and objective. Subjective, those which the patient himself feels, but which the physician cannot demonstrate. Objective symptoms are those which the physician can, by various methods of examination, find out for himself. Although the existence of subjective symptoms cannot be proved, experience helps one to judge, by the way the patient talks about them, or conducts himself while he alleges

he has them, whether they exist to the degree they are complained of, if at all. Some symptoms essentially subjective are related by the patient in so characteristic a way, or exert so characteristic an effect on his conduct or personality, that they are practically objective. To be accepted as existing in fact, subjective symptoms should harmonize with objective symptoms.

Every variation from the normal which the individual experiences may be regarded as a subjective symptom. But only a few of the more important ones require mention.

Subjective Symptoms.—PAIN.—Pain may be constant, or occur only in attacks, or only result from pressure or movement.

The reaction to pain varies greatly in different individuals. Some suffer in nutrition and are incapacitated by pain which, judged by ordinary standards, must be slight; while others endure what we know are the severest pains (e. g., trigeminal neuralgia) for years, without giving up their work or losing in weight or color. The endurance of pain, consequently, is a matter of personal reaction, if not of personal character. It is therefore difficult to estimate the severity of pain.

The physician is never in a position to absolutely deny that pain exists—he may, however, believe it to be slight or absent, if the patient does not describe it in a characteristic way, or if it fails to agree with the other symptoms complained of. The value of the Mannkopff sign for true pain (q. v.) is questionable. Inquiries regarding pain should be made as to its character, distribution, time of appearance, duration, and effects on the general organism. Pain can cause the heart to beat faster or slower, can cause redness of the affected parts, or twitchings in the neighboring muscles.

PARÆSTHESIA.—Sensations of pins and needles, of tickling, of burning, of feeling as though a part were asleep, are

symptoms of organic, and also of functional, diseases. They may cause much discomfort. They are never objective. The same line of inquiry as for pain is to be followed in their investigation.

VERTIGO.—As currently used, the term vertigo applies to two symptoms which, although closely allied and interdependent, are distinct. In one sense vertigo is the sensation of dizziness. In the other it is the disturbance in equilibrium which may or may not accompany the subjective disturbance. As a symptom, therefore, it is both subjective and objective. The mechanism, upon the disturbances of which it depends, is made up of the semicircular canals, the vestibular nerve, and the connections of the vestibular nerve in the pons, medulla, cerebellum, and cerebrum. These disturbances may be due to structural damage to these parts, or to temporary alterations in their blood supply or pressure. Vertigo, therefore, while it may be present in some degree in all physical disturbances, is especially important in diseases of the ear and of the nervous system.

Most diseases of the middle ear are accompanied with more or less giddiness, but the more severe forms are due to affections of the labyrinth. These may be due to disease, or to injuries of that region. The vertigo is usually intense, is both subjective and objective, and is associated with nerve deafness.

Vertigo is a sensation from which every one suffers, at some time or another, as the result of sudden changes in position, or of slight errors in diet, or of other trivial causes. Also, no condition is more easily magnified, intensified, and prolonged by the subject focusing his attention upon it. Since in neurasthenia and hysteria the main mental characteristic is introspection and self-analysis, it is not surprising that in these disorders we find vertigo a prominent symptom without traces

of permanent or adequate underlying physical cause. In these diseases vertigo is chiefly subjective.

In sharp contrast to the vertigo which occurs in them is that which results from organic disease of the brain. In brain injuries, vertigo results from interferences with the nervous connections of the semicircular canals. It may be due to direct injury to these parts or to changes in blood pressure or in general brain pressure. It has great variation in intensity and character. If the sensation is referred to one side, if there is a tendency to fall to one side, and if the other symptoms are in agreement, the diagnosis of organic injury is sometimes possible from the vertigo alone. But only rarely. For most patients after trauma capitis complain of dizziness and the establishment of its origin must usually be made by the associated symptoms.

Objective Symptoms.—It is impossible to devise any scheme of examination which is in every way satisfactory, but I have found the following, slightly modified from Strümpell's, the most useful:

General appearance (facial expression, manner, etc.).

Mental state.

Cranial nerves.

- Olfactory nerve.
- Eyes—2d, 3d, 4th, and 6th nerves.
- 5th nerve.
- 7th nerve.
- 8th nerve.
- 9th, 10th, 11th, 12th — nerves which include speech, deglutition, secretion of saliva, and taste.

Paralysis, rigidity, tremor, morbid movements, gait.

Sensation (pain, touch, temperature, muscular sense).

Reflex action (superficial and tendon reflexes).

Atrophy of muscles.

Trophic disturbances.

Electrical examination.

General Appearance.—In many cases the general manner, the facial expression, the speech, the attitudes, the gait, point at once to the condition from which the patient is suffering, and indicate the special functions which should receive the most careful investigation. Many nervous diseases have characteristic physiognomies, and cause pathognomonic movements. From the manner and facial expression alone it is often possible to decide as to the existence of real or supposed pain, of insomnia, or, indeed, if the patient is seriously ill. The observation of the face in certain diseases (bulbar palsy, paralysis agitans, general paresis) is often of itself sufficient for diagnosis. Such indications are always of value as clinical guides, and may prove to be important supports of the final diagnosis; but too much weight should not be assigned to them until their significance has been verified by the results of more extended examination.

Mental State.—The examination of the mental state consists partly in observation of the patient's general conduct, and partly in questions.

Does the patient care for his dress and person? Is he excited and active, or depressed and quiet? Is the mood changeable or apathetic? Does the patient assume peculiar attitudes or have peculiar movements or expressions? Is he obstinate or easily led? Is he intelligent or stupid? if the latter, is there reason to suppose that he was always so? Is the speech rapid or slow, or incoherent, or is the patient mute? Is he attentive and quick of comprehension? Having the patient read and write is a useful adjunct in throwing light on these questions. Many of the questions which have to be put in the examination

of the mental state sound trivial, and almost silly, if the patient is sane and of reasonable intelligence. To avoid giving offense, a certain amount of tact is necessary on the part of the physician. Questions should be asked as casually as possible, during pauses in the routine physical examination.

If more than one examination is possible—and several are often necessary—the same questions should be asked at the succeeding examinations as at the first, and notes should be made as to how the different replies agree.

In the replies (or lack of them) to his questions, the physician should satisfy himself that the patient is not feigning; that he is not controlled in his answers by delusions; whether, if the patient fails to answer, it is because of depression, or because, as in maniacal conditions, no attention is paid to the questions put. It should not be forgotten that in any of these latter conditions the person under examination may give incorrect answers or no answers at all, and still be perfectly aware of all that is passing.

In insanity there is invariably more than one mental faculty affected. In a general way, however, we can investigate separately consciousness and orientation, memory and intelligence, and must always look for illusions, hallucinations, and delusions.

Coma, or unconsciousness, is usually readily apparent. In the lighter degrees, it may be necessary to test specifically for it, by noting how the patient responds to questions and to various visual and tactile stimuli.

Orientation, closely allied to consciousness, is the ability of an apparently conscious person to appreciate matters connected with himself and his environment. It is dependent upon a variety of mental faculties. To examine for it, the patient is asked his name, age, residence, and occupation; the date, namely of month and of week; where he is at present, how

long he has been there, where he came from and how—then where he was a week ago, a month ago, a year ago. Lack of orientation in varying degrees may be demonstrated by the foregoing questions. The patient may show himself entirely unappreciative of his surroundings; or he may realize them at one examination and not at another; or he may be only partially disoriented. Thus he may know the facts directly connected with himself, yet not know the date.

The foregoing questions also, reveal clews as to memory. In addition, with the accident as a starting point, inquiry should be made as to the patient's memory of it, of events prior to it, and of subsequent ones. Regarding the accident itself, care should be taken to be certain that the patient's statements are products of his own knowledge, and not the promptings he has subsequently received. After an accident in which unconsciousness was a direct result, memory is apt to be very cloudy as to all events relating to it, both those which transpired during the period of partial unconsciousness as well as such as took place while the patient was apparently conscious. In retrograde amnesia, there is loss of memory for events preceding the accident. Such memory-loss frequently follows severe brain injuries and is limited to the occurrences of a few hours or, sometimes, several days before. In some rare cases of hysteria, retrograde amnesia extends back for much longer periods. Questions intended to determine the condition of the memory for events which are in direct relation to the accident readily suggest themselves.

In addition, inquiry should be made regarding occurrences in early life. The names of friends, of places of residence, of occupations, of dates and occurrences generally, of years back. Under this heading, also, comes the inquiry as to the retention of facts of general knowledge. In this regard it is manifestly impossible to formulate a series of questions applicable to all

cases. But, be he native or foreign, cultured or ignorant, there are certain things that most adults can be assumed to have known. Among them are the days of the week, of the month, the ability to count, the alphabet, the names of prominent persons, contemporary or historical, the names of important geographical features, of capitals, the dates of battles, the reasons for wars, etc. With due allowances for previous education, some or all of these can be asked. The events subsequent to the accident should be inquired into with similar detail. The time which has elapsed, the treatment pursued, how, when, and where, the doctor's name, the progress of convalescence, etc.

The retention in memory of present impressions is closely related to attention. This can be tested by inquiry as to occurrences which have just taken place; also, by asking the patient to write one or more sentence at dictation, and noting if letters or words are left out. Memory-loss may be present in all degrees. Events may make so little impression on the mind that the patient cannot recall them under any circumstances; or, with prompting, he may be also to recall them fairly well.

Personal investigation by the examiner is necessary for proof of loss of memory. For some patients—e. g., in general paresis—may claim good memory, while in reality the memory is faulty. Others—e. g., in neurasthenia—may say the memory is poor, while in reality it is perfect. In melancholia the memory is generally good, though the patients often say they can remember nothing. In other forms of insanity the memory may be perfect.

This fact is often lost sight of by cross-examiners, who, in testing the mental state of a witness, examine the memory chiefly or solely, whereas in many affections the memory is perfect, should be assumed to be so and the examination con-

fined to other mental faculties. Illusions, hallucinations, and delusions are important mental symptoms.

An illusion is a false sensory impression produced by actual perception of real objects, but misinterpreted or exaggerated. The patient hears the door close, but thinks it is a cannon; sees the pattern on the wall paper, but thinks it a devil's face grinning at him. Illusions are especially characteristic of delirium and are usually fleeting.

An hallucination is an apparent perception for which there is no corresponding object. The patient hears voices when there are none, sees objects not apparent to others, smells odors which do not exist.

Hallucinations may be fleeting or fixed. In inquiring for them it is advisable to put the questions in a way that the patient may believe it is the sense organs, and not the mental faculties, that information is required about. For example, the patient is asked if his hearing is acute, if he ever has noises in the ears, if these noises ever form themselves into articulate sounds and the sounds into words, and if so, what the words are.

Delusions are false beliefs. There may be therefore both sane and insane delusions. Of insane delusions, the commonest are those of persecution. The patient thinks there is a plot against him, that his food is poisoned, that he is being followed, that persons are talking against him, that he is under hypnotic or spiritualistic influence, etc. Insane persons are often very secretive about their delusions, so that painstaking questioning and several examinations may be necessary before they can be brought out.

The ability to read, to write, to comprehend, or to repeat written or spoken words, to recognize objects by any of the special senses, gives important information in regard to aphasia or psychical speech disturbances. Dysarthria, or in-

terference with speech through physical causes, becomes apparent upon examination of the cranial nerves.

SMELL.—*Olfactory Nerve.*—The sense of smell is impaired or lost in injuries to the olfactory nerves and in hysteria. It should be remembered, also, that it may be diminished in diseases of the nose, and diminished or lost in injuries to the nasal bones. In testing for it one nostril should be taken at a time, and the other nostril tightly closed.

The substances ordinarily used are musk, peppermint, asafetida, and acids. They should be in solution, and kept in bottles with rubber corks.

THE EYES.—Although examination of the eyes includes the examination of several nerves, they may be most conveniently considered together. In all doubtful cases, testing of the various ocular functions should be intrusted to some one especially familiar with them. Every physician, however, ought to be able to examine for and understand the evidences of prominent ocular symptoms, and should be able to use both the ophthalmoscope and the perimeter.

The first step in the examination of the eyes is to notice the size of the palpebral fissure and the prominence of the eyeball. The condition of the upper lid as to elevation or drooping (ptosis), the ability to close or to open the eye, render important information regarding, respectively, the seventh and third nerves. The pupils should be examined for actual and comparative size one to the other, and for irregularities of outline such as may have been caused by preceding iritis. The contraction of the pupil under the influence of light is usually tested either by covering the eye and then observing, when it is uncovered, if the pupil grows smaller, or, if the iris is very dark, the converse of this procedure is often easier—i. e., after the eye has been exposed to light, it is covered with a dark object, and it is then observed if it dilates in the shadow.

The power of accommodation is determined by having the patient alternately look at a near object and then at a distant one, and observing the change in the pupillary circumference. All these tests should be tried at first with each eye separately, while the other is closed, and then with both eyes open.

The Argyll-Robertson pupil responds during efforts at accommodation, but not to light. It is one of the most important signs in nervous pathology, as, when sight is preserved, it is only found in locomotor ataxia, cerebral syphilis, and dementia paralytica.

Paralysis of the ocular muscles may be at once apparent by pronounced deviation of one or both eyes. In slighter degrees its presence is shown by the patient's inability to perform the movements controlled by certain muscles. It is accompanied by double vision.

The information obtained by means of the ophthalmoscope is always valuable, and not infrequently permits a diagnosis which otherwise could not have been made. In recommending its general use, however, it must be emphasized that it is an instrument requiring considerable skill and constant practice. To understand the significance of the pictures which it reveals implies its almost daily use in both health and disease. In the hands of a novice, normal variations may be regarded as evidences of morbid conditions, and pathological changes, significant even when slight, may escape detection. Few general practitioners have the opportunity to acquire the skill which is necessary to obtain reliable results from ophthalmoscopic examination, and it is accordingly advisable to have their observations and conclusions submitted to the control of a competent ophthalmologist, who shall at the same time determine refractive errors if they exist, and the presence or absence of such insufficiencies of the ocular muscles as only appear by the use of prisms.

The limits of the field of vision are obtained by the perimeter. The use of this instrument requires no particular skill. It is, however, a too frequently forgotten fact that perimetric examinations are only of value when supplemented by general examination of the eyes.

A limitation of the visual fields can only be diagnosticated as functional when examination has proved an absence of organic disease or defects which might explain it.

There are many varieties of perimeters, of which the most useful, from the neurologist's standpoint, is the one devised by Schweigger (Fig. 1). It can be taken apart, is easily portable, and furnishes sufficiently accurate results. This instrument is held by the patient so that the cupped end of the upright, *a*, is placed just under the lower rim of the orbit, and the patient is directed to look through the hole, *b*. The movable arc, *c*, can be placed at will at different meridians, which are indicated by the pointer, *d*. The object carrier, *e*, is fitted with jaws, into which can be inserted the disks containing little squares of different colors and of white. In examining for the field of vision the eye not under examination is covered, and the object is removed from the periphery of the field toward the center. As soon as it is perceived, the degree is read from the scale and is marked on the perimetric chart.

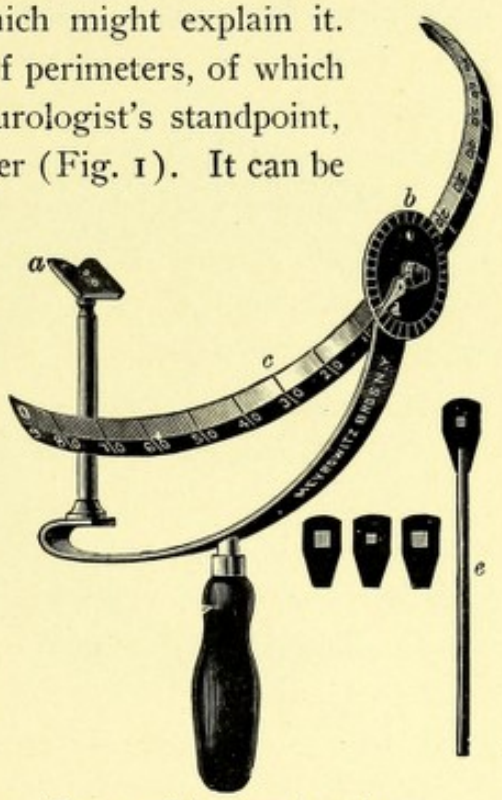


FIG. 1.—Schweigger's perimeter.

As indicated by the chart (Fig. 2), the normal field of vision for white on the horizontal meridian is 90° on the

temporal side, and 60° on the nasal side; on the vertical meridian it is 55° superiorly and 70° inferiorly. The outline of the entire field as indicated in the chart is fairly constant in normal eyes. It is subject to slight variations in different individuals within normal limits, but a diminution of 10° must be regarded as of pathological significance.

The fields for the different colors are smaller than those for white. They range in size from the field for blue, the

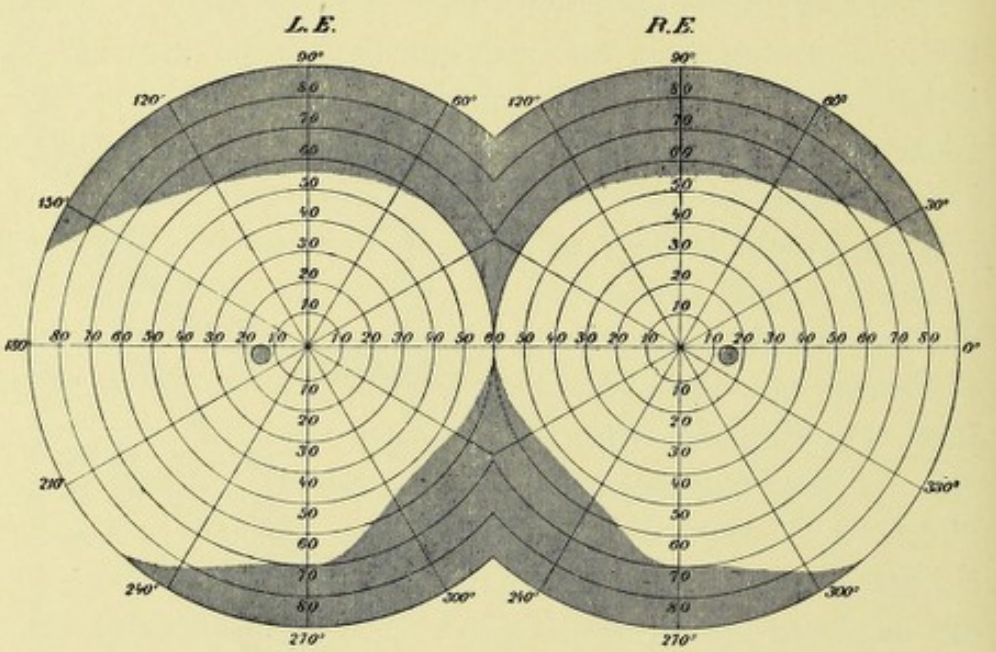


FIG. 2.—Normal field of vision.

largest, through those for yellow, orange, red, and green in the order named, to that for violet, which is the smallest. It is usually convenient to limit the examination of the color fields to those for blue and red.

The methods of examination of the other cranial nerves, with the exception of the eighth and the various gustatory fibers, will be found in the description of their injuries, so that they need not be given here. A few words must be said, however, regarding the senses of hearing and of taste.

HEARING.—*Auditory Nerve.*—Tests of hearing should be made for one ear at a time, the other ear being tightly closed. Lesions of the auditory nerve can be distinguished from those affecting the external or middle ear by the presence or absence of "bone conduction." When deafness is the result of inflammations, or defects which affect the ear without involving the auditory nerve, the patient can hear the tick of a watch or the sound of a tuning fork, when either of these objects is placed on the mastoid process or on the vault of the cranium, very much better than when it is held directly opposite the auditory meatus; on the other hand, when the auditory nerve is affected, either in its course or in its terminations within the labyrinth, the deafness is equally pronounced in whichever situation the testing object is held. In the first case, in which the auditory passages are obstructed, the bone conducts the sound to the auditory nerve; in the second case it is the nerve itself which is affected, and deafness consequently exists independently of conduction.

TASTE.—The sense of taste depends upon several nerves, and it is rarely if ever lost as a result of organic injuries which do not cause coma or extreme laceration of the mouth and pharynx. Injuries to the trigeminus, or to the facial nerve outside the skull, may cause loss of taste in the anterior two-thirds of the tongue, on the same side; to the glossopharyngeal, in the posterior two-thirds. Loss of taste also occurs in hysteria. Flavors are recognized by means of the olfactory nerve. The gustatory function has to do with the qualities salt, sweet, bitter, and sour, and certain metallic sensations only.

In examination for the sense of taste the test solutions (sodium chloride, 10 per cent; sugar, 5 per cent; tartaric acid, 10 per cent; strychnine, 0.1 per cent) are applied to the tongue by means of a glass rod while the patient's eyes are covered and his nostrils closed. The tongue is held outside the mouth, and

the examiner places drops of the testing fluids on it and inquires if the patient recognizes them. The examination is often unsatisfactory. Many normal persons cannot taste when the tongue is outside of the mouth, yet when it is withdrawn it is placed against the palate, and it is then impossible to localize gustatory abnormities if any exist. The galvanic current is a preferable means of testing for disturbances of taste. A very weak current, such as may be obtained from a single cell, is made to pass through the tongue from wire electrodes placed a short distance apart. In this way the situation of any loss of the ability to recognize the metallic sensations may be discovered.

Paralysis.—After the initial shock has passed away, traumatic paralysis from cerebral lesions is rarely absolute. Even when the use of the paralyzed member is greatly impaired, the patient can usually succeed in producing some slight movement. In injuries to the spinal cord, on the other hand, complete paralysis is frequent, so that the limbs lie useless and as though dead. In peripheral nerve lesions the paralysis is so distributed that some muscles may be absolutely paralyzed, while the motor power in others is as good as it ever was. The paralyses of hysteria also have characteristics of their own.

When the patient is unconscious, the examination for paralysis must, of course, be made without his assistance. If the coma is not profound, he may be seen to move the limbs of one side while those of the other side remain motionless; or the limbs of one side may remain unaffected by such peripheral irritations as pricking, pinching, or tickling—annoyances which the limbs of the other side endeavor to escape. Even when the patient is absolutely comatose, by observing differences of passive muscular resistance—such as are shown by the way in which the limbs, when lifted up, fall again to

the bed, or by the condition of the muscles as revealed by grasping them with the hand—inequalities of muscular power on the two sides can usually be recognized. When the patient is able and willing to assist the examiner, the task is much easier. If the loss of power is complete and generally distributed, it is self-demonstrative; but if partial, or if limited to certain muscles or groups of muscles, it may require considerable care and skill to elicit its exact character. The object may best be accomplished by directing the patient to perform certain movements and to repeat them in rapid succession, and to make all the resistance of which he is capable against the force exerted by the examiner. In this way may be found not only the situation of the paralysis but also its degree. The methods of examination for paralyses of the face and tongue will be described when speaking of those affections.

In the neck the trapezius and sterno-mastoid are the commonest muscles involved. Paralysis of either of them is indicated by an inability to move the head and neck in the various directions, or by abnormal attitudes. To detect palsy of the muscles about the shoulder joint, the patient should be required to make all the movements possible at this point, such as putting the hand on the head, the chest, the opposite shoulder, and the back, and the pressing of the arm firmly against the side. He should try to maintain these positions while the examiner attempts to overcome them. By comparing the two sides in this way, degrees of relative weakness may be demonstrated. Similar procedures are applicable at the elbow and wrist joints. A means of detecting slight degrees of paralysis in the extensors and flexors of the wrist and fingers is to have the patient rapidly open and close the hands for as long a time as he is able. By this test is shown unilateral weakness as well as the stiffness of the joints which is so important an indication of certain forms of injuries to nervous structures. The grip may

be tested by having the patient grasp and squeeze with both hands the two hands of the examiner. Weakness in the interossei and lumbricales shows itself by impairment in the finer movements of the fingers. The patient has difficulty in buttoning his clothes, in picking up pins or other fine objects, in writing, and in similar movements which require muscular strength and precision.

The examination of the muscles of the abdomen, back, and lower extremities is conducted on similar lines. The patient, while lying at full length, should be requested to raise himself

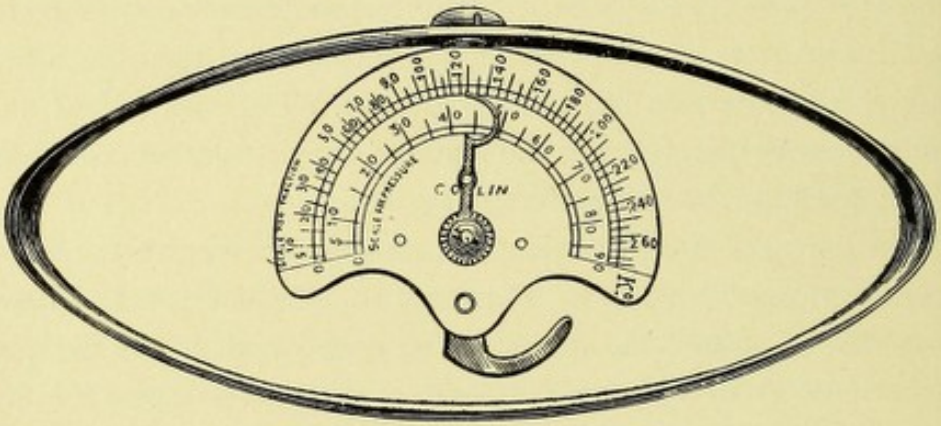


FIG. 3.—Hand dynamometer.

to the sitting posture without using legs or arms; he should be told to " bear down " while the hand of the examiner is on the abdominal wall; he should be observed while getting into a chair and while getting out of it without using the hands. The movements at the hip, knee, and ankle joints should be carefully noted. Assuming and leaving the " squatting " position tests the muscles on the front and back of the thigh; they may also be tested by stepping with one leg on to and down from a chair without assistance. The muscles of the legs may be tried by forward, backward, and lateral movements of the foot. For the examination of these motor functions various instruments are employed, notable among which is the hand dynamometer

(Fig. 3), used to determine in kilogrammes or pounds the strength of the grip. Dana has devised an apparatus by which the strength of the muscles of the lower extremities may be similarly measured. While these instruments are often convenient, they are by no means essential for securing an accurate idea of the muscular power, and, in my experience, equally reliable results may be obtained without them.

RIGIDITY AND CONTRACTURES.—The rigidity or spasticity which appears after lesions of the central neuron, either in the brain or in the spinal cord, and which eventually leads to contractures, begins to appear in a few weeks after the receipt of the injury. It is characterized by a stiffness of all the muscles and by an overaction of the ones which are the least paralyzed. There is a consequent limitation of movement at the joints, which may ultimately render them absolutely immobile. This late rigidity is unmistakable. The contractures, usually in flexion, to which it gives rise are highly characteristic. These are often of vice-like rigidity, causing marked disability and deformity. When of cerebral origin, contractures involve all the muscles of a limb, though unequally. When due to the paralysis of a single nerve, the contracture exists in the opposing muscles to the ones paralyzed.

Cerebral lesions which cause paralysis may induce rigidity in the paralyzed limbs at once. This is called early rigidity, and is indicative of an irritative lesion of the upper motor pathway. When the loss of power is well marked it can easily be detected that the limbs are paralyzed as well as stiff; but it not infrequently occurs that the affected limb is rigid, and still the patient can move it. It is in such cases that rigidity is an important diagnostic symptom. Its existence, if the patient is conscious, can easily be demonstrated by having him execute rapid movements which require a considerable flexibility in the muscles, and observing whether they are done with

normal ease and speed. When there is coma, the presence of the rigidity must be determined by the way the limbs feel. This is easy to do if the stiffness is pronounced, as the limb then feels abnormally hard and resistant to the touch. But when present in slight degree only, it is usually observed by no one except by him who looks for it carefully, and who is accustomed to looking for it.

Tremor.—In the examination for tremor one should note the character, i. e., whether coarse or fine, whether it is constant or intermittent, whether it occurs during rest, whether intended movements increase it, whether coördinated or choreiform, and the effects upon it of external influences.

Fibrillary tremor, or fibrillation, is a very fine, rapid, wavelike contraction of individual muscle fibers. It causes no movement, and although its occurrence is involuntary it is often observed by the patient himself. It is seen chiefly in the face, in the muscles around the shoulders, and in those around the base of the thumb. Although a nearly constant symptom in muscular atrophy, and in most atrophic spinal-cord lesions, it is not pathognomonic of any disease, as it may occur in asthenic conditions which are brought on by excesses of any kind. Before ascribing to tremor a too serious significance, the physician should remember that it occurs as a result of many factors. Exertion, excitement, cold, alcoholism, etc., may all cause tremor of various kinds and degrees.

Tremor of the face is observed during the examination of the head. In the extremities it exists chiefly in the arms and hands, though it may also occur in the legs. By causing the patient to stand with his arms stretched out in front of him and his fingers spread apart, by having him execute movements with the arms and legs, the distribution and character of the tremor may be ascertained. Tremor of the hands is further

shown by the handwriting; and of the legs in attempts made by the patient to touch objects with the toes, to describe circles on the floor, to lift the foot to a given height, etc.

MORBID MOVEMENTS.—Convulsions, athetosis, spasmodic twitchings, and similar purposeless movements, when resulting from accidents, are symptoms of so pronounced a character that there is rarely any difficulty in referring them to the lesions or conditions of which they are the results.

A distinction should be made between a tic and a spasm. Spoken of in this sense, a spasm is an involuntary contraction of a muscle or muscles supplied by a single nerve. Spasms are usually reflex in character, and often a reflex cause for them can be discovered. A tic is a complicated, coördinated, involuntary and recurring contraction of a group of muscles supplied by a number of nerves. Tics, therefore, are not true reflexes, but are of cortical origin. They usually develop as a result of emotional causes or from accentuated habits. Habit is always a factor in their persistence.

Some of these symptoms are constant. Others, such as epileptic convulsions, are periodic and are rarely seen by the physician. In making his decision as to the character of these latter, he must depend largely upon information from outside sources. In hospitals, where the patients are under the constant observation of the house staff and of trained nurses, there is rarely any question as to the reliability of the facts reported to him. But in the absence of observations by skilled assistants he should be extremely cautious lest he attribute too much weight to symptoms he does not see.

GAIT.—If the patient is able to walk, valuable and positive information concerning the condition of the motor mechanism may be obtained by the observation of the gait. Aside from such individual peculiarities of locomotion as are due to disorders of the bones or joints, there are certain types of gait

which are absolutely typical as expressions of morbid conditions and which can never be successfully simulated.

The Ataxic Gait is the most characteristic of these. It occurs after lesions to certain sensory fibers and is especially seen in locomotor ataxia. In that disease there is no loss of motor power, but there is loss of the sense of position, so that the patient does not walk well because he does not know where to put his feet. More force than necessary is used in bringing the legs forward; the feet are lifted too high and thrown too far outward, and finally come down to the floor, the heel first and then the toe, with a sharp slap. In slight degrees of ataxia, although the patient may get around fairly well, and can often walk rapidly and go long distances, there is difficulty in walking in the dark or in going downstairs. When the ataxia is more pronounced, the patient walks with his eyes riveted upon his feet, trying in this way to direct the movements of the legs. When the ataxia is still more exaggerated the patient is only able to walk with the assistance of a cane or of an attendant, or locomotion may be altogether impossible.

Gait of Motor Paralysis.—The gait of motor paralysis has essential differences according to whether it is the central or peripheral motor neuron which is affected.

With injury to the central motor neuron, the paralysis is usually only partial, and is associated with stiffness; in lesions of the peripheral neuron it is more complete, but the muscles are soft and all rigidity in them is absent.

The types of gait dependent upon affections of the upper neuron are hemiplegic and, when both sides are affected, spastic. The hemiplegic gait is usually spastic, but the term spastic is generally understood to apply to cases in which both legs are affected.

Hemiplegic Gait.—In cerebral hemiplegia, after the acute effects of injury have passed away, the leg ordinarily regains

quickly much of its power, but the descending degeneration in the motor pathway causes it to become very stiff. In walking, the patient throws the weight of the body toward the unparalyzed side and circumducts the paralyzed leg so that it describes a segment of a circle in its forward movement. From this peculiarity it is sometimes called the " mowing gait." The foot is dragged at its tip and inner portions only, and there is little movement at the knee or ankle joints.

Spastic Gait.—The spastic gait is the gait of double hemiplegia. It occurs as the result of bilateral lesions of the brain, or of lesions of the spinal cord above the lower dorsal region.

Its characters are essentially the same as those of the hemiplegic gait, except that both legs are involved and that the degree of paralysis is greater than in hemiplegia. In walking, the patient stiffly circumducts one leg until the foot has crossed in front of the foot of the opposite side. The same thing is then done with the other foot. From this crossing of one foot in front of the other the spastic gait is sometimes called " crossed-leg progression." The soles of the feet leave the floor but little or not at all, and the power of locomotion is consequently impaired seriously. If the patient carries a cane he keeps it in front of him, and leans on it with both hands.

The Equine or Stepping Gait is the type of gait in flaccid paralysis of the anterior tibial group of muscles. It is due to lesion of the peripheral neuron, and is seen in peripheral nerve palsies and in injuries of the spinal cord in or below the lumbar enlargement. It is never associated with rigidity. As a result of the paralysis, when the lower limb is raised the foot drops, so that the toe touches the floor. The toe is then dragged, or the leg is raised higher than normal, in order that the foot may clear the floor. There is, accordingly, pronounced " knee action," and hence the term *stepping* or *equine* gait.

In addition to these characteristic and constant anomalies

of progression there are other disturbances of gait which are frequently observed and which, when present, are at once suggestive of the conditions to which they are due. Of these may be mentioned the careful steps of the patient with lumbago; the inert dragging of the feet in hysteria; the quick yet careless walk often seen in the early stages of dementia paralytica; and the festination of paralysis agitans, of which Trousseau said that " the patient was running after his center of gravity."

Intimately allied to the gait is the ability of the patient to maintain his equilibrium. Normal persons cannot hold themselves perfectly motionless when standing with closed eyes, but in them the swaying is slight. When the swaying is pronounced it constitutes a symptom of importance and is called the Romberg symptom.

If the patient is in bed and unable to use the legs, it is, of course, impossible to make these tests. But many persons remain in bed for long periods of time, although they are perfectly able to walk. It is always desirable to have the patient make an attempt, at least, at walking; when this is not possible, much information may be obtained by observing how the patient lies in bed, how the limbs are held, and whether their attitude indicates pain.

Sensation.—Although the clinical value of the anomalies of cutaneous sensation, especially as observed in the functional nervous diseases, has been subjected to much adverse criticism, the fact remains that anæsthesia, whether of functional or organic origin, is one of the most valuable aids in neurological diagnosis, and no examination of a nervous case can be regarded as satisfactory in which the presence or absence of anæsthesia has not been definitely determined. For sensory examinations there is a variety of instruments which are of greater service to the anthropologist and experimental psychologist than to the clinician. It is not only usually impos-

sible to exercise in the clinic the exactness of the laboratory, but it is generally true that any deviation from normal sensibility is of very questionable clinical value when it is so slight as to require instruments of precision for its determination.

The æsthesiometer, the instruments for recording degrees of pressure, the concealed needles for testing sensibility to pain, and the metal piles for determining the degree of thermo-anæsthesia are rarely if ever necessary in clinical examinations; a needle, a piece of cotton, and two test tubes, one filled with hot and the other with cold water, are usually sufficient apparatus, and furnish results as reliable as those obtained by more delicate instruments.

During the examination the patient's eyes are covered, and he is asked to indicate *when* he feels a touch rather than *if* he feels it. It is advisable from time to time for the examiner to pretend to touch the patient without actually doing so, and then noting whether or not he responds; for the patient, although he cannot see, may say, on hearing the rustle of the coat sleeve, that he felt contact when none was made. It is often impossible or inadvisable to extend the examination over the whole cutaneous surface; the report should then state what parts were not examined and the reasons for the omissions.

The limits of the anæsthesia are usually sharply defined in organic cases. In functional cases the boundary between parts which are anæsthetic and those which have retained normal sensibility is often difficult and sometimes impossible to accurately determine. Inasmuch as a thorough examination is fatiguing to both patient and examiner, it is advisable in such cases to be satisfied with an approximation of the anæsthetic limits rather than to overfatigue the patient, because if he becomes tired his answers are less reliable. The results of sensory examination are best recorded by anæsthesia charts, which are printed on pads with gummed backs, or which may

be stamped out with rubber dies. It is most convenient to mark the affected areas with ink or pencil on the patient, and then to transfer them to the chart. Anæsthesia of different characters may be indicated by vertical or horizontal lines, by crosses or circles, or by various other devices.

The different forms of sensibility should be examined sequentially. The condition of the sense of touch of the skin, as well as the patient's ability to indicate the part touched, is ascertained by light brushes with the cotton. The state of the conjunctivæ and the lips, tongue, and throat may be best examined at the same time. Slowed conduction is essentially a symptom of chronic processes.

For the *sense of pain*, examination with a needle furnishes the desired information in most cases, although when simulation is suspected other devices (see Part III) are sometimes resorted to. In testing the sense of temperature two test tubes, one filled with cold water and the other with water at a temperature of between 120° and 130°, will enable the physician to discover any differences in the perception of either heat or cold, as well as the patient's inability to distinguish between these two varieties of sensory stimulation.

The *muscular sense* is a complex function, and is usually abolished when anæsthesia is total. It includes the sense of position of the limb and the appreciation of the amount of exertion necessary for the performance of voluntary movements. When the muscular sense is lost and motor power is retained, the patient, in attempting movements of the limbs, throws them about, often with great force, but without the ability to properly and quickly direct them toward the objective point. This condition of ataxia is seen in the gait of tabes, and may be demonstrated for the upper extremities by directing the patient to perform definite movements with the hands and observing how accurately he does them. It may further

be brought out by attempts of the patient to put the limb of one side in a position conforming to that in which the limb of the other side has been placed by the examiner, or by his inability to tell with closed eyes the exact position that a limb is in.

Astereognosis is the loss of the sense of spatial relations. As a brain symptom it will be again referred to. The patient is unable to recognize objects by the sense of touch. To examine for astereognosis the patient's eyes are closed, different objects are placed in his hands, and he is asked to give the names and the uses of each of them.

REFLEXES.—The condition of the pupillary and palpebral reflexes are determined at the time of the examination of the eye; the pharyngeal reflex, at the time the throat is examined for anæsthesia.

There remain a series of reflexes termed superficial, and deep or tendon reflexes, which should be examined together. They are extremely valuable, and often constitute the one link necessary to complete the diagnostic chain. The most important superficial cutaneous reflexes are the cilio-spinal, those of the abdomen, the cremasteric, and the plantar.

The *cilio-spinal* consists in a dilatation of the pupil when the skin just above the clavicle is irritated by pinching or by the faradic brush. Its loss is indicative of lesion to the cervical sympathetic either in its course along the vertebral column, in its passage into the spinal cord through the first or second dorsal nerve roots (possibly also the eighth cervical) or in the first dorsal and lower cervical segments of the spinal cord. The *abdominal* reflexes are epigastric, abdominal, and hypogastric. Their action in health is seen by the contractions of the skin induced by quickly stroking these regions of the abdomen with the head of a pin. The *cremaster* reflex occurs as the result of irritation of the inner side of the thigh, either by

striking or by pinching, which causes a contraction of the cremaster muscle and an ascent of the testicle. In the plantar reflex, irritation of the sole of the foot causes a flexion at the knee and hip joints and a more or less violent agitation of the whole limb.

The most important of the deep or tendon reflexes are those concerned in the contraction of the triceps, the supinator longus, the biceps, the extensors of the wrist, and the quadriceps extensor of the leg. For their elicitation it is necessary that the limb be pendent and free, the muscles relaxed, and that

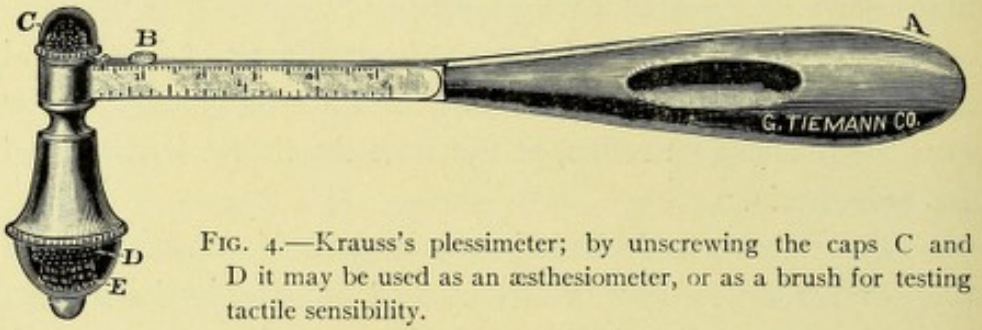


FIG. 4.—Krauss's plessimeter; by unscrewing the caps C and D it may be used as an æsthesiometer, or as a brush for testing tactile sensibility.

light taps be applied over the tendons of these muscles, preferably with a small hammer provided with a rubber tip, known as the plessimeter (Fig. 4).

The *triceps*, or *elbow-jerk*, and the *supinator* and *biceps* reflex are not necessarily present in health, although they may be. Their presence or absence is readily determined by taps, applied for the former just above the olecranon process, and for the latter (Fig. 5) just above the lower end of the radius on its outer surface.

The *knee-jerk*, or patellar or quadriceps reflex, is by far the most important of all. In health, a light tap upon the patellar tendon induces a quick but slight extension of the leg. The action is said to be hypertypical when it can be elicited by taps on the muscle above the knee. There is no absolute standard by which it may be determined when a hypertypical knee-jerk

becomes exaggerated. Very great exaggeration is usually associated with ankle clonus, and with the Babinski phenomenon, and even in the absence of these additional signs, when light taps bring about quick and forcible muscular contraction, the condition must be regarded as exaggerated. Since absence

FIG. 5.—Showing method of obtaining the biceps and supinator jerk.

of knee-jerk is occasionally observed in persons who are apparently normal, this symptom alone is not sufficient for the diagnosis of disease.

When the examination has not been painstaking it may be concluded that the knee-jerk is absent, although the em-

ployment of the proper means for its elicitation would have demonstrated its presence. Such an error is particularly frequent when the reflex activity is diminished, either as a personal peculiarity or as a result of disease.

In the larger number of cases the knee-jerk may be obtained by having the patient sit in a chair, one leg crossed over the

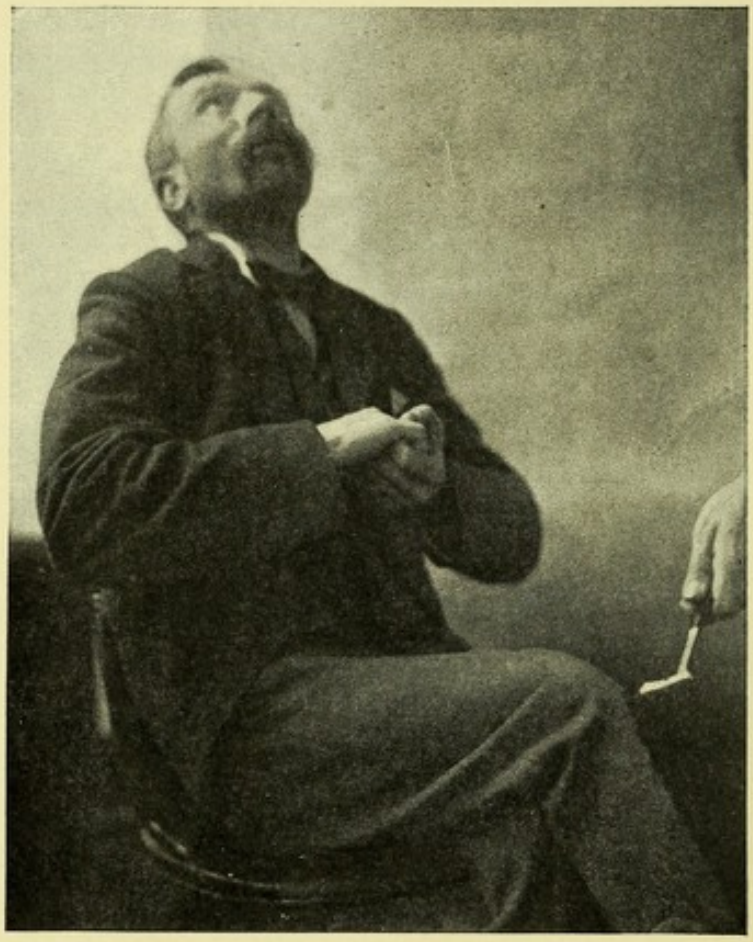


FIG. 6.—Showing the Jendrassik method of obtaining the knee-jerk.

other, while the physician taps the patellar tendon. If there is no response, the Jendrassik (Fig. 6) method of reënforcement is resorted to. The patient, having crossed the legs, pulls forcibly on his closed hands and looks up at the ceiling. Before striking the tendon the examiner should satisfy himself that

the patient's hamstring muscles are relaxed. If none of these methods is successful they may be varied by having the patient sit on a table so that the legs hang over the edge; or the examiner may place one arm under the leg to be examined, so that the hand rests on the knee of the opposite leg. In this way the leg is elevated from the floor, and at the same time the condition of the hamstring muscles is ascertained. If the leg is held perfectly limp and no reflex can be obtained by any of these methods, it is fair to infer that the knee-jerk is lost. Such an inference is not justifiable, however, if the flexor muscles of the legs cannot be made to relax, as sometimes occurs in persons who persistently refuse to understand what is said to them. For such cases the most that can be said is that the knee-jerk was not obtained.

The Babinski reflex consists in extension of the great toe (dorsal flexion) when the sole of the foot is irritated. This is the reverse of the normal, in which plantar flexion results from such irritation. The absence of the Babinski reflex does not negative organic disease. But when present, it indicates impairment in the pyramidal tracts and is especially useful in the differential diagnosis of hysteria. The only exception is in very young children, in whom the Babinski may occur normally.

Foot or Ankle Clonus is usually regarded as an evidence of impaired conduction in the pyramidal tracts, although it may rarely occur in functional cases. In these latter, however, it rarely presents the exaggerated characteristics which stamp it as a symptom of organic disease. In organic disease foot clonus is usually associated with an increase in the knee-jerk. In rare cases of lesions low down in the cord, clonus is present, with absent knee-jerk. To examine for it the patient sits down and the examiner supports the under surface of the knee with the left hand and grasps the toes with the right.

The foot is then sharply flexed. If ankle clonus is present, there ensues a series of rapidly alternating contractions of the flexors and extensors of the ankle.

TROPHIC DISTURBANCES.—*Trophic Disturbances of the Skin.*—These are directly connected with the peripheral circulation and with the sweat glands. Limbs or different parts of the body may be hot or cold, cyanosed or flushed, either continuously or alternately. Nutrition may fail, giving the "glossy" skin of neuritis, or the bedsores of spinal-cord disease. There may be an increase or absence of sweating; when one-sided, this indicates organic disease of the sympathetic. The changeable trophic disturbances are particularly frequent in the functional diseases.

Atrophy of Muscles.—Atrophy of muscle is recognized by sight, by touch, and by measurement. Occurring in localized muscles, or groups of muscles, it is indicative of disease of the peripheral neuron. In injuries to the anterior horns of the spinal cord, to the motor nerve roots and peripheral nerves, it is more or less conspicuous and prompt in appearance, varying with the severity of the lesion. Atrophy of whole limbs or segments of limbs is less common and less pronounced. Occurring thus, it is usually one-sided, and its degree is determined by comparing the circumference of the affected side with that of the sound side. Contrary to the general impression, in normal persons there is very little difference in the circumference of the limbs of the two sides. Hitchcock, from the measurements of 4,890 students at Amherst College between the ages of sixteen and twenty-six years, has established that the right arm is 7 millimeters (about one-quarter inch) larger than the left, that the difference in favor of the right forearm is the same, that the right thigh is 3.3 millimeters (about one-eighth inch) larger than the left, and that between the right and left calf there is practically no difference (1 millimeter). It is probable that in

persons who do heavy work with one arm only, these differences are slightly increased.

Disuse of a limb, for any cause, may bring about a slight degree of wasting. This does not usually exceed one-quarter of an inch for the arm or one-half an inch for the thigh. The atrophy in hemiplegia following cerebral hemorrhage is also usually very slight. After injuries to joints, an atrophy may ensue, more marked than the atrophy of disuse, and evidently

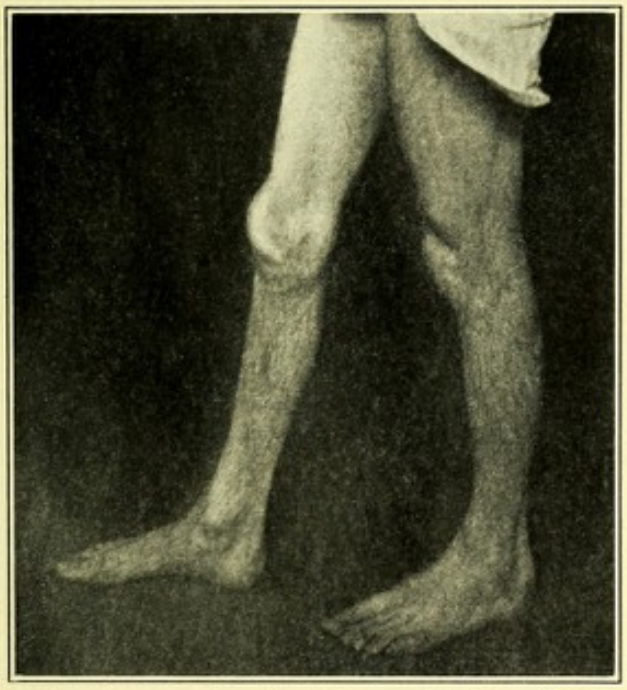


FIG. 7.—Case of reflex atrophy of the leg resulting from injury to the knee.

due to some trophic disturbance in the muscles. The muscles situated centrally to the joint are the ones commonly affected, though the whole limb may atrophy. The extensors are more frequently affected than the flexors, and may show excess of myotatic irritability, e. g., increased knee-jerk. The knee is the joint most frequently affected in this way. When the joint trouble is over, the patient usually recovers, though recovery is slow.

The following case (Fig. 7) illustrates this condition:

A previously healthy man, aged thirty-one, received an injury to the right knee by being struck by a baseball. The pain was severe and constant at first, but soon diminished in intensity. Four months later it again became severe, so that a cane was necessary in walking. There was no redness or swelling in the joint, but the leg was growing smaller. At an operation performed eight months after the accident no pus was found, but the bone was "softened," and some of it was chipped off. After the operation the pain gradually became less, but as a result of overexertion (walk of a half mile) the knee again became painful. At the time of my examination, two and a half years after the accident, the joint symptoms were at an end, and the limb was painful only after exertion. But the patient was unable to do any work requiring standing or walking, and used a cane and one crutch almost constantly. Examination showed an atrophy of the whole lower extremity. The circumference of the right calf was one inch less than that of the left, and of the right thigh one and a half inches less than that of the left. There was a general reduction in both galvanic and faradic excitability, without reversal of the normal reactions, and general weakness in the limb. There was no further evidence of nervous disease. The knee-joint was freely movable, not painful, and presented no signs of internal disease.

Electrical Examination.—In cerebral injuries it is often immediately evident that the battery can throw no light upon the character or situation of the lesion. On the other hand, electrical examination is indispensable for all affections of the lower motor neuron, and is often of great assistance in the diagnosis of the paralyses of hysteria.

Much information can be obtained from a simple faradic battery, although it is usually desirable to test with both the interrupted and the constant current. The sponges in the electrodes must be kept constantly moist, and one of them should have a small head and be furnished with an interrupter. A galvanometer is a useful addition to the galvanic battery, but it is indispensable only when the paralysis is bilateral, so that the reactions on the injured side cannot be compared with

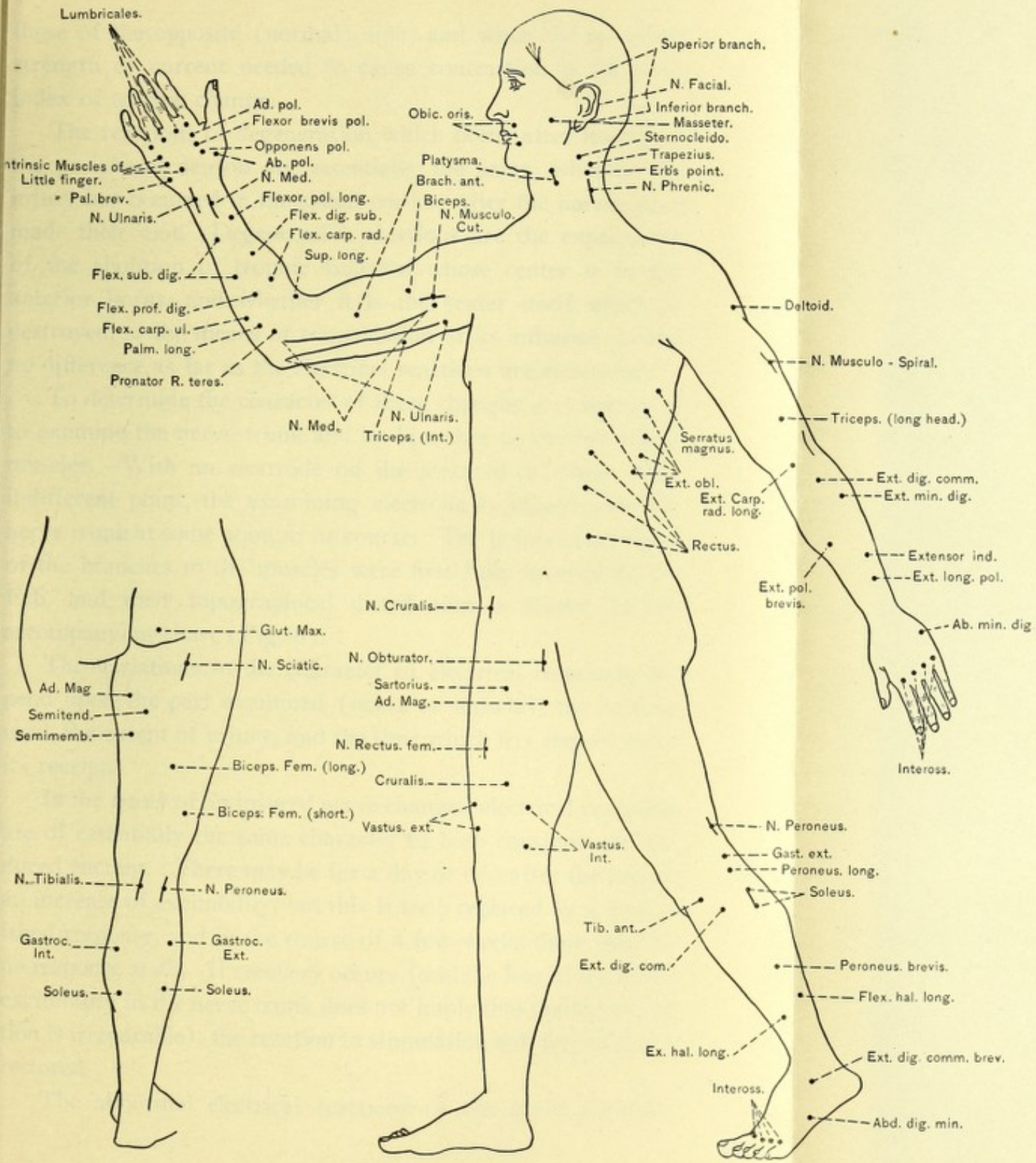


FIG. 8.—Diagram showing the superficial position of the large nerve trunks and the motor points for the muscles.

those of the opposite (normal) side; and when the increased strength of current needed to cause contraction is the only index of trophic change.

The reactions of degeneration which occur after lesions of the peripheral neuron are essentially the same, whether the injury has occurred in the spinal cord or after the nerves have made their exit. Degenerative reactions are the expressions of the abolition of trophic function whose center is in the anterior horns, and whether it is the center itself which is destroyed, or the means of transmission of its influence, makes no difference as far as the electrical reactions are concerned.

To determine the character of these changes it is necessary to examine the nerve trunk and its branches to the individual muscles. With an electrode on the sternum or some other indifferent point, the examining electrode is placed over the nerve trunk at some point in its course. The points of entrance of the branches to the muscles were first fully ascertained by Erb, and their topographical distribution is shown in the accompanying chart (Fig. 8).

The variations in the character of electrical responses depend upon the part examined (nerve or muscle), the current used, the extent of injury, and the time which has elapsed since its receipt.

In the *trunk* of an injured nerve changed electrical reactions are of essentially the same character to both constant and induced current. There may be for a day or two after the injury an increase of excitability, but this is soon replaced by a diminished response, and in the course of a few weeks there may be no response at all. If recovery occurs (and the loss of electrical excitability in the nerve trunk does not imply that the degeneration is irreparable), the reaction to stimulation will be gradually restored.

The abnormal electrical reactions of the *nerve termina-*

tions in the muscles are found by placing one electrode over an indifferent point and the examining electrode over the motor points of Erb, which correspond in position with the entrance of the nerve ending into the muscle.

At these points the electrical evidence of nerve injury differs with the two currents. Response to the *faradic current* may be hyperactive for a day or two after the injury, but it then rapidly decreases until about the second week; then there is no longer any response at all. When regeneration begins, often about the second month after injury, there occurs a gradual return of faradic excitability.

The *galvanic reaction,* on the other hand, after a day or two of normal or increased response, falls with the faradic; but instead of becoming extinct, like the faradic, it begins to rise in the second or third week. The increased response to galvanism may become and remain so marked that very weak currents produce forcible contractions. The increase of galvanic excitability may persist for many months and eventually be replaced by total absence of response; or about the same time that the muscles show a return of contractility to faradism the galvanic response decreases until it reaches and remains at the standard of health.

The accompanying diagram (Fig. 9) may graphically represent the behavior of the electrical reactions for the first four weeks after a moderately severe injury of a peripheral nerve. It is seen that in the muscle the response to faradism is hyperactive for a few days, then rapidly sinks in about the second week. The galvanic curve, on the other hand, after an initial rise and fall, begins to rise after the second week, and remains for some time above the normal. In the nerve the behavior of the two currents is seen to be the same.

These changes in electrical reactions indicate degenerative processes of greater or less severity in the nerve trunk and

nerve endings. They are quantitative. Still more important are the qualitative changes as seen in the " reaction of degeneration."

The reaction of degeneration is found in the muscles only, and consists in a reversal of the normal reaction to galvanism.

In health the contraction induced by closing the circuit is greater when the cathode is over the motor point than when the circuit is completed with the anode in that situation. The

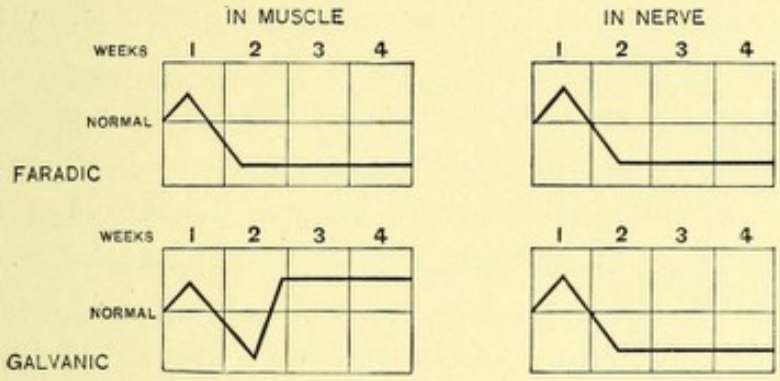


FIG. 9.—Diagram to illustrate the electrical reactions occurring in a nerve after a moderately severe injury.

cathode-closure contraction is greater than anode-closure contraction, or, graphically:

$$CaCl > AnCl.$$

In disease these reactions may be reversed, so that

$$CaCl = AnCl,$$
$$\text{or } CaCl < AnCl.$$

In degeneration of nerves the contractions of the muscles to both galvanic and faradic currents are often slow and worm-like, instead of being quick and tonic, as in health. This ver-

micular reaction, like the reaction of degeneration, is indicative of serious though not necessarily irreparable injury. Such are the changes in electrical response most frequently observed after nerve injuries.

Lumbar Puncture.—This little operation is of great value in diagnosis, especially in injuries of the brain, to determine the existence of intermeningeal hemorrhage. Performed with care as to asepsis, it is without danger. It is somewhat painful, and may be preceded, if necessary, by freezing or cocainization of the skin. The instrument consists of a needle, with stylet, at least three inches long, and of about one-sixteenth inch diameter.

The patient lies on the left side, near the edge of the bed, the head bent and thighs flexed as much as possible. The puncture in adults is best made between the third and fourth lumbar arches; in children, one or two spaces lower. The fourth lumbar spine is readily identified, being on a level with the crest of the ilium. The instrument is inserted about three-quarters of an inch to the right of the median line, and is pushed upward and inward. The stylet is withdrawn, and the fluid flows, drop by drop, or more freely, varying with the pressure. From 5 c.c. to 10 c.c. may be withdrawn. The fluid should be examined microscopically for blood, cellular elements, etc.

Determination of Blood Pressure.— Arterial pressure is raised in acute compression of the brain, in depressed mental states, in conditions of severe pain, and usually in arteriosclerosis; it is lowered in states of mania and in shock. It is measured by the sphygmomanometer.

The approximately normal mean blood pressure, as determined by Thayer with the Cook Riva-Rocci sphygmomanometer from 276 healthy individuals, arranged by decades, is as follows:

1–10 years	104.6	mm.
10–20 "	128.7	"
20–30 "	136.9	"
30–40 "	140.8	"
40–50 "	142.2	"
50–60 "	154.8	"
60–70 "	180.0	"

These measurements were taken with the patient at rest in the recumbent position. All such measurements should be taken in this way.

Janeway says that with his instrument, with the broad cuff, the readings, in adults, should be 20 mm. to 30 mm. lower.

Referring to his own instrument, Janeway says: " I regard with suspicion any pressure above 135 mm. in a young person; 145 mm. in an older one, when found on several examinations and with due precautions."

When used for the determination of the general condition of the vascular system, several observations a day for several days, are desirable. In head injuries, the pressure should be measured every fifteen or twenty minutes, as long as the patient is in danger. A sudden rise in pressure of 20 mm. or more, in the victim of a head injury is strong indication of compression.

Estimation of Injury.—In the clinical descriptions to follow an effort will be made to give, as far as possible, the prospects for recovery from the various diseases. But a word should be spoken here concerning the effects of injuries upon the social and industrial life of the individual. By the passage of a law in Germany in 1884, workmen and employees (with the exception of those of commercial, of domestic, and of a few other callings) whose annual wages do not exceed 2,000 marks are insured against accidents incident to their various occupations. Such persons are entitled from the fourteenth week after the accident to an indemnity of two-thirds of their wages, payable monthly. All preëxisting diseases which are brought

into activity or whose course is hastened by such an accident must be indemnified for under the law. The insurance money is paid by the " Berufsgenossenschaft "—that is, by an association of the employers of the various trades. The establishment of the amount of indemnity is usually based upon a medical certificate and fixed by the Berufsgenossenschaft. The injured workman may appeal from this.

If, after settlement, an important change in the injured person's condition takes place, the indemnity may be readjusted, either to the advantage of the workman or to that of the employers.

The amount of indemnity is graded in accordance with the disability. In cases of total disability, the full amount is paid; when the disability is partial, only a part of the amount. If the workman is disabled for his special occupation, but can support himself, though not so well, at some other, payment is made in proportion to his lessened earning capacity.

The following table shows approximately the proportionate indemnity values in various injuries:

	Per cent.
Severe head injuries, with concussion of the brain....	50 to 100
Epilepsy	50 " 100
Slight head injuries which cause headaches and dizziness	30 " 35
Loss of one eye.....	25 " 33½
Loss of both eyes.....	100
Loss of one eye when the other eye was already blind..	100
Deafness in one ear with partial deafness in the other (dynamite explosion).....	40
Crushing of chest with fracture of ribs, involving diaphragm and lungs.....	60 " 75
Rupture; one side, 10 per cent; both sides, 15 per cent.	
All ruptures preventing use of the abdominal muscles...	50
Paralysis of the extremities following fracture of the spine. The allowance varies with degree of disability.	
Paralysis of one leg.....	70
Pain in the back, diminishing working capacity.....	20

Such figures as are given in the above tables are based upon actual amounts allowed to injured workmen. They cannot be accepted as absolute standards, but they suggest questions to be considered in the estimation of injury—namely, how far has the accident compromised the individual's working capacity, his pleasure of life (suffering), his chances of material success. Such questions are too general to be answered when considering the prognosis of any single disease, as they ask how much has this individual been affected in his social life by the accident. They are personal and social as well as medical, and many of them the physician can answer in part only.

Is the injured person affected in a way to compromise his particular calling? The permanent paralysis of the hand would practically ruin a surgeon, but it would not affect the wisdom of the rulings of a judge. Was the injury such as to permit of light work, but, if excessive work were attempted, would symptoms ensue of a character to compromise working capacity? The age of the injured person must be considered. It is in general more of a misfortune for a youth than for an old man to be permanently incapacitated. The years in the immediate vicinity of thirty probably represent the highest economic value of American manhood. From an analysis of 147 cases of awards made in suits at law for wrongful death, which awards were confirmed by supreme courts or courts of appeal, Leighton arrives at this conclusion. From his cases he constructed a curve the height of which corresponded with the size of the verdicts. The curve rose sharply after the age of puberty was passed, culminating at thirty. After thirty a gradual decline took place till the age period of fifty-five to sixty was reached, when the declivity became marked, remaining so to the end.

Social conditions must be considered. Disfigurements

(e. g., facial paralysis) would not interfere with working capacity, but they would lessen the chances of a young woman's marrying well. Has the injured person been rendered less capable of caring for himself or have the ordinary dangers of life been increased? The loss of an eye might not seriously interfere with vision, but it would be ruin were the other eye lost later. Such questions come up in regard to all injuries. For, after all, it is the effect of the accident upon the injured man, rather than the disease itself, which is the important consideration. If he is to be permanently incapacitated, it makes little difference to him whether his trouble be functional or organic. And it is upon such general effects that all compensation must be based.

PART I

ORGANIC EFFECTS OF INJURY TO THE NERVOUS SYSTEM

CHAPTER I

ACUTE INJURIES OF THE BRAIN

Causes and Symptomatology—Injuries to the Cranial Nerves—General Diagnosis and Prognosis of Brain Injuries

INJURIES to the brain have to be considered in two classes. First, the acute injuries, which are frankly surgical, with fairly well-known pathology and characteristic symptoms. Second, the complications and sequelæ of head injuries, in which the time interval between cause and effect is lengthened, and consequently the certainty of the injury as a cause becomes more or less open to question. The longer the time interval, and the less the ultimate symptoms conform to clearly cut clinical types, the more is doubt thrown on the injury as a cause. It is concerning the latter conditions that medical and surgical opinion is most apt to vary. As a result, they are frequent subjects of litigation.

CAUSES

The classical cause of injury to the brain is fracture of the skull (Fig. 10). As a result of this, brain injuries are too often considered in relation to the injury of the bone rather than of the brain itself. Too often is the severity of the lesion

measured by the kind of fracture and its site rather than by the evidences of mischief done to the intracranial contents. This is manifestly an error. A man may sustain a fracture of the skull, either at the base or vertex, and escape with slight or with no cerebral symptoms. He is little or none the worse for the accident, as the fracture itself is no menace to life or future capacity. But once the brain itself is injured, no one

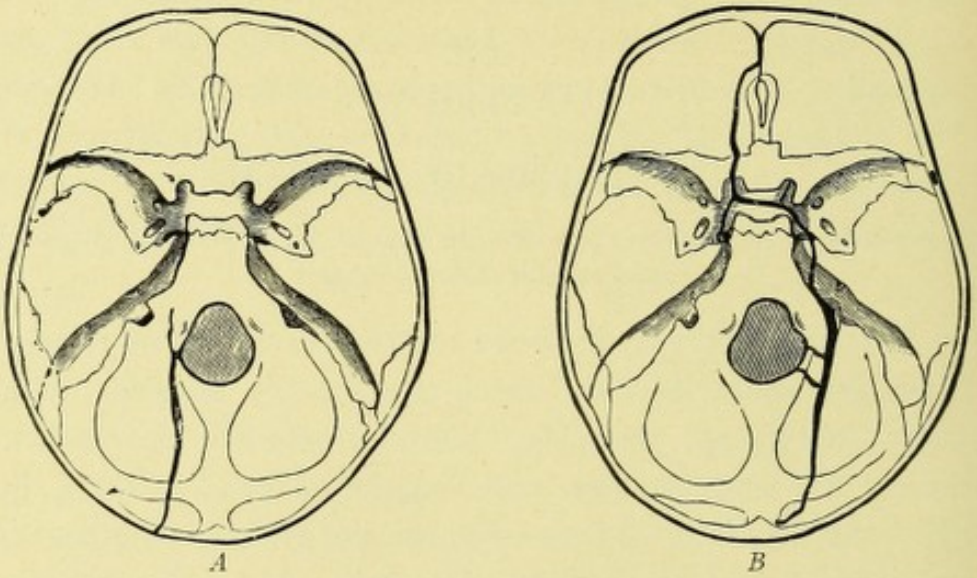


FIG. 10.—Partial (*A*) and complete (*B*) longitudinal fracture of the base of the skull.

can predict positively the outcome. And that the brain may sustain serious and even fatal injury without bone lesion is well known. Consequently, while the majority of injuries to the brain are associated with, or secondary to, skull fractures, the seriousness of the condition is to be measured in terms of brain, not of bone.

Brain injuries are the results of physical violence to the head. The violence may be direct, as from falls or blows upon the head, or they may be transmitted, as by falls on the buttocks. Blows from small, heavy instruments, such as clubs, are apt to cause fracture. In instruments with a larger sur-

face, such as sand bags, fracture is often absent. Blows delivered by various parts of rapidly moving vehicles are frequent causes of fracture. Penetrating wounds, such as stab wounds, either through the bones of the vault or through the orbits, are not uncommon. Bullet wounds are frequent and important causes.

Symptoms

General Symptomatology.—Injuries of the brain are usually associated with some of the purely surgical symptoms of scalp wounds, skull fracture, ecchymoses, escape of cerebrospinal fluid, and the like. In addition, symptoms characteristic of brain injury are headache, vertigo, and vomiting.

After many head injuries, sugar appears in the urine. Of 212 cases of head injuries, collected by Higgins and Ogden, there was glycosuria in twenty. This followed both simple scalp wounds and fractures of the skull. Of twenty-four cases of fracture of the vault, and of twenty-one cases of fracture of the base, five in each class showed glycosuria. The appearance of the sugar took place from eight to twelve hours after the injury. It disappeared in some cases within twenty-four hours, and in others lasted from five to six days. In some cases such glycosuria is accompanied with an increase in the quantity of urine. Polyuria may exist alone, without sugar. In one personally observed case of fracture at the base of the skull it so existed and persisted for several months.

There are other general symptoms of brain injury which will be described under the special forms. But more extended mention is required by mental symptoms, focal symptoms, and injuries to the cranial nerves.

Mental Symptoms.—Mental symptoms are naturally the most prominent evidences of disturbances of the organ which is the seat of the intellectual faculties. In injuries to the spinal

cord, or to the peripheral nerves, consciousness is usually retained; but in all severe cranial injuries some disturbance of consciousness is to be looked for. The term "coma" is applied to the condition in which the patient is completely unconscious, and can be aroused but little, or not at all, by peripheral irritations. In the milder degrees of insensibility, when he can be made to answer questions, it is called "stupor." From the presence of unconsciousness alone, it is usually impossible to determine whether or not there has been a gross structural lesion of the brain. After a blow on the head a man may remain unconscious for several days, and finally recover without any further evidences of cerebral trouble; or the coma following a similar accident may be less profound, although permanent paralysis, mental impairment, or death is to be the result.

Brain injury sometimes, although rarely, occurs without loss of consciousness. Occasionally cases are reported in which the injured persons are rendered unconscious for a brief period and then rally, without presenting further symptoms, for several days or possibly a week or two, when they die suddenly or develop the symptoms of meningitis. But, as a rule, brain injury is synonymous with more or less complete coma, which follows the accident immediately. From the initial unconsciousness a large proportion of the patients who die never emerge. Sometimes the initial coma lessens, the patients regain sufficient consciousness to answer questions, but relapse again into unconsciousness from which there is no further rallying. Profound and persistent coma is an ominous prognostic sign, although I have known it to last a week and still be followed by complete recovery. The most characteristic mental state is semicoma or stupor with delirium. In this the patients lie quietly. They resent being disturbed, and if disturbed become resistant and irritable. There is frequently

a muttering, hallucinatory delirium which, especially in alcoholics, may become sufficiently violent, with shouting, screaming, etc., to require restraint. The faculty of attention is nearly always seriously impaired. The patients may be aroused and made to answer questions intelligently, but the attention soon wanders and they relapse again into stupor or delirium. Comprehension of persons and places is disturbed in varying degrees. Headache may or may not be complained of. Some patients recognize their condition and their surroundings; others think they are anywhere but where they are, mistake the doctors and nurses for their friends, or fail to recognize their friends. Sudden impulses, such as to get out of bed, to rush from the room, etc., are very common. These are often conspicuous in patients who are fairly clear most of the time. They are illustrative of the changeability in symptoms of brain injuries. A patient who at one moment talks rationally and coherently, at the next may be totally irrational and irresponsible. Mental confusion may be the most prominent symptom in injuries which are soon to prove fatal.

A short time ago I had the opportunity of examining at the Harlem Hospital a young man who had been struck by a billiard cue over the right parietal eminence. There was a scalp wound over the place where he had been hit, but no discoverable evidences of fracture. At the time I saw the patient, two days before his death and several days after the accident, he was apparently entirely conscious although very much confused. He understood everything that was said to him, but it was difficult to hold his attention; when told to put out his tongue or perform other acts he obeyed promptly, but he did not seem to know what he was doing, and had to be spoken to two or three times before he executed the movement. There was general increase of the deep reflexes, but there was a total absence of evidences of local injury to the brain. Soon after this he became comatose and died. The autopsy showed a fissured fracture of the right parietal bone and a large subdural hemorrhage on the opposite side of the brain.

When the patient comes to himself it is usually found that the memory for the period of mental symptoms is impaired. Even when consciousness is apparently unaffected, disturbances of memory take place.

Thus, a forty-year-old woman fell on her head. She appeared perfectly normal, but suddenly, when an ice bag was put on her head, she was much astonished, and wanted to know what it meant and why she was in bed. After some thought she remembered she had fallen from the window, but everything else since the accident she had forgotten, and this loss of memory remained permanent.

An alienist received a blow in the face while making his visits in the wards. He continued his visits, made notices in his book, etc., but when he got home it was discovered that he had forgotten the whole thing (Näcke).

In the condition known as retrograde amnesia the memory is lost for a period some time prior to the accident. Thus, in one of my cases, the patient's mind was a blank from the time he bought a railway ticket until he found himself in an ambulance, although fully half an hour had elapsed between his last recollection and the accident. In another, the memory was imperfect, and certain events were entirely obliterated for a period of four days prior to being thrown from a horse.

Abel reports the case of a puncture of the anterior part of the brain in which, immediately after the injury, the patient's life for twenty years before the accident was completely obliterated from his memory. He failed to recognize his wife and friends, and maintained that he had never worked on a railway, though he had been a railway employee for twenty years. A year after the accident great improvement was apparent.

Retrograde amnesia is not a symptom of brain injuries exclusively. It occurs in hysteria and in a variety of non-traumatic cerebral diseases.

The duration of the mental symptoms in those who recover from cerebral injuries is variable in the extreme. Some

patients emerge from the initial coma clear and have no further symptoms. Others continue in the condition of semi-coma for days and even weeks. In others the condition persists still longer, so that long after the physical strength is regained there are serious mental anomalies.

None of the mental symptoms of head injuries is necessarily indicative of permanent organic brain lesion, although it is generally true that the more serious the mental symptoms the more probable becomes the existence of deep-seated cerebral lesions.

Mental symptoms may or may not be associated with focal symptoms.

Focal Symptoms.—The most certain and definite information concerning injuries to the brain is furnished by the symptoms of lesions confined to circumscribed areas (see Figs. 11 and 12). Although no individual region of the brain acts independently of other parts, there are certain districts which have specialized functions, and injury to these causes definite and constant symptoms. The most sharply limited of these districts are the ones which preside over the faculty of speech and the ones concerned in voluntary motion.

Speech Disturbances.—Most traumata which act on the brain in a way to abolish or impair the activity of the centers of speech cause at the same time such pronounced mental defects that examination for the highly specialized cerebral functions is unsatisfactory or unavailing. However, such selective injuries sometimes occur without seriously interfering with general intellectual power, and when the acute general effects of injury have passed away, there may be left symptoms of focal lesions to parts concerned in the faculty of language or other specialized regions.

The best defined of the cerebral centers for speech may be seen in the accompanying Figs. 11 and 12. They include

the second and third left frontal convolutions, injury to which causes motor aphasia or an abolition of the power of voluntary speech without paralysis of the muscles of articulation, or

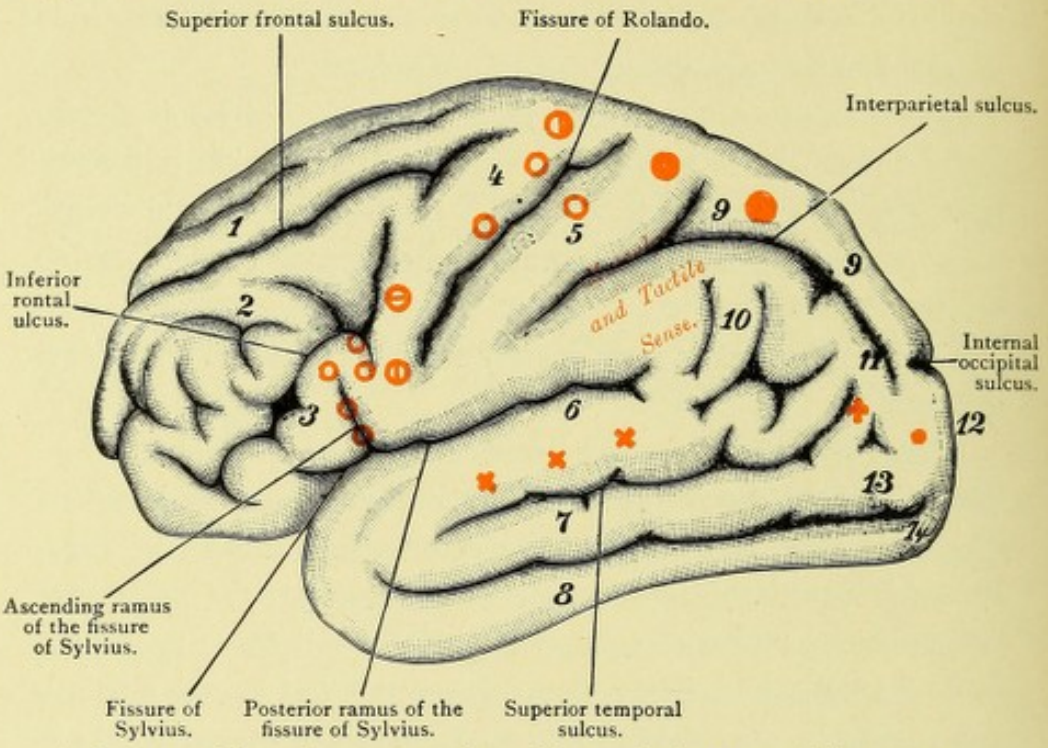


FIG. 11.—Schematic representation of the cerebral cortex and its centers. (After Tillmanns.)

1. First.
2. Second.
3. Third.
} Frontal convolution.

4. Anterior.
5. Posterior.
} Central convolutions.

6. Upper.
7. Middle.
8. Lower.
} Temporal convolutions.

9. Upper.
10. Lower.
} Parietal convolutions.

11. Gyrus angularis.

12. Upper.
13. Middle.
14. Lower.
} Occipital convolutions.

○ In 4 and 5 on both sides of the fissure of Rolando, motor area for the upper extremity.

◑ Motor area partly for the upper and partly for the lower extremity (great toe).

● Motor area for the lower extremity.

ⓗ Cortical area for the hypoglossal nerve.

ⓕ Cortical area for the facial nerve.

○ (3) Motor aphasia.

✕ (6) Sensory (auditory) aphasia with word-deafness.

+ (11) Aphasia with word-blindness.

• (12) Region of the visual area (see also Fig. 11).

Between 5 and 10, cortical center of stereognosis.

without loss of understanding of spoken or written language. Motor aphasia is the most common of the isolated speech dis-

turbances due to traumatic causes. It is almost always associated with right hemiplegia. Injury to the first or second left temporal convolution causes word deafness or the loss of understanding of spoken words, without any disturbance of hearing

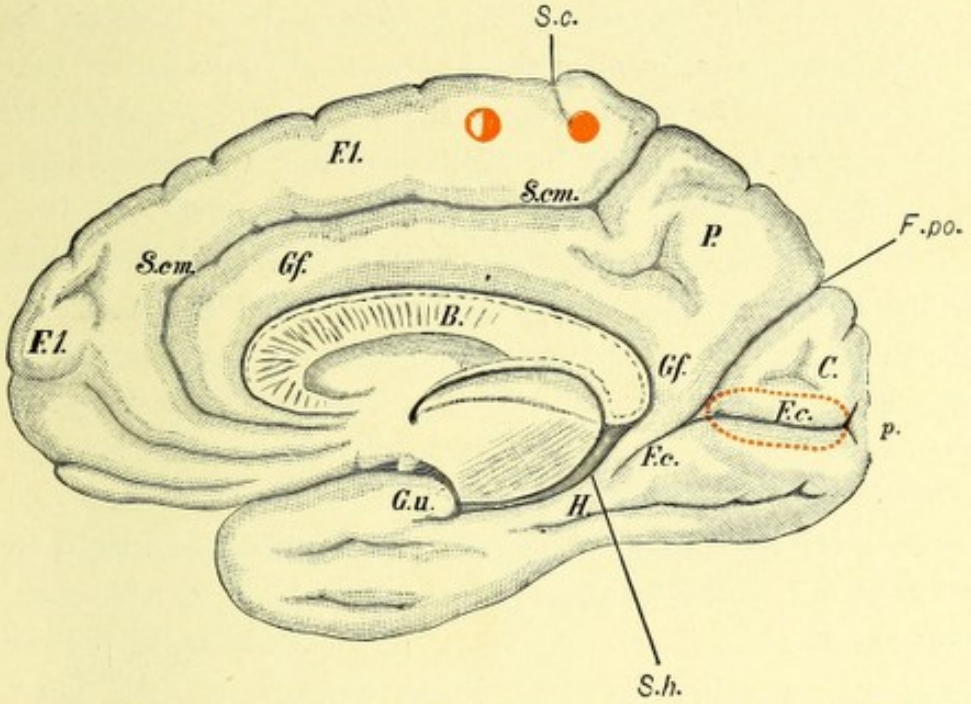


FIG. 12.—View of the right cerebral hemisphere from the median side; *B*, corpus callosum divided longitudinally; *G. f.*, gyrus fornicatus; *H*, gyrus hippocampi; *S. h.*, sulcus hippocampi; *G. u.*, gyrus uncinatus; *S. c. m.*, sulcus calloso-marginalis; *F.*1., first frontal convolution; *S. c.*, termination of the fissure of Rolando; in front the anterior central convolution with the motor area partly for the upper and partly for the lower extremity, and behind the posterior central convolution with the motor area for the lower extremity; *P*, præcuneus; *C*, cuneus; *F. po.*, parieto-occipital sulcus; *p*, polus; *F. c.*, calcarine fissure; in the posterior part of this the visual area is shown by a red dotted line. (After Tillmanns.)

or without serious impairment of the power of speech. Injuries to the occipital lobes sometimes cause a loss of power of the understanding of written signs, and if situated unilaterally, near the calcarine fissure, hemianopsia. Mind blindness and mind deafness are rare conditions, in which, without other mental defect, there is an inability to recognize familiar objects or sounds.

Motor Symptoms.—*Paralysis.*—Loss of power of voluntary movement is the most constant and most valuable sign of focal injury to the brain. It is a very frequent accompaniment of cerebral traumata, and, even when slight in degree, its meaning is so unmistakable that examination for it should be scrupulously made in all cases, although it may seem at first to be absent. The nature of paralysis may be best understood by referring to the way in which voluntary movements are normally performed.

Voluntary movements are presided over by the motor cells, which are situated in the gray matter around the fissure of Rolando. It is in the cells of this region that the motor pathway has its beginning, and the motor pathway (Fig. 13) is the cerebro-spinal tract of which our knowledge is most complete.

It descends through the brain as a well-defined bundle of nerve fibers which terminate in the basal ganglia, the pons, the medulla, and the anterior horns of the spinal cord. Before reaching the spinal cord the larger number of these fibers decussate, those for the cranial nerves, in the pons and in the upper part of the medulla, and those for the spinal cord, in the lower part of the medulla. By this crossing, the cerebral fibers for motion have their termination in the opposite side of the cerebro-spinal axis to that from which they came. The continuation of the motor tract from the gray matter of the brain axis and spinal cord is in the peripheral nerves, which are distributed to the muscles.

The entire tract for voluntary motion, from cortex to periphery, is composed of aggregations of nervous units, which are called motor neurons. A neuron, a term proposed by Waldeyer to include the nerve cell in its entirety, consists of (*a*) the cell body, (*b*) the protoplasmic processes or dendrites, which are generally supposed to convey impulses *to* the cell

and are consequently afferent, and (*c*) the axis cylinder of the cell, or the neuraxon, which is known to convey impulses

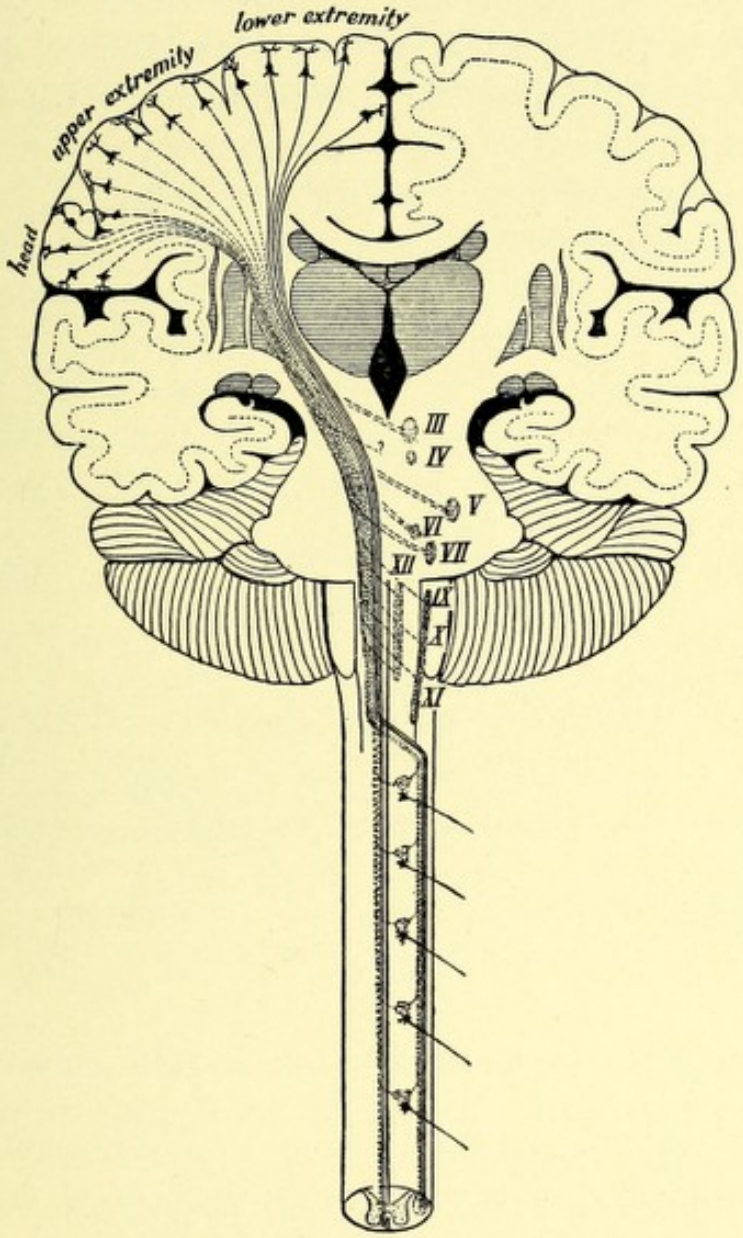


FIG. 13.—Schema illustrating the course of the cerebro-spinal motor path. (After Van Gehuchten.)

from the cell and is consequently efferent. The cerebro-spinal motor pathway is principally made up of two sets of these

neural elements, which are called respectively *central, upper,* or *secondary* neurons, and *peripheral, lower,* or *primary* neurons (Fig. 14). The cell body of the central neuron is situated in the motor cortex of the brain, and its neuraxon descends through the motor pathway, to be connected by fine terminal

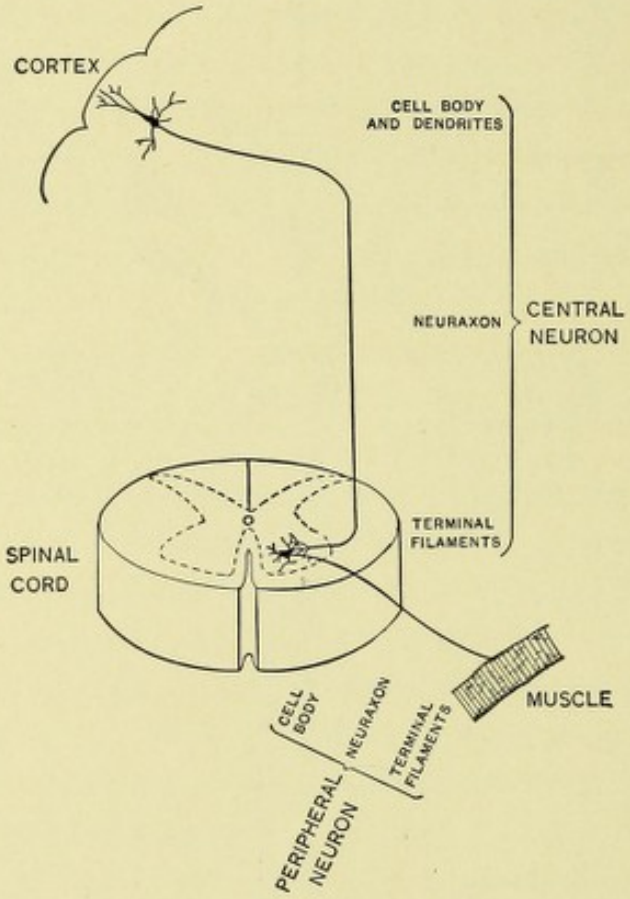


FIG. 14.—Schema to illustrate the arrangement of the motor neurons.

filaments with the peripheral neuron. The peripheral neuron connects the gray matter of the brain axis or of the spinal cord, as the case may be, and the periphery. Its body is in the gray matter, where it is closely associated with the terminations of the central neuron; its neuraxons form the motor fibers of the peripheral nerves. A voluntary movement is the result of some change, the nature of which is unknown, in the

cortical part of the central neuron, by which an impulse is liberated and caused to descend the neuraxon. Its energy is then transferred by means of the terminal filaments to the peripheral neuron, along which it passes to the muscle fiber, which it stimulates and causes to contract. When for any reason the creative power of the cell body of the central neuron is abolished, or the conductivity of its lower part or of the peripheral neuron is lost, the muscles which are controlled by these elements cannot receive the stimulus necessary for their contraction, and are consequently paralyzed.

The clinical manifestations of paralysis vary according as the lesion exercises its inhibitory or destructive action upon the central or the peripheral neuron.

SYMPTOMS OF CENTRAL NEURON INJURY	SYMPTOMS OF PERIPHERAL NEURON INJURY
Paralysis,	Paralysis,
Rigidity,	Flaccidity,
Increase of tendon reflexes,	Loss of all reflexes,
Babinski phenomenon,	
Loss of peripheral reflexes,	
Preservation of normal electrical reactions,	Degenerative electrical reactions,
Atrophy slight or absent,	Atrophy early and decided,
Trophic disturbances not prominent.	Trophic disturbances prominent.

Exception must be made for the distinctive character of these symptoms in so far as that after acute injuries all reflexes may be absent for a time; paralysis may remain flaccid for hours or days; and that at least five days are necessary for the development of degenerative electrical reactions. The pure type of central neuron paralysis is rarely seen except in injuries occurring in or above the basal ganglia of the brain. In these situations none of the neuraxons have reached their lowest destination, and since a peripheral neuron does not begin until

its central neuron ends, there are no peripheral neurons in the vicinity of the lesion, and the paralysis accordingly corresponds to the central neuron type. The pons, medulla, and spinal cord, on the other hand, contain such peripheral neurons as are making their exit from the cerebro-spinal axis (cranial and peripheral nerves), and also those central neurons which are still descending to be connected with the peripheral neurons which are situated at lower levels. Consequently, while injuries of the cerebrum present symptoms referable to lesions of the central neuron only, those at the base of the brain or in the spinal cord usually give evidence of interference with both neurons. Thus an injury to one side of the pons, in a situation below the crossing of the facial nerve, causes a facial palsy on the same side as the lesion (peripheral neuron), while the paralysis of the arm and leg is on the other side of the body, and of central neuron type.

In addition to the types of paralysis, according as one or the other neuron is affected, there are certain means of localization of the injury afforded by associated signs, which are present when the lesion is in some situations and which are absent when it is in others.

Cortical injuries commonly cause paralysis of the arm and leg, less frequently of the arm, leg, and face. When the cortex is injured, convulsions are common, as is seen in traumatic epilepsy or in the twitchings of muscles recently paralyzed; there may also be a loss of the sense of position and some disturbance of cutaneous sensation. Monoplegia rarely results from trauma of the brain; when it occurs, it is due to an elective injury to the cortex.

In the *tracts* below the cortex smaller lesions cause more extensive paralysis, as in those situations the motor fibers are more closely packed together and the pathway is smaller.

Lesions of the *internal capsule* cause hemiplegia of the

opposite side; when situated posteriorly there may also be diminution of cutaneous sensibility and hemianopsia. Lesions at the *base* of the brain, by pressing upon or destroying the neuraxons of those central neurons which preside over voluntary motion in the limbs, may cause paralysis of them of various distributions. But in addition to the palsies of the limbs, central neuron type, there are usually palsies of the cranial nerves, peripheral neuron type.

The pons, as well as the cortex, contains a convulsive center, and consequently lesions at the base of the brain, which irritate the pontine region, are followed by muscular twitchings or by general convulsions. Pontine lesions also usually cause anæsthesia.

In brain injuries, paralysis affects the extremities and the cranial nerves. The paralysis of the extremities exists much more frequently as weakness, or increased rigidity, than as profound loss of power. It usually takes the form of hemiplegia, though monoplegia, or even polyplegia, are, sometimes seen. Polyplegia seems regularly fatal. With hemiplegia, on the other hand, death is by no means inevitable. If the patients recover, the hemiplegia rapidly recedes; and, as a rule, recovery of power and balance is much more complete than is the recovery from hemiplegia of apoplectic origin.

Sensory Symptoms.—Pronounced anæsthesia is rarely if ever observed as a focal symptom of traumata of the brain.

After severe injuries, when the coma is profound, there is often a generalized diminution or a total loss of cutaneous sensibility, as is shown by the patient's indifference to the application of painful stimuli. But this is to be interpreted as a disturbance of the receptive centers, due to abolition of mental function, rather than as an indication of any interruption of conducting paths. This assumption receives additional proof from the fact that sensibility almost invariably returns

with a restoration to consciousness. If there is a well-marked hemiplegia, complaints of subjective sensations of pricking or numbness in the paralyzed side may be made, and by examination with the needle or with cotton it may become evident that there is some blunting of sensibility on the paralyzed side. But, although the patient does not feel so well on that side as on the other, he can still distinguish between hot and cold, can tell when he is touched, and give some expressions of pain upon the application of painful stimuli—evidences of hypæsthesia rather than of anæsthesia. Hemihypæsthesia, associated with disturbances of the sense of position, is a not infrequent result of injuries of the motor cortex.

Lesions which result from external violence and which affect the cerebral sensory tracts probably never cause complete hemianæsthesia, with involvement of the head, trunk, and extremities, such as is frequently seen in hysteria. In cortical injuries the resulting anæsthesia is never total and is rarely pronounced in degree. Pressure upon the posterior third of the hinder limb of the internal capsule may bring about complete loss of sensibility to all stimuli on the opposite side of the body; or pressure upon the pons in the neighborhood of the fifth nerve may cause " crossed hemianæsthesia "—i. e., loss of sensibility in the distribution of the fifth nerve on the side of the lesion and anæsthesia of the arms, trunk, and legs of the opposite side. Cases presenting these symptoms have been reported, but, as far as I know, they have been the results of tumors or of non-traumatic vascular disturbances exclusively. It cannot be denied, however, that similar symptoms might result from laceration of the brain, or from hemorrhages into it which were caused by quickly acting external violence, but if such a possibility exists, evidence in its favor has escaped my notice; and, indeed, it seems hardly probable that any trauma could act in a way to produce so small and so

elective a lesion as would be necessary to cause the pure type of organic hemianæsthesia, without at the same time so seriously injuring other parts of the brain that the unilateral character of the sensory loss would not be maintained, or that the sensory symptoms would become masked by more serious disturbances.

As will be mentioned in succeeding pages, hemianæsthesia—profound, total, and sharply limited by the median line of the body—is not uncommonly observed in the victims of railway and allied disasters. To it Charcot ascribed a pathognomonic value in the diagnosis of hysteria, a view which was opposed in Germany. Without entering here into further discussion of the question, it may be safely asserted that when pronounced hemianæsthesia appears as one of the clinical results of an accident it is in all probability hysterical, and that if this symptom is ever the result of an organic injury to the brain, the inevitable association of other symptoms of cerebral lesion will leave no doubt as to its nature and origin. An important sensory symptom is astereognosis. The recognition of objects by the sense of touch is called stereognosis. This faculty has its cortical seat in the cells of the middle third of the posterior central convolution and in the adjacent part of the inferior parietal lobule. Injury to this region, or to the neurons which descend from it, with the other sensory neurons, in the internal capsule, causes astereognosis. As an isolated symptom in traumatic cases it is rare. Donath has recorded a case in which it, together with hemiplegia, resulted from a stab wound which entered the brain above the right ear.

Reflexes.—As a rule, after injuries to the head which have been severe enough to cause profound coma, both the skin and tendon reflexes are temporarily lost. The general absence of reflexes indicates that the brain has been seriously injured, but gives no indication as to the seat or the character of the lesion.

When the initial coma is less profound, or when it is beginning to pass away, the condition of the reflexes is capable of furnishing more definite information.

All the reflexes on the paralyzed side may be absent, while those of the unaffected side remain about normal, or—and especially if the examination is made a few days after the accident—there may be a unilateral increase of tendon reflex activity on the side of the paralysis, while the abdominal reflexes of that side remain unelicitable. The unilateral absence of abdominal reflexes, with increase in the knee or wrist jerk, is a valuable diagnostic sign of hemiplegia, and may be present when the loss of motor power is very slight.

Increase in activity of the tendon reflexes is apt to be a permanent condition after brain injuries. The Babinski reflex is often present very early, though, unless the spasticity remains pronounced, it passes away.

Pupils.—Abnormality of the pupils is a very common symptom of brain injuries. Usually it exists as inequality, the larger pupil being on the side of the injury. The pupils may be evenly contracted, though they usually dilate before death. There may be alternating dilatation and contraction or the pupils may be immobile.

Injuries to Cranial Nerves

Injuries to the cranial nerves do not necessarily indicate brain injury. These nerves may be compressed and consequently paralyzed by fracture through the foramina which give them exit from the skull. With few exceptions, however, injuries sufficiently severe to fracture the skull are accompanied by brain lesions with which the peripheral palsies may or may not be associated. The causes of traumatic intracranial paralysis of the peripheral nerves are fractures at the

base, hemorrhages at the base, bullets, and penetrating wounds. The nerves may be lacerated, compressed, or divided, and usually more than one nerve is affected. The symptoms ensue immediately or, occasionally, not until some time after the accident. To explain these latter cases is often difficult—only rarely are they due to septic meningitis.

Olfactory Nerves.—Injury to these nerves is rare. It occurs as the result of penetrating wounds of the frontal and nasal region, of hemorrhages in the anterior fossa of the skull, and by contrecoup from injuries to the posterior pole of the skull. The symptoms are loss of smell, usually associated with impairment of taste.

Optic Nerve.—Injury to these nerves is more frequent, probably, than clinical records indicate. Slight symptoms referable to it readily pass unnoticed in comatose patients, or in those not submitted to special examination. The nerve is almost always affected in front of the chiasm, so that hemianopic symptoms are rare. Optic nerve disturbances after fracture at the base are most frequently due to involvement in the fracture of the thin walls of the optic canal. Displaced portions of bone may crush and destroy parts of the nerve, causing permanent blindness in the corresponding portions of the field. It is probable, also, that amblyopia may be caused by hemorrhages at the base, which push their way into the optic canal without this latter being fractured.

Hemorrhages into the sheath itself only occur when associated with fracture of the canal. According to Leber, if the lesion is behind the entrance into the nerve of the arteria centralis retinæ, atrophy never appears. If in front of this arterial supply, the disk begins to show atrophy at the end of a few weeks.

The type of contraction of the field is not the concentric as in ordinary atrophies, but rather a loss of the upper, lower,

or particularly the lateral halves of the field. The visual disturbances are for the most part one-sided, corresponding to the side of the injury, which is usually the frontal, temporal, or parietal regions.

It is rather difficult to estimate the frequency with which substantial recovery from the initial amblyopia or blindness occurs. The prognosis depends upon the nature of the injury. Lacerations by bone splinters or foreign bodies are irremediable. Hemorrhages into the nerve itself probably are also. When atrophy appears, there will be no restoration in the atrophied portions.

But initial blindness is sometimes recovered from. I am inclined to think that ophthalmologists, who most frequently see the cases which have gone on to atrophy and permanent blindness, regard the prognosis in every case as more serious than do surgeons, who see the patients immediately after the injury, and may therefore witness defects in sight which are transient. Knotz reports a series of ten clinical cases giving symptoms of fracture at the base and visual disturbance. In half of his cases vision was affected in both eyes, though the eye corresponding to the side of the external injury showed more serious and more permanent symptoms. Of these ten cases, in eight there was amblyopia on the side of the injury, but in only one was the amblyopia permanent. On the side opposite to the injury amblyopia occurred five times, but was always transitory. In one case there was immediate and permanent blindness on one side. In another there was immediate blindness in both eyes, but vision was perfectly restored in the eye opposite the injury, and on the same side there was left a contraction of the visual field. Of Van Nes's three cases of unilateral opticus injury, immediate one-sided blindness resulted in all. In one, death resulted; in another, complete restoration in six weeks; in the third, optic-nerve atrophy.

Traumatic affections of the optic nerve are frequently accompanied by palsies of the motor nerves of the eye and by deafness.

Nerves to the Ocular Muscles.—After the facial, paralyses of the motor nerves of the eye are the commonest traumatic cranial nerve palsies. If variations in the pupils are considered as belonging to palsies of the motor nerves, then these latter are the most frequent focal signs of all. Irrespective of the condition of the pupils, the order of frequency of involvement of the motor nerves of the eye is the third, sixth, and fourth.

FIG. 15.—Double third-nerve palsy, showing outward and downward deviation of both eyes and wrinkling of the forehead.

The Third Nerve.—Palsy of this, either total or in some of its fibers only, is one of the most frequent evidences of injuries in the vicinity of the orbit and at the base of the brain. From the controlling influences which this nerve exercises upon the eyelid, upon most of the extrinsic muscles of the eyeball, and upon the size of the pupil an abolition of its function causes striking symptoms. When the nerve is the seat of a complete lesion there are ptosis, outward and downward rotation of the eye from the overaction of the external rectus and the superior oblique, and dilatation of the pupil (Fig. 15). Complete third-nerve palsy, however, is comparatively rare in acute conditions. More frequently the nerve shows a partial and elective palsy. Thus there may be a drooping of the upper lid, so that the affected eye cannot be opened as wide as the normal eye; or there may be only a slight dilatation of the pupil on the injured side, so that there exists an inequality in the size of the pupils, the larger being on the side of the injury; or there

may be a weakness in the extrinsic muscles supplied by the third nerve, with paralysis of accommodation.

The Fourth Nerve.—Injury to the fourth has been very rarely recorded. It sometimes occurs in conjunction with other cranial nerve palsies, as in a case of Knotz, in which there was total ophthalmoplegia, interna and externa.

The Fifth Nerve.—The trigeminus usually escapes intracranial lesion, and is never injured alone in that situation. Paralysis of the masseters, pterygoids, and temporals, and anæsthesia of the face are the main symptoms of fifth-nerve paralysis. Taste fibers, coming from the anterior two-thirds of the tongue, and leaving the tongue by means of the chorda tympani pass, in some cases, through the second or third branch of the trigeminus. Disturbances of taste, therefore, should be looked for in the trigeminus injuries. Neuralgic pains, dryness of the eyelids, herpes, and neuroparalytic ophthalmia may also result from intracranial injury of the fifth.

The Sixth Nerve.—The sixth is most frequently injured together with the seventh, both commonly being associated with deafness. The sixth is sometimes, also, injured alone, usually on one side, although bilateral palsies of it are by no means rare. Palsies of any of these nerves cause diplopia.

The prognosis of the palsies of the motor nerves of the eye is usually good. It is rare for them to persist for more than a few weeks or months as serious disabilities. Usually, in that time, double vision passes away, if it existed, and the eyelid, if there was ptosis, raises itself sufficiently for effective use. In most cases of traumatic ptosis, however, weakness sufficient for identification remains in the eyelid, although the patient may be unconscious of it.

The Facial Nerve.—Intracranial facial paralysis is the commonest single cranial-nerve palsy in fracture at the base.

With Heer it occurred in seventeen per cent of the cases, with Van Nes in twelve per cent, with me in ten per cent. The whole nerve is involved, but the lower branches suffer most; the paralysis is also more apt to be permanent in the lower branches. Sometimes the facial palsy remains as a permanent disfigurement. More frequently, however, it, like the other cranial-nerve palsies, is temporary, although it, like the others, usually leaves some trace by which it may be identified by the practiced eye. The nerve may be affected at any point in its passage through the petrous portion of the temporal bone. Most frequently, however, it is injured below the branching of the superficial petrosal nerve. When injured at the geniculate ganglion there is paralysis of the soft palate, which occurred in one of my cases. Disturbances of taste are rarely mentioned. When present they are transitory. The nerve is usually injured on one side only. Bilateral facial palsy, from fracture at the base has, however, been observed. Rarely the facial is injured within the skull by penetrating wounds.

Eighth Nerve.—Injuries to the ear itself are so frequent in fractures at the base that the diagnosis of an acusticus lesion is extremely difficult. Deafness is particularly frequent with palsies of the optic, of the sixth and seventh together, and of the seventh alone. It can only be positively identified as of nerve origin when there is nerve deafness.

Frequently several cranial nerves are paralyzed together. A characteristic clinical type of such multiple nerve lesions, sometimes seen after fractures at the base, is illustrated by the following case (Figs. 16, 17).

The patient came to the Vanderbilt Clinic complaining of deafness, double vision, and the drawing of the face to one side. He told us that twelve weeks previously he fell twelve feet, striking on the buttocks; he lost consciousness for some time, and on coming to himself

observed the symptoms above mentioned, together with bleeding from the left ear. Our examination showed: partial paralysis of left external rectus (sixth nerve), peripheral paralysis of left side of face (seventh nerve), deafness in left ear with loss of bone conduction (eighth nerve). The appearance of the patient was highly characteristic: the left side of the face was flattened, a condition which was caused by a contraction of the muscles of the right side. When the patient looked straight in front of him there was a left internal

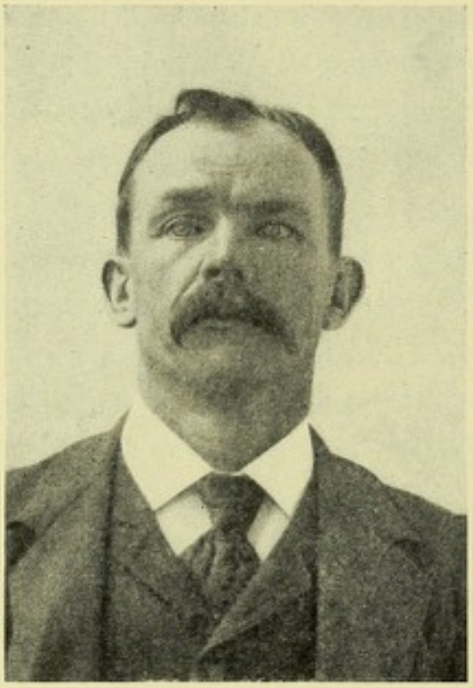


FIG. 16.—Photograph showing paralysis of the left sixth and seventh nerves, due to a fracture of the skull. Patient trying to look straight ahead.

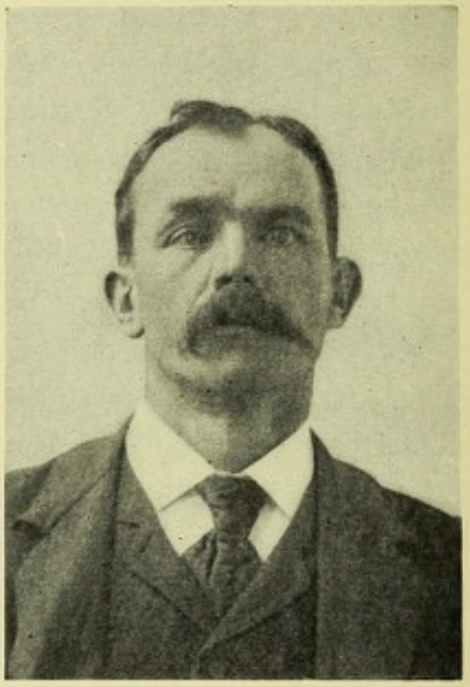


FIG. 17.—Same case. Patient trying to look toward the left.

squint caused by overaction of the internal rectus; when he tried to look toward the left, the right eye executed the movement normally, but the left looked straight ahead from inability of the left external rectus to rotate the eyeball outward.

Complete injuries of the glossopharyngeal and vagus within the skull are not consistent with life. It is within the realm of possibility, however, for certain branches or connec-

tions of these nerves to be affected in injuries at the base in a way to give focal symptoms.

In injuries at the base, the throat should be examined with a view to determining paralysis of the palate (Jacobson's nerve), changes in salivary secretion, loss of sensibility in the throat, and loss of taste (glossopharyngeal). Intracranial lesions of the pneumogastric are excessively rare. Stierlin has reported a case of fracture at the foramen magnum in which, together with paralysis of the right side of the tongue (hypoglossal) there existed a pronounced weakness of the right vocal cord (pneumogastric). There was also a paralysis of the right side of the soft palate, although the glossopharyngeal was not injured.

General Diagnosis

In two classes of cases, only, is diagnosis of injury to the brain difficult. First, when the symptoms immediately following the injury are slight and the unconsciousness transient. Then the severity of the condition is often overlooked. This frequently happens, and it is not unheard of for persons with severe brain injuries to walk to hospitals or to be arrested for disorderly conduct.

In a case at Roosevelt Hospital, a man fell off a cable car, was rendered unconscious, and was brought to the hospital. There were no evidences discoverable of fracture of the skull or of local brain injury, and the next day, as the patient was feeling perfectly well, he was discharged from the hospital. He continued to feel well for two weeks, when he suddenly died. An autopsy by the coroner showed a fracture running transversely for the whole width of the middle fossa of the skull.

In the majority of cases with late accession of prominent symptoms the cause is meningeal hemorrhage. In order that such cases be not passed unrecognized, it is imperative that

every injury to the head, of any severity, should be at once carefully examined for all the signs of interference with brain function, and should be kept under close and skillful observation for at least thirty-six hours after the accident. This observation should relate especially to the mental condition, to twitchings, to the access of rigidity in the limbs, and to any increase in the blood pressure.

The second point of difficulty in the diagnosis of brain injury has to do with the cases in which persons are found unconscious. There is no history.

In the summer of 1896 one of the female inmates of the New York Workhouse, aged sixty, was found on the floor of her cell unconscious, presumably having fallen from her bunk to the stone floor, a distance of about four feet. There was a deep scalp wound over the left parietal eminence. She was transferred to the hospital, and when I saw her she was in partial coma, with a right hemiplegia unassociated with aphasia. The question was, Did the patient fall and receive a scalp wound as the result of an apoplectic attack, or was her condition due to a fracture of the skull, with resulting injury, or compression of the brain? There were no external evidences of fracture. There were no palsies of the cranial nerves, except that the right pupil was larger than the left. The hemiplegia, which was complete, involved the arm, leg, and face; there were no convulsions, and only slight disturbances of sensation. All these symptoms indicated with probability, but not with certainty, a primary cerebral hemorrhage rather than a laceration or traumatic compression of the brain. The autopsy, held ten days after the accident, confirmed this opinion. There was a large hemorrhage in the left internal capsule, but no injury to the other parts of the brain, and no fracture of the skull.

In such cases diagnosis is often difficult. Certain surgical symptoms, such as tangible fractures of the vault, escape of blood or of cerebro-spinal fluid from the ear, are naturally conclusive evidence of fracture, and consequently of brain injury. Subconjunctival ecchymosis, subcutaneous ecchymoses—especially behind the ears—and blood-stained cerebro-spinal fluid

(lumbar puncture) are also strong evidences of fracture. Scalp wounds are suggestive, but not conclusive, of traumatic origin.

In the absence of surgical signs, the question becomes that of the differential diagnosis of coma. The forms of coma which may be mistaken for brain injury are those due to alcoholism, to Bright's disease, to diabetes, to cerebro-spinal meningitis, hysteria, and apoplexy. In the coma of the general medical diseases underlying symptoms are obtainable. In alcoholism the pulse is rapid and full and the odor of alcohol unmistakable. In so many cases of fracture at the base alcoholism is present, that one should be extremely chary of excluding brain injury simply because alcoholism is present. In hysteria the coma is profound, without disturbance of pulse or respiration. In apoplexy the paralysis is usually extensive and pronounced, and there are present the evidences of arterial diseases. Except in simple concussion, the focal signs are the chief neurological means of distinguishing brain injuries from the toxic comas and from apoplexy. Focal signs play no part in the toxic comas. Some are usually present in brain injuries. In apoplexy they are also present, but more pronounced than in brain injury. In this latter we find a slight drooping of an eyelid rather than a pronounced ptosis—a slight inequality of the innervation of the two sides of the face rather than a complete drawing of the face to one side. In the limbs, slight rigidity or slight weakness on one side is more common than the outspoken hemiplegia of apoplexy. The same is true for the reflexes. Pathological changes exist, but usually slight in degree.

General Prognosis

Only the most general statements can be made in regard to the prognosis of injuries which vary so greatly in degree, character, and cause. It is manifestly impossible to formulate a prognosis at once applicable to momentary losses of consciousness from slight blows, and to conditions resulting from extensive intracranial lacerations and hemorrhages.

In a study undertaken by me some years ago, an attempt was made to formulate some facts as to the fatality of brain injuries of a certain uniformity of type. Fracture at the base was chosen as the index, since it represents an extreme degree of violence and probably a similarity in the brain lesions. That this latter is true is shown by the great similarity in symptomatology, and also in conclusions as to mortality by different observers. Heer in 58 cases had 29 deaths, or a mortality of fifty per cent; Van Nes in 82 cases had 39 deaths, or a mortality of forty-seven per cent; Phelps, in 286 cases had 176 deaths, or a mortality of sixty-one per cent; Dr. R. F. Weir and Dr. W. T. Bull, having kindly given me access to their histories at the Roosevelt Hospital, I found that 60 cases were received there during the years 1900 and 1901. By adding to these 9 cases of which I have private records, the resulting total is 68 cases. Of these 40 died, a mortality of fifty-eight per cent.

Thus a fatal result is to be looked for in more than half of the cases of fracture at the base. The combined tables of Heer, Van Nes, Phelps, and myself, comprising a total of 494 cases, show a mortality of fifty-seven per cent. In this connection it must be stated that some hospital records show a much higher mortality. In the great majority of cases death is due directly to the injury, without intercurrent conditions. Of the 40 fatal cases of my series, 13 died on the day

of injury and 12 died on the following day. Thus, sixty-two per cent of all deaths occurred within one day. Of Heer's 27 deaths, 13 occurred within twenty-four hours of the injury. Of my other deaths, 6 occurred within two days and 7 in between three and five days. According to these figures, ninety-five per cent of the patients who die from fracture at the base do so within five days of the receipt of the injury. Those dying after longer periods usually have pneumonia or meningitis, although this latter condition is infrequent in New York. Age does not seem to be an important feature in prognosis. In my series, the average age of those who recovered (4 patients being under ten years) was thirty years, and of those who died (5 patients being under ten years) twenty-seven years. The immediate prognosis is doubtless influenced by the patient's general condition of health, especially as regards conditions predisposing to pneumonia. I have no statistics on this point further than that two patients, known to have had nephritis before the accident, made good surgical recoveries. It seems probable, however, that a healthy man has a better chance of withstanding a brain injury than does an infirm man. Alcoholism affects both immediate and ultimate prognosis profoundly. Alcoholic patients are more apt to die, because they are more prone to pneumonia, because their heart action is weak, and because in them delirium is more violent, thus preventing them from resting quietly. The patients who do not die, recover with reasonable promptness. It is not unusual for a patient with fracture at the base with simple concussion to be able to be up in five days. As a rule, however, patients are in bed ten days; sometimes, of course, for many weeks. English found the average time for returning to work, after simple concussions, four and one-half weeks; after cerebral contusion, nine and one-half weeks; after laceration, thirteen weeks. A man may return to work, however,

and not feel himself; but usually after a severe injury it is twelve months, or even two years, before the patient feels as well as before the accident.

The foregoing statistics represent in a general way the long-run outcome of such brain injuries as are caused by violence which fractures the skull at its base. Injuries which cause fractures of the vault have a much greater variation in severity and character, and generalizations are hardly possible. In fracture at the vault the brain is more apt to be lacerated by splinters of bone, there is much greater tendency to secondary hemorrhage and to infection. At the same time, these injuries may result from slighter degrees of violence than fractures at the base, and involve very much less general contusion of the brain, so that recovery is more frequent in fractures of the vertex than in those at the base. Oftentimes fractures of the vault are associated with fractures at the base.

Statistics of Acute Head Injuries.—Hudson Street Hospital, 1896–1904

HEAD INJURIES.	Cured.	Improved.	Transferred.	Died.	Total.
I.—*No Injury to Skull:*					
Concussion of brain	3	152	26	2	183
Traumatic meningitis	...	....	....	1	1
Laceration of brain	....	....	....	2	2
II.—*Fracture of Skull:*					
Unclassified	1	6	1	11	19
Acute meningitis	....	....	....	1	1
Hemorrhage of brain	....	....	....	1	1
Circumscribed, simple, no brain injury	4	9	....	....	13
Circumscribed, simple, brain injury	....	....	....	4	4
Compound Fracture of Skull: (*including bullet wounds*)					
Unclassified	....	1	....	2	3
Circumscribed	22	77	9	....	108
With laceration of brain	3	3	1	45	52
Fracture at Base:	3	54	31	166	254

A general idea as to the immediate prognosis of acute head injuries is given by the cases on page 88 from the Hudson Street Hospital for the years 1896 to 1904 inclusive.[1]

Certain symptoms are always of grave import in all head injuries. Profound and persistent coma, active delirium, high fever, rapid pulse and respiration usually foreshadow a fatal outcome. Polyplegia is regularly fatal. With hemiplegia, on the other hand (in fractures at the base at least), death is by no means inevitable, as recovery occurs in a good number of the cases. As the patients get better, the hemiplegia rapidly recedes; and, as a rule, recovery of power and balance is much more complete than is the recovery from hemiplegia of apoplectic origin. Certain conditions of the pupils seem of particularly grave significance. I have noted high mortality in cases with persistently and equally contracted pupils, with immobile pupils, and with pupils presenting alternating contraction and dilatation.

[1] Those statistics were kindly furnished by Dr. P. R. Bolton.

CHAPTER II

ACUTE INJURIES OF THE BRAIN—*Continued*

Clinical Types: Concussion, Contusion, Laceration, Compression, Wounds.

Concussion of the Brain

Concussion of the brain is a term applied to the abrogation of various cerebral functions brought on by a sudden change in position of the brain. The causes are any sudden forces acting upon the skull. The force may be applied directly to the skull or be transmitted, as occurs in falls on the buttocks.

Pathology.—The pathology of the condition is not definitely known, though it is commonly supposed to be connected with changes in position of the cerebro-spinal fluid with resulting injury to the cerebral substance. Anatomical lesions sufficient to explain concussion are yet to be demonstrated. Cell changes and microscopic hemorrhages are found in some, but not in all cases. But they are not sufficient to explain the profound disturbances in the functions of the cortex and of the medulla which may be the symptoms of concussion. We can nevertheless safely assume that minute and widespread lesions are at the bottom of the condition. Concussion of the brain is a nearly constant accompaniment of all brain injuries; and nearly all brain injuries, therefore, in which, in addition to concussion, gross lesions such as hemorrhages, contusions, lacerations, etc., are present, have a mixed symptomatology.

The greater the physical violence, the greater the concussion effects, and the greater, also, the probability of the coex-

istence of severe contusions or lacerations. It is probable that even in contusions and lacerations of the brain, concussion, quite as much as the visible injuries to the brain structure, is accountable for the serious symptoms.

The primary effect of concussion is upon consciousness. It may disturb it profoundly, but it does not affect the specialized areas of motion, speech, sight, etc. Consequently, when these areas give evidence of injury some condition other than concussion is present.

Clinical Forms.—Concussion occurs in varying degrees. In the mildest form, after a blow, there is a dazing or confusion which is only momentary. Or the patient may be several minutes completely unconscious, and then slowly regain consciousness. The face is pale, the muscles are relaxed, the pulse is of low tension, and may be rapid and irregular; the respiration is shallow. On coming to himself the patient has peculiar sensations in the head, complains of headache and dizziness, and feels weak. He may have forgotten not only the accident itself, but events which took place some little time before. If the concussion were not complete, and if, during it, he were made to answer questions, he may retain no recollection of it afterward. In other cases the symptoms are practically identical with those of physical shock. Consciousness is not completely lost, the patient may be aroused, but he is irritable and apathetic. The face is pale, the skin moist and cold, the pulse soft, rapid, and feeble, the respirations shallow, Recovery shows itself by warmth returning to the body surface, strengthening of pulse and respiration, and clearing of the mental state. This change may be ushered in by nausea and vomiting.

In the more severe degrees concussion is a much more serious matter. The patients lie for hours or even days in coma from which they can be only partially aroused, if at all.

The symptoms already mentioned are intensified. The pulse may be slowed and loss of sphincter control is frequent. These cases are always difficult to differentiate from those in which gross lesions coexist with the concussion. The longer the symptoms persist, the greater the probability that concussion is not the only factor at work.

Treatment.—The treatment of concussion sums itself up in heat to the body by hot bottles, etc., lowering of the head, and stimulation.

Contusions and Lacerations of the Brain

Contusions of the brain consist in bruisings, by which nerve tissue, connective tissue, and blood vessels are injured. As a result there ensues softening, with, eventually, the formation of a scar. With an external wound sepsis, in the form of meningitis and abscess, is a serious danger. Failing an external wound, these injuries rarely become infected, though the possibility of infection, even under such circumstances, exists, and should be held in mind. In lacerations the brain substance is actually torn, so that there is a loss of continuity. These injuries are usually the results either of fractures of the skull, of bullet wounds, or of penetrating wounds. Independently, however, of the destructive action of injury by broken bone or by entering foreign bodies, the brain may be both bruised and torn by the force of the original blow. This is shown by the cases in which no bone injury exists, and by the cases of extensive laceration and contusion (especially in the temporo-sphenoidal lobes) in parts some distance from the seat of fracture.

Symptoms.—Such injuries are invariably associated with the symptoms of concussion; to them are almost always added some focal symptoms. In text-books on surgery it is stated

that localizing signs are frequently absent. In my experience some localizing signs, other than those directly connected with consciousness, are generally present. Often they are very slight, but even a slight rigidity of a limb, or a difference in tendon reflex, are sufficient for a diagnosis of a lesion more serious than concussion. The symptoms of compression frequently complicate or supervene upon those of laceration or contusion. In lacerations and severe contusions the coma is usually profound, and the temperature is often high (102° F.) from the outset.

Treatment.—The surgeon should always endeavor to determine whether the cerebral symptoms point to lesions in deep and inaccessible portions of the brain or whether they are cortical. Deep-lying contusions and lacerations, unassociated with compression, cannot be helped by operation. When fractures of the vault, with external wounds, are present, the greatest care as to cleanliness and free drainage is imperative. Depressed bone should be elevated and free splinters of bone removed. The dura, if torn, may be sutured. Failing scalp wounds, depressed bone should be elevated if focal symptoms can be traced to it.

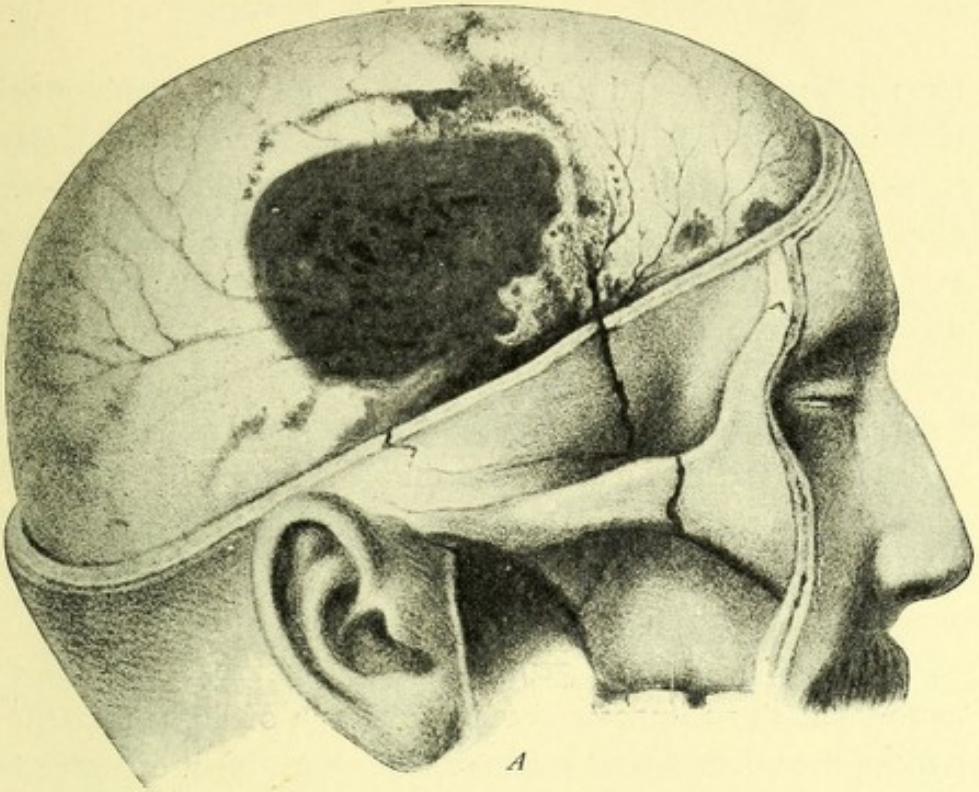
As to whether simple depressions of the bone, without external wound and without symptoms of local brain injury, merit operation, opinions differ. In my opinion, such depression when over the motor area, unless very slight, should always be elevated.

Compression of the Brain

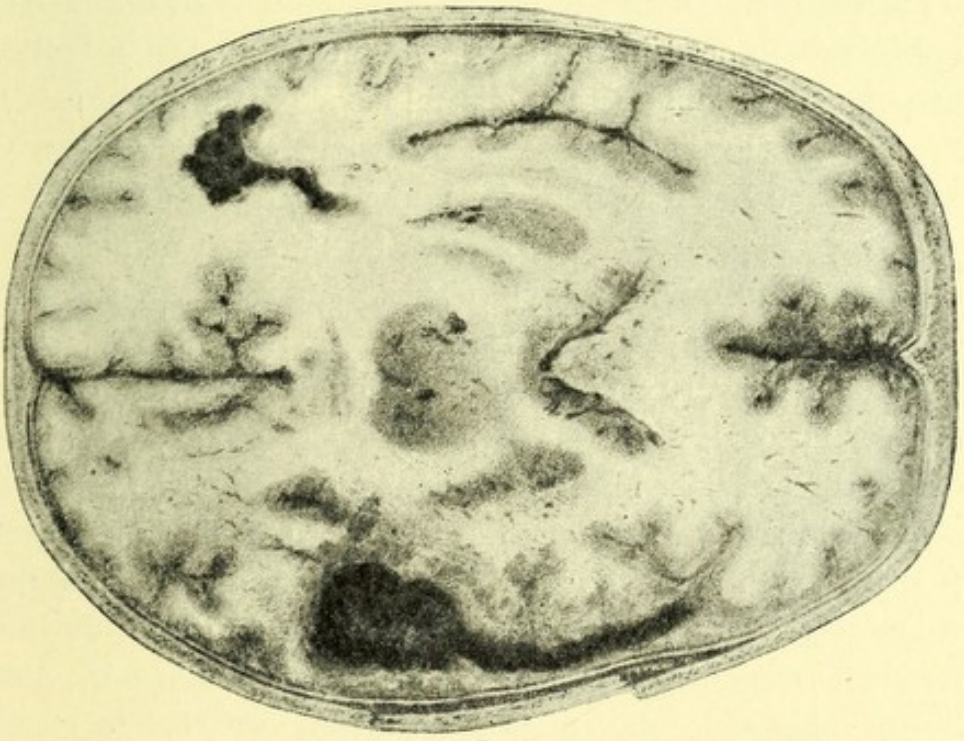
The skull, the case of the brain, is fixed and unyielding, entirely incapable of being acted upon by any force from within in a manner to increase its cubic contents. The brain substance itself is, in all probability, incompressible. Conse-

quently, when the cranial cavity is encroached upon, either by an increase in the amount of the cerebro-spinal fluid, or by foreign bodies such as blood clots, the increased pressure is exerted solely upon the arteries, veins, sinuses, capillaries, and lymph channels of the brain. The circulation in these is consequently impeded, and venous stasis and anæmia result. It is from defects in the cerebral circulation, therefore, that the symptoms of increased brain pressure arise. This increase in brain pressure can be measured in chronic cases, such as hydrocephalus with patent foramen of Majendie, by a manometer attached to a cannula inserted in the subdural space by means of lumbar puncture. In acute hydrocephalus, or acute compression, also, when not of high grade, the increase can be determined in the same way. But when the increase in pressure is acute and extreme such measurements are no longer possible. This is so because the brain is forced downward by the increase in the cranial contents until the medulla engages in the foramen magnum. There it acts as a ball valve, preventing the communication of the cerebral subdural space with that of the spine, and, by closing the foramen of Majendie, preventing communication of the subdural spaces with those of the cerebral ventricles.

For practical clinical purposes, therefore, the diagnosis of increased brain pressure is made by other means than those of direct measurements. As has been said, when there is an increase of intracranial pressure—i. e., when the cranial cavity is encroached upon by an excessive secretion of cerebro-spinal fluid or by foreign bodies—the circulation in the cerebral blood vessels is seriously impeded. The arteries are narrowed and the current in them slowed; the veins and capillaries are venously congested. In other words, there exists cerebral anæmia. In the cortex the effects of this are seen in the various mental symptoms of compression; in the medulla, by symp-



A



B

FIG. 18.—Fracture of the skull, with epidural, *A*, and intra-cerebral, *B*, hemorrhage, and softening. (Anger.)

toms referable to interference with respiration, and, especially important in this connection, to the circulation. Every sudden rise in brain pressure causes a rise in blood pressure, due, in all probability, to irritation of the vaso-motor center by anæmia. Cushing believes the action of the center under these circumstances to be of essentially compensatory character. He says that the rise in blood pressure tends to counteract the bulbar anæmia which the increased brain pressure has caused; and that when the brain pressure reaches a degree that the increased blood pressure can no longer counteract, the bulbar anæmia increases to the point of causing death by paralysis of respiration.

Symptoms.—Rise in general blood pressure, therefore, is to be rated as an important early symptom of brain pressure. By means of the sphygmomanometer (see p. 54) it can readily be measured; and the use of this instrument is of the utmost importance in head injuries, giving as it does valuable indications for operation. Rise in general blood pressure is an early, though not always the earliest, symptom. In later states of compression, shortly before death, the blood pressure falls.

Of the other early symptoms of compression of the brain are congestion of the face, headache, nausea, vomiting, restlessness, delirium, varying degrees of unconsciousness, slow pulse, and irregular breathing. The pupils are contracted at first, or unequal, the larger pupil generally being on the side of the compression. Later, the pupils are dilated and do not respond to light. These are the general rules, though subject to many exceptions. Even in the early stage the veins of the optic disk may show congestion and tortuosities. In the terminal stage of compression there is profound coma, with loss of sphincter control. There is choked disk. The temperature, normal or subnormal at first, may rise. Phelps maintains that high fever is not a part of the symptomatology of

hemorrhage, but rather indicates laceration. The pulse becomes rapid, the blood pressure falls, the breathing grows deep and stertorous, becoming of the Cheyne-Stokes type, and death ensues through failure of respiration, œdema of the lungs, or foreign-body pneumonia.

The severity of the symptoms and their duration depend upon the degree of compression, and the rapidity with which it is exerted. Moderate pressure may cause no symptoms other than irritability and headache, and be an entirely recoverable lesion. Extreme and rapidly exerted pressure, such as is caused by large epidural hemorrhages, may be quickly fatal. In slowly exerted pressure death may be postponed for several days; or, if the bleeding stops, the patient may recover. It is said that diminution of the cranial contents by one-sixth of their volume is invariably fatal. The symptoms of traumatic compression are usually, though not always, combined with those of concussion, and often with those of alcoholism. When the compression is in a specialized region of the brain, focal symptoms are added.

Causes.—The most important causes of traumatic compression are hemorrhages. Depressed fractures of the skull, when not associated with hemorrhage, contuse and lacerate the brain, but probably do not cause sufficient diminution of the cranial contents to give compression symptoms. It is possible that simple increases of the cerebro-spinal fluid may occur after injuries (meningitis serosa traumatica) causing increase of brain pressure. But, for practical purposes, the one cause is hemorrhage (Fig. 18).

Hemorrhage arises from laceration of the intracranial vessels. The vessels are lacerated by splinters of bone or by foreign bodies; rarely they are directly torn, there being no fracture in the vicinity of the bleeding. The laceration is usually on the side of the injury; sometimes, however, by contrecoup on

the opposite side. Hemorrhages take place external to the dura, internal to the dura, and in the brain substance. The external or epidural hemorrhages come from the meningeal vessels (middle meningeal artery) and are usually in the immediate vicinity of a fracture of the vertex. They often appear as very large clots. They give pronounced and increasing focal symptoms and are the most favorable for operation. Subdural hemorrhages are usually from the pial vessels. The blood spreads under the dura, often covering one-half of the convexity. Such cases have a high mortality and are unfavorable for operation. They are frequently associated with fractures at the base. Traumatic hemorrhages into the brain are complications of lacerations of the brain. If the bleeding is extensive the symptoms of compression set in at once. More frequently, however, the patient recovers from the effects of the concussion, and the symptoms of compression do not set in until some time after the accident. The length of the interval is usually only a few hours. It may, however, be protracted for two or three days.

The symptoms vary with the vessel affected. The middle meningeal artery, or one of its branches, being close to the bone and distributed over the whole lateral aspect of the skull, is the vessel most exposed, and consequently bleeding from it as a complication of fractures of the vault is the commonest cause of compression by hemorrhage. The hemorrhage may be extensive, several ounces or more, or small. The symptoms are those of brain compression generally, plus such focal symptoms as may be occasioned by pressure on specialized regions. The commonest seat of the hemorrhage is over the motor region. Consequently irritative and paralytic motor symptoms are prominent. Muscular spasms, succeeded by slowly progressive paralysis with rigidity of the limbs of the opposite side, are highly characteristic.

When the extravasation is in the anterior or posterior poles of the brain, localization by means of the nervous symptoms is more difficult, and reliance has chiefly to be placed on the situation of the external injury. Although successful trephining has been performed over such highly specialized centers as those for speech and the spatial sense (astereognosis) the vast majority of cases are accompanied by too great perversion of intellectual function to make such diagnosis possible.

Hemorrhage from the dural sinuses is an occasional cause of intracranial hemorrhage. The deep-lying sinuses are inaccessible to surgical relief, and are only ruptured in extensive general injuries. The superior longitudinal sinus, however, is not infrequently lacerated in fractures of the vertex and by penetrating wounds; the lateral sinus, by direct lacerations and by transmitted force. The diagnosis is generally possible by the external appearance of dark fluid blood. When the hemorrhage is intracranial only, the diagnosis depends upon the nature of the injury, upon the symptoms of compression, and upon, possibly, the gradual development of hemiplegia.

Hemorrhages from the pial vessels are invariably associated with general brain contusions. As causes of symptoms of their own they are relatively unimportant, although they may be of sufficient size to give rise to symptoms of brain compression. It is doubtful if they can be distinguished, clinically, from extradural hemorrhage by any means other than lumbar puncture. If the hemorrhage is at all extensive the cerebrospinal fluid will be found to be blood-stained. Hemorrhages in the internal parts of the brain are associated with lacerations of the brain substance, and will be considered under that heading.

Prognosis.—The prognosis of head injuries accompanied by compression of the brain by hemorrhage is always very grave. V. Bergmann reports a mortality of ninety per cent

in non-operated cases. In the larger number of fatal cases the primary unconsciousness is recovered from for a brief period only, or not at all. The symptoms of compression, which are severe, come on rapidly, and death ensues in from

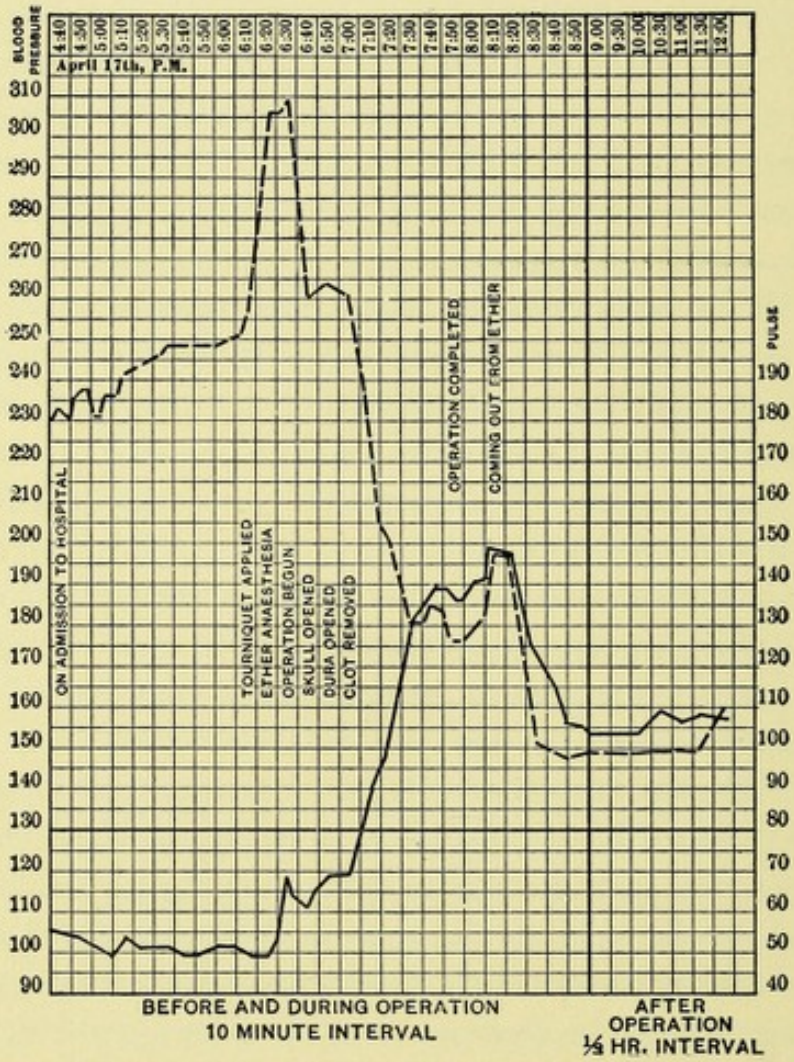


FIG. 19.—Case of compression of the brain by hemorrhage. Chart taken before, during, and immediately after operation. Broken line represents blood pressure in mm. of Hg. Solid line represents pulse rate. (Cushing.)

twelve to forty-eight hours. The prognosis is naturally better when the symptoms develop slowly, as in such cases the bleeding is either more apt to stop spontaneously, or there is more time in which to decide on operative interference.

Treatment.—There are two methods of treatment. One, expectant, during which the patient is kept as quiet as possible, morphine is given if necessary, ice is applied to the head, and the symptoms carefully watched. If the symptoms increase, as shown by a rapid rise in the blood-pressure curve, or in the accession of other symptoms, the expectant plan must be abandoned. The purpose of operation is to remove the clot and to check the bleeding point, if possible.

If the symptoms point to involvement of specialized centers, such as that for speech or for a particular portion of the motor tract, the skull should be opened over these centers. Usually, however, the clot covers a large part of the convexity and is readily accessible through an opening made over the middle of the motor region. The middle meningeal artery, or one of its branches, may be tied through this opening.

The relief of symptoms by removing the compressing body is often immediate. The change in the blood pressure is shown in Fig. 19, which is the chart of one of Cushing's cases.

Wounds of the Brain

Wounds of the brain by bullets, sharp objects, etc., belong to the domain of surgery, especially to military surgery, rather than to neurology. Bullet wounds cause concussion, laceration, and hemorrhage, and, secondarily, compression. Long-range and spent bullets can cause cerebral symptoms without fracture of the skull. Generally, however, the skull is fractured. When the bullet enters the skull the dangers of infection are great, and abscess of the brain is a very frequent complication. The symptoms are those of brain injuries generally. Any of the cranial nerves may be injured, but the optics are affected with especial frequency. In some cases of Mauser-bullet injuries, single nerves may be picked out with remarkable exactness,

without any general brain symptoms except the preliminary ones of concussion, etc. The location of the bullet, even with the x ray, is always extremely difficult.

The after results of bullet wounds are those of brain injury, generally, except that bullet wounds are particularly prominent in the causation of abscess and of traumatic epilepsy.

Treatment.—Cleanliness and drainage are the chief features. In bullet wounds, the bullet should be removed if possible. This is usually both difficult and hazardous.

CHAPTER III

THE COMPLICATIONS AND SEQUELÆ OF BRAIN INJURIES

Traumatic Meningitis—Abscess of the Brain—Nonpurulent Encephalitis—Traumatic Apoplexy—Traumatic Epilepsy—Tumors.

Traumatic Meningitis

Clinical Forms.—Meningitis from trauma is almost entirely a surgical affection. It may affect the dura (pachymeningitis) on its external or internal surface. In external pachymeningitis the inflammation is limited, is in direct association with the external wound, and is due to retained wound secretions, to infection, or to foreign bodies. Internal pachymeningitis is a chronic and productive rather than a septic inflammation. It may be either localized or diffuse. Localized, it is in relation to the external injury, is the cause of localized headache without general brain symptoms, and may be relieved by operation. The diffuse variety is similar to the hemorrhagic pachymeningitis of non-traumatic origin. There is headache and blunting of the intelligence, and, if hemorrhage occurs from the newly formed tissue, focal symptoms. Trauma as a cause of this form of meningitis is not definitely established.

The commonest variety of traumatic meningitis is leptomeningitis, or that affecting the pia. It is acute, and of septic origin, with tendency to spread. It may begin over the convexity or at the base, though it is rare at the base. When it begins over the convexity, the infection takes place through the external wound; when at the base, through traumatic communications with the orbit and the nose, rarely the ear. The

cerebro-spinal fluid is turbid if the meningitis is extensive. The symptoms begin abruptly, consisting of headache, chill, stiffness of the muscles at the back of the neck, fever, and rapid pulse. At first there is delirium, later coma.

Under the term serous meningitis have been described cases in which brain symptoms develop, usually immediately after an injury to the head. The symptoms are general, indicating compression, though they may consist in one-sided convulsions or paralysis. The post-mortem appearances are limited to large collections of fluid beneath the dura.

This condition is obscure, is rare, and can hardly be recognized *intra vitam*.

Prognosis.—The prognosis is for all varieties of generalized meningitis grave, and the treatment consists in cleansing as far as possible the sources of infection.

Traumatic Abscess of the Brain

Abscess of the brain consists in a focal suppuration of the brain substance. In the only variety considered here the pyogenic infection gains entrance by traumatic injury to the head. It is not probable that cerebral abscesses occur by metastasis from suppurating peripheral wounds without general visceral involvement. Mere contusions of the scalp, without fracture, are not sufficient to cause abscess. There is generally an injury to the bone, although the infection may be transmitted from a scalp wound through uninjured bone. The commonest causes are infected wounds of the skull or its coverings. Depressed fractures of the vertex, the entrance into the cranial cavity of foreign bodies, such as splinters of bone, bullets, punctured wounds in which pieces of the puncturing object remain in the brain, are frequent causes. The more open and free the external wound, the less is the danger of abscess. Non-oper-

ated cases are followed by abscess with especial frequency. In 112 reported cases, mainly treated without effort to remove the bullets or fragments of bone which penetrated the brain, deep abscess resulted in nearly ten per cent (Phelps). Fractures at the base are rare as causes. When active, they are so by means of infections which gain entrance through fractures involving the ethmoid cells, the pharynx, orbit, or rarely, if ever, the ear. In some late abscesses, it is possible that areas previously the seat of hemorrhagic softening become infected through the blood.

Thus Daiber reports the case of a man who fell and sustained a contusion of the right occipital region. He was unconscious for five days. For some time afterwards he suffered from dizziness, but was practically well for ten years. Then he had an attack of unconsciousness lasting five minutes, with convulsions, especially of the right side. A few months later he became paralyzed on the right side, with Jacksonian seizures, and died in twenty-three days. The autopsy showed an abscess in the left motor area, which probably took place through the breaking down and infection of a contrecoup hemorrhage, as there was no trace of any skull injury.

Traumatic cerebral abscess is most common in adult males. It is rare in children. The danger of its occurrence as a complication of head injuries is, after all, remote, except where foreign bodies have gained entrance to the brain. Of 400 cases of fracture of the skull, English saw but three abscesses develop. The percentage of traumatic abscess to abscess of other origin has been estimated at from sixteen per cent to fifty per cent.

Pathology.—The focus of suppuration is single in ninety-three per cent of the cases (Macewen). It is small, or may attain the size of a hen's egg or even larger. It grows rapidly or slowly. Slow-growing abscesses are usually encapsulated. The brain substance in the abscess region is softened, and may contain hemorrhages. If superficial, it is directly under the

external wound, and forms part of a general infection in which scalp, bone, and membranes share. If deep, it is subcortical, and may be separated from the external wound by an inch or more, often connected to the periphery by a narrow line of suppuration. Deep abscesses are particularly common in bullet

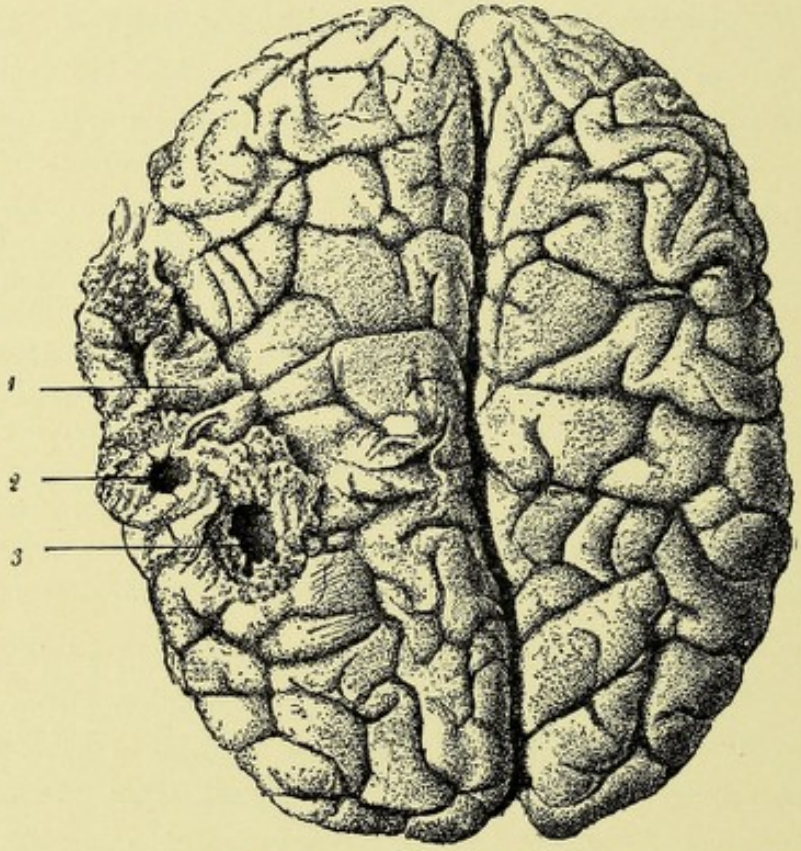


FIG. 20.—Abscess of the brain, from an injury in the right parietal region. (Roncali.)

wounds; they are found chiefly in the frontal and occipital lobes and beneath the motor area.

Symptoms.—In a general way, cerebral abscesses may be divided into (1) superficial and (2) deep. The superficial abscesses are early in appearance, and are surgical rather than neurological. They constitute a complication rather than a sequel of the injury. Situated in the cortex, they are direct

extensions of the suppuration in the scalp, bone, and membranes. Symptoms referable to them are often masked by the general and local symptoms of the injury. They consist in the general symptoms of infection, and may also present, though less clearly and fewer in number, the same signs as will be described under deep abscess.

The symptoms of deep abscesses are slower to appear. While they may set in within a few days of the trauma, the average time of appearance is from three to five weeks (von Bergmann). Not infrequently the interval is months or years. English reports the case of a woman who, ten years after a depressed fracture of the vault, died after operation. The abscess was situated in the temporo-sphenoidal lobe, beneath the seat of fracture. During the ten years she had been perfectly well, with the exception of occasional headaches, and, eight years after the accident, of a single temporary attack of acute pain in the neighborhood of the old injury, with tingling in the opposite arm, and vomiting. Even longer intervals have been known. The symptoms must be considered in connection with the original injury and the length of time before the infection makes itself symptomatically recognizable. After severe cranial traumata, the original symptoms of concussion may persist and mask those of the abscess; or the patient may seem to be recovering, and be suddenly taken with headache, vomiting, rise of temperature, etc. In some cases sudden death occurs without premonitory symptoms. The shorter the interval the more acute is the onset, although in general the invasion is less abrupt than in abscesses of otitic origin. In cases which are first recognizable long after the injury, the invasion is usually very slow indeed. In such cases, in the interval, the patient may feel fairly well, or have indefinite subjective cephalic sensations, or headache, or temporary attacks of aphasia, or one-sided numbness, or other

evidences of focal lesion. General recurrent convulsions have in a number of cases been the only premonitory symptom. The symptoms, once developed, may be divided into general and local. The general symptoms are those due to the infection. Of these, fever is fairly constant. It is not usually very high (101° F. to 102° F), although it may go to 104° F., or even higher. The curve is interrupted and irregular, and often sinks below normal. The shorter the time after the injury, and the more rapid the invasion, the higher and more irregular the curve is apt to be. In acute cases it begins suddenly, often with a chill, although chills are less frequent than in otitic abscesses. In some cases the temperature has been found higher on the affected side of the head. The pulse is slow, as a rule, but in some cases with high fever, and in many terminal stages, it is rapid. Still, a pulse of 50 or 60 is more common than one above 70. It increases in frequency when the abscess is evacuated. Vomiting is frequent. It occurs both as the result of gastric irritability, and in the projectile form of cerebral vomiting. Constipation is the rule. There is generally rapid emaciation and loss of strength. Leucocytosis is high. Unless the abscess has ruptured into the subdural or ventricular cavities, the cerebro-spinal fluid, as examined by lumbar puncture, is clear, though under increased pressure.

The local symptoms are those which indicate an increase in brain pressure, together with focal signs which vary with the region affected. Of the symptoms of increased brain pressure, headache is the most constant, and the earliest. Like other symptoms, it develops gradually or quickly. It may be general, but, as a rule, it is more marked on the affected side. It is described as severe, though generally not so severe as in brain tumor. It is variable, being much more intense at some times than at others. Sometimes it is associated with pain and stiffness in the muscles at the back of the neck. In such cases

the abscess is situated in the posterior pole of the brain. In rare cases headache is absent. Dizziness is not constant, though it occurs.

Optic neuritis is an important sign and present in thirty to thirty-five per cent of the cases. It is more frequent than choked disk (Oppenheim). It is often unilateral, corresponding to the side of the abscess. It only occasionally interferes seriously with vision.

The characteristic mental state of cerebral abscess is that of indifference, apathy, and somnolence, which is sometimes preceded by restlessness and confusion. In a large number of cases the mental state, headache, and slow pulse are the only definite symptoms. When the stupor is profound, it may be responsible for loss of control of the sphincters.

The focal symptoms depend upon the region of the brain involved. In "silent regions" they are absent. Also, in chronic cases, the brain adjusting itself to gradually increasing pressure, they may be slight or absent. Focal symptoms may also be due to the œdema which surrounds the abscess. These disappear upon evacuation of the abscess, but may cause errors in localization.

The most frequent sites of traumatic abscess are the frontal lobes and the motor region. Motor symptoms predominate as focal signs, and the face, or one limb, of the opposite side may be paralyzed, but more often there is hemiplegia. The paralysis is of the central neuron type with increased tendon reflexes, and appears as a weakness rather than a complete loss of power. Irritative phenomena are very common. Rigidity and resistance of the limbs of one side is highly characteristic. Jacksonian seizures are also common, and general convulsions are sometimes the chief symptom. Associated with the paralysis, or rigidity, or local spasm may be sensations of numbness and tingling, usually transitory, but sometimes constant. If

the abscess is in or near the motor speech centers, there is motor aphasia.

Traumatic abscesses in the temporal lobes are rare. If they occur here, the focal symptoms are those of temporal affections generally, plus, in many cases, the effects of pressure on the motor tract.

In the occipital lobes traumatic abscess is more frequent. In this situation they furnish few or no focal signs, although hemianopsia has been observed. Focal lesions in the occipital lobes may give symptoms strongly suggestive of cerebellar involvement. Such a case is the following, reported by Reverdin and Vallette:

A boy was struck by a stone in the back of the head a little to the right of the median line. The wound suppurated, and there was a small sequestrum of bone. Four months later he developed headache, vomiting, and the symptoms of meningitis. These symptoms, which improved somewhat, were followed by stiffness in the muscles of the back of the neck, and rigidity of the legs, which only lasted a short time. Then ensued somnolence, general torpor, and unsteadiness of gait. In the right eye there was external strabismus and a slight squint, with an enlarged left pupil. No diplopia and no hemianopsia, but bilateral choked disk. The left knee jerk was exaggerated. There was ataxia of the legs and a tendency, in walking, to fall to the right. The autopsy showed an abscess in the right occipital lobe.

Isolated traumatic abscess of the cerebellum is so unusual that such a diagnosis should be made only with the greatest care.

Course and Prognosis.—Superficial abscesses, being generally merely a part of the original injury, may evacuate themselves externally. But spontaneous recovery from deep abscess of the brain is a negligible quantity. Once begun they go on until death, or until relieved by operation. The symptoms are subject to variations in intensity, but their course is constantly progressive. They may exist for several months, sometimes very much longer, before proving fatal. Sudden death

occurs from rupture of the abscess into one of the encephalic cavities, or with nothing but the internal hydrocephalus to account for the suddenness. Gradual death occurs as the result of the general intoxication or exhaustion.

Diagnosis.— The diagnosis is always difficult, and only in rare cases can it be made with absolute certainty. The history of the injury is naturally most important, though it may be so indefinite as to be valueless. If the examination of the head shows extensive scar formation or depression of bone, the diagnosis is much helped thereby. Great probability for the diagnosis is given if evidences of suppuration are still present.

It should be remembered that fracture of the vertex of the skull may occur, and give rise to abscess, without leaving trace of external wound. Depressions in the skull, therefore, even without scar, must be looked for. The conditions which most often cause confusion in diagnosis are epilepsy, certain psychoses, meningitis, and hemorrhage.

Both general and focal convulsions, which occur after head injuries, may be due to abscess, although there is nothing in the convulsions themselves to stamp their origin. Every case of post-traumatic convulsions, therefore, should be investigated for the general symptoms of abscess. In abscess, the parts affected by the convulsions are apt to show loss of voluntary motor power coming on, not directly after the injury, but more or less simultaneously with the seizures. In epilepsy there are wanting the symptoms of internal hydrocephalus, such as severe headache, vomiting, choked disk, slow pulse, etc.

Certain post-traumatic psychoses, especially those of the epileptic type, with headache, momentary losses of consciousness, dullness of the general mental state, may be mistaken for abscess. They can only be correctly recognized by consideration of the general symptoms, and by the absence of focal symptoms.

Meningitis is confused chiefly with superficial and early abscesses. However, in late, deep abscess in the occipital region there is sometimes rigidity of the muscles of the back of the neck.

From post-traumatic hemorrhage abscess also offers great difficulties of differentiation. In general it may be said that hemorrhage usually appears earlier than abscess; that it may stain the cerebro-spinal fluid; that it is more sudden in its onset and indicates more extensive lesion, and that the evidences of extreme internal hydrocephalus are absent. In hemorrhage leucocytosis is often present, but in less degree than in abscess; in hemorrhage, also, the evidences of general infection are absent.

Nonsuppurative encephalitis distinguishes itself from abscess by its course, viz., a sudden and brief accession of general symptoms, which pass away leaving a permanent paralysis.

The **treatment** of cerebral abscess is operative, the guiding signs being the external injury and the focal symptoms.

Without operation brain abscess is hopeless. Operation has materially modified the mortality, and offers a legitimate hope of cure in every case. Every case of head injury with persistent or late appearing symptoms should be investigated with especial care for abscess; and operation should be undertaken when the symptoms are sufficient to make the existence of abscess probable.

Non-purulent Encephalitis

This disease involves the cerebral cortex chiefly, although sometimes the midbrain, medulla, and cerebellum are affected. Its pathological anatomy is similar to that of anterior poliomyelitis. The blood vessels degenerate, their walls are infiltrated

with round cells and red blood cells, there are capillary hemorrhages, and degeneration of ganglion cells. The disease is usually focalized, and most frequent in the region of the motor cortex.

Symptoms.—The symptoms are usually ushered in by a day or two of *malaise,* headache, dizziness, and apathy. The apathy rapidly deepens to stupor, and often to coma. There is then a sudden rise of temperature (102° F. to 104° F.), with rapid pulse, following which focal symptoms show themselves. These depend upon the region of the brain involved, but aphasic symptoms and monoplegia are particularly frequent. There may be hemiplegia, or cranial nerve palsies, or ataxic symptoms referable to the cerebellum. Optic neuritis has been observed. Convulsions are rare. Encephalitis has a high mortality. The average duration in non-fatal cases is about three weeks. If death does not occur, the focal symptoms may improve greatly, or persist.

Ætiology.—Non-purulent encephalitis most frequently follows infectious diseases, and itself is doubtless an infectious disease. The relation of trauma to it, therefore, presents the same questions as trauma and infection generally. There are, however, a few instances of typical encephalitis, following trauma to the head, in which no other cause was discoverable.

Diagnosis.—Inasmuch as the brain symptoms have begun within a few days or weeks from the injury, hemorrhagic encephalitis may be confused with either brain abscess, meningitis, or late apoplexy.

It is possible that some of the obscure, yet, doubtless organic, sequels of head injuries belong to the category of hemorrhagic encephalitis.

Friedmann has described the case of a previously healthy man who fell on the left side of the forehead, and who, without loss of consciousness, developed on the right side paralysis of the third, sixth,

seventh, and eighth nerves. He complained of severe headache and dizziness, and on getting up from bed (in fourteen days) was uncertain in gait. He gradually but constantly improved for six months. Then the headache again became so severe that he had to go to bed. At this time there was paralysis, with rigidity and tremor, in both legs. In a few days the constant headache disappeared, being replaced by a periodic headache, which came mornings and evenings. The headache could not be controlled by drugs. During the attacks of pain the face became very red, and the patient sweated. The attacks continued for several months, until the headache again became constant, so that the patient had to go to bed. With depression as the only preceding mental symptom, coma gradually developed, and after a few days the patient died.

Macroscopically the brain was normal. Microscopically the small vessels and capillaries were filled with blood, there were minute fresh hemorrhages about the vessels, and the vessel sheaths were filled with blood cells and the vessel walls infiltrated with small round cells.

Cramer reports a similar condition. The patient had convulsions as a child, but was, nevertheless, intellectually and physically well developed. In twelve to fourteen days after a fall from a horse, he developed, without warning, great excitement and hallucinations. There were at the same time twitchings and a varying degree of clouding of the intelligence. The excitement continued, and the patient died in coma. The autopsy showed adhesions of the dura, venous hyperæmia of the pia, and flattening of the convolutions. The cortex was markedly reddened and ingested, and the base was full of blood. There was no loss of fibers in the cortex, but the ganglion cells showed a loss of Nissl bodies. There were fresh hemorrhages in gray and white matter. Many veins and capillaries showed in their sheaths fresh blood pigments with leucocytes in the adventitia.

Traumatic Apoplexy

Ætiology.—In addition to the hemorrhages in the membranes or in the brain substance which are the immediate and direct results of laceration, the indirect effects of trauma as a cause of cerebral hemorrhage merit consideration. When apoplexy occurs, vascular disease has preëxisted, as a mere increase in blood pressure does not cause normal arteries to break. To this perhaps exception must be made for the late

apoplexy of Bollinger. In how far injuries contribute to the arterial degeneration which leads to apoplexy is a vexed question, and one difficult to decide.

The general causes of such degeneration, which is of slow course, are subtle and manifold. Advancing age, all chronic diseases, syphilis, some general infections (typhoid fever), and many poisons (lead, alcohol, and tobacco), contribute to this end. Psychic effects, such as grief, worry, excitement, are doubtless of great importance also. With all these factors to be considered, it is well-nigh impossible to determine what place, if any, should be assigned to an injury of years before. Yet not only is injury to the head advanced as a cause, but it has even been maintained that general injuries, which at first give the symptoms of traumatic neurasthenia, result in progressive cerebral endarteritis.

After many accidents certain general factors in arterial degeneration are called into play for which the accident is only indirectly responsible. The patient is liable to the various psychic symptoms of worry, anxiety, etc., to be described on a later page. In addition—and this is especially true in the laboring classes—he is apt to be idle. As always, idleness means less exercise, less fresh air, less concentrated effort, and more alcohol and more tobacco—all factors in the production of arterio-sclerosis. Consequently trauma, if a cause of chronic arterio-sclerosis, acts in more ways than one.

Symptoms.—There are three varieties of vascular disturbance occurring after injuries to the head in which the connection between cause and effect is close if not indisputable. These are: 1. Apoplexy occurring simultaneously with the injury. 2. Apoplexy occurring shortly after the injury. 3. Apoplectiform symptoms occurring a long time after the injury.

1. *Apoplexy Occurring Simultaneously with the Injury.*—In these cases, rare, but of great medico-legal importance, the

patient, immediately after a blow on the head, develops the symptoms of a hemorrhage of the internal parts of the brain. Usually the blow is not severe, and all evidences of injury to the scalp or skull may be wanting. The symptoms are those of apoplexy, and may be quickly fatal. Or there may be hemiplegia involving arm, leg, and face. Such hemiplegia is more complete than that from pure traumatic lesions, and there are wanting evidences of irritation and the symptoms of concussion. In addition, unmistakable signs of general vascular degeneration are present.

In a recent case the patient was struck on the head in a street brawl. He became momentarily unconscious and completely hemiplegic. Examination a few days later showed left hemiplegia, normal mental state, and no general brain symptoms, but a well-marked arterio-sclerosis of the peripheral arteries, and a hypertrophied heart with a systolic murmur at the apex.

The explanation of these cases consists in a preëxisting weakness of the walls of the cerebral arteries, by miliary aneurisms or otherwise, which, when subjected to a sudden rise in blood pressure, give way. It is possible that the rise in blood pressure is due to the mechanical effect of the blow. But in many cases attendant circumstances leave little doubt that the psychic factors of fright and excitement are the most active exciting causes.

2. *Apoplexy Occurring Shortly after the Injury* (*Traumatische Spät Apoplexie*).—This condition, described by Bollinger, comes on a few days or weeks after a head injury. The hemorrhage, which in Bollinger's cases was fatal, takes place in the neighborhood of the fourth ventricle and the aqueduct of Sylvius. Bollinger maintains that the hemorrhage is preceded by local softening, which was caused by unequal pressure of the cerebro-spinal fluid.

A considerable proportion of these cases have been in

young persons. The injury is invariably to the head. It may or may not cause unconsciousness. In any event, the patient recovers and is able to return to work. After a few days or weeks there begin headache, somnolence, and coma, with paralysis of the extremities or of the cranial nerves; or, after a similar period of freedom, an apoplectic stroke may occur without any warning. Since the introduction of the term of traumatic late apoplexy, many cases have been described which the term originally was not intended to include—cases of ordinary hemorrhage, secondary to laceration, and cases in which the blow, or the shock attending it, caused the rupture of preëxisting miliary aneurisms. These are not cases of late apoplexy. Also a variety of peculiar and non-fatal symptoms, such as attacks of somnolence, of stupor, of cranial nerve palsies, etc., are placed in this rubric, though that they should be here is a question of doubt.

3. *Apoplectiform Symptoms Occurring Long after Injury.*—The symptoms in this class point to a slow increase in vascular occlusion in parts long before the seats of traumatic insults, and are those of slow thrombosis. It is generally seen in cases of fracture of the skull, with extensive laceration of the brain. The increase in symptoms begins after a lapse of many years. The following cases illustrate the condition:

A man sustained a compound depressed fracture of the vertex by being hit by a brick in 1861. The left leg was paralyzed, but recovered in a few weeks. Ten years later the leg again became weak, and finally almost completely paralyzed. The arm, also, lost considerable power, and the intellect became blunted. (English.)

Frost reports the case of a man who, twenty-six years after a compound fracture of the skull, with extensive loss of substance, slowly developed paralytic symptoms in the limbs of the opposite side. The autopsy showed general degeneration in the cerebral arteries, but more marked on the affected side of the brain. In this region, also, the brain showed cavities, the results of small areas of softening.

Traumatic Epilepsy

Epilepsy is one of the most frequent affections of the nervous system, and is among the commonest of chronic diseases. It is estimated as occurring in one of every 500 of the population. In a definite number of cases no cause can be found to explain the convulsions; then the epilepsy is called idiopathic, and is regarded as a disease *sui generis*. In a small percentage of the total number of cases the convulsions can be shown to depend upon gross intracranial lesions (Jacksonian epilepsy), or upon the irritation of the cortical cells by toxic substances circulating in the blood. In such cases the convulsions are the expressions of definite pathological states.

When the attacks occur as the immediate result of physical injury, epilepsy is called traumatic; the trauma causes some morbid alterations in the brain substance which find their clinical expressions in recurring convulsions.

The frequency of traumatic epilepsy, as compared with the idiopathic form, is somewhat difficult to determine. If we are to accept as traumatic every case which has the history of a fall or a blow on the head, received years before the appearance of convulsions which are from the first general in character, the category of idiopathic epilepsy would become very restricted; for few persons reach adolescence without a fall or an injury of some kind, and almost every epileptic patient can be made, if closely questioned, to admit some such history. According to the books at the Vanderbilt Clinic, a large percentage of all the patients had falls in infancy, or frights, or other slight injuries, but they are not regarded as examples of traumatic epilepsy, and it seems doubtful if even one per cent of the total number of cases of the disease could be proved, with reasonable probability, to be so. It is convenient to discuss the subject under three heads:

(1) Traumatic true epilepsy. (2) Traumatic focal or Jacksonian epilepsy. (3) Traumatic reflex epilepsy.

1. Traumatic True Epilepsy.—Under this heading are included cases symptomatically identical with idiopathic epilepsy, and without visible lesion of the brain, but which first appear after an injury to the head. The convulsions are general from the outset, and recurrent.

The division between traumatic general epilepsy and traumatic focal epilepsy is in some respects artificial, but for practical purposes is necessary. General convulsions, which occur in sequence to an injury to the skull, when neither the injury itself nor the symptoms which immediately follow it indicate gross lesion of the brain, are classed as general or true epilepsy. The brain may have sustained gross lesions, but they cannot be recognized. When convulsions occur in sequence to a skull trauma which we know has injured the brain (as evidenced by immediate symptoms, such as paralysis, or, by the nature of the accident, such as the entrance into the brain of foreign bodies), even though the convulsions be general from the first, they are none the less classed as focal epilepsy; for in these latter cases, though the convulsions are general, they result from a focal lesion.

Ætiology.—Of 3,000 cases of true epilepsy studied by Gowers, blows and falls on the head are ascribed as causes in 108. A third occurred under ten years of age, rather more than a third between the ages of ten and twenty years.

Wildermuth found four per cent of 189 cases due to skull injuries.

Of 300 cases of head injury, English found 21 cases developed epilepsy after a year or longer. Of these, 7 were Jacksonian in type. As convulsions play a minor rôle in the acute symptoms of fracture at the base, we hardly expect epilepsy to be a frequent result of that injury, and it does not seem

to be one. The statistics of the Craig Colony for Epileptics show that of the 826 cases at that institution, in only one is there any suggestion of a fracture at the base.

Of the immediate causes of the disease, injuries to the head are the most important. The records of the Franco-Prussian War show that from 8,985 head injuries there developed 46 cases of epilepsy. The same records show 17 cases among 77,461 persons wounded in the body or extremities. Thus head injury is not everywhere regarded as the sole cause.

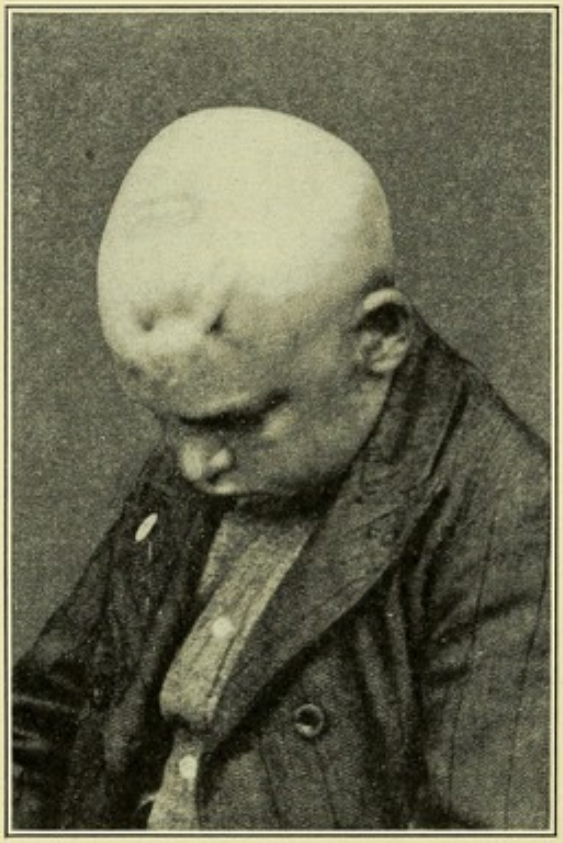


FIG. 21.—Case of traumatic general epilepsy from fracture of the skull. (Craig Colony.)

In most authentic cases, the injury has been to the head either as blows or falls on the head, and sufficiently severe to cause primary unconsciousness. Fright, also, is an important excitant of epilepsy, and must be considered in connection with all traumatic cases. After fright, the convulsions are general and ensue quickly. In true traumatic epilepsy, predisposition is an extremely important factor. As Gowers says, the exciting cause is only the spark which ignites the powder. Alcoholism constitutes the most prominent of the forms of acquired predisposition. General convulsions are a not uncommon symptom in alcoholism, and are often first called into play by a physical injury.

Cases of traumatic general epilepsy are always difficult of exact clinical interpretation; but before they are received as traumatic, due consideration should be given the following points:

In the first place, it must be shown that the patient had never had general convulsions before the injury, except such convulsions as may occur in infancy without being considered as expressions of idiopathic epilepsy. General convulsions are so common in infancy that it would be manifestly unjust to regard as epileptic persons who had had fits in the first year or two years of life, but who had subsequently been free from them.

The general condition of the patient, as regards alcoholism or other degenerative evidences, immediately before the accident, must be considered, especially if the injury is supposed to have been slight.

The injury itself is naturally the most important causal element. To merit consideration as a cause, it should be sufficiently severe to bring about some loss of consciousness.

The time elapsing between the receipt of injury and the first epileptic manifestations cannot be considered as a definite guide in diagnosis.

Some few cases, which in other respects are satisfactory as illustrative of traumatic epilepsy, have had no fits until several years after the receipt of the injury. The most that can be said in this respect is that the larger number of cases have developed within a year after the accident, and that most of the cases which appear after longer periods of time are much less certainly of traumatic origin.

In doubtful cases the age of the patient may be of assistance in diagnosis. Idiopathic epilepsy is essentially a disease which begins in childhood and adolescence. Sixty per cent of the cases occur before twenty years of age, and only thirteen

per cent after thirty. This is true, irrespective of causes. When we come to consider traumatic factors we find that they figure as causes of general convulsions chiefly in youth and adolescence. General convulsions occurring for the first time in an adult after an injury to the head, without evidences of focal lesion, are only to be accepted as traumatic and as true epilepsy after careful inquiry has eliminated all other possible causes, notably alcoholism.

Symptoms.—The symptoms of traumatic general epilepsy develop at varying periods after the injury. In about one-half the cases the first fit takes place within a month. After a year or more the possible activity of other causes always obscures the diagnosis, although a number of cases have been reported as traumatic in which the interval was ten years or longer. The symptoms present no differences from idiopathic epilepsy, consisting in *grand mal* attacks, *petit mal* attacks, equivalents, and secondary mental disturbance. Between the attacks, at first, the patient is normal, or presents such symptoms of predisposition as he previously had had, or the symptoms of traumatic cerebrasthenia. The ultimate tendency of the disease, however, is toward augmentation in the number of the seizures and toward mental deterioration.

About one-third of the cases of idiopathic epilepsy have mental disturbances aside from those associated with the unconsciousness of the attack or the ultimate deterioration. According to Wagner, such mental disturbances occur three times more frequently in traumatic than in idiopathic epilepsy. They usually come on within a few months of the injury, and with especial frequency in predisposed individuals. General convulsions are often absent, though they may appear ultimately. The psychic attacks usually come unheralded, although they may be preceded by irritability or mental depression. The symptoms of the attacks vary greatly in range and dura-

tion. Memory for what occurred during them is obliterated. Courtney suggests dividing them into *petit mal* and *grand mal* attacks.

The *petit mal* psychic attacks are of short duration, and are characterized by automatic acts. Courtney describes such a case:

> A man, eighteen years of age, always well and without discoverable predisposition, fell from a tree. He was dazed, and walked several miles before coming to himself. A few years later he began to have "fainting spells," during which he lost consciousness. Since then he has had recurrent attacks of automatism with loss of memory. Between attacks he does his ordinary work. In an attack observed fifteen years after the accident, the patient, without convulsive movement, became very quiet, sitting in a chair, and reading a paper upside down. The face was congested, the pupils did not respond to light, the knee jerks were absent. He paid no attention when spoken to; given small pieces of paper, he tore them up. Then he signed a note for $10,000, saying that he wished to do so, and knew what he was doing. The attack lasted about fifteen minutes. When over, he had no recollection of anything he had done. Said he had never seen the note before.

The *grand mal* psychic attacks are explosive, characterized by sudden anger, by violence, by automatic acts, and resemble in many ways the explosive diathesis of Kaplan. Between the attacks the patients are normal, or depressed and anxious, or troubled with hallucinations, or complain of headaches, loss of memory, etc.

The *grand mal* psychic attacks may last for weeks or months. Although of distinctly mental character they are properly classed as epileptic when associated with some of the motor phenomena of epilepsy. But when all convulsive symptoms are absent, it is difficult or impossible to say whether they should be classed with the epilepsies or with the insanities. Long observation extending over months or even years, directed toward the evolution of the symptoms, is necessary

before definite classification is possible. The following case may be one of traumatic psychic epilepsy or of traumatic insanity.

C. R., a milk dealer, thirty-six years of age, while going his rounds in his milk wagon, March 1, 1905, had a collision with a trolley car. As a result of this he was thrown forward, his head hitting with considerable force against one of the iron bows of the top of the wagon. He was somewhat dazed, but talked to the men of the trolley company. He obtained another wagon and continued his rounds that day, and on subsequent days. There was at that time a swelling of the right side of the head, from one to two inches above and behind the occipital protuberance. He applied some "horse liniment," which probably contained cantharides, to the bruise. At any rate, soon after its application the bruise "opened" and ran for a few days. From the first, the patient complained of pain in the head at the site of injury, which was constant, and increased by any contact; but he gave no other symptoms and continued his work. Ten days or two weeks after the accident the first attack showed itself. Without any warning, further than complaining of a heavy feeling in his head, he left his work and went home. He said that he had delivered his milk (which he had not), fell in a heavy stupor for several hours (from which he could not be aroused) and, when he came to himself, remembered nothing of the attack. In subsequent attacks he did a variety of impulsive and purposeless acts. On one occasion he slipped out of his window to the balcony, walked three-quarters of a mile in his night clothes, until taken to a hospital by a police officer. When arrested he did not know who he was, insisted that he must go out to deliver milk, did not know where he had come from, or why he was on the street at that hour. On coming to himself he recognized the officer (whom he knew). In another attack he closed and fastened the windows and doors of his room, and turned on the gas without lighting it. When the door was forced open he did not remember what he had done. On one occasion he emptied his milk cans in the sewer, and came home and said he had delivered his milk. For a time he had an attendant, and when out walking with him he would have sudden attacks of shivering. After these he would suddenly start off to run, would run up or down the street or across lots, evidently without purpose. When restrained he would shiver, and then ask his attendant where he was. He did not remember that he had attempted to run, or done anything unusual.

In May, 1905, he was examined by a physician who stated at the outset that he was an expert on insanity. This seemed to worry the patient very much. After a long and tiresome examination the patient began to tremble all over, was unable to stand or walk without assistance, and could not write his name. In an attempt to take him to the hospital he jumped out of the carriage, ran away, and was finally found, seven hours later and two miles away, without coat or collar, his clothes torn and covered with mud. When found he had no idea of what he had done or where he had been. In one attack he is said to have frothed and bled from the mouth. This is the only suggestion of convulsions in the history.

In other attacks, after being sullen and morose for a day or two, he would become very quiet, and unwilling to answer questions. In such attacks he was extremely fearful that something was going to happen to him, that people were after him, that he was going to be locked up. He would move about the room, and take every chance to escape from the interview. In some, he was ugly and threatened violence. At the termination of my examination he threw his watch at me in the street from a second-story window. The attacks frequently had distinct auræ and exciting causes. The pain in the head regularly became worse. Pressure over the scar would always bring on an attack. Frequently the attack was heralded by a visual aura. On several occasions he thought (incorrectly) he saw his horses and wagon, and rushed after them. Other forms of auræ are bright balls and shadows before the eyes. Sometimes the aura alone occurred, and no attack followed. Sudden excitement or fear often brought on attacks. All attacks have begun at night. Before and during an attack the pupils are widely dilated. At the end of an attack the patient falls into a stupor from which he awakes practically normal. During one of the attacks he was taken to Dr. Prout's sanitarium at Summit, N. J. Two days after admission he woke up in a normal state. He was bright, talkative, wanted to know where he was, how long he had been there, and who brought him; he wanted to return at once to work.

As to duration, the attacks have shown a steady tendency to become longer, and the intervals between them shorter. In the beginning they lasted for from a few minutes to less than an hour. Recently, the briefest has been two days and the longest two weeks. At the beginning there was an interval between the attacks of several weeks; now it is seldom more than a few days.

The case presents a remarkably clean-cut example of amnesic automatism alternating with a normal mental state. During the attack

the patient generally remembers nothing about the accident, though in some attacks he has hinted vaguely at some injury having been done him, and vowed vengeance on whoever brought it about. It can hardly be called a case of double personality, for in many of the attacks, especially the longer ones, he recognizes his own identity, his family, etc. During the shorter attacks he apparently has a blurred conception of personal consciousness or none at all. When out of the attack he can recollect nothing which occurred during the attack, but remembers all other things, the accident included. When out of the attack he seems perfectly normal, and able to conduct his business. To this the exception must be made that recently, even when out of the attack, he has shown a tendency to be inert, listless, and rather stupid. This may be in part due to the large doses of bromide he has taken. This drug tempers the severity and shortens the course of the attacks.

To connect the symptoms with the injury seems unavoidable. Prior to the accident the man had shown no mental symptoms, and did not drink. He had had no serious disease, and never had presented any epileptic manifestations. He had established and successfully carried on a paying milk business. Insanity or other neuropathic factors in this family history were not demonstrable. At the time of the accident he received an injury to the head which left a scar about two inches long. The local symptoms referable to this have always been pronounced. Headache, referred to the injured spot, has been constant, both during and between attacks. The pain has always become worse before attacks, and pressure on the scar would bring on an attack. His physician, Dr. S. J. Keefe, of Elizabeth, to whom I am indebted for most of the history, excised the scar, finding a depression in the bone three inches in diameter. Although he strongly urged it (as was eminently indicated) he could not obtain consent to trephine or explore the skull further.

At the time of my physical examination the patient was in an attack. He was moody, fearful, suspicious, and inclined to be violent if troubled too much. The pupils were dilated, but responded to light and accommodation. The knee jerks were active, but there was no clonus and no paralysis. There was a coarse tremor of the tongue, and a tremor of all extremities, increased by movements. In some attacks this tremor became so pronounced that the patient fell when he attempted to walk. Between attacks it was absent.

2. Traumatic Focal (Jacksonian) Epilepsy.—The term Jacksonian or focal epilepsy indicates irritation of the cerebral

cortex by a focal lesion. When the cortex is subjected to local irritation it is normal for the muscles supplied by it to be thrown into spasm. From this point of view Jacksonian epilepsy is a symptom, and not a disease. But, in traumatic cases at least, the spasms have a tendency to recur and ultimately to become general, and to affect the individual in much the same way as general epilepsy does. So traumatic Jacksonian epilepsy merits a separate clinical description.

Pathology.—Our knowledge of the pathology of Jacksonian epilepsy is much more definite than that of the idiopathic form. It has been carefully studied by Van Gieson. There is frequently a fracture of the skull so that splinters of bone press upon the cortex; or without fracture there may be an exostosis beneath the site of the original injury. The membranes may be adherent to one another, and thus form the starting point of wedges of connective tissue which grow downward at the expense of brain tissue. The nerve cells in the affected area are found in various degrees of degeneration, or they may have entirely disappeared, having been replaced by islets of neuroglia which have developed in the course of the arteries that pass perpendicularly from the surface into the brain cortex. These morbid changes are fairly constant in Jacksonian epilepsy, and are sufficient to permit the disease to be regarded as due to organic cerebral lesion; but in furnishing a pathological anatomy they do not explain the paroxysmal nature of the symptoms. If the cerebral lesion is sufficient to cause paralysis, that symptom can very easily be understood by remembering that injury to the cortex has injured or killed the cells which are the essential factors in voluntary motion. But why cells, whether they do or do not retain the power of causing voluntary movement, should from time to time become the seat of irritation and thus cause convulsions, remains unexplained.

ÆTIOLOGY.—In the ætiology of Jacksonian epilepsy predisposition is less important than in the general form. It is probable that in the predisposed the tendency of the convulsions to become generalized and progressively worse is more pronounced. But the first Jacksonian seizure occurs independently of predisposition. Males, in the active periods of life, are the most liable to severe head injuries, so they furnish the larger number of cases of Jacksonian epilepsy. Blows on the head with clubs or heavy instruments, striking the head in falls or in other ways, are not uncommonly followed by the disease. In most of the cases the skull is fractured, either in the internal or external table, so that the brain is directly pressed upon. But in some cases Jacksonian epilepsy apparently develops as a result of an injury to the head which has not fractured the bone.

Thus, in a case reported by Lloyd and Deaver, the patient was struck on the right side of the head at the age of sixteen; five years afterwards he began to have fits, which commenced with numbness and spasm in the left arm, extending to the left side of the face; consciousness was not often lost during the attack. The skull was trephined over the hand and arm center; there was no fracture, and the brain and meninges appeared normal.

Similarly, in the case of Starr's, the patient was hit on the left side of the head with a sand bag, and was rendered unconscious for twelve days. The skull was not fractured, as was proved by operation, but in three months the patient began to have attacks of aphasia and convulsions in the right hand.

Pistol-shot wounds, when not fatal, may be the starting point of focal epilepsy. In such cases it is difficult to tell whether the bullet, or the fragments of bone it carries with it, or the resulting scar, is the immediate cause.

Thus a previously healthy young man was accidentally shot by a pistol, the .22-caliber bullet entering the right eye. The eye was enucleated, and a piece of the orbital wall was extracted from the brain. The bullet was not found. Left hemiplegia developed, and

in two years recurring Jacksonian convulsions began and became general.

Abscess formation, so common after bullet wounds, may first manifest themselves by Jacksonian attacks months after the injury.

INTERVAL.—The fits may ensue almost immediately, or may be separated from the occurrence of the original injury by a period often varying from months to years.

Thus in a case of Starr's, a boy eighteen years old, previously healthy, received an injury of the right parietal bone by being struck on the head by a heavy block of wood. In three weeks he began to have frequent attacks, which were characterized at first by tingling and numbness in the left hand, a sensation which extended up the arm to the shoulder, and then down the trunk and leg, never involving the face. The subjective sensations were followed by twitching of the muscles of the arms without involving other parts and without being accompanied by loss of consciousness. The attacks lasted about a minute, and between them there was neither paralysis nor anæsthesia. It was not possible to determine by palpation of the scalp whether the bone was fractured or not; but an operation showed that there was a linear fracture of the right parietal bone, and that immediately over the hand center there was imbedded in the dura a splinter of bone one inch long and three-quarters of an inch wide.

In contrast to this case, which developed so quickly, is another one, equally typical of Jacksonian epilepsy, but in which the fits did not appear until three years after the accident.

A man, twenty-four years of age, sustained a fracture of the skull on the right side about the middle of the coronal suture. He recovered from the acute effects of the injury, but three years later began to have epileptic attacks. The fits began with a movement of the left hand and with a turning of the head to the left; the patient then lost consciousness, and the convulsion became general. Operation over the right arm center showed that a splinter of bone was indenting the dura, that the dura was thickened, and that the brain was yellower and more œdematous than usual.

Some cases have been reported as Jacksonian in which the

time elapsing between the occurrence of the injury and the first appearance of convulsions has been much longer, being five or ten or even a greater number of years. In such cases, inasmuch as the fits are almost always general, it is more difficult to prove the causal relationship of the said cause to the convulsive symptoms. So long an interval, however, is unusual. Of forty-four cases of traumatic epilepsy which were operated upon (collected by Mason for Gray), most of the fits began in a few weeks or months after the injury. Five cases appeared after an interval ranging from three and a half to fourteen years, but in all of these, except one, the convulsions were general and the influence of the trauma was consequently obscure. The exception is the case of Lloyd and Deaver, to which reference has already been made.

In the interval between the accident and the appearance of the first fit the patient may be perfectly well after recovery from the acute effects of the injury. If the motor tract has been directly injured, there may be hemiplegia; or, of the dura has been irritated, there may be the headaches characteristic of pachymeningitis.

Thus a patient at the Vanderbilt Clinic, previously healthy and without nervous predisposition, when a boy of eleven years of age was thrown from a wagon, striking his head. Since that time he has had severe generalized headaches and *petit mal* attacks, both of which later became frequent. The physical examination was negative. There were no scars or evidences of fracture of the skull, no paralysis or other indication of disturbance of the motor tract.

Usually, however, in the cases which give no sign of direct organic injury, the latent period passes without symptoms.

Symptoms.—The most common history of Jacksonian epilepsy is somewhat as follows:

A man, previously healthy, is struck on the head by a heavy instrument, or falls so that his head receives a severe

blow, which inflicts a scalp wound. He becomes unconscious, and may suffer for some time from the symptoms of cerebral concussion, or from those of compression. He eventually recovers, and finds that one side of the body is paralyzed. The paralysis usually rapidly improves, so that when the patient is finally examined for it nothing remains except a comparative one-sided weakness or stiffness, or a unilateral increase of the deep reflexes. Some weeks or months after recovery from the effects of the accident the patient begins to have convulsions in some portion of the body opposite to that of the head injury. The twitchings, which are usually preceded by a feeling of numbness or tingling in the affected part, begin in a few muscles and then spread to others. They are expressive of irritation of a localized portion of the brain cortex, and are called, after their original describer, Jacksonian. In the early stages of the disease the fits often remain local, but they have a tendency

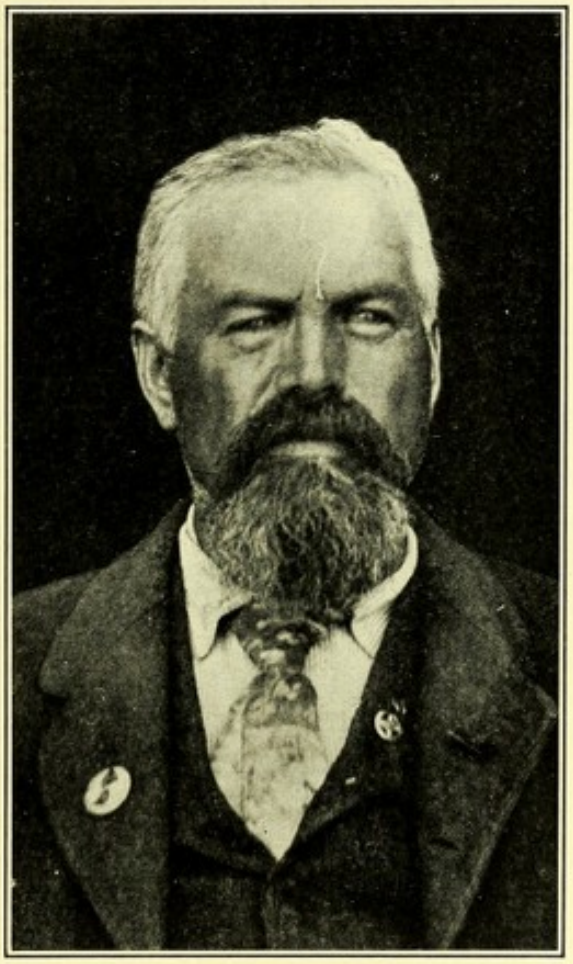


FIG. 22.—Case of traumatic Jacksonian epilepsy. At age of 44, fracture of frontal bone. (Shown by white line.) Two months later, Jacksonian seizures in right side of face. Later the seizures became general. (Craig Colony.)

to progress, so that they eventually involve the whole body, although usually the attack continues to begin in the part first affected. Consciousness is not lost in the earlier attacks. The tendency of such a case, however, is in all respects progressive, so that after the disease has existed for a number of months or years the attacks become exactly similar to the *grand mal* of idiopathic epilepsy, with the possible exception that they may continue to begin as a localized spasm.

On examination of the head there may be found the scar of the original scalp wound and a depression in the skull. The scar is usually not tender, and pressure upon it does not cause a fit. The skull may be fractured, however, even on its convexity, without leaving any sinking in of its surface. When a depression exists, the injured portion of the brain is not necessarily immediately beneath it. The bone may be splintered, or hemorrhage may have occurred in such a way that the cerebral lesion is situated at some distance from the site of the external injury. The frequency of this occurrence is fully recognized by surgeons, who, in operating for traumatic epilepsy, open the skull at a point opposite the portion of the cortex which supplies the muscles that first become the seat of spasm, instead of being guided by the depression in the bone. They follow the medical rather than the surgical indications.

The convulsions of Jacksonian epilepsy begin in single muscles or groups of muscles of the face (including those of the eyes and neck), of the arm, and of the leg. They may at first remain localized, but tend to spread. From the face they spread to the arm and then the leg; from the arm, to the face and then the leg; from the leg, to the arm and then the face. If the spasms pass to the other side they become general.

The convulsions are tonic and then clonic, and last from a few seconds to several minutes, or may last as long as two

hours. In the partial attacks, unconsciousness and the post-epileptic stupor are lacking; but the muscles which have been the seat of the spasm generally show marked weakness for some time after the attack.

Jacksonian attacks may succeed each other at brief intervals, or be separated by periods of months or even years. In some cases the seizures are general, with loss of consciousness, from the outset. This occurs even when there are definite symptoms indicating focal injury, such as a fracture of the vault with hemiplegia of the opposite side. It occurs also, with special frequency, in pistol-shot wounds which have produced no focal symptoms. The following case offers an example of this:

A man, thirty-nine years of age, without predisposition, was shot at the age of twenty-five by a .22-caliber pistol, the ball entering the left frontal region. Four years later he had, during three days, twenty or thirty attacks of momentary unconsciousness and rigidity of the muscles, without falling. He was then free from attacks for eight years. But at the end of that time regularly recurring general attacks began and persisted. Between attacks the patient had no symptoms.

The typical symptoms of Jacksonian epilepsy are subject to variations. The fits may take other forms than those of muscular spasm. *Petit mal* is a common and sometimes the sole symptom. Attacks of aphasia or of headache may be substituted for convulsive phenomena, as is shown by the after-history of Case VI of Starr's "Brain Surgery":

J. R., aged forty, was struck upon the left temple and sustained a fracture of the skull in August, 1889. When he recovered consciousness he was found to be paralyzed upon the right side and aphasic. In the course of the following six months the hemiplegia slowly subsided and the speech gradually improved, so that he was able to go about, but was still unfit for work. About a year after the accident he began to have convulsions; some of these were gen-

eral, with loss of consciousness, but later they became localized and remained so for some time. They increased in frequency until, when seen in December, 1892, the patient was having several every week. The attacks began with a twitching of the muscles about the mouth upon the right side; a drawing of all these muscles toward the right, with twitching of the eyes, and a gradual extension of the spasm to the right side of the neck and to the right arm and hand. During the attack he did not lose consciousness, but he could not speak, and experienced a sensation of tingling in the face and mouth. After an attack he appeared to be weak and was not able to talk as well as before. Examination on December 10, 1892, demonstrated a slight paresis on the right side of the face, the tongue not deviating, and some weakness in the right arm, but no affection of the leg; no disturbance of sensation; increased reflexes upon the right side. A depressed fracture of the skull, running backward two inches about over the position of the Sylvian fissure, was evident upon palpation. The posterior limit of the fracture was an inch below the location of the motor area of the face. At the operation a considerable amount of injury to the meninges and to the brain substance was found on the left motor region. The patient was trephined four times.

A note made by me on January 22, 1897, four years later, in the Vanderbilt Clinic records, says that "since the last operation there have been no convulsive seizures, but six or seven times a year the patient has attacks of headache which last from five to six hours. The pain is intense and consciousness is sometimes lost temporarily. During one of these attacks the patient attempted suicide on account of the pain."

When the epilepsy remains focal there is much less blunting of the intelligence, and less tendency to the development of psychoses, than when it is general.

Course.—The course of Jacksonian epilepsy is progressive. The tendency is for the fits to become uninterruptedly more frequent and severe, and to be coupled with a constantly increasing failure of mental power. In some cases the disease appears to remain stationary, and very rarely the fits are said to have ceased altogether, either spontaneously or through the influence of drugs. However, until very recent years recovery

was practically hopeless. With the advances which have been made in cerebral surgery the prognosis has become somewhat more favorable. Operations have in certain cases been the means of lessening the number and severity of the attacks, and a few patients appear to have been permanently cured by the removal of sources of cortical irritation. But the results of trephining for epilepsy, although tangible, have not been sufficiently brilliant to warrant the disease being regarded as anything but a very serious affection, which in by far the larger number of cases is incurable. The chances for successful treatment are greatest when the operation is done immediately after the accident; then, by the immediate removal of splinters of bone or blood clots, the brain will be irritated for as short a time as possible. In any case in which the fits have once appeared and existed for any length of time the hopes for recovery are slight. The disease in itself is not a direct menace to life, but its victims are in constant danger of accident. They are "risks" which life-insurance companies regularly refuse to accept.

3. Traumatic Reflex Epilepsy.—Under this term are grouped cases of recurring convulsions, the results of peripheral injuries. They are extremely rare, and doubt is often expressed as to the injury being more than a coincidence. I have never myself made the diagnosis of traumatic reflex epilepsy.

The records of the Franco-Prussian War showed 17 cases of epilepsy developing among 77,461 persons injured in the trunk or extremities.

Siemerling says such cases are particularly frequent after injuries to the scalp, with adherent scars. They are reported also after injuries about the neck and to the nerves. The scars and wounds are painful, and are the seats of auræ which precede the fits. The fits are general. Cures have been reported by excision of the scars.

Treatment of Traumatic Epilepsy

The treatment of traumatic epilepsy is medical and surgical. The medical treatment consists in the administration of antispasmodic remedies, notably the bromides. More important are general hygienic measures such as outdoor life, simple occupations, attention to general health, and, whenever the patients are able, plenty of muscular work. The ideal of treatment is colony life. The surgical treatment consists in elevation of depressed bone, excisions of pieces of diseased cortex, and the removal of foreign bodies. It is only useful in recent cases, and then only when followed up by careful medical treatment. For reflex epilepsy, it is recommended to remove peripheral sources of irritation.

CHAPTER IV

GENERAL PHYSICAL AND MENTAL RESULTS OF HEAD INJURIES

Pathology—Frequency—Traumatic Cerebrasthenia—Symptoms—Traumatic Insanity—Direct and Auxiliary Causes—Primary Traumatic Insanity—Traumatic Defect Conditions—Treatment.

Pathology

After head injuries there may remain cicatrices in the scalp, with extensive scar formation. The skull may show no evidences of bygone trauma, or there may be depressions, defects of various degrees, splintered bone, osteitis, or exostoses. The membranes are frequently fused to themselves, to the bone, and to the brain, and may be formed into cysts. The brain may show foci of sclerosis, especially in the tips of the frontal and parietal lobes, the seats of election of diffused traumatic hemorrhages. It may also contain cysts, or loss of substance with scar formation. Arteriosclerosis is a frequent finding. Microscopically, the brain may show degeneration in cells and fibers. Whether these lesions are progressive, with the tendency to extend to other parts of the brain than those actually injured, or whether, after the original œdema disappears, the blood absorbed, the scar formed and the resulting degeneration in fibers and cells completed, they remain practically stationary, is a question pathology has failed as yet to answer. Clinical evidence favors the latter alternative, although in a few rare cases, as that of Frost's, already referred to on page 117, a progressive degeneration sets in years after the original injury.

Any of the above-mentioned pathological conditions may be found when mental symptoms, during life, were absent. So there is no characteristic pathology for the late clinical effects of brain injuries.

The purely surgical after-results of injuries to the head are not of great importance. Cicatrices of the scalp occasionally are painful and require operation. The defects in the bone, even when large enough to render visible the pulsation of the cranial contents, rarely give trouble. But after every severe injury or commotion of the brain there result changes in blood supply, in pressure, and in the constituent parts of the brain. These changes can, and often do, pending the brain's readjustment, cause a variety of annoying symptoms, such as headache, dizziness, irritability, etc., which begin shortly after the return of consciousness, but which are chiefly noticed when the patient resumes his normal vocation. These symptoms must be regarded as incidental to the process of repair, and consequently reckoned as part and parcel of the original injury. In the great majority of cases they gradually disappear, so that the patient is practically as well as before the accident. In a few they remain stationary or get worse, or are complicated by others of a more serious character. As a general rule paralytic symptoms are at their worst during the period of acute symptoms, and from then on improve. Traumatic hemiplegia usually recedes rapidly, the restoration of power and balance becoming more complete than is the case with hemiplegia of apoplectic origin. Some increase in tendon reflexes almost always remains. Destruction of specialized areas of the cortex (e. g., speech center) are permanent. The results of injury to the cranial nerves have already been given.

It is not safe to give a bad prognosis as to the ultimate mental state in these cases too early. After severe injuries a year at least is necessary for full restoration, and a bad prog-

nosis given before that period may easily prove erroneous. Thus in a case described several years ago, a patient sustained a fracture at the base, with left hemiplegia and ptosis on the right side. The physical symptoms disappeared in a few weeks, but the patient remained slovenly, unable to care for himself, to talk intelligently, or to understand what was said to him. He was seriously demented and improvement seemed improbable. After a few months, however, these symptoms also passed away, and the patient's mind cleared sufficiently for him to leave the hospital.

I also have records of the case of a child who, after a fracture at the base, showed defects in memory, in concentration, and behavior which persisted for several months, but which then disappeared, so that the child became as well as before the accident.

Frequency

Available facts regarding the general late effects of head injuries are not voluminous. Of the after-histories of 15 patients, with fracture at the base, published by me (the inquiry being made from one to two years after the injury), in 10 there was complete recovery, except in so far as complete restitution of parts originally paralyzed had not taken place. In 5 there was complaint of increased nervousness and irritability.

Bullard obtained the after-histories of 19 cases of fracture of the base. Of these, 16 made apparently perfect ultimate recoveries. In 2 patients who were alcoholic there was a greatly increased susceptibility to alcohol, and symptoms of irritability and violence. In one case, a schoolboy was reported as being unable to learn since the injury.

Important information on this subject has been contributed by English. Of 200 cases of head injuries consist-

ing in fracture of the skull or contusion or laceration of the brain, after a year or more after the injury he found no effects in 79, slight effects in 92, and marked effects in 29. Under marked effects are included the physical symptoms of dizziness, headache, etc., in addition to purely mental symptoms. Of 164 patients in whom he obtained records as to working capacity, he found 115, or seventy per cent, doing the same work at practically the same wages. Nineteen patients were doing lighter work, and 11 were only able to do light work. Ten, engaged in trades demanding balancing power (workers on ladders, cabmen, etc.), had given up their trades chiefly on account of dizziness. Nine were totally disabled. In 8 of the 300 cases, mental symptoms amounting to insanity existed.

For evident reasons, the question of post-traumatic cerebral symptoms concerns chiefly men in the active periods of life, engaged in the heavier kinds of manual labor.[1] In the aged, permanent cerebral effects are more apt to follow. Children, on the other hand, usually make good recoveries unless there has been extensive brain laceration. The most important factor as regards the injury is its severity. Whether the skull is fractured or not seems to be a matter of secondary importance, except in so far as fracture serves as an index of severity of injury. A better index is the immediate mental symptoms. Depressed or comminuted fractures of the vault are less apt to be followed by late results if operated on early. In how far laceration or destruction of the brain by other means affects the permanent mental symptoms is rather difficult to say. Certain it is that large portions of the brain may be destroyed without affecting mental capacity. There is very little satisfactory evidence that injuries of any one part of the

[1] Brain injuries and consequent mental defects from accidents at birth are left out of account.

brain are especially apt to be followed by mental defect, although Phelps believes that injuries of the left frontal lobe are especially apt to be followed by intellectual and emotional disturbances.

These late symptoms of head injuries may be divided into three classes. The confines of any one class cannot be sharply drawn, as they overlap and merge into one another. But all may be included in the three classes which are: (1) Traumatic cerebrasthenia. (2) Traumatic insanity. (3) Defect conditions.

Traumatic Cerebrasthenia

The term cerebrasthenia is comprehensive, as it is made to include certain physical affections, such as dizziness and headache, together with changes in mental make-up, which, while they may not merit the term insanity, render the patient different and less capable than before. In a general way the term refers to the condition of nervous ill health in which most survivors of head injuries find themselves for some time after the injury. Chief among the symptoms are headache, dizziness, irritability, ease of fatigue, change in character, and intolerance of alcohol.

Headache.—The patients frequently complain of peculiar sensations in the head, such as feelings of numbness, or tingling, or flushing, or constriction, or dullness, or actual pain. Pain most frequently is dull and more or less constant, similar to neurasthenic headache. Again, it is of a character to suggest definite focal disease. Under such circumstances it is localized, more or less paroxysmal, and radiating in character. There may be tenderness to pressure over the seat of the pain. Pain of this character may be due to bone disease, or to exostoses, or to meningeal adhesions, and often merits operative interference.

Dizziness.—Dizziness, with or without faintness and nausea, coming chiefly in attacks, is a particularly annoying symptom. It is brought on or intensified by stooping or sudden movements, especially when there is loss of substance in the skull. Recurrent dizzy attacks are extremely common; they usually pass away in time and in themselves are entirely insufficient for the diagnosis of epilepsy. They may be sufficiently severe to debar the victim of them from engaging in occupations which require good balancing power, such as working on heights, etc.

Irritability.—An increased irritability is frequently spoken of as a result of head injuries. Boys become quarrelsome and pugnacious. They lose their temper readily or fly into unreasonable rages. Kaplan has given the name "explosive diathesis" to the states in which the patients have sudden outbreaks of anger and even violence, during which they are irresponsible. These explosive periods generally follow the use of alcohol. Their similarity to psychic epilepsy has already been referred to. In contrast to such states are others in which the patients are depressed, silent, and moody, but only irritable when disturbed.

Sleep.—Sleep is apt to be imperfect and disturbed by horrible dreams, which may or may not relate to the accident. Somnolence occurs, though it is a rarer symptom. Somnambulism has been observed in children.

Physical and Mental Endurance.—Endurance is regularly lessened for a time after head injuries. The patients find they cannot do the physical work they did before the accident, or else only with great fatigue; mental labors are particularly exhausting and fatigue of any character makes all symptoms worse. These patients are worse in hot weather, and high temperatures are not well borne. There is also a general nervousness, the patients becoming upset and disturbed by trifles,

and unduly sensitive to all sensory stimuli. Such conditions seriously impair working capacity.

It has been maintained that persons who have received head injuries are more subject to tuberculous meningitis, to cerebro-spinal meningitis, etc.

Memory.—In the majority of cases there is no affection of the memory, except as to facts immediately connected with the accident. In others, there is a memory loss similar to that encountered in neurasthenia. The patients are forgetful through inattention and so make mistakes, and get in the habit of writing down what they are to do. In other cases there seem real defects in memory, notably for names. In some cases the arithmetical faculty is reported as diminished.

Change in Character.—The symptoms already mentioned imply a change in character. In addition some patients undergo further changes. They become moody, eccentric, they are irascible and hard to get along with; their tastes change, a sober man takes to drinking, a cautious man becomes reckless, they are prone to fall into the hands of quacks, or to become adherents of social or religious extravagances. Changes of this latter character are rare and usually late in appearance, so that it is difficult to say what other factors besides the injury are accountable.

Tolerance to Alcohol.—An almost universal and frequently permanent effect of head injuries is a lessened tolerance to alcohol. Intoxication occurs more easily than formerly, and the other symptoms are made worse by drinking. Also, the symptoms just enumerated are more pronounced and more frequent in alcoholics. In the poorer classes the idleness enforced by convalescence furnishes in itself a temptation to intemperance, and many of these are cases of alcoholism quite as much as of traumatic cerebrasthenia.

Such symptoms as have now been described usually pass

away in a few months, although after severe injuries one or even two years may be necessary for complete restoration to health. A certain number of patients remain eccentric, peculiar, uncertain in their business transactions, unstable in their purposes, and prone to take up fads.

In persons predisposed to insanity the symptoms of traumatic cerebrasthenia, instead of getting better, pass into those of other well-recognized forms of mental disease, such as paranoia, melancholia, one of the forms of manic depressive insanity, dementia præcox, and the like.

Traumatic Insanity

Most of the statistics on the subject of insanity as a result of head injuries are obtained from cases already in asylums. To accept such statistics as evidence for a traumatic origin of insanity is misleading in the extreme. The families and friends of insane patients are ever eager to avoid the stigma of inheritance, by advancing accidents, no matter how trivial, as causes of the mental disease; histories obtained from the patients themselves are manifestly unreliable. So routine asylum statistics which show percentages reaching as high as ten per cent cannot be accepted.

An exception must be made for the statistics of Meyer, which are compiled with especial care. Of 3,000 admissions, he found 23 cases, or less than one per cent, in which mental symptoms followed a more or less severe traumatic injury. But a close analysis even of his cases shows that the evidence that the insanity resulted from injury to the head, unassociated with other causal factors equally important, is meager. From Meyer's statistics it would seem that trauma capitis is the chief cause of insanity in less than one-half of one per cent of the cases.

A more reliable method of investigation consists in obtaining the after-histories of cases of head injuries. Werner collected the following relative to the frequency with which insanity follows head injuries:

Reported by.	Number of head injuries.	Number of patients who became insane.	Variety of injury.
Wagner	96	3	Fractures of the skull.
Kohler	193	0	44 severe injuries with 22 defects.
Kramer	173	0	Very severe injuries.
Stolper	981	12	138 severe complicated and uncomplicated fractures and severe commotion. In the 12 cases of mental disease 11 had severe head injuries.
Report of German army, 1870–71	8,985	13	Injury or commotion of the head.

These statistics of Werner's, taken together, show that about one-fifth of one per cent of head injuries are followed by insanity. Of the 15 cases of fracture at the base reported by me, in one only were the primary mental symptoms protracted, and in that one perfect recovery occurred after a few months. I endeavored to correlate the number of head injuries occurring in New York City with the number of patients in the city insane asylums having a distinct traumatic history. Taking fracture at the base of the skull as an easily recognizable form of injury, and as a reliable index of serious commotion of the brain, I estimated that 150 recovered cases of this injury were discharged every year from the various hospitals in New York. If insanity were an important consequence of such an injury we would be certain, in view of the class of persons involved, to find evidences of it in the New York City asylums. Yet such evidence is wanting.

Of more than 2,000 patients at the Manhattan State Hospital, East, over three-fourths of whom are men, in no case is there history or evidence of fracture at the base. It may be

said, incidentally, that there are but two cases of fracture of the vault. In one patient, depressed, hypochondriacal, and controlled by hallucinations of hearing, the symptoms developed eighteen months after injury; in another, probably paranoia, nine years after the injury. It is probable that the percentage of 1 person in 1,000, as shown at Manhattan State Hospital, East, is not greatly in excess of the percentage of skull fracture, of both vault and base, in the general population. The Central Islip branch of the Manhattan State Hospital is chiefly a service of chronic cases, most of the patients, about 3,000 in number, whose insanity is of long standing, having passed through the Ward's Island institutions. Histories in these cases are consequently somewhat indefinite. Dr. C. A. Smith, superintendent of the hospital, has kindly sent me the following *résumé* of the cases in which trauma figures:

Total number of cases with traumatic history.....	14
Number of cases with no external evidence of injury...	8
Number of cases with scars or depressions over vault...	6

In none of the cases is it possible to make a diagnosis of fracture at the base.

There is no reason to suppose that the mortality in persons who survive the acute symptoms attendant upon fracture of the base is particularly high, and the majority of such persons, being of the indigent class, would be obliged to go to the State hospitals for their district did they become insane.

Therefore, while freely admitting the possibility of error in computations of this character, the apparent absence of such cases from the State hospitals would indicate that fracture, at the base at least, was almost a negligible quantity in the ætiology of insanity.

From the foregoing it seems reasonable to believe that the vast majority of patients who survive the immediate effects of severe head injuries make substantially complete recoveries as

far as their mental state is concerned; and that serious mental symptoms persist in considerably under one per cent.

Direct and Auxiliary Causes.—In every case of supposed traumatic insanity it is imperative to inquire as to the predisposing and auxiliary causes, which so often outrank in importance the injury itself. The most prominent of these are hereditary predisposition, alcoholism, general somatic diseases, epilepsy, if it develops, and the mental or nervous shock with which the injury was associated.

Hereditary predisposition makes itself noticed in two ways: first, that mental symptoms in sequence to injury are much more frequent in the predisposed; and, second, that mental symptoms in such persons may first appear after injuries which in themselves are certainly too trivial to produce them. In such cases the injury to the head is often not of great severity. Guder has especially described these cases and shown the triviality of the blows as compared with the symptoms which followed them. Emotional factors at the time of the accident were often pronounced, hence the great excitement, the brooding anxiety, and hysterical characteristics. The patients give the history or present the stigmata of hereditary predisposition, and careful questions usually elicit the fact that prior to the accident they had shown mental symptoms. The physical injury, therefore, is only an auxiliary cause, and frequently an unimportant one. The number of these cases is considerable, and it is probably from them that most of the statistics as to traumatic insanity are obtained. The prognosis depends upon the fundamental disposition.

Alcoholism is an extremely weighty factor in all head injuries. In alcoholics the initial delirium is extremely active and apt to be prolonged. The mental symptoms which sometimes persist in alcoholics owe their existence to alcohol more than to trauma. The impulsive tendencies in traumatic neurasthenia

and in traumatic insanity are especially conspicuous in drinkers, and almost invariably show themselves in even slight degrees of intoxication.

General somatic diseases do not have a very important part in ætiology. Meyer reports cases in which, long after the injury, febrile attacks have brought out mental symptoms; and some cases are on record in which, when the patient was senile or had other evidence of degeneration of the cerebral arteries, a rapidly progressing dementia set in a few months after the accident. The effects on the mind of epilepsy following trauma are spoken of elsewhere. Psychoses sometimes develop in persons who have been crippled or disabled by severe surgical injuries. These hardly merit the name of traumatic as they are the results of brooding and despair.

Some patients who are suffering from or have suffered from traumatic cerebrasthenia, and in whom predisposition may not be demonstrable, years afterwards, under special stress, develop transitory mental disturbances. Meyer describes cases of this kind.

A man with marked family history sustained a fracture at the base at the age of twenty-two years. At twenty-five, after a blow on the abdomen, he developed possible hallucinations. At the ages of forty and forty-one he had two attacks of semidelirium, each of which was ushered in with inflammation of the respiratory tract.

A girl, several years after two cranial injuries, during an attack of *grippe* developed delusions with hypochondriacal depression and attitude of persecution with possible hallucinations and confusion of facts and of current events. Recovery.

The element of mental shock and fright does not enter in most severe cranial injuries, as in them consciousness is immediately lost. When consciousness is not lost, fright as a cause may be very important. The mental effects of fright will be spoken of on a later page. They may so closely resemble those

of physical injury to the brain that it may be impossible, with both factors present, to determine which one was the more active.

The important factor of the injury itself is often difficult to determine. We know that blows which cause unconsciousness have affected the brain; and that those which cause contusions and wounds of the scalp, or injuries to the cranial bones, may do so. Injuries of this character may be responsible for true primary traumatic insanity. The less severe the blow, the less probable it becomes that any mental symptoms which follow it are the results of physical injury to the brain; and the more on the alert the examiner should be for predisposition, for preëxisting psychoses, and for other auxiliary causes.

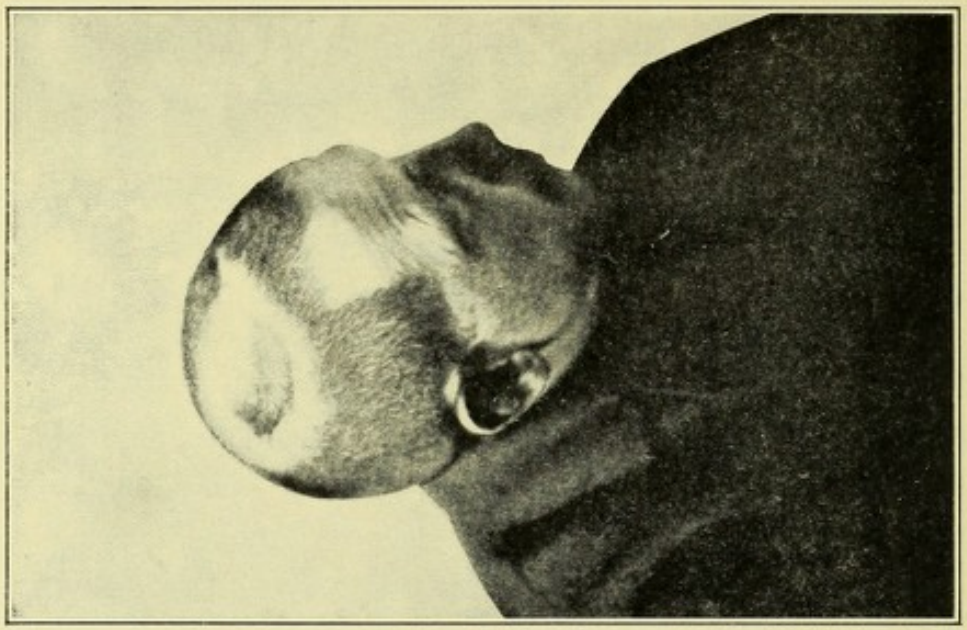
When reliable information regarding the injury is wanting, certain general facts must be relied on.

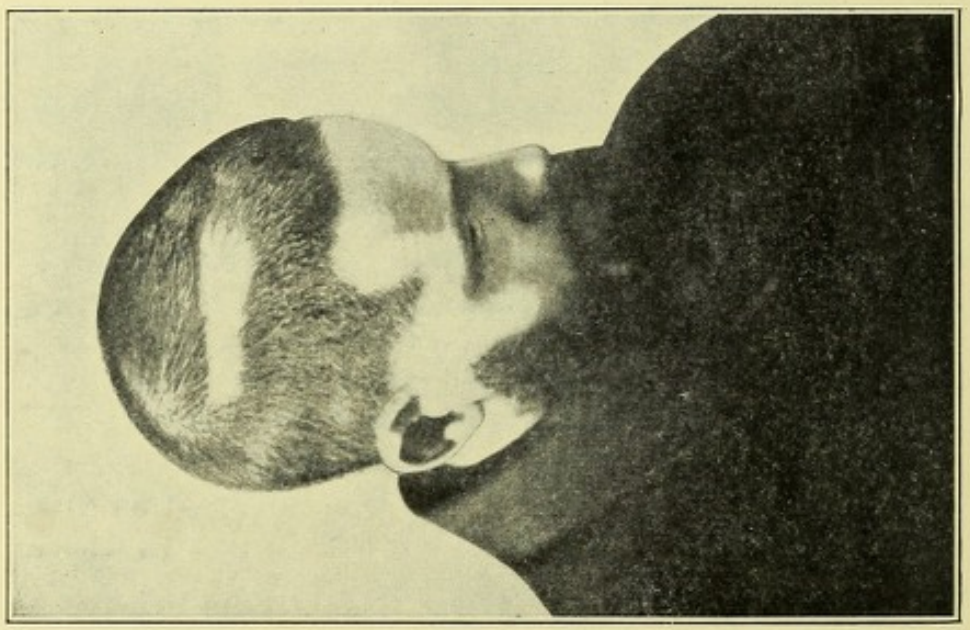
Although our knowledge of insanity is extremely imperfect, it has reached a certain point of practicality so far as head injuries are concerned. We recognize certain vesanic types, notably delirious and confusional states and dementia, as the legitimate results of injury. We are also almost in a position to deny that the systematized insanities, such as paranoia, or such emotional states as melancholia, ever result from traumatic causes alone.

Primary Traumatic Insanity

There are a few forms of mental disturbance worthy of the name insanity, resulting from injury to the head, and in which it can be shown that the patient was not predisposed, and that important auxiliary causes were absent. In these trauma is unquestionably the sole cause; and these types alone constitute true or primary traumatic insanity. The symptoms in these

conditions are of a delirious, confusional, and impulsive character. They may be continuous, constituting a fairly typical





FIGS. 23 and 24.—Cases of insanity following depressed fracture of vertex. (Manhattan State Hospital.)

though protracted delirium, the intervals of normal mentality being brief or wanting; or they may be distinctly periodic with almost normal intervals. In the persistent delirious forms the

head injury is invariably severe, with usually profound coma, followed by delirium. The initial delirium may remit, so that the patient is mentally clear for several days or weeks; or the original delirium may continue and persist, in milder form, after the patient is well enough to leave the bed.

The two following cases illustrate varying grades of this affection:

An old gentleman, about January 12, 1902, was caught between two trolley cars, was thrown to the ground and rendered unconscious. He was taken to the Roosevelt Hospital, where I saw him about one week later, in consultation with Dr. G. E. Brewer. The patient had been delirious and somewhat noisy since coming to the hospital. After being in the hospital a few days, he suddenly developed paralysis of the right external rectus.

The mental state at the time of my examination was soporific with impulsive tendencies. He lay in bed with the eyes closed, but could answer questions, although the answers were often not pertinent. The impulsive tendencies showed themselves by calling out and shouting, by suddenly trying to get out of bed, etc. These latter symptoms grew worse rather than better, so that the patient deteriorated mentally in spite of physical improvement. He was taken from the hospital in March, but was so irresponsible and uncontrollable that the question of commitment to an institution for the insane came up. On March 17, 1902, I examined him again at his home. He had no recollection of having seen me before, although he said that he knew me perfectly well, and that my name was Mr. Brown. He said that he had not been in a hospital at all, but that he had been staying in hotels and thought he had been away on some long journey, although he did not know where. He did not know where he was or what the season of the year was. With this lack of orientation, he would wander about by himself, would break the furniture and smoke incessantly. Physically he seemed perfectly well. With the exception of waking up at night and becoming excited, he slept well. Immediate commitment seemed inadvisable, and soon after this the patient began to improve, and in six weeks was perfectly well. His son, writing in January, 1903, says that as far as he can determine his father's intellect is now as good in every way as before the accident.

In February, 1903, the patient came to my office for examination.

I could discover no evidences of mental defect or perversion. His memory for the period of time that he was in the hospital and for a few weeks subsequently was imperfect and cloudy, although he remembered seeing me and some of the replies he made to my questions.

For the second case I am indebted to Dr. H. S. Noble, superintendent of the State Hospital at Middletown, Conn.:

E. N. B., painter, married, aged forty-six, admitted to the hospital May 21, 1891; duration of insanity, six months; alleged cause, trauma to head. The patient has drank moderately for some years. The present attack, which is his first, resulted from an injury received nearly six months ago. As he was coming downstairs in the hospital, where he was at the time employed, he fell headlong to the bottom. He was picked up unconscious, and lay confined to bed for several weeks. At the time of injury he bled profusely from the left ear and was quite restless, thrashing about and trying to get out of bed. He suffered extremely from headache and intolerance of light, and after consciousness was partially restored he was delirious for several weeks. As he gained strength and began to go about, his mind gave evidence of serious impairment. The expression of the face was changed and he was easily confused and bewildered, wandered about the city and neighborhood without special purpose, and often did not return home till late in the evening. He was irritable and fretful and abusive to his family, accusing his wife of having been unfaithful to him, and on several occasions he threatened her. His malady was increasing and without lucid intervals or remissions. Although on one or two occasions when under the influence of anger he threatened to drown himself, he nevertheless was not regarded as suicidal, nor was he destructive or filthy.

May 30, 1891.—Has thus far been quiet, cheerful, and contented. Seems slightly elated, but has expressed no delusions. Meets his wife pleasantly and cordially when she calls upon him.

June 1, 1891.—Is a trifle loquacious and shows an emotional instability. Consciousness is clear and he is completely oriented. Memory is unimpaired for both recent and remote events.

July 27, 1891.—Is given limited parole on the grounds, as he is now able to control his emotions much better than at first. Judgment is somewhat faulty, and his capacity for mental work is diminished.

September 1, 1891.—Is at present in a fairly comfortable condi-

tion, but he still shows some mental impairment, is a trifle silly, and self-control is lacking. Taken home by his son on trial.

Absent thirty-eight days.

October 9, 1891.—Brought back this evening with the corroborative report that he is no longer fit to be at large. He wanders about the streets constantly, drinking in the saloons, and is unable to fix his mind upon any kind of occupation. He is extremely irritable and abusive to his wife, and accuses her of having been unfaithful to him. Has occasionally threatened violence to members of his family.

From this date on the patient improved, and was discharged from the hospital in April, 1892. The following year he did light work at home, returning then to the hospital as a painter. The past ten years (January, 1903) he has worked steadily as a painter at the hospital, though there is still a slight degree of dementia manifest, as evidenced by dull comprehension, impaired judgment, diminished capacity for work, and slight emotional unstability.

These cases of protracted delirium present superficial differences in symptoms, but the fundamental conditions are very similar. Sleep is interfered with by dreams. Consciousness and orientation, at first seriously disturbed, become gradually reëstablished. But dream states persist in which the patients are quiet, not particularly depressed, inattentive, or in which they show anxiety. These dream states are apt to be interrupted by periods of great agitation, in which the patients under sudden impulses become excited and prone to commit acts of unreasoning violence, such as getting out of bed, rushing to the window, striking the attendants, etc. Impulsiveness and sudden changes in behavior are highly characteristic of traumatic insanity, and make constant watchfulness imperative in the care of these patients. Periodicity is also characteristic. A patient who at one moment talks rationally and coherently, at the next may be totally irrational and irresponsible. The statements of these patients are always to be accepted with reserve as there is a tendency to make misstatements, and also to invent whole stories. Delusions may occur, but they are not well systematized, and hallucinations are not prominent.

The physical symptoms present all the features of severe nervous shock. Vasomotor symptoms and changes in pulse rate and blood pressure are prominent. It is when the physical symptoms begin to pass away and the patient is able to be up and about that the case comes to be looked upon as distinctly mental. From then on the progress is usually toward betterment, with frequent relapses brought about by undue stimulation, excitement, or fatigue. At the end of a few months, in nearly all of the reported cases, substantial recovery has occurred. In some cases the characteristics of traumatic cerebrasthenia remain; in others, there is a mild degree of dementia, as shown by emotional instability, by defects in memory and judgment, and by loss of originating effort. Capacity for mental and physical work is also generally lessened.

In their periodicity, their impulsive periods, their tendency to fabrications, their dream states, these cases of protracted traumatic delirium have striking resemblances to the epileptic psychoses. The resemblance becomes still closer when the trauma has been directly to the cortex. Under these circumstances headache is a much more prominent symptom. It is often circumscribed to the region of the injury. An increase in the pain usually foreruns an accession of mental symptoms. Two cases of Meyer illustrate this.

In one of these a man, thirty-eight years old, cheerful, social disposition, was struck on the head by a fifty-seven-pound weight. He was not knocked down, but there was a hematoma of the scalp. Shortly afterwards the patient had attacks of headache and dizziness, and a few times he vomited his coffee in the morning. His character changed, he becoming sulky, irritable, intolerant to alcohol. He had attacks of headache during which he became very violent and even homicidal. During one attack he had delusions of persecution. About a year after the accident he was operated upon. Under the seat of a small scar on the right parietal eminence the remains of an old subdural clot were found and removed. After this the headaches and violence disappeared, but there remained undue susceptibility to alcohol.

A woman with a family history of insanity sustained, at the age of forty-eight, a severe cranial injury, although the skull did not seem to be fractured. She was stunned, but recovered and began to have attacks of a sensation as though she was whistling or whirling around, followed by staggering and loss of consciousness. There were also slight headaches. In the course of years she became more emotional, restless, sleepless, careless of lamps and lights, and she often declared that the people with whom she lived did not use her well. Ten years after the accident she was having occasional convulsions, and after these she was more emotional and dazed. Her memory was much weakened. She finally died in status epilepticus.

In some cases the resemblance to psychic epilepsy is so close that the case might be indifferently described as traumatic insanity or traumatic epilepsy. Such a case has been described on page 124.

Traumatic Defect Conditions

Under this heading fall conditions of interference with intellectual processes due to destruction of special or extensive regions of the brain. With this are included the mental disturbance consequent upon aphasia, those secondary to epilepsy if it develops, and the rare cases of dementia which result directly from losses of large portions of the brain. In these latter the dementia is not excessive, and is coupled with physical weakness. As examples may be mentioned the celebrated crowbar case, and the case of Frost, in which the patient lived for many years, though incapacitated, after the loss—through an injury—of a large portion of a hemisphere. Certain it is that marked dementia rarely, if ever, results from loss of brain substance alone, without the dementia having been long preceded by symptoms of perverted mental function.

Treatment.—While the patients are confined to bed, the treatment for all these cases is the same as for injuries to the head generally. Later, it becomes a question of management,

with avoidance of exciting and fatiguing influences. Most patients are inclined to return to work too early. After a severe injury several months at least should elapse before work is resumed. At the same time, as many of these cases resemble neurasthenia very closely, there is always the danger of hypochondriasis. Consequently, it is advisable for the patients to resume their occupations as soon as is consistent with general health. The impulsive tendencies should be borne in mind, and the patients be so protected that they can injure neither themselves nor others.

Headache is best treated by the ice coil or by the coal-tar products. The dizziness can be often relieved by small doses of bromide, combined with the fluid extract of ergot, taken four times daily. For insomnia, if it exists, full tepid baths (94° F.), of one-half hour duration, or cold packs are useful. If drugs must be resorted to, the following powder will be found efficacious:

℞ Codeine ..... gr. jss.–iij.
Chloralamid ..... ʒj.
Trional ..... gr. xxx.

M. Fiat in Chart. No. VI. div. Sig.: One powder at night.

For the general nervous symptoms outdoor life and occupation are the best remedies, though care is necessary that the patients do not become overfatigued.

Surgery has proved useful in some instances, either by the removal of depressed portions of bone, or exostoses, or blood clots.

The indications for surgical interference are external evidences on the skull, or localized pain, or paralysis, or convulsive phenomena.

Laplace reports two cases of successful surgery for trau-

matic insanity. In one, after a fall on the head, with secondary headache and a complicated psychosis, he demonstrated by trephination an exostosis on the injured spot. Cure resulted. Syphilis could be positively excluded. In the second case, in which dementia with tendency to falls resulted from a kick on the head, he found great thickening of the bone and hyperæmia of the dura. Recovery in three weeks.

CHAPTER V

INJURIES TO THE SPINAL CORD

Pathology—General Ætiology—General Symptoms—Focal Diagnosis.

THE degree of violence necessary to cause injury, either directly or indirectly, to the spinal cord is very great. No organ in the body is so strongly secured against danger. It is held in place above and below by attachments of the dura mater, and at the sides by its thirty-one pairs of nerves. Between the two membranes which surround it is a thick column of fluid that envelops it in its whole extent, and which acts as a cushion in dissipating the force of any shocks which may be received by the spine. The vertebræ (Fig. 25) through which it passes are thick and strong, are held in place by tough ligaments, and are everywhere padded by massive layers of muscles.

The accuracy and compactness of the articulation of its parts enable the spinal column to withstand great degrees of strain and force before its own integrity or that of the spinal cord is disturbed. Yet, notwithstanding the solid strength of the vertebræ, their nicety of adjustment, the massed layers of muscles, and the other efficient means of protection, the spinal cord may become the seat of serious injuries. But the injuries which overcome the barriers by which it is shielded from danger are the results of excessive violence that acts directly upon the spinal column.

Thus from severe falls or blows upon the head there may result spinal-cord injury, even though the skull escapes fracture. Falls on the buttocks, either from heights or as occa-

sionally occur in consequence of elevator accidents, may produce a like result. Penetrating wounds, or blows on the back from heavy objects, may be immediately followed by symptoms of impairment or loss of spinal function. By very severe

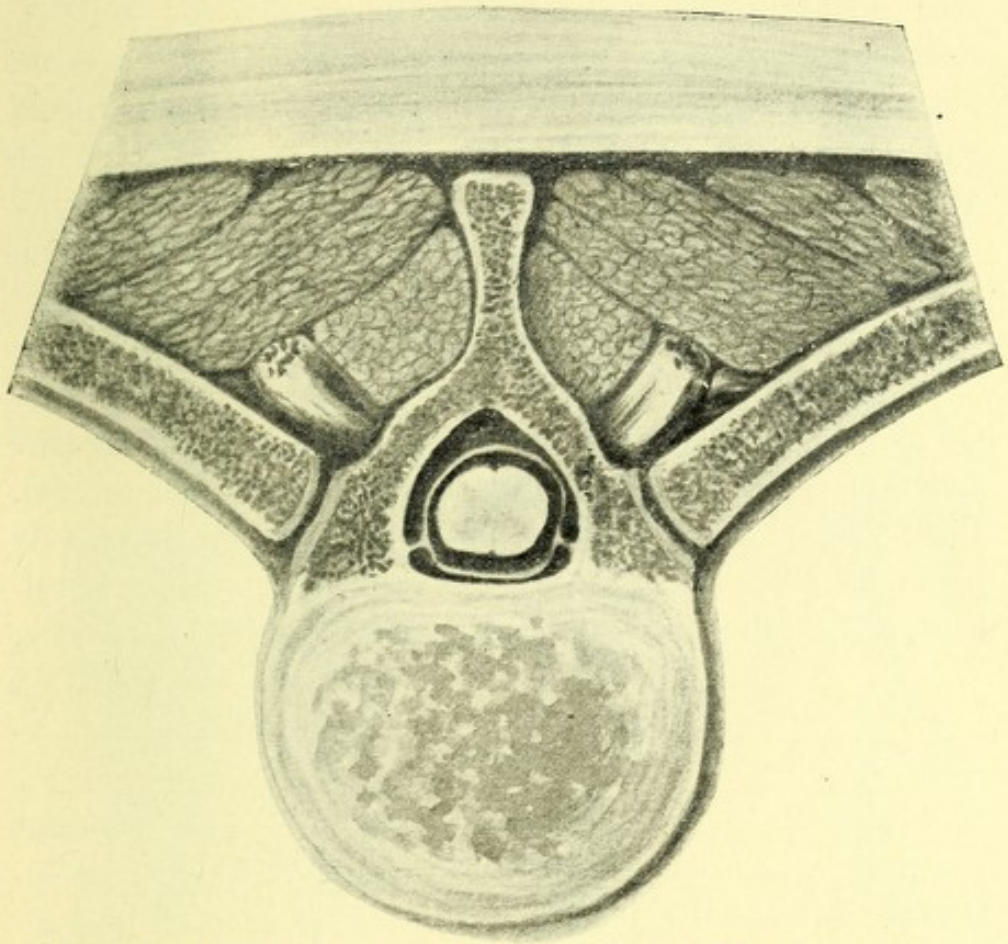


FIG. 25.—Transverse section of the structures surrounding the spinal cord at the line of the ninth thoracic vertebra.

and sudden twists or wrenches it is possible for vertebræ to be dislocated or for some of the intravertebral structures to be torn. But the violence which causes these injuries is extreme, and differs both in character and in degree from such violence as may be received by the sudden stopping or starting of a train, or from slipping on a banana peel, which is not infrequently advanced as a cause of "spinal" injury.

It is obviously difficult to determine the degree of force which has been exerted on the spinal column in any given accident. Yet, to prove the existence of an injury to the spinal cord, it is necessary to show that the force has in all probability been sufficient to break through the vertebræ or to cause stretching or laceration of the structures which they surround. Such proof is reënforced when the symptoms are in accord with those we now recognize as indicative of lesion of the spinal cord.

Although the diagnosis of the exact character of the lesion is often difficult, it is almost always possible to determine whether the spinal cord has or has not been injured.

Injuries to the spinal cord are comparatively infrequent. Of over 70,000 cases of general injuries reported by Wagner and Stolper, 0.71 per cent only involved the spinal column. Gurlt found them in 0.33 per cent of general fractures. As the cord escapes damage in one-third of the injuries to the column, it is readily apparent that traumatic cord lesions are rare.

Pathology

Contusion, laceration, and hemorrhage are the important features in traumatic lesions of the spinal cord and its membranes. Injuries to the cord membranes are of much less significance than injuries to the membranes of the brain. Their pathological interest concerns hemorrhage almost entirely. Tears and contusions, while frequent, are lost in the graver injuries to the cord itself.

Hæmatorrhachis.—As a result of rupture of the blood vessels of either dura or pia, hemorrhage outside the cord or extramedullary hemorrhage occurs in considerable volume and extent. The most frequent variety is epidural. The blood may attain the size of large clots (Figs. 26, 27). Clinically

this condition cannot be recognized. It is doubtful if it occurs as an independent affection, it being almost invariably associated with serious lesions in the cord itself. It practically never occurs independently of fracture. All the weight of pathological and experimental evidence is against hæmatorrhachis itself being the cause of any but transitory symptoms. Consequently, surgical operations for its relief are distinctly uncalled for. The same is true for subdural extramedullary hemorrhages.

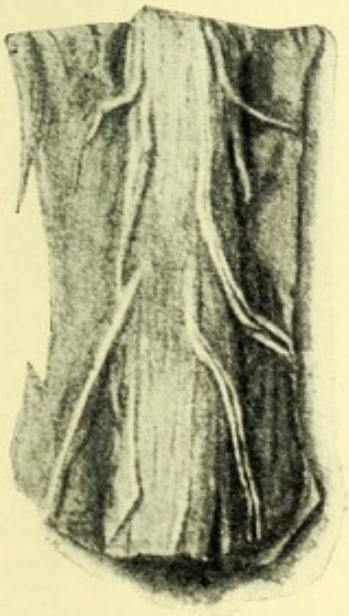


FIG. 26.—Intradural hemorrhage, associated with contusion of the cord.

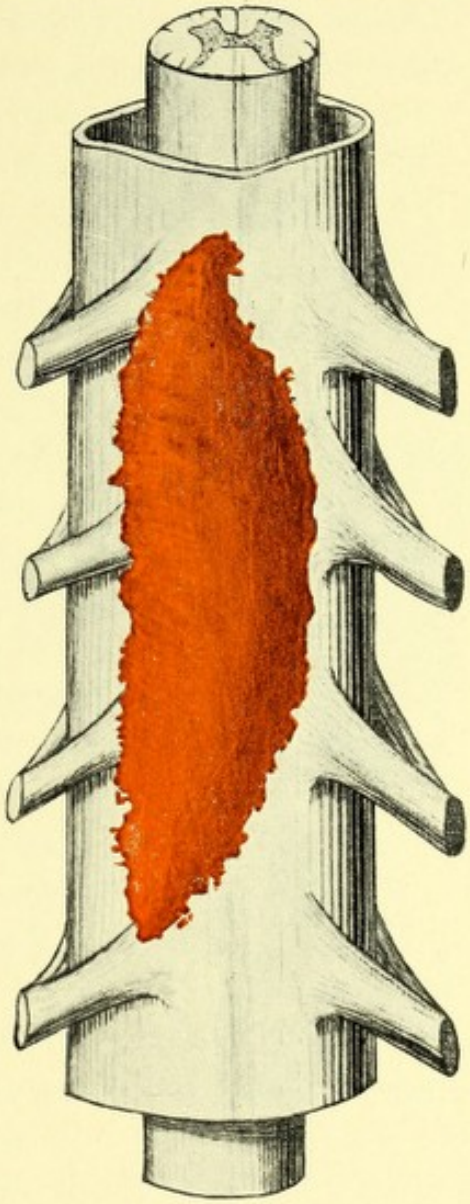


FIG. 27.—Extradural hemorrhage.

Contusions and Lacerations.—Contusion, laceration, and crushing of the cord are all of practically the same character, but vary considerable in degree. According to the extent of

injury, they are commonly called total transverse lesions and partial lesions. As there are no clinical symptoms which permit a total transverse lesion to be recognized as such during life, and as, unless the cord be actually divided, some fibers escape destruction in the most extensive mutilations, it seems wiser to content ourselves with dividing these cases into severe and partial.

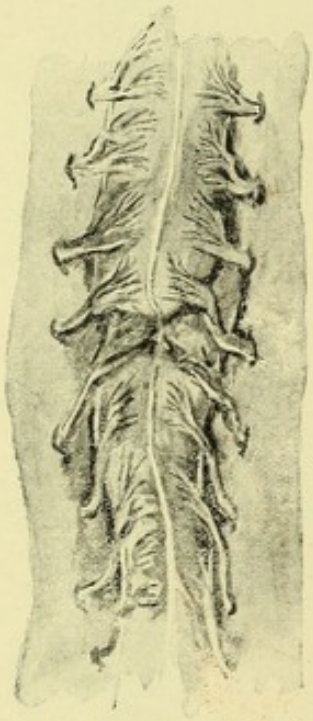


FIG. 28.—Crush of the spinal cord, due to fracture of the cervical vertebræ. (From a drawing kindly furnished by Dr. Van Gieson.)

The gross appearance of a contused or crushed cord varies. It may at first appear perfectly normal in color, contour, and consistence, the discoloration and softening only showing on section. Usually, however, there is a distinct transverse depression at the site of injury, with membranes and cord congested, and the cord showing yellowish spots of softening (Fig. 28). The disintegration in the cord considerably exceeds the point of actual compression. In severe cases the whole cord in the neighborhood of the injured spot is so softened that it can with difficulty be removed, *in toto,* from the canal. On section, the medullary substance is softened, the normal configuration of the gray matter is disturbed, and there are hemorrhagic extravasations in and about the place of greatest injury. When death is delayed, the softening gives place to scar tissue, so that the diameter of the cord is lessened. Microscopical sections, appropriately stained, show large myeline drops, red and white blood corpuscles, large epithelioid cells with granular bodies, broken and degenerated nerve fibers, and degenerated ganglion cells—the traumatic de-

generation. In addition, above and below the lesion, if death has been delayed for five days or more, may be seen ascending and descending degenerations.

Does the spinal cord regenerate? It is well known that intramedullary fibers can be bruised and contused and yet the symptoms which accompanied the original injury pass away almost completely. But to prove that the nerve fibers of the spinal cord, which have once been actually severed in their continuity, ever regenerate in a way to reëstablish useful function is much more difficult. In human cases, and in experiments, when the injury consists of contusion, it is impossible to determine whether the return of conductivity was due to actual regeneration and reuniting of divided ends, or to regeneration in fibers which had been bruised only. The latter alternative seems the more probable, inasmuch as in no animal experiment has it been possible to completely divide the spinal cord and then secure a restoration of conducting function. At the same time, in both sections and contusions of the spinal cord new nerve fibers are found in the nerve tissue. There is nothing to show, however, that such sprouts form links of union between the divided ends of fibers. Schmaus has shown that a divided spinal cord can be reunited by nerve fibers in case some of the blood-vessel-carrying tissue is left; that it is along this tissue, namely, in the lymph spaces of the blood vessels, that the new fibers push their way. Such fibers are extremely few in number. In spite, therefore, of a recently reported case by Stewart, in which a bullet had completely divided the spinal cord, and in which, after suture of the ends of the spinal cord, the patient is said to have had some return of conductivity the fifth day after the operation, there seems little justification in believing that the nerve fibers of the spinal cord, when once divided, regenerate in a way to give useful function.

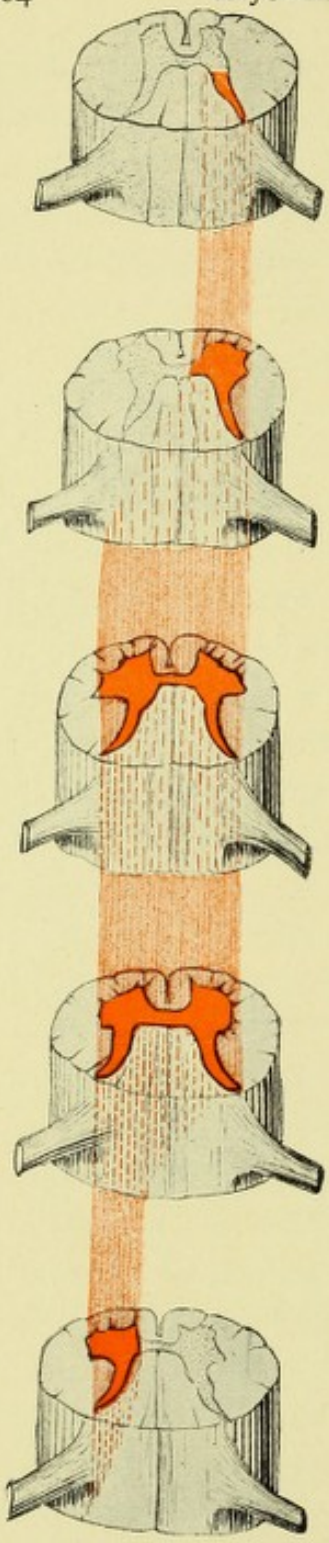


FIG. 29.—Diagram to illustrate the course of the blood in a case of hematomyelia.

Hematomyelia.—Hemorrhage into the spinal cord, or hematomyelia, occurs as a complication of general crushes of the cord, being merely an incident in the general destruction. It also occurs when there is no discoverable injury to the bones, and when the cord shows but little trace of bruising. While the pathological anatomy, as far as the hemorrhage is concerned, is practically the same in both cases, the clinical distinction between the two varieties is very great. Hematomyelia is usually focal. It occurs chiefly in the gray matter, since this latter is more highly vascularized and less supported by connective tissue than the white.

The ventral and dorsal horns are the favorite sites of the bleeding. The gray commissure, often, and the central canal, usually, escape. It is easier for hemorrhage to pass up and down the cord in the gray than it is for it to push its way out into the white matter. The common picture is a focus of hemorrhage in the gray matter of one or two segments from which columns of blood ascend or descend the central parts of the cord (Fig. 29). These columnar extensions proceed through two, three, or even four or more segments, in one or both direc-

tions. They are not equal in length, one penetrating for a longer distance than the other, the longer one usually going brainward. The seat of the extension is almost always in the gray matter, usually in the dorsal or ventral horn, on one side of the cord only; the extension above the seat of the most extensive hemorrhage is usually opposite the one below

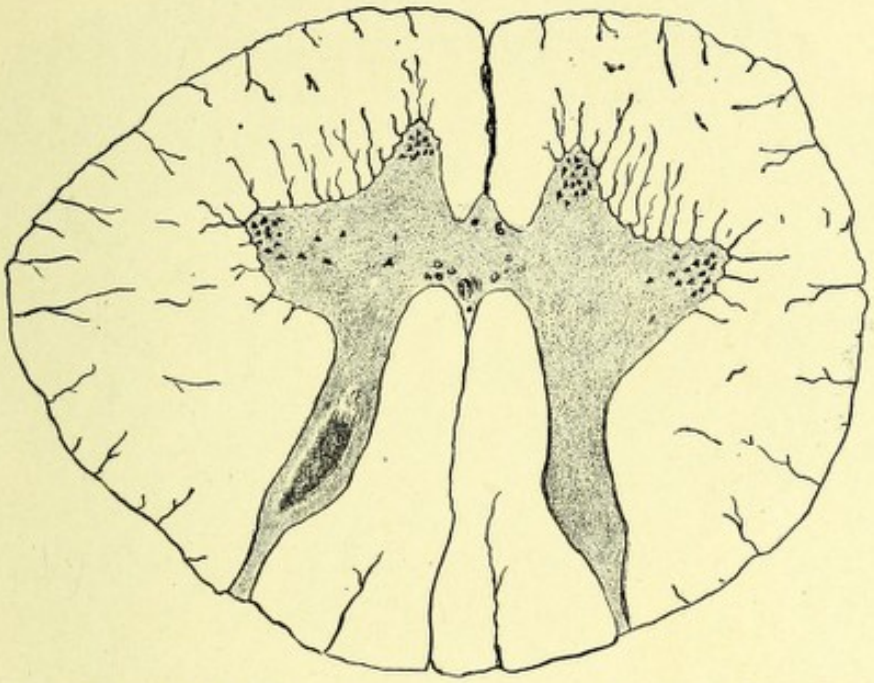


FIG. 30.—Showing inferior termination of the posterior column of blood in a case of traumatic hematomyelia.

(Fig. 30). When the original hemorrhage is profuse it may escape, at the focus of greatest injury, beyond the limits of the gray, and infiltrate the white columns to a greater part of the circumference of the cord, or may appear as smaller collections of punctate extravasations. Similarly, though much less frequently, the columnar extensions, instead of remaining in the gray matter, extend laterally, most frequently into the white matter behind the middle commissure. In addition, in the neighborhood of large hemorrhages there are usually minute extravasations disseminated in the white matter. Occa-

sionally, also, large isolated hemorrhages into the white matter are observed. They are more frequent in the dorsal columns just behind the gray commissure. The size of the

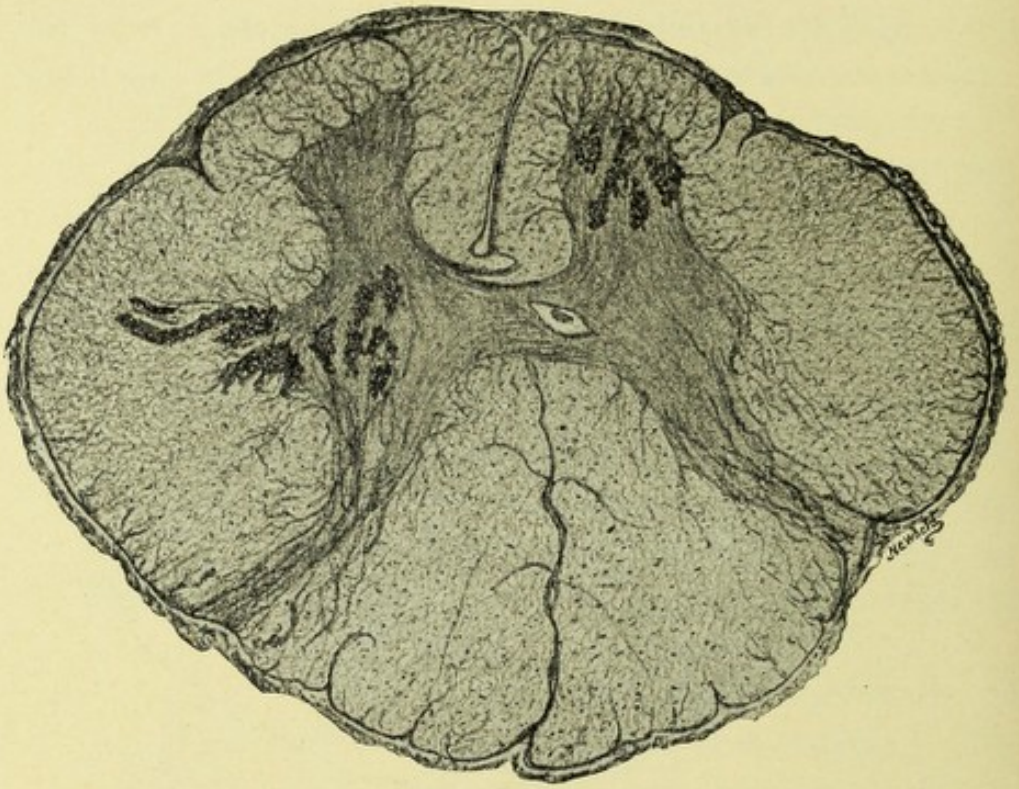


FIG. 31.—Small hemorrhages into first thoracic segment of spinal cord. Spinal column uninjured. No spinal-cord symptoms. Death due to general injuries.

individual hemorrhage varies greatly. In some of the reported cases the cord at the affected level has been completely hollowed out by the blood clot. In others the blood, though extending widely, has left some of the cord free. In others, as in Fig. 31, the hemorrhage is extremely small and more marked on one side than on the other. The effect upon the cord varies with the extent of the hemorrhage. When small, it causes no visible inflammatory or degenerative reaction (Fig. 32). Large hemorrhages may be surrounded by areas of softening. In many cases of large hemorrhages, however,

the action of the hemorrhage seems chiefly mechanical. The blood pushes its way through the cord substance, destroying as it goes. As it disintegrates and is absorbed, together with the destroyed nervous tissue, there results a cavity in the cord. Figs. 33, 34, and 35 show the various stages in the formation of such cavities.

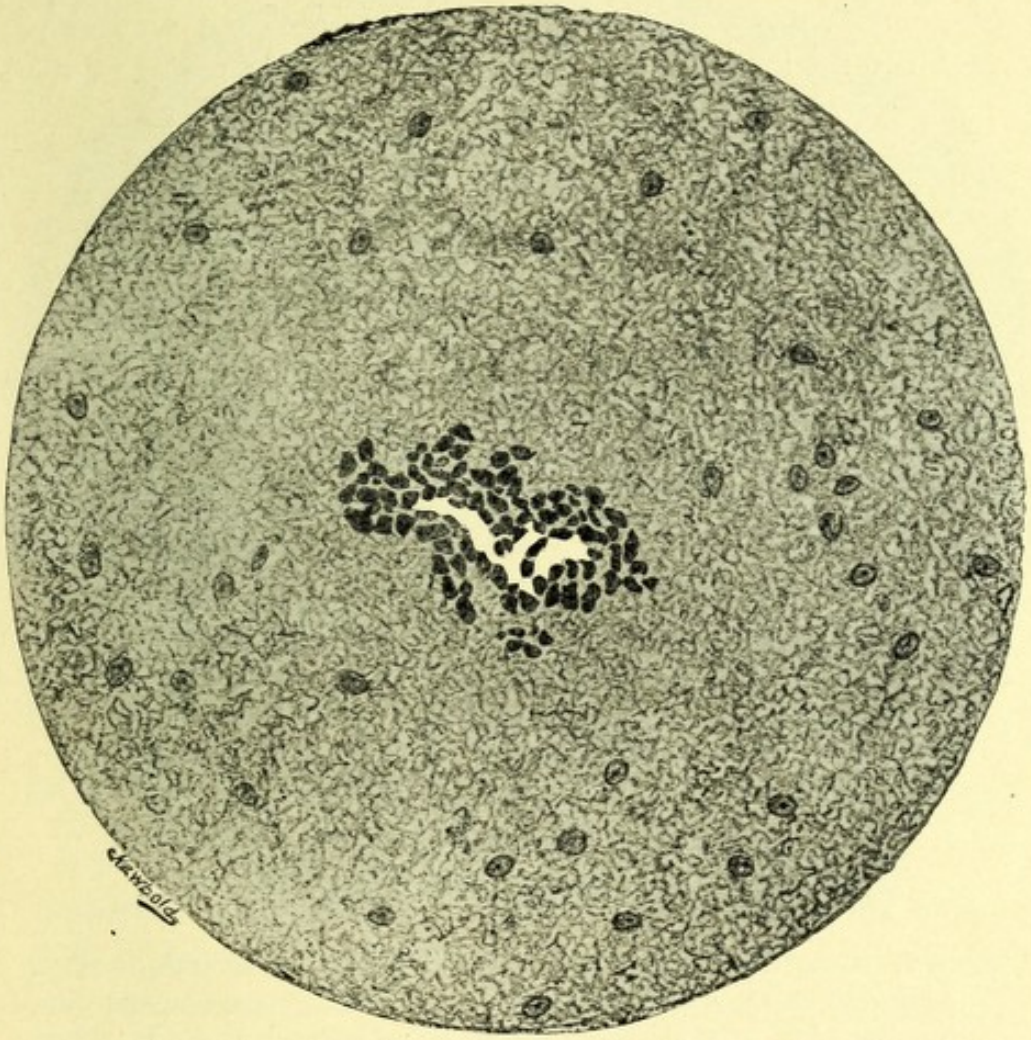


FIG. 32.—Small hemorrhage into spinal cord. No inflammatory or degenerative reactions.

Such hematogenous cavities have no epithelial lining and are not ordinarily connected with the central canal. They may resemble very closely the conditions found in syringomyelia, and the symptoms to

which they give rise are often surprisingly like those of syringomyelia. Some writers have gone so far as to refer to such cases as traumatic syringomyelia. They should, however, be kept distinct in name, as they are distinct in origin, from the chronic progressive and probably congenital spinal disease, and should be designated as primary hematomyelia.

Localized collections of hemorrhage from injury are found very much more frequently in the regions of the cord subjacent to the lower cervical vertebræ, i. e., in the region of the

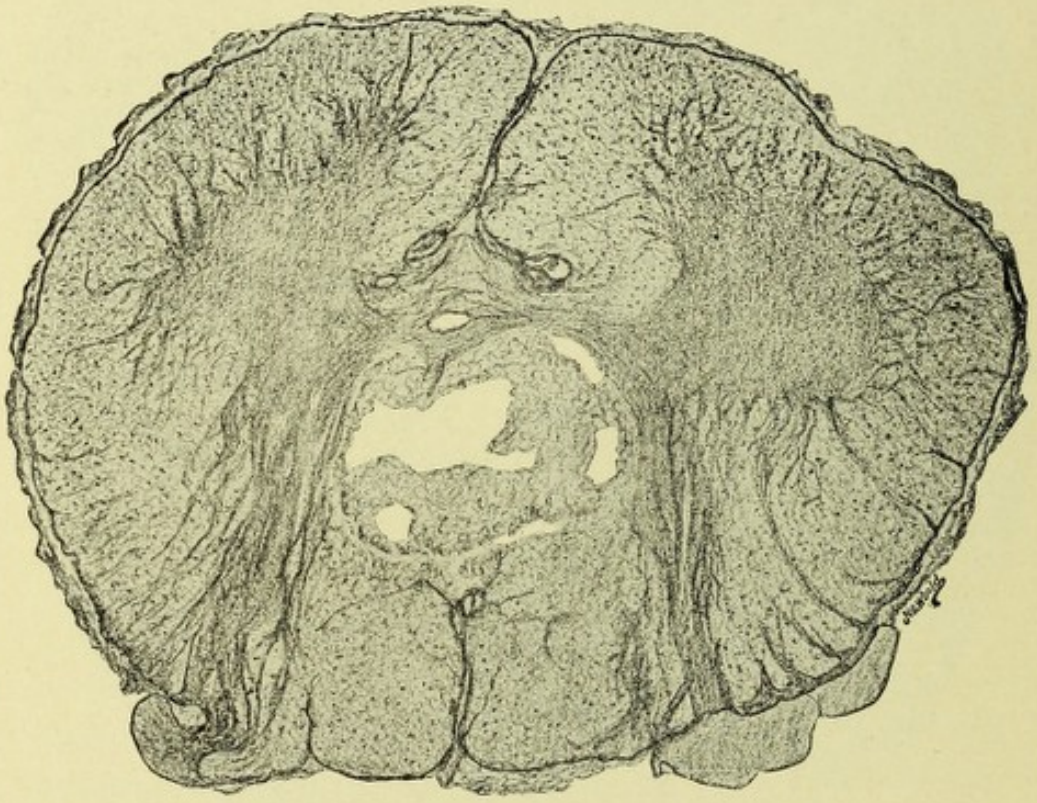


FIG. 33.—Case of fracture of fifth cervical vertebra with laceration of sixth cervical segment of the spinal cord. Ascending and descending hematomyelia. Section from fourth cervical segment shows beginning cavity formation. Blood and detritus not yet absorbed.

greatest spinal movements. The corresponding spinal-cord segments are the fifth, sixth, seventh, eighth cervical, and first and second thoracic. It is possible also that primary traumatic

hematomyelia occurs in the lower lumbar region and in the conus medullaris. In the majority of the cases of hemorrhage in this region, however, there have been serious lesions in the bones.

In some few cases of fracture of the spine in the lower dor-

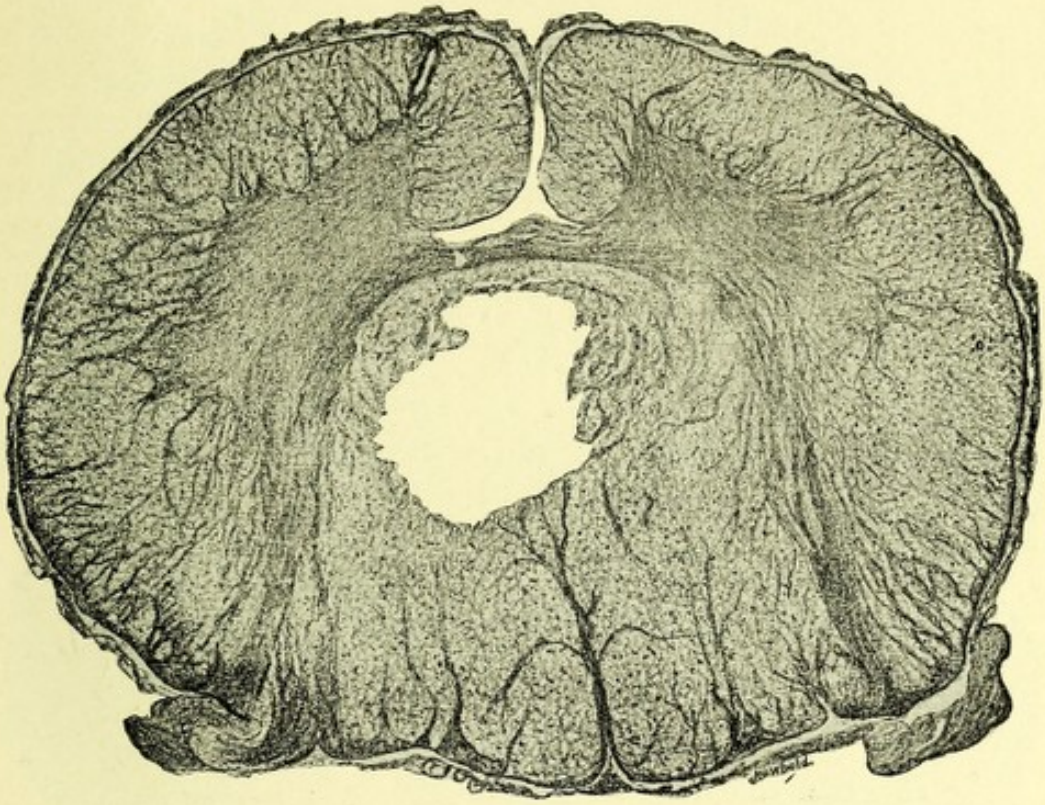


FIG. 34.—Same case as in Fig. 33. Third cervical segment. Cavity formation further advanced.

sal and lumbar region there appear, in addition to the symptoms of more or less complete paralysis in the legs, evidences of a partial lesion in the cervical region, although the cervical vertebræ remain intact. Such cases can best be explained by primary hematomyelia in the cervical region, due to forced movement in the neck, without fracture, which occurred at the same time as the fracture in the lower region.

In contrast to the previously described type of hemato-

myelia is the disseminated type, in which there are extravasations throughout the whole spinal axis. This variety of hematomyelia has been much speculated upon as affording an anatomical foundation for the traumatic neuroses. It has been found at autopsies on infants born after prolonged and difficult labor. In the cases described by me the patients had suffered fatal traumata, but gave no evidence of spinal-cord lesion in the brief interval of life granted them after the accident.

A *résumé* of the lesions found is as follows: Punctate hemorrhages scattered up and down the cord, generally too small to be recognized by the naked eye (Fig. 36). In some places there were only two or three free cells. In a few sections the blood lay in little cavities, but its most common distribution was an infiltration of a small number of blood cells between the neuroglia and nerve fibers.

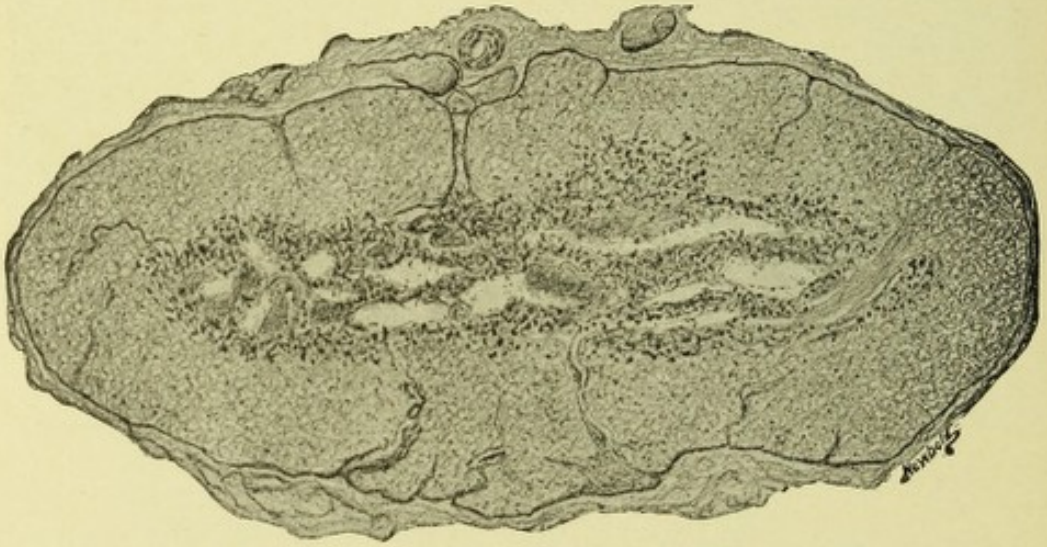


FIG. 35.—Primary traumatic hematomyelia. Death seven months after injury. Section shows late result of cavity formation. Central parts of cord, where hemorrhage had occurred, are being filled up with new tissue.

In one case there was some softening, with tendency to cavity formation. The largest extravasations were in the pia and around the nerve roots. In the spinal cord, although occurring in the gray matter, they were most frequent in the white matter, especially in the posterior columns. The situation of the hemorrhages bore little

or no relation to the location of the large blood vessels. The blood vessels were prominent and filled with blood. In all other respects the spinal cords appeared normal.

Such lesions as the foregoing cannot with certainty be recognized clinically. They are mentioned here as proof that general injuries

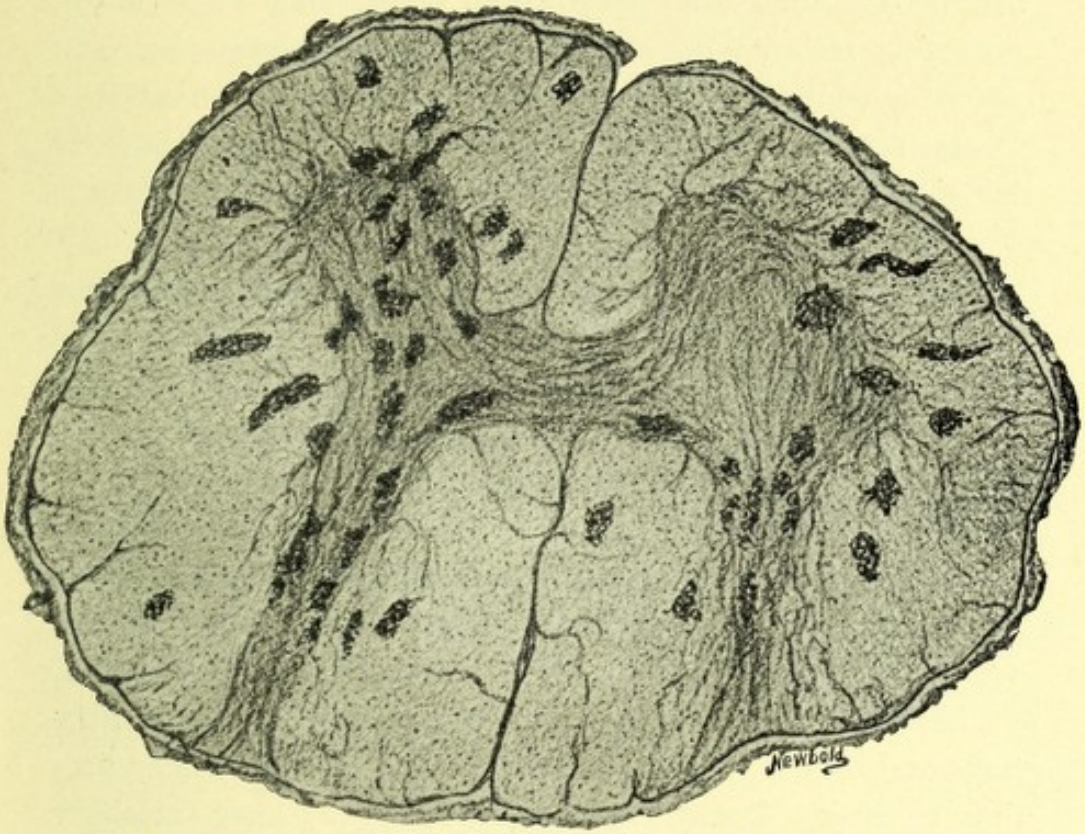


FIG. 36.—Disseminated hemorrhages into the spinal cord from injury.

can cause disseminated lesions throughout the spinal axis, a fact important to be borne in mind in connection with the clinical cases described under "unclassified forms."

GENERAL ÆTIOLOGY

Traumatic lesions in the spinal cord vary with the method of their production. In fractures and dislocations of the spine the spinal canal undergoes a sudden narrowing, so that the cord is compressed, contused, or lacerated. It is a fact too often lost sight of, that in many, if not in most cases injury

to the spinal cord as a result of fracture and dislocation of the spine is of momentary production; that the displaced bones, after doing the damage, spring back into place, so that the spinal canal does not remain permanently contracted. The condition in the cord, consequently, is the result of a sudden squeezing rather than of a long-continued compression. In severe cases the cord is reduced to a pulp, or even cut in two. In less serious cases, as in fracture through a vertebral body without displacement, or of a lamina, or of a spinous process, or of ligaments, the broken fragments may encroach but little upon the spinal canal, so that both cord and membranes escape with slight bruising only. Loose fragments of bones, except when caused by bullets, are unusual. In about one-third of all cases of fracture and dislocations the spinal cord escapes uninjured. Hemorrhage in cases of fracture is usually of secondary importance. As a general rule, the more serious the laceration of the cord, the less conspicuous clinically is the hemorrhage. It is most frequent in the cervical region, but even here it may be not at all prominent in severe lesions.

Stab wounds and pistol-shot wounds are similar to fractures and dislocations in their effects on the cord. They produce contusion, laceration, and hemorrhage. They act either by impinging directly upon the cord or by causing a splintering of bone. They most frequently cause partial lesions, most of the cases of Brown-Séquard paralysis being due to these agencies. They present no essential pathological characteristics, except that they are more apt to be followed by infection, or may be the starting point of adhesions, and thus cause serious and progressive compression of the cord. In both bullet and stab wounds there is usually much contusion of the cord, in addition to the actual destruction. In both these varieties of injury, also, hematomyelia may form an important feature.

Stretching of the cord without discoverable lesion in the spine, or distortion, is an injury limited to the cervical region, and results from sudden forced flexion or extension of the neck. The mechanism of production consists in the cord being actually stretched so that its component parts are torn in their long axes; or in the cord being drawn too tightly over the posterior surface of the body of a vertebra or of a vertebral disk. This variety of injury is most frequent in the region of the fifth and sixth cervical vertebræ, and is particularly effective in causing hemorrhage. It is impossible to conceive the blood vessels of the spinal cord being torn without the nerve fibers also being injured. Yet in many of the cases the hemorrhage is undoubtedly the most important lesion. Distortion may also lacerate the cord without hemorrhage, causing symptoms similar to those of hemorrhage. But such is not the general rule, and hemorrhage usually has a very important place in this class of injuries—so important that it has gained the distinction of constituting a definite clinical type of spinal-cord injury.

General Symptoms

Before passing to the detailed description of injuries to the spinal cord, some general statements regarding the symptoms which are common to all varieties of lesion will not be out of place. As the accident which gives rise to the spinal symptoms is usually severe, and frequently general in its effects, there is apt to be more or less cerebral concussion. Histories in these cases are always difficult to obtain, and usually incomplete, and the paralysis makes objective examination difficult.

Motor Symptoms.—Of these, the most important is paralysis. The paralysis resulting from trauma to the spine comes on, in most cases, instantly. If the injury is general, involving the head as well as the back, the patient is often rendered

unconscious and picked up paralyzed. Otherwise he is either knocked down, and finds himself unable to rise; or else, if injured while walking or standing (as in blows, stab wounds, etc.), the legs give out from under him and he sinks at once to the ground. In a few exceptional cases there is an interval between the receipt of the injury and the resulting paralysis. The commonest example of this is for the injured person to be able to get up and walk about, though with some difficulty, and then, after a few hours, to find the strength in the legs failing, and to become, in a brief period of time, totally paraplegic. Such cases are explained by slight contusion, which is followed by more destructive hemorrhage. Again, a paralysis, slight perhaps at first, is made complete by a sudden change in position. This is doubtless due to additional laceration of the cord brought about by fresh pressure from a fractured fragment. In excessively rare cases the paralysis is slight at first and rapidly improves, so that the patient is able to be up and about again. But after a few weeks the muscular power begins again to disappear from the parts originally affected, and the patient goes on with the symptoms of slow compression.

The degree of paralysis in spinal-cord injuries varies greatly. Immediately after extensive lesions the common and distressing picture is that of complete paraplegia. This may continue unchanged till death. Frequently the paralysis shows a predilection for the extensors, so that from the outset the patients are able to slightly flex the toes or fingers, or become able to do so soon after the accident. In partial lesions the paralysis may be profound and extensive at first, but rapidly improve. In other cases of partial lesions the paralysis is never anything more than weakness. In the commonest distribution the paralysis involves all muscles supplied by the affected segment and by all lower-lying segments. Thus, if

the cervical region is affected, there is paralysis of all four extremities; if the dorsal region, of both legs (paraplegia). In partial lesions there are exceptions to this, in that the paralysis may affect one arm and one leg chiefly (Brown-Séquard paralysis—spinal hemiplegia), or both arms chiefly (diplegia brachialis), or one arm chiefly or one leg chiefly (monoplegia). But even in those partial lesions in which the paralysis is chiefly one-sided, all muscles below the injury are weakened, at first at least.

In character, the paralysis varies with the seat and with the extent of the injury. In the lower lumbar and sacral regions the type, whether the lesion is partial or extensive, is that of peripheral neuron paralysis, viz., flaccid, with rapid atrophy and degenerative electrical reactions. At higher levels, when the injury to the cord is extensive, the paralysis is always flaccid at first. But as recovery sets in in these cases, and from the first in cases of partial lesion, classification as to the neuron type becomes possible. Thus, in the upper lumbar region both central and peripheral neuron types may be combined. In the dorsal region, where the motor nuclei are relatively unimportant, the central neuron type prevails. Injuries to the cervical region commonly give the peripheral neuron type in the hands, with the central neuron type in the lower extremities. As has been said, extensive lesions, and oftentimes partial ones at first, give flaccid paralysis, with absolute loss of muscular tone and absent reflexes. A satisfactory explanation of this is yet to be advanced.

The paralysis is very prone to be complicated by contracture, either when the flexors were not completely paralyzed, or when, in recovery, more power is restored to them than to the extensors. In partial lesions, immediately after the accident this is seen as a tightening of the hamstrings or of the Achilles tendon. It occurs through an excess of muscle tone

in the flexors which the weakened or paralyzed extensors are unable to resist, with the result that the segments of the affected extremity become fixed in flexion. The resulting contractures are often totally disabling.

Phenomena indicative of irritation are frequent in muscles partially paralyzed, and also occur in those entirely bereft of voluntary motor power. Twitchings and jerkings of the legs, especially at night, are frequently complained of by paraplegic patients. Fibrillary tremors are also common in muscles whose motor nuclei are affected. Both fine and coarse tremors are present in limbs which are regaining motor power.

Sensory Symptoms.—The sensory symptoms of spinal-cord injury consist of pain, hyperæsthesia, parasthesia, and anæsthesia. Pain is local, and due to the injury of muscles, ligaments, and bones of the back; or it is radiating, due to irritation of the posterior nerve roots. Local pain is dull and aching in character, constant, and greatly intensified by movement. It is often absent, and when present may annoy the patient but little. But in nearly all cases of injury to the spinal cord there is at first a point of increased sensitiveness over the vertebral spines of the injured region. Several spines may be hypersensitive, but there is usually one over which pressure causes more pain than over any of the others. As recovery proceeds, the pain lessens and often entirely disappears. Radiating pains occur only in partial lesions. They are particularly frequent in the arms, though they also occur in the legs. They are sharp and shooting, and are of the great intensity characteristic of nerve pains. Only occasionally spontaneous, they are generally brought on by movement of the extremities or of the spinal column. They are sometimes the chief symptom of partial lesions. They may last for weeks or months, but finally disappear entirely. Hyperæsthesia may be associated with radiating pains. More frequently, however,

it, in common with parasthesia, is part and parcel with anæsthesia. Thus, when cutaneous sensibility is impaired, rather than lost, as in cases of partial lesions or in recovery from total anæsthesia, hyperæsthesia and parasthesia are often prominent. It is a very good index that recovery has begun, when patients who have been deeply anæsthetic complain of numbness, tingling, and pain to touch in the affected parts. In severe lesions there is often a zone of hyperæsthesia directly above the total anæsthesia. Subjectively, this may be complained of as a girdle sensation.

In a large percentage of all spinal-cord injuries anæsthesia, in greater or less degree, is present. It is well to remember, however, that it is sometimes wanting. In extensive lesions it is profound from the outset, involves all forms of cutaneous sensibility, and remains throughout, unchanged. It occurs in areas characteristic of the segmental supply of the spinal cord. The upper limits are less pronounced than lower down. In many cases, however, it exists as a diminution rather than as a loss of sensibility. This fact is not generally enough appreciated; for a hyperæsthesia, if in characteristic areas, and if associated with other symptoms, is just as valuable for diagnosis as anæsthesia. Many cases are pronounced as free from sensory disturbances when painstaking examination would show that the sensibility, below a certain line or in a certain distribution, was blunted as compared with other parts of the body.

Elective anæsthesia is found chiefly, if not exclusively, in lesions in the central parts of the cord. It takes the form of loss or diminution in the perception of thermic and painful stimuli with normal perception of touch. If the sense of touch is impaired, it is pretty safe to say that the temperature and pain senses will be impaired also. Elective anæsthesia is sometimes found directly above the superior limit of total

anæsthesia in transverse lesions. It indicates central hemorrhage above the place where the cord is crushed across.

Déjérine has reported a case in which, with traumatic paraplegia of the lower extremities, there existed the syringomyelic dissociation on the right side of the chest and on the inner surface of the right arm. The autopsy showed, above the crushed part of the cord, a cavity in the gray matter extending from the ninth dorsal to the seventh cervical segment.

Anæsthesia is a more fleeting symptom than paralysis. It sets in abruptly, but is apt to quickly grow less. In recovery its surface extent shrinks, its degree lessens, and it may entirely disappear. When permanent, it is a direct and serious menace to life, and argues against recovery.

The distribution of these various forms of sensory disturbance, so important for localization, will be described under Focal Diagnosis.

Reflexes.—When a lesion involves the region of the cord in which a reflex center is situated (as, for example, the lumbar region, the center for the knee jerk), the reflex is diminished or abolished, varying with the severity of the lesion. When, however, the cord is involved higher up, the behavior of the reflexes whose centers are lower down is variable. The reason for the variability has been the source of much discussion.

The clinically important reflexes are the plantar and the knee jerk. The abdominal reflexes are variable and uncertain. The cremasteric is diminished or lost only (not exaggerated), and then only when its center is invaded. The plantar reflex, namely, the flexion of the great toe on tickling of the sole of the foot, is abolished in lesions of the sacral region. In higher situated lesions it is inverted, becoming the Babinski phenomenon (q. v.). The knee jerk is diminished or lost in injury to the second and third lumbar segments or

their roots. But the behavior of the knee jerk in lesions situated above these segments varies with the extent of the injury and with the time after the accident. Very slight injuries in the dorsal and cervical regions may cause an exaggeration of the knee jerk, with foot clonus; immediately. In more severe lesions in these regions the knee jerk is diminished or abolished at first, to become exaggerated later. The jerk may be absent for weeks or even months, and then return and become exaggerated. The longer it remains absent the more severe is the lesion. In extensive lesions, as in total transsection of the cord in the dorsal and cervical regions, it is lost immediately after the accident. But even in such cases, with complete division of the cord, the knee jerk may return after a number of weeks, if the patient live so long. It is important to bear in mind that absence of the knee jerk cannot of itself be construed as evidence of a complete transverse lesion.

Genito-urinary Symptoms.—Of the symptoms affecting the genito-urinary tract, the most important are those referable to the bladder. The nervous mechanism of micturition is still obscure. The vesical sphincter, by its continuous tonicity, enables the urine to collect in the bladder. Micturition is accomplished by the action of the detrusor urinæ, aided by the abdominal muscles. Both the sphincter vesicæ and the detrusor are under nervous control. The detrusor, doubtless, has a special spinal center in the lower sacral segments, which has central connections, and which is also excited reflexly (full bladder). It has generally been assumed that the sphincter also has a special center, closely adjacent to, though distinct from, that of the detrusor. But against such an assumption is the fact that injuries of the spinal cord never, primarily, do away with the action of the sphincter. Retention, not incontinence, is the early condition in traumatic lesions even when the region of the vesical center is itself destroyed. To explain

this, Müller assumes that the sphincter receives its innervation from the sympathetic. Retention of urine is present in a large proportion of spinal-cord injuries. At first it is usually pronounced. The expedients of pressure on the abdominal walls, or of having the patient stand (if he is not paraplegic), prove ineffectual, and the catheter is resorted to. The sensation of wanting to urinate is usually, though not always, absent. There is rarely the pain due to bladder distention so common in mechanically obstructive diseases. After a time, if the bladder paralysis persists, retention gives way to incontinence. The incontinence occurs as a more or less constant dribbling, or the intermittent discharge of a few drams of urine at a time. The meatus is constantly wet, and the odor of urine about the patient is hardly to be disguised. This incontinence is more than an overflow, as the persistent bladder distention decreases considerably with the onset of incontinence. Infection is an almost unavoidable consequence of long-continued catheterization, and consequently cystitis, pyelonephritis, and calculi are common complications of these injuries. In some few cases all bladder symptoms are entirely absent; in others they quickly disappear, or are very slight, consisting only in the requiring of an extra effort for voiding the urine. This may be so when the other initial symptoms, such as paralysis and anæsthesia, are fairly well marked. When it is so, it is a very certain indication that the lesion is partial, and a sound reason for a good prognosis. In a few rare cases of lesions very low down in the spinal axis, retention of urine may be the only symptom of the lesion.

The bladder itself shows the effect of its loss of nerve influence. Its epithelium desquamates, it is easily injured and very liable to infection. The urine is prone to alkalinity, and the formation of phosphatic stones is common. As a rule the penis is swollen and flaccid, and sexual and testicular sensations

are abolished. Priapism, with or without sensation, is not rare, especially in severe lesions, in high lesions, and when the patients are young. At the time of the accident there often is an ejaculation of semen. This is the result of the sudden and violent stimulation analogous to the ejaculations observed after death by the electric current. The effect of spinal-cord injuries upon the secretion of urine has not been accurately determined, though the weight of evidence is in favor of the quantity of urine being lessened.

Trophic and Vasomotor Disturbances.—In limbs the seat of traumatic spinal paralysis there are apt to be dryness and scaliness of the skin and interference with the growth of the nails. Sometimes the temperature of the skin is temporarily raised. This eventually gives way to lowered temperature, and, as a rule, the paralyzed parts are cold to the touch. Cyanosis is sometimes observed, especially in lesions of the lumbar region. Some elevation of body temperature is apt to set in a few hours after the injury. If this does not exceed 101° F. it is not a particularly alarming symptom. The higher the situation of the lesion, the higher is apt to be the temperature rise. In cervical lesions, very high temperatures (108°–110° F.) have been recorded as occurring within a few hours of the injury. When pulmonary complications set in, the temperature rises in nearly all cases. In cases of long standing, fever occurs as a result of infection. The most important trophic symptom in spinal-cord cases is the bedsore. It appears almost exclusively in parts the seat of pressure. Thus it is most frequent over the buttocks, the heels, and the external malleoli, but appears also, in cervical lesions, on the elbows and shoulders. Bedsores, usually, though not always, are confined to extensive lesions. They sometimes develop very rapidly, so that within a few days, or even within twenty-four hours of the accident, they are well under way.

More commonly, however, they develop slowly. The part which is the seat of pressure becomes slightly reddened, the redness extends in circular form, becoming darker in color until it assumes a bluish tinge. The skin over the reddened area is moist, and vesicles form, which, when they break, leave an ulcer in the skin. The ulcer may extend rapidly in diameter and into the underlying tissues. Infection in the ulcer is common, is attended by fever, and is often the immediate cause of death.

Herpes zoster, occurring in the distribution of one or more nerves, is also an occasional trophic symptom.

Digestive-Tract Symptoms.—Dryness of the mucous membranes of the lips and mouth is a common occurrence in spinal-cord injuries, though it may be due in part to the fever. Sordes sometimes form on the lips. The stomach, as a rule, presents no symptoms. The most important symptom referable to the intestines is distention of the abdomen by gas. It does not occur in lumbar and sacral lesions. In cervical and dorsal lesions more or less of this distention is quite common, and it is often an alarming symptom because it embarrasses respiration. Its development is aided by the weakness of the abdominal muscles, but its real origin is due to paralysis of the sympathetic fibers of the intestines. It takes some little time to develop, usually not showing itself sooner than twelve hours after the accident. It may gradually subside, or persist until death.

Some interference in defecation is almost constant in spinal-cord injuries. The most common is constipation, although incontinence of fæces may be present from the first. In partial lesions constipation alone may persist. In the severer lesions, with more extensive paralysis of the sphincter ani, the constipation eventually gives way to incontinence. Paralysis of the sphincter is readily demonstrated by digital

examination. On inserting the finger the sphincter fails to contract around it. On withdrawing the finger the anal orifice remains open. The incontinence may be absolute, so that the

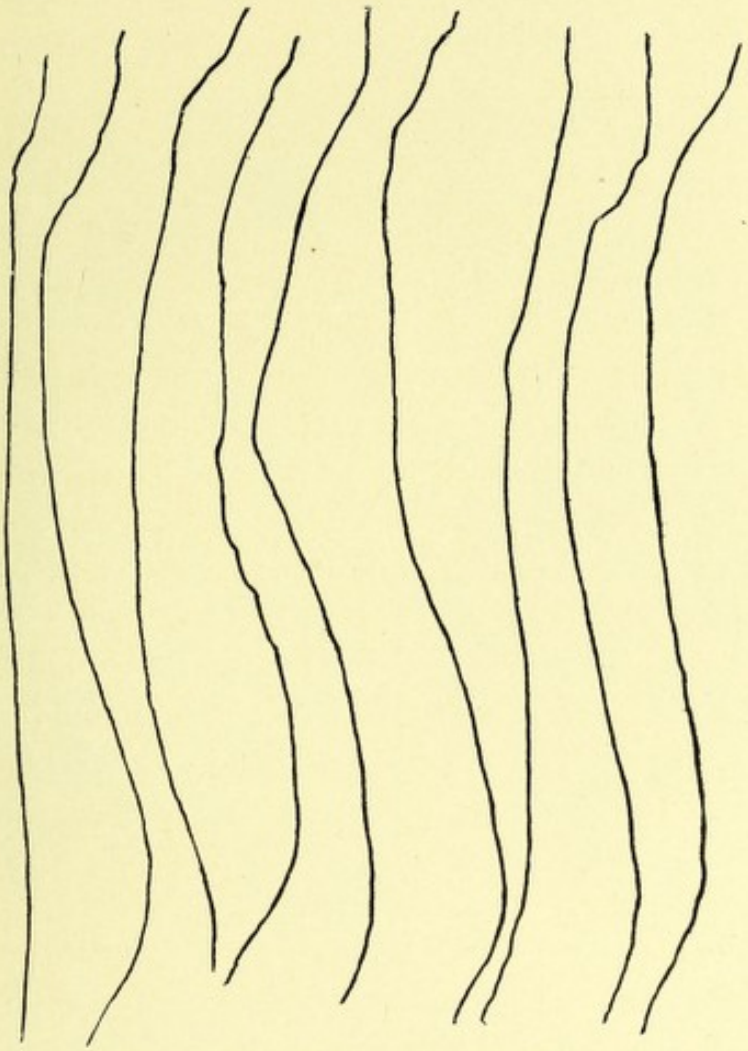


FIG. 37.—Showing variations in dorsal curves in healthy students of gymnastics. All taken facing to the right. (Walton.)

fæces pass away without the patient's knowledge, or the patient may feel the desire for defecation, but almost simultaneously the fæces pass. In other cases control is maintained, except under unusual excitement or in urinating or after laxatives have been taken. Incontinence of fæces is one of the most

distressing of human accidents, and, in addition to indicating a severe lesion, adds another menace to life through infection.

The other general symptoms need receive but brief mention. The pulse is usually full and at first accelerated. Later it may return to its normal rate, but is apt to be intermittent.

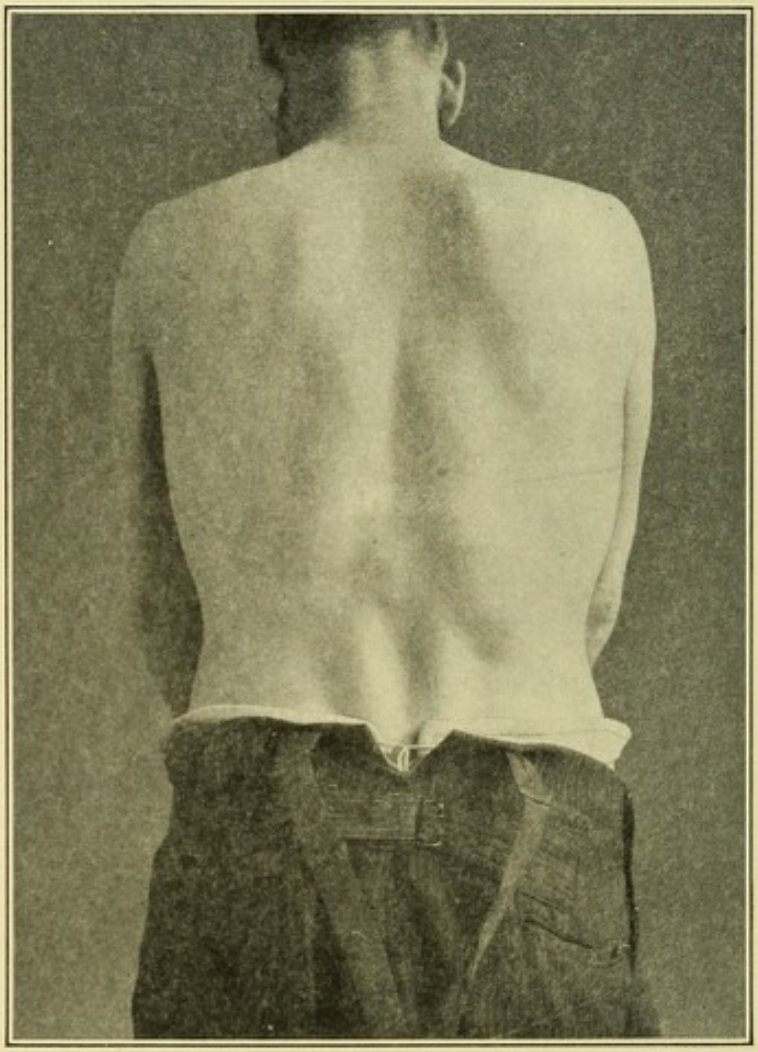


FIG. 38.—Showing prominence of vertebral spines in fracture-dislocation in lumbar region. Partial lesion of the spinal cord.

It becomes more rapid again following infection, but the high fever characteristic of cervical lesions does not necessarily accelerate it. Respiratory anomalies at first are the results of

paralysis of the muscles of respiration or of distention of the abdomen by gas. Later they are the expressions of œdema

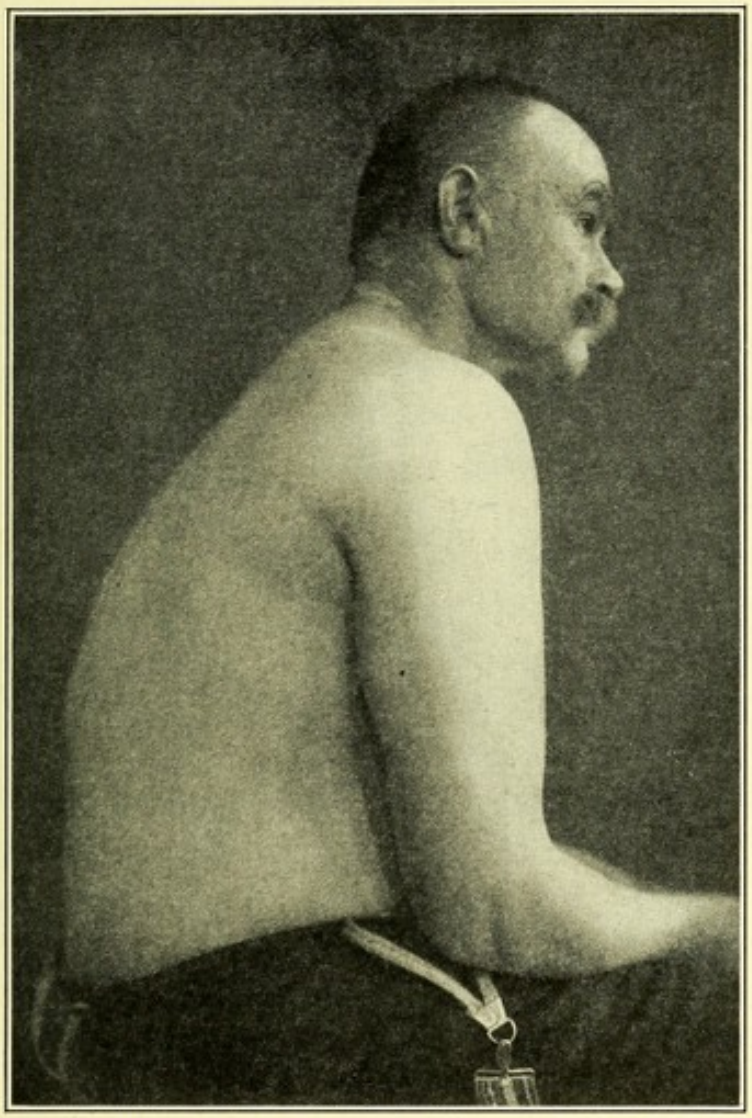


FIG. 39.—Showing attitude and deformity in fracture-dislocation in lumbar region. Partial lesion of the spinal cord.

and congestion of the lungs, as a result of which so many of the patients die. Myosis is a common symptom of injury to the spinal cord at any level. The abnormalities of the pupil and of the palpebral fissure, due to lesion of the cervical sympathetic, will be described with cervical lesions.

Focal Diagnosis

For purposes of prognosis and of operative treatment it is essential that the site of the injury in the cord be accurately located. The symptoms assisting to this end are surgical and neurological, of which the latter are the more important.

Surgical Focal Signs.—These are similar to the evidences of bone injury in other parts, except that fractures of the vertebræ generally cause less displacement than fractures of other bones. It is not at all unusual for the cord to be severely injured from fracture without the spinal column showing deformity. In dislocation, deformity is more pronounced. It consists in the lack of normal alignment in the vertebral spines, or in the depression or undue prominence of one or more of them. Before pronouncing as pathological irregularities in the spinal column, one should be familiar with the wide variations, in health, of the dorsal curve. This is well shown in Fig. 37. In fracture dislocation the deformity is often so great as to show the broken back at once (Figs. 38, 39). The deformity, when paralysis is not complete, causes characteristic attitudes, independently of those due to muscular paralysis (Fig. 40). Swelling over the seat of the injury is not marked,

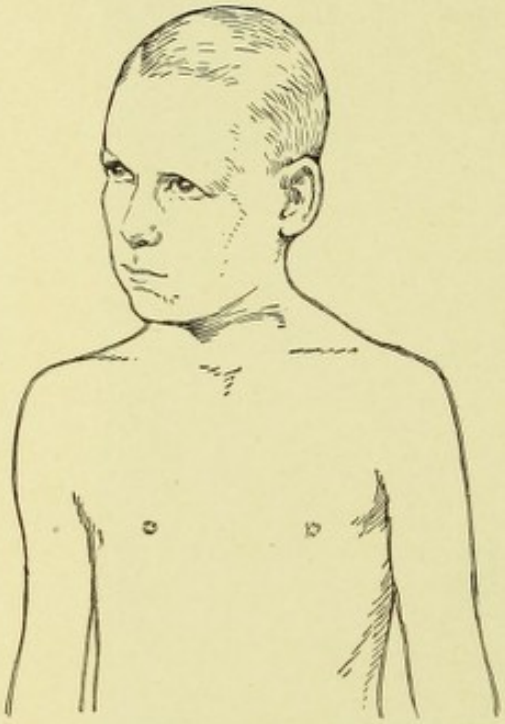


Fig. 40.—Showing position of the head in a case of dislocation of the atlas. (Drawing after Kocher.)

and may be entirely lacking. In occasional cases a fluctuating tumor, containing serum, forms rapidly after the accident, over the seat of the injury. Discoloration due to extravasated blood is not constant, and is much more frequent in injuries due to direct violence than to falls. There is frequently a red spot in the median line corresponding with a prominent spinous process. Undue mobility of the injured region is rarely demonstrable, and then is usually limited to loosened spinous processes or laminæ. Crepitus is only occasionally made out. Examination for it is hazardous.

The *x*-ray, in the hands of an expert, is often of great use. In bullet wounds it is a distinct aid in topical diagnosis, and is the only means of determining the place of final lodgment of the bullet. It is also serviceable in fractures. In partial lesions, when there is doubt as to the existence of fracture, it may establish the diagnosis. It is particularly successful in the cervical region, though excellent pictures have been taken even of the lumbar region. By it Fractures have been revealed which were not diagnosticable by other means.

Neurological Focal Signs.—The neurological focal signs are more to be relied upon than the surgical ones. The former point to the place which is the seat of injury in the cord, while the latter, such as deformity, etc., merely indicate that part of the affected bone which is accessible to external examination. The part of the spine which creates the more serious injury to the cord may be several inches higher up or lower down. In many cases there is an agreement between the surgical and neurological focal signs. For topographical diagnosis it is essential to be familiar with the normal relations of the spinal-cord segments to the vertebræ. The spinal cord itself stops at the lower border of the body of the first lumbar vertebræ, though in children it extends somewhat lower. The nerve roots consequently vary in length and direction, becom-

ing longer and less horizontal the lower down they are in the cord. A segment is the part of the cord contained between two sets of roots. Individual segments vary somewhat both in size and in their relation to the vertebræ. Their general arrangement is shown in Fig. 42.

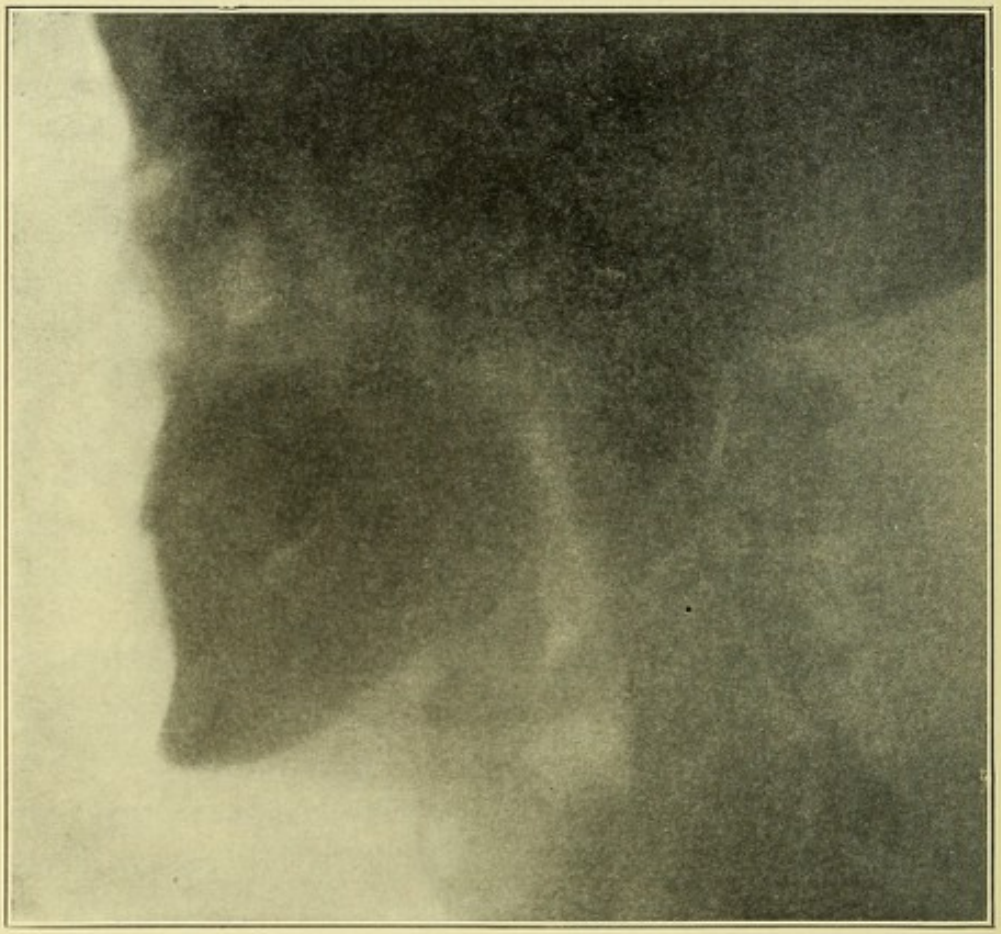


FIG. 41.—Forward dislocation of the sixth cervical vertebra. Head held rigid with chin to right of center. No spinal-cord symptoms. (Markoe.)

Starr quotes Chipault as giving the following rules for determining the relation of the segments to the spinous processes of the vertebræ: "In the cervical region add one to the number of the vertebræ, and this will give the segment opposite to it; in the upper dorsal region add two; from the sixth to the eleventh dorsal vertebræ add three. The lower

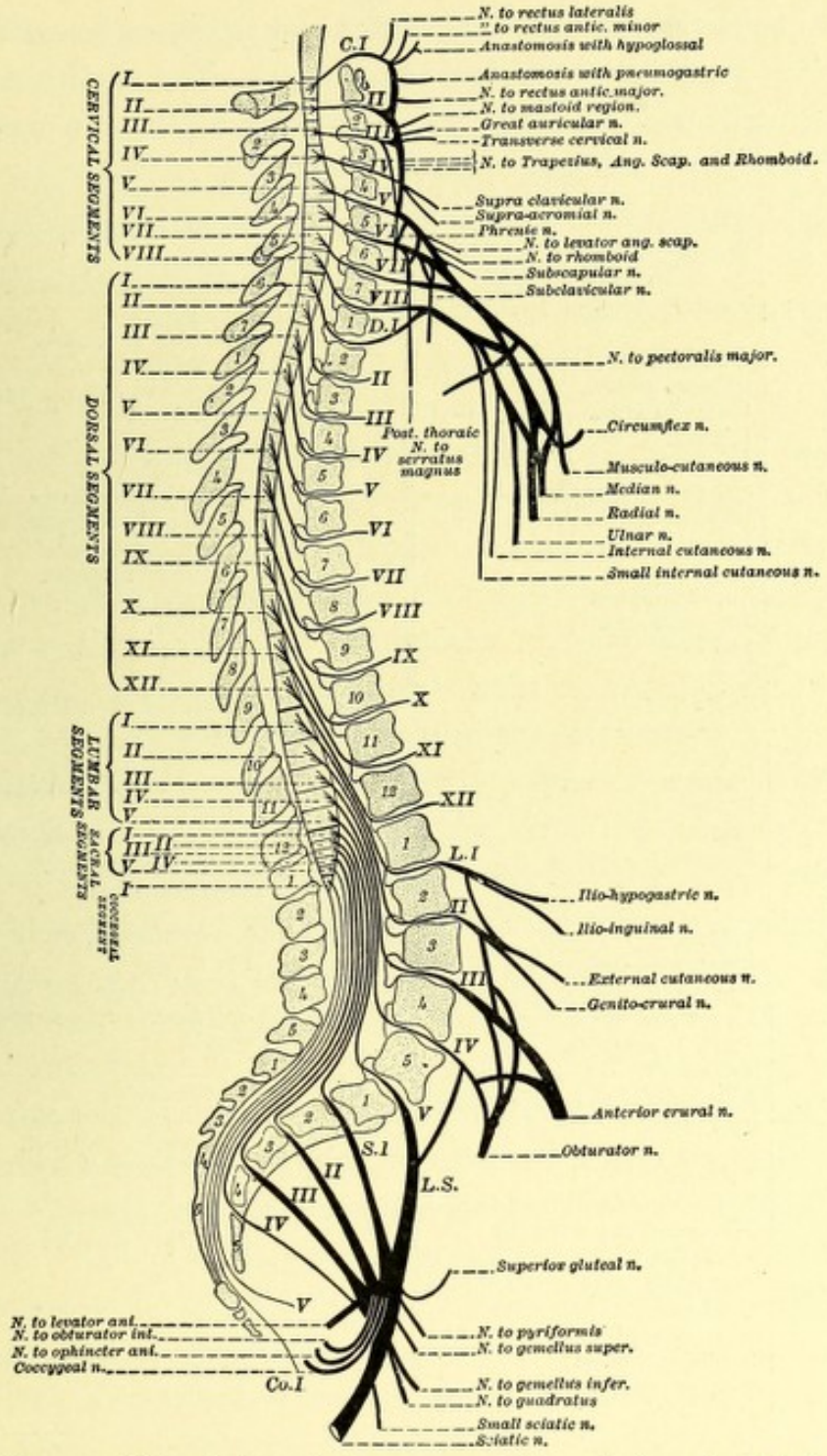


FIG. 42.—The relations of the segments of the spinal cord and their nerve roots to the bodies and spines of the vertebræ. (Déjérine et Thomas, modified by Starr.)

part of the eleventh spinous process and the space below it are opposite the lower three lumbar segments. The twelfth dorsal

Segments.	Muscles.	Segments.	Muscles.
C. ii–iii.	Trapezius. Sternomastoid. Levator anguli scapulæ. Deep muscles of neck.	C. viii.	Flexors of wrist and fingers. Interossei. Extensors of thumb (?).
		D. i.	Extensors of thumb. Interossei. Thenar and hypothenar muscles.
C. iv.	Diaphragm. Supraspinatus. Infraspinatus. Deltoid. Biceps. Supinator longus. Rhomboids. Scaleni.	D. ii.	Intercostals Muscles of abdomen and back. Erectors of the spine.
		L. i.	Abdominal muscles. Iliopsoas. Cremaster. Sartorius.
C. v.	Supraspinatus. Infraspinatus. Deltoid. Biceps. Supinators. Pectoralis major (clavicular part). Serratus magnus. Rhomboids Scaleni. Brachialis anticus. Teres minor.	L. ii–iii.	Flexors and adductors of thigh. Sartorius (?).
		L. iii–iv.	Extensors and adductors of thigh. Abductors of the thigh. Quadriceps femoris. Tibialis anticus.
C. vi.	Biceps. Brachialis anticus. Pectoralis major (clavicular part). Serratus magnus. Triceps (?). Extensors of wrist and fingers. Pronators.	L. v–S. i.	Flexors of knee. Glutei. External rotators of thigh. Long flexors of foot and toes. Peronei.
		S. i–ii.	Calf muscles. Peronei. Small muscles of foot. Erector penis (S. ii, Müller).
C. vii.	Triceps. Extensors of wrist and fingers. Flexors of the wrist (?). Pronators. Pectoralis major (sternocostal part). Subscapularis. Latissimus dorsi. Teres major.	S. iii–iv.	Perineal muscles. Ejaculator muscles (S. iii, Müller). Bladder. Rectum.
		S. v.	Levator ani. Sphincter ani.

spinous process and the space below it are opposite the sacral segments."

The important neurological focal signs are paralysis of muscles, anæsthesia, and disturbances of the reflexes.

Paralysis.— Every spinal-cord segment contains motor nuclei for several muscles. No single segment, however, contains all the nuclei for any one muscle, as every muscle is represented in at least two segments, and some muscles, as, for example, the biceps, have nuclei in a large number of segments. This fact furnishes at once a means of differentiating spinal-cord from peripheral-nerve lesions. It is common to see a complete paralysis of a single muscle from a lesion of a peripheral nerve, but such an occurrence is impossible from a traumatic lesion of the spinal cord. Further, muscles which are supplied by the same nerve do not necessarily have contiguous spinal nuclei; so that the distribution of the paralysis from injury of a nerve trunk is different from the distribution of the paralysis from injury to any one spinal-cord segment.

The paralysis which serves as a localizing sign, as it is a paralysis of individual muscles due to involvement of their spinal nuclei, is of the peripheral neuron type. The important muscles have their localization in the spinal cord approximately as indicated on page 190. Traumatic lesions, unless minute, are immediately fatal when situated above the fourth cervical segment.

Anæsthe ia.—As the sensory neurons are afferent, destruction of them, at any level, renders it impossible for sensory impressions received below the site of injury to reach the brain. In the spinal cord, destruction of a segment causes anæsthesia in all parts in and below the affected segments. That the diagrams of sensory supply of the spinal cord, as constructed by different observers differ considerably, is not astonishing. In most of the cases from which the conclusions as to sensory supply were drawn microscopic examinations were not made, and consequently the exact limits of the lesion

were not accurately determined. Further, the size of the segments themselves permits different anæsthetic fields, according to whether the lesion ceases at the upper or the lower limit of the segment. This is particularly true for the cervical region, where the individual segments attain a length of from one-quarter to one-half an inch. Where the segments are much smaller, as in the lumbar and sacral regions, the personal equation of the observer must also be a cause of variation in results. It is a matter of considerable difficulty to determine the limits of these segments, and it is in these regions that the widest statistical differences occur. Also, the sensory areas in different subjects doubtless vary considerably. But, even when the foregoing opportunities of variation are fully taken into consideration, the segmental distribution of anæsthesia remains as our most accurate method of focal diagnosis, and one upon which both physician and surgeon can confidently rely for practical purposes. In constructing a diagram for localization, it must be borne in mind that it is impossible, in the present state of our knowledge, or of our means of acquiring it, for absolutely definite and unvarying areas to be laid down. A scheme which harmonizes the differently constructed schemes of various authors, and which is reënforced by facts of development, proves the best one for working use, even if it allows some latitude for the precise limits of the various anæsthetic areas. Such a scheme, elaborated by Seiffer, seems the most useful of any hitherto published. It is represented in Fig. 43. Some explanations in regard to it must be given here.

Bilateral lesions of the spinal cord above, and involving the fourth cervical segment, are immediately fatal. Consequently the limits of anæsthesia due to injury to segments above the fifth is obtained from one-sided affections. Anæsthetic areas of these regions are indicated in the diagram. Below the lower boundary of the sensory area, supplied by the fourth cervical segment, the cutaneous surface is divided

by Seiffer's scheme into districts. The lines which divide these districts form the basis of orientation for the individual segments. These lines are:

1. The neck-trunk line.
2 and 3. The axial lines of the arms (ventral and dorsal).

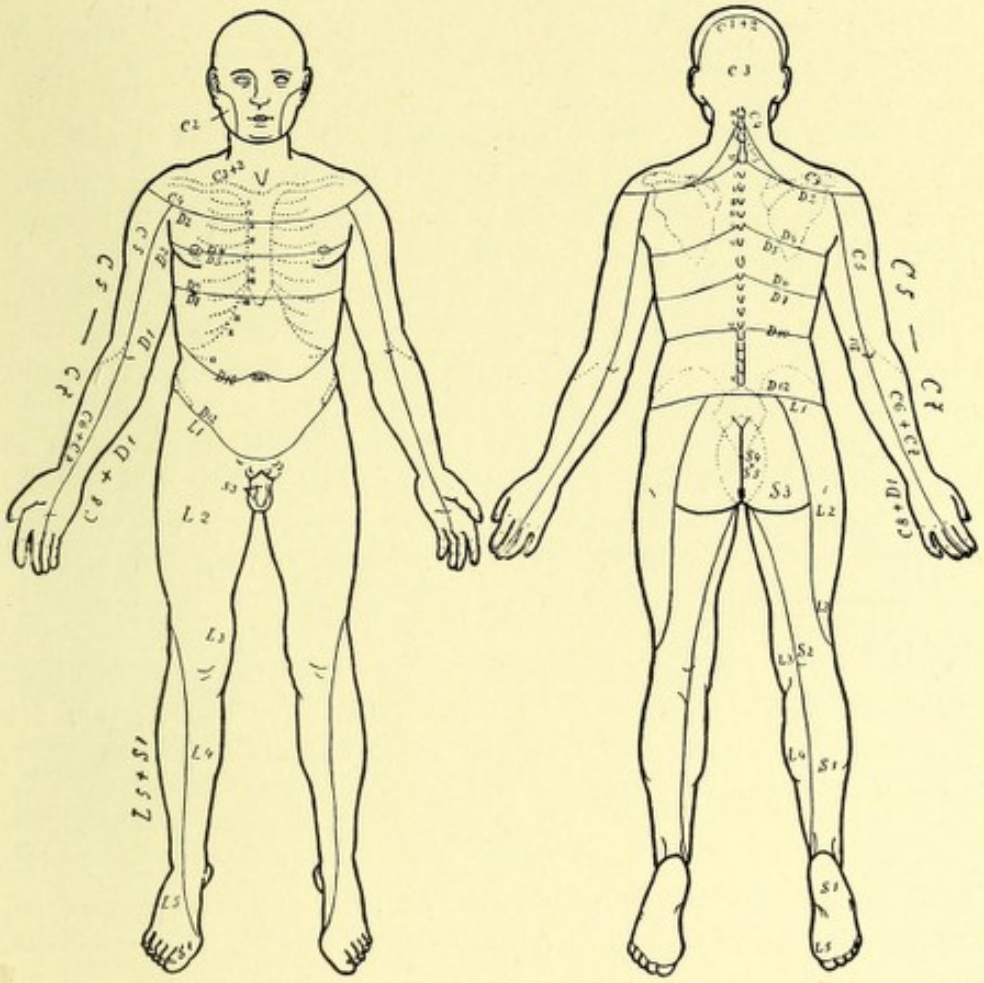


FIG. 43.—Diagram showing sensory supply of the spinal-cord segments. (Seiffer.)

4. The intermammillary line.
5. The xiphoid line.
6. The umbilical line.
7. The trunk-legs line.
8. The sacral circle.
9 and 10. The axial lines of the legs (ventral and dorsal).

The Neck-trunk Line.—In front, this line runs across the chest, chiefly in the second intercostal space, with a slight convexity down-

ward. It passes up over the upper part of the deltoid, so that the ventral axial line of the arm is perpendicular to it. This line is of especial importance in front, as there it constitutes the upper limit of anæsthesia in lesions of no fewer than five segments—viz., C. v, vi, vii, viii, and D. i. After destruction of any one of these segments the upper limit of anæsthesia in front is always the same, and corresponds with the neck-trunk line. Diagnosis as to which of these five segments is affected, cannot be made from the trunk anæsthesia in front. On the back and the back of the neck the sensory distribution of the above-mentioned segments, while not quite so uniform as on the chest, still presents only slight variations. From the upper part of the deltoid the neck-trunk line passes backwards, and then splits into two branches, the upper one ascending sharply to the fifth cervical spinous process, the other branch going to the spinous process of the seventh cervical vertebra. The line before its division, and its lower branch, form the upper boundary of the second dorsal segment, the lower boundary of the fourth cervical segment being formed by the line before its division and by its upper branch. In the small pyramidal space included by the branches of the lines from the two sides are to be found the sensory supply of segments C. v, vi, vii, viii, and D. i. Since the superior limit of anæsthesia caused by injury to any of these segments is the same in front, and since it varies so little behind, diagnosis as to the particular one of these segments affected depends upon the distribution of anæsthesia in the arms.

The Axial Lines of the Arms give the key to the differential diagnosis of the segmental functions which are so closely packed together about the neck-trunk line. These lines run down the middle of the arm, one on the ventral, the other on the dorsal side. The part of the cutaneous surface lying external or radial to them is supplied by segments C. v, vi, and vii; that lying internal or ulnar to them, by C. viii and D. i.

The fifth cervical segment supplies a greave-shaped area on the outer side of the upper arm, the sixth cervical a similar area on the outer forearm. The first dorsal segment has a greave-shaped area along the ulnar side of both arm and forearm. Segments C. vii and viii must occupy positions midway between the preceding. For none of these can more definite boundaries be laid down, and except for the general line of division, as indicated in the figure, we are not in a position to give the segmental supply of the fingers.

The Intermammillary Line.—This line, extending between the nipples, marks the boundary between the fourth and fifth dorsal seg-

ments. Parallel to it and passing through the tip of the xiphoid process, is

The Xiphoid Line.—Between this and the preceding are contained segments D. v and vi, and below this line are collected, in order from above downward, D. vii, viii, and ix.

The Umbilical Line, passing with slight undulation around the body at the level of the umbilicus, is about the level of D. x. The remainder of the dorsal region is contained between it and the next line, called

The Trunk-leg Line.—This line in front corresponds with the inguinal fold. Behind, it runs nearly horizontal, passing back below the crest of the ilium. It forms the natural boundary between the trunk and the extremities, and marks the end of the dorsal region and the beginning of the lumbar. It is somewhat analogous to the neck-trunk line in that it, in the middle of the body behind, marks the upper limit of a number of segments—viz., L. i, ii, iii, iv, and v, and S. i and ii. These segments, with the exception of the part of the trunk-leg line mentioned, have different superior boundaries, and are also to be further differentiated by the axial lines of the lower extremities.

The Axial Lines of the Lower Extremities.—These lines are similar to those of the upper extremities, except that they do not run in the middle of the limb. The dorsal axial line begins at the middle line of the body in the anal region, passing down the back of the leg behind the internal malleolus to the inner border of the foot. The ventral, or anterior axial line, is also posterior at first. Starting from the junction of the middle and outer third of the trunk-legs line, it passes over the middle of the gluteal region down the back of the thigh to just above the knee. It here becomes anterior, descending the leg in a slight sacral curve, passing beneath the lower border of the patella, gaining the tibial ridge, and then passing to the inner border of the foot, about the base of the great toe.

It is even more difficult to define exact sensory areas in the lower extremities than in the upper. This is in large part due to the close proximity which both lumbar and sacral segments have to each other, thereby making lesions closely confined to one segment unusual.

S. i, ii, and iii occupy the posterior part of the lower extremity contained between the axial lines. These areas are measured from above downward. Thus S. iii has its superior boundary in the trunk-legs line and in the boundaries of S. iv and v (to be immediately described). Below it is S. ii. Thus both of these are posterior, except that S. iii supplies the anterior part of the genitals, below the part sup-

plied by L. i. S. i forms the transition between the lumbar and sacral regions. It is to be sought on the sole of the foot (perhaps somewhat above it posteriorly), and it may extend outward to the external part of the calf of the leg. L. i, ii, iii, iv, and v occupy the anterior surface of the lower extremity, the anterior axial line dividing L. iv from L. v and such part of L. i as may reach in front. Posteriorly, the lumbar segments occupy the regions not occupied by the sacral. L. v probably has a representation on the sole of the foot.

S. iv and v are contained in a saddle, or circle-shaped area of somewhat uncertain size, situated at the caudal extremity of the trunk.

Reflexes.—The behavior of the reflexes under varying degrees and conditions of spinal injury has already been given. It is only necessary here to give the general segmental location of the reflexes. The most important of them are approximately as follows:

Segments.	Reflexes.	Segments.	Reflexes.
C. v–vi.	Biceps. Supinator longus.	D. ix–xii.	Abdominal.
		L. i–ii.	Cremasteric.
C. vi.	Triceps.	L. ii–iii–iv.	Knee jerk.
C. vi–viii.	Extensors of wrist.	L. iv–v.	Gluteal.
D. vii–ix.	Epigastric.	S. i–iii.	Foot clonus. Babinski (plantar).

Brown-Séquard Paralysis.—As a result of hemorrhage, of gunshot and stab wounds, and less frequently of ordinary fractures of the cervical and dorsal region, the resulting symptoms indicate a lesion in one-half of the cord only—Brown-Séquard paralysis. They consist in a crossed paralysis of motion and sensation, distributed as follows:

On the side of the injury: Paralysis, with (usually) increased tendon reflexes. Loss of muscular sense (sense of

position). Hyperæsthesia, often higher than the anæsthesia of the opposite side.

On the opposite side: Anæsthesia. The anæsthesia never includes the sense of position. It is frequently also elective, the pain and temperature sense being alone affected, tactile sensibility remaining normal. There is some reason to suppose that unilateral lesions in the sacral, and possibly in the lower lumbar region, give a slightly different picture. From a case of his own, and from two earlier reported cases, Weisenburg believes that in these regions a one-sided lesion causes disturbances of motion and sensation in the lower extremity, external genitalia, and perineum on the same side, and disturbances of sensation in perineum and external genitalia on the opposite side. This is explained by the sensory fibers of the pudendal plexus decussating lower down than do the sensory fibers for the limbs.

CHAPTER VI

INJURIES TO THE SPINAL CORD—*Continued*

Fractures and Dislocations of the Spine—Wounds of the Spinal Cord—Pott's Disease—Spondylitis Traumatica.

For clinical convenience, injuries to the spinal cord may be divided into two general classes, according as to whether the evidences do or do not point to lesions in the vertebræ, viz.:

I. Spinal column injured by fractures and dislocation of the spine, causing secondarily contusion, laceration, hemorrhage, and softening of the cord. Such secondary injuries as Pott's disease and spondylitis must receive mention here also. Under this head, also, naturally fall such injuries as stab wounds and bullet wounds. This class contains the larger number of spinal-cord traumata.

II. Spinal column uninjured, but spinal cord the seat of hemorrhage (primary hematomyelia), and, according to some authorities, concussion.

Fractures and Dislocations of the Spine

Although this class of cases is usually referred to in terms of bone, it must be distinctly borne in mind that the bone injury is of secondary importance. It is the injury to the spinal cord which is significant. Cases of fracture or dislocation, without involvement of the cord, are fairly frequent, but are of no more, and oftentimes of less gravity than fractures and dislocations of other bones. In cases with spinal-cord involvement the original bone deformity, and that which ensues from

rarefying osteitis, are much less important than the paralytic symptoms. The symptoms of injury to the cord have important differences, according to the region affected. They

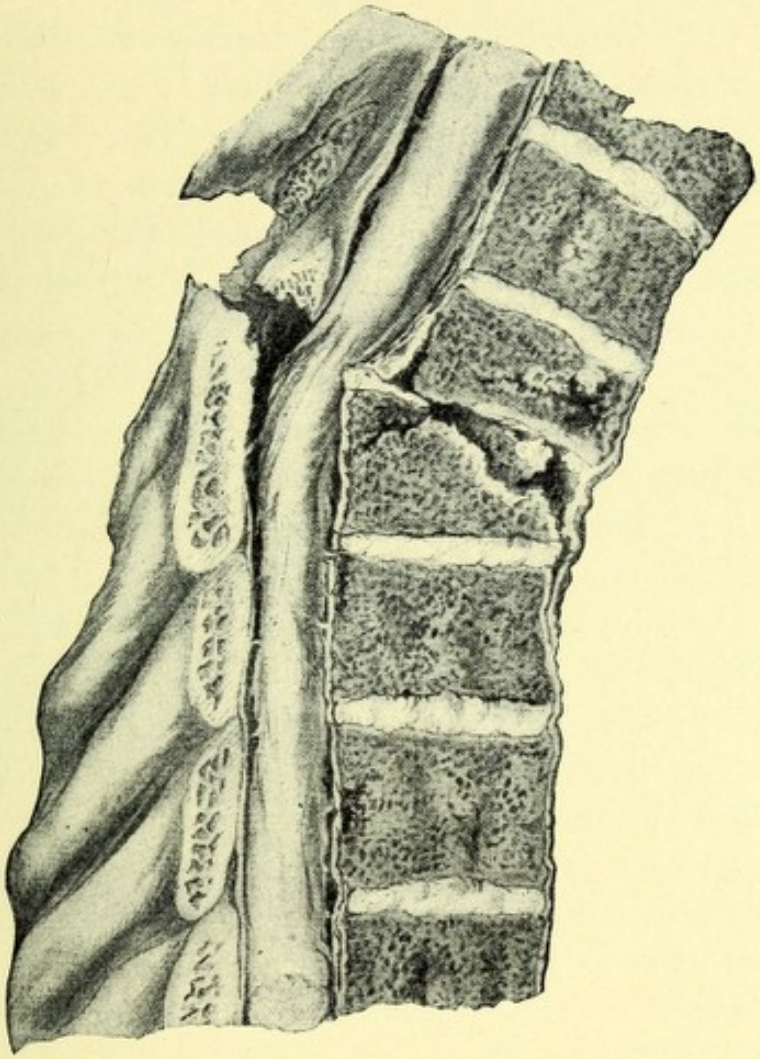


FIG. 44.—Fracture of the fourth and fifth lumbar vertebræ. Compression of the spinal cord. (Wagner and Stolper.)

may be divided into the following regions: Cervical, dorsal, lumbosacral, and cauda equina.

Cervical Lesions.—Of 136 cases of fracture and dislocation of the spine referred to by Wagner and Stolper, 34, or about twenty-five per cent, involved the cervical region, the

vertebræ affected being the third, 10 times; the fourth, 7 times; the fifth, 5 times; the sixth, 11 times; and the seventh, once. Of 82 cases of Burrell's, 28 were in the cervical region; of 270 of Gurlt's, there were 98 in the cervical region.

The most frequent cause of these fractures is sudden forced flexion of the neck, which is brought about by falls on the head or by heavy weights falling on the head. Direct violence to the neck is also a cause. The bone lesion consists most frequently in fracture of the body of the vertebra and dislocation. Dislocations occur also without fracture. Isolated fractures of parts other than the body, such as the spinous processes and the laminæ, also occur as the results of direct violence to the neck. They are associated with considerable contusion, with extravasation, and often present the signs of mobility and crepitus. In fractures of the cervical region, more than elsewhere, sudden movements are apt to increase the injury to the cord. These injuries cause considerable malposition of the head, which becomes particularly noticeable if the patient recovers. The head is bent forward (Fig. 46), or is rotated to one side or the other (Fig. 40). In forward dislocations of the lower cervical vertebræ the projection in the posterior pharyngeal wall can sometimes be felt by digital examination through the mouth. From the nature of the accident there is apt to be more or less cerebral concussion, and in those injuries the patients are frequently unconscious for a time.

Owing to the presence in the cervical cord of sympathetic fibers for the pupil, the palpebral fissure, the eyeball and face, cervical lesions have certain characteristics not found in lesions situated lower down. The symptoms referable to injury of the cervical sympathetic are paralytic in character, and consist in myosis, narrowing of the palpebral fissure, sinking in of the eyeball, flushing of the face, and loss of the ciliospinal reflex, which consists in dilatation of the pupil by peripheral

stimulation. In lesions involving both sides of the cord the myosis is the striking symptom. In unilateral lesions, on the other hand, all the symptoms of paralysis of the cervical sympathetic may be present. As cervical lesions frequently result from distortion, and as distortion is a variety of injury which may affect the cord completely or partially, there is a great variety in the fullness of the symptom complex in these cases.

Severe transverse lesions are characterized by high temperature, abdominal type of breathing, priapism, retention of urine, and end fatally, with few exceptions, in a few days or even a few hours. The focal signs are readily deduced, from what has been said on focal diagnosis, but a brief *résumé* of the more characteristic symptoms as related to injury of the different segments may be given. Transverse lesions of and above the fourth cervical segment are immediately fatal from paralysis of the phrenic and diaphragm.

Fifth Cervical Segment (fourth cervical vertebra).—Paralysis of all extremities, except perhaps scapular muscles. Patient can shrug shoulders. Diaphragmatic breathing. Anæsthesia from second intercostal space, including all of arms.

Sixth Cervical Segment (fifth cervical vertebra).—The biceps, the supinator longus and brevis, the brachialis anticus, and the deltoid, escape complete paralysis. The patient can flex forearm and raise arm, and lies with forearm and hands slightly flexed, with the upper arms slightly abducted and rotated from the trunk. Anæsthesia the same as in fifth-segment injury, except that the radial side of the arm is not involved.

Seventh Cervical Segment (fifth cervical vertebra).—The triceps is not extensively paralyzed, neither are pronators of arm. The patient, therefore, can extend and rotate forearm.

The arms lie folded on the chest. Distribution of anæsthesia not to be absolutely differentiated from that of C. vi.

Eighth Cervical Segment (sixth and seventh cervical vertebræ).—Paralysis limited to below elbow; possibly of fingers only; possibly, also, of fingers and wrist. Anæsthesia in ulnar distribution (Fig. 45).

First Dorsal Segment (seventh cervical vertebra).—Pa-

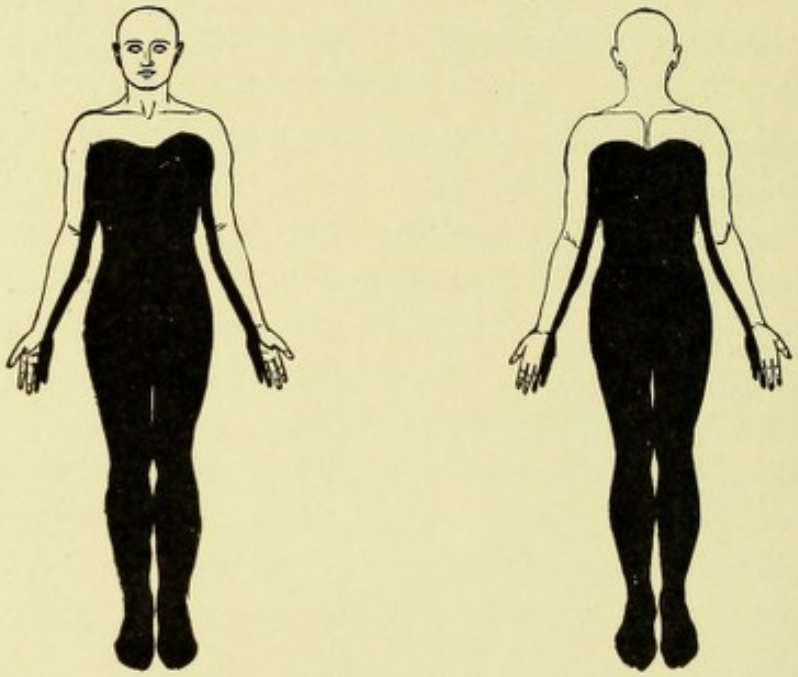


Fig. 45.—Anæsthesia in a case of fracture of the cervical vertebræ, with injury to the eighth cervical segment.

ralysis both motor and sensory, that of ulnar nerve distribution.

These types represent the severe and fatal degrees of injury. More common than bilateral and symmetrical symptoms are cases in which some of the symptoms are wanting on one or both sides. Of motor paralysis, one side is usually more profoundly paralyzed than the other. One arm is especially apt to retain considerable motor power. Diplegia brachialis,

or paralysis of both arms without involvement of the legs, is a symptom complex characteristic of primary hematomyelia rather than of fracture. The type of spinal hemiplegia is sometimes observed. In very rare cases localized motor paralysis is wanting. The following is an example:

The patient, W. A. B., thirty-six years of age, fell about fifteen feet into an areaway, on December 9, 1898, landing on the head. He was picked up unconscious. The scalp was badly contused and lacerated; there were great pain and tenderness and some swelling of the neck. There was no paralysis, but both arms and both legs were weak, the weakness being especially noticed on the left side. There were tingling of the fingers and numbness of the arms and legs. There were no disturbances of the bladder or rectum.

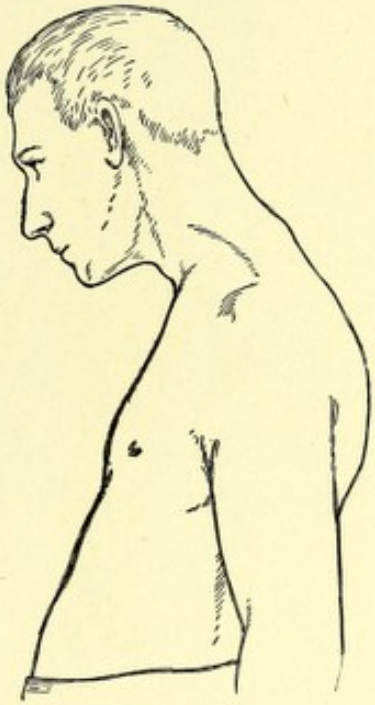


FIG. 46.—Attitude in a case of partial lesion of the spinal cord, due to injury of cervical vertebræ.

Examination, January 25, 1899, Vanderbilt Clinic: The patient (see Fig. 46) holds the head bent forward and is unable to make free movements with the neck. Efforts at movement cause severe pain. The seventh cervical spine is so unduly prominent that, although there is no crepitus, false motion, or other evidences of fracture, it seems certain that there has been either fracture or dislocation in this vicinity. The symptoms pointing to a nervous lesion are a partial hemianæsthesia on the right side, and a contracted pupil, with some sinking in of the eyeball and narrowed palpebral fissure on the left (ciliospinal center). The hemianæsthesia is very indistinct in its upper boundaries. It does not involve the face, and begins at about the second intercostal space. In the upper extremity it is more definite, and in the leg it is pronounced. It includes all qualities of cutaneous sensibility. There is also anæsthesia of the index finger of the left hand. All the muscles of the extremities seem somewhat weak, but there is no demonstrable paralysis. There is no localized atrophy, no fibrillation, no foot clonus. The knee jerks

are lively, especially the left, but not excessively exaggerated. The absence of paralysis, of sphincter troubles, and of marked changes in the tendon reflexes, shows that the injury to the cord was extremely slight. It is unusual for such slight injuries to be caused by a fracture or dislocation. But that a bone lesion, causing pressure upon the outside of the cord, was at the bottom of the symptoms in this case, seems probable. There were deformity and extreme pain in the back, with paralysis of the cervical sympathetic; the motor disturbances, slight though they were, involved all four extremities, and the hemianæsthesia bespoke a lesion affecting the long afferent sensory paths.

In general, however, paralysis is the cardinal symptom least likely to be wanting. More frequently the bladder escapes, or there is no anæsthesia.

The fewer the symptoms the better the prognosis. The preservation of the knee jerks and of bladder control are of particularly favorable omen.

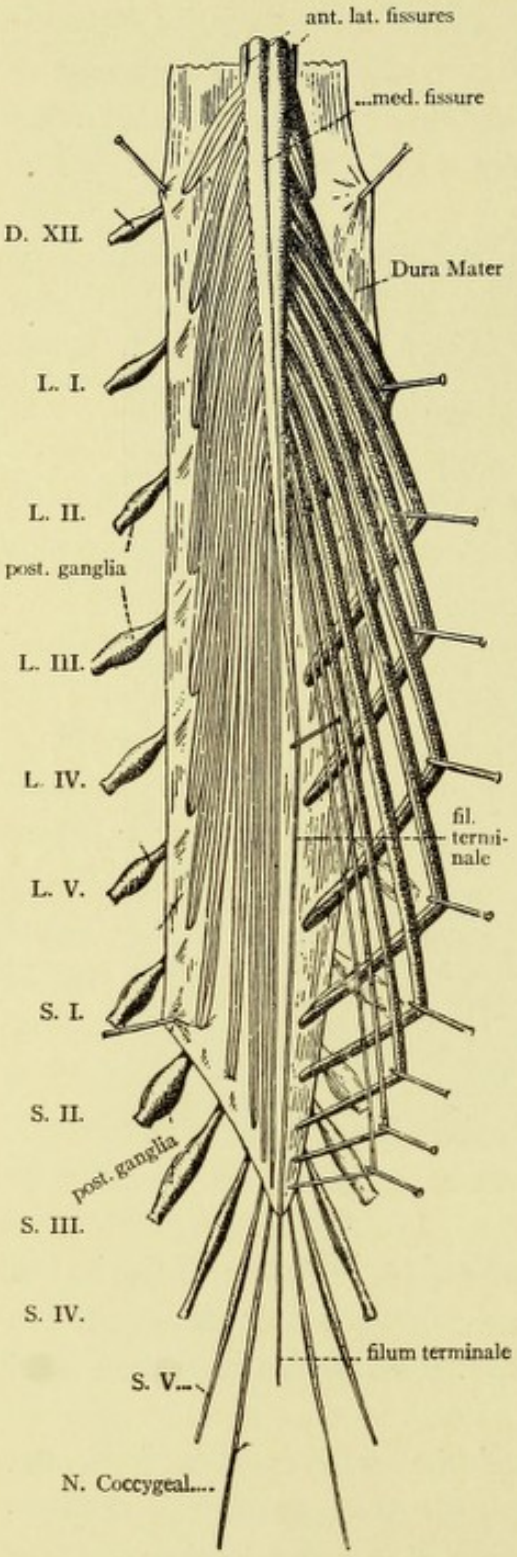
Dorsal Lesions.—This region of the spinal cord, considered surgically, extends from the second through the twelfth dorsal segment, and is protected by the part of the spinal column which extends from the first through the tenth dorsal vertebra. Fractures in this region are almost exclusively of the vertebral bodies, and the external deformity is generally pronounced. They may be complicated by fractures of the sternum and ribs. They result from direct violence, distortion as a cause being unknown in this region. Here, also, in contrast to the cervical region, the cord is never injured without injury to the bones. The bone injury consists almost entirely in fracture of the vertebral body, with serious mutilation of the cord. This region is less frequently involved than the cervical, there being, in Wagner and Stolper's tables, 26 cases out of 136; in Burrell's, 31 out of 82; in Gurlt's, 71 out of 270. The vertebræ affected were the fourth, 9 times; the tenth, 8 times; the eighth, 4 times; the fifth, 3 times, and the sixth, twice.

Injuries to the dorsal cord have no characteristics demanding special mention, except that they are almost always severe, and that, varying with the altitude of the lesion, the intercostals or the abdominal muscles may be paralyzed.

FIG. 47.—Fracture of arches of fourth and fifth lumbar vertebræ.

Lumbosacral and Cauda Equina Lesions.—Injuries to this region interfere with the movements and sensibility of the legs, and with the functions of the bladder, the rectum, and the sexual organs.

The lumbosacral region of the cord extends from the upper border of the eleventh dorsal vertebra to the lower border of the first lumbar. It is divided into five lumbar and five



sacral segments, each segment giving off a pair of nerve roots, which go to form the cauda equina, and the last sacral giving off also the coccygeal nerve. The three lower sacral segments are called the *conus medullaris.*

The important functions presided over by the lumbosacral cord are crowded into small compass, the entire length of all ten segments not exceeding three and one-half or four inches. The cauda equina is much longer. The roots of the lumbar and sacral segments which make it up descend directly downward from their origins, the lower the root the more internal being its situation (Fig. 48). They pierce the dura opposite the same intervertebral and sacral foramina by which they emerge from the spinal cord. From the first

FIG. 48.—Diagram of the lumbosacral region of the spinal cord and of the cauda equina, after opening the dura. One-half natural size. (Van Gehucten.)

lumbar nerve, behind the eleventh dorsal vertebra, to the space between the sacrum and coccyx, where the coccygeal nerve emerges, is a distance of from eight to ten inches. No single nerve root is so long as this, but the sacral roots have a course in the spinal canal of from five to six inches. The cauda is covered throughout by the dura. The roots which compose it have distinct motor and sensory functions, as the union of them to form mixed nerves takes place outside the dura, where also, on the sensory root, before its union with the motor, the posterior ganglion is situated. The cauda completely envelops the cord, so that no injury to the lumbosacral region can occur without an accompanying injury to the cauda; but the cauda below the termination of the cord may be injured alone. In such cases, from focal signs alone, it may be impossible to determine whether the injury is to cord or to roots. If, for example (see Fig. 48), the cauda is subjected to complete transverse compression just below the last sacral segment, the fourth, and possibly the third, lumbar nerve will be deprived of its conductivity, and the loss of function will be the same as though the segments themselves had been injured.

The compactness of the constituents of the cord, the enveloping of it by the long roots, and the fact that the bone lesions are usually extensive make topical diagnosis of injuries in this region very difficult. The lesions themselves are comparatively frequent. More than half of all fractures and dislocations of the spine take place below the tenth dorsal vertebra. The vertebræ most frequently involved are the last two dorsal and the first lumbar. Injury of these three give mixed cord and cauda lesions. Lower down, pure cauda lesions occur. Gurlt's tables show, of 270 cases, 27 cases of fracture of lumbar vertebræ, and 17 of lumbar and dorsal together. In Burrell's 82 cases, 23 were of the lumbar region.

These injuries result from extreme violence. Men in the

active periods of life are almost invariably the victims. The bone lesion involves the vertebral bodies chiefly, and fracture and dislocation usually occur together. Deformity is marked.

The chief object in diagnosis is to determine whether cauda or cord have been chiefly or solely affected. In cauda lesions, naturally, the prognosis is much better. There are the following points of distinction: The external deformity may give a clew as to the part of the canal infringed upon. If the compression is at or below the second lumbar vertebra, the lesion is of the cauda only. Cord lesions are, as a rule, painless. Cauda lesions are apt to be painless at first, but sharp pains shooting down the legs frequently set in with recovering function. In cauda lesions, also, the anæsthesia is more widespread, more irregular, and less profound than in cord lesions. There is, in general, less symmetry in cauda than in cord lesions. Involuntary twitchings of the muscles is characteristic of cord lesions, and atrophy is more pronounced in these than in those of the cauda.

The course of the case may throw some light on the diagnosis. As in other injuries of the cord, the early symptoms do not necessarily represent the extent of the permanent damage. For example, an injury to the conus, in addition to the paralysis of bladder and rectum and the anæsthesia characteristic of conus lesions, may present a general weakness of the legs, diminution of knee jerk, and scattered areas of anæsthesia in the legs. In the course of a few days these latter symptoms, indicating injury to the cauda, may greatly improve, or pass away entirely, leaving the conus symptoms alone as permanencies.

Fractures of the sacrum or of the pelvis may cause symptoms suggestive of intravertebral lesions. Thus, a fracture of the sacrum may compress the roots going to form the sacral plexus, or compression or traction on the sciatic may cause

paralysis of that nerve. The latter diagnosis presents no difficulties. In the former, the surgical examination of the bones, externally and by rectum, and the frequently unilateral character of the symptoms, are usually sufficient to prevent confusion.

From the charts on motor and sensory localization the symptoms of injury to the lumbar and sacral segments can

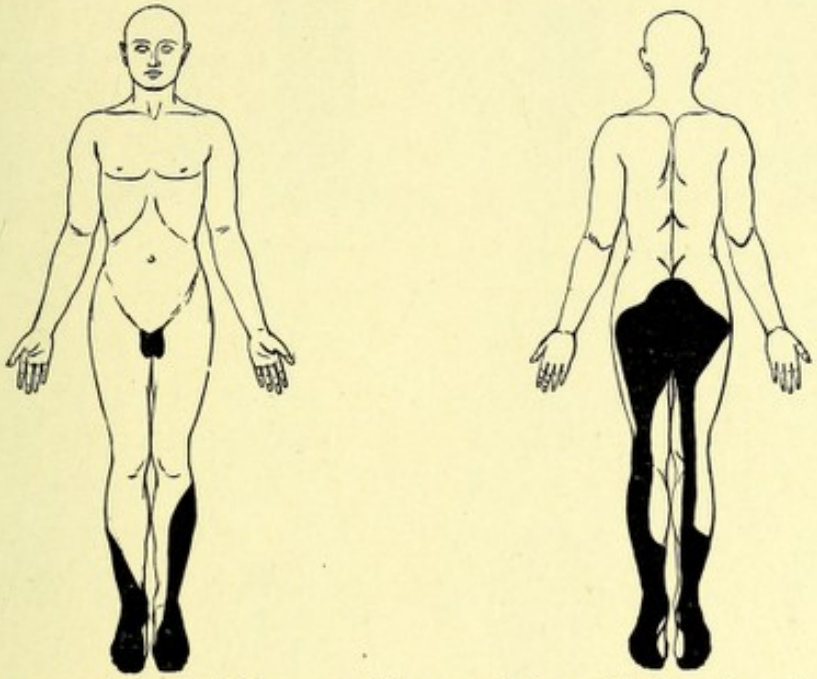
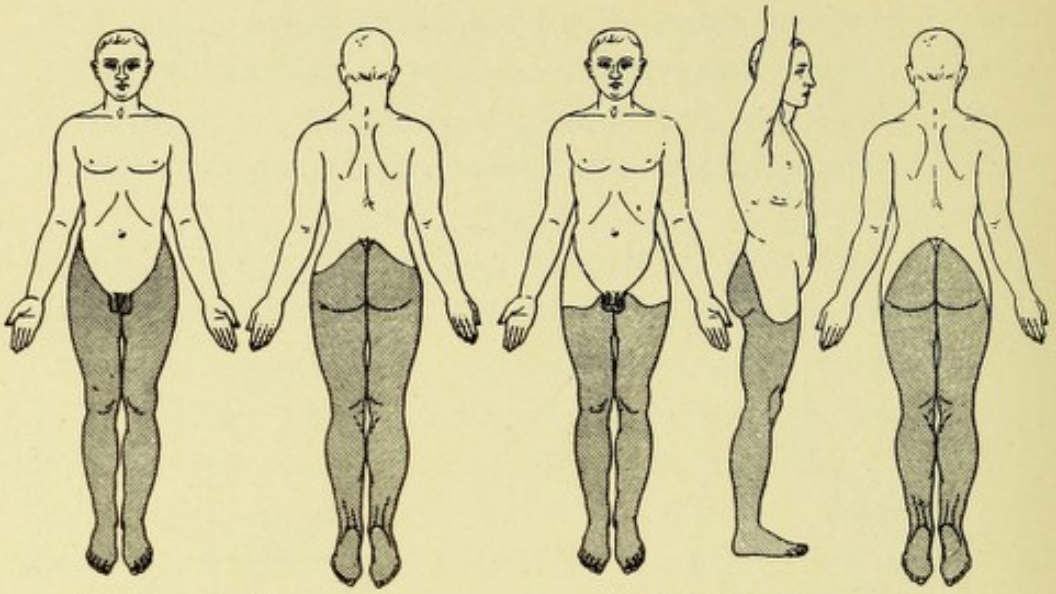
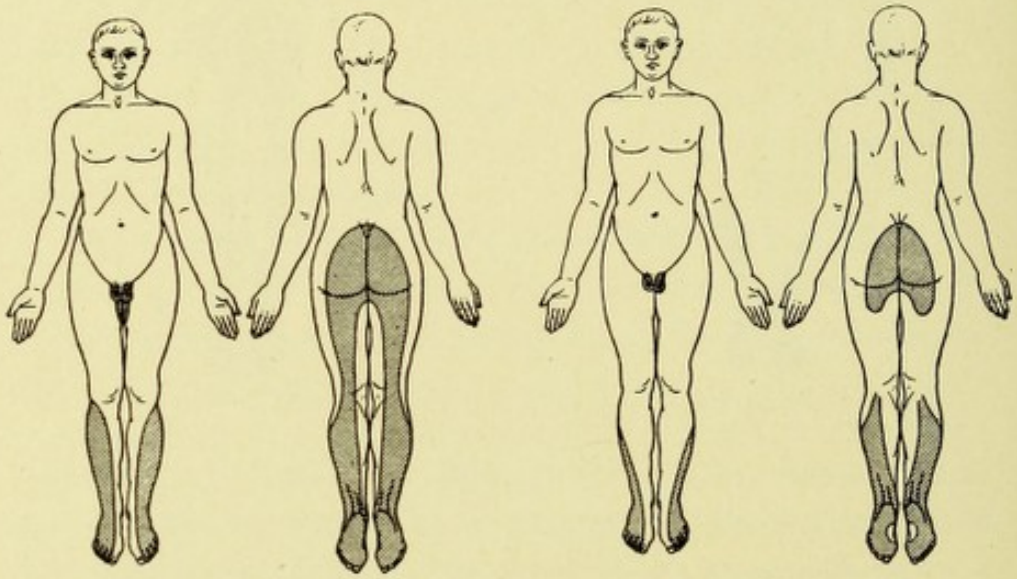


FIG. 49.—Anæsthesia in a case of fracture of the twelfth dorsal vertebra.

readily be deduced. In injury to the upper half of the lumbar cord there is complete paraplegia. This may be produced by fracture dislocation of the tenth and eleventh, or eleventh and twelfth dorsal vertebræ, or by fracture of the twelfth dorsal vertebra (Fig. 49). In injury to the lower half of the lumbar region of the cord, sensibility is preserved on the anterior aspect of the thigh, and the adductors and quadriceps are only partially paralyzed, or not paralyzed at all. The knee jerk is not lost if the lesion is below the second, third, and fourth segments, and if the roots from these segments are not



A. Second lumbar segment. B. Third lumbar segment.



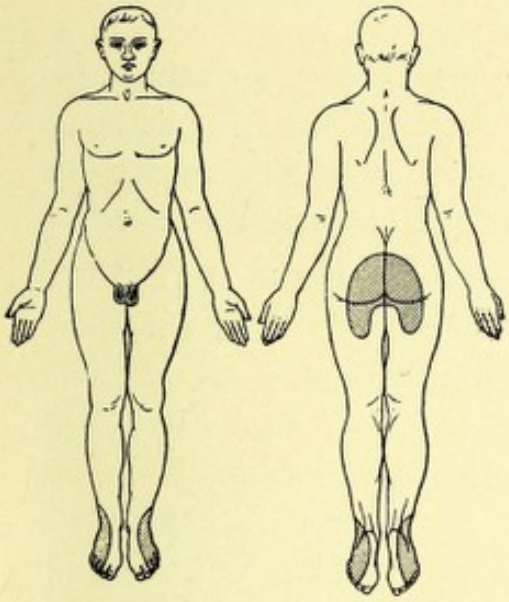
C. Fifth lumbar segment. D. First sacral segment.

FIG. 50.—A.–D. Anæsthesia in lesions of lumbo-sacral region. (Müller.)

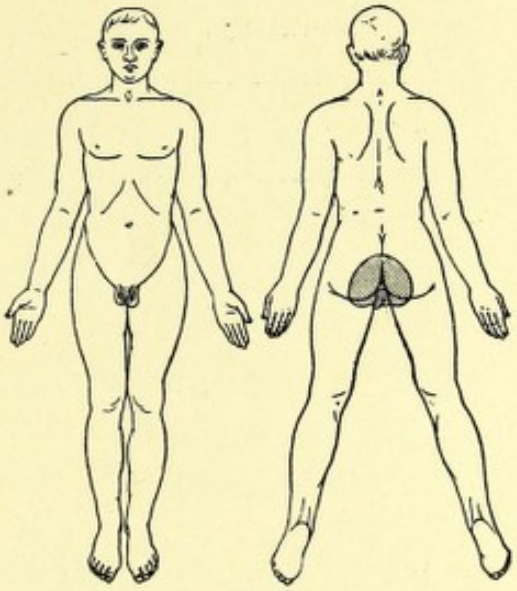
affected. Rare cases of conus or cauda lesions have been reported, in which, with absent knee jerk, there was a foot clonus.

Fractures and dislocations of the first and second lumbar vertebræ are the ones usually responsible for sacral lesions. Injury of the two upper sacral segments gives the anæsthesia

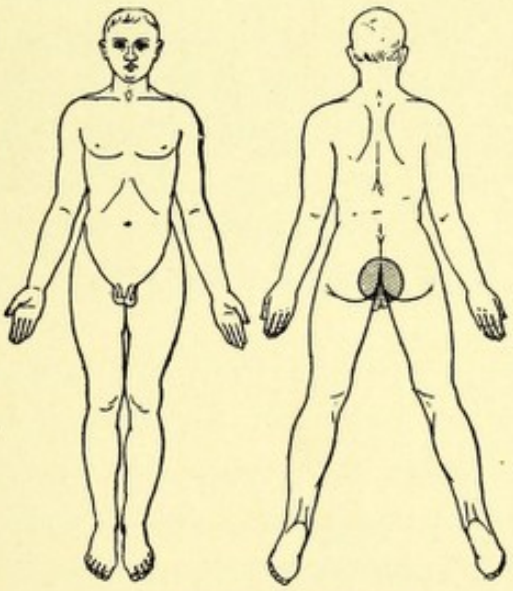
and paralysis indicated on the charts, without loss of knee jerk, provided the lumbar roots are not affected. These two segments have been termed the epiconus. Injury of it causes paralysis, especially pronounced in the peronei muscles.



E. Second sacra segment.



F. Third sacral segment.



G. Fourth sacral segment.

FIG. 50.—E.–G. Anæsthesia in lesions of lumbo-sacral region. (Müller.)

Pure conus lesions are characterized by lack of paralysis of the limbs, by paralysis of the bladder and rectum, a saddle-shaped area of anæsthesia over the buttocks, and with anæsthesia of the penis and scrotum. In all pure sacral lesions the testicular sensation is retained, as the testicle receives its sensory innervations from the lumbar plexus.

The center for erection being in the second sacral segment, and for ejaculation in the third sacral, if the lesion is below these segments procreation is possible. Müller reports the case of a patient with traumatic paralysis of bladder and rectum who became the father of two children. Müller believes that preservation of procreative power points to a low-lying lesion in the conus, rather than to a cauda lesion.

Prognosis in Fractures of the Spine

In a certain small percentage of injuries to the spinal column the cord escapes. The prognosis of such cases concerns surgery. Fractures and dislocations with involvement of the cord are always most serious injuries. Of 44 cases received at the Hudson Street Hospital during the years 1896–1904,[1] 25 died, 8 were transferred (final outcome unknown), 9 were discharged improved, and 2 were discharged cured. Of 8 cases received at St. Luke's Hospital between the years 1895–1901, 2 died. Most of these were received some time after the injury.

Gurlt's famous statistics, from 270 cases, show a mortality in eighty per cent, and Burrell's, from 82 cases, a mortality of seventy-eight per cent. Of Burrell's twenty-two per cent of cases in which death did not occur, one-half the patients were able to resume work.

This mortality is high. But it should be remembered that

[1] For the statistics thanks are due to Dr. P. R. Bolton.

in many cases, other severe injuries, in themselves sufficient to cause death, are often associated with the spinal fracture. On the other hand, some patients recover from the immediate effects sufficiently to leave the hospital. They are then lost to view, though a considerable portion carry with them such serious conditions as bladder or rectal paralysis, or paralysis of the legs, from which, doubtless, many eventually die. Due allowances being made for statistical errors, it can safely be said that the direct or proximate mortality of injuries to the spinal cord is well over seventy per cent. Some writers, on the basis of statistical compilations, have endeavored to prove that in operated cases the mortality was less. Their conclusions are far from convincing. It is hard to believe that such operations, undertaken early, turn the stream of life in favor of the injured man. Death occurs within five days in more than half of the fatalities.

In the prognosis of individual cases the extent of injury and the region of the cord affected are the most important. The extent of the injury is shown, to a certain extent, by the symptoms. A total lesion at any level, except in the lowest segments, is certainly fatal, sooner or later. But total lesions are not the rule. More commonly some function remains, as shown by anæsthesia being only partial, or by some motion still being possible in affected parts, or by the preservation of sphincter control. Naturally, the fewer symptoms present, the more reason is there to suppose the lesion not of great extent. Absence of bladder paralysis is a most favorable sign, such cases rarely proving fatal, and usually being susceptible of great improvement in the symptoms which exist. It is also a favorable sign when motor paralysis is partial. Preservation of sensibility indicates a partial lesion, but less reliance is to be put upon it than upon the others from the point of view of prognosis. Bedsores are always serious, and every patient

with them is in danger. Extreme age, or previous ill health, compromise the prognosis. Except in cases of very partial lesion, or in those which are quickly fatal, the prognosis cannot be reasonably definite until some little time after the accident. Every amelioration of symptoms gives ground for hope that the permanent lesion is to be less severe than the early symptoms indicated. The sooner the symptoms become less or disappear, the better the prognosis. Pain and tenderness in the legs are often heralds of restoration of function. If after ten days or two weeks there has been no improvement, the chances for substantial recovery are very much less. This is not universally so, as in some cases no improvement is noticeable until weeks or even months after the accident. But, as a general rule, if a relatively good recovery is to be made, indications of it are apparent within two weeks of the accident. Once improvement has begun, it may progress indefinitely for years.

With the same degree of injury, the higher it is situated the worse is the prognosis, severe lesions in the cervical region having the highest mortality, those in the lumbosacral the lowest. Fractures of the first eight dorsal vertebræ have as high a mortality as the cervical. This is due to the fact that complete lesions are relatively more frequent in the dorsal region than elsewhere. Injuries above the fourth cervical segment are immediately fatal. In the lower-lying cervical segments extensive injuries are rarely survived more than a few days, the patients sometimes dying in from twenty-four to forty-eight hours. In rare cases, however, with symptoms of extensive mutilation in the cervical region, death does not occur, and a certain degree of recovery takes place.

Thus, a young man, in diving, fractured the fifth and sixth cervical vertebræ. There was immediate paralysis of arms and legs and of the bladder, and anæsthesia, profound in the legs but indistinct in its upper boundaries. The patient was very ill for a few days, with

temperature 106° F. and pulmonary symptoms. There soon ensued some slight improvement in paralysis and anæsthesia. Six weeks after the accident laminectomy was performed. The improvement in the paralysis continued, the patient eventually recovering (four years) sufficiently to sit in a wheel chair, and to hold books in the hand.

In another patient, seen two weeks after the accident, the symptoms were almost the same as the preceding, except that no improvement whatever had occurred. At the end of seven months the man was still living, but had improved but little.

In many cervical cases it is evident almost at the outset that the injury is not extreme, and that life is not threatened. Such patients usually stay in bed a week or ten days, and then are able to get about. The legs may remain weak and stiff for a long time, and there usually remains some permanent paralysis in the upper extremities. In many such cases the bladder is affected temporarily or not at all. In most dorsal lesions the patients improve little or not at all, the bladder paralysis results in cystitis, bedsores develop, and death ensues, after weeks or months, from exhaustion and septicæmia. In my experience lesions in this region are more uniformly fatal than in any other, though death is often long delayed.

In injuries to the lumbosacral cord, death, if it occurs, is usually delayed many months. In this region partial lesions are also frequent, but the degree of functional recovery is less satisfactory than in cervical cases, there usually remaining some drop foot and serious bladder disturbances.

Injuries to the cauda equina are spoken of, in many textbooks, as universally hopeful, especially if operated upon. While such injuries have a better prognosis, both as to life and as to functional recovery, than cord injuries, they are sometimes fatal after a long period of time, and quite frequently improve little or not at all. The chances for recovery are much improved by operation, but even after operation many cases show no change.

In the patients who do not die from fracture of the spine functional recovery is rarely complete. Even after slight injuries some parts remain weakened, and spastic if the injury were above the lumbar enlargement, or flaccid if at or below the lumbar region.

Many patients can get about on crutches, some with only a cane, and others—but these latter are more rare—can walk unassisted. In one case, after a partial paraplegia from a fracture of the sacrum, the patient, at the end of a year, was completely restored, so that he could walk and play tennis almost as well as before. A number of cases are on record in which, even with slight paralytic effects, the patients begot children. Probably in ten per cent, or perhaps a little more, of all cases of fracture of the spine the patient is able eventually to resume his former occupation. On the other hand, many live badly crippled and disabled for years.

Wounds of the Spinal Cord

Gunshot Wounds.—Bullet wounds of the spinal cord are apt to be associated with lesions in the viscera. Except in the case of balls entering from behind, the bullet usually, before reaching the spine, penetrates important organs in the abdomen or thorax. The bullet may impinge directly upon the spinal cord, lacerating it or cutting it in two. Frequently it works its damage by fracturing the bone, splinters of which pierce or compress the cord. The bullet may then pursue its course, finally lodging far from the spine. The x-ray is extremely useful in determining the position of the bullet's final lodgment. If the bullet does not remain in the spinal canal, the symptoms it causes are the same as those of contusion and laceration of the cord generally. If it remains there, it may become the starting point of suppuration, which usually re-

mains local; or of adhesions, which cause a progressive compression of the cord.

Thus a man was shot in the abdomen and became immediately paraplegic. After a number of weeks he began to have very severe pains in the legs. An x-ray showed the bullet in the fourth lumbar vertebra. A laminectomy, performed by Dr. W. T. Bull, showed the ball lodged in the body of the fourth lumbar vertebra, but projecting into the canal. It had formed the starting point of adhesions, which were compressing the cauda equina through the dura. The bullet was removed and the adhesions broken up. The pain became less within a few days and finally disappeared. When last heard from, the patient was regaining motor power. (Dr. Alvarez.)

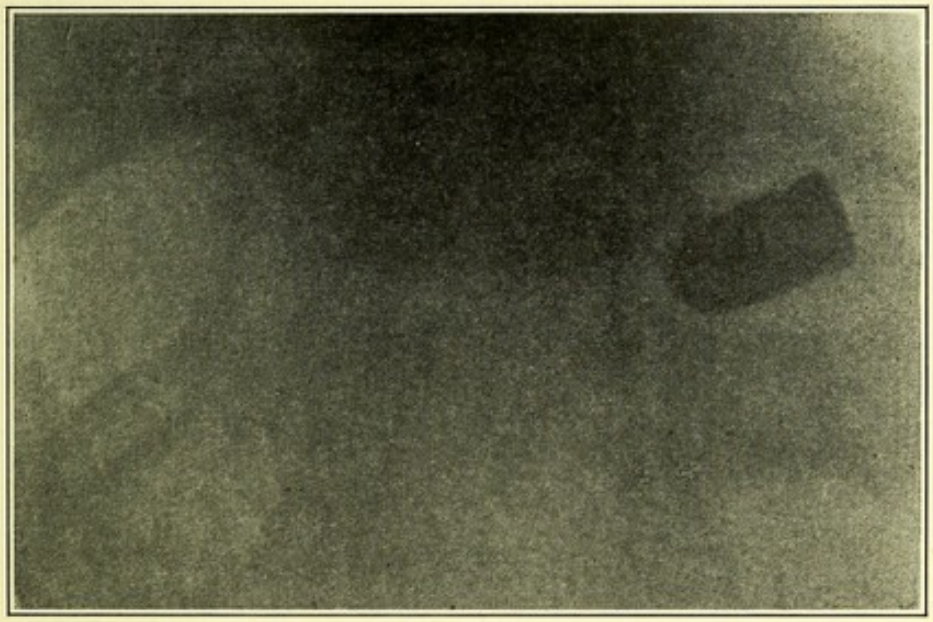


FIG. 51.—Bullet wound of dorsal region. Symptoms of spastic paraplegia which rapidly improved after operation.

The symptoms of bullet wounds are those of spinal-cord injury generally. They vary in extent and character with the extent and character of the lesions.

Partial lesions are not rare. It is possible that a bullet striking the vertebra may cause central hemorrhages within the cord. The same rules for localization hold good as for other

injuries. It should not be forgotten, however, that the localizing signs of anæsthesia, paralysis, etc., may point to a lesion a little higher up in the cord than that caused directly by the bullet. This is due to the extension upward, from the point where the cord was directly injured, of softening or of hemorrhage.

Stab Wounds.—Stab wounds of the spinal cord occur with special frequency in the cervical region. The wound of the cord may be on the same or on the opposite side of the external injury. The symptoms are usually of the Brown-Séquard type. The mortality, as ascertained in 46 cases reported by Roeseler, is, for the two upper cervical vertebræ, seventy-one per cent; for the fourth upper cervical vertebra, fifty-three per cent; for the three lower cervical vertebræ, twenty-three per cent, or forty per cent for the whole cervical region. In the dorsal region the mortality is thirty-one per cent.

In recovery, the first symptoms to disappear are the bladder and rectal paralysis, and then the paralysis of the limbs, the leg recovering more completely than the hand. On an average, the patients who recover are able to walk in eleven weeks. The recovery of motor power is usually not perfect, some weakness and rigidity remaining. The power of copulation and procreation may be preserved. In fatal cases death is due to paralysis of respiration, to bedsores, and to cystitis.

Tuberculosis of the Spine—Traumatic Pott's Disease

Spinal caries, or Pott's disease, consists in a chronic focal tuberculous inflammation of the vertebræ, chiefly of the bodies. It may be associated with abscess. When sufficiently pronounced the vertebra crumbles. Sometimes by direct pressure, but generally by extension of the tuberculous process to the

external surface of the dura mater, the spinal cord is involved. Such involvement, most frequent in the dorsal region, occurs in about one-third of all cases. It consists partly in mechanical compression, partly in œdema brought about by disturbed circulation.

Symptoms.—The symptoms are referable to the bones and to the spinal cord. They come on weeks or months after the injury. The bone symptoms consist in pain, especially on movement, tenderness and rigidity of the back, all of which vary greatly in degree. The pain may at first be referred to other parts than the spine, e. g., abdominal pain, in dorsal caries; pain in the knee, in lumbar caries. Deformity, sometimes absent, is usually easily distinguishable. A sharp kyphotic protuberance of one or two spines is the most characteristic deformity.

The spinal cord symptoms are those of partial compression myelitis, varying in distribution and character with the region of the cord affected. In the dorsal region, the commonest, they consist in spastic paraplegia, the legs are weak, and the reflexes are increased. There is diminution of sensibility rather than complete anæsthesia. The bladder may or may not be involved. Pain may be confined to the back or radiate down the back. Bedsores and atrophy herald the later stages.

Ætiology.— In a large proportion of cases of spinal caries injury is advanced as a cause. In a large proportion of these, again, there is no reasonable connection between the trauma and the spinal disease. In a certain small percentage, on the other hand, the relation between the two is undoubted. Spinal caries, being a tuberculous disease, the lodgment of the tubercle bacillus in the body is a necessary preliminary to tuberculous inflammation. The injury, consequently, is only an exciting cause. At the same time, persons in previous good

health, and without the history of tuberculosis, develop spinal caries as the result of injury.

The injury itself consists in a blow on the back or a sudden severe strain of the back. No age is exempt, though young persons are the most frequently affected.

Prognosis.—The prognosis is always serious. Whitman says that twenty per cent die during the active stages of the disease or during the first few years. Recovery, on the other hand, occurs.

Treatment.—The treatment of Pott's disease belongs to orthopedic surgery. Rest, either in bed, with splints, or plaster jackets, is the essential requisite.

Traumatic Spondylitis (Nontuberculous)

Kümmel has described a traumatic vertebral affection which in many ways resembles Pott's disease, but which, in all probability, consists in a nontuberculous rarefying osteitis. The ætiology is similar to that of the tuberculous form. The vertebræ between the third and seventh dorsal are the ones most frequently affected, although affections of both cervical and lumbar have been observed.

After the injury there is pain in the back, of varying degrees of severity, which lasts from two to eight days. This gradually disappears, but after a few weeks, or even months, it returns. It is severe, and is referred to the injured spot and to the nerves radiating from it. The parts are painful to pressure, and any movement aggravates the pain extremely. Gradually a kyphosis appears. This involves several vertebræ, making a more diffuse deformity than is seen in Pott's disease, and the angle formed by the kyphosis is less acute. If the patient is suspended, the general kyphosis disappears, though one vertebral spine still remains prominent.

For the pathological anatomy it is assumed that by the trauma the vertebræ are sufficiently injured (possibly fractured) to cause interferences in nutrition, which result in a rarefying osteitis, with consequent giving away of the spinal column.

Varying with the severity of the original injury, the cases have different courses. Some patients have severe and continuous pain from the outset. In these there is doubtless fracture or serious compression of the vertebræ, without, at first at least, cord involvement.

The spinal cord is usually not affected in this disease, though cases are on record in which both paralysis and involvement of the sphincters have ultimately appeared.

The prognosis is much better than in Pott's disease. Most cases, after months, recover very satisfactorily, and are able to resume work.

The diagnosis rests between Pott's disease, fracture of the spine, and back sprain with nervous symptoms of functional character. The differences from Pott's disease have been given. In severe cases the diagnosis from fracture of the spine is difficult or impossible. The absence of initial spinal-cord symptoms shows that the fracture, if one exists, does not necessarily entail serious consequences. From lumbago it is differentiated by its course, and by the absence, in lumbago, of actual spine deformity. The presence or absence of functional nervous symptoms does not materially help the diagnosis, as they may occur in either condition.

The treatment is practically the same as for Pott's disease.

CHAPTER VII

INJURIES TO THE SPINAL CORD—*(Continued.)*

Injuries to the Cord without Bone Lesion—Hematomyelia—Concussion—Treatment of Spinal-cord Injuries.

UNDER this heading only two conditions require consideration, viz., primary hematomyelia, called primary to distinguish it from the hemorrhages secondary to bone injuries, and concussion of the cord.

PRIMARY FOCAL HEMATOMYELIA

Primary hematomyelia may be, as shown in the remarks on pathology, either focal or disseminated. The disseminated form cannot with certainty be recognized during life, so the following applies to focal hematomyelia only.

Frequency.—This affection is certainly more frequent than is generally recognized. It receives brief if any mention in text-books on surgery. Yet of 21 cases of spinal-cord traumata examined by Thorburn, 6 were interpreted as examples of hematomyelia. In none of these were evidences of fracture present during life; in the only fatal case the column was found after death to be intact. Of 7 fatal cases of spinal injuries examined by Parkin the column was normal in 1. Of Stolper's 22 autopsies on various spinal-cord injuries 2 were examples of primary focal hematomyelia.

But the percentage of primary cases, as shown by autopsy, by no means expresses their relative frequency, because this is the form of injury to the spinal cord in which recovery

occurs most often. Neither can we as yet determine the frequency from clinical reports, because it is only recently that attention has been directed to the most important diagnostic sign, i. e., dissociated anæsthesia. When search for this symptom becomes part of the routine examination in surgical wards, we may expect to hear more of primary focal hematomyelia from trauma.

Ætiology.—With the exception of pistol-shot wounds primary focal hematomyelia results almost exclusively, when the exact cause can be ascertained, from sudden forced flexions or extensions of the neck. As examples may be cited falls on the head, the dropping of heavy weights on the head, or, as occurred in three personally observed cases, by diving into shallow water. Thoburn explained the mechanism of the lesion's production by a momentary dislocation of the cervical vertebræ. In view of the already stated fact, that the symptoms in these cases differ materially from such as are seen when fracture or dislocation can be proved, and that in the autopsies there were absolutely no traces of any change of position in the vertebræ, this theory is hardly satisfactory. I believe that the hemorrhage occurs not from external compression but from a rupture of one or more of the blood vessels of the central gray matter, brought about by the stretching of the cord during the forced flexion or extension of the neck.

Symptoms.—As neither fracture nor dislocation belongs to primary focal hematomyelia, but little need be said as to these conditions. There may be œdema, subcutaneous extravasations, stiffness, pain, etc., in the neck, the results of the sudden and excessive strain to which the cervical vertebræ and superimposed structures have been subjected; but crepitus, false motion, or pathological irregularities in the vertebral spines are conspicuous by their absence. The symptoms directly referable to the spinal cord, as they are the results of an

internal and generally circumscribed injury, differ from those of crushes of the cord in which both white and gray matter are commonly implicated. If the hemorrhage is so extensive that it bursts beyond the gray matter and involves the cord, either directly or by pressure in its whole cross section, the clinical evidences of paralysis, anæsthesia, etc., would be identical with those of a transverse lesion. When, however, as is usually the case, the hemorrhage exceeds the gray confines but little or not at all, the clinical picture has a distinct individuality, of which the most salient features are an initial motor paralysis which soon becomes modified, and anæsthesia for temperature or pain, or both, without impairment of the sense of touch.

The paralysis of voluntary motion, with few exceptions, ensues instantly upon receipt of the injury. The muscles involved at the level of the injury (since the hemorrhage is most constant in the lower cervical region) are those of the forearm and hand, and sometimes of the upper arm; those below, of the lower extremities. The paralysis of the upper extremities has a different anatomical basis from that of the lower. The first is due to the blood being poured directly into the spinal nuclei of the brachial nerves, thus constituting a destructive lesion. From such a condition perfect recovery is improbable, and, in fact, most of the patients remain permanently paralyzed, though the paralysis may be very limited in some of the muscles of the upper extremity. The paralysis of the legs, on the other hand, is the result of conditions often permitting recovery. The most feasible explanation of this paralysis, from which recovery may be expected, is that the hemorrhage, although occupying the central portions of the cord, exerts sufficient pressure, or is followed by œdema sufficient to compress for a time the pyramidal tracts which lie a little distance from it, and which are carrying the long cerebral fibers des-

tined for the muscle nuclei situated at lower levels. When the hemorrhage ceases, the commotion it has caused in its vicinity subsides and the pyramidal fibers are permitted to resume their wonted function. The distribution of the paralysis varies with the situation and extent of the hemorrhage. If below the first thoracic segment—an unusual situation—the arms escape. If above the second thoracic segment, the paralysis affects the muscles whose nuclei are below the upper limit of the hemorrhage. If the hemorrhage is very small and on one side of the cord only, the paralysis may affect one arm chiefly or only; if larger, but still confined to one side, there is spinal hemiplegia.

The condition of diplegia brachialis traumatica probably belongs to primary hematomyelia. In this condition, after an injury in the cervical region, there develops at once a flaccid paralysis of both arms, the legs escaping, or being only slightly involved. There is severe pain in the neck, which radiates down the arms. The common pathological explanation of these cases is extramedullary hemorrhage; or tearing or crushing of the nerve roots.

How a hemorrhage situated without the cord could cause such symptoms it is difficult to see; neither is it probable that all the ventral roots going into the formation of the brachial plexus could be so seriously injured as to cause bilateral brachial palsy, without at the same time there being other conspicuous symptoms on the part both of the spinal cord and of the spinal column. In contrast to these improbable hypotheses is the one which postulates that the lesion consists of hemorrhage among the nuclei of the brachial nerves. This explains perfectly all the symptoms, and its correctness seems almost incontestable in view of the demonstrated frequency with which hemorrhage involves the gray matter of the cervical region.

In many cases of hematomyelia the hemorrage is sufficient to cause, at first, paralysis of both arms and of both legs. When the patient is seen immediately after the accident the paralysis is flaccid in character. The muscles are soft and yielding, without rigidity, and the paraplegia is complete. The knee jerks are diminished or lost. When the lesion is very small, the knee jerks may be hypertypical or exaggerated at once. In the lower extremities the paralysis soon undergoes a change, and from being flaccid it becomes spastic. The knee jerks, if they were diminished or lost, return and become exaggerated, and ankle clonus is added.

With the establishment of the spasticity strength returns to the legs. Some patients are able to walk a few weeks after being completely paraplegic. The recovery in the legs comes sooner than that in the arms; in the arms it is also less perfect than in the legs, there usually being left a permanent atrophic paralysis in some of the brachial muscles.

A patient recovering from this affection may present a picture very similar to that of progressive muscular atrophy (amyotrophic lateral sclerosis), viz., a paralysis of the upper extremities of the peripheral neuron type, with atrophy, fibrillation, and degenerative electrical reactions, and of the lower extremities of central neuron type, with rigidity, increased knee jerks, and ankle clonus.

The clinical characters of the impairment of cutaneous sensibility in primary focal hematomyelia vary. We expect that in all cases, whether chronic or acute, in which a lesion is confined to the structures surrounding the central canal, the ability to recognize thermic and painful stimuli will be impaired or lost, while tactile sensibility remains normal Such is the condition in primary focal hematomyelia, except in the few cases in which the blood clot is so extensive that it causes the total anæsthesia of a transverse lesion.

The hemorrhage may be of considerable size without doing so, however.

In characteristic cases, while sensibility to touch is not affected, there exists thermo-anæsthesia or analgesia, or both together. The distribution of these disturbances is in general the same as that for the total anæsthesia of transverse lesions.

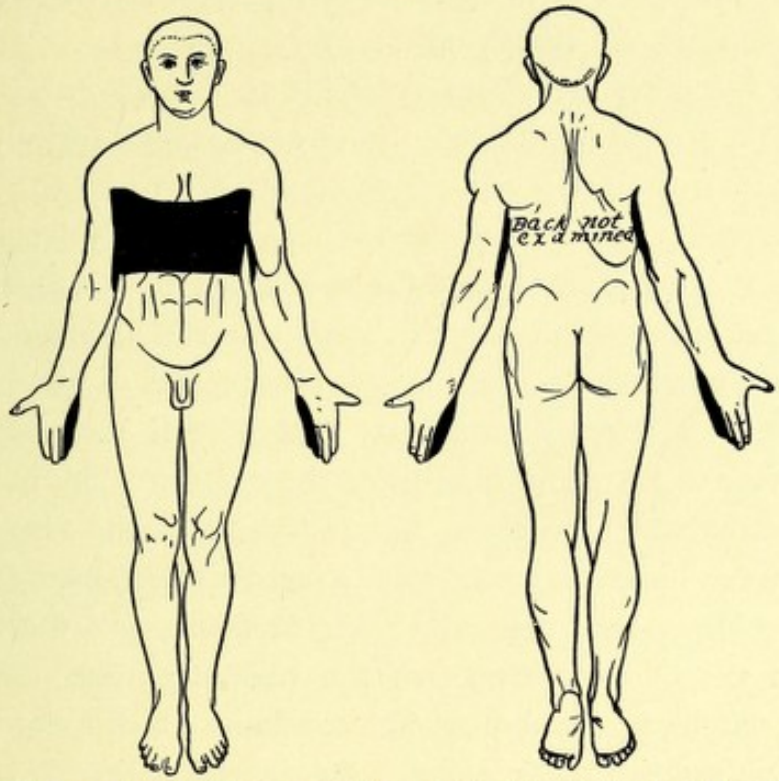


FIG. 52.—Loss of temperature sense in a case of primary focal hematomyelia from injury. (Verified by autopsy.)

The Brown-Séquard type is often seen, viz., a motor paralysis of one arm and leg, with loss of pain sense and temperature sense in the arm and leg of the opposite side. The degree of thermo-anæsthesia and analgesia is not constant. As a rule, analgesia is the lesser of the two; in recovery, also, it is the first to improve or disappear. From personal experience I can speak only of the loss of temperature sense. That has generally been, for the first few weeks after the accident, complete,

so that the patients were not only unable to distinguish between heat and cold, but felt no discomfort from contact with the test tube of boiling water, although other kinds of painful stimuli were appreciated at their true value. When the symptom was incomplete at first, or when time had permitted it to become so, it appeared as delayed perception. The tendency of these affections of the pain and temperature sense is toward improvement, and sometimes recovery is perfect.

Of the other symptoms referable to the sphere of sensation, pain is most prominent. Some cases run an entirely painless course; in others, pain, both spontaneous and on motion, is referred to the site of the lesion. The sharp lancinating pains down the arms or legs, the so-called irritative root symptoms, are not frequent in pure central traumatic hematomyelia; neither are numbness, tingling, nor other forms of paræsthesias frequent. The patient may say that he felt such perverted sensations for a short time after the accident, but that they were temporary; he most frequently states that he "feels everything," being ignorant of the fact that he cannot tell heat from cold, or recognize either one of them. As the hemorrhages are in the vicinity of the centers of the ciliospinal reflex, pupillary phenomena are constant. If the amount of blood on the two sides is equal, the degree of myosis on both sides is equal. More frequently, however, the myosis, like the paralysis, is more pronounced on one side. Other evidences of lesion of the ciliospinal centers are less frequently mentioned.

The other symptoms which sometimes occur in primary focal hematomyelia may be dismissed with brief mention. Thus, retention of urine, incontinence of fæces, bedsores, priapism, œdema, cyanosis of the extremities, distention of the abdomen, chills and fever, all have been observed. These are the general symptoms of any injury of the spinal cord, and one

or all of them may be present in the special affection under discussion. In it, however, they are generally characterized by short duration and by one or more of them being absent. Thus, the incontinence of fæces may stop in a day or two; control of the bladder is usually slower in returning, but it generally does return. Bedsores do not occur in mild cases.

Diagnosis.—The nature of the accident, the extent and character of the initial palsy, the sphincter defects, the absence of external signs of bone lesion, may all be the same in other kinds of spinal-cord injuries as in primary focal hematomyelia. The chief distinguishing guide, immediately after the accident at least, is to be sought in the sphere of sensation. When the other symptoms are in accord with an acute injury, and when there is anæsthesia for pain or temperature, in characteristic areas, without any impairment, of the sense of touch, the diagnosis can be made, I think, without hesitancy. The fact that such a dissociation of sensation occurs in other spinal-cord affections, in certain forms of neuritis, or even in hysteria, can hardly cause confusion to anyone who looks at the case from every point of view. The chief difficulties to certainty of diagnosis will be met in cases in which the hemorrhage is very small and in those in which it is very large. In the first category it is entirely possible for a hemorrhage confined to the anterior horns to cause no sensory symptoms, and few or no symptoms of general commotion, and the diagnosis would therefore have to be made from the paralysis and wasting of one upper extremity. Similarly, if the hemorrhage were in a posterior horn, the resulting sensory defect might easily be overlooked.

Thus, a man was injured in an elevator shaft by the balance weight coming down slowly, so that he could not extricate himself. With head sharply flexed on the chest, he was pressed down to the floor. His symptoms were: Laceration of the scalp, where the weights

had pressed; severe pain and tenderness in the neck, with limitation of movement; tingling in the tips of the fingers of both hands. He could move all the extremities freely, although they felt weak; he could pass the water normally; the bowels moved regularly. He remained in the hospital for two weeks, the treatment being directed to the stiffness and pain in the neck and to the scalp wounds. He had no bedsores. When discharged from the hospital the pain in the neck was still troublesome, and he felt weak generally, conditions which have been gradually improving.

Examination, January 24, 1899, Vanderbilt Clinic, two months later: The neck is held somewhat forward on the chest and is very stiff. Attempts to cause movement in it in any direction, either on the part of the patient or of the examiner, cause sharp pain. There are, however, no demonstrable evidences of fracture or dislocation in the vertebræ. The only positive indications of destructive lesion in the nervous system consist in a patch of anæsthesia over the right shoulder and a foot clonus on the left side. The anæsthesia (Fig. 53) corresponds to the deltoid region. In the area marked black on the chart it is profound and total, so that touches are not felt, pin pricks cause no pain, and temperature sense is abolished. Surrounding this patch of total anæsthesia in front, behind, and above there is a narrow zone of diminished sensibility, which merges into normally sensitive skin. Below, the whole right arm is not so acutely sensitive as the left, although there is no objective anæsthesia. In the trunk and legs feeling is the same on the two sides. The foot clonus on the left side is rhythmic and fairly forcible. It is not particularly persistent, and is easily exhausted. The tendon reflexes are everywhere very lively. The left knee jerk is more active than the right; elsewhere there is little comparative difference between the tendon reflexes of the two sides. The man is weak generally, but he is not conscious of, nor was I able to demonstrate, a greater weakness on one side than on the other. Fibrillary twitchings manifest themselves upon use of the muscles of the upper extremities. They seem equal on the two sides. There is no atrophy. The measurements of the limbs of the left side are somewhat less than those of the right, but not beyond physiological limits. There have never been rectal or vesical difficulties.

In contrast to the minute lesions are the extensive ones, which lead quickly to death. It is entirely possible for a large central hemorrhage to have the same effect, by pressure or by

actual destruction, in causing symptoms, both motor and sensory, identical with those of general crushes. It seems, however, that such cases are unusual; that the fibers for sensibility, here as elsewhere, are much more resistant to pressure effects than are those of motion. Between these two extremes are the

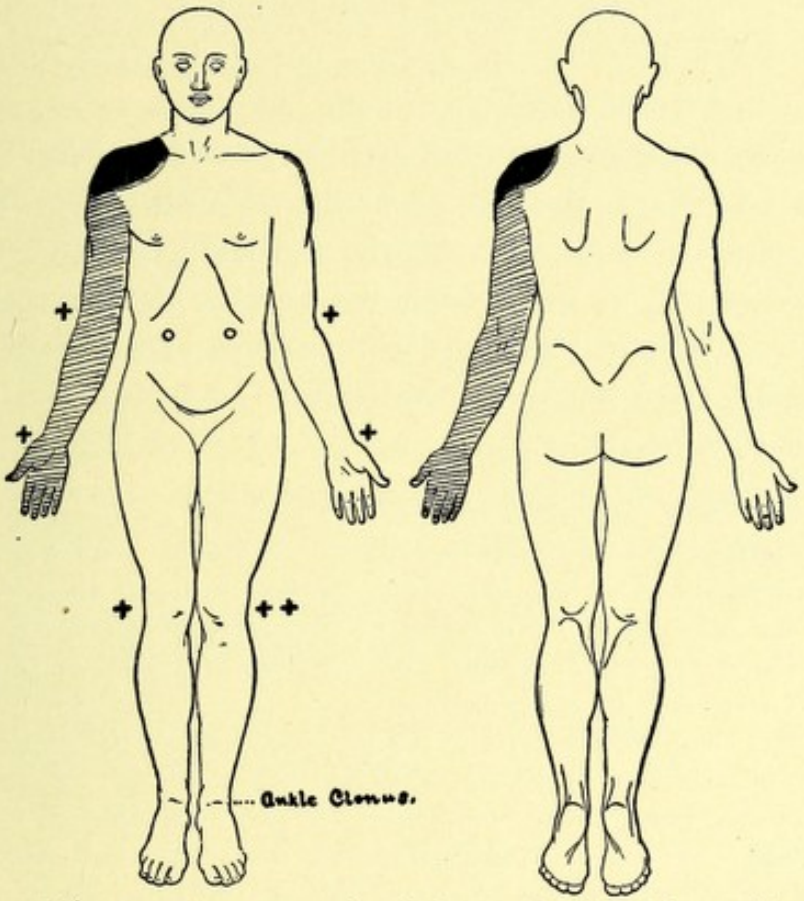


FIG. 53.—Diagrammatic representation of the anæsthesia and the condition of the deep reflexes in a case of primary focal hematomyelia from injury.

most frequent cases of primary focal hematomyelia, which, in addition to the sensory symptom complex, have other characteristics of their own. Among these may be mentioned the strong tendency toward improvement of muscular power, more rapid in the legs than in the arms; frequent unilateral distribution; the comparatively quick return of sphincter control, and the general absence of bedsores.

The condition known as diplegia brachialis traumatica has usually been explained by assuming an extradural hemorrhage. In the absence of post-mortem evidence of the existence of such a lesion as an isolated condition, and in the light of our present knowledge of hematomyelia, it seems to me that the clinical condition must henceforth be recognized as resulting from hemorrhages into the cord and not outside it. The symptoms consist in a flaccid paralysis of both upper extremities, appearing as the immediate result of an injury to the neck. The legs are not involved, there is generally no anæsthesia, and the sphincters are implicated slightly and temporarily or not at all. Pain may be prominent, but it may also be slight or absent. The knee jerks may be normal or hyperactive. The following, from the Vanderbilt Clinic, is a typical illustration of this condition:

A man, fifty-four years of age, was run into on the street by a light carriage on December 2, 1899. He thinks that one of the shafts struck him in the neck from behind, and that then the carriage ran over him. As a result of this accident there were fracture of two ribs, scalp wounds, and paralysis of both upper extremities. The patient stayed in bed ten days. During that time, he says, there was complete paralysis in the shoulders and in the upper arms, and great weakness in the forearms and hands, though he could use these to a certain extent. The right side was more affected than the left. The neck was stiff and painful on movement, and there were severe pains between the shoulders and in the neck, which darted down the arms as far as the elbows. At no time was there any paralysis of the legs, of the bladder, or of the bowels; nor was the patient himself conscious of any impairment of cutaneous sensibility. There were no bedsores. On January 8th, the following notes were taken as to his condition:

The patient walks naturally, though the head and neck are held rather stiff. There is a slight prominence of the seventh cervical spine, but no marked tenderness. The movements of the neck are fairly free and cause no pain. Both upper extremities are weak, and around the shoulders there is almost total paralysis. This latter affects the deltoid, the spinati, the rhomboids, and the supinatores

longi. These muscles are pronouncedly atrophied, are the seats of fibrillary twitchings, and respond sluggishly to faradism. In the other muscles of the upper extremity the affection is weakness rather than paralysis, and evidences of degeneration of muscle tissue are wanting. The knee jerks are normal. It is impossible to demonstrate positively pathological disturbances of sensibility. The symptoms, therefore, were almost purely motor, and were confined to the upper extremities. It is evident that the pyramidal tracts for the legs were not destroyed nor pressed upon.

Prognosis.—That this class of cases merits careful observation and record is shown by the favorable course which they so frequently follow. To call good the prognosis of any injury to so vital a structure as the spinal cord may seem exaggerative; yet, compared with secondary injuries, the prognosis of primary focal hematomyelia is extremely favorable.

That recovery is the rule is shown by the infrequency of autopsy records compared with the large number of clinically reported cases. To cite instances bearing on prognosis:

A patient of Thorburn's, paralyzed in legs and arms on December 19th, was discharged on January 16th, "cured, except for some feebleness of the grasp."

Barling reports the case of a man who fell about ten feet, striking on the head and neck. The right upper extremity was almost completely paralyzed, and there was general weakness in the right lower extremity. Analgesia in the right leg was the only sensory symptom. There was no evidence of fracture of the spine. In six months the patient resumed his occupation as a fireman on an engine, and "was as well able to do his work as before the accident."

A patient of Minor's, injured on July 6th, walked on September 5th following, and left the hospital on September 8th. Another patient of the same physician, totally paraplegic at first, could stand alone seven days after the accident.

The following are personal observations:

Case I.—A young man, upon diving into shallow water and striking the bottom with his head, instantly lost power in arms and

legs. When examined two years later, much of the paralysis had disappeared, there being left an atrophic weakness of the small muscles of both hands and a spastic paresis of the left leg. There were no signs of fractured vertebræ. The patient could use both hands fairly well and walked easily, though with a limp. In this case there was undoubtedly a bilateral hemorrhage in the nuclei of the first thoracic segment of the spinal cord, which had also pressed on the left pyramidal tract.

Case II.—A strong man fell thirty feet through a hatchway, and was instantly paralyzed in both legs. Arms unaffected in any way; complete motor paraplegia of legs, with retention of urine; diminution (almost extinction) of both knee jerks. Sensibility to touch and pain were normal, but a pronounced thermo-anæsthesia was present below the nipples. There were no bedsores. Recovery was very rapid. The retention of urine disappeared in ten days, and in six weeks the patient could walk unsupported. In two months the following notes were made: The man now walks easily and without a marked limp, although the right leg is stiff. All movements of the lower extremities are performed quickly and with good force. Both knee jerks have now become exaggerated, and there is also double ankle clonus. Thermo-anæsthesia has in large part disappeared, though traces of it are still present in the legs, especially the left. At the first examination the vertebral column appeared normal. At this second examination there was a kyphosis in the upper dorsal region. The patient had secured a place as night watchman, and said he could perform his duties without fatigue. Thus, in two months from the receipt of the injury, this man had recovered from a complete motor paraplegia with bladder paralysis, and had again become a breadwinner.

Case III.—A healthy man, on July 5, 1898, struck his head on the bottom in diving, and became paralyzed in all four extremities. He would have drowned had not his friends pulled him out of the water. His condition improved greatly, but for several days after the accident he had been unable to move hand or foot, to pass his water, or to control his bowels.

Two weeks later sphincter control had already returned, there had been no bedsores, and the patient, though still bedridden, could move both legs and the right arm freely but feebly. In the right upper extremity complete paralysis of the deltoid, of the triceps, and of all the muscles of the forearm remained. The knee jerk was absent on the

right side and much diminished on the left. The right pupil was smaller than the left. The only objective anomaly of sensibility was a slight thermo-anæsthesia in the left upper extremity. The spinal column appeared normal. Within four weeks of the receipt of this injury the patient could get around on his legs. In six weeks he had regained so much power in the legs that he could walk with ease, without cane or crutches and without noticeable defect, although the legs were somewhat stiff and not so strong as they had been before the accident. He had, however, walked up four flights of stairs without assistance. Recovery in the left hand had kept pace with that of the lower extremities, but the right hand and arm, though much better, still showed signs of serious involvement. Any movement with them the patient could make, but all the muscles of this member were unmistakably feeble, as shown in the grip, in movements of the fingers, etc. There was a marked atrophy also of the thenar eminence. The patient said that he had, with difficulty, been able to sign his name, but that for practical purposes the right hand was of little service to him. Both knee jerks were now exaggerated and there was a foot clonus on the right side. He complained at this examination, as he had done at the previous one, of pain in the back of the neck. The pain was dull, not particularly increased by movement, and never very severe. These were the only symptoms left in a patient who, forty-six days before, had been unable to move hand or foot, to control the bowels, or to empty the bladder. The disturbances in the perception of thermal sensation had vanished, the restoration of rectal and vesical function was complete, and the man was nearly ready to resume his occupation.

Case IV.—A man, thirty-one years of age, fell off a cart, striking on the right side of the head, on July 21, 1899. He was immediately paralyzed in both arms and in the left leg, and for three days following there was retention of urine. On September 6th, less than eight weeks after the accident, when examined at the Vanderbilt Clinic, the right arm had nearly entirely recovered; the left arm was still weak, as was the left leg, though the patient could walk without much difficulty. There was no deformity of the vertebræ, though the movement of the neck caused pain. Tactile sensibility was everywhere normal, but there remained considerable impairment of thermal and pain sensibility in the right side of the body, i. e., on the side opposite to the motor palsy, thus constituting the Brown-Séquard type. Thus, in less than eight weeks after an accident which had caused complete paralysis in three members, one arm had recovered almost entirely,

the other arm was useful to a certain extent, and the paralyzed leg had regained sufficient power to make locomotion easy.

In the case of diplegia brachialis referred to on page 232, in three years later the only remaining symptoms were some wasting over both deltoids and spinati, with slight weakness and fibrillary contractions in these muscles.

Case V.—A football player had a severe collision with another player, so that he sustained a forced flexion of the neck. He immediately had pain in the neck, with pain and tingling down the arms, and weakness of the arms. My examination showed weakness in both triceps and extensors of the wrist and fingers of both sides and unequal pupils (ciliospinal center). No other symptoms. In three months he made a complete recovery.

These cases show how complete recovery may be in primary focal hematomyelia from trauma, even when the initial symptoms are of an alarming character. The prognosis as to life and as to restoration of function is directly proportional to the extent of the hemorrhage. With this latter excessive, tearing and lacerating the cervical cord in nearly total cross section, the patient usually dies in a few hours or a few days. These are the cases most frequently seen at autopsy. When, on the other hand, the amount of the hemorrhage is small, the degree of possible recovery, as shown in the above-cited cases, is truly astonishing.

Immediately after the accident it may be extremely difficult to determine the extent of actual injury undergone by the cord. The destruction may involve the white matter considerably and the clinical type of central affection still be preserved. But whenever the symptoms remain sufficiently elective to make the diagnosis of primary focal hematomyelia possible, the outlook is more favorable than for any other variety of spinal trauma of like initial severity. More light as to prognosis is shed in the few days following the acoident. The early restora-

tion of sphincter control, the quick return of voluntary motor power in some muscles, the absence of the tendency to bedsores, all speak for a favorable termination eventually.

Concussion of the Spinal Cord

Concussion of the spinal cord has been a much agitated question in the past. Volumes have been written about it; it has had a prominent place on the programmes of many medical congresses; enormous sums of money have been paid for it in personal-injury claims. Now its clinical importance is rapidly on the wane. No question in neurology has been surrounded with a greater confusion of ideas. In the early days, when the analysis of nervous symptoms was imperfect, and the lesions underlying them were not known, the introduction of the term concussion, as an explanation of all kinds of traumatic nervous affections, instantly obtained wide favor. As a result of concussion persons could be paralyzed or killed, could become the victims of chronic degenerative diseases, or of affections of preëminently mental characteristics. The scope of the term in those days was almost limitless. With advancing knowledge, and as many of the diseases formerly blanketed by concussion took on an unmistakable individuality of their own, the limitations of concussion became more sharply defined. But even to-day the word is loosely used. In the clearest writings on this subject it is often difficult to tell whether the author intends concussion to designate a cause of spinal symptoms, a clinical entity by itself, or a certain pathological condition in the spinal cord *sui generis* and distinct from other lesions. If, in discussions on this subject, the three possible meanings of the term—a cause, a clinical condition, and a pathological lesion—be kept distinct, the subject may still present difficulties and uncertainties, but it will be at least comprehensible.

Concussion as a Cause.—Concussion of the spine is invoked as a cause of nervous symptoms in the general neuroses, in certain chronic degenerative diseases, in affections indicating disseminated and focal lesions in the spinal cord. The physical characteristics of the injury include general severe shaking and wrenching of the body, and blows over the spine. Such violence is met with in accidents in which the individual is violently thrown about, in falls, and in blows from heavy objects. The relationship of such accidents to the neuroses and to certain chronic degenerative diseases is gone into at length elsewhere in this volume. Here it is sufficient to explain that, in the complex study of the causes of disease, we are often forced to draw our conclusions as to cause from the symptoms which exist and the way they develop. We know that in hysteria, for example, no symptoms are present which indicate visible organic changes in structure; we assume, therefore, that the injury has not caused physical interference with the mechanism of the spinal cord. Further, as hysteria frequently results from fright and similar agencies, we infer that it is the emotional and not the physical element of accidents which call it into play. Similarly with the chronic degenerative diseases. For the determination of concussion as a cause of them we rely upon an analysis of recorded cases. As a result of such an analysis we find that certain ones (e. g., progressive muscular atrophy) probably do, and certain others (e. g., locomotor ataxia) probably do not, result from concussion of accidents.

That concussion accidents are active in the production of traumatic spinal-cord lesions, both disseminated and focal, has been thoroughly illustrated in the foregoing pages. The only comment necessary here is that it acts, not by any mysterious molecular disarrangement, as the original supporters of the theory maintained, but by actual gross contusion, or laceration of the constituent elements of the cord, or by hemorrhage

into its substance. That such lesions may be produced without visible alterations in the bony case of the cord is now well known; and the better it becomes known, the less we will hear of concussion of the spinal cord.

Concussion as a Clinical Entity.—The clinical individuality of concussion of the brain is firmly fixed. It is almost as well established that no such condition, parallel in symptomatic behavior and outcome, exists in the spinal cord. The question is not whether the fibers and cells of the spinal cord are of different function and amenable to different laws than nerve fibers and cells situated elsewhere, but whether the protecting envelopes do not shield the cord from certain injuries to which the brain, and even the peripheral nerves, are liable. Temporary loss of function from trauma is shown in the brain by loss of consciousness; in the nerves, by numbness, tingling, and even motor weakness, or paralysis, in the distribution of the nerves. Brief duration and prompt restoration are the qualities of the condition. It is not probable that such a state of affairs ever occurs in the spinal cord. No spinal-cord case at all comparable to concussion of brain or of nerve is known to me, either personally or in literature. Once spinal-cord symptoms manifest themselves as a result of injury, they show, either by the extremely slow return of function or by the permanence of the symptoms, that gross structural changes have occurred.

Concussion as a Pathological Condition.—Is there such a pathological condition as concussion of the spinal cord recognizable *sui generis* and distinct from other lesions? Or, if the term be objectionable, can concussion bring about a lesion in the spinal cord whose visible characteristics permit it to be recognized as different from the lesions caused by laceration, by contusion, or by hemorrhage? The answers given by human pathology are in the negative. In the cases which have been regarded as examples of the condition by A. Westphal,

and by Willard and Spiller, the vertebræ were fractured, and there were cord lesions, with hemorrhages in the immediate vicinity of the fracture. These investigators maintained that the cord lesion was the result of concussion, and not of compression by the loosened vertebræ. They based their opinions upon the absence of evidence of external bruising of the cord. The lesions found did not differ from those of crushes of the cord generally. If such proof is to be accepted as evidence, the condition must be regarded as not uncommon, for every pathologist is familiar with the fact that the spinal cord frequently retains its normal contour even after it has been seriously compressed. That slight and temporary compression is amply sufficient to cause a loss of spinal function is shown by the complete (and generally temporary) paraplegia which follows operations on the spine in which the cord may have merely been touched by the operator.

Gowers is the warmest partisan of spinal concussion, yet the proofs he adduces are far from satisfactory. Most of the cases he cites date from a period of which the neurological contributions must be accepted with considerable reserve. In one of the more recent ones (Fischer) there was an extradural hemorrhage, extending from the cervical to the lumbar region, of which Gowers makes no mention. In Gowers's own case, the pathological picture is that of myelitis. "A lady was shaken in a railway collision. She seemed immediately after the accident to have suffered no injury, but in a few days paraplegia developed, and from its consequences she died, six weeks after the accident. Throughout the dorsal regions of the cord I found indications of subacute myelitis, chiefly in the white columns, varying in its extent in different regions, but in most part confined to the lateral tracts."

Schmaus records the microscopic examination of the spinal cords of four individuals who died from the effects of traumatic

paraplegia. In three the spinal column had not been fractured. In all these cases the patients had survived the injury, and had lived, with paralytic symptoms, for several months. All of the spinal cords had cavities in the gray matter. It seems possible that these cavities were not the results of the breaking down of gliomatous tissue (as Schmaus interpreted them), but were rather the spaces left after hemorrhages which occurred at the time of the original injury, and that the cases themselves were simply cases of myelitis secondary to hematomyelia.

The results of animal experimentation more successfully sustain the contention of concussion as a definite lesion. In fact, Schmaus's celebrated experiments have been largely received as finally settling the question. Schmaus induced paraplegia in animals by tapping them on the back with a hammer without inflicting injury to the spinal column. After death no hemorrhages were found, but there was swelling of the axis cylinders in the parts of the cord opposite the external injury. Schmaus concluded "that anatomical changes of the specific nerve elements may be brought about by purely traumatic means; that swelling and degeneration of the axis cylinders, breaking up of myelin, softening, and gliosis with cavity formation, may result directly from injury, independently of accessory causes." Experience has shown, however, that the utmost conservatism is necessary in accepting the deductions from experiments upon the nervous system of animals as applicable to the nervous system of man. In guinea pigs and in rabbits, the animals that Schmaus made use of, the protection of the spinal cord by skin, muscle, bone, and ligaments, after due allowance is made for comparative differences in size, is very much less secure than in man. And the possibility of causing paraplegia without gross lesions in these animals does not justify the conclusion that a similar result might be expected were man the animal experimented upon. Schmaus's results, further-

more, are not identical with those of a subsequent observer. Bikeles, in a series of experiments conducted along similar lines, found that swelling of the axis cylinders, a condition to which Schmaus attached great importance, was absent.

Were the point at issue one as to whether or not the fibers and cells of the spinal cord were susceptible of a temporary depression of their function under the influence of properly applied violence, valuable deductions might be drawn from animal experimentation. Such, however, as has already been said, is not the question. The problem has to do with the structures which protect the human spinal cord rather than with the functions of the spinal cord itself, and must consequently find its solution in the hospital, in the dead house, and in the laboratory of human pathology.

Leyden, who is often quoted as a believer in concussion, now regards it as secondary to hemorrhage.

Such is the most important evidence for the existence of the pathological condition known as concussion of the spinal cord. The writings of Watson, of Vibert, and of many others who are frequently cited in this connection cannot be regarded as having materially added to the sum of knowledge. In view of the intense interest with which the question has been for so many years surrounded, and in consideration of its importance both to medicine and to law, the facts seem very meager.

To the impartial observer the conviction must be inevitable that the weight of evidence is against the existence of the condition.

Treatment of Injuries to the Spinal Cord

After injuries to the spinal cord the patient should be disturbed as little as possible and every effort should be made to avoid movement in the spine itself. In severe lesions the pa-

ralysis is so extensive that means to secure immobility in bed are rarely necessary. In partial lesions the spine may be held still by means of sand bags. Orthopedic appliances are rarely necessary. For the paralysis itself there is no treatment. But attempts to counteract the establishment of contractures are always to be made. Tendons which are beginning to show undue prominence and rigidity should be gently stretched several times a day. Drop foot may be combated by having the feet held at right angles to the legs by sand bags, or by straps which pass from the knee to the soles of the feet. Attention to the condition of the skin is a most important requisite of treatment. This task falls generally to the nurse, to whose attentive skill many of these patients owe their lives. The bed should be a water or air bed; the sheets kept free from wrinkles, crumbs, and other foreign objects. (The patients, being anæsthetic, do not complain of these things.) Change of position should be made as often as is practicable without disturbing the spine. The buttocks, internal malleoli, and heels, being the election sites of bedsores, should be frequently inspected, rubbed with alcohol, and protected by cotton or air cushions. Once the skin is broken, the greatest care is necessary to keep the ulcer and its edges clean and freed from all pressure. There is no satisfactory treatment for anæsthesia. For the pain, internal analgesics are of chief service. In partial lesions the cautery applied over the painful region may benefit. The use of the catheter should be delayed, if possible. When used, as is generally necessary, no operation in surgery merits more careful asepis. To avoid the catheter Cushing recommends a suprapubic or perineal cystotomy, with permanent drainage. Constipation can usually be controlled by ordinary laxatives. Distention of the abdomen with gas can sometimes be reduced by the use of calomel and hot stupes. For incontinence of fæces there is no treatment.

The remaining question in the treatment of injuries to the spinal cord concerns operative procedure. The one operation is laminectomy, bloodless reduction of dislocations not being recommendable. For gunshot wounds there is a general agreement of opinion that early operation is desirable. Such operation is naturally contraindicated when the bullet has seriously involved other vital structures besides the spinal cord, or when the general condition of the patient is such as to make his surviving the operation improbable. If the x-ray shows that the bullet is not lodged in the canal, the purpose of the operation is less definite, but it may nevertheless be performed. Prewitt has tabulated 49 cases of gunshot wounds of the spine, treated since the aseptic era. Of this number, 24 were subjected to operation, with 11 recoveries and 13 deaths. Twenty-five were not operated upon, with 8 recoveries and 17 deaths. With fracture and dislocation of the spine the indications for operation are far less plain. Thoughtful and experienced men hold diametrically opposed views regarding it. The disagreement is of long date, and nothing in the recent advances either in surgery or neurology has thrown light upon it. Those who advocate operation say that the removal of pressure at least gives the cord a better opportunity for repair, and that in many operated cases more or less complete restoration of function follows operation. The opponents of operation reply that the injury sustained by the cord is done at the moment of injury; that the paralysis, etc., is the result of a sudden squeezing, and not a permanent compression; they cite the 21 autopsies of Bowlby in which no compression by displaced bone and no narrowing of the canal was found; they say that to relieve permanent compression, if it exists, is unavailing, since, as a result of the original injury a certain amount of softening and attenuation of the cord is bound to occur, which by itself insures plenty of space in the canal. They say that clinically reported

instances of recovery after operation are not convincing, as recovery may begin long after the patient has been paraplegic, independently of operation.

Somewhere between the two extreme positions the wisest course lies. In fractures and dislocations of the cervical and upper dorsal regions operation rarely seems to be of benefit, and should not be undertaken unless there is reason to suppose there has been comminution of the bones. In the lower dorsal and lumbar regions operative procedures hold out more hope of help. This is especially true for the dorsolumbar region, as here the removal of pressure from the nerve roots and cauda equina may be of unquestionable advantage. But even in these regions it is advisable to defer the operation till several weeks after the injury, in order to determine the extent of spontaneous repair.

Certain it is that laminectomy for fracture and dislocation of the spine is not a life-saving operation. The most that can be expected from it is that the restoration of functional capacity may be more complete as a result.

It is obviously contraindicated to operate for hematomyelia; and hematorrhachis, frequently spoken of as the ideal condition for operation, probably never exists as an independent affection.

CHAPTER VIII

INJURIES TO THE PERIPHERAL NERVES

Ætiology—Symptoms—Relative Frequency—Prognosis—Treatment—Injuries to Individual Nerves—Combined Paralyses of the Upper Extremity—Paralyses of the Lower Extremity—Injuries of the Sympathetic Nerves—Traumatic Neuralgia

THE pathological changes that occur in a nerve which has been divided, and so cut off from its trophic center, cannot be detected with the microscope before the lapse of from five to eight days. Then the myelin surrounding the axis cylinder may be seen to be breaking up, a process of degeneration which progresses, and which may be ultimately associated with the destruction of the axis cylinder and replacement of nervous tissue by connective tissue. Such a process is degenerative rather than inflammatory, and it is only by the addition of infectious elements that to the degeneration of nerve fibers are added the ordinary vascular manifestations of inflammation. Nerve disorders which take place under these latter conditions may properly be called traumatic neurites, but the simple degenerative process should be spoken of as nerve degeneration, or palsy, and not as nerve inflammation.

There are two theories as to the regeneration of nerves. By one, the central theory, regeneration implies integrity of the trophic centers, and is limited to the proximal end of the nerve. The regeneration reaches the distal end by the outgrowth of the axis cylinders, which push their way from the central into the distal end. Consequently, by this theory, no regeneration can occur without the divided ends being united directly by suture or by implantation. According to the second or periph-

eral theory, which is ably defended by Ballance and Stewart, regeneration of both axis cylinder and medullary sheaths takes place in both central and peripheral ends, although in the peripheral end the regeneration is imperfect unless the ends have been united. By this theory, the new nerve fibers do not depend on trophic centers, but are formed from cells in the nerve sheath itself. By either theory, union of divided ends is necessary for complete repair.

When nerves are cut, regeneration may occur if the ends are properly united. Repair in nerves injured by blows or crushes, but not divided, may ordinarily occur in the course of several months. In both instances there may be restoration of function in varying degrees.

ÆTIOLOGY

Nerves may be injured by being cut, lacerated, bruised, stretched, or compressed. The most frequent injuries are bullet wounds, cuts from sharp instruments or pieces of glass, falls, excessive muscular action, and pressure from various causes.

In the more severe forms of traumata the general condition of the patient has, of course, but little influence upon the development of paralytic symptoms. But slight blows, falls, and pressure effects are very much more likely to be followed by loss of peripheral nervous function in enfeebled and alcoholic persons than in persons in robust health. In pressure palsies, particularly, alcohol is a potent predisposing agent.

SYMPTOMS

Disturbances of motion are the most important symptoms of peripheral nerve injuries, and they are such as are caused by interference with the lower motor neuron. Loss of con-

ductivity in the neuraxon abolishes the transmission by it of the impulses of motion and nutrition, so that the muscles are paralyzed and undergo atrophy, although the cell-body portion of the neuron, situated in the spinal cord, remains unaffected. Although the motor symptoms of peripheral nerve palsy are in many ways similar to those which may result from disease of the spinal cord itself, they differ so essentially in distribution and in association with other symptoms that there is rarely any difficulty in determining whether it is the spinal or peripheral part of the neuron which is involved by the lesion.

Although in all injuries to the spinal nerves there is usually an association of both sensory and motor symptoms, motor paralysis is the most pronounced and the most important. It occurs immediately or soon after the injury, and is always flaccid in character. It is limited to the muscles supplied by the injured nerve, although usually all the muscles are not involved to an equal degree. Complete loss of power of a whole limb is observed only in cases in which the injury has been extremely extensive and severe. Muscular weakness becomes apparent not only through the inability to perform certain movements, but through various abnormal positions of parts deprived of their muscular power. Through continuous overaction of opposing muscles the paralyzed muscles may eventually become tense and the tendons of the overacting muscles prominent, thus forming contractures; but spasticity, such as is ordinarily seen in cerebral palsies, never occurs. Deformities may result from the overaction of the opposing muscles, as is seen by the drawing of the mouth to one side in facial paralysis, or from the effects of gravity, as is the case with the drop wrist of " Saturday-night " paralysis.

Atrophy in muscles slightly paralyzed often does not appear at all, but when a nerve has been seriously injured its occurrence is constant. After injury to a peripheral nerve the mus-

cles may very quickly lose their firmness and consistency; it is usually, however, two or three weeks before the atrophy becomes apparent to the eye.

Tremor is a frequent symptom of peripheral nerve injury; it occurs in the smaller muscles of the hand, but more frequently in traumatic lesions affecting the muscles around the shoulder joint: It is fine, fibrillary, and very much intensified by movement and fatigue.

Sensory symptoms are usually not prominent in traumatic palsies of nerves; even when a nerve is divided anæsthesia does not invariably result. In most of the isolated peripheral palsies the chief sensory symptoms consist of tingling and numbness in the areas supplied by the injured nerves. Perception and localization of touch, pain, and temperature are usually little or not at all impaired. Anæsthesia may occur in the regions supplied by the nerve, but it is chiefly found in cases in which inflammation is added to the degenerative process, or in which the nerve injury has been particularly severe. The same is true of spontaneous pain. Neuritis can cause most intense pain. But in the nonseptic palsies of traumatic origin pain is only an occasional symptom. It is sometimes, however, the most prominent result, so that the condition is spoken of as traumatic neuralgia rather than palsy. This is particularly frequent after injuries to the fifth and to the sciatic nerves. Tenderness over the affected nerve trunk and muscles is also occasionally present, and may be a distressing symptom. In lesions of the sensory portions of nerves it is the lower sensory neuron, which is situated between the posterior spinal ganglion and the periphery, that is affected. Consequently, when it is injured, the symptoms of disturbed function occur in the course of the nerves themselves. From the description of anæsthesia and paræsthesia resulting from spinal-cord injury, it will thus be seen that the distribution of sensory symptoms is entirely dif-

ferent in lesions of the peripheral nerves from that observed in lesions of the spinal cord.

The tendon reflexes in peripheral nerve palsies usually remain unchanged, because the muscles concerned in reflex action are rarely involved in these disorders. There is never any increase of reflex activity, although a degenerating muscle may contract to slight mechanical stimuli. The knee jerk is the only tendon reflex constantly present in health, and an isolated peripheral palsy of the extensors of the leg is extremely rare.

Electricity is the most valuable agent at our disposal for the diagnosis and prognosis of peripheral nerve injuries. It is only in disease or injury of the lower motor neuron that the reaction of degeneration occurs; in primary disease of the muscles and in cerebral palsies electrical irritability remains practically unchanged. All severe injuries of the peripheral nerves, however, like those of the spinal cord, are associated with changes in electrical reactions. When the protoplasmic portions of the neuron (the anterior horn cells) are extensively destroyed, as in infantile spinal paralysis, degenerative reactions are constant and occur rapidly; but in chronic affections of the anterior horns—e. g., progressive muscular atrophy—the cells degenerate slowly and individually, and consequently electrical excitability of the muscles is retained for a long time, and only disappears when all the muscle fibers have atrophied.

The electrical examination of every case of peripheral nerve palsy is indispensable for clinical purposes, and should be carried out with both the faradic and galvanic current.

The changes in electrical excitability after injuries to the peripheral neuron have already been described (p. 50). They vary in character, time of appearance, and duration, but the variations are usually in direct ratio to the extent of injury. After severe injuries the initial period of hyperexcitability is short; after slight injuries it may continue for several weeks

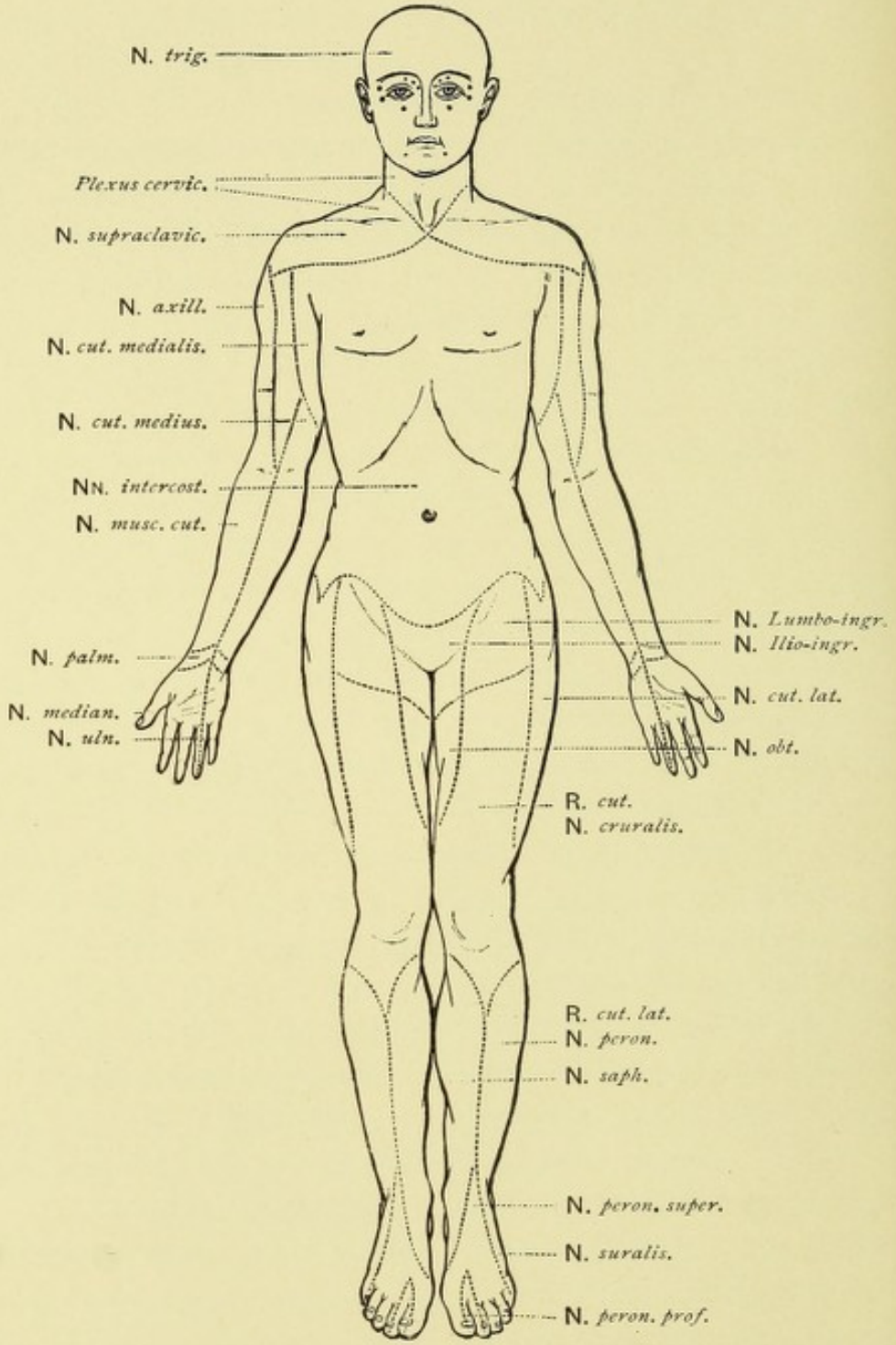


FIG. 54A.—Peripheral distribution of sensory nerves.

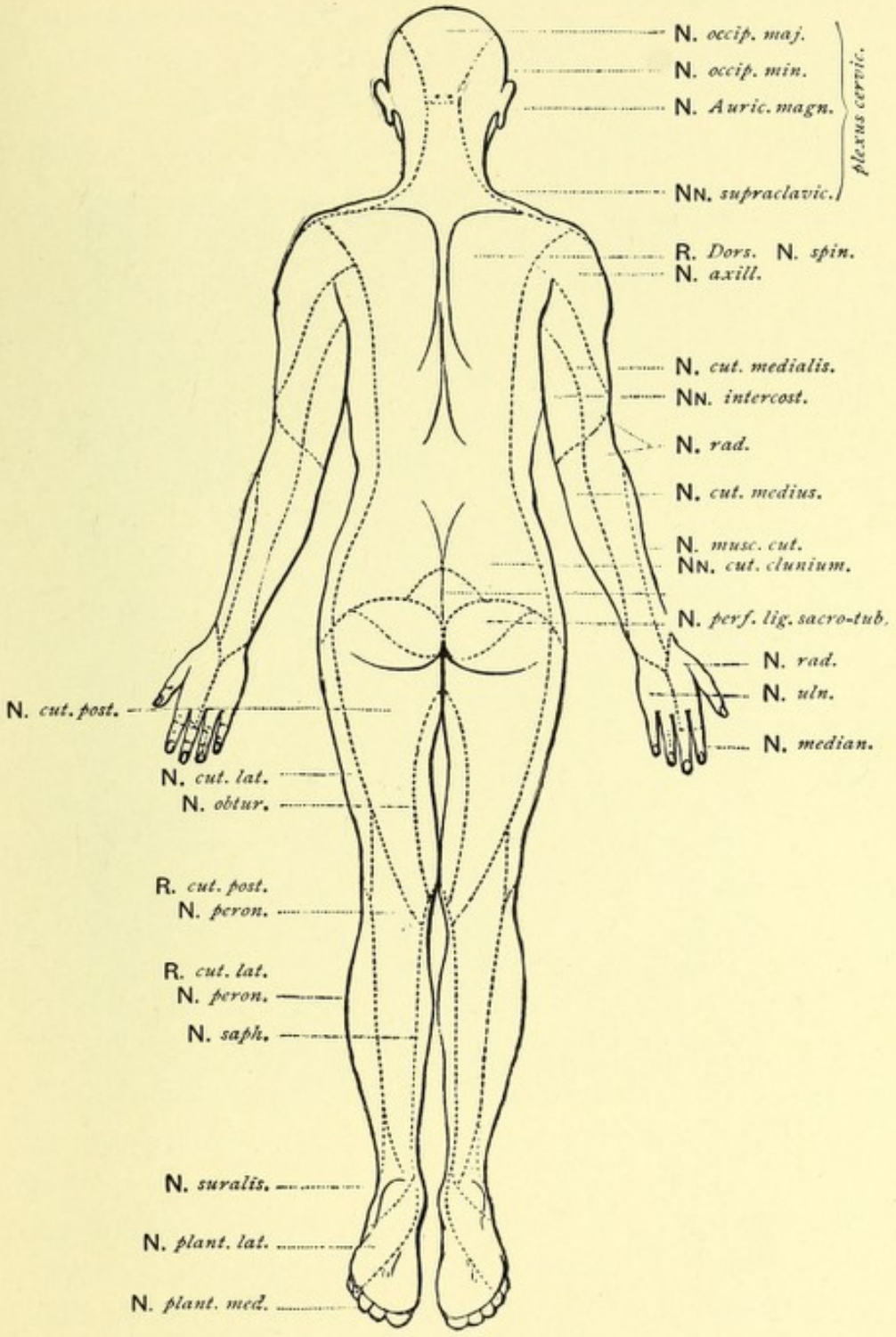


FIG. 54B.—Peripheral distribution of sensory nerves.

and then return and remain at the normal. If, after six to eight days, there is no decrease of excitability, we may infer that recovery will be complete and rapid. If after two weeks the faradic irritability of the nerve is entirely lost, the duration

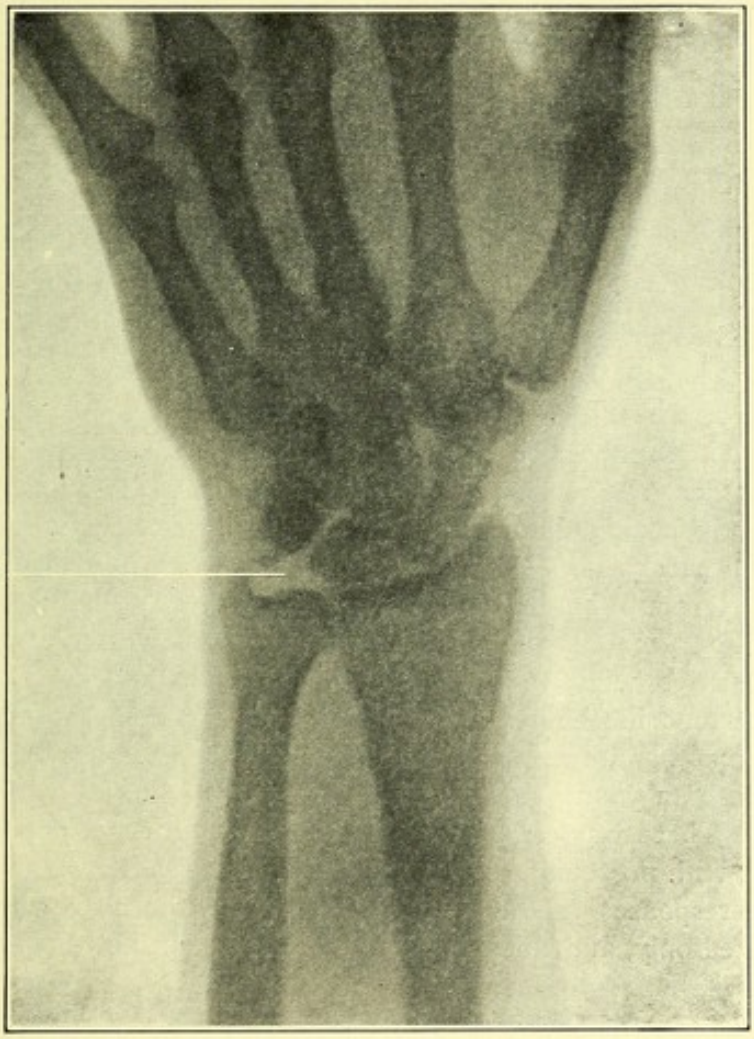


FIG. 55.—Splinter of glass causing injury to the ulnar nerve. The star shows location of the splinter.

of the paralysis will be long. In severe injuries there may be for several months no response to faradism or to galvanism applied to the nerve, or to faradism applied to the muscle, while the galvanic reactions in the muscles may be increased or reversed. If after three or four months these electrical

alterations persist, and if at the same time there has been no improvement in the paralysis, it is always certain that recovery, if it occurs at all, will be very tedious. Very frequently, however, there is a return of motor power before there is any improvement in the degenerative reaction. Such a return renders the prognosis more favorable.

Occasionally, after very severe contusions or partial lacerations, the nerve may be totally bereft of all function for months, and give complete degenerative reaction. If the injury occurred during operation, or as the result of a cutting blow, it may be impossible to decide whether or not it is divided. In such cases operation or time alone can decide.

Thus, as a result of a fracture at the elbow, a young girl became paralyzed in the muscles supplied by the musculospiral nerve. Voluntary power was gradually being restored when, after an operation to correct a bone deformity which had been left, the musculospiral was again paralyzed. The surgeon thought it due to pressure at the time of operation, but could not be positive that he had not cut the nerve. Four months after the operation there was total paralysis in the muscles below the elbow (musculospiral) and no reaction to either current in the nerve. After several weeks recovery began spontaneously and eventually became complete.

Similarly, a young man cut the outer side of the leg with a piece of glass. There was complete paralysis in the external popliteal distribution, with profound anæsthesia in the great toe, R. D., and failure of nerve response. Yet operation showed that the nerve sheath had been nicked only, and the nerve badly contused but not divided.

In the case of foreign bodies, such as pieces of glass, bullets, etc., the x-ray is of great value in diagnosis and localization.

A young man fell on fragments of broken glass, cutting the right wrist, and sustaining immediate paralysis of the ulnar nerve for both motion and sensation. No glass was found in the wound, and no operation was done. The symptoms rapidly improved so that he could use the hand freely; but it never regained complete power. Six years later, while tying up a package, the patient had the sensation

as though something snapped, felt a severe pain on the ulnar side of hand, and again was paralyzed in the muscles supplied by the ulnar. A piece of glass was evidently in the wrist in such a position as to injure the nerve when the wrist was suddenly moved. The x-ray (see Fig. 55) confirmed this. A splinter of glass was found at the operation, pressing on the nerve. Since the operation the strength in the hand has been greater than it was before the second palsy.

In most injuries the paralysis ensues immediately. After fractures, however, the loss of function may come on gradually,

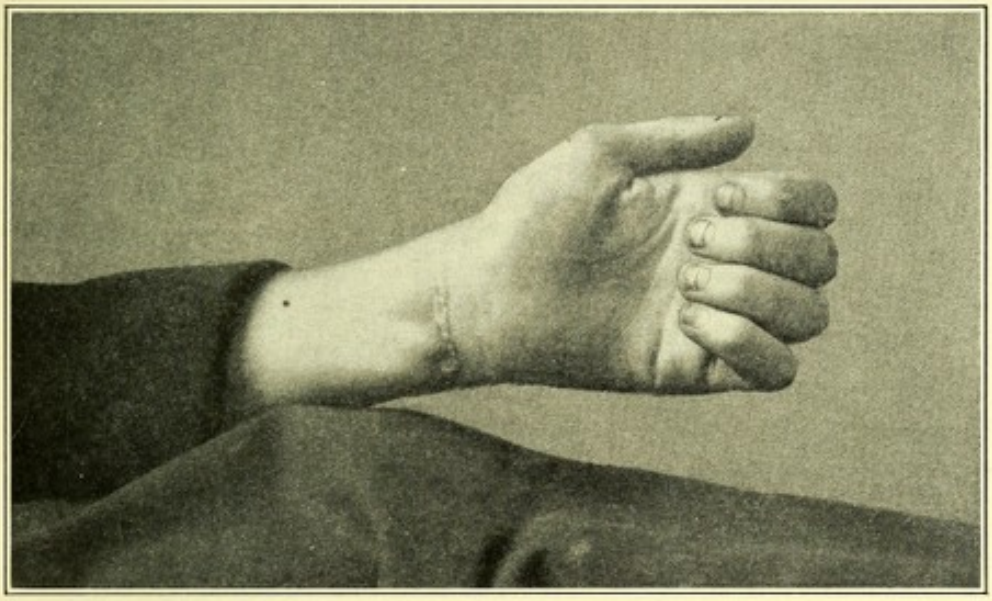


FIG. 56.—Trophic disturbance of the nails after an injury to the median and ulnar nerves at the wrist.

the nerve being caught in the callus and gradually compressed. In wounds, also, the connective-tissue growth and contraction may cause a gradual increase in symptoms.

Thus a man was shot through the upper arm, sustaining injury to the median nerve, as shown by anæsthesia and partial paralysis. A year later he began to have severe pain in the tip of the thumb. An operation showed that the nerve was not divided, but was tightly bound down by adhesions.

Vasomotor and trophic disturbances are common accompaniments of severe injuries to nerves, and consist in the condi-

tion known as glossy skin, in œdema, in the impairment of the growth of the nails, in the lowered vitality of the skin as shown by the occurrence of cutaneous inflammation and subnormal temperature, and by the atrophy of muscle.

Relative Frequency

The following table, made up from 157 consecutive cases of nerve injury in men at the Vanderbilt Clinic, shows the relative frequency of injury to different nerves. Cases of paralysis from pressure or from section of the nerves at operation are not included. Under the brachial plexus are included both injuries to the plexus itself and to two or more nerves in the arm:

Facial	2	Median	10
Brachial plexus	68	Posterior interosseous	1
Circumflex	15	Anterior crural	1
Musculospiral	29	External popliteal	4
Musculocutaneous	1	Anterior tibial	2
Ulnar	24		

Prognosis

The prognosis of nerve injury depends upon the general health of the patient and the extent of injury to surrounding parts and to the nerve itself.

Regeneration of nerves occurs more quickly in the robust than in the person exhausted from any cause. Injury to surrounding parts interferes with the reparative process.

The extent and character of injury which the nerve itself receives are naturally the most important prognostic considerations. From the effects of gradual pressure, as in the "sleep palsies," the nerve usually recovers perfectly in a few weeks or months. The same is true for blows and falls by which there has been no serious injury to bones or joints. With severe

fractures and dislocations, especially about the shoulder joint, the prognosis is always more serious, and in a considerable proportion of cases complete restoration of function does not occur. The prognosis is much better for injuries of single nerves than for those of plexuses. Of 23 cases of plexus paralyses observed by Bruns, recovery occurred in six only. After very slight injuries, return of function may be practically complete in three or four weeks. But in the severer grade of injuries, with pronounced paralysis and impairment of the electrical reactions, not much return of power is to be looked for before two months. The nerve may be practically dead for six months or longer, and still perfect recovery ensue. In recovery, the sensory symptoms always disappear first, and then the motor. The atrophy, like the degenerative electrical reactions, are slower in disappearing.

The prognosis for complete division of nerves depends in large part upon the length of the interval between the injury and the approximation of the divided ends by operation. The results of early suture are very often, though by no means always, successful. Although, in my own experience, complete recovery has occurred after a year's interval, and although there are reported cases of secondary suture after a still longer time, with good recovery, the earlier the operation is done the better are the chances for a good functional result. Of 15 cases operated upon in Bergmann's clinic (Hector) almost complete recovery resulted in 7; in 6, distinct motor or sensory symptoms remained, and in 2 there was no improvement. Of the 7 cured cases, the suture was done soon after the accident in 5, and in the other 2 within three months. Of the 6 partially successful cases, in 2 the suture was done promptly, in 4, after more than three and one-half months.

Treatment of Nerve Injuries

The treatment of nerve injuries is medical and surgical. The medical treatment of the paralysis consists chiefly in rest, massage, and electricity. Rest is obtained by relieving the paralyzed muscles of all tension. Thus, in palsies about the shoulder joint the arm should be carried in a sling; in palsies of the extensors of the wrist and fingers, if there is any tendency to contracture on the part of the flexors a splint, holding the weakened muscles extended, should be applied. Massage of the paralyzed muscles often seems useful, if skillfully done. Electricity is a more generally accessible remedy. If the muscles respond to faradism, this current may be used, but it is my conviction that the galvanic current is a greater stimulus to nutrition and consequently more effective. The current should be only strong enough to cause slight contractions. Fifteen or twenty contractions of each muscle, obtained with that pole over the motor point which gives the best contraction, once, or better twice, daily, are sufficient. There is little reason to doubt that regeneration is much hastened, if not in some cases inaugurated, by these means.

For painful affections of nerves, heat is the most important consideration. The part should be kept wrapped in cotton, and hot applications made, when possible, at frequent intervals. Hot fomentations, hot-water bags, hot electric cushions, all are serviceable. Rest is even more imperative than in paralytic conditions. The mild constant galvanic current often relieves. If the pain persists, counter irritation, in the form of applications of tincture of iodine, of mustard leaves, or, best of all, light cauterizations with the Paquelin cautery, are indicated. Internal medication consists of the various analgesic remedies, notably the coal-tar products, quinine, and general tonics. In some cases of protracted neuralgia change of scene and climate alone effect a cure.

For divided nerves, suture is the one remedy. For purposes of identification of the distal end, a small faradic battery will be found useful at operation. Suture should be done as soon as the diagnosis is possible. When, two or three months after the accident, the diagnosis between a bad contusion and a section of a nerve still cannot be made, an exploratory operation is indicated. In some cases of bad contusion of nerves an excessive growth of connective tissue prevents regeneration. This is particularly apt to be the case in injuries about the shoulder. In such cases operation may be tried. The nerve ends cannot be directly united, but should be connected by implantation of animal nerves, or by tubes of decalcified bones. After certain joint injuries, adhesions about the joint may prevent regeneration, or be the cause of severe neuralgias. Breaking up the adhesions under ether, with subsequent massage, effects a cure in some cases. Finally, nerves caught in callus should be freed by operation.

Injuries to the sympathetic are rarely, if ever, amenable to treatment.

In Erb's obstetrical palsy, with laceration of the cervical nerve roots, improvement in position and some restoration of function has been obtained by union of the divided ends, even when the operation was done long after the injury. In long-standing paralyses of the facial, some restoration of function has been obtained by anastomosis with the hypoglossal (Clark).

Injuries to Individual Nerves

The symptoms of injury of the cranial nerves within the skull have been given in the chapter on Brain Injuries. Some of these nerves may be injured in their superficial course also, and then give a somewhat different symptomatology.

Fifth Nerve.—Traumatic extracranial paralysis of the tri-

geminus, a rare condition, is usually limited to one branch of the nerve. Stab wounds, bullet wounds, and blows are the causes. The symptoms are pain and anæsthesia in the distribution of the branch affected.

Seventh Nerve.—Traumatic extracranial facial paralysis is not very common. Blows on the ear or over the mastoid process, stabs or cuts in this region, or fractures of the jaw, may cause paralysis in all the peripheral branches of the nerve. Injury to the face may cause loss of power in either of the facial branches. A lady was operated upon by infraorbital incision for rebellious one-sided neuralgia of the fifth nerve. When seen several months afterwards the neuralgia remained, but to it had been added an incurable paralysis of the same side of the lips and cheek; the cervico-facial branch of the seventh nerve had been cut at the operation.

FIG. 57.—Facial palsy on right side. Attempt to wrinkle the forehead.

FIG. 58.—Same case—attempt to close the eyes. (Drawn after Starr.)

The symptoms of facial palsy vary with the extent of injury. A blow on the ear or mastoid may cause paralysis of all branches. Then the paralyzed side of the face will be flattened, the prominence of its cutaneous and muscular folds diminished, and the face drawn toward the sound side. The

eye cannot be closed because of loss of power in the orbicularis palpebrarum, a condition which not uncommonly results in a troublesome conjunctivitis, and exposes the eye to injury from foreign bodies. The forehead cannot be wrinkled on the affected side. The mouth, when closed, is usually drawn toward the sound side, causing an inequality which becomes still more evident when the patient laughs or opens the mouth wide. In paralysis of the lips and cheeks the patient complains that food runs out of the paralyzed side. He cannot close the lips perfectly, as may be shown by attempts at whistling, etc. If the temporo-facial branch alone is affected, the paralysis will be limited to the forehead and eye; if the cervico-facial, to the lips, cheek, and platysma myoides. Abnormal electrical reactions occur very quickly.

Blows around the ear affecting the facial nerve may also cause deafness, tinnitus aurium, and dizziness, indicating disturbances of the auditory nerve; but extracranial facial paralysis is not associated with symptoms of injury to any other nerve except the eighth. The taste is not affected; impairment of sensation may occur in the posterior auricular region, but never on the face.

The diagnosis is simple. Cerebral facial palsy never involves the occipitofrontalis to a marked degree, and does not seriously interfere with the muscular movements involved in the display of the emotions. When a person with cerebral facial palsy laughs, the innervation of both sides of the face is usually almost equal, so that the differences of the facial folds are scarcely perceptible; also, paralysis of the seventh nerve, when of cerebral origin, is usually associated with hemiplegia of the same side. In nuclear disease, or in injury of the nerve in its course from the pons to the exit from the skull, there are usually associated symptoms of palsy of other cranial nerves, as well as disturbances of taste or salivary secretion.

Hysterical facial paralysis is very rare, and is always associated with other hysterical stigmata.

The prognosis varies with the character and extent of the injury. If the nerve has been cut, it will not regenerate unless the ends become approximated soon after the injury. Many cases do not entirely recover even after a slight blow. Gowers reports the case of a boy who was struck behind the ear with a book, in whom the resulting facial palsy was permanent. The prognosis should be very guarded, and controlled by electrical examination. Cases which show no sign of improvement after four months will probably never recover entirely.

Glossopharyngeal and Pneumogastric Nerves.—Sometimes after intracranial injuries at the base, more frequently after bullet wounds in the throat, or stab or bullet wounds high up in the neck, but rare in any situation, there result symptoms referable to affection of one or both of these nerves, such as anæsthesia of the throat, paralysis of the palate, disturbances of taste, and changes in the salivary secretion. Both the glossopharyngeal and vagus doubtless have varying distributions, and consequently no one of the preceding symptoms are sufficient for identification of the nerve affected. Injury of the spinal accessory (q. v.) high up may cause some of the above symptoms. Isolated traumatic paralysis of the glossopharyngeal has not been observed. When paralyzed, it is in part only, and then in association with other nerves. Neither are there reported isolated paralyses of the trunk of the pneumogastric as a result of injury, except when, at operations on the neck, the nerve has been divided, with or without intention. High up in the neck this nerve may be injured, the hypoglossal being injured with it.

Hirsch has reported a case in which a man shot himself in the mouth. The ball (as shown by x-ray) lodged opposite the fourth cervical vertebra. There resulted, all on the left side, the sympa-

thetic symptom group: atrophy of one-half of the tongue (hypoglossal), paralysis of the left vocal cord, and persistently rapid (108) pulse (pneumogastric).

The only symptoms which can be identified as indicating isolated affection of the pneumogastric are paralysis and anæsthesia of the larynx, and disturbances affecting respiration and heart action. Bilateral injuries would be quickly fatal; unilateral injury of the glossopharyngeal or pneumogastric in the neck is usually accompanied with fatal hemorrhage. From the results of unilateral sections at operation, however, we infer that when life is spared the initial tachycardia and embarrassment of respiration eventually subside, the functions of the divided nerve being carried on by its uninjured fellow.

Traumann has described a case in which the tachycardia persisted for several weeks, as long as the patient was under observation.

Spinal Accessory Nerve.—The spinal accessory nerve has two nuclei; one in the upper five cervical segments of the cord, which sends impulses to the trapezius and sternomastoid muscles; the other, situated in the medulla, is in reality part of the nucleus of the pneumogastric. Roots from both nuclei unite to form the accessorius nerve. The nerve trunk thus formed divides shortly after its exit from the skull into two branches, one of which, the external, supplies the sternomastoid and trapezius; the other, a short branch, enters into the trunk of the pneumogastric. Injury of the spinal accessory on one side, therefore, after the union of its various roots and before its bifurcation, paralyzes the sternomastoid and trapezius, and at the same time gives the following symptoms referable to the vagus, viz., paralysis, and possibly slight disturbances of sensation, of the larynx; paralysis of the palate, difficulty in swallowing, and tachycardia.

Traumann has described a case in which, as the result of a stab wound under the right ear, there was difficulty in swallowing, hoarse voice, paralysis of the right vocal cord and of the palate, and paralysis of the trapezius and sternomastoid. In addition the right hypoglossal nerve was cut, as indicated by paralysis of the right side of the tongue. The spinal accessory was doubtless injured just after its exit from the skull and before its division into its external and internal branch.

More frequently, however, the spinal accessory is injured in its external branch alone, the only symptoms being those referable to the two cervical muscles.

The paralysis of the sternomastoid is usually complete; that of the trapezius is more apt to be partial, owing to this latter muscle receiving supplemental supply from the cervical nerves. This supplemental supply is not constant, as cases are on record in which total palsy of both muscles resulted from section of the spinal accessory.

When both muscles are paralyzed the resulting incapacity is extreme; all power for lifting heavy weights is gone, and the patient is unable to elevate the arm more than 90°. The head is held slightly forward. The shoulder is depressed, the arm hangs heavily with slight forward rotation, the scapula is drawn away from the spine and rotated on its horizontal axis. There is a sinking in of the line extending from the occiput to the acromion process, and a loss in the fullness at the base of the neck.

The causes of spinal accessory paralysis are stab and pistol-shot wounds of the neck, fracture of the cervical vertebræ, and, possibly, injuries at the base of the skull.

Hypoglossal Nerve.—Extracranial traumatic paralysis of this nerve is usually unilateral, and always due to punctured or pistol-shot wounds. That traumatic paralysis of the hypoglossal is rarely observed may be accounted for by the close anatomical relations of this nerve with the pneumogastric, the

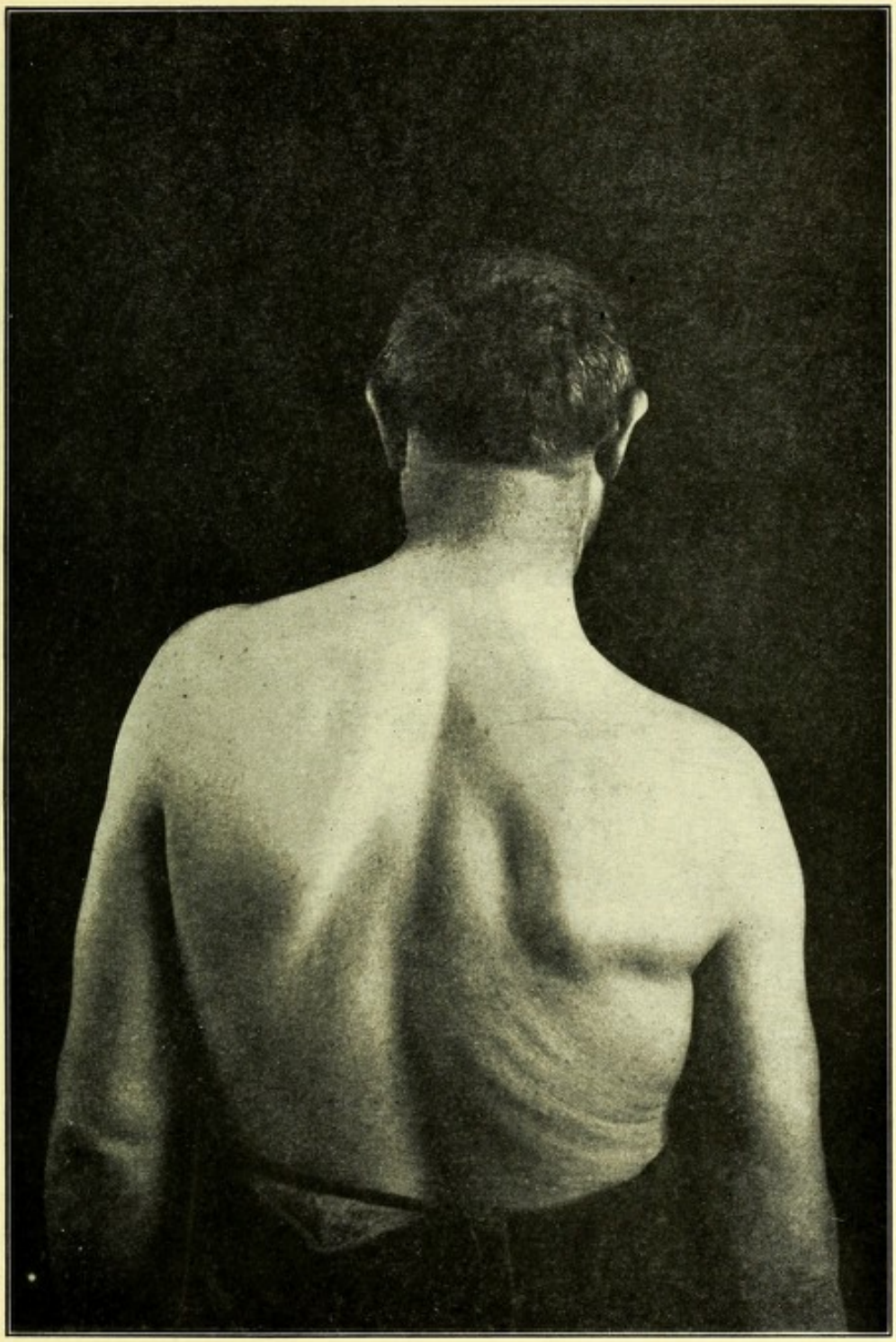


FIG. 59.—Paralysis of right spinal accessory nerve. Due to accidental section of the nerve at operation.

internal jugular vein, and the internal and external carotid arteries. Because of this association, most injuries of the nerve in its deep course prove fatal from involvement of one or other of those important structures. It is only after it has passed under the occipital artery, to proceed beneath the stylohyoid to the tongue, that it may be injured without great danger of involving the large blood vessels and the pneumogastric.

A patient at the Vanderbilt Clinic presented classical symptoms of left-sided paralysis of the tongue. He had been stabbed in the neck several years before, and sought medical advice for an aneurism of the left occipital artery. The stab wound must have divided the nerve near the occipital artery, but only have caused slight injury to the blood vessel.

Unilateral hypoglossal paralysis abolishes motor power in the corresponding side of the tongue. When the tongue is protruded it deviates toward the paralyzed side. Eating and talking may thus be seriously interfered with. Sensory and gustatory abnormities do not occur. The affected side of the tongue diminishes in volume, and its surface is traversed by deep furrows in which collect epithelium and dried secretions.

Traumatic hypoglossal paralysis can only be cured by operation.

Phrenic Nerve.—Paralysis of the diaphragm, resulting from injury to the phrenic nerve, which arises from the third, fourth, and fifth cervical nerve roots, is the one important symptom of injury to the cervical plexus. The other branches of the cervical plexus are chiefly sensory; their distribution may be seen by reference to the chart (see Fig. 54). Unilateral paralysis of the diaphragm, although an unusual accident, may result from shot wounds and stabs of the neck. The symptoms are dyspnœa on exertion and unilateral loss of the abdominal type of breathing. Litten was the first to describe

a bilateral wavelike movement caused by contraction of the diaphragm, which may be seen on the thorax of normal individuals. The movement, which is very slight, descends from the sixth rib on inspiration, to occur again on expiration. It is not visible in paralysis of the diaphragm. The prognosis of phrenic paralysis is grave.

Posterior Thoracic Nerve.—This nerve supplies the serratus magnus. Although isolated paralysis of the posterior thoracic is not common, it may result from falls on the shoulder or from injuries to the neck. A woman at the Vanderbilt Clinic developed it immediately after delivery. Such occurrences are explained by compression of the nerve through muscular contraction. Traumatic paralysis of the serratus magnus is always unilateral. The symptoms are characteristic. At rest, the internal edge of the scapula is prominent, and its lower angle is nearer the vertebræ than on the unparalyzed side. When the arm is held forward the scapula has the peculiar prominent position which has received the name of "winged scapula." Ability to raise the arm above the horizontal may be preserved. Recovery occurs in about one-third of the cases only.

Circumflex Nerve.—The circumflex nerve supplies the deltoid, and is sometimes paralyzed from injuries around the shoulder. As a result the shoulder is flattened and, as movements of the deltoid become impossible, the arm can hardly be raised at all. If there is much atrophy, a subglenoid dislocation of the humerus is usually present. The circumflex also supplies the teres minor, but paralysis of this muscle is unimportant. The electrical reactions, sensory symptoms, and prognosis in paralysis of the circumflex are similar to those of other nerves.

Musculocutaneous Nerve.—Isolated paralysis is very rare. The motor symptoms are referable to the biceps and the brachi-

alis anticus, and the sensory symptoms to the sensory distribution of the nerve.

Median Nerve.—Isolated paralysis of this nerve is uncommon, but median-nerve palsy is not infrequently associated with palsies of other nerves affected by injuries around the shoulder joint.

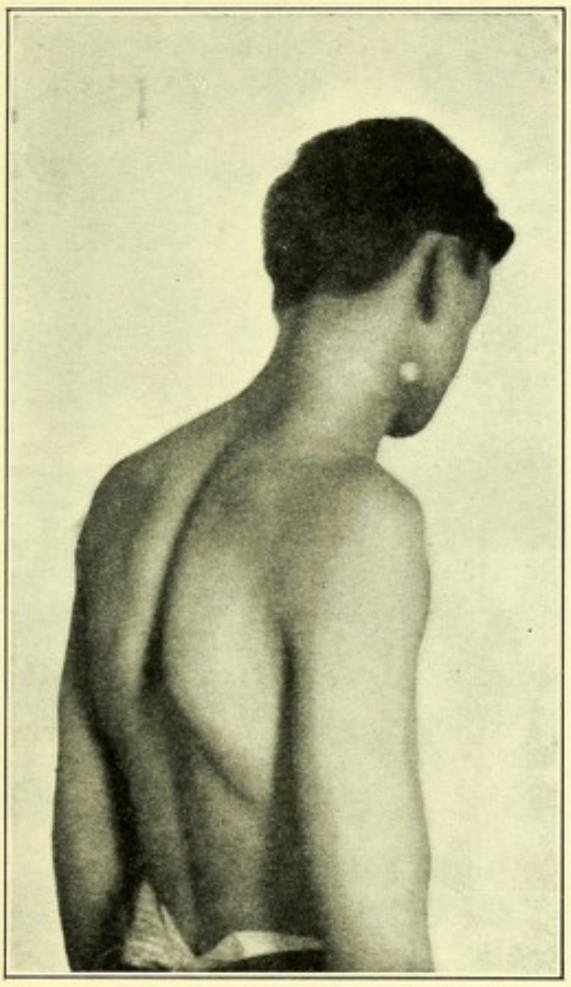


FIG. 60.—Atrophy of deltoid and drooping of shoulder due to paralysis of the circumflex nerve.

The symptoms are paralysis or weakness in the muscles supplied by the nerve. From paralysis of the pronators, the arm is slightly rotated outward, and pronation is impaired or impossible. From paralysis of the flexor carpi radialis, flexor sublimus digitorum, and of one-half of the flexor profundus digitorum, flexion of the wrist and fingers is interfered with. The nerve supply by the ulnar of the flexor carpi ulnaris, of the inner half of the flexor profundus digitorum, and of all the interossei, permits, when the ulnar nerve remains intact, some flexion of the wrist and of the terminal phalanges of the two inner fingers and flexion of the first phalanges of all the fingers. Through overaction of the extensors of the thumb (posterior interosseus) and of the

adductor (ulnar) the thumb assumes the position of extension and adduction, being in the same plane as the fingers. Sensory symptoms are usually unimportant; they occur in the sensory distribution of the nerve. According to Bernhardt, paralysis of this nerve is associated with trophic disturbances (atrophy, glossy skin, etc.) with especially frequency. The electrical reactions and prognosis have no individual characteristics.

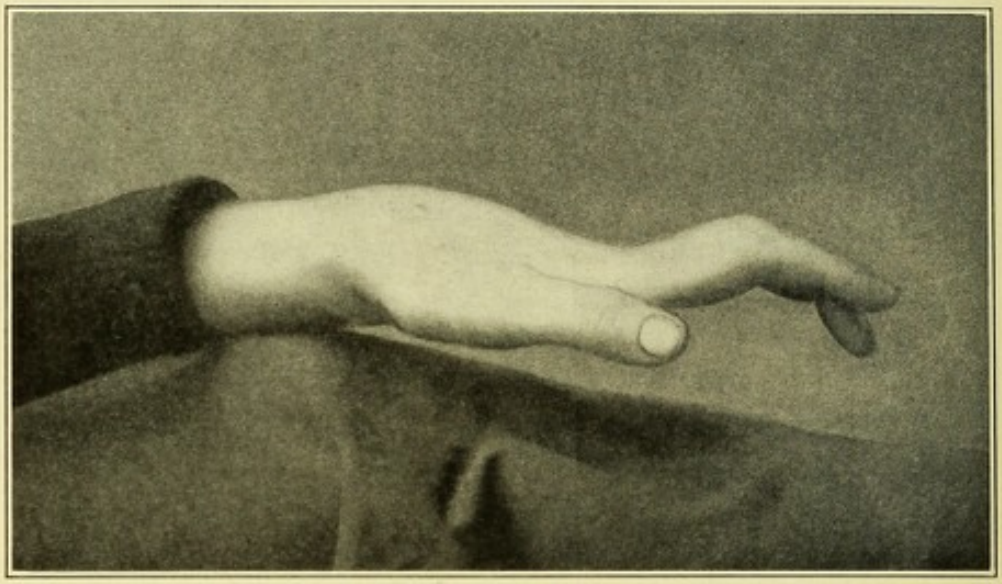


FIG. 61.—Deformity of the hand caused by injury to the ulnar and median nerves at the wrist.

Ulnar Nerve.—The ulnar nerve arises from the inner cord of the brachial plexus, derived from the eighth cervical and first dorsal segments of the spinal cord. From its origin in the axilla, until it passes through the olecranon groove, its course is superficial. In the upper part of the forearm it is covered by the flexor carpi ulnaris, but in the lower half of the forearm it is covered only by integument and fascia. Its superficial situation renders it very liable to injury. Paralysis may follow blows, cuts on the arm or forearm, dislocation, and fractures of the humerus or clavicle; it may be compressed by callus, or strained by continued or sudden forced flexion of the

forearm; it may be dislocated at the elbow, or be involved by fractures and dislocations at the elbow, in the forearm, or at the wrist. Of three cases of ulnar palsy recently coming to the Vanderbilt Clinic, one was caused by a piece of steel hitting the inner side of the arm, and two by falls on the elbow. The records of the Vanderbilt Clinic show that of all peripheral paralyses due to direct and immediate violence, ulnar paralysis is second in frequency. Unlike the musculospiral, it is rarely the seat of pressure or sleep palsy.

The symptoms are chiefly motor, and their distribution may be readily inferred by recalling the distribution of the nerve. Loss of power in the flexor carpi ulnaris causes weakness in flexion of the wrist; loss of power in the inner half of the flexor profundus digitorum causes weakness in flexion of the two internal fingers. In complete ulnar paralysis the little finger cannot be moved at all.

The most characteristic and disabling effect of paralysis of this nerve is paralysis of the interossei, all of which it supplies. The interossei flex the first and extend the second and third phalanges of the fingers, which actions are necessary in writing and in most of the finer movements of the hand. The loss of them causes very serious inconvenience. The interossei also adduct and abduct the fingers and adduct the thumb, movements which can no longer be performed when the ulnar is paralyzed.

Atrophy of paralyzed muscles and overaction on the part of opposing muscles are early complications in ulnar palsy. In beginning stages the condition is manifested by slight overextension of the first phalanges of the fingers, especially the little finger (Fig. 62), and an inability to firmly extend the terminal phalanges. If the palsy is complete and permanent, the deformity becomes very marked, constituting the claw hand or *main en griffe* of Duchenne.

The sensory distribution of the ulnar nerve is limited to the inner side of the anterior and posterior surfaces of the hand. Posteriorly it extends a little above the wrist and includes part of the dorsal surface of the middle finger, and the ring and little fingers; anteriorly it is limited to the hand and two inner fingers.

The sensory symptoms which occur as a result of ulnar

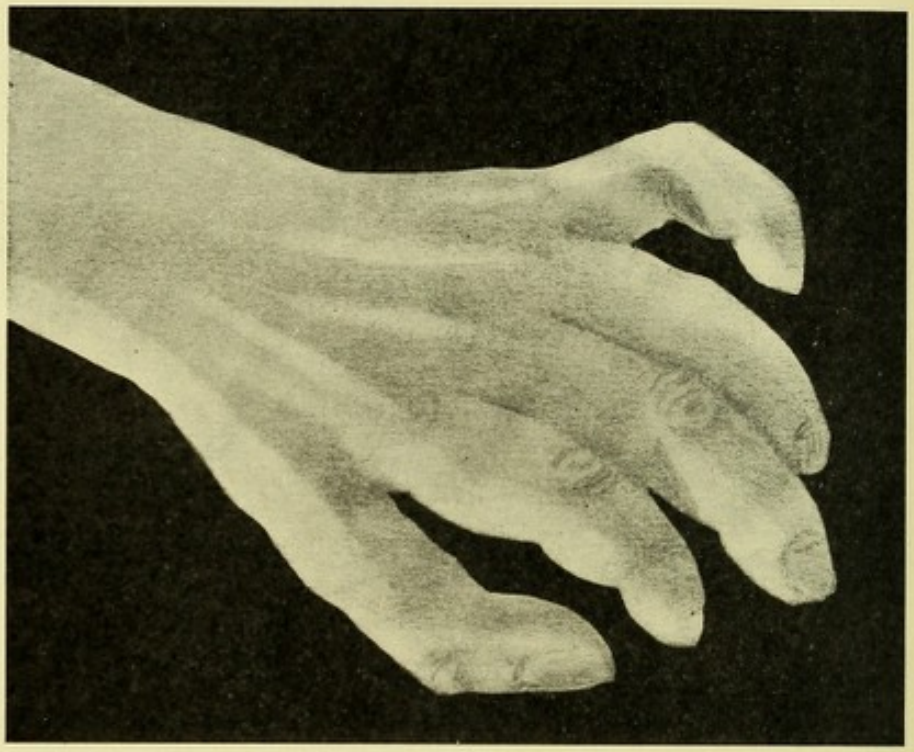


FIG. 62.—Beginning *main en griffe*. From an injury to the ulnar nerve.

palsy are chiefly subjective—numbness, tingling, and similar sensations. There may be some diminution of cutaneous sensibility, but complete anæsthesia is unusual, unless the nerve is inflamed as well as injured.[1]

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[1] The ulnar nerve seems particularly liable to the somewhat unusual affection known as ascending neuritis, or neuritis migrans. In this extremely painful disease a peripheral nerve injury, with external wound, is followed by extensions of the paralysis and the pain up the course of the nerve or to other nerves. In a

When ulnar palsy occurs as a result of pressure from dislocated bones it usually recovers in a few weeks or months, if the dislocation is reduced and if the nerve is not too seriously lacerated, or compressed. In fractures there is more danger of the nerve being lacerated, and consequently the prognosis should be given with greater reserve. If the paralysis be due to pressure exerted by callus, operation will be necessary for complete recovery. Electrical examination may be found useful in deciding upon the necessity for operation. From ordinary blows the nerve usually recovers perfectly; the prognosis when the nerve has been divided is the same as for other nerves.

Musculospiral.—The musculospiral nerve arises from the posterior cord of the brachial plexus, and carries in its trunk fibers from all the nerve roots which form the brachial plexus, with the exception of those from the first dorsal and those from the fifth cervical. Fibers from the fifth cervical are sometimes included (Quain). It is probable that in this nerve, as in many others, the cells from which the motor fibers of the nerve arise are not limited to the spinal-cord segment from which the nerve root springs, but may come from the anterior horn cells of any one of several segments.

Its situation in the axilla exposes the musculospiral nerve to injury; dislocations, and fractures of the humerus and clavicle are frequently followed by paralysis of this nerve. In the arm the most frequent cause of musculospiral palsy is pressure, whether caused by a crutch or by weight of the body during sleep. Blows and wounds to the forearm, also fractures of the bones, may abolish its function. Musculospiral paraly-

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patient at the Vanderbilt Clinic an ulnar neuritis developed, probably from a cut on the little finger, and eventually the nerve became paralyzed for all its functions. The pain was intense, and the swollen nerve could be felt as a round cord in the whole of its peripheral course.

sis has also resulted from falls on the hand and from sudden and forcible extension of the arm.

The symptoms are generally familiar. If the nerve is injured in the axilla or upper part of the arm, there will be pa-

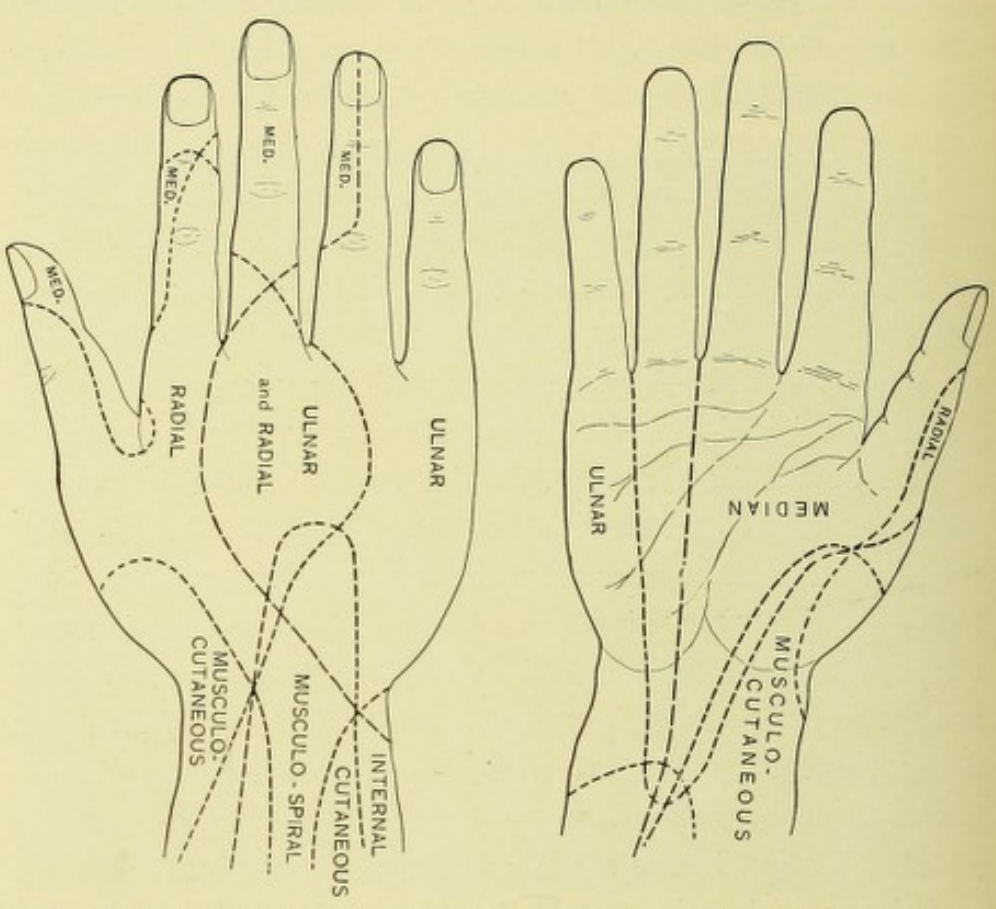


FIG. 63.—Distribution of the sensory nerves in the hand. (After Quain.)

ralysis of the triceps as well as of the muscles situated lower down. If the nerve is injured below the branch for the triceps, that muscle will escape. The other muscles involved are the supinators of the arm, the extensors of the wrist and fingers and of the thumb.

Complete musculospiral paralysis causes the familiar drop wrist (Fig. 64). When drop wrist continues for any length of time there develops a prominence on the back of the hand

due to thickening of the tendon sheaths. The hand hangs helpless, and all efforts on the part of the patient at extension of the wrist or of the first phalanges of the fingers are ineffectual. If the hand is supported, thus giving a *point d'appui* to the interossei, the terminal phalanges can be extended and the fingers separated. The power of abduction and extension of the thumb is lost. It frequently happens that the extensors are not completely paralyzed; when this is the case the paralysis is least marked in the index and little fingers. Paralysis of the supinators, especially of the supinator longus, causes the arm to pronate when any efforts at flexion of the wrist or fingers are attempted. In this way the grip is very much lessened in force. By paralysis of the supinator longus the

FIG. 64.—Drop wrist, from palsy of the musculospiral nerve.

power of flexion of the forearm is diminished. If the triceps is involved, extension of the arm is impaired or lost. The methods of examination for most of these muscular functions are obvious. The most satisfactory test for weakness in the supinator longus is for the patient, with the ulnar side of the forearm resting upon a table, to attempt to raise the forearm against the resistance of the hand of the examiner.

The sensory symptoms of paralysis of this nerve are usually limited to numbness and tingling of the radial side of the forearm and hand and of the back and outer side of the arm. The extent of the atrophy depends upon the severity of the injury. The electrical reactions are the same as for the other nerves. In the larger number of cases the injury is of moderate severity, without pronounced reactions of degeneration.

The diagnosis of traumatic musculospiral paralysis ordinarily presents no difficulties except when a history of accident is unsatisfactory. Then it is well to remember that lead paralysis is usually bilateral, and only exceptionally affects the supinator longus and extensor longus pollicis. Also lead poisoning causes electrical changes of serious import. In progressive muscular atrophy the paralysis is rarely complete, and its distribution is ulnar as well as radial. In progressive muscular atrophy the electrical reactions remain for a long time normal.

The prognosis of musculospiral paralysis is in general very favorable; unless the injury has been unusually severe, recovery is the rule.

Combined Paralyses of the Upper Extremity

Blows or falls on the neck or shoulder or arm, and dislocations and fractures of any of the bones of the upper extremities, may cause paralysis in more than one nerve.

Thus a patient came to the Vanderbilt Clinic with paralysis of the musculospiral, median, and ulnar nerves, which came on immediately after a fall on the palm of the hand.

The musculospiral and ulnar are the nerves most frequently involved together in injuries around the shoulder joint,

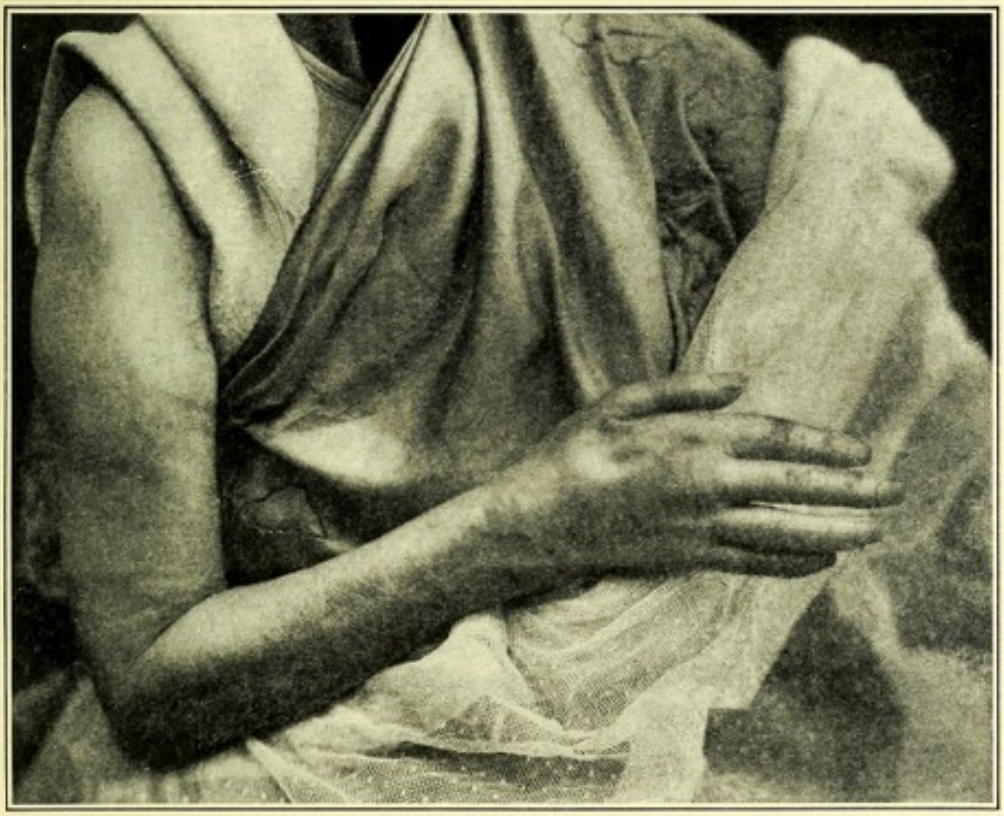


FIG. 65.—Injury to the brachial plexus from fracture of the clavicle. Paralysis of all the nerves of the upper extremity.

although paralysis of the circumflex, or the median, or the musculocutaneous may be added. The symptoms of these combined paralyses are the sum of the symptoms of lesions of the individual nerves. They are usually the result of severe injuries, and the prognosis is accordingly serious (Fig. 65).

Injuries to the neck, falls upon the point of the shoulder, and less frequently dislocations of the shoulder, sometimes cause a peculiarly distributed paralysis, first described by Erb,

and often called Erb's palsy. The muscles most frequently affected are the deltoid, biceps, brachialis anticus, and supinator longus. In several of the Vanderbilt Clinic cases the supra- and infraspinati were involved as well. In these cases there is slight inward rotation of the arm. All these muscles, except the last two, receive their innervation through the fifth and sixth cervical nerve roots. The suprascapular nerve, which supplies the supra- and infraspinati muscles, receives some fibers from the fourth cervical segment, but most of its fibers come from the fifth and sixth nerve roots, and so it may easily be injured when those roots are affected.

Hoedemaker has suggested that in injuries to the shoulder paralysis of these nerves may occur by the fifth and sixth nerve roots being compressed between the transverse processes of the sixth and seventh cervical vertebræ and the middle of the clavicle.

In cases where the paralysis is severe this affection is very disabling. The arm cannot be raised from the side, and the forearm cannot be flexed or strongly rotated outward. From paralysis of the deltoid the shoulder of the affected side is lower than its fellow, and there may be a slight subglenoid dislocation of the humerus. Atrophy is often an early symptom, and there is usually marked fibrillary twitching in the muscles, when, if not completely paralyzed, they are put in action.

The sensory symptoms are never prominent; there may be numbness and tingling in the region of the shoulder, or in the radial distribution of the forearm and hand. The electrical reactions soon show degenerative changes.

All these muscles may, in health, be made to contract by applying the electric current at a point in the neck called Erb's point (see Fig. 8); after injury, disordered electrical reactions soon become manifest at this same point. A Vanderbilt Clinic case after a fall on the right shoulder com-

plained of some weakness in the shoulder muscles of that side, especially in the morning. The man was left-handed, and examination showed only a comparative weakness of the right deltoid, biceps, and supinators. This weakness might have been a normal difference between the right and left side, and the diagnosis was not clear. But when the galvanic response in these muscles, obtained through Erb's point, was found considerably exaggerated on the right side, it became evident that the patient was suffering from a very mild injury to the upper part of the brachial plexus.

FIG. 66.—Characteristic deformity and interferance with growth in Erb's obstetrical palsy. (Drawing after Clark.)

The diagnosis of this affection is extremely simple if the patient is examined for muscular power and by electricity. In severe cases, in which the deltoid is so relaxed that it permits a slight subglenoid dislocation of the shoulder, the condition is sometimes mistaken for a simple dislocation. Such errors are the results of superficial examination.

The general prognosis of this form of paralysis is serious, although a certain proportion of patients recover. In my experience, however, such recovery has always been tedious, extending over many months.

A form of Erb's palsy in which the prognosis is always bad is obstetrical palsy (Fig. 66). This is caused by undue stretching of the plexus during delivery, usually by traction on the child's arm. The greatest strain comes on the cord formed by union of the fifth and sixth cervical roots. If these roots only are injured the distribution of the palsy is the same as that given above. But in many cases lower-lying roots are also injured, so there results disability of the whole arm. The roots may be merely lacerated, or actually torn across or out of the cord. In either case the prognosis is bad for the muscles paralyzed, the child usually growing up with shortened and deformed arm.

Another form of brachial-plexus paralysis involves the first anterior dorsal root. Through this root pass the sympathetic fibers for the eye. The condition was first thoroughly studied by Klumpke, and is called Klumpke's, or the lower-arm type of brachial-plexus palsy. It consists in a paralysis of the small muscles of the hand, and evidences of disturbance of the sympathetic on the same side of the face. There are myosis, diminished palpebral fissure, loss of the ciliospinal reflex, sinking in of the eye, and flattening of the face. There are no vasomotor disturbances. Klumpke's paralysis, as an isolated paralysis of the brachial plexus, is a great rarity. It occurs from causes similar to those already mentioned in lesions affecting the fifth and sixth cervical roots, causing Erb's palsy. The lower roots of the brachial plexus are affected much less frequently, however, than the upper, and Klumpke's paralysis is much more uncommon than the variety named after Erb.

Klumpke's paralysis is only infrequently seen in its pure form. It is usually more comprehensive, and differs in other ways from the variety of brachial-plexus lesion first described by that observer. Thus, in a case at the Vanderbilt Clinic, a man fell on the right shoulder, fracturing the clavicle. After the bone lesion had healed

the patient presented the following symptoms: Left myosis without other evidences of sympathetic disturbances; difficulty in raising and flexing the arm, and an almost total disability in performing the finer movements of the fingers and in flexing the wrist and hand. There was slight anæsthesia along the inner side of the arm. The deltoid and biceps reacted to faradism, but the muscles supplied by the median and ulnar nerves presented degenerative electrical reactions. Thus, in this case, myosis was the only evidence of disturbance of the sympathetic, but in addition to palsy of the circumflex and musculocutaneous nerves there was affection of the median and ulnar.

Paralyses of the Lower Extremity

Any of the nerves of the lower extremity may be paralyzed through injury. But such palsies are very much less frequent than those of the upper extremity, and only a few of the more important ones will be described. The characteristics of the others may be readily inferred from the function of the particular nerve affected. Injuries of the sacral or lumbar plexuses as such are unusual.

Hartmann has described a stab wound, which, entering the pelvis by the sacrosciatic notch, severed the third and fourth sacral roots and the obturator and crural nerves.

Crural Nerve.—This nerve supplies the internal iliac, the psoas, the sartorius and part of the pectineus, the quadriceps femoris, and the sartorius. Injury of the nerve within the pelvis, from fractures of the vertebræ or of the pelvis, or by wounds, causes a paralysis of all muscles. More frequently, however, the nerve is injured in its course in the thigh by fracture of the thigh, and by wounds, so that the only important muscle completely paralyzed is the quadriceps. There results inability to flex the thigh or extend the leg. Walking is only possible when the patient drags the affected leg. The knee jerk is lost, and anæsthesia exists in the distribution of the nerve.

Sciatic Nerve.—Fractures of the vertebræ and pelvis, dis-

locations of the hip (especially thyroid dislocations), and fractures of the femur are among the important causes of paralysis of the sciatic. Stab or bullet wounds are rarely mentioned as causes. Although the sciatic supplies the rotators of the hip and the flexors of the leg, these muscles are seldom completely paralyzed by injury to the nerve. Much more frequently the paralysis makes itself evident in lower-lying muscles, especially those supplied by the external popliteal nerve, less frequently those supplied by the posterior tibial. The knee jerk is unaffected, and anæsthesia is usually limited to the distribution of the anterior tibial.

External Popliteal Nerve.—This nerve, from its exposed situation at the knee, is particularly liable to injury from incised wounds, from fracture of the fibula, either directly or by callus, and from sudden exertion or strain, as in jumping or making false steps. The symptoms consist in paralysis of the muscles on the front and outer side of the leg. The foot cannot be raised, there is stepping gait, and tendency to inward rotation of the foot. There is a strong tendency to early contracture in the tendo Achillis. The distribution of the anæsthesia is shown on the chart.

Posterior Tibial Nerve.—Injury of this nerve is rare. When paralyzed, there is loss of plantar flexion of the foot and toes.

Injuries of the Sympathetic Nerves

The sympathetic nerves consist of a chain of ganglia which extends on either side of the vertebral column, from the sacrum to the skull. The individual ganglia are connected together by longitudinal strands. They receive, by means of the nerve roots, communicating fibers from the spinal cord; and they send, by means of the cerebro-spinal nerves, filaments which break up into many fine branches, forming the sympathetic

plexuses, which are distributed to the smooth muscle fibers throughout the body, especially to those of the blood vessels of the stomach, intestines, air passages and lungs, the genito-urinary organs, the dilator of the pupil, the smooth muscle of the eyelid, the sweat and secretory glands. The heart muscle is also supplied by them, together with the pneumogastric. The sympathetic connects the cerebro-spinal centers with the parts to which it goes. For example, the cervical sympathetic helps transmit the impulses from the ciliospinal center in the cord to the pupil.

The sympathetic is divided into cranial, cervical, thoracic, abdominal, and pelvic regions. Of these, the cervical, with the upper thoracic, is the only region upon which reliance can be placed for topical diagnosis. Lesions of the sympathetic lower down, which may readily occur in all injuries to the vertebræ, are doubtless concerned in the tympanites, the cyanosis, the coldness or heat of the extremities, etc., often seen in traumatic paraplegias. But such symptoms are not topical. The evidence that the thoracic viscera can be affected by sympathetic injury is meager, though the following case of Wallenberg's is very suggestive in this connection:

A previously strong man was stabbed in the back with the result (as shown by the anæsthesia) that the third dorsal nerve was injured. There was at first dyspnœa and precordial oppression, and for a long time afterwards slowing of the pulse.

Lesions of the cervical sympathetic have highly characteristic symptoms which are almost exclusively paralytic in character (see Fig. 67). When complete, they consist in narrowing of the palpebral fissure (sympathetic ptosis), sinking in of the eyeball, contraction of the pupil, with possible slowed light reaction, loss of the ciliospinal reflex, flattening of the side of the face, redness and dryness of the skin of the side of the

face. Except in cases of complete division, these symptoms are rarely, if ever, all present. Reddening of the cheek is very rare, though the cheek may be warmer. Lack of sweating is not unusual. There may be excessive flow of tears on the affected side. The injury which produces them may affect the spinal cord, the spinal nerve roots, the anterior divisions of the spinal nerves, the sympathetic ganglia themselves, or the cords which connect them. When the spinal cord is the seat of lesion, the symptoms, due to involvement of the ciliospinal center, are usually confined to contraction of the pupil and slight narrowing of the palpebral fissure. This is seen in injuries to the lower cervical and upper dorsal region. The causes are those of spinal-cord injury, generally. The symptoms characteristic of injury to the spinal-cord nerve roots are the same, though usually more pronounced, plus the sinking in of the eyeball. The spinal impulses which have to do with the pupil and eyeball leave the cord by the first dorsal root chiefly, possibly also by the eighth cervical and second dorsal. When these roots are injured we have the Klumpke type of paralysis (q. v.). Injuries to the roots result from falls and blows on the shoulder, with or without dislocation and fracture, and rarely from wounds.

FIG. 67.—Paralysis of left cervical sympathetic. Narrowed palpebral fissure, small pupil, sinking in of eyeball and flattening of the face. (Drawn after Oppenheim.)

Injuries to the neck may give the complete symptomatology of sympathetic lesions. The causes are stab and bullet wounds and fractures of the clavicle. That injuries within the skull can cause the same set of symptoms is a matter of con-

siderable doubt. A few cases are on record in which, associated with vascular lesions of the basal ganglia and cortex, there were present some symptoms similar to those mentioned for lesions of the cervical sympathetic. Authenticated cases of such effects from trauma are not available.

Traumatic affections of the cervical sympathetic are not disabling. The excessive secretion of tears is the only really annoying symptom. The prognosis varies with the cause. In spinal cord and root affection there is usually great improvement, though symptoms enough are left to be detected. In wounds of the neck, the symptoms may be permanent.

Traumatic Neuralgia

Injuries of nerves in which paralysis is a pronounced feature usually run their course without pain. Notable exceptions to this are cases in which there has been much laceration of tissue, and in pistol-shot wounds, in both of which there results extensive scar formation. As a rule, however, it is in slighter injuries, when paralysis is not pronounced, that nerve pain is most frequent.

Symptoms.—The symptoms are those of neuralgia generally. Along the course of one or more nerves there are sensitiveness to touch, particularly at certain points, perhaps slight anæsthesia, and more or less constant pain, which usually becomes, in attacks, very severe. The pain may set in at once, but usually does not become severe till some days after the injury. In lacerated wounds the interval may be even longer.

Thus, by the bursting of the barrel of a shotgun, a gentleman sustained a severe laceration of the left hand, particularly on the ulnar side. Three weeks later he began to have neuralgic pains in the ulnar distribution, which soon became excruciating.

Prognosis.—The prognosis of traumatic nerve pain is good, in that the patients eventually recover. But recovery is always slow, and relapses are not uncommon. The persistent pain and loss of sleep make for conditions of great general nervousness, or even hypochondriasis, and for drug habituation. This is particularly the case in individuals of neurotic temperament, who furnish a large proportion of the cases of traumatic neuralgia. Of the cranial nerves, the fifth is the only one liable to traumatic neuralgia, and that very rarely. Traumatic nerve pain of this nerve has been mentioned as a sequel of fractures at the base of the skull, but more frequently arises from injury to one of the peripheral branches, particularly bullet wounds. It does not have the severe course of the nontraumatic variety.

Injuries about the elbow, and especially about the shoulder joint, are the ones most frequently followed by pain. These injuries may be fractures and dislocations of the bones, which irritate the nerve at once, or which, later, by the formation of scar tissue, compress it.

There is a form of brachial neuralgia, or neuritis, in which, after very trivial injuries, such as slight twists of the elbow joint or strains of the muscles, severe symptoms develop. It occurs generally in neurotic or gouty persons. The pain begins in the joint, or the muscles surrounding it, and runs down the arm. Certain movements, varying in different cases, cause intense pain, and the muscles often develop considerable spasm. There are usually painful points about the shoulder or in the arm, and there may be slight anæsthesia. There may be atrophy of some of the muscles. General symptoms of joint disease are lacking. This affection has a very tedious course toward recovery, and is almost invariably attended with insomnia and general nervous symptoms. It causes considerable disability, but always is recovered from. It is frequently met with in litigated cases.

In intercostal neuralgia the pain follows the course of the intercostal spaces, the points of especial tenderness being just external to the spine and in the axillary line. The pain is more constant and less paroxysmal in character than in other neuralgias, but is intensified by all movements, such as deep breathing, etc. It results from injuries to the spine or to the ribs, and from contusions and wounds to the chest. It may be followed or accompanied by herpes.

While any of the nerves of the lower extremity may become painful as a result of trauma, the sciatic is the only one so affected frequently enough to require special mention. Injuries to the nerve itself through stab wounds, bullet wounds, fractures of the pelvis, and dislocation or fracture of the femur, or indirect injuries, such as blows or falls on the buttocks, and straining of the thigh and hip muscles, may bring about sciatica. The pain follows the nerve distribution completely or partially. The patient can often trace with the finger the exact course of the nerve, from its exit from the pelvis down the back of the thigh, around the leg, and down the peroneal region to the heel. Or there may be places in the nerve's course which are not painful, or the pain may be limited to the hip and thigh. There is often, also, pain running up over the back of the pelvis. The pain is generally constant as a dull pain, greatly increased by movement (walking), and subject to exacerbations, especially at night. Scoliosis, with concavity toward the affected side, often results, probably from the pain or spasm, and is curable.

The painful pressure points are at the sciatic notch, at the middle of the nerve trunk in the thigh, and at the external malleolus of the fibula. Sciatica is sometimes mistaken for other conditions, though such errors should not occur. It is distinguished from locomotor ataxia by the character of the pain, and by the absence of the general symptoms of the latter dis-

ease. The pain in spinal-cord tumor is usually bilateral, is accompanied with paralysis, bladder disturbance, and anæsthesia. In sciatica, anæsthesia is very rare, is scattered, and not complete. In sciatica there is sometimes slight wasting, but no true paralysis, and the gait is that characteristic of pain.

CHAPTER IX

TRAUMA AS A FACTOR IN THE CAUSATION OF CERTAIN CHRONIC DEGENERATIVE DISEASES

General Paresis—Locomotor Ataxia—Progressive Muscular Atrophy—Paralysis Agitans—Multiple Sclerosis—Tumors—Syphilis—Diabetes Mellitus.

WHILE the exact diagnosis of the acute conditions which have been described in the preceding pages sometimes present difficulties, their ætiology is in most cases easy of demonstration.

When an organic injury is immediately followed by symptoms of well-recognized pathological conditions in the nervous system there is rarely any question as to the propriety of associating the two occurrences in the direct relationship of cause and effect. The ultimate termination depends upon the nature of the injury and upon the part of the nervous system which has been affected by it.

In the general diseases of the nervous system the establishment of trauma as a cause is a much more difficult matter. In many of them trauma is doubtless a considerable factor, but in none is it a necessary one. They are for the most part chronic, and the time interval between the accident and the first appearance of symptoms is usually sufficient to admit the operation of other causes. In most of these diseases, also, the post-mortem appearances indicate a destroying agent more subtle, widespread, and persistent than trauma. At the same time our knowledge of the protean causes is imperfect. We are not, therefore, in a position to deny that under certain circumstances trauma might call them into play.

The origination from injury of diseases of the nervous system is necessarily so obscure that it cannot be even approxi-

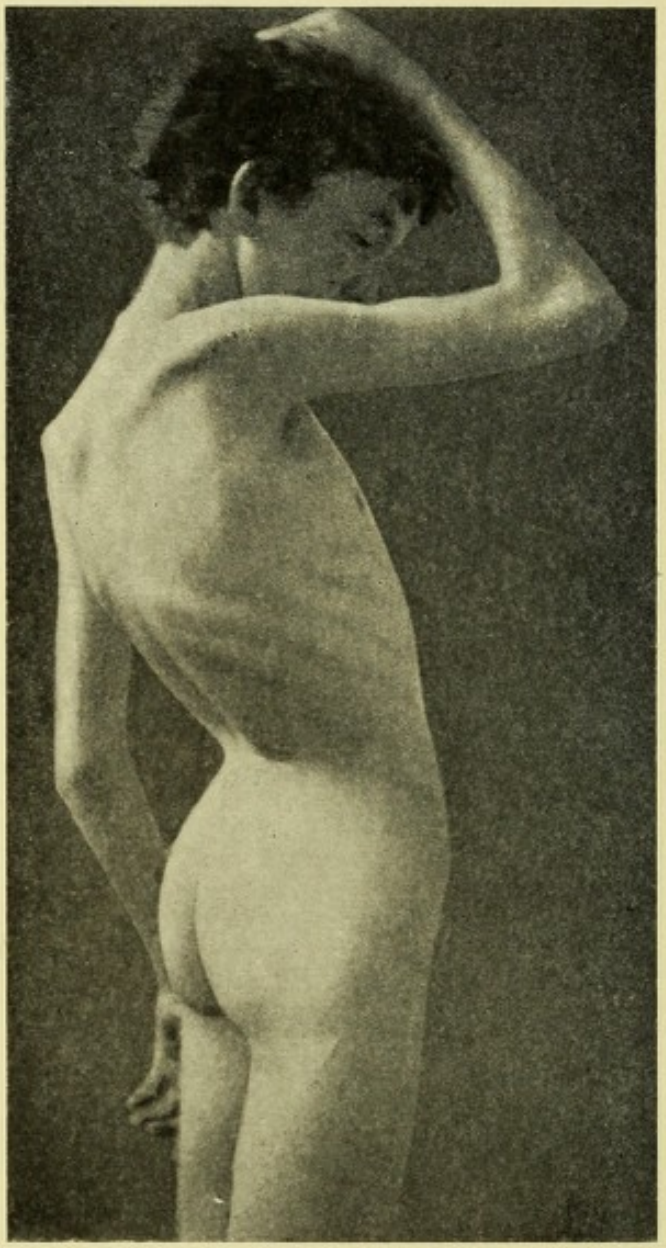


FIG. 68.—Deformity of the back caused by syringomyelia.

mately decided except for those disorders which are comparatively frequent. Thus, although there is no doubt but that spinal hemorrhage may give rise to conditions in the spinal

cord similar to those found in syringomyelia, there is as yet only very meager clinical evidence for the existence of traumatic syringomyelia.

Laehr reports two cases, in each of which the disease

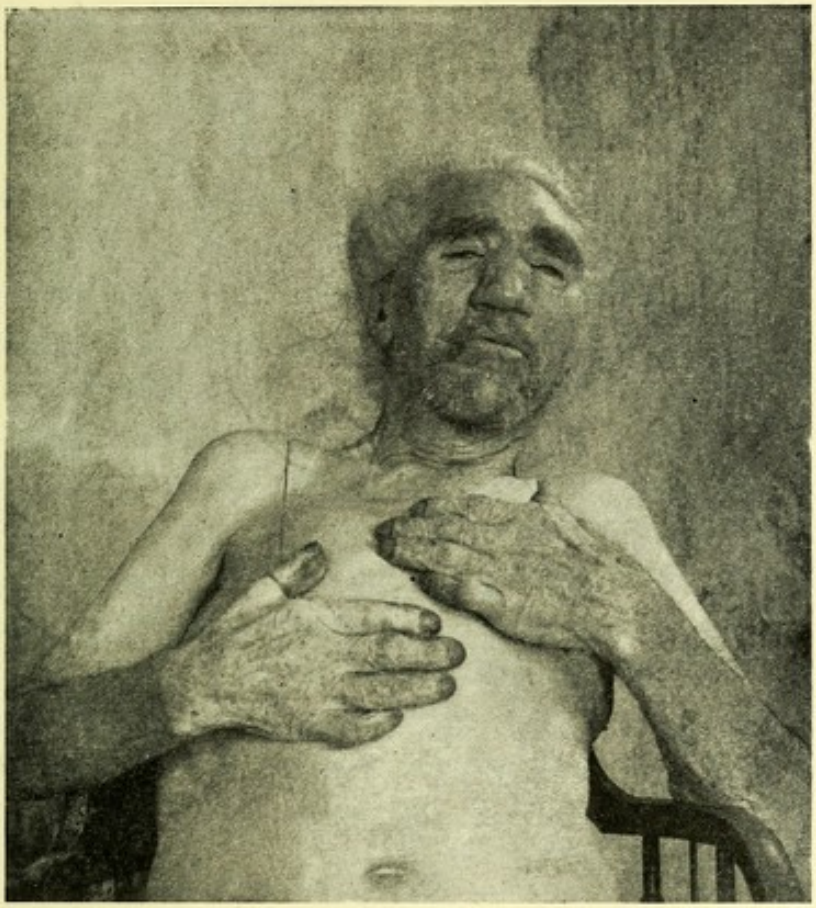


FIG. 69.—Acromegaly. (Photograph of a female patient who died in the Incurable Hospital.)

already existed at the time of the injury; and Huisman has recently described as traumatic a case in which the relationship of the injury to the spinal disease is not clear. A similar uncertainty exists in regard to the traumatic ætiology of ataxic paraplegia and chronic progressive spastic paraplegia.

So little is known about the pathology of acromegaly (Fig. 71), and the disease is itself so rare, that knowledge of the part

played by injury in its causation is still largely speculative. Unverricht, however, describes a case of typical acromegaly in a patient who had received an injury, and whom the examining physician had regarded first as a simulator and later as an example of "traumatic neurosis." Unverricht adduces several other cases of alleged traumatic origin.

There is, however, a group of comparatively common chronic degenerative diseases of the nervous system of which injuries are regularly spoken of as causes, and which, by reason of the symptoms being often observed for the first time after an accident, have important forensic relations. In some of them the occasional occurrence of trauma as a sole cause seems probable; in others the possibility of a traumatic origin is extremely doubtful, as is indicated by the general character of the disease and by the absence of well-authenticated cases which have occurred in this way.

So many factors must be taken into account in a consideration of these diseases and their relationship to injury that they require individual description. These diseases are: General paralysis of the insane, locomotor ataxia, progressive muscular atrophy, paralysis agitans, multiple sclerosis, tumors of the nervous system, syphilis of the nervous system, and diabetes mellitus.

General Paralysis of the Insane—General Paresis —Dementia Paralytica

An inquiry which has for its object the determination of the influence of trauma upon the development of general paralysis of the insane is beset with many difficulties. The disease has an initial stage, that may last for months or years, during which the patient is not only not incapacitated for work, but may conduct himself so rationally that no suspicion is entertained that

he is already suffering from a disease which is soon to destroy both mind and body. From the insidiousness of its onset, it is usually impossible to say even approximately when the morbid process began. In nontraumatic cases, when the first marked symptoms consist of an attack of acute maniacal excitement or of acute mental depression, or of some other acute manifestation, there is every reason to suppose that the disease had already existed, though unsuspected, for some time. Similarly, when an injury to the head is quickly followed by an outbreak of the symptoms of the disease, it is never possible to say with even reasonable certainty that the trauma did anything more than hasten into activity a process which was already existent, and whose ultimate development was inevitable, irrespective of traumatic agency.

Prodromal Stage.—Inasmuch as it is so often the subject of medico-legal inquiry, the prodromal stage was called by Le Grande du Saulle "the medico-legal period." How easily it may pass unsuspected by the general public or by medical men who are not especially versed in mental diseases, is shown by an incident related by Le Grande du Saulle, and quoted by Hamilton:

> Two brothers went to the office of a Parisian alienist, and the elder had a private consultation, the result being that he was informed that the other had the incipient signs of paretic dementia, and that death would occur in three or four years. They departed, and the result was that a policy of insurance was procured for 100,000 francs. Three years later the elder brother pocketed the results of the robbery.

The prodromal symptoms of general paresis are both physical and mental. At first the physical symptoms may be nothing more than a slight tremor of the face and tongue, with some indistinctness of speech. Very commonly there is an early myosis, or a pupillary inequality, or the pupils may show the

Argyll-Robertson phenomenon. The knee jerks are lost in about one-third of the cases; in the others they may be normal, or may, in common with other tendon reflexes, be exaggerated. The earliest variations from the normal mental state are a slight forgetfulness, inattentiveness, carelessness, or irascibility. The patient's character changes without his noticing it. "Der Kranke wird ein Anderer, und er merkt es nicht." The delusions of grandeur and extravagant acts, so common in the more advanced stages, are only occasionally prominent in the beginning of the disease.

FIG. 70. — Showing characteristic facial expression in the early stage of general paresis.

These mental signs, although they may not pass unnoticed by the associates or by the family of the patient, rarely excite any particular remark until they have existed for some time, and have become disagreeably prominent. Then inquiry fails to fix within weeks or even months just when the changes in personality began.

To determine the exact part played by any exciting cause in a disease whose beginning is practicably never determinable, appears well-nigh impossible. By the time the symptoms of paresis are sufficiently pronounced for the patient to be brought (he rarely comes of his own accord) for medical examination, the disease has already existed for some time; just how long a time it is impossible to say, but certainly long enough for the patient's own statements to have become unreliable, and for his friends to have forgotten when they first noticed changes in

his character. Consequently, whether any alleged cause was so far responsible for the disease that, had the cause not existed the disease would not have developed, will be a matter of individual opinion, and must be largely determined for each case. The most that can be done here in considering the influence of trauma in the causation of the disease is to briefly observe the general ætiology and to review the evidence for traumatic agency.

General Ætiology.—As the clinician is hampered by being unable to recognize the disease until it has already existed for a considerable length of time, and as the pathologist has failed to reveal the fundamental character or causation of its morbid anatomy, the ætiology of general paresis remains imperfectly understood.

Among the general ætiological factors the influence of syphilis is the most easily demonstrable. There is a variation in the observations of different investigators as to the percentage of general paralytics in whom a history of preceding syphilis is obtainable, but all agree that the percentage is large.

In Germany it is given by		
Mendel and Schnell	75 per cent	Quoted by Oebeke.
Binswanger	49–72 " "	Quoted by Oebeke.
Ziehen	33–43 " "	Quoted by Oebeke.
Gudden	surely 35.7 per cent; probably 9.6 per cent.	
Hirschl (Austria)	70 to 90 per cent.	
	56 per cent history of syphilis.	
	25 " " syphilis probable.	
In France it is given by Régis	70–90 per cent.	
In America it is given by Peterson	60–70 " "	
by Bannister	89 " "	

The frequency with which syphilis has preceded general paresis is certainly more than a coincidence, for in other forms of mental disease the history of lues is not obtainable in over

fifteen to twenty per cent. In what way the syphilitic poison causes the cerebral degeneration is unknown. Pathology has failed to reveal whether it acts simply as a predisponent, which requires the addition of some exciting cause to induce the disease, or whether the late poisons of syphilis are of themselves sufficient. The latter alternative seems the less probable from the fact that while syphilis is very frequent, general paresis, compared to it, is rare. If syphilis alone were capable of producing general paresis, it would be reasonable to suppose that the relative frequency of the occurrence of the two diseases would be more equal. Furthermore, general paresis is different, both clinically and in its pathological anatomy, from the affections of the brain characterized by syphilitic lesions. However, important as is the relationship between syphilis and paresis, it is not absolute. There are cases which give no evidence or history of syphilis, and in which the preëxistence of syphilis is not even probable. Fournier, who believes syphilis to be the sole cause of tabes, admits that paresis may occur without the patient having had the venereal disease.

There are other predisposing factors which must be considered as affecting causation. Paresis is much more frequent in males than in females (four to one, Mickle). In women of the higher classes of society it is very infrequent. The disease is sharply limited by age. An overwhelming majority of the cases occur between the ages of thirty and fifty; the disease is very rare before twenty-five, and rarely, if ever, begins after sixty. Heredity may often be found to be an important factor—not in the sense that general paresis is directly transmitted, but rather that the individual is provided with a brain which is particularly liable to succumb under stress.

The causes most commonly regarded as exciting in the ætiology of general paresis are mental excitement, worry, and overwork. The general class of persons among whom the dis-

ease is most common, the greater frequency with which it occurs in the inhabitants of cities, and the fact that it appears at an age when mental and bodily strain are at their highest tension, substantiate the hypothesis that these agencies are at least powerful contributing causes.

Trauma as a Cause.—The influence of accident and injury is universally regarded as important, and trauma appears in all books on the subject as one of the principal exciting agents. General injuries are sometimes described as determining causes, but it seems extremely improbable that any injury can be regarded as sufficient to induce general paresis unless the blow was directly applied to the head, or unless there was a considerable shaking up of the cranial contents, such as may occur in concussion accidents. In only a few of the reported cases has the skull been fractured.

The percentage of traumatic cases, as it is given by different observers, to the total number of cases of general paresis varies greatly. Schläger says: "One-seventh of all cases of mental disease induced by head injuries are cases of general paralysis." Meyer found 15 cases of injury to the head in 76 cases of general paresis, in which the causes "were clearly made out." In 80 male cases, Krafft-Ebing found cranial injury to be the cause in 6. Mickle quotes these authors, and from a study of the English lunacy reports finds six per cent of the cases in both sexes as due to head injuries.

Christian observed 43 cases of general paresis in 100 cases of injuries to the skull. This percentage is too excessive to merit serious consideration. In 500 cases of insanity Schläger ascribed a traumatic ætiology to 49, of which 7 developed general paresis.

Gudden reports 45 cases in which a history of head injury was prominent. In 6 of these the trauma had occurred in childhood, years before the symptoms of the general brain disease.

In 18 the trauma and the paralytic symptoms were separated by a period varying from six months to twenty years; and in 21 the paralytic symptoms developed in direct sequence (few months) upon the head injury. The accident was generally accompanied by loss of consciousness, and in 4 cases the skull was fractured. Of 175 cases reported by Hirschl, trauma had exerted an influence in 7.4 per cent.

Kaplan found a traumatic history in 4.4 per cent of the cases of the paresis. But he dismissed such statistics as valueless, as a larger percentage than that is found in sane persons. To prove this he examined 100 hospital patients between the ages of fifty-five and seventy-five, suffering from internal diseases, but free from all mental symptoms. No less than seven per cent of these had received severe head injuries between the ages of thirty-one and forty-three.

From the above reports of traumatic general paresis it appears that the injury was incurred in the great majority of cases by falls or by blows on the head received in brawls. Now, the disease is characterized by momentary attacks of dizziness or unconsciousness, which may cause the patient to fall and strike his head, by an inattention which exposes him to a variety of accidents, and especially by an excitable mental state, which frequently leads him into those animated discussions, as a result of which somebody's head is often injured. And, indeed, it seems as though the receipt of head injury was a result of the disease rather than the cause of it in by far the larger number of the published cases. The following case illustrates a history of the disease such as is usually described as traumatic, but in which the patient was undoubtedly suffering from paresis before the occurrence of the accident:

A. G., forty years of age, came to the Vanderbilt Clinic on December 18, 1893. Syphilis and alcoholism denied. The patient says he was well until six months previously, when he fell from a build-

ing. He did not lose consciousness, but was in bed two weeks with a swollen ankle. No paralysis. Two months later right hemiplegia and motor aphasia suddenly developed, lasted for two weeks, and then as suddenly disappeared. A similar attack of paralysis, also temporary, occurred a short time afterwards, and was followed in a few days by an attack of motor aphasia, unaccompanied by loss of muscular power. The day before I saw the man he had had convulsions in both arms without any loss of consciousness. Examination showed no paralysis, no ataxia, pupils equal and reacting to light, knee jerks absent; temporal limitation of the visual fields; tremor of face, tongue, and hands very marked; speech and expression characteristic of general paresis.

There was no question as to the diagnosis of the disease from which the patient was suffering, but the apoplectiform character of its course indicated very conclusively that the fall had not, as the patient claimed, caused the disease, but had been a result of one of the earliest seizures.

How necessary it is to consider every factor before ascribing to trauma a place in the causation of general paresis is well shown by the following case. Inasmuch as it is soon to become the subject of litigation, there will be no mention of names, dates, or places in describing it:

A man, thirty-three years of age, consulted a physician on account of an injury which he had received six weeks previously. The patient denied having had syphilis, and said that he had always been a strong and healthy man until he met with an accident in which he was struck on the head and knocked down by a passing vehicle. As a result of the injury he was carried to a hospital, and lay there unconscious for eight days. He then quickly recovered and returned to work, although still troubled with the headaches, for which he sought medical advice. The examination showed tremor of the face, lips, and tongue, thickness of speech, inequality in the size of the pupils, though both responded readily to light and during efforts at accommodation. All the tendon reflexes were active, but there was no paralysis. The general appearance and manner were those of a patient with general paresis. The gait was careless, the memory poor, the attention defective. The diagnosis and the evaluation of the influence of the injury were, however, postponed until further information could be obtained.

From the house surgeon of the hospital where the man was taken after the accident it was learned that on admission the patient was delirious and was paralyzed in one arm, but had no external injuries except a slight abrasion on one cheek. The mental condition resembled that of acute mania, with great excitement, delusions, and hallucinations. These soon passed away, however, as did also the paralysis of the arm.

Witnesses of the accident said that the man fell against the vehicle, instead of the vehicle running into him. His wife admitted that for a year before the accident her husband had been for-

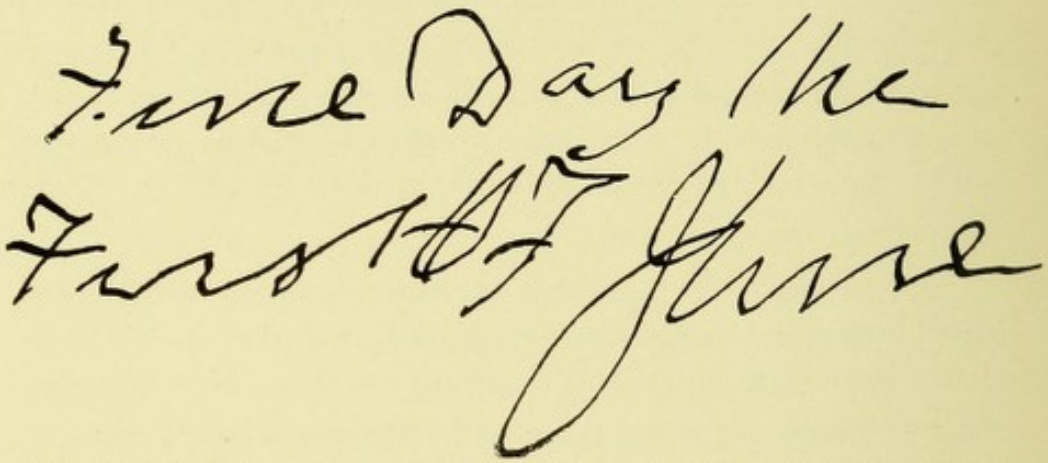


FIG. 71.—"This is a fine day for the first of June." Handwriting of a patient in the early stage of general paresis.

getful, irritable, and subject to headaches; that she had noticed that one pupil was larger than the other; that there had occurred from time to time paralysis of one arm or of one leg, or loss of the power of speech, but that these symptoms would pass away completely in a day or two. She also admitted that, having been pregnant seven times, she had never given birth to a living child, the abortion always occurring in the sixth or seventh month. These facts, together with subsequent observations of the patient, during which the symptoms became more marked, left little doubt as to the correctness of the diagnosis of general paresis; but they proved conclusively that it was not traumatic general paresis. The patient was in all probability syphilitic, as shown by the barrenness of his wife, who is a strong, healthy-looking woman. The mental disease had begun at least a year before the accident, as shown by the attacks of paralysis, the headaches, the inequality of the pupils, etc. The accident was in all probability a result of the disease, inasmuch as the wit-

nesses said that the patient fell and was then hit by the vehicle. Accordingly, in this case, in the absence of external evidences of injury, it is reasonable to maintain that the accident exerted little or no influence upon the course of the disease. It would probably not have occurred to a healthy man, but was one of the unavoidable penalties which are paid by persons who are unable to take care of themselves.

Cases similar to these have undoubtedly been the cause of much misapprehension as to the part played by injury in the causation of general paresis. The patient may himself believe in the truth of the history he tells regarding the occurrence of the accident, but the mental state of a disease which is characterized by delusions, hallucinations, and confusion render untrustworthy all the statements which may be made by anyone suffering from it.

If the disease is sufficiently advanced to be diagnosticated, there is no more reason for the physician to accept the patient's statements regarding accident or injury than there is to believe the expressions of grandiose ideas or of depression, or the assertions of magnificent health in a person who is evidently doomed. Consequently, no case of general paresis can be reasonably regarded as of traumatic origin unless the testimony of credible witnesses shows that the injury was the result of an accident which, as far as the victim was concerned, was unavoidable.

When it can be shown that the head injury was in no way the result of the disease, but was the consequence of one of the many accidents to which all people are commonly exposed, it is necessary to determine as clearly as possible in how far it can be held as responsible for the development of the paresis.

The inquiry should be especially painstaking as regards three points:

1. In the first place, to avoid gross errors in diagnosis, it must be proved that the course and character of the symptoms

are identical or closely allied with those of general paresis. After some head injuries, especially if the skull has been fractured or there has been intracranial hemorrhage or brain laceration, there develops a condition of delirium and excitement, at the subsidence of which the patient may pass into a condition of dementia. Associated with the mental symptoms there may be the dysarthria, the evidences of localized paralyses, the tremors, and other signs similar to those which occur in general paresis. These patients may die, in which event the lesions found are more localized and more prominent than those of general paresis; or they may improve, and live in good physical health. Under these latter conditions, essential clinical differences from general paresis soon become evident. There is a constant tendency to mental improvement rather than to retrogression; the patients gain strength rather than lose it, and the delusions and grandiose ideas characteristic of the late stages of paresis are wanting.

2. The question of predisposition is very important in cases alleged to have resulted from trauma.

In nearly all the reported cases of general paresis which have followed injury, in which the receipt of the injury of itself might not have been regarded as a symptom of the disease, there was evidence that a strong predisposition, either hereditary or acquired, had preëxisted. The hereditary predisposition has been proved by the family history, by congenital syphilis, or by the stigmata of degeneration. The acquired predisposition has consisted in the results of preceding syphilis. Of all these factors syphilis is the most important. To deny a traumatic origin to the disease because the patient has had syphilis appears to me to be equivalent to saying that the patient would have had paresis whether his head had been injured or not. Such a view is not warranted by our present knowledge. Similarly to what will be said concerning locomotor ataxia,

there are so many syphilitic persons who receive severe head injuries, without developing general paresis, that in any syphilitic person in whom head injury is quickly followed by the symptoms of the mental disease it seems more reasonable to credit to the latter cause an influence at least equal to that of the venereal infection. However, this matter will always be one of individual opinion. Most of the cases of paresis occurring after head injury have had syphilis, and it is never possible to decide positively as to the relative value of the two factors. The number of recorded cases of persons who were syphilitic, yet who presented no symptoms of brain disease until after the receipt of a head injury, is not small. Thus:

Mickle tells of the case of a soldier with a long syphilitic history who suffered a severe injury to the head in July, and on the 29th of that month was admitted to the hospital with maniacal symptoms. Seven weeks afterwards he was discharged from duty, but was readmitted, in the January following, with general paresis, the onset of which had been characterized by the mental symptoms which followed closely upon the cranial injury.

In similar cases the syphilis had preceded the paresis by a period varying from one to twenty years, and the first mental symptom had appeared in a few months or years after the accident.

3. Can a person who has never had syphilis or marked hereditary predisposition develop general paresis as the direct consequence of injury to the head?

Dr. L. C. Pettit, of the Manhattan State Hospital, Ward's Island, New York, whose extensive researches bearing upon this disease are well known, tells me that "from a study of 2,000 clinical histories, he is forced to believe that head injury is not to be regarded as a sole cause of the disease. In over 100 autopsies made on paretics he has only once seen anything which seemed to be correlative to such a cause. In one case,

over the region of the right gyrus occipitalis superior there was a depression, about the size of a silver 25-cent piece, of the inner table of the skull, which was the result of injury. In exactly the same area on the left hemisphere there was necrosis of the cerebral cortex."

In reference to trauma as a sole cause, Mickle says: "In cases which have come under my own observation, where cranial injury has conduced to general paresis, it has, in the majority, seemed to play the part of a predisposing rather than of an exciting cause. In speaking of cranial injury as a predisposing cause of general paresis, we may suppose that, in consequence of latent residual results of the immediate effects of trauma, either the cerebral tissues are simply less resistant to the influences of the ordinary causes of the pathological process which underlies general paresis, or that this process springs more fully into being by assisting in, and in its turn being assisted by, the intensification and extension of slight local inflammation or hyperplasia sequential to the brain injury; or, again, assisted by morbid vasomotor effects of that injury."

The views of these observers seem to be substantiated by clinical experience. I have been unable to find in all literature a single case of traumatic general paresis in which sufficient details were given to justify the absolute conclusion that the head injury was the one cause of the disease.

The number of cases in which there is even a possibility that the trauma was the exclusive cause is very small. The most complete one is reported by Fox; but that observer himself admitted that the disease might have existed previously to the receipt of the injury:

The patient was a man, forty-nine years old, married eighteen and a half years; eleven children; wife miscarried once with twins. Family and personal history clear. No syphilis. Formerly a cool,

collected, precise, sober, and particularly neat man, who had always enjoyed very good bodily health. On December 27th he was thrown out of a trap, striking on his head. His wife asserts that he was in perfect mental health up to the time of the accident, and that some unusual irritability which had been noticed was simply due to a heavy cold, which his previous excellent mental health had unfitted him to bear patiently. Whether this was or was not the case, it is certain that, a day or two after the accident, various symptoms suggestive of general paresis appeared; so that, if the disease had not an exclusively traumatic origin, its development and first manifestation at least were determined by accident. The first day after the fall he complained of pain in his head, but after the first day made no complaints, and said he was never better in his life.

Sixteen days after the accident, when admitted to the hospital, the physical and mental symptoms of general paresis were easily recognizable. The disease pursued a characteristic and fatal course.

Another case is reported by Van Deventer (quoted by Bechholm):

A stonecutter, thirty years old, father alcoholic, patient himself slightly alcoholic, but no history of syphilis. He was struck in the left temple by a piece of marble and lost consciousness momentarily, but continued with his work after a few moments. The next day he became aphasic for twenty-four hours, restless, and unwilling to stay in bed. Had headache and insomnia. A few days later had an attack of violence and confused ideas of persecution. He entered the asylum three weeks after the accident, presenting symptoms of amnesia, megalomania, with hallucinations and persecutory ideas. Seven months later occurred clonic spasms and apoplectiform attacks; seventeen months later there were difficulties of speech, tremor, inequality of the pupils, and weakness of all the muscles. The patient died, twenty-five months after the accident, with symptoms of complete dementia.

Neither of these cases is conclusive evidence of the existence of head injury as a sole cause of general paresis.

In default of better proof of the traumatic origin of a disease which is so common, and which has so frequently been the subject of medico-legal inquiry, the conclusion is unavoidable that if trauma is ever the sole cause of general paresis,

such a causal relationship is extremely unusual and difficult of proof, and is to be accepted only after scrupulous inquiries have eliminated all of the many opportunities of error.

In spite of these facts, cases of general paresis advanced as traumatic are litigated for large verdicts. If the testimony is to the effect that prior to the accident the man was strong and able to manage his affairs, and that after it, no matter how trivial it may have been, he soon became changed, ultimately being incapacitated, or dying from the disease, the jury waives aside purely philosophical considerations and awards damages graded upon the patient's age and previous earning capacity.

Locomotor Ataxia—Tabes Dorsalis

Although locomotor ataxia is one of the commonest of the chronic degenerative diseases of the nervous system, there is very little definite knowledge regarding its pathogenesis, and many difficulties stand in the way of a complete understanding of its nature. The disease is not in itself a direct menace to life, and in the cases which come to autopsy the morbid process has existed so long and has become so extensive that it is impossible to tell when it had its beginning. Another difficulty in the study of its pathology is that, although only certain portions of the cerebro-spinal axis are involved, there is nothing about the microscopic appearances of the lesion itself which are exclusively characteristic. Its fundamental anatomical character is a slowly progressing degeneration of the posterior columns of the spinal cord, usually associated with an inflammation of the pia mater and a degeneration of the posterior nerve roots and of the spinal ganglia. It is essentially a degeneration of nerve fibers and nerve cells, associated with a growth of connective tissue and sometimes with degeneration of the walls of the blood vessels. All these morbid changes are found in

other diseases. Even the clinical fact that syphilis frequently precedes tabes receives no substantiation from the microscopical anatomy of the latter disease. From a study of the spinal cord alone in a case of tabes it would be impossible to say whether the patient had or had not had syphilis. Whatever the pathological nature of tabes may be, it is certainly something more than spinal syphilis. It is conjectured that the ultimate products of the syphilitic virus exercise an elective action on certain portions of the spinal cord or of its nerve roots, and that the tabetic degeneration is in reality syphilitic. This view is largely speculative and lacks anatomical proof. In the absence of such proof it is unjustifiable to claim an absolute causative influence for syphilis.

But though the morbid anatomy of tabes has been of little or no service in determining the causation of the disease, it has yielded an important result in showing that the character of the lesion is degenerative rather than actively inflammatory, and that it requires a considerable time for its development. The nerve-fiber degeneration, the connective-tissue growth, the vascular abnormities, all bespeak a process which is essentially slow, and argue against the possibility of the disease reaching a marked degree of development within a few days or weeks.

Preataxic Period.—The clinical difficulties which we meet in attempts to elucidate the mysteries surrounding this disease arise from the insidiousness of its onset, the extreme chronicity of its course, and the uncertain character of its causes. Locomotor ataxia is among the most chronic of degenerative nervous diseases. It may exist, without seriously impairing the general health of the patient, for ten, twenty, thirty, or even a greater number of years after its existence has become recognized. How long it may last before any symptoms whatsoever are apparent is not definitely known, but certainly for a con-

siderable length of time. A patient of mine, fifty-two years of age, who had syphilis thirty years ago, has been in the preataxic state for five years. Both knee jerks are absent, there is loss of sexual power, the right pupil is larger than the left and presents the Argyll-Robertson phenomenon, and there is a diminution of sensibility in the ulnar distribution of both hands. These signs are sufficient for a diagnosis of tabes, although, aside from a slight swaying of the body on standing with closed eyes, there are absolutely no disturbances of motion, and there have never been characteristic pains. The patient came to me originally for treatment of alcoholism, and the tabetic symptoms were discovered during the course of a routine examination.

The pain is usually the cause of medical advice being sought, although there are other frequent initial symptoms observed by the patient, such as slight ataxia, difficulty in urination, and disturbances of vision.

The condition of the bones and joints which predisposes them to disease or fracture is sometimes the means of first calling attention to the existence of the spinal degeneration.

Some time ago a patient came to the Vanderbilt Clinic with extreme enlargement of the right knee joint, which he said was the result of a fall received a month previously. Although he did not know it, the man presented classical symptoms of tabes, which must have existed long before the accident; and although the particular lesion, which has proved to be a Charcot joint, may have been to a certain extent influenced by the injury, it occurred in an individual predisposed by tabes to its development.

The photograph in Fig. 72 is of a Charcot joint in a patient who presents nearly all the symptoms of advanced tabes. He says he was perfectly well until he fell on the right knee, as a result of which it became enlarged, a condition which was followed by other characteristic symptoms. As the accident occurred years before the patient came under observation, it is impossible to prove the incorrectness of his belief.

Even in the clinically early stages, during which the patient is usually conscious of one symptom only, or of no symptoms at all, a careful examination will show that there are already present objective evidences of structural disease which must have been present for a considerable length of time, and which must, in all probability, have antedated any subjective discomfort. In default of such objective evidences, the diag-

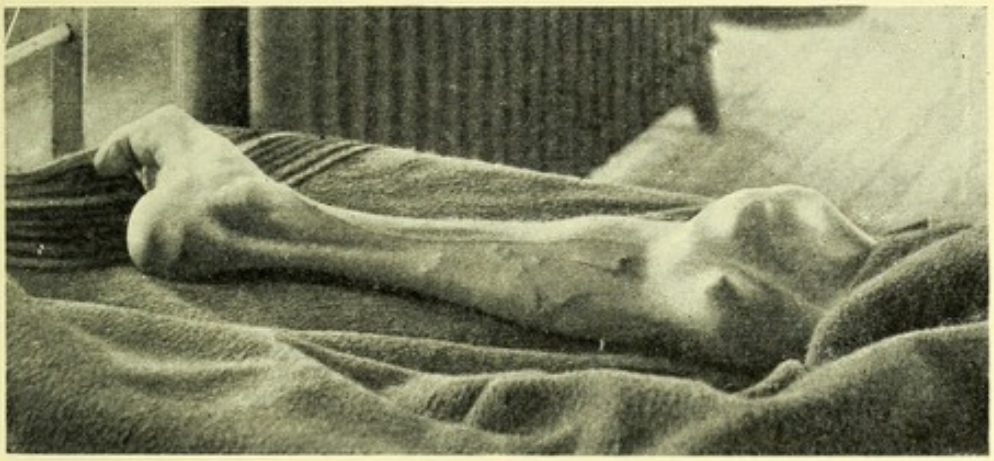


FIG. 72.—Charcot joint, the alleged result of trauma.

nosis of tabes cannot with certainty be made. The most common of diagnostic signs are the loss of one or both knee jerks, the Argyll-Robertson pupil, and the Romberg symptom. One or all of these three symptoms—the cardinal symptoms—are very constant, and usually appear early, but none of them would be noticed by the patient, and would ordinarily be overlooked in any medical examination in which a determination of the condition of the nervous system were not a primary object. Similarly with tactile anæsthesia of the trunk; the patient is, in early stages, unconscious of it until it is demonstrated by the physician. It is often one of the earliest of tabetic manifestations.

That the early symptoms of tabes are often overlooked is a fact of common experience. It occurs not infrequently

that persons suffering from incipient tabes, when the only subjective symptoms are pain or perhaps slight unsteadiness of gait, are seen by physicians who are unfamiliar with the diagnosis of nervous diseases, and who treat such cases without knowing upon what fundamental conditions the symptoms depend. No examination of the knee jerks or of the pupils is made, and the condition is regarded as sciatica or "spinal congestion." There can be no doubt that in many cases the cardinal symptoms exist for months or years without the patient's knowledge, and the physician who eventually finds any one of them finds not a recent symptom, but one whose character has permitted it to exist a long time unsuspected; or the patient may, while in the preataxic stage, have consulted a physician for other troubles, but unless at that time he complained of subjective nerv-

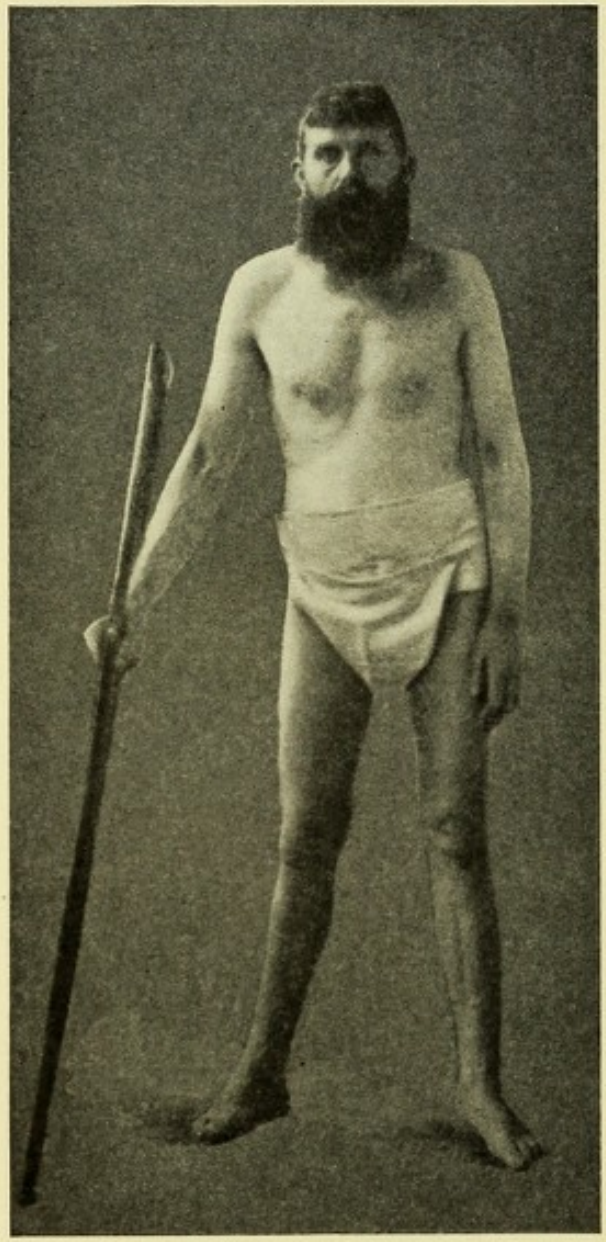


FIG. 73.—Attitude of static ataxia.

ous symptoms as well, there would probably have been no examination of the pupillary or patellar reflexes. Without such examination it would be impossible to assert that the patient was free from spinal disease. Similarly, the fact that the patient had been a successful candidate for life insurance would not be an absolute guarantee that he was at that time free from beginning posterior spinal sclerosis, because in

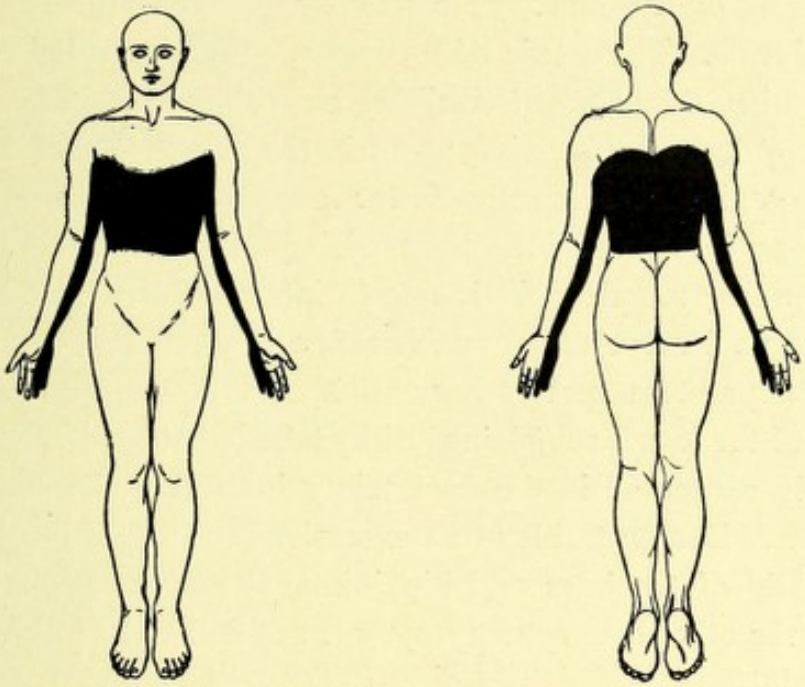


FIG. 74.—Tactile anæsthesia in a patient in the early stage of locomotor ataxia, whose only other symptoms are slight unsteadiness of gait and loss of knee jerk.

the routine examination for life insurance there is rarely any attempt at examining for nervous symptoms which are not immediately apparent.

That an examination by a competent physician, who is also on the alert for nervous disease, is necessary before anyone can be pronounced free from tabes, may seem equivalent to saying that all men are ataxic until they are proved not to be so. Extravagant as it may appear, that such a demand is essential in any attempts to fix the beginning of the disease will

be thoroughly indorsed by anyone who has studied the literature of the cases which are said to arise from injury, or who has had under his care cases of tabes which have remained for years in the preataxic stage, with few or no subjective symptoms, but in which symptoms have suddenly become active and severe as a result of some slight injury or shock.

Apart from the question as to whether it can or cannot be caused by injury, tabes has certain important features relative to accidents. The ataxia of the legs predisposes to falls, especially when the patient is walking on a slippery surface, or is stepping down, or is going about in the dark. The condition of hypotonia is also an active factor in causing mishaps. Hypotonia is a looseness of the joint arising from relaxation of the surrounding muscles. It is due to interference with the posterior spinal nerve roots, and is reflex in mechanism. It is a serious symptom, and is responsible for the sudden "giving away of the legs" complained of by tabetic patients. It is also actively concerned in the uncertainty in station characteristic of tabes. Demonstrable as an excessive mobility of the joints, especially of the knee and hip joints, it may be pronounced while the other locomotor symptoms are still well in the background.

In addition to these features of tabes, which predispose to accidents, must be considered the effects of accidents on the disease itself. The fragility of the bones in tabes makes them easy of fracture, and fractures in tabetics are common. The disease also is frequently made very much worse by physical violence and mental distress of all kinds. Preëxisting symptoms are intensified, and others, which had not been complained of before, are brought to light by such factors. The symptoms most commonly aggravated or made to appear by such circumstances are ataxia and pain. In the case of a patient who before an accident was in the preataxic stage of the disease,

although ignorant of it, having had no subjective symptoms, and who soon after an accident begins to have pain or to become unsteady in gait, the inference that the accident was the cause of the entire disease would be, from his point of view, only natural. Errors of interpretation of this character are doubtless frequent. The rule that tabes is made worse by accidents is, however, not absolute. One patient, in the preataxic stage, after a severe railway accident, was so badly bruised and contused that he kept his bed for a month, but there was no accession of tabetic symptoms.

Ætiology.—It is evident that there must be considerable uncertainty concerning the ætiology of a disease which may be in operation for months or years before its existence is known. This uncertainty is not materially relieved by what we know about the causal agents of tabes. Of the various factors which have been named as responsible for the spinal degeneration, syphilis is least open to destructive criticism. But even to the influence of syphilis is attributed a different importance by various observers. Erb finds preceding syphilis in over ninety per cent of his cases; but of 108 cases observed in Leyden's clinic, Storbeck finds but 23 which are surely syphilitic, 22 which are doubtful, and 63 which are " surely not syphilitic," though how he may be sure of that is not apparent. However, all writers agree that syphilis, either as a direct or as a predisposing cause, occupies a prominent place among the ætiological factors of tabes, although it cannot be said that tabes never develops unless the patient has had syphilis. Such a possibility is, indeed, generally admitted, and Gowers says that in ten per cent of the cases syphilis may be excluded with confidence.

Although there can be no doubt as to a relationship between syphilis and tabes, it is very difficult to determine whether the venereal disease acts as an exciting or as a predisposing cause of the spinal degeneration. In favor of the theory that

it is an exciting cause is the occasionally observed fact that tabes follows immediately upon syphilis without any other causes being discoverable. The evidence for it being a predisposing cause is, in the majority of cases, much stronger. Tabes does not usually appear until all distinctive syphilitic manifestations have ceased. When syphilitic and tabetic symptoms occur together, specific treatment usually causes the former to subside, but has no effect on the latter. The number of persons affected with syphilis is very large compared with the number of persons who develop tabes. The anatomical character of tabes is entirely different from that of recognizable syphilis. In the cases which are presumably free from syphilitic infection, it is usually impossible satisfactorily to prove any particular predisposition to the spinal disease, or to any diseases of the nervous system. Tabes itself may be said to be never directly transmitted. Neither is the pathogenesis of this affection explained by the other ætiological factors. The disease occurs chiefly in the male sex (ten to one), and most commonly appears in the middle decades of life (thirty to fifty).

Of the various exciting causes which are commonly mentioned in discussions on the ætiology, trauma is the only one of which we need speak, except to say of the others that their causal relationship to the spinal disease is rarely clear. If trauma is a cause of tabes, what would be its most probable mode of action? Our ignorance concerning the factor of predisposition renders any satisfactory answer to this question impossible. In the surely syphilitic cases, if the syphilis were considered an exciting cause of tabes, the action of trauma could only be regarded as an injurious influence acting upon a disease which was already inevitable. But if the syphilis had acted merely as a predisposing cause, it would be entirely permissible to maintain that the degeneration would not appear unless some

exciting factor were added. Our ignorance in regard to the nature of the syphilitic factor, and our total inability to explain the pathogenesis of the cases in which syphilis probably never existed, render it impossible to tell the part that trauma might play in the evolution of tabetic symptoms which had been absent before the injury. With a total absence of clinical evidence adequate to establish the existence of tabes as a direct result of injury, speculations as to the possibility of such a condition are more or less futile. If it is permissible to reason from analogy and from the study of those cases published as traumatic, in which, although they are incomplete as evidence, the influence of trauma in the development of the disease cannot be denied, it would seem that, if tabes is to result from injuries at all, it will be found to be from injuries to the peripheral nerves. It has been shown by Soukanoff and others that posterior spinal degeneration not infrequently develops from disorders which originally were clinically typical of multiple nerve inflammation. Now most of the cases of tabes reported as traumatic, which, although they do not prove a traumatic origin for the disease, might have been traumatic, have developed after gunshot or other wounds of the limbs, which may have involved the nerves. It may be possible that injury to a peripheral nerve can cause a neuritis which ascends to the posterior spinal ganglion, inducing disturbances there that are to become the starting points of degeneration in the spinal cord. Such a view of pathogenesis is, however, purely speculative.

If trauma stands in any causal relation whatsoever to tabes, such a connection must be very infrequent. Of 281 cases of Erb, trauma is mentioned as a sole cause in only one. Now tabes has received more careful attention than almost any other disease of the nervous system, and several monographs appear every year on its ætiology alone. Yet among the thousands of

cases which have been reported in detail, there are only a few dozen which are alleged to have been solely due to injury. Of the 185 full clinical histories of the disease recorded in the books at the Vanderbilt Clinic, up to the year 1897, in 3 cases only is there mention of an injury which might be regarded as causal: of these, one, with the Charcot joint, has already been mentioned; in another, there was an interval of *twenty-nine years* between the occurrence of the accident and the first appearance of spinal symptoms. The third is unique, and since it presents such evidences as would be accepted as final by any one unfamiliar with the insidious character of the onset of the disease, it must be recorded in detail:

The patient, a railway employee, thirty-nine years of age, fell off a freight car in October, 1896, striking on the buttocks and sustaining a fracture of the pelvis and some injury to the left leg. For one month he was in bed in the hospital, bolstered up with sand bags, with an extension splint on the left leg. Then he got up, and in trying to walk around the bed he fell to the floor and sustained another fracture. He was in bed again for four weeks, and when he got up this time found he could not use his legs properly, a condition which has remained about stationary. He came to the Vanderbilt Clinic in July, 1897, presenting the ordinary symptoms of locomotor ataxia —viz., ataxia and loss of muscular sense in the legs, without motor paralysis, Romberg symptom, loss of knee jerks, difficulty in passing water, and numbness in the legs (no objective anæsthesia). The left pupil was larger than the right and responded slightly during efforts at accommodation, but not to light. The right pupil was immobile. Vision: R. E., $\frac{20}{70}$; L. E., $\frac{20}{50}$ (for field of vision see Fig. 75). The ophthalmoscope showed pronounced optic-nerve atrophy in both eyes, most marked on the right side.

When questioned as to possible causes, the patient made the following replies:

Syphilis.—He denied emphatically every symptom of any venereal disease. He was married at the age of twenty-four, and his wife gave birth to two healthy children. One miscarriage came at the eighth month, but it followed immediately upon a fall, and consequently cannot be regarded as an evidence of syphilis.

Condition Previous to the Injury.—The patient could not be made

to admit the existence of any tabetic symptom previous to the injury. He was positive that he had never had any difficulty in walking, either on the street, on the stairs, or in the dark; he did not fall; he never had shooting pains. He said that for several years before the accident his eyesight had not been so good as formerly, but he could read the paper without difficulty, and that three or four months before the accident he successfully passed the examination of the eyes for color perception which was demanded by the railway of

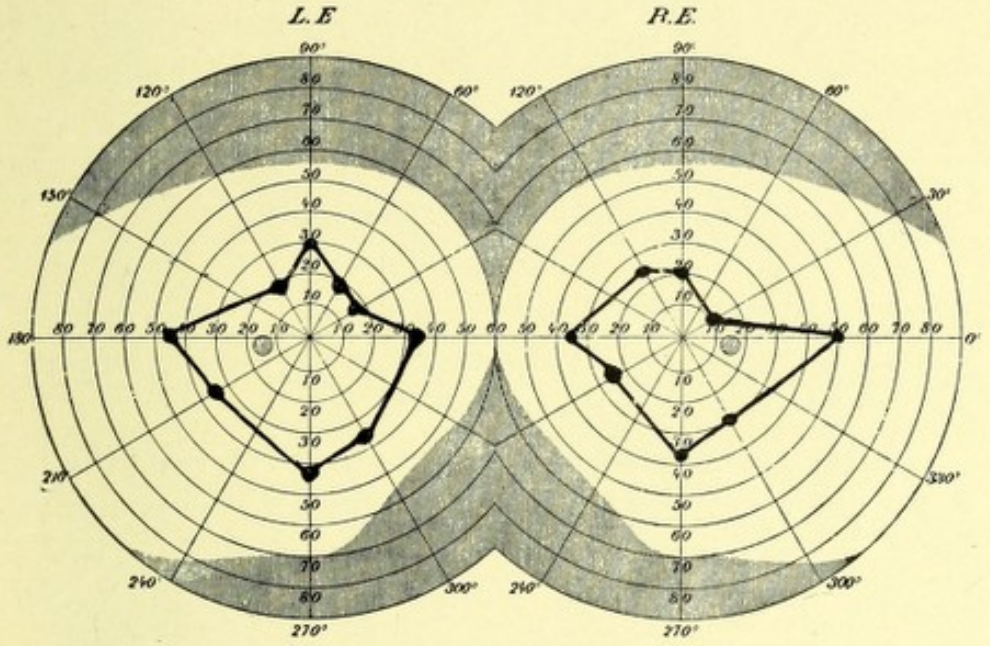


FIG. 75.—Field of vision in a case of locomotor ataxia which developed immediately after an injury.

which he was an employee. He stated that many of the employees were rejected at this examination.

After the Accident.—The first symptom he observed was failure of sight. In a week or ten days after he had been in the hospital his vision became so poor that he could no longer read the paper, and since then he has been unable to read any but very large type. The other symptoms of tabes were only observed when he left the bed, and are essentially those which have been enumerated. The patient had never thought of bringing a claim against the company, and his whole manner spoke against his having any desire or motive for telling anything but the truth.

Thus an apparently healthy man, not predisposed to nervous disease, receives an injury and rapidly develops symptoms of locomotor

ataxia. The accident might be regarded as the sole cause, were it not that, on account of the latent character of the disease, no injury can be said to be causal, unless, previously to its receipt, tabetic symptoms were shown to be absent.

In spite of the absence of adequate and reasonable proof, in most text-books on nervous diseases trauma receives an important place among the exciting causes of the disease. For purposes of scientific interest, and also because in negligence cases injury may be alleged to be the cause of the disease by persons who, though honest, are ignorant of symptomatology, or by persons with fraudulent intent, it is very essential that the merits of the question be clearly understood.

The subject has received careful study by Prince, who criticises all previously reported cases with a view of determining if in any case the disease could have been reasonably presumed to be of traumatic origin.

He analyzed the 40 cases of tabes which had been published as traumatic, and rejected them all as being insufficient as evidence, although he admits that 12 of them might have been of traumatic origin. But not a single case fulfills satisfactorily the conditions which must be complied with if we are to insist on anything like reasonable proof.

The most important of these conditions is to show that tabes had not preëxisted. If this cannot be done, the insidious character of the disease would place the weight of evidence with him who claimed that it had antedated the accident. As has already been indicated, such proof is usually lacking, yet without it any claim of tabes caused by injury lacks basis. Prince expresses the opinion, which is indorsed by Bernhardt, that tabes cannot be regarded as traumatic if the patient has had syphilis. Inasmuch as we are ignorant of how syphilis acts in the ætiology of the spinal disease, and in the absence of proof that injury can cause tabes in syphilitic persons, this

view appears unwarrantable. In regard to the severity and character of the injury we are equally in the dark. It is generally unprofitable to speculate as to how the human body will react to injurious influences if we have no clinical data upon which to rely.

From general principles it would seem reasonable to expect that an injury to cause so general a disease as tabes would have to be physical and reasonably severe. Psychic shock seems hardly sufficient to induce nerve degeneration.

To be serviceable as evidence, the interval between the receipt of the trauma and the first appearance of symptoms would have to be no longer than a few weeks or months. If tabes results from nerve injuries, the interval should be occupied by symptoms of neuritis.

Since the writing of Prince's paper a few other cases have been reported. In one, by Bernhardt, tabes had existed before the injury, but had been made worse by it; in another, by Hitzig, there was no examination until a year and a half after the accident; and in one, by Craig, there seems to have been some doubt as to the correctness of the diagnosis of tabes.

Lammers has reported a case which developed under observation:

> A man, forty-nine years of age, not syphilitic, previously vigorous, slipped, while carrying a heavy log, and lacerated the muscles of the left thigh. In four weeks he recovered sufficiently to return to work. Then pain in the muscles that had been injured set in, and became so severe that he had to stop working altogether. In less than a year from the accident the Argyll-Robertson pupil and the Romberg symptom were demonstrated. The knee jerks at this time were weak and soon became abolished, and ataxia was added. One year later tabes was fully developed. Lammers suggests the possibility of this case originating through a peripheral nerve injury.

Such conclusions as we are justified in drawing in regard to this matter are general. There is no really satisfactory proof

that tabes has ever been the sole result of trauma. On the other hand, there are a few well-recorded examples of the disease in which, while the evidence is insufficient for positive proof, such a relationship cannot be denied. It seems probable that in most of the so-called traumatic cases the patients were in the pre-ataxic period at the time of the injury. As has been pointed out, tabetic patients bear injuries ill, and in many cases, after injuries, there is a sudden accession of painful and disabling symptoms, so that the disease becomes well marked. This fact must be given due weight in any medico-legal consideration of the disease.

Progressive Muscular Atrophy (Creeping Palsy)

Including Amyotrophic Lateral Sclerosis.

The researches of recent years have very much enlarged the field occupied by the disorders in which wasting of the muscles occurs, so that to-day the term progressive muscular atrophy might be construed to include atrophies which are progressive, although different in course and pathology from the original disorder described by Duchenne. There are at present two chief groups of idiopathic muscular atrophies, one of which is spinal in origin, and the other of which commences in and remains localized to the muscles without any involvement of the spinal cord. The latter variety, more commonly called progressive muscular dystrophy, occurs in families, is a disease of childhood and youth, has a different muscular localization from the spinal type, and, as it is rarely, if ever, suspected of being of traumatic origin, requires no mention here.

A less frequent type of progressive muscular atrophy is called, after its earliest describers, the Tooth or Charcot-Marie type. Its pathology is obscure, though the weight of evidence

favors its being due to a chronic neuritis rather than to spinal-cord disease.

The most prominent anatomical feature of progressive muscular atrophy (spinal) is a degeneration in all parts of the peripheral motor neuron, with a resulting wasting of the muscles. Associated with the degeneration of the peripheral neural element there almost constantly occur degenerative changes in the lower portion of the central neuron, which descends from the cortex in the motor tract, and which is continued through the spinal cord in the pyramidal tracts.

According as the morbid changes are most pronounced in one or the other of these two neurons, the clinical type of the disease varies (compare page 71). Thus, if the primary (or peripheral) neuron is chiefly affected, the atrophy and weakness will be extreme and reflex irritability will be lost, in accordance with the type of pure atrophic paralysis. On the other hand, if it is the pyramidal tracts which are the more involved, the character of the paralysis will be spastic, with a certain rigidity of the muscles and an increase of the tendon reflexes.

To the spastic form of progressive muscular atrophy has been given the name of amyotrophic lateral sclerosis. It is a convenient clinical term, but, in spite of a recent monograph by J. B. Charcot in defense of its autonomy, I have been unable to convince myself that any differentiation of the two conditions is justifiable on anatomico-pathological grounds. In by far the larger number of cases there are morbid changes both in the anterior horns and in the pyramidal tracts, and the clinical differences which are observed, according as one or the other neuron is the more affected, seem to be variations from a common condition rather than expressions of separate morbid entities. Accordingly, in speaking of the traumatic origin of progressive muscular atrophy, the term will be used so as

to include the clinical type known as amyotrophic lateral sclerosis.

Symptoms.—The symptoms of progressive muscular atrophy consist of atrophy, weakness, and fibrillation of the affected muscles, with, in the early stages, a slight alteration of electrical excitability only. Objective sensory symptoms are never prominent, but there may be pain, especially if the process is rapid. Pain has usually been mentioned when the disorder was sequential to trauma. The bladder and the rectum are seldom involved in this disease, and there is rarely if ever complete loss of sphincter control.

The symptoms vary with the locality of the lesion, and several types have been described which depend for their individuality upon the situation of the muscles that are first attacked. The Duchenne-Aran type is by far the most frequent. In it the locations in which the disease first appears and the order in which it progresses are fairly constant. Beginning on one side in the muscles at the base of the thumb, and in the interossei, it skips up to the shoulder to affect the deltoid and perhaps others of the shoulder muscles. The muscles of the arm and forearm may next be involved, or the disease may first pursue in the upper extremity of the opposite side the same course that it followed in the extremity first attacked. Very frequently the deltoid is the earliest to wither, the small muscles of the hand joining in the atrophy at a later period. In more advanced stages the process may move upward, giving the picture of glosso-labio-pharyngeal paralysis, or it may descend to involve the legs.

Course.—The course of progressive muscular atrophy is chronic, although very much more rapid than that of locomotor ataxia. In many cases the progress becomes slower as the disease advances, and in some it seems to stop altogether. When complicated by bulbar palsy, death soon ensues from fail-

ure of respiration, or from "foreign-body" pneumonia. If the atrophy remains limited to the hand and shoulder muscles, the patient may live, incapacitated, for many years.

Ætiology.—Even less is known about the causation of progressive muscular atrophy than about that of tabes. Syphilis is spoken of as a cause, but its influence is not proved, and is certainly very much less important than in tabes. Exposure to cold and wet, and overexertion, have sometimes preceded the first appearance of the disease. From the fact, as first shown by Ballet and Dutil, and later by Hirsch, that the chronic disease may first appear in muscles which had been affected by acute infantile spinal palsy, it seems as though preceding inflammatory conditions of the spinal cord might be a predisposing cause.

The causal relationship of trauma to progressive muscular atrophy is fairly well established. A considerable number of cases are on record in which injury of a limb was quickly followed by an atrophy of its muscles, a condition which later became general.

Less frequently blows on the back or concussion accidents have been the only discoverable cause of the typical symptoms which shortly after ensued.

Diagnosis.—To prove progressive muscular atrophy to be in all probability the result of injury, it must be shown: 1. That the patient has muscular atrophy. 2. That he was free from the disease a short time before or immediately after the accident. 3. That the accident caused a considerable shaking of the body or injury to a limb.

1. The first requirement is a question of diagnosis, about which there is usually little difficulty. If the disease is of the lower Duchenne-Aran type, the two conditions to be eliminated are injury to the ulnar nerve and lead palsy. It will be remembered that the ulnar nerve supplies the interosseous muscles,

and that injury to it causes *main en griffe*, which is also a frequent symptom in progressive muscular atrophy. If the history told by the patient is reliable, ulnar-nerve palsy and progressive muscular atrophy will rarely be confused. In the peripheral lesion the atrophy develops very much more rapidly, the loss of muscular power is greater, and the subjective sensory sensations are more prominent. The diagnosis may, however, usually be made by objective signs alone. In any palsy of a peripheral nerve which is sufficiently severe to cause pronounced atrophy, the electrical conductivity of the nerve is very much diminished or lost. Unless nerve injuries are very severe fibrillary twitchings are not marked, and in an injury to the ulnar nerve there is never any involvement of the deltoid.

The differentiation from lead palsy may be more difficult. If there are no constitutional evidences of plumbism, it is sometimes an exceedingly delicate matter to decide between lead palsy and a progressive muscular atrophy in which the extensors of the wrist and fingers have become involved on both sides. In lead palsy the supinator longus and the extensor ossis metacarpi pollicis escape. But they may also be the only muscles to escape in the spinal disease. In a recent case at the Vanderbilt Clinic, in which the wasting and paralysis were bilateral and which by its course has been proved to be one of progressive muscular atrophy, these two muscles were the only ones of the extensor group which had not been involved by the paralysis. Such cases, however, are unusual; most frequently progressive muscular atrophy is at first unilateral, or very much more marked on one side than on the other. If it has developed in both hands, there are also characteristic atrophies about the shoulder muscles, the extensors of the wrist not being affected until a later period of the disease.

If traumatic progressive muscular atrophy begins in the

deltoid or other of the shoulder muscles, it may, if the history be unreliable, be confused with some of the palsies of the brachial plexus. The distribution of the palsy and the results of electrical examination will usually permit a recognition of the two conditions. Increase of the tendon reflexes, both at the knee and in the upper extremity, frequently occurs in progressive muscular atrophy, but is never a part of the peripheral disorder with which it might be confused. Its existence is therefore a valuable aid in diagnosis.

2. The validity of a claim of progressive muscular atrophy caused by injury depends very largely upon the ability of the claimant to prove himself to have been free from the disease at the time of the accident. Such proof is more easily attainable than it is for the tabetic to prove that at any given time he had no symptoms of tabes; for the early symptoms of progressive muscular atrophy are essentially objective, and are of a character to be noticed not only by the patient but by his friends. A marked sinking in between the first finger and thumb may occur in a month's time, and although the atrophy is usually more rapid in its development than the loss of power, even a weakness of the interossei is so annoying and disabling, and causes so serious an impairment of those finer movements of the fingers which are essential for the commonest daily duties, that it could not exist without becoming the subject of remark. If a person who develops, soon after an accident, progressive muscular atrophy, type Duchenne-Aran, could prove that he had continued to do manual work satisfactorily up to the time of the injury, it would be very good evidence that he was then at least free from the disease. In most of the reported traumatic cases the patients were active workers up to the time of injury.

3. The accident which has most frequently induced the disease has usually been severe. I know of no case in which fright

alone is said to have been a cause, and in most the injury was very appreciable.

Illustrative Cases.—The kind of accident and the mode of the first appearance of symptoms may be illustrated by the following cases; some have been seen at the Vanderbilt Clinic, and others are derived from various sources. For two reasons no effort has been made to include all the cases published as being due to trauma: First, because the clinical evidence in favor of a traumatic origin of the disease is too well established to make such a review necessary; and, secondly, because many of the cases have been described in a way that renders it impossible for the reader to determine whether the disease had been caused by injury, or whether, indeed, it were unquestionably progressive muscular atrophy. The following cases, therefore, have been chosen, inasmuch as they seem to prove with a considerable degree of probability that progressive muscular atrophy may result from injury, and because they illustrate the clinical courses of such cases:

Raymond quotes from Friedreich the case of a man who, after a bruise of the right hand, developed an atrophy in that hand which followed a progressive and ascending course and was eventually complicated by bulbar palsy. He also refers to Poncet's report of a soldier who was shot in the right shoulder. The muscles of the wounded shoulder soon became atrophied, and the left shoulder became involved later.

Clarke describes a case which, in addition to furnishing a good example of traumatic progressive muscular atrophy, illustrates the predisposing influences of certain occupations, and shows the injurious effect of depressing mental influences upon the course of the spinal disease:

A woman, thirty-eight years of age, married, seamstress, of delicate constitution, fell downstairs and hurt her right hand, and especially the thumb. One year later there was pain in the right arm,

neck, and shoulder. She was unable to hold her needle, yet there was no pain in the hand or forearm. At this time the muscles of the arm and forearm began to waste, but improved again in two months. The sudden death of her husband caused her a severe mental shock, and she then complained of pain in the neck, and that both arms and both legs felt weak, cold, and sometimes numb. Loss of power of the neck muscles ensued, and the right arm soon became almost completely useless.

When admitted to the hospital the right arm was paralyzed and wasted, and the examiner "could feel only fat, skin, and bones." No reaction to faradism. No stiffness.

Examination.—Left arm: Little power in the hand. The patient can extend the fingers slowly and feebly. All the thumb muscles are wasted. The muscles of the lower two-thirds of the forearm are wasted, as are the supinators. She can wriggle the arm along the chest. There is a fair quantity of muscle on the upper arm. Interossei react to a slight current; extensors feebly.

Neck: Muscles can bend the head only a little. There is slight power in the left trapezium, but none in the right. Muscles on the scapulæ are gone. The facial muscles act slightly. Palate moves slightly. Tongue protrudes, but is atrophied on both sides. Deglutition imperfect. The right leg is wasted, but retains slight power of movement. There is considerable strength in the left leg. Patient died four months after admission to the hospital, and about two (?) years after the receipt of the injury.

A case of the late Professor Eisenlohr's is interesting, as it shows the early appearance of the symptoms of bulbar palsy after an injury to the upper part of the spinal column and follows the clinical type of amyotrophic lateral sclerosis in the extremities:

A locksmith, forty-nine years of age, asserts that he had been well previously to the receipt of a blow on the neck in the spring of 1877. In July of the same year there appeared a gradual weakness of the arms and hands, which extended two months later to the legs. In October began disturbances of articulation. Examination in January, 1878, showed the following conditions:

The lower part of the face is flaccid, the lips are constantly open, and blowing and whistling are impossible. The tongue is atrophic and its movements are limited. The articulation of all consonants is

bad. Movements of the soft palate are very slow. Swallowing and chewing normal. The shoulder and upper arm muscles are moderately atrophied on both sides. The muscles of the forearm are markedly atrophied on both extensor and flexor surfaces, the interosseous spaces of both hands are deeply sunken in, and the thenar and hypothenar eminences are flattened. The hands and fingers are almost immobile. Great loss of power in both arms. Sensibility is normal. The reflexes are active. Electrical excitability is diminished. Similar changes, though less marked, are observed in the lower extremities. Death occurred in a few months as the result of the bulbar palsy. The autopsy and subsequent microscopical examination showed a degeneration of the anterior horns of the spinal cord and of both pyramidal tracts.

Progressive muscular atrophy sometimes results from head injury, or at least from accidents in which the head was injured. In such cases there has usually been considerable general violence, and it is impossible to tell how much of it has been exerted on the spine.

Mann reports the case of an Englishman who at the age of twenty-one fell from the rigging to the deck of a ship. He was rendered unconscious, but sustained no fractures of the bones. Previously to this accident the patient had been perfectly well. Denied syphilis or nervous disease in his ancestors. One month after the fall the deltoid began to waste, then the biceps and the lower-arm muscles, and finally the muscles of the legs. One year later he was seen by Sir William Gull, who thought the patient could not live twelve months. The disease came to a standstill, however, for seventeen years after the accident the patient was treated by Dr. Mann for another condition.

In a case of Bullard's, a teamster, sixty-one years of age, a staging fell and struck the side of his head.

The patient had been in previous good health; there were no syphilis or rheumatism or other causes of constitutional disease discoverable, although signs of phthisis appeared while the patient was under observation. There was no loss of consciousness at the time of the accident, but soon after it the patient felt pains in the head, neck, and left shoulder, which were followed by weakness and

atrophy of the muscles of the left arm and hand. Seven months after the injury he was first seen by Dr. Bullard. The muscles of the shoulder and of the back on the left side were markedly atrophied, those of the upper arm less so. The forearm was still less affected. *Main en griffe.* Thenar and hypothenar eminences flattened. In the interossei there was diminished faradic reaction. Fibrillary twitchings were marked. The patient gradually grew worse, and the left arm became affected. One year later the atrophy in the muscles of the upper part of the back and the fibrillary twitchings became very marked. Reaction to faradism was diminished, but sensation remained normal.

A case by Ziehen is less convincing:

Man, twenty-one years of age, healthy, received a severe blow in the chest and was then thrown into a ditch, where he lay unconscious for some time. This was in March, 1888. He suffered from fatigue off and on, which was increased, on one occasion especially, when he stood for some hours in the river fishing (in July, 1889). He soon became unable to do heavy work, and appeared at the clinic in 1890, showing a well-advanced case of progressive muscular atrophy.

Ziehen maintains that the atrophy was caused by the original injury, and that the standing in cold water only aggravated a process which had become established. The interval of time, however, which elapsed between the accident and the date of the medical examination was too long to permit an inference of anything more than a possible relation between the original trauma and the spinal disease.

The relationship between preceding inflammations and the development of progressive muscular atrophy is shown by the following case:

An electric lineman, thirty-five years of age, a steady drinker, who had had the initial lesion of syphilis in 1885, came to the Vanderbilt Clinic on October 30, 1896. He said that in 1888 he fell forty feet from a pole, landing on the back and buttocks. He then walked a few steps, but his legs soon gave out from under him and he was carried to bed, completely disabled in the lower extremities. For three months he was in bed, unable to move his legs. The urine had to be drawn with a catheter. He then recovered and went back to work, and continued at work until July, 1896. He told us that

for the past three months he had noticed a gradually increasing weakness in the legs and thighs, without pain, but with soreness across the back and stiffness at the ankle, hip, and knee joints; paræsthesia existed in the sole of the left foot and in the ulnar distribution of the fingers, with considerable weakness in the left arm, a condition which was also just commencing to be felt in the right hand.

Examination.—Gait is paretic and spastic; there is atrophy of the right calf muscles, less marked on the left side. Double ankle clonus and exaggerated knee jerks. Atrophy of the first interossei muscles of both hands and beginning *main en griffe*. Fibrillary twitchings on the inner sides of both arms.

The atrophy and loss of power, which had begun and remained most marked in the legs, had already involved the small muscles of the upper extremities. If one be permitted to speculate as to the probable genesis of the disorder, it would seem not improbable that the original injury (in 1888), coming so soon after the syphilitic infection, had caused hemorrhage, or an outbreak of localized syphilitic inflammation in the spinal canal in a way to exert pressure upon the spinal cord. From this condition the patient apparently recovered. There was left, however, a *locus minoris resistentiæ*, as was shown by the progressive muscular atrophy first appearing in the muscles which had previously been the seat of the temporary paralysis.

The following case, which was under our observation at the clinic for many months, developed progressive muscular atrophy three months after a blow on the back:

The patient (Figs. 76 and 77) says that, as far as he knows, there have never been any nervous diseases in his family, and that previous to his present illness he himself had always been strong and well. As a young man he had gonorrhœa, but denies all symptoms of syphilis. He was married at twenty-nine years of age, and has one healthy child. His wife never had any miscarriages. He is a moderate drinker. At the age of thirty-one, eight years ago, the patient weighed one hundred and seventy-five pounds, and was healthy and strong. As the proprietor of a rag shop he was daily engaged in handling heavy packages, an exercise which he could perform without unusual effort or fatigue. The man is intelligent and straightforward, has no object to deceive, and every statement he makes relative to his physical condition previous to the accident indicates that he was a healthy, active, muscular man.

In 1889—that is, about eight years ago—while lifting one end of a bale of cotton weighing 800 pounds, he slipped and fell so that one corner of the bale struck him in the small of the back. He suffered no pain, and was able to get up and return to work, and for three months observed no ill effects from the accident.

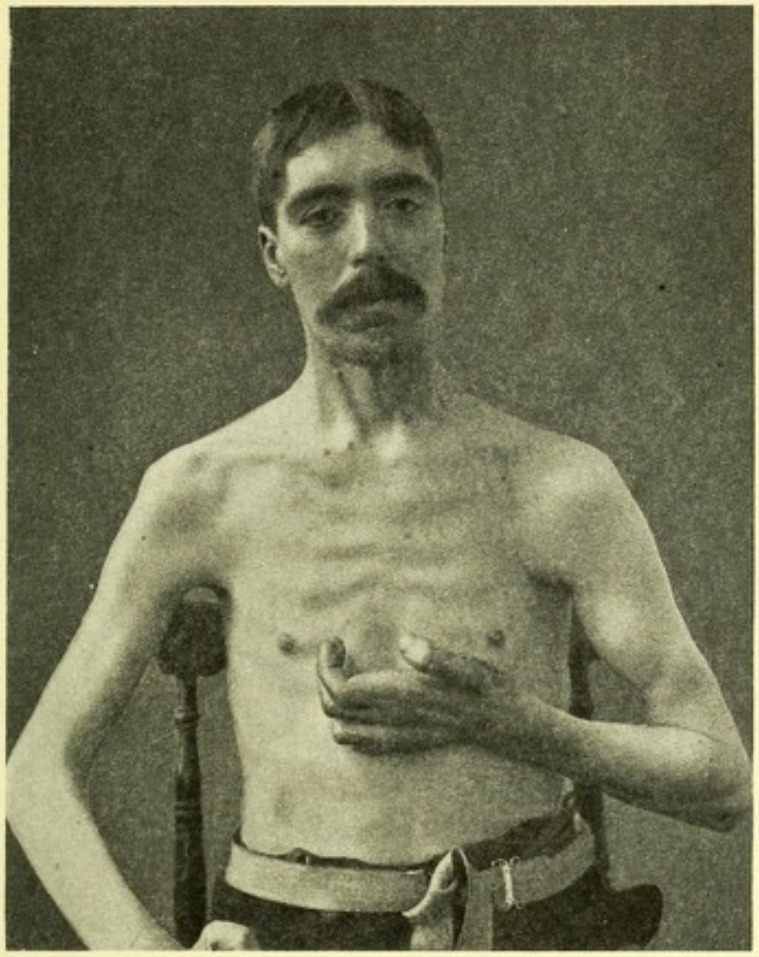
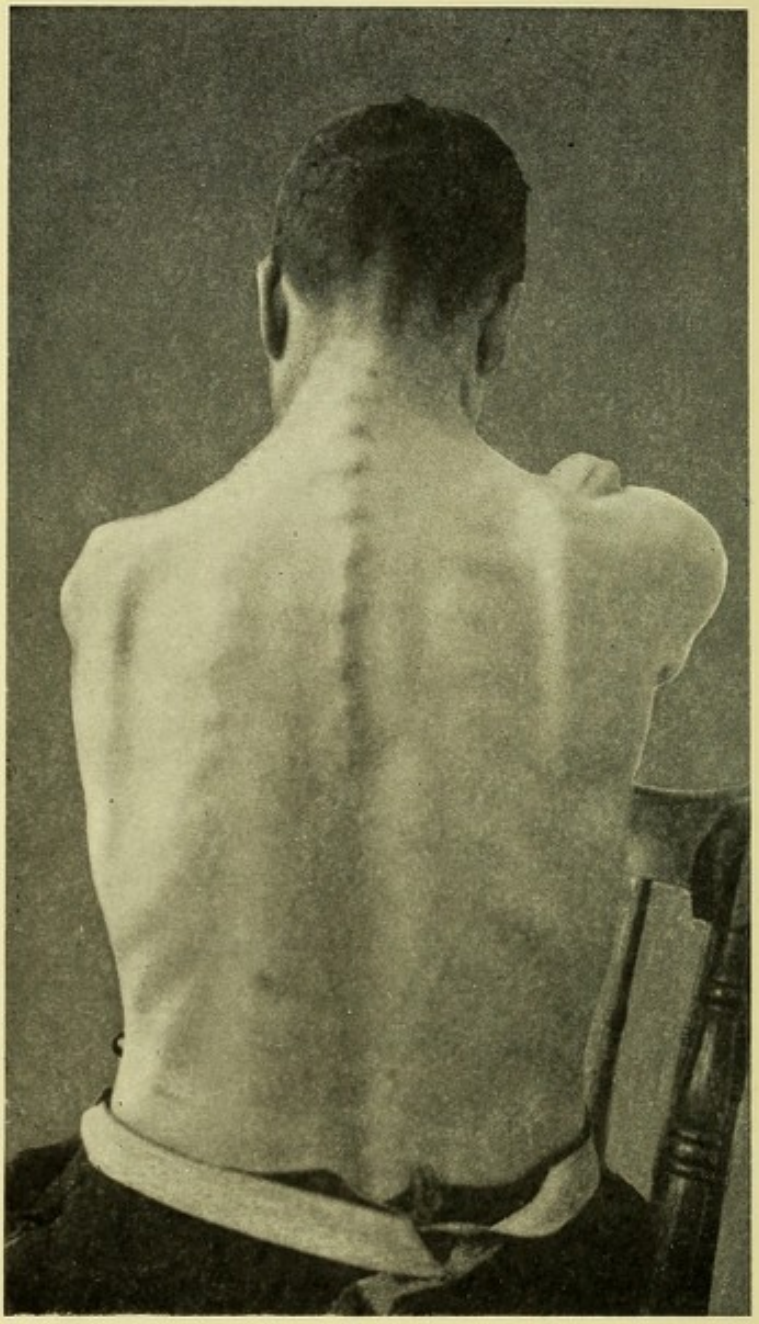


FIG. 76.—Case of progressive muscular atrophy which began three months after an injury to the back. The photograph shows the wasting of the shoulder and chest muscles and the *main en griffe*.

Then he began to have pain in the small of the back and in the muscles of the right arm, with some loss of power, especially for movements of extension of the wrist and of the fingers. The weakness of the right upper extremity soon became so marked that he had to give up working with it. One year after the accident the left hand and arm became similarly affected. At this time he also noticed the hands were becoming very much thinner between the thumbs

and fingers, and that there was a decided sinking in around the thumbs. The fingers became stiff, which was observed particularly



F.G. 77.—Same patient as in Fig. 76, showing wasting of the muscles of the back.

upon attempts to extend them. He soon became so incapacitated that he was obliged to give up all heavy work. With the exception

of slight pain, there were never any sensory disturbances, and the functions of the bladder and rectum remained normal.

The condition of wasting has been progressive, and to-day the man presents a picture of progressive muscular atrophy, type Duchenne-Aran, with beginning involvement of the leg. There is extensive atrophy of the thenar and hypothenar eminences and of the interosseous muscles. Both hands are in the attitude of *main en griffe*. The dorsal surface of the thumb is in the same plane as the back of the fingers, and its opposability is lost. There is also an atrophy of the flexors and extensors of the wrists, the deltoids, the pectoral muscles, and the muscles of the back, especially of the right side. There is also a beginning atrophy of the left anterior tibial muscles, so that the patient has the "stepping" or "equine" gait on that side. The atrophied muscles are the seat of fibrillary twitchings, which become very marked on exertion. All the muscles, with the exception of those which are wholly atrophied—as are some of the interossei—and of the ones which are held stiff by contractures, react readily to faradism. The knee jerks are present, and there are no sensory disturbances. The patient is practically deprived of all use of the upper extremities. The paralysis and deformities of the fingers and hands prevent any fine coördinated movements, and, on account of the loss of power in the shoulder muscles, he can with difficulty lift his hands to his head or hold his arms out from his body. As a consequence he can do no manual work, cannot dress himself, and can only with difficulty feed himself. His condition is slowly but steadily becoming worse, and has recently become complicated by tubercular disease of the right lung.

The only medico-legal question which might arise in regard to this case is whether the condition is solely due to the injury. There can be no doubt as to the correctness of the diagnosis; every symptom is present, and the patient presents a typical picture of the Duchenne-Aran type. The symptoms appeared a short time after injury in a man who had undoubtedly been strong and well before, and who was in no way predisposed to disease. The injury was sufficiently severe to cause considerable violence to the back, and it seems to me entirely reasonable to believe that had it not been received the spinal disease would not have developed.

Paralysis Agitans (Parkinson's Disease—Shaking Palsy)

Examination of the properly prepared brain and spinal cord of a person dead with paralysis agitans shows degenerations in nerve fibers and nerve cells and in connective tissue. But since the disease occurs almost altogether in persons who have reached the age when such degenerations often occur without causing the symptoms of any clinical entity, it still remains to be proved whether, in paralysis agitans, the anatomical alterations are causes or effects, or simply accompaniments of the malady. But, although the pathology remains unexplained, the fact that in it the central nervous system appears differently than in health, together with the fact that the clinical manifestations indicate the action of a common structural cause, are sufficient to permit paralysis agitans to be classified without impropriety among the organic nervous diseases.

Symptoms.—The symptoms which are the most constant, and which give the disease its clinical characters, are tremor, rigidity, and weakness. The tremor is rhythmical and comparatively coarse, there being between five and seven excursions in a second. It continues during rest, ceases during sleep, and although not at all or only slightly aggravated in intended movements, it is made very much worse by emotional influences. The tremor usually originates in the thumb and index finger of one hand, and from there it spreads to other fingers. Later it involves the wrist and the muscles of the whole upper extremity. After it has persisted for a longer or shorter time in any one part its usual course is that of extension. The mode of extension, which is fairly though not absolutely constant, is from the arm to the leg on the same side, and then to the arm and the leg on the opposite side. In the rare cases in which

the tremor begins in the leg, it may follow the hemiplegic type, or it may involve the opposite leg, before passing up to the arm.

The head and shoulder muscles are rarely involved; these

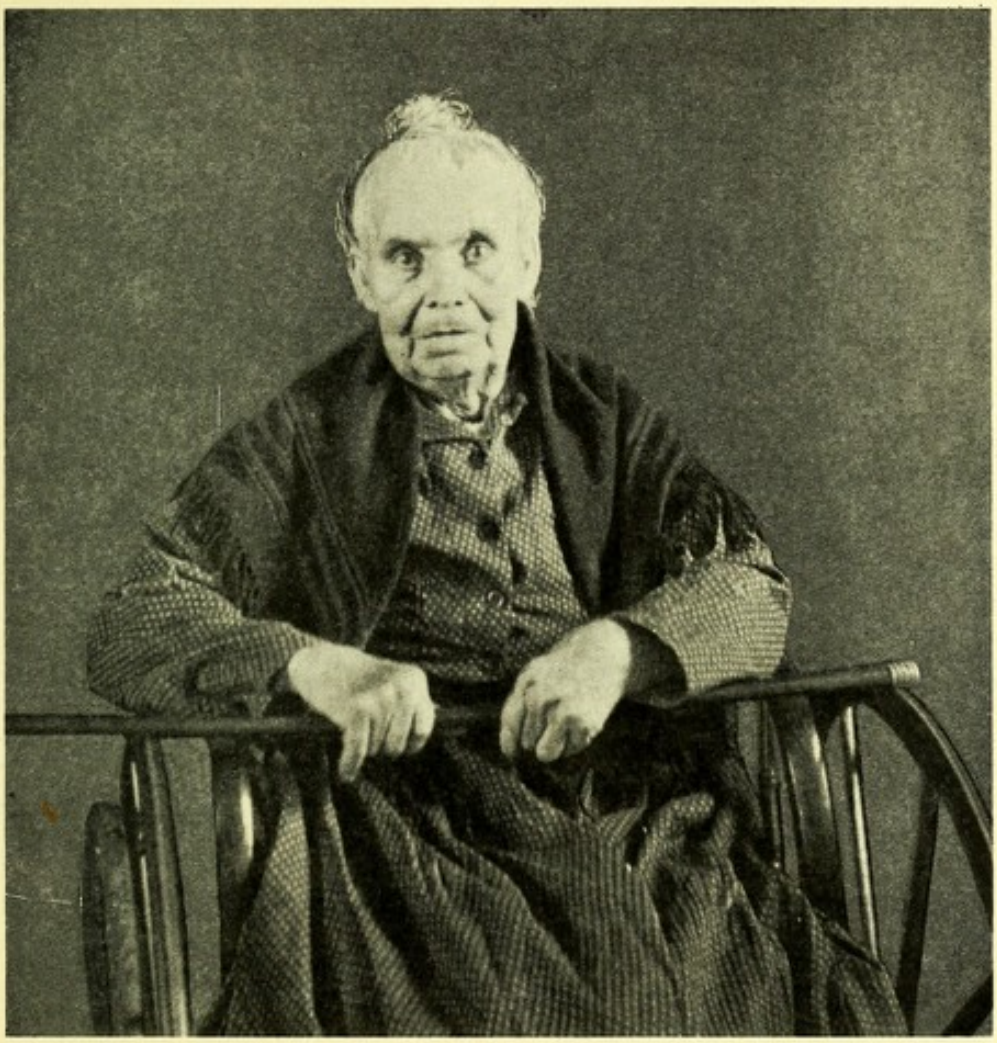


FIG. 78.—Showing facial expression and position of the hands in paralysis agitans.

parts are caused to oscillate by the transmitted movements from the limbs, but it is only in very exceptional cases that they are themselves the seat of tremor.

Muscular rigidity and weakness are symptoms of equal importance to the tremor, and occasionally cases are met with

in which they are present without the tremor, furnishing the seeming paradox of paralysis agitans without agitation.

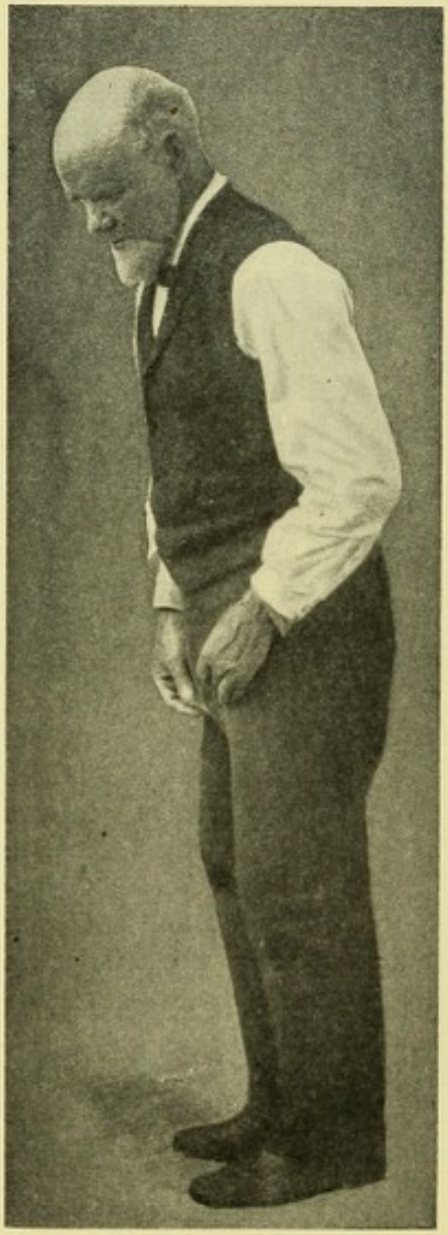


FIG. 79.—Showing characteristic attitude in paralysis agitans

In the larger number of cases, however, the rigidity and weakness develop in the same parts and at the same time as the tremor. The rigidity consists in a stiffness of the muscles without increase of the reflexes, and is consequently different from spasticity; it can usually be overcome without difficulty by the physician. The loss of power is never absolute, but all the movements of the affected muscles are slow and uncertain.

When the disease is well developed the muscular conditions give rise to characteristic appearances. The face (Fig. 78) is expressionless and drawn. The eyes are wide open, and there is very little play of the features in talking or in emotional expressions. The voice, from stiffness of the muscles of the larynx, changes its pitch but little and is monotonous. The head is bent forward (Fig. 79), and there is frequently a bending forward of the spine. The limbs are flexed at the large joints; the fingers are flexed at the first phalanges, and

extended at the second and third (interosseous position). The gait is shuffling, the patient walking with short steps, and having difficulty in starting, or, having once started, in stopping himself. Festination, or the tendency to constantly increase the rapidity of the gait, and retropulsion and propulsion, or the tendency to fall backward or forward, as the result of slight pushes, are common symptoms. There are no changes in reflex action, no atrophy or disturbances of sphincter action. Cutaneous sensibility remains normal, although the patients frequently complain of subjective disturbances of sensation. The disease follows a chronic and progressive course, and is incurable, although it may last for an indefinite number of years. The symptoms have been described in some detail, because the order of their development after injury is our chief proof of the traumatic ætiology of the disease.

Ætiology.—There are few disorders of the nervous system in which predisposition is so seldom demonstrable as in paralysis agitans. In a small proportion of the cases the disease itself, or epilepsy, or insanity, may be shown to have existed in relatives, but generally persons who become the subjects of Parkinson's disease have healthy ancestors, and have themselves been previously healthy. The disease occurs most commonly in the decades of life between forty and sixty, and is more frequent in men than in women (five to three). According to Gowers, exciting causes can be found in one-third of the cases, of which the only ones requiring description here are nervous shock and physical injury.

Gowers believes fright to be the essential feature in the genesis of this disease.

In support of this view he cites the case of a man who was waked by a bell on account of a fire; for a year and a half the same bell always caused transient tremor, which then became permanent, and passed into the typical form of paralysis agitans.

Also, the case of a woman who, at thirty-seven years of age, was sitting quietly at work, when a stream of water suddenly flowed from a tap onto her left wrist. She was much startled; the left arm immediately began to shake, and the tremor persisted, passing to the leg, and afterwards to the limbs on the left side.

That the disease often appears and becomes permanent soon after a severe shock, without any physical injury, there can be no doubt, but more frequently there has been physical injury as well. Of 139 cases observed at the Vanderbilt Clinic, fright is assigned as the sole cause in 7. On the other hand, of 26 traumatic cases examined by Walz, there is no mention of fright alone as a cause, when unaccompanied by physical injury.

The injury, which is usually moderately severe, may be of different kinds. Paralysis agitans has immediately developed after falls, injuries to nerves, stab wounds, contusions, fractures, etc. Walz's analysis showed, in 26 cases, general concussion, 6; wounds (stabs and cuts), 7; burning and freezing, 1; sprains, twists, and fractures, 4; contusions, 8.

Some connection between the part injured and the locality of the earliest symptoms is almost unexceptional. If the injury were limited to one hand, the tremor and stiffness begin in that hand; if there were fractures of bone, or contusions or injuries to nerves of one extremity, the symptoms first appear in that extremity.

Thus, a male patient, aged sixty, at the Vanderbilt Clinic, was in a runaway accident and was thrown out, striking the left arm and shoulder. Tremor began soon after in the left arm, and eventually extended to the leg.

Such a relationship might be regarded as coincidental if it were true only of the upper extremity, where the disease regularly begins; but it is also almost always true for those

other parts in which nontraumatic paralysis agitans only very exceptionally has its initial symptom.

Charcot reports two cases in which the influence of the injury upon the development of symptoms appears unquestionable. In one case:

A woman fell out of a wagon and received a contusion of the left side. She soon had severe pain, referred to the sciatic nerve, and in a short time the whole limb began to shake. The tremor was permanent, and extended to all the other members.

In the other case, a woman suffered a dislocation of the jaw. The paralysis agitans, which soon developed and eventually became general, first appeared in the jaw.

When the injury is general, such as a concussion of the whole body, the symptoms begin in one or both of the upper extremities.

Thus, in a case described by Walz:

Male, aged sixty-one, laborer. No syphilis or alcoholism. Heredity negative. Was struck on the back of the head by a package weighing about sixty-five pounds, which had fallen from a fifth-story window. The patient was knocked down and rendered momentarily unconscious. After ten minutes he got up and walked home. For two weeks he was in bed, with pains in the upper extremity from the neck to the fingers, with rhythmical movements of both hands and of the head. The patient recovered sufficiently to get up, but he could not work, and he was granted a pension, in accordance with the German law, for "paralysis agitans, which was the direct result of injury." The patient died from spasm of the respiratory muscles four years after the accident. The autopsy report says: "There are no local findings, and certainly no hemorrhages of old or recent date. No marked atheroma, and nothing abnormal in the dura or bones."

Paralysis agitans often develops at the site of an injury received years before. Examples of this are very common:

A patient of mine, when a young man, received a severe mutilation of the right hand. The wound healed perfectly, and although

the right hand remained practically useless, the patient for twenty years had no symptoms of nervous disease. At the end of that time the right wrist and arm began to shake, and general paralysis agitans eventually developed. From the Vanderbilt Clinic records may be quoted the case of a man who, at the age of forty, cut the ulnar side of his right wrist. Paralysis agitans appeared twenty-two years later; it began in the right hand. Also that of a man, three fingers of whose left hand were amputated at the age of thirty, and in whom the first symptom of paralysis agitans, coming on at the age of sixty-three, consisted of tremor in the operated hand.

In such cases, on account of the long-time interval, the injury can only be regarded as a predisposing cause.

The intermediate or the "bridge" symptoms, which exist between the receipt of the injury and the appearance of definite symptoms of paralysis agitans, consist in general nervous shock, and in pain, weakness, and some stiffness in the parts which are to become the seat of the tremor.

They usually appear soon after the injury. To refer once more to Walz's analysis, tremor appeared at once, or within a day, in 8; soon, or in a few days, in 7; after one to eight months, in 7, and after one to four years, in 4.

The tremor is the first pronounced symptom to appear; the other symptoms are of gradual development, and do not vary essentially in their course from the symptoms of the disease when it occurs independently of traumatic influences.

It is impossible to explain the genesis of traumatic paralysis agitans further than to note the sequence of events and to infer that the injury stands in some causal relation to the disease. Walz believes that the disease can appear only in persons whose nervous systems are already deteriorated. While the truth of such a theory cannot be denied, it cannot be proved, because it is rarely possible to demonstrate any predisposition in the patient.

While it may seem reasonable to suppose, at least after

slight injuries, that the disease would have eventually developed under any circumstances, there are no means of knowing that such would have been the case.

Multiple Sclerosis

Multiple sclerosis is a chronic degenerative disease of the nervous system, of which the underlying pathological condition is scattered areas of neuroglia growth throughout the cerebrospinal axis.

Symptoms.—The characteristic symptoms are nystagmus, intention tremor of the hands, scanning speech, and a condition of spasticity in the extremities. Other symptoms occur, varying with the situation of the neuroglia growth. Fully developed, no disease is more characteristic; but the symptom complex is often incomplete, so that, in many cases presenting one or two symptoms only, the diagnosis is conjectural.

Ætiology.—As regards cause, most of the cases follow infectious diseases, notably typhoid, or else develop without an adequate cause being discoverable. It is assumed by some that the abnormal islets of neuroglia are congenital, and that no cause acts other than by stimulating them to growth.

As in all chronic nervous diseases, trauma figures in the ætiology. In an analysis of 109 cases at the Vanderbilt Clinic, Jelliffe found 13 cases with preceding trauma, and quotes the statistics of Hoffman as giving ten per cent, and of Klausner as giving twenty per cent, of traumatic cases. It is doubtful if trauma could be shown, with reasonable probability, to stand in so high a causal relation. It is not to be gainsaid, however, that the typical symptoms of multiple sclerosis develop in direct sequence to severe general injuries, and to injuries to limbs. Jelliffe suggests that trauma and infection may be associated in causation. At a later page reference will be made to multiple

hemorrhages in the cerebro-spinal axis from severe injury being the possible cause of symptoms in many ways similar to those of multiple sclerosis; and of other cases in which there develop multiple foci of softening due to arterial disease. But both these latter have material differences in their symptomatology from protean multiple sclerosis.

Tumors of the Nervous System

According to the theory of Cohnheim, tumors have their origin in undeveloped embryonal cells which remain inactive until, through the agency of some exciting cause, they are stimulated to a disordered growth. The nature of the essential exciting causes is unknown, but that trauma is one of them is commonly believed. There is little question as to the possibility of long-continued irritation inducing malignant neoplasms, especially in the mucous membranes. To prove that a single acute injury may bring about such a result is very much more difficult.

In tumors of the brain, a preceding trauma figures in the history in from five to twenty per cent of all cases. But such cases are not distinguished from others either as regards clinical cause or the pathological characters, or location of the tumor, or the age or sex of the patient. Doubtless, in many of them the injury was without influence, and only hastened the new growth. Illustrative cases are the following:

In a recent autopsy held at the almshouse upon the body of a woman, seventy years of age, who had never given any symptoms of cerebral disturbance, there was found a large endothelioma upon the internal surface of the dura mater, on the right side of the middle fossa of the skull, lying adjacent to the squamous portion of the temporal bone just posteriorly to the greater wing of the sphenoid. On the opposite side of the skull was an old, undepressed fracture. The question is, of course, open as to whether the situation of the

tumor were entirely accidental, or whether at the time of the fracture sufficient force was exerted by *contrecoup* to incite to morbid activity embryonal cells in the dura mater, with the resulting formation of an endothelioma (Fig. 80).

A still more suggestive case has been reported by Carara, in which a man, previously well, received a blow on the head and died of tumor of the brain within five months. The patient, who was thirty-nine years of age, was struck with a stick on the left posterior

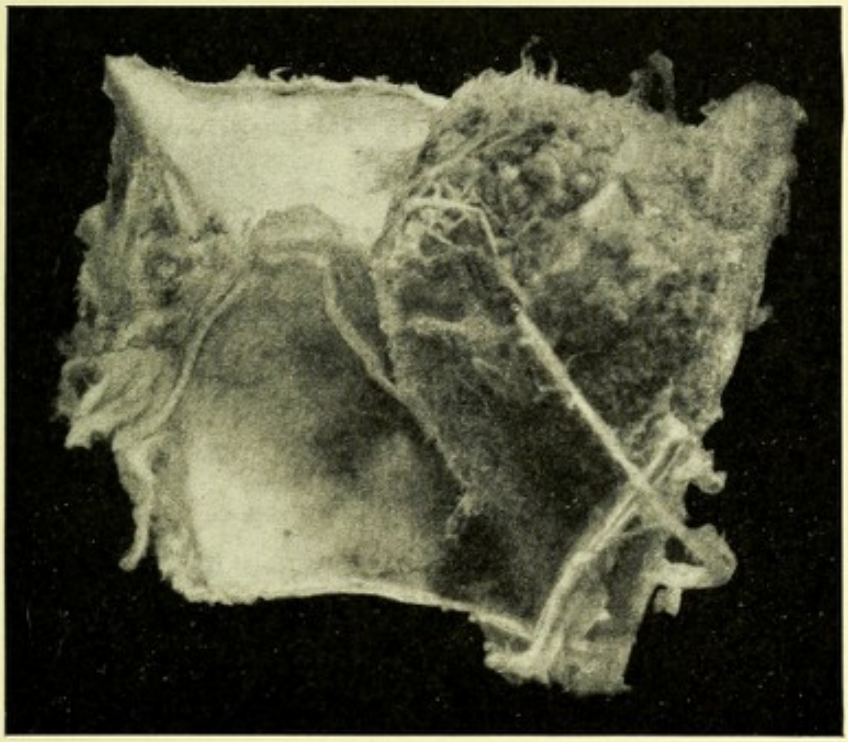


FIG. 80.—Endothelioma of the cerebral dura mater. Possibly of traumatic origin.

parietal region. The scalp wound healed in about six weeks, but there soon occurred headache, deafness in the left ear, and dizziness and numbness of the left side. These symptoms became worse, and the patient entered the hospital, where it was found that, in addition to the symptoms already mentioned, there were a weakness and diminution of cutaneous sensibility in the left arm and leg. The reflexes were normal. The patient began to have convulsive seizures, and soon after this he died. The autopsy showed that the skull was not fractured, but that there was a glioma in the right side of the brain, in the neighborhood of the Rolandic fissure. Whatever may be one's opinion, it is impossible to decide whether the blow on the head was

the sole cause, or whether the tumor had begun to grow before it was received.

The following circumstances point to a direct causal relationship between brain tumor and trauma. Absence of symptoms prior to the accident. Brain tumor may be latent and symptomless for some time, but, as a rule, less so than other chronic diseases. Pain or dizziness, or other symptoms, are usually of early appearance. Tumors being of relatively slow growth, symptoms, in traumatic cases, are not to be looked for until after an interval of at least several weeks. If symptoms appear immediately after the accident, it is more probable that the tumor preëxisted. The injury should be to the head, and of considerable severity. The situation of the tumor should be in anatomical relationship to the injury to the head. In fatal cases, the microscopic examination of the tumor, by establishing its probable duration, may throw some light upon its relationship to the injury.

Practically the same may be said of tumors of the spinal cord as for tumors of the brain. In a small number of cases the injury is the starting point of the symptoms, and without doubt stands in close causal connection to them.

Sarcoma of bone is the most frequent variety of tumor traceable to injury. In the skull these tumors are not common, but in the vertebræ they are comparatively frequent, and cause pressure effects on the cord. Lowenthal has collected 7 such cases with distinct traumatic history.

In the peripheral nerves, fibroma or fibroneuroma is the one important variety of tumor. This may occasionally develop after contusions or wounds of single peripheral nerves.

Syphilis of the Nervous System

By Saenger, and others, syphilis, as an important factor in predisposing to the traumatic neuroses, has been duly emphasized. Whatever rôle it may play in the ætiology of these baffling affections, in their symptomatology it plays none. From their clinical manifestations, or from their course, the preëxistence of syphilis can neither be affirmed nor denied. It is not intended to speak of these disorders under the present heading, but rather of those in which syphilitics, after definite traumatic injuries, develop nervous symptoms of characteristic type. The earliest writers on syphilis of the nervous system have emphasized such a relationship. Yet, in the extremely voluminous literature of the syphilis question, there are but few instances recorded in which it is made plain. From a considerable experience with general traumatic injuries of the nervous system, I have come to the conclusion that trauma as a direct excitant of nervous syphilis is extremely rare. That it does sometimes so act, however, cannot be denied.

Nonne reports a case of brain tumor and one of basal meningitis, both valuable in their ætiological relations. In the first, a young woman, two years after infection and six weeks after a fall on the head, from which she was unconscious for an hour, developed symptoms of brain tumor. Remedies proving unavailing, operation was resorted to, and a gumma was found in the anterior central convolution. The second case, which was verified at autopsy, was that of a longshoreman, who, with the history of syphilis three years previously, developed the symptoms of a progressive basal meningitis. These came on as a result of a fall which had caused a severe cerebral concussion.

The following case was personally observed. A single man, thirty-five years of age, two years after contracting syphilis, for which he had received thorough and apparently effective treatment, was hit on the head with a mallet. He received a slight scalp wound, which just reached the periosteum. He was not rendered unconscious, and did not complain of headache. He developed, however,

a condition of somnolence and inattention. He would lie in bed till the afternoon; he lost interest in things, and was more or less oblivious to his surroundings. About three weeks after the accident he suddenly developed a left facial palsy, and the right pupil became larger than the left.

Under energetic specific treatment these focal symptoms and the great somnolence vanished, and during the five years the patient has been under observation no focal symptoms have reappeared, with the exception of slight sluggishness of the pupils. Mentally, however, the patient underwent a complete change from the outset. Immediately after the accident, as has been said, somnolence and apathy were pronounced, which disappeared for a time under specific treatment. In the succeeding months, however, the patient became irritable and very erratic, and developed delusions of persecution. He finally threatened homicide against one particular person, who he thought had used him ill, so that it was necessary to have him committed to an institution. This idea of injury, which was an insane delusion, faded away with time, and the patient's liberty was restored to him. But he remained partly demented, and to the present time has been entirely incapable of resuming his business. Specific treatment has proved, for the mental symptoms, entirely ineffectual. The absence of physical signs and the long persistence, without material change of the dementia, argue the diagnosis of cerebral syphilis, of which the immediate exciting cause was trauma.

As regards syphilitic affections of the spinal cord after trauma, there is very little reliable information available. Syphilitic affections of the vertebræ are excessively rare, and still more rarely affect the cord. They are difficult to differentiate from tuberculosis. An example of this is the following:

A young man, one year after syphilitic infection, fell heavily on the back. He sustained no immediate injuries, but a month later the legs began to get stiff, and he gradually developed a well-marked spastic paraplegia, without anæsthesia or bladder disturbances. Pain in the back was never prominent, but occurred after sitting in one position and after exertion. It was referred to the eighth, ninth, and tenth dorsal spines, which, two years after the accident, when examined by Dr. Gibney, were unduly prominent.

After two years of treatment in bed with plaster jacket the deformity disappeared, but there was no change in the spinal cord symptoms.

It is far from clear, in this case, whether the injury precipitated a specific meningomyelitis or whether there was caries of the vertebræ.

Syphilis of the peripheral nerves is the rarest form of nervous syphilis. In the diagnosis it should be borne in mind that syphilis, when it attacks the nervous system, is usually disseminated and widespread; that, although the chief symptoms may be focal, careful search often reveals others which indicate lesions elsewhere.

Diabetes Mellitus

As a traumatic affection diabetes mellitus must be considered from two points of view. First, as a temporary condition, resulting from injuries to the head, already referred to (p. 61). Such a condition, recorded examples of which are numerous, might be more properly designated as traumatic glycosuria, as many of the symptoms of diabetes are absent, and as the progressive course of the latter disease is not followed. The injuries are usually severe, the sugar appears promptly, but, most frequently, in small amounts, and the urine does not contain acetone or diacetic acid, often found in advanced diabetes.

Glycosuria appearing in this way has doubtless much in common with that produced by the Claude Bernard puncture of the medulla. The nervous control of the storage of glycogen in the liver and muscles, presided over by the medulla, is temporarily interfered with. In clinical traumatic cases, the medulla being rarely, if ever, demonstrably injured, we assume that it is acted upon indirectly. Certain it is that traumatic glycosuria is not a localizing brain symptom.

Contrasted with the cases of temporary glycosuria are those in which, after injuries, diabetes, characteristic in course and symptoms, appears.

There are numerous cases of this character in the literature.[1] They have occurred in sequence to injuries to the head, to general injuries, and to fright.

Ebstein reports the case of a locomotive engineer, who, as the result of a collision, developed a traumatic neurosis, and three months later the symptoms of diabetes, which persisted until death.

Naunyn gives the history of a man, twenty-eight years of age, who had passed a life-insurance examination in the summer of 1894, and who, three weeks after a severe head injury a year later, developed excessive thirst and polyuria. In January, 1896, sugar was demonstrated, and persisted for three months, the period of observation.

Reibel describes the case of a woman, who, during the fright incidental to the siege of Strasburg, developed itching, which was followed by the other symptoms of diabetes.

All these cases are open to the objection that it is not proved that diabetes did not exist at the time of the accident.

Clinical and experimental data point to chronic disease of the pancreas, possibly also of the liver, as the causes of diabetes.

Trauma, therefore, as a factor, is most important in cases in which the disease develops slowly, and within weeks or months after the accident. A patient who immediately after the injury presents all the symptoms of pronounced diabetes, in all probability suffered from the disease before.

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[1] In a series of 3,161 cases, tabulated from different authors by Sternberg, there were 79, or 3.4 per cent, with history of a preceding trauma. These statistics probably exaggerate the proportion of cases which could withstand critical analysis.

PART II

FUNCTIONAL EFFECTS OF INJURY—THE NERVOUS DISORDERS WHICH MOST FREQUENTLY FOLLOW RAILWAY AND ALLIED ACCIDENTS—THE TRAUMATIC NEUROSES

CHAPTER I

INTRODUCTORY CONSIDERATIONS OF THE TRAUMATIC NEUROSES

History—Nomenclature—Pathology—Causation—Symptoms

History

An eventful pathological history attaches to those nervous disorders which most frequently follow railway and allied accidents, and in which there is no reason to suppose that there have been gross lesions to the nervous system. Explained at first on a theory of disturbance of vascularity of the spinal cord, they were later thought to depend upon multiple areas of inflammation disseminated throughout the whole cerebro-spinal axis. They have been successively regarded as nearly altogether organic, nearly altogether functional, or nearly altogether feigned. They have been named after two men—Erichsen and Oppenheim—who held very different views as to their nature. They have been, and still are, called by many different titles, chief of which are railway spine, railway brain, concussion of the spinal cord, concussion of the brain, spinal anæmia, spinal irritation, traumatic neurosis, traumatic hysteria, trau-

matic neurasthenia, and traumatic hysteroneurasthenia. These disorders have become uninterruptedly more frequent and more important. They are accident neuroses, and we live in an age of accidents; they often result from actionable negligence, and the frequency of actions brought for personal injuries is constantly increasing. More intimate familiarity with the general diseases of the nervous system has perfected our powers in the diagnosis of all nervous affections, and it now seems that most of these functional traumatic neuroses can be properly classified, even though they may not be perfectly understood.

Such a classification and description will be the object of the following pages, and may be best accomplished by first passing in review the various opinions of men who have contributed to this subject.

Although concussion of the spine had been described by Abercrombie, Ollivier, and others, the first important writer on railway injuries was Erichsen. Erichsen was a surgeon, and wrote at a time when nervous disease was but little understood. Gross lesions, such as hemorrhage and acute softening, were of course recognized, but almost nothing was known of the microscopical pathology of the nervous system. Had Erichsen been the most expert neurologist of his time he could have known the finer anatomy of none of the degenerative diseases except locomotor ataxia. The knowledge of nervous symptomatology was correspondingly imperfect. From Erichsen's descriptions it is very hard to tell what many of his patients suffered from. He evidently did not recognize the differences between ataxia and paralysis, for in the case of posterior spinal degeneration, which will be referred to later, he describes the symptoms as paralytic, when in reality they were ataxic. He says little about electrical reactions, does not examine the reflexes, and makes very cursory mention of anæsthesia. Erichsen (I quote from the later edition of his book)

recognized that these disorders were not necessarily the exclusive results of railway accidents.

Of his 53 cases, only 17 were injured on railways. In most of the others the violence acted immediately on the spine, as in direct blows, injury to the back by the falling of trees, etc., or in falls from considerable heights on the head or on the buttocks.

The violence was generally severe, and entirely capable of fracturing bone or rupturing ligaments, and not the kind of violence which is most frequently associated with railway injuries. Many of his cases give evidence of severe organic lesion of the nervous system, and would to-day undoubtedly be regarded as depending upon visible anatomical changes of a morbid character. They would now be explained not as Erichsen explained them, by an assumption of molecular changes in the spinal cord, but by the occurrence of such visible damage as may be caused by compression of nervous tissue from splinters of bone, or by hemorrhages resulting from laceration of blood vessels, or from the wounding of any of the intraspinal structures. Of only a few of Erichsen's cases can it be said with reasonable certainty that they were functional—not dependent upon local conditions of an organic character—but due to a functional disorder of the whole nervous system. These latter Erichsen did not recognize for what they really were.

With the exception of one case of syphilis of the nervous system, he apparently did not appreciate the possible influence of trauma upon hastening the clinical manifestations of latent nervous disease. He also failed to distinguish between symptoms of spinal and cerebral origin. For example, he reports a case (No. 48) of paraplegia, with involvement of the face, as due to spinal injury.

Confusion will invariably result in the mind of anyone who

begins the study of traumatic nervous disease by a perusal of Erichsen's work. He will there find grouped together cases which are evidently of very diverse character. Erichsen makes of concussion both a cause and an effect. For him, concussion is not only the shock, the jar of the accident, but it is also the disarrangement of molecular structure of the spinal cord which may result in alterations of blood supply (anæmia, hyperæmia) sufficient to give clinical symptoms.

Erichsen's theory of concussion of the spinal cord as a pathological entity was purely hypothetical. Microscopical technique is more advanced now than it was at the time he wrote, yet it has thus far failed to disclose not only molecular pathology but any such thing as molecular structure. Molecular disorders are still in the domain of speculative science. The significance of local spinal anæmia and hyperæmia, to which he ascribed much importance, is absolutely unknown. It is not intended to cast any discredit upon Erichsen or his work, but, writing at the time he did, it would have been impossible for him to formulate a theory which could serve as an authority to-day. The following case, his one autopsy for the determination of the pathology of spinal concussion, seems to prove this:

> Patient, fifty-two years of age at the time of his death, a man of active business habits, had been in a railway collision. Immediately after the collision the patient walked from the train to the station, which was close at hand. He had received no external sign of injury, no contusions or wounds, but he complained of a pain in his back. Being very unwilling to give in, he made every effort to get about in his business, and did so for a short time after the accident, though with much distress. Numbness and a want of power in the muscles of the lower limbs gradually but steadily increasing, he soon became disabled. His gait became unsteady, *like that of a half-intoxicated person*. . . . In the latter part of his illness some weakness of the upper extremities became apparent, so that, if the patient was off his guard, a cup or a glass would slip from his fingers.

. . . There was no paralysis of the sphincter of the bladder until about eighteen months before his death.

This is apparently the description of a case of locomotor ataxia, and the autopsy by Lockhart Clarke, instead of furnishing an explanation of the pathology of spinal concussion, as Erichsen supposed it did, added one more to the list, at that time small, of the recorded cases of primary degeneration of the posterior columns of the spinal cord. Dr. Clarke says, in his autopsy report:

The membranes at some parts were thickened, and adherent at others to the surface of the white columns. . . . On making sections . . . of all the white columns, the posterior were exclusively the seat of the disease. These columns were darker, browner, denser, and more opaque than the antero-lateral, and in their preparations under the microscope this appearance was found to be due to a multitude of granular corpuscles and isolated granules, and to an exuberance of wavy fibrous tissue disposed in a longitudinal direction.

Erichsen did not appreciate that many serious disturbances of general nervous function may result from injury and shock when there has been no local damage to the nervous system itself.

Thus, Case VI, which he describes as "concussion from a fall out of a bathing machine," might be construed as a case of hysterical paraplegia, or of astasia-abasia:

A young lad, fourteen years old, fell in shallow water, striking the sandy bottom. He received no mark of external injury, and walked home. His legs gradually became weak and numb. Erichsen found, ten days later, that "he was quite unable to stand, but when lying in bed could kick out his legs quite in a natural way." He recovered completely in four months.

Erichsen's work, however, possesses more than a merely historical value. It called attention to the possibility of fractures of vertebræ and of tearing of ligaments without external evidences of injury, and proved that serious nervous symptoms

may result from injuries which at first appear trivial. But he left almost untouched the complicated questions involved in the greater number of railway injuries, and, through a failure to recognize the frequent subjective character of the symptoms, his work is to be regarded as a contribution to surgery rather than to neurology. It remained for subsequent writers, more experienced than he in general nervous diseases, as well as in the forms which most frequently result from railway accidents, to explain the chain of symptoms which are most commonly recited by injured persons to claim agents, and which are now called the neuroses of traumatic origin.

While Erichsen's book marks the beginning of the study of the nervous disorders following railway and allied injuries, it was a long time before their true nature was understood. Ten years after Erichsen's first edition, Erb still believed in molecular disturbance as the chief element of concussion. Westphal, in 1878, reports three cases, in two of which the symptoms were so widely disseminated and so pronounced that he ascribed them to multiple areas of cerebro-spinal softening.

Riegler, in 1879, thought that in many cases the symptoms were exaggerated or assumed. In the absence of exaggeration or feigning, he regarded the symptoms as due to organic lesions.

In America, Hodges was the first to object to Erichsen's pathological views. In 1880 he read a paper before the Boston Society for Medical Improvement, in which he said that "we have no knowledge to justify the proposition that what is popularly called 'concussion of the spine' is due to a molecular disarrangement of the cord by an assumed shake or jar. That vascular disturbances (anæmia, hyperæmia, hemorrhages, spinal apoplexy), followed by meningitis, myelitis, or degenerative changes of the spinal cord, are shown by experimental pathology, and to a certain extent by post-mortem examinations, to be the true cause of symptoms heretofore commonly

designated as concussion of the spine." He thought Erichsen's book "presents an exaggerated picture of its symptoms and consequences which are not justified by our present knowledge of the subject." Hodges also suggested the possible functional character of many of the symptoms.

It remained, however, for Page to prove, as conclusively as such things can be proved, that although falls, jars, and shocks might cause organic injury to the spinal cord, such a result is relatively infrequent; that railways accidents, in which the factors of concussion and fright are so intimately mingled, more frequently cause nervous disorders of a functional than of an organic character. Page was surgeon to the London and Northwestern Railway of England, and had had a very large experience in railway cases. His experience was essentially different from Erichsen's, in that, as a corporation surgeon, he saw many cases of trifling injury, and had to be constantly on his guard against imposture and exaggeration. His work bears the marks of intelligent and impartial observation, and although he may have been to a certain extent prejudiced in favor of the railway company many of his views have been substantiated by subsequent observers. Page saw the errors of Erichsen's work, and emphasized the important fact that there had been no satisfactory pathological proof of disease of the spinal cord resulting from concussion, as distinguished from recognizable injury. He showed that the discussions regarding spinal concussion were entirely empirical, and that there was no proof to show that such a condition, comparable to the demagnetization of a magnet by a blow of a hammer—a favorite simile of Erichsen's—was ever caused by direct blows on the back or by a general shaking up of the whole body. He fully admitted the possibility of intraspinal hemorrhage without external evidences of violence, but he claimed that such injuries occurred in a relatively small proportion of railway cases, and did not

need to be explained by any theory of molecular disarrangement. The larger number of Page's cases had received slight injuries, and the symptoms which came on immediately or soon after the accident consisted chiefly of pain in the back, associated with the common subjective disorders of neurasthenia, but without any of the localizing signs of organic injury. He also recognized the existence of disturbances of motion dependent upon idea or loss of will power. His chapter on Functional or Neuromimetic Disorders gives several interesting cases which would to-day be called traumatic hysteria. One of these may be quoted, to show the long duration which is possible in functional paralysis:

"Case of functional motor paraplegia. Extreme emotional disturbances." Man, aged forty-one, who was naturally very excitable, was in a very severe collision in which the carriage he was in was smashed to pieces. There were no evidences of serious physical injury, but the patient was very nervous, and in bed for several days. Complained of pain in the back. No tenderness. He dwelt on the fear of paralysis, and steadily lost power in the legs. Eight months after the accident he was quite unable to walk, and failed entirely to make any requested movements of the legs or feet during examination. There was no paralysis of bowels or bladder, and the sensory impairment was at best slight. There were no bedsores or atrophy of muscles.

Nine months after the accident the patient had a sudden attack of aphonia, which lasted three weeks and disappeared as suddenly as it came. He was troubled by excessive emotional irritability. Page's report to the company, nine months after the accident, was, that "the cause of the paralysis seems to lie in the directing power of the will, rather than in lesion discoverable of the brain or spinal cord." The man ultimately recovered, but was ill four to five years. His recovery came suddenly. He had been unable to get out of his chair without help until one day he got up without knowing it. His son said to him, "Why, father, look what you've done!" "Good God," he replied, "I have got up myself!" From that day he could get up without difficulty.

Page publishes an appendix to his book, in which are tabu-

lated the ultimate results of 234 cases as observed from two to five years after the accident. He says that most cases recover completely, although recovery may be much delayed.

He admits, however, that the injured man may never be as well as he was before, and that many patients permanently retain evidences of impaired health. From an examination of his table it would seem that complete recovery did not occur in more than seventy per cent of the cases.

Page's book quickly obtained wide favor, and a second edition appeared in 1885. At this time, also, the possibility of hysteria occurring as a result of injury and shock became recognized, and attention was called to the fact that some of the cases of "concussion of the spine" were purely hysterical. Putnam had an accident case of hysterical hemianalgesia and paraplegia, and emphasized the necessity of looking for hysteria in all concussion cases. Walton reported a case of hysterical anæsthesia brought on by a fall, and in an article entitled "Spinal Irritation" he suggested a probable cerebral origin for many of the symptoms of these disorders, and proposed the term "railway brain" as a substitute for "railway spine." Dana, in December, 1884, gave the first complete *résumé* of this class of cases. His conclusions—which, with very slight modifications, have been proved to be correct—are: "That the term 'spinal concussion' is misleading and often incorrect, and the symptoms which are usually associated with that name are really symptoms of traumatic neurasthenia, hysteria, and hypochondriasis, associated more or less with symptoms of injury to the vertebral muscles and ligaments and to the spinal nerves; that, in other words, concussion is mental shock and physical bruising." Dana believed in "concussion of the spine" as occurring in rare instances, and recognized the possibility of injuries and jars causing myelitis without there being any injury to the spinal column.

In the following year, Thomsen and Oppenheim, assistants of Westphal, in support of the theory of an organic basis for these disorders, maintained that hemianæsthesia was not a pathognomonic symptom of hysteria, but might result from organic cerebral injury, and consequently could not be regarded as proof that many of these cases were not organic in character It must be remarked, however, that the cutaneous insensibility studied by these authors in patients suffering from cerebral disease differed in essential particulars from the hemianæsthesia of hysteria. It occurred as a general hypalgesia, or diminution of sensibility to pain, which was more marked on one side than on the other, rather than in the form of the sharply defined unilateral loss of sensation so commonly observed in hysteria. Also, nearly all the cases examined by them presented psychical anomalies—hallucinations, apathy, depression, etc.—conditions which render any examination of sensations unsatisfactory and unreliable. Consequently, the paper by these authors can in no way be regarded as overthrowing the theory of a hysterical character for many of the traumatic affections under discussion. In 1885 Oppenheim returned to the views of Westphal, namely, that the symptoms of "railway brain" were due to disseminate foci of inflammation or softening.

Strümpell, writing in 1888, described a general traumatic neurosis and a local traumatic neurosis.

The general traumatic neurosis, which is apparently a mixture of hysteria and neurasthenia, he illustrates as follows:

> A healthy man falls from a height, is unconscious or dazed, and has to be carried home. There are no serious injuries discoverable. Recovery is very slow, and the patient stays in bed for some time. Then he finds himself unable to work. He becomes very nervous. He is melancholy and depressed, and ceases to take an interest in his surroundings. He loses his energy and will power, and becomes agitated on slight provocation. There may be considerable loss of memory. There is difficulty in fixing and holding the attention.

Sleep is disturbed by dreams. There are cutaneous anæsthesia and hyperæsthesia (especially of the back), and there may be a dissociation of sensations. The function of the special senses is impaired.

The motor difficulties consist in weakness rather than paralyis. Tremor and stiffness of the muscles are frequently present. The skin reflexes are often absent, and the deep reflexes are generally exaggerated. There are various functional disturbances of the vegetative organs, and complaints are made of loss of sexual inclination and power. Many of these cases are incurable.

Under the head of local traumatic neurosis Strümpell described the various local manifestations, such as paralysis, contracture, hyperæsthesia, joint affections, etc., of hysteria.

Interest in the causative relation of trauma to nervous disease reached its height when, in 1889, Oppenheim published his celebrated monograph, "Die traumatische Neurosen" ("The Traumatic Neuroses"), a book which has been widely read, and which has exerted no little influence upon subsequent discussions of nervous disorders following accidents. Oppenheim did not attempt any description of direct lesions of the nervous system. He only delineated the clinical forms which result from accident and injury, and which affect the nervous system "through sudden vibration or in reflex ways." He admitted the existence of traumatic hysteria and traumatic neurasthenia, but to the forms of functional traumatic disorder, which are not typical for either hysteria or neurasthenia, he gave the name of *traumatic neurosis.*

The symptoms of Oppenheim's traumatic neurosis are chiefly a combination of the symptoms of severe neurasthenia, with some hysterical manifestations. They may develop immediately after an accident, or their appearance may be delayed for weeks or months. The first disturbances are purely subjective, such as pains in the back, or headache, anxiety, and restlessness. Depression and general worry may amount to hypochondriasis, without, however, direct impairment of the

mental power. The patients are sleepless, dizzy, and subject to " weak spells " ; they have bad dreams ; tremor occurring as a simple vibration, or in the form of tic convulsif, chorea, epilepsy, etc., are not infrequently observed. Disturbances of voluntary motion may usually be explained by pain caused by movement, though the muscles are nearly always weak and may be entirely paralyzed ; there may be a weakness of all four extremities, or hemiparesis, or paraparesis, or monoparesis ; the paralysis differs from organic paralysis in important particulars, one of which is a peculiar rigidity of the muscles. The knee jerks are frequently exaggerated, but never lost. Atrophy may occur.

Disorders of speech are limited to a form of stuttering. Inequality of the pupils is frequent, but they rarely fail to react to light. Loss of sensibility of the skin and mucous membranes occurs, but does not follow the distribution of organic anæsthesia. The visual fields may be contracted. Vasomotor disturbances are prominent. The bladder is rarely involved, although there may be difficulty in passing water, or incontinence or retention of urine. The sexual function is generally impaired. The condition of the skin reflexes is not constant. They may be active, but are usually diminished or lost in anæsthetic areas. Cardiac irritability is frequent. Oppenheim deduces the symptomatology from 42 cases which he had observed. Very few of the patients entirely recovered.

Oppenheim's monograph was followed by the appearance in America of a book by Clevenger, of Chicago, entitled " Spinal Concussion." Clevenger proposed the name of " Erichsen's disease " for these disorders. He says : " Erichsen's disease is a group of mainly subjective symptoms of a nervous and mental nature, sufficiently characteristic to enable it to be recognized as a traumatic neurosis, distinct from other traumatic neuroses, with which it may or may not be associ-

ated. The most common cause of Erichsen's disease is a concussion of the spinal column, including its contents and nearest appendages."

The term "Erichsen's disease" has not met with favor as a designation for any symptom group, and Clevenger's work is said by Gilles de la Tourette to be "*le triomphe de la confusion des idées en pareille matière.*"

Since 1889 the neuroses following accident or injury have been a subject of contention, especially among neurologists. The views of Strümpell and Oppenheim created much discussion. By some, the existence of a special traumatic neurosis, with characteristic symptoms, was unreservedly accepted. Many indorsed the theory with certain modifications, and a few spoke emphatically against the individuality of any neurosis which occurs only as a result of injury and shock.

Eisenlohr and Schultze, especially, questioned the value of the individual symptoms upon which rested the theory of a clinical entity for a special neurosis after trauma. Schultze declared that "while a variety of neuroses and psychoses may be induced by trauma, the traumatic neurosis as such has no existence." Bruns also adopted this view, believing it more correct to make the diagnosis of traumatic hysteria or traumatic neurasthenia than to speak of a traumatic neurosis. But Bruns admitted that, even after the most careful efforts at differential diagnosis between the various neuroses of traumatic origin, there remained a certain number of mixed forms which cannot be satisfactorily classified.

The opposition to the admission into medicine of a traumatic neurosis as a distinct disorder became more and more pronounced. At the Twelfth International Medical Congress, held at Wiesbaden in 1893, it was generally accepted "that, even after slight injuries, general functional disorders not infrequently develop. These neuroses are not disease pictures of

any particular variety, but all may be classified under the name of other well-recognized neuroses, of which the most important are hysteria, neurasthenia, hypochondriasis, and their mixtures."

The literature of the traumatic neuroses is now very large. Page, in 1891, published a new and shorter book. In this country Knapp has embodied the results of his extensive study and experience in an excellent chapter in the "Text-Book of Nervous Diseases by American Authors." Dana also discusses the subject very thoroughly in Hamilton's "Text-Book of Legal Medicine." Dr. Outten, in Witthaus's "Medical Jurisprudence," writes with an authority acquired through large experience as a railway surgeon. Crocq has written an essay on the "Névroses traumatiques," which has been crowned by the Belgian Academy. An able summary of the questions involved has been given by Saenger.

Strümpell has cast doubt upon the value of many of the individual symptoms of the traumatic neuroses. His new position was attacked by the men who took part in the discussions at Hamburg and Frankfort in the spring and summer of 1896.

Angell has said the following of railway spine: "Railway spine is a convenient and picturesque term which has hypnotized juries, even as the shock has hypnotized the plaintiff. It is dramatic but not accurate. The damage done is not to the spine or spinal cord, but to the mind. "It is a psychical disorder, not a physical one, although it has a physical expression in its symptomatology."

Hamilton has recently embodied the results of his experience in an impartial monograph, classifying many of the functional conditions under the term "aboulia."

Such is the history of the views regarding the functional nervous disorders which most commonly follow railway and allied injuries. From the time that they were recognized as

depending upon functional nervous disturbance, of which the structural changes are more minute and more baffling than the coarse lesions of hemorrhage, softening, or inflammation, they became the object of study by neurologists rather than by surgeons.

Discussions concerning them have been held with activity, and at times with acerbity. It is not surprising that, without the guidance of visible pathological anatomy, the views of different observers regarding the nature of symptoms are not entirely unanimous. But the progress of general neurology has been constant, and year by year our understanding of the genesis and character of these neuroses becomes clearer. To-day, although not perfectly understood nor classified with absolute security, they have obtained a firmer foothold in clinical medicine than they ever had before.

The larger number of cases can be made to agree with well-recognized and well-defined clinical types, so that the course of any individual case can be foretold with a certain degree of accuracy.

The following is a summary of the present understanding of the essential features of the traumatic neuroses:

Nomenclature

There is now a nearly complete unanimity of opinion that there is no such thing as a special neurosis excited by trauma. Injury and shock may be followed by symptoms of any of the well-recognized neuroses or psychoses, or the symptoms of one or more of these disorders may be blended in the same case; but the theory of a special traumatic neurosis lacks clinical foundation and has been generally abandoned. Even the severe forms of nervous disturbance which sometimes follow injury, the observation of which led Oppenheim to coin the term " trau-

matic neurosis," do not need to be explained by assigning them to a special nosological place of their own. Some of them are mixtures of hysteria and neurasthenia, and others may eventually be shown to depend upon structural changes in the central nervous system, of which the clinical manifestations are associated with symptoms of hysteria and neurasthenia. These cases present no symptoms which cannot be explained on such an hypothesis. There is nothing sufficiently individual about them to warrant the use of the term "the traumatic neurosis."

If this view is correct, if there is no individual traumatic neurosis, and if trauma is to be regarded in this connection merely as an exciting cause of other well-known nervous disorders of functional character, the comprehensiveness of the term traumatic neuroses might be very much enlarged. Besides neurasthenia and hysteria with their mixtures and allies, and possibly some forms of organic disease whose pathology is still to be explained, it could include neuralgia, chorea, exophthalmic goiter, or any disease which is a neurosis and which may develop as a result of injury and shock. Such a nomenclature would, however, be entirely at variance with the original significance of the term, and would leave us without any generic designation under which to classify the forms of functional nervous disease which have obtained such prominence in medical jurisprudence. For the present, at least, it seems impossible to dispense with an expression which, although inexact, has come to be generally recognized as applying only to a particular class of cases. But, while retaining "traumatic neuroses" as a generic designation, the physician should not be satisfied in saying of any given case that it is one of the traumatic neuroses, but should continue the examination until he can say, in all probability at least, which one. It is only by repeated efforts at exclusive diagnosis that the pathology

and classification of these disorders will be rendered still more plain.

Strümpell has suggested that "traumatic neuroses" be changed to "accident neuroses." Against this may be urged that they do not necessarily result from accidents. Injuries received in battle can hardly be called accidental. Strümpell's reason for the substitution of "accident" was because a trauma is not always demonstrable. But the wider definition of trauma includes psychic as well as physical injury, and consequently traumatic can be with propriety applied to those neuroses which result from psychic shock as well as from physical injury.

In the following pages these disturbances will be discussed under the heads of

1. Traumatic neurasthenia.
2. Traumatic hysteria.
3. Unclassified forms.

Insanity from psychic shock, while not a traumatic neurosis, can best be considered here also. In traumatic neurasthenia the mental state is subject to wide variations. The disorder may take on the type of hypochondriasis, less frequently that of melancholia. These variations will be here described as variations of a common morbid condition.

Hysteria following traumata is not always of the pure type seen when the affection develops in young women from nontraumatic causes. When owing its origin to injury or fright, it is more commonly associated with pronounced neurasthenic symptoms, such as are usually present in nontraumatic hysteria occurring in men. Although traumatic nerrasthenia and traumatic hysteria frequently occur together (traumatic hysteroneurasthenia), for purposes of convenience they will be described separately. Under "Insanity from Nervous Shock" will be considered some of the pathological effects upon the mind of violent emotion, notably fear.

The uncertainty regarding the group of cases called unclassified is explained by the name. Further information is necessary before they can be assigned to any nosological category. To the other functional nervous disorders, which are neuroses, and which may arise from trauma, such as exophthalmic goiter, chorea, etc., no mention will be made. They are not included by the general term " traumatic neuroses," and require no description in a work of this character.

Pathology

The possibility of injury to nervous tissue, and the symptoms caused thereby, have already been considered in Part I.

The belief, which was first systematically formulated by Page, has now become general that in by far the larger number of the cases of nervous disorders which follow railway and allied accidents there is no gross injury to nervous tissue. Physical concussion may be a cause of subjective nervous symptoms, and if severe may indirectly cause organic injury. But the conception of spinal concussion as a cause for general nervous symptoms and as a pathological condition is without foundation and has been almost entirely abandoned. By far the larger number of cases of the traumatic neuroses may be explained by the theory that the symptoms are those of hysteria or neurasthenia, functional disorders of which the pathology is unknown. There are, however, a few cases in which the symptoms are somewhat different from those of hysteria and neurasthenia, and which may depend upon the structural lesions of the brain and spinal cord, although our knowledge concerning their pathology is still largely speculative.

ÆTIOLOGY

There are so many causative factors common to all of the neuroses of traumatic origin that they may best be described in a general way together. Causal characteristics special to any individual form will be given under its description.

Accidents which become the subject of medico-legal inquiry are more frequently followed by functional nervous disorders than by organic injuries to the nervous system. An analysis of 100 successive cases which were seen by Walton, where nervous symptoms were complained of and in which the question of damages had arisen or was likely to arise, gives the following results: "The date of the examination was from one week to three and a half years after the accident, and the nature of the trauma ranged from slight jars and moderate blows on various parts of the body to violent collisions and severe falls. In 17 cases there were unmistakable evidences of injury to the spinal cord. In the remaining cases the symptoms, although often in many cases pronounced, differed in essential respects from the symptoms of organic nervous diseases."

Although the traumatic neuroses may follow any accident in which injury and shock have been prominent factors, they have been chiefly studied in connection with railway accidents. Interest in them has advanced with the extension of railway travel. Until 1866, when Erichsen's book appeared, little attention had been given to them. It is only in recent years that railway travel has assumed proportions sufficiently extensive to create for railway injuries a medical literature of their own. Improved mechanical appliances, perfected systems of signals, and the enforcement of military discipline among employees all tend to reduce the accident percentage. But these advances are more than counterbalanced by the constantly increasing traffic

on all kinds of surface vehicles, so that the number of persons killed and injured becomes constantly larger.

The following table is taken from the Tenth Annual Report of the Interstate Commerce Commission (Washington, 1896). It contains the total number of persons killed and wounded on railways in the United States during the eight years 1888–1895:

Comparative Summary of Railway Accidents for the Years ending June 30, 1895, 1894, 1893, 1892, 1891, 1890, 1889, and 1888.

Year.	Employees.		Passengers.		Other persons.		Total.	
	Killed.	Injured.	Killed.	Injured.	Killed.	Injured.	Killed.	Injured.
1895....	1,811	25,696	170	2,375	4,155	5,677	6,136	33,748
1894....	1,823	23,422	324	3,034	4,300	5,433	6,447	31,889
1893....	2,727	31,729	299	3,229	4,320	5,435	7,346	40,393
1892....	2,554	28,267	376	3,227	4,217	5,158	7,147	36,652
1891....	2,660	26,140	293	2,972	4,076	4,769	7,029	33,881
1890....	2,451	22,396	286	2,425	3,598	4,206	6,335	29,027
1889....	1,972	20,028	310	2,140	3,541	4,135	5,823	26,309
1888....	2,070	20,148	315	2,138	2,897	3,602	5,282	25,888

In the year ending June 30, 1902, there were 8,588 persons killed and 64,662 injured. One employee was killed out of every 401, and 1 was injured out of every 24. One passenger was killed out of every 1,883,706, and 1 injured out of every 97,244. Surface transportation lines swell these figures enormously. From a comparison of steam- and electric-road casualties, Hamilton states that it would appear that the trolley lines have six times as many deaths and nearly ten times as many injuries as the steam roads. It should not be forgotten, however, that electric roads carry nearly eight times as many passengers as steam roads.

Railway collisions or derailments are particularly well adapted to cause every variety of injury. Decapitation, dismemberment, bruises and crushes of the flesh, fractures, dislo-

cations and twists of the bones and joints, cuts from broken glass, burns and scalds from fire and escaping steam, may immediately induce death or cause disabilities and disfigurements of all degrees. To the physical dangers of such disasters is added mental shock. It is needless to emphasize how lasting must be the terrible impression which is made by the suddenness of the accident, the terrific shaking, the crash of breaking wood and glass, the cries of the wounded, the noise of escaping steam, and the uncertainty and terror of such catastrophes. But while death or physical injuries of every character and degree may result from these accidents, the number of persons who receive in this way organic injury of the nervous system is not great when compared with the number of persons who, although hurt but little or not at all at the time of the accident, eventually develop some nervous symptoms. It is a matter of general remark that the functional disorders occur most frequently when there has been no gross physical injury. In railway accidents there are usually a large number of persons who neither die nor yet are badly hurt. They may be severely frightened, and violently thrown about, and receive twists or wrenches of the spine, or blows upon the back or head; but whatever physical injury is incurred, it does not cause them great pain or disability at the time. Yet it is these persons, who were considered at the time of the accident as fortunate in escaping unscathed or with slight injury, who eventually complain of the symptoms of some one of the traumatic neuroses. There is no reason to suppose that in such cases the nervous system has received any structural damage, for in organic nervous injury there is usually immediate loss of nervous function. In the traumatic neuroses, on the other hand, with the exception of some cases of hysteria, there is an interval between the occurrence of the accident and the appearance of pronounced symptoms.

In many cases actions for personal injuries are brought when there has not only been no physical injury, but even when there has been no general accident. The sudden starting or stopping of a train, the slipping in an aisle or from steps covered with ice, or similar trivial mishaps, are alleged as causes of neurasthenic symptoms. A woman in Brooklyn recently brought suit for $10,000 for alleged injuries (none were visible) received by being thrown from her seat to the floor by the sudden stopping of a trolley car.

But although many escape from railway accidents without severe physical injury, nearly all are frightened; and fright is as fruitful a cause of some functional nervous disorders as physical injury.

Under the term psychic shock are included sudden fright, grief, remorse, and even joy. This factor is one to which it is always difficult to ascribe an appropriate causal value; one, also, which is often invoked as a sole cause, when it is really only one of many.

The importance of emotional factors in the causation of either somatic or mental diseases cannot be minimized. No one, be he physician or layman, can doubt the active part played in the production of disease by the contributing causes of the emotions. It is seen in daily social life, in the routine practice of medicine, and especially after great catastrophes and tragedies, such as explosions, battles, conflagrations, earthquakes, tornadoes, etc. In Reibal's paper descriptive of the siege of Strassburg a graphic account of such effects is given.

The bombardment of Strassburg lasted from August 15 to September 28, 1870. The terror to the inhabitants of the town was indescribable. Shells, bombs, fuses, were sent almost uninterruptedly into the heart of the city. To this were added the fires, which raged everywhere. At times the city streets were said to be practically full of molten iron and lead and burning

débris. Reibal describes 7 cases of cerebral disturbances, 2 of which will be again referred to, in which a fatal issue was the direct result of the bombardment. In 4 of these cases the patients were already infirm. He also observed 2 cases, in women, of epileptic convulsions; neither had had convulsions previously. In one case the convulsions disappeared, in another they became regular in occurrence. These convulsions were regarded as truly epileptic, and were differentiated from hysterical convulsions. Reibal also saw one patient in whom itching, as the initial symptom of a fatal diabetes (see p. 346), developed while she was huddled in a pit where she, with others, had sought refuge from the flying shells. Subsequently to the siege, also, he noted a great increase in the number of cases of diabetes, and of deaths, from cerebral causes, in infants.

In fright, the blood first leaves the body surface, there is tumultuous and rapid action of the heart, and probably an increase in blood pressure. Increased blood pressure in frightened animals has been demonstrated by Conty and Charpentier, and by Bezald and Dainilewsky. After the initial period of excitement, the effects upon the circulation are different, doubtless, in different cases. In some there is persistent tachycardia. Squires reports the case of a girl, frightened by a plate falling on her, in whom the pulse rate changed from 75–80 to 135, remaining at the latter rate for two years, up to the date of the communication. In other cases of psychic shock the pulse is slow, a fall in blood pressure following the initial rise. Vascular instability is shown in the changing character of all the symptoms.

But, important as emotional factors are in causation, to prove them unique factors is rarely possible. Psychic shock may doubtless induce heart failure or apoplexy, but only when vascular disease preëxists. Sudden death, with emotional factors as a cause, is not mentioned in Brouardel's "Death and

Sudden Death," and I feel safe in saying that the case is yet to be advanced in which life ceased as the immediate consequence of such causes alone. General medical diseases are rendered worse by emotional strains, and resistance to them is lessened thereby, but they do not originate in such causes. Of the diseases of the nervous system, the neuroses are the only ones in which fright and similar factors can stand as sole causes. Chorea commonly, and paralysis agitans and exophthalmic goiter in a few well-authenticated instances, have been the unique results of fright. Fright figures prominently, also, in the ætiology of epilepsy, though it probably causes true epilepsy only in persons predisposed to the development of the epileptic habit.

Hysteria and neurasthenia are the neuroses in which emotional causes figure most prominently in ætiology. In the majority of cases, associated with the fright or other similar cause is some bodily injury, trifling though it may be. With this physical factor present, the emotional cause has more or less importance attributed to it, varying with the personal equation of the observer. Thus, dependent upon the mental inclinations of different writers, the traumatic neuroses arise from causes chiefly material or chiefly psychic.

Although accidents on railways stand in the most conspicuous causal relation to some of the traumatic neuroses, any of these disorders may follow mishaps or catastrophes of diverse character in which the elements of physical commotion or injury and psychic shock are prominent.

New inventions and elaborated mechanical contrivances are constantly supplying additional causative factors. They may follow accidents in elevators, in theaters, in machine shops. The introduction of swift surface transit in the streets has caused the annual number of these disorders to very materially increase.

The rapid development in recent years of the industrial uses of electricity has added another to the already long list of exciting causes. Electric currents of high potential are now of such indispensable service in city life that the danger of receiving shocks from street currents, although chiefly limited to electric linemen, is one to which every citizen is more or less exposed. It is not probable that electric currents which do not induce almost instant death ever cause organic nervous disease. No such case has ever come to my personal observation, and a search through literature for the record of such a case has proved fruitless. As far as the nervous system is concerned, it is the functional affections which result from accidents by electricity, and it is the general public, rather than the employee of the company, which suffers.[1] The lineman may be seriously burned or be instantly killed by touching live wires or by receiving in various ways currents of high voltage; but he resembles the brakeman in this respect, that he receives his injury and dies of it, or quickly gets over the effects of it. Constant familiarity has so eliminated the element of fright that for him it is a physical injury, and rarely is followed by any symptoms of functional nervous disease. In persons unfamiliar with electrical appliances the effects of powerful electrical shocks are essentially different from those experienced by the employees of electrical companies. Such a one, who touches or is touched by a conductor which is not insulated, or which he supposed to be not insulated, receives, independently of any electric shock, a fright as severe as the fright caused by a railway collision. Thus, a case is reported by Dana of a patient who had read of the killing of a man by an electric wire. A few days after, he was walking along the street when sud-

[1] In New York City the placing of the electric-light wires underground, which was brought about largely by the efforts of H. P. Brown, Esq., has very materially diminished the number of electrical accidents to the general public.

denly a dead wire fell and hit him on the head. The blow was not very severe but the man fell unconscious, and when he was aroused he was found to have typical hysterical symptoms. Of all the cases of neurasthenia or hysteria which have followed contact with wires carrying high voltage currents, in only a few is there reason to suppose that the full strength of the current passed through the body. It is very difficult to determine the exact strength of current necessary to cause extensive burns or instant death. In the executions at Sing Sing the currents are not of higher voltage than some of those which pass through city streets, yet the effects of them on condemned murderers are not always identical. In some of the men death has been almost instantaneous, without any charring or burning of the body; in others, the flesh has been considerably burned before life was pronounced to be extinct. It is impossible to say, simply from the examination of a person who has received a strong electric current, how strong the current was which passed through him. It may have been weak, and still have caused extensive burns, or it may have been fatal without leaving external physical traces. It is certain that the victim of an accident rarely receives the full strength of a current of high potential. The contact is usually imperfect and only momentary, and the larger part of the effects of the electricity are dissipated without coming in contact with the body at all. Clinical observation shows that the greater number of persons who develop neurasthenia or hysteria as the result of accidents with electric wires have not been seriously injured. There are sometimes burns of the clothing or of the hands, but these are rarely severe, and more often are entirely absent; and in many cases circumstantial evidence, or the evidence of witnesses, proves that the patient received no electric current at all.

That fright is a most important element for the occurrence of the traumatic neuroses appearing as sequelæ of electrical

accidents receives additional proof from the almost universal agreement of opinion that such accidents are most frequently followed by the symptoms of hysteria, the fright neurosis *par excellence*. Neurasthenia, however, is sometimes the result of real or supposed injury from electrical currents. Knapp reports such a case:

> A coachman, forty-two years of age, strong, healthy, courageous, and not at all nervous, was driving a team of horses which fouled a live wire and were thrown. He got out and freed the horses, but probably received no physical shock. He was very much frightened, though he was not thrown down himself, and walked home. After the accident he slept badly; was nervous and apprehensive. There was a marked tremor of head and hands, loss of sexual desire, pain in back, exaggeration of knee jerks, and rapid heart action. After a duration of three months these symptoms entirely disappeared, and the man became as well as ever. They were symptoms of essentially neurasthenic character, and, as such, are not of the most common type observed after electrical accidents.

In gaining new causes, the traumatic neuroses have not lost old ones. Any one of them may result from a fall from a horse, from a carriage, or from slippery steps, or falls of any kind in which the person is frightened and he is hurt or his back is twisted. They very frequently follow falls in which the victim strikes on the head, on the back, or on the buttocks. Blows of any kind, but especially such as fall upon the head or the back, are often followed by functional nervous symptoms.

Meteorological disturbances, and especially lightning, are frequently the cause of nervous manifestations in persons of a neurotic temperament, and sometimes seem to be the only ætiological factor for well-marked neuroses. What has been said in regard to the place occupied by powerful electric currents among the exciting causes of the traumatic neuroses may, with certain modifications, be repeated for lightning. Like high potential currents, it may instantly kill, or cause extensive

burns. But the persons who, after lightning strokes, develop any of the traumatic neuroses, of which in this connection hysteria is the most frequent, usually bear no traces of physical injury and probably have not been struck at all.

Seismic phenomena are associated with the two factors—physical vibration or violence, and fright—most essential for the occurrence of the traumatic neuroses, and they are frequently followed by symptoms of functional nervous disease.

Charcot observed typical cases of hysteria after the earthquake in Nice. The earthquake in Charleston, S. C., and vicinity, in 1886, was followed by many cases of functional nervous disorder.

Porcher quotes as follows the report which a physician of Camden, S. C., made to the State Board of Health one month after the earthquake shocks of August 31, 1886:

"They" (the earthquake shocks) "at first naturally created much consternation among our population, and have undoubtedly had a very deleterious effect upon sick and feeble persons, being followed by much nervous prostration and other unpleasant symptoms. Even upon well, robust people their effects have been striking in some instances. Some have described their sensations as similar to those experienced after a shock from an electric battery; others have experienced a very marked feeling of debility in their lower extremities; others have had vertigo, nausea, etc. Some, again, who were not affected by these unpleasant symptoms in the beginning are now troubled by them."

From the various medical reports of these earthquake shocks, it appears that most of the nervous symptoms which developed as a result of them were neurasthenic in character.

Hughes observed, after the St. Louis cyclone of 1896, "cases of paræsthesia, hyperæsthesia, analgesia and hysteroidal shock, neurasthenia and some of the so-called traumatic neuro-

ses, and 'railway-spine' symptoms, such as follow the perceptibly uninjured after railway accidents."

As has been said, after sieges, battles, conflagrations, similar symptoms are of common occurrence.

In determining the causation of the traumatic neuroses there are several considerations of importance in addition to the character of the accident.

Among the factors active in the production of general nervous diseases, an important place is occupied by predisposition, either hereditary, or acquired through excesses of any kind. In neurasthenia and hysteria originating from causes other than trauma, it may often be discovered that previously to the appearance of symptoms the resisting powers of the nervous system has become enfeebled through various causes. When these disorders result from traumatic influences, predisposition to nervous disease, either hereditary or acquired, is often difficult to prove. Traumatic neurasthenia frequently appears in persons previously healthy and active, and it is often impossible to discover any predisposition in persons who develop hysteria after trauma. The question of predisposition is, however, very cursorily treated in many reported cases of this character, although it is of the highest importance, both for scientific proofs and medico-legal purposes, to know whether organic disease or an enfeebled nervous system had preëxisted. It should make a great difference in a verdict if an injury had merely caused an outbreak in symptoms which had previously existed, though latent.

Occupation and mode of life exert a certain influence on the occurrence of these disorders. Railway employees, with the exception of locomotive engineers and railway postal clerks, are less prone than passengers to develop hysteria or neurasthenia after accidents. This is explained in part by the fact that the employee, having become more or less accustomed to

the ordinary mishaps incident upon railway travel, can bear with a certain degree of composure any accident in which he is not severely injured. Also, when an employee is slightly injured, his one thought is to get well and to return to work as soon as possible. For obvious reasons it is bad policy for him to bring suit against the company—a limitation which acts to his advantage, for by means of it his convalescence is not delayed by the anxiety and vexations which commonly attend suits for damages. Engineers, as distinguished from other railway employees, do not share in the comparative immunity from functional nervous disorders, an exception which may be in part explained by the constant nervous strain to which their responsibility exposes them.

Railway postal clerks are frequently victims of neurasthenia. The character of their work demands that they be on their feet, subject to the constant vibration, swaying, and jolting of the rapidly moving train. At the same time, the sorting of the mail requires close attention and concentrated mental effort. They are consequently exposed to the two most fertile causes of nervous exhaustion.

Contrary to the generally received impression, the traumatic neuroses are relatively more frequent among the poorer classes. As a reason for this it may be urged that laboring men are more constantly exposed to injury. But, aside from this unquestionable fact, it seems as though there were something in the mode of life of the poorer classes which rendered them particularly susceptible to evil results from slight traumata. Probably faulty hygiene furnishes the predisposition, of which the existence is so often hard to prove. Men are more frequently affected than women, a difference which can be explained by the very much greater frequency with which men are exposed to accidents of all kinds.

The physical condition of the person at the time of the

accident exerts a very important influence. The fact is well established that those who are asleep, or under the influence of liquor, experience the fewest serious results, both physical and mental, from railway and allied accidents. On the other hand, persons suffering from chronic disease of any kind, and particularly disease of the nervous system, are ill fitted to sustain physical or mental shocks. The original disease may be made worse, or to it may be added symptoms of subjective and functional character.

Thus in an unpublished case of Peterson's, which he kindly permits me to quote, a lady had suffered for some time from amyotrophic lateral sclerosis, although the disease had only annoyed her by interfering with the finer movements of the hands. One day, while sitting in a restaurant, she was struck on the head by a revolving fan which had become detached from its support on the ceiling. As a result of this there supervened a condition of extreme nervousness, irritability, sleeplessness, and despondency—symptoms previously absent. Also tremor, of which she had never complained before, became very marked after any exertion or fatigue.

The influence of suggestion by physicians is frequently traceable in the causation both of traumatic neurasthenia and of traumatic hysteria. In many cases it seems as though these disorders owe their appearance, in large part at least, to the fact that the patients have been told by physicians that they may some day have trouble with the spinal cord as a result of the accident. The examples of the bad effects of such ill-advised statements are numerous.

Thus, a gentleman was slightly jarred by the sudden stopping of a sleeping car, but was not thrown from his berth and felt no immediate ill effects of the mishap. On general principles, however, the next morning he consulted a physician, who told him that his spine had been concussed and that he might eventually have serious symptoms as a result. A few days afterwards the patient began to have pain in the back, and finally became a confirmed neurasthenic.

Similarly, a park policeman was thrown from the back platform

of a horse car, striking upon the pavement with his face. He thought his jaw was broken, and immediately sought medical advice. The doctor said that there was no serious injury about the head, but that he feared trouble from the spinal cord. A few months after this the patient came to the Vanderbilt Clinic, presenting a pronounced picture of traumatic hysteria.

Intimately allied with suggestion as a causative factor of the traumatic neuroses, and by some considered the most important of all, is the question of litigation. In how far and in what way the hope of compensation for personal injuries received in accidents influences the development of functional nervous symptoms will be considered in succeeding pages. In speaking here of ætiology, it is sufficient to say that there is a far greater probability that functional nervous disturbances will appear, or, if they have already appeared, that they will be made worse in any case which becomes the subject of medico-legal inquiry. Upon this point all authorities agree. There are still many differences of opinion in regard to the pathology, symptomatology, and prognosis of the traumatic neuroses, but the belief in litigation as a very potent causative factor is universal. It surrounds the patient with the influences from which he should be free, and prevents him from pursuing the course of treatment best suited to permit a return of health and of self-control. It is the physician's duty to do all in his power to favor an adjustment of claim, rather than have his patient run the risk of becoming a chronic invalid through the vexations and annoyances which are invariably associated with suits for damages.

Symptoms

The symptoms of the traumatic neuroses are essentially those of traumatic neurasthenia and traumatic hysteria. Inasmuch, however, as these two disorders often occur together in

accident cases, and since there has been so much discussion relative to the clinical value of their manifestations, it will be necessary to make some preliminary observations on the general character of the symptoms, although they will be fully described in later chapters. Many of them are purely subjective. In private practice, when the physician has to deal with subjective symptoms, he is usually safe in believing that they really are causes of annoyance or suffering to the patient, although it may be evident that they are to a certain extent exaggerated. When the question of litigation enters, however, the case becomes entirely different; then, instead of looking for the customary exaggeration of chronic invalidism, the examiner must be on his guard against malingering. To ensure correct results from the examination of litigation cases it is necessary that the physician be cognizant of fraud, familiar with the methods of diagnosis of nervous disease, and aware that the functional disorders may be serious or incurable affections. The examiner, whether he be an expert called by a claimant or be employed by a corporation, should undertake the examination without prejudice and without bias. His position is always delicate and often difficult. It would be superfluous to dwell here upon the moral duties of an examiner in litigation cases. That is a question in ethics rather than in medicine. The physician owes it to his client to exert every effort in his behalf. He owes it to himself to give an opinion based solely upon his own convictions. By whomsoever employed, he will rarely err if he keeps constantly in mind that his function is scientific, and that he is not to be influenced by the effect his opinion will have upon the financial aspect of the case.

It is often impossible to decide, from a single examination, what the merits of the case really are, and in doubtful cases an opinion should not be given until after several have

been made. In general practice the physician often finds himself unable to determine the exact nature of a disease until he has observed it during a considerable period of time. How, then, in any accident case in which the symptoms are almost entirely subjective, and in which exist the strongest motives for exaggeration or deceit, can he hope to be always correct in diagnosis and prognosis from examining the patient once only? In Germany, laboring men who are the victims of accidents can be placed in the hospital until the exact nature of the disorder becomes plain. Such a procedure is impossible in America. With us, only those persons go to the hospital who are seriously injured, and about whose injuries there could be no question of doubt. The others must, for the greater portion of the time, be free from medical observation. This fact makes it imperative that the physician be cautious about too unreservedly accepting the patient's statements, or about placing too high a value upon such symptoms as are purely subjective in character. The history of how injuries were received, or the accounts of physical conditions, are often unreliable when told by persons unfamiliar with the significance of symptoms or with the general workings of pathology. To a certain extent in general medical cases, and to a greater extent in functional nervous troubles, when there is no thought of litigation, the physician must be guided by the results of his own examination rather than by the description of suffering and discomfort given by the patient. In cases in which the strongest motives for exaggeration and deceit are present, how much more conservative must he be about too fully accepting statements of personal ill health which permit of no objective proof!

The history may be unreliable even when the patient believes it to be true. By constantly rehearsing in his mind the details of the accident as he remembers them, or as they have been told him by witnesses, by the sympathetic inquiries and

solicitations of friends or interested persons, the patient may come to believe that he knows for himself what has in reality been told him or suggested to him by others.

It is, of course, not meant that the examiner should necessarily disbelieve everything that a plaintiff tells him; but the history of the traumatic neuroses has shown, and the nature of the cases demands, that the statements of patients in litigation cases should be subjected to some corroborative proof before they are unreservedly accepted as true. The proof of the reality of the painful sensations from which the patient says he suffers may often be found in his manner and general bearing. His whole appearance may indicate ill health, although there is no one objective symptom to prove its existence. It is then that the physician must be guided by his own experience in nervous disease. But when there are no physical traces left by the suffering through which a plaintiff says he has gone, or to which he asserts he is still subject, there is no way in which it may be decided as to the unreality of the symptoms, except in so far as repeated medical examinations may show their existence to be improbable, or in so far as information from outside sources may prove that the claimant only conducts himself like a sick man when he is being watched. Thus must be determined the truth concerning such symptoms as sleeplessness, spontaneous pain, nervousness, lack of interest, and general fatigue. Some of the symptoms commonly called objective are also, in large part at least, subjective.

Excitability of the heart and rapid pulse, although objective signs, are not in themselves necessarily evidences of disease.

Palpitation of the heart is particularly frequent in persons who use to excess tea, alcohol, tobacco, or other stimulants.

In most people the heart becomes more rapid at the time

of a medical examination, and in those of a nervous temperament the heart action may become tumultuous from very slight excitement. The same observation holds good for anomalies of respiration.

Anæsthesia is the symptom about the diagnostic value of which there has been the greatest contention.

It has been urged against it that the symptom is subjective, inasmuch as, with the exception of tests which are painful, the physician gains his information from the patient, who, through inattention or through the influence of suggestion, or when actuated by ulterior motives, may make answers which are not true, and thus be credited with symptoms which do not exist. Yet, although anæsthesia is a subjective symptom in the sense that the coöperation of the patient is often necessary for the establishment of its existence, it is converted into an objective symptom when the replies of the person under examination show it to be in accord with the other disease evidences which may be present, and in agreement with the loss of sensibility recognized as characteristic of definite clinical types. There is no question as to the objectivity of anæsthesia as observed in spinal-cord lesions or in classical cases of hysteria. When, however, the loss of sensibility is slight, occurring in small areas with indefinite boundary lines, or in the form of a general blunting of the sense of touch or of pain, its diagnostic value is not great and can easily be overestimated.

Acuteness of cutaneous sensibility is subject to wide variations, which, clinically at least, must be regarded as within normal limits. It varies with age, sex, and race, with different individuals under similar circumstances, and with the same individual under the changing effects of weather, time of day, fatigue, and similar temporary conditions. Certain diseases, such as general paresis and chronic intoxications, notably alcoholism, in which pronounced anæsthesia is not usually present,

are often accompanied by a certain degree of blunting of sensibility. When due allowance is made for these variations anæsthesia remains as a symptom of positive and often of pathognomonic usefulness. The physician who is familiar with the facts will appreciate the appropriate value of the symptom, and will not be apt to ascribe to slight impairment of sensibility an unwarranted diagnostic importance.

Pain or hyperæsthesia is subjective except in so far as it leaves unmistakable evidences in disturbances of nutrition. A test usually considered as of considerable value for the determination of the genuineness of pain and for converting it into an objective symptom has been named, after its original describer, Mannkopff, although its applicability in the traumatic neuroses was first pointed out by Rumpf. It consists in observing the pulse rate before, after, and during pressure upon an area alleged to be painful. If the pulse becomes more rapid while the pressure is being made, it is supposed to be proof that the pain is real and has reflexly caused the heart to beat more rapidly. The application of the test is illustrated by Rumpf in the following case:

A man, thirty-three years of age, fell from a roof. After the various early symptoms of injury had disappeared the patient still complained of great weakness and pain in the head, back, and left breast, which was much increased by any contact. The pain was entirely subjective, but was converted into an objective symptom by the Mannkopff test. To avoid confusing the increase of the pulse rate, due to fear or other psychic influences, with the increase caused by the perceptions of real pain, the pulse was first counted with the patient in the recumbent posture without any pressure being applied to the painful areas. The rate was: In the first quarter of a minute, 20; in the third quarter of a minute, 24; in the fifth quarter of a minute, 29; in the seventh quarter of a minute, 26; in the ninth quarter of a minute, 26; in the eleventh quarter of a minute, 25. Then firm pressure was applied to the alleged painful area in the back, and the pulse at once increased to 33 beats in the first quarter. On removing the pressure it sank again as follows: In the first quarter

of a minute, 32; in the third quarter of a minute, 30; in the fifth quarter of a minute, 28; in the seventh quarter of a minute, 27; in the ninth quarter of a minute, 25. In addition to an increase in rate, the pulse became smaller and at times irregular.

Rumpf regarded the Mannkopff test as of great value in the detection of simulation; but observations made by Strauss, in which the pulse waves were carefully recorded by means of the sphygmograph, have shown that, in many cases of unquestioned pain, pressure over the painful areas causes no increase in the heart's action; so that Strauss's conclusions, which accord, I think, with those of most clinicians, is that the Mannkopff symptom is not constant even in cases in which there is no reason to doubt the real existence of pain or hyperæsthesia. If the symptom is present, as it often is in traumatic lumbago, or as it may be in hysteria, it is a valuable aid in diagnosis. But if it is absent, we are by no means justified in concluding for that reason that the pain or hyperæsthesia is assumed. In making the test the pulse should be carefully counted for some little time before exerting the pressure, in order to eliminate as far as possible any acceleration which may be due to psychic influences. This test may be extremely painful. The Mannkopff test is used almost entirely for pain in the back. In addition to pain in the back, pain is complained of in the traumatic neuroses in many other parts. These are of great variety, but headache is the most constant.

The visual disturbances of the traumatic neuroses have been the subject of much controversy. There is no reason to doubt that in neurasthenia there is commonly an asthenopia which prevents any long-continued use of the eyes, and that some of the visual anomalies of hysteria, although very difficult to explain, are pathognomonic of that disease.

The chief contention among neurologists has been in regard to the diagnostic value of the results of perimetric ex-

aminations. The two conditions most commonly observed in such examination of cases of the traumatic neuroses have been the concentric contraction, without structural disorder of the eye, and the shifting or fatigue contraction of the visual fields (see Chapters II and III). The first of these conditions was originally described by von Graefe and the second by Förster.

In regard to both of them arises, as it must arise in regard to any functional symptom, the question, Do they occur in normal individuals, and do they occur in other diseases in a way to impair their diagnostic value for the traumatic neuroses?

The recent investigations of Koenig indicate very positively that concentric limitation of the visual fields is not found in persons with normal nervous systems. From the examination of 216 nonnervous cases and of 10 pathological cases Koenig concludes that it does not occur in healthy persons, that it may be the only symptom of hysteria, and that, when constant, it is typical of hysteria, even when the limitation is only slight in degree.

This ocular condition is observed in many of the disorders of the nervous system, of which the most prominent are tabes, dementia paralytica, epilepsy, trigeminal neuralgia, and alcoholism. But occurring in this way, even if the routine examination of the eye fails to disclose any other visual defects, the associated symptoms will prevent the contraction of the visual field being ascribed to hysteria alone. A consideration of these facts, together with the great improbability of the simulation of the condition, seems to justify the acceptance of contraction of the visual field, when there are no morbid alterations of eye structure, and when symptoms of other forms of functional or organic disease are absent, as a pathognomonic symptom of hysteria.

The significance of the shifting type of contraction is not

so well established. Peters has found it present in many persons presumably healthy. Koenig, however, and still more recently Müller, incline to the view that, if at all pronounced, this symptom is certainly indicative of retinal fatigue; and that although it may occur in healthy men, in them it is less constant and less pronounced. As Müller says, when it is constantly present at repeated examinations in persons supposedly healthy, it is time to look for nervous symptoms. Vertigo, a symptom commonly complained of in both hysteria and neurasthenia, has been described on page 20.

The tendon reflexes, and especially the knee jerk, become the subject of discussion in most accident cases. Loss of knee jerk does not occur as the result of functional disease. Exaggeration, on the other hand, is the rule. Unless associated with other symptoms, slight exaggeration cannot be regarded as of any particular pathological significance; when associated with ankle clonus it usually, though not invariably, indicates organic disease; when associated with other symptoms it lends confirmatory evidence as to the existence of morbid functional states.

Vomiting or spitting up of blood-stained fluid immediately after the accident occurs in a certain proportion of the cases. While alarming in appearance, this symptom has no particular significance unless it is the result of injury to the thoracic contents or to preëxisting disease of the lungs. It is observed in neurasthenia, but more commonly in hysteria. The discharge comes from the mucous membrane of the mouth and throat, and, as Strümpell has shown, is characterized by the small number of red blood cells and by the free mixture of mucous epithelium and bacteria which are collected in the buccal and pharyngeal mucous membrane.

The other symptoms of the traumatic neuroses will be so fully described in succeeding pages that they require no men-

tion here. It is to be remembered, however, that it is not by the consideration of any one symptom that a complete comprehension of any case of functional nervous disease following an accident is to be obtained. The case must be looked at in its clinical entirety. Attention must be given to the kind of accident and the extent of physical injury as well as to motives for simulation or exaggeration. It is not sufficient to observe objective symptoms without inquiring whether they could not have antedated the accident. It is not possible to form a conclusion from one subjective symptom.

By refusing to express an opinion without having looked at the case in every aspect, the physician will rarely find himself in error.

CHAPTER II

TRAUMATIC NEURASTHENIA

Ætiology—Pathology—Symptoms: Mental, Motor, Sensory (Traumatic Lumbago), Special Senses, Reflexes, Vascular and Digestive Disturbances—Diagnosis—Prognosis

NEURASTHENIA is a condition of irritable weakness of the nervous centers, as a result of which they become less tolerant of external impressions and of the effects of fatigue. It is a condition rather than a disease; but although it most commonly occurs without there being any structural lesion of important organs, its clinical picture is so typical and its symptoms are so well marked that the disorder, when pronounced, is usually spoken of as though it were a disease. The clinical manifestations of neurasthenia are the results of a loss of potential nervous energy. From whatever cause it may have been induced, the patient presents the symptoms of a diminished power of resistance to the influences of fatigue and a hyperexcitability of the nervous centers. He is quickly exhausted by mental and physical exertion, and reacts too strongly to all forms of peripheral irritation. The neurasthenic is incapable of prolonged mental or physical work, and such efforts are quickly followed by confusion and fatigue. All the receptive centers are hypersensitive, so that there is a morbid increase of response to all peripheral stimuli, and the individual becomes intolerant of such slight irritations as pass in health, if not unobserved, at least without being followed by sensations which are painful.

Neurasthenia may exist in varying degrees. Temporary

nervous exhaustion naturally follows excessive work of any kind. Anyone who has become overfatigued by mental strain or by excessive work will present many of the symptoms of neurasthenia; but in the majority of persons the evidences of exhaustion which may be present at night, after a hard day's work, have vanished in the morning, when sleep has permitted a restoration of vigor to the nervous system. When the overwork is continued for too long a time, however, or when the rest is inadequate, the symptoms which at first were temporary may tend to become more and more permanent, and the tired man or woman, instead of presenting such effects of overwork as may be easily repaired, may become a clinical type of pronounced neurasthenia—a condition which requires more than ordinary rest for its cure. Under the name of nervous prostration neurasthenia has come to be a generally familiar affection. It is commonly spoken of as a disorder which is directly amenable to the will, in that a person suffering from it might be well if he could bring himself to believe that he were not ill. This is true in part only. The nervous exhaustion has to a great extent robbed the patient of the ability to control his own mental processes; but to restore the will power there must be a return of energy to the nervous centers, by the exhaustion of which it has been lost.

ÆTIOLOGY

Since the description of neurasthenia by Beard, the disorder has been generally recognized. It may complicate any of the chronic diseases; it frequently results from overwork, anxiety, and excess of any kind, and in some cases it develops without apparent cause. The fact that it may develop primarily as a result of injury and shock has only been recognized in recent years. When occurring in this way it is called trau-

matic neurasthenia, and differs only in slight particulars from neurasthenia due to other causes. The back injury and the effects of litigation have given certain peculiarities to traumatic neurasthenia, but the underlying conditions and essential symptoms are the same in all varieties of the disorder.

Traumatic neurasthenia is by far the most frequent functional nervous affection which occurs as a result of accident. Its causation has already been described in a general way. The disorder is infrequent in the old or in the young, most commonly occurring in the active periods of life. It has been reported as occurring in children. Vibert records two cases of " traumatic neurosis " observed in children aged respectively three and a half and five years. From Vibert's description these cases seem to present the symptoms of psychic epilepsy rather than those of neurasthenia; and the functional nervous disturbances following accidents, which I myself have observed in children, have been suggestive of a more profound affection of the nervous system than neurasthenia. Men are much more frequently affected than women. Nervous predisposition, although demonstrable in some cases, does not appear to be an essential factor in the development of the disorder.

The accident is the most important causative factor of traumatic neurasthenia. The fact that it very frequently follows accidents on railways gained for it the original name of railway spine.

Although it also frequently follows any of the accidents in which the factors of fright and physical commotion are prominent, there is something about a railway accident which seems particularly well adapted to call into action the peculiar chain of nervous symptoms which are known as neurasthenic.

The reasons for this cannot altogether be explained by the medico-legal aspects of railway accidents, although traumatic neurasthenia has become more frequent with the constant in-

crease in litigation. It may be in part explained by physical reasons. Railway collisions and derailments usually put unusual strain upon the back, and pain in the back is generally an early and the most constant symptom of the disorder. In the course of nontraumatic neurasthenia, also, the backache is a prominent symptom. It may be that a railway accident, in causing strain of the spinal ligaments and muscles, and consequently pain in the back, furnishes at the outset one of the most characteristic symptoms of neurasthenia, to which general nervous shock and subsequent events add others. The fact remains that most railway accidents do cause back strain through the violent shaking of the whole body, and traumatic neurasthenia usually begins with pain in the back. In severe collisions the victim may be thrown backward and forward many times before the car ultimately comes to rest. The legs or the trunk may be caught between the seats or other objects and held there for a long time, and the free portions of the body may sustain violent wrenches and twists, which are transmitted to the spine. Even when the passenger is merely thrown from his seat to the floor, or pitched against the seat in front of him, the suddenness of the force and the involuntary resistance which is offered by the muscles of the trunk and back put considerable strain upon the spinal ligaments and muscles. The sudden stopping or starting of a train may cause the spine to be considerably wrenched. Thus some cases of back strain occur when there has been no serious accident. In addition to the local injury, railway catastrophes usually cause the most severe mental shock that can be imagined. An association of these two factors, the physical and the psychical, is the commonest cause of traumatic neurasthenia.

Can shock alone, when the back is not injured, cause traumatic neurasthenia? In a future chapter it will be shown that pure nervous shock without physical injury may cause a vari-

ety of mental symptoms; but as a cause of neurasthenia it is unusual. If a person is very much frightened by any accident, he may in future be morbidly nervous about reëxposing himself to that especial variety of danger. A victim of a severe railway collision may never again be able to feel at ease while on the railway, although he may never have been physically injured in traveling. It is a fact of common experience that runaway accidents, even when no injury is received, may cause a timidity about driving which is never overcome. But the morbid fears that result from accidents in which no physical injury has been received are, in the majority of cases, systematized, and relate only to the particular form of accident by which the person has been frightened.

When general neurasthenic symptoms develop from fright alone, it is probable, in most cases at least, that the person was in a nervous condition at the time of the accident, or that some injury was received which passed unobserved.

However, when the nervous shock is severe, neurasthenia sometimes results, although the physical injury be but trifling. Schaefer relates the case of a locomotive engineer in whom the fear of an impending collision and overexertion in stopping the train produced typical symptoms. In the following case, also, the physical element must have been unimportant.

A Russian Hebrew, aged thirty-nine, came to the Vanderbilt Clinic on June 3, 1896. He said that until the preceding May he had always been healthy, and came of a long-lived and healthy family. He had no bad habits, was married, and had several healthy children. Denied venereal disease. He is a furrier by trade. It is not probable, however, that this occupation had predisposed him, by chronic mercurial poisoning, to nervous disease. Although some workers in furs become poisoned by inhaling the mercury with which furs are prepared, such a result only occurs when the furs are heated and the mercury is volatilized. Our patient had been exposed to no such danger, and denied, furthermore, all symptoms of hydrargyrism. This man had no motive for malingering. He has never had any

thought of or cause for bringing an action; he is a member of no lodge or society. When not at work, the only money he gets is drawn from what little he has saved or what is earned by his wife. Since an accident which must have, at best, been attended with slight physical commotion, he has been totally unable to work. I saw the man in February, 1897—nearly one year after the accident—when he told the following story and presented the following symptoms:

At the time of a fire at Bleecker Street and Broadway he was leaning out of a window watching the disturbance. His head and shoulders were out of the window, the abdomen resting on the window sill and the feet on the floor.

As a result of the explosion of two boilers which were in the burning structure opposite, the building in which he was at the time was jarred, but not enough to break any glass or to cause the plastering to fall from the walls. The man was very much frightened, and he felt the concussion through his whole body, but he was not dislodged from his place. Upon getting out of the window frame he fell down, but quickly picked himself up, and was busy and constantly on his feet for the rest of the day. That evening he went to bed and stayed there for two weeks, feeling weak and sick. Soon after the accident the patient began to have pain in the back and has had it ever since. There was no vomiting, no loss of sensation, no trouble with the bowels or rectum, and no paralysis. When well enough to leave his bed he tried to resume work again, but could not do so on account of the trembling of the fingers and the pain in the back.

He is a man of fairly good color, but has the anxious, tired look of neurasthenia. The back is painful in its whole extent—from the cervical to the lumbar region. Pressure over the spinous processes is painful, but does not cause the heart to beat more rapidly. The skin over the whole back is hypersensitive. There are no evidences of organic disease. The pupils are equal and react readily to light. The optic nerves are normal. There is a slight peripheral limitation of the visual fields, although it is difficult of determination. There is nowhere any loss of sensation. There is marked tremor of the face and in the hands, constant, but becoming intensified by movement. The gait is slow and uncertain; the knee jerks are normal. Appetite good; bowels regular. During his illness the patient has lost eighteen pounds.

In this case there was some physical shaking, although it must have been very slight.

In some rare cases there seems to have been no injury at all. Thus, in a case of Page's:

Neurasthenia induced by fright. Previous anæmia. A lady, aged twenty-four, was in a collision which took place at night without the slightest warning. The baggage fell all over the carriage, and her husband was thrown against her, but she herself was neither thrown from her seat nor injured in any single part. As soon as she got out she was much alarmed at seeing a carriage had been smashed to pieces, and then she watched a man being rescued from the *débris* in which he was buried. She went on her journey, and the next day felt, to use her own words, as though she had passed through something terrible; and from that time onward she became sleepless, lost her appetite and strength, suffered from pain in the back of the head and at several spots down the spine, and was quite upset by any attempts at household work or by reading and writing. Thinking she had not been hurt, her friends urged her to do as much as possible, and not to give way, but she steadily got worse rather than better; and although not in the least hysterical, it was not until proper treatment was begun that improvement set in. The case was complicated by previous dysmenorrhœa and anæmia, both of which were increased for a time by the accident.

These cases show that neurasthenia may result from fright when the physical injury has been insignificant. It may, on the other hand, follow trauma by which the patients were hurt before they knew that they were in danger, and so escaped the fear of impending injury. These latter cases are unusual, for shaking of the whole body or injury to the back rarely occurs without there being at the same time some terrifying factors. Few participators in railroad accidents escape without being very much frightened; and when physical injuries are received in any way there is usually a period, even though it be momentary, of antecedent alarm. The fright, however, is often trifling, as when caused by the sudden starting of a train, or a fall on the sidewalk or from the steps of a carriage. As is well known, neurasthenia may follow such accidents.

The gravity and duration of the symptoms are not always

in proportion to the severity of the accident, although in general the more severe the accident the more rebellious the resulting neurosis.

As neurasthenia is a condition, it may exist in very widely varying degrees of severity. Many persons find themselves after an accident more nervous and more easily fatigued than they were before; but the impairment of general health or strength may be so slight that they do not complain, and, by continuing their work and thus keeping their attention away from their own troubles, they institute, without knowing it, the very best treatment they could have. It is in such cases as these that the anxiety attendant upon bringing claims or litigation may render very serious a disorder which otherwise would have been trivial.

Contrasted with the frequency with which neurasthenia remains a mild affection is the fact that it may be very severe, and render its victim totally incapacitated for work.

In recognizing that many cases are transitory and easily repaired by proper means, sight should not be lost of the fact that the disorder may be intractable, or even incurable.

Pathology

Little is known of the pathology of neurasthenia. It rarely kills, and there are no recorded autopsies in which lesions were found adequate to account for the neurasthenic symptoms. In the autopsies which have been made on persons who died during the course of the disorder the nervous system has not been examined with sufficient care to discover any morbid appearances in the ganglion cells. Hodge and, more recently, Lugaro have shown, however, that visible alterations of form and structure occur in the ganglion cells of animals as a result of fatigue. And since fatigue is the most prominent fea-

ture in the clinical picture of neurasthenia, it is to be inferred that the pathology of the disorder is to be sought for in nutritional disturbances of the ganglion cells. It would be useless to speculate here as to how these changes are brought about, or what their essential characteristics are. It is enough to say that it seems probable that to explain the disturbances of function there are structural changes which may eventually be seen and to a certain extent understood; but until our knowledge regarding the pathology of neurasthenia is more exact and full, it must continue to be classed with the functional diseases.

Symptoms

In describing the symptoms of traumatic neurasthenia it must be understood that they can only be regarded as belonging to that category when organic injury of the nervous system can be excluded.

The mode of onset of the symptoms is subject to many variations. The character of the accident, and the influences by which the patient is surrounded, both before and after its occurrence, cause great differences in their development. Persons who are asleep when the accident occurs are spared much of the shock and are less liable to injury. The same is true for persons who are drunk. The two conditions, sleep and intoxication, render the development of neurasthenia less probable.

Immediately after the accident there may be considerable prostration and shock. The patient does not lose consciousness, but is pale, dizzy, tremulous, and nauseated, with cold skin and rapid pulse. If there is head injury, and sometimes when the head has not been injured, consciousness may be lost for a variable length of time. If the body has been severely bruised or lacerated, the earliest symptoms may be entirely

surgical. The latent period, including the time between the accident and the first appearance of neurasthenic symptoms, varies in duration. It is usually only a few days, though it may last for several weeks.

The ultimate development of neurasthenic symptoms is to a great extent modified by those influences which surround the patient immediately after the accident. Those patients do best who can put themselves immediately in a physician's hands, and who can declare themselves independent of claims for damages. Litigation, while it may help the pocket, does so by making large demands on health. By avoiding it, neurasthenic symptoms, instead of becoming worse, with a tendency to chronicity, may oftentimes be entirely avoided.

The symptoms of traumatic neurasthenia are various, and will require examination in detail. When the disorder dates from the time of a railway disaster, the story is often somewhat as follows: A previously healthy man while riding in a railway car is suddenly startled by the screech of the locomotive, and almost immediately afterwards experiences a sudden physical shock, is thrown violently from his seat to the floor, where he is flung backward and forward, receiving blows upon the head and back. Or he may have been caught between two seats and his back twisted or wrenched. When the car has finally come to rest he finds himself very much dazed and confused, though usually he does not suffer much pain. He frees himself, or is extricated from the wreck, and may assist in rescuing others, or may at once go to some place of shelter. If there are houses near by, he usually walks to one of them unless he has been severely wounded. If the accident has occurred in the open country, away from any habitations, he may be exposed to the weather until assistance arrives. He is usually nauseated, and may vomit, and sleeps badly the following night. In a day or two he begins to have pain in

the back. From that time on occur, in varying degrees, the symptoms of neurasthenia. The history will, of course, vary with the kind of accident, but the salient features of it are usually that the patient was at first more frightened than hurt, and that the symptoms appeared only some time after the accident.

Mental Symptoms.—The mental symptoms constitute the most characteristic feature of the disease. In the milder forms, the fretful, querulous invalid may be regarded as hypochondriacal, and as a bore who thinks only of himself, but not as out of his mind. When the symptoms are developed in their highest degree they bring the patient dangerously near the border land of insanity. The neurasthenic is irritable, introspective, depressed, and inattentive; he quickly tires of any prolonged effort; he is emotional and fearful, and does not sleep well. A previously strong, healthy, and active man may, after some trivial accident, become entirely changed. Little things which previously passed unnoticed become matters of annoyance. Slamming of doors, loud talking, slight jars and noises, become to him unbearable. An unexpected sound may cause his heart to palpitate very violently. Strong light hurts his eyes. He likes best to sit or lie in a darkened room, removed from all causes which may irritate him. Altruism is buried under the load of preoccupation with self. Some patients take to their beds almost immediately after the accident and stay there for weeks or months. In a case which I saw in consultation with Dr. A. W. Warden, in reference to a claim against the (then) Metropolitan Traction Company, the patient remained in bed for many months.

She was a single woman, thirty-eight years of age, of neurotic temperament, who previously to the accident had always been reasonably well. On May 13, 1896, an open car in which she was sitting was struck by a cable car. The jar was considerable and the woman was thrown forcibly against the back of the seat. She was

very much frightened, but was not thrown from the seat, nor did she experience any severe pain at the time. She felt sick and faint, but was able to walk. She went immediately to bed, where she stayed for five months, a prey to the customary neurasthenic symptoms. Pain in the back, difficulty in urinating (partly due to a pre-existing retroversion of the uterus), difficulty in breathing, nausea, vomiting, and flushings of the face were, at one time or another, prominent symptoms.

Our examination in August failed to detect any evidences of organic nervous injury. There was no anæsthesia, no limitation of the visual field, no paralysis, no disturbances of the functions of the bowels or bladder, except such as might be explained by the local pelvic conditions. The patient was depressed, tremulous, and anxious. The back was hypersensitive in its whole extent. Pressure on the vertebral spines in the thoracic and lower cervical regions caused very decided expressions of pain. The patient could walk, but was afraid to do so. The gait was slow, hesitating, and uncertain. She complained of dizziness, but could stand perfectly well with closed eyes. The after-history is interesting. From the time of the accident until October—five months—the patient was in bed. In October she began to walk a little about the house, and in November her claim was satisfactorily adjusted. But after receiving the money the improvement, instead of continuing, stopped, and in February, 1897, Dr. Warden told me that she was in bed again, worse than she ever had been.

The neurasthenic is fretful, fault-finding, and peevish. His chief, and too often his sole, interest is in matters directly connected with his own condition. He observes all his symptoms, and from superficial reading of medical books he often thinks himself the victim of innumerable diseases. Solicitude for family and business is made to yield the foremost place in his thoughts to the anxiety about his own health.

Introspection is almost constant, and in certain varieties of neurasthenia, in which the manifestations are chiefly mental, constitutes the chief symptom. The patient notes and speculates upon the variations in his feelings. He talks the most willingly to those who will listen the most patiently to his complaints. He constantly visits the physician, though he is rarely

satisfied with one, but goes from one doctor to another. Anyone who, even when in fairly good health, is persistently on the alert from some deviation from the normal of his own physiology will rarely fail to detect it. But the healthy mind can usually satisfactorily account for and dismiss without thought such trivial symptoms as a slight palpitation of the heart or an irregularity of the digestive apparatus. The neurasthenic thinks over such symptoms, and, in accordance with the well-known psychological law that attention intensifies sensations, they are made worse. As a consequence, he becomes depressed. Neurasthenic depression is different from the depression of melancholia, although these two conditions may occur together. In neurasthenia the depression is in regard to the patient's own prospects and chances of recovery. Unlike the melancholiac, the neurasthenic rarely loses hope of ultimate restoration to health, but he is discouraged at its long postponement. Every new symptom, or every aggravation of a preëxisting symptom, adds to his low spirits. Yet he rarely becomes absolutely despairing. If suicide occurs, it is probably the result of something more than traumatic neurasthenia in a person previously well.

Added to these symptoms are often many others, the most characteristic of which is mental fatigue. The neurasthenic mind usually becomes quickly tired, and is incapable of prolonged effort. The patient may find it almost impossible to keep his attention for any length of time on any subject not directly connected with his own state of health. Many patients are entirely unable to keep at work. When a neurasthenic begins to read, he soon lays the book aside. If he plays a game, his interest is not in it and he quickly wishes to stop. Besides the lack of interest in any mental effort, protracted intellectual work is followed by an increase of the various subjective symptoms of which neurasthenics so frequently complain.

Through the combined influences of fear, lack of interest, and lack of attention, the will power undergoes more or less impairment. The neurasthenic can ordinarily make the simplest decisions only after much hesitation and doubt. Important questions to be acted upon excite him, and he frequently declares himself incapable of deciding them. There is, however, no real impairment of intellectual capacity, except such as may be accounted for by hesitancy and lack of close attention. The memory remains good, though the patient often believes the contrary. There is little change in the quality of the reasoning powers, when the patient can be brought to use them.

Fear is a conspicuous symptom, but it rarely takes systematized forms, except in so far as all circumstances which relate to the accident are held in dread. On the contrary, the condition is one of timidity and shrinking rather than of active fear. The patient is afraid to go out, lest he become dizzy; he is afraid to meet people, lest they tell him how badly he is looking; he is afraid of anything which causes his mind to revert to the accident in which he was injured. If he were injured on a railway, he is very unwilling to undertake a railway journey; if in an elevator, he prefers the safer method of walking upstairs. It is important to remember that the fears of traumatic neurasthenia are rarely systematized. This is especially to be borne in mind in view of the recent work of Janet. Under the term psychasthenia Janet has grouped an important set of cases (long well known), in which, along with well-marked neurasthenic symptoms, are present imperative conceptions. These imperative conceptions are of great variety, but fear is prominent among them. There is fear of contamination (mysophobia), fear of closed places (claustrophobia), fear of making decisions (*folie du doute*). Such cases are frequently classed as neurasthenic. They occur in persons with well-marked neurotic taint, almost always hered-

itary. Rarely, if ever, are they called into play by physical injury, a fact important in considering traumatic neurasthenia. These imperative conceptions are quite different from the impulses, epileptic in nature, which follow certain head injuries, and which force the patient to sudden purposeless acts. The following case, though somewhat out of place here, may be cited to show the difference:

Dr. Starr has kindly communicated to me the history, that of a young man, twenty years of age, previously strong, active, and healthy, who, during a football game, was thrown on his head. He was momentarily stunned, but continued playing. The team of which he was an important member was badly beaten, and its defeat was, in part at least, explained by the fact that the injured man made mistakes in the signals and so broke up the whole system of team play. The young man himself remembered nothing of the latter part of the game. After this injury he slept badly, was nervous and irritable; a few weeks later he suddenly disappeared, and nothing was heard of him for four days. When he returned to his family he could give no clear account of his absence. He said that he had felt as though some one were after him, and as though it were necessary for him to get out of the way. He was seen at this time by Dr. Starr, who found him quiet, composed, with the ordinary symptoms of neurasthenia. A short time after this the patient had an attack of great excitement, during which he felt himself obliged to go out and walk, and felt like knocking down everyone he met in the street.

Since the football game, six years ago, this young and apparently strong man has led a life of invalidism. He has wandered about to various water cures without fixity of purpose or power of concentration. His conduct has been erratic, and he is in constant dread and fear of indefinite things. There have never been any periods of unconsciousness. He is depressed, hypochondriacal, and melancholic, and is said to have attempted suicide.

In neurasthenia the speech is sometimes indistinct and thick, bearing a superficial resemblance to that of general paresis. But, unlike the general paralytic, the neurasthenic can at once correct his speech defects when his attention is called to them, and then he can articulate perfectly well. The sleep

may remain normal. Ordinarily, however, it is broken, and in traumatic neurasthenia is frequently attended with nightmares. The bad dreams relate to the accident with special frequency. Neurasthenics are very emotional. Sudden noises, the sight of trifling accidents, or even thoughts of the accident of which they themselves have been the victims, may be followed by evidences of great excitement. Under such circumstances the patients become tremulous and confused, can speak only with difficulty, and are conscious of precordial oppression and palpitation of the heart. They are rarely subject to outbursts of laughter, but tears come easily.

It is only in the severer forms of the neurosis that all of these symptoms are present. They vary in number and degree in individual cases. But usually the picture of traumatic neurasthenia is a picture, as Knapp has said, "of the complaining, irritable, nervous invalid." When he tries to work he finds himself incapable of keeping his attention upon what he has to do. Efforts to concentrate the attention are followed by an increase of subjective symptoms. He may be almost constantly a prey to the annoyances of peculiar subjective sensations, and he describes his sufferings as more severe than they really are. He becomes alternately hot and cold; he has flushing of the face, his ears ring, he gets dizzy, his head aches, and he becomes confused. As a result of the discomfort from which he constantly suffers, and from a fear that any unusual excitement may make it worse, he prefers staying at home to going into crowded places or on the street. He shuns society, and after his condition becomes generally known society, unwilling to listen to constant complainings, shuns him. In traumatic hysteria the mental state has many characteristics similar to those of traumatic neurasthenia, but there are also some differences. The hysteric is more silent under his sufferings, resembling in this respect more closely the type of melancholia. He broods

more, but he talks less. The hysteric has to be questioned in order to find out the nature of his mental inquietude. The neurasthenic describes his sensations and feelings with exaggeration, with an unnecessary elaborateness of detail, and seeks opportunities to voice his complaints; the hysteric talks about them only when he is interrogated.

That some of the symptoms of neurasthenia might be controlled there is no doubt; but many of them are beyond the control of the patient and cause him much discomfort. Those thoughts must indeed be painful which can make a man, previously strong, healthy, and active, lose all inclination and ability for his work, and become more or less oblivious to the interest of his household.

The facial expression often tells the story. It indicates not so much bad health, as discouragement, anxiety, and fatigue. In it can be read the eternal worry and self-questionings which are going on in the mind. The face may be pale, though very often, if the cheeks were red previously, they do not lose their color.

It is a fact which is often hard to bear in mind, that the complaints and actions of the neurasthenic depend upon actual disturbances of the nutrition of the ganglion cells, for which he is largely irresponsible. He makes the complaints because the ganglion cells are exhausted, and he keeps the ganglion cells from being repaired because he so constantly thinks of his own troubles.

Thus he finds himself in a vicious circle. By constantly thinking of his own ills he renders his condition worse. Yet the same fatigue of nervous tissue which has caused the change in his character and health robs him of his independence and prevents him from withholding his attention from the trivial annoyances which are passed over unheeded by healthy men. It is only by an appreciation of this fact that the neurasthenic

can be treated with the consideration he deserves, and not summarily dismissed as a person whose only disease is too much thinking about himself.

Most of the mental symptoms of neurasthenia are subjective. The physician becomes cognizant of them chiefly through the confidences of his client; and although an experienced physician can usually tell in how far the recital is genuine, he has no means of being positive that all the complaints to which he has to listen are really believed in by the patient. It is the fact of subjectivity of symptoms which in negligence cases may tempt men to become malingerers, and which often gains for the neurasthenic patient the name of exaggerator or imposter.

Motor Symptoms.—Paralysis does not occur in traumatic neurasthenia, for gross lesions of brain, spinal cord, or peripheral nerves are absent, and functional paralyses occur exclusively as a result of hysteria. Yet although no individual muscle or group of muscles completely lose their powers, or present degenerative electrical reactions or atrophy, the vitality of the whole muscular system is very much lowered. Muscular force is only slightly impaired. A more striking symptom consists in this—the quickness with which the muscles become fatigued. Single movements may be performed with nearly all their original energy, but muscular power becomes rapidly and progressively exhausted. The muscular fatigue cannot be referred to the muscles alone. In every voluntary movement the stimulus passes from brain cortex along nerve fibers to muscles; and in the muscular fatigue of neurasthenia all these elements—nerve cell, nerve fiber, and muscle cell—share; so that the apparent muscular weakness of neurasthenia is only another expression of the general condition of the nervous system in that disorder. When the muscles become tired there is invariably an increase of other symptoms; the general nervous-

ness becomes worse and the subjective discomforts are intensified. The speed with which the muscles tire may be seen in many ways. Sometimes on standing only for a few moments the legs seem to give way, or there is some shaking of the body when the eyes are closed. The gait, except in so far as it is affected by lumbago, presents nothing unusual. The patient moves slowly, but he lifts his feet from the floor and walks steadily. He cannot, however, walk far. A man who before the accident could walk miles without fatigue, may become extremely exhausted by the walk of a few blocks. Some patients walk little or not at all for many months. In exaggerated cases the muscular efforts necessary for the performance of the ordinary duties of the toilet are fatiguing, and the patient may even be obliged to sit down several times while dressing himself. Fatigue may also be shown by the finer coördinated movements of the fingers being imperfectly performed. There may be difficulty in writing, in buttoning the clothes, etc. This is partly due to tremor, and partly to the rapidity with which all the muscles, but especially the smaller ones, become exhausted. Fatigue of the muscles, therefore, is among the commonest of neurasthenic symptoms. It is not, however, constant. Some neurasthenics can endure muscular exertion fairly well. In such, fatigue effects are more particularly mental. As a result of mental effort the nervous symptoms are intensified, the patients becoming excitable, sleepless, and confused.

Tremor is a frequent symptom. It resembles very closely the tremor seen in alcoholism or in toxic conditions generally. It is fine, rapid, and regular, becomes somewhat more pronounced on intended movements, and is much intensified by emotional influences and by fatigue. At times it disappears altogether. Its most frequent situations are the tongue, the face, and the hands. The tremor observed in the eyelids when

they are half closed, commonly spoken of as a neurasthenic symptom, occurs too frequently in normal individuals to be regarded as a diagnostic sign of any great value.

Sensory Symptoms.—Among the various symptoms complained of in traumatic neurasthenia, pain in the back is the most frequent and the most prominent. It exists in two forms: First, it may be of a dull, aching character, nearly constantly present, and affect the whole back, or, as more commonly occurs, be limited to the neck or to the lower part of the spine. Its most frequent seats are in the cervical and thoracico-lumbar regions. It is extremely indefinite in character, changing about from day to day. Even at the time of a medical examination the point of most marked hypersensitiveness to touch changes its location by several vertebræ within a few minutes. For example, at the first examination the tenderest spot may be found over the spinous process of the third thoracic vertebræ, and ten minutes later the seventh thoracic spine is found the most sensitive. The pain is aggravated by bodily or mental fatigue or by digital pressure over the vertebral spines, and is generally similar to the backache frequent in simple neurasthenia. Pain of this character is in large part an expression of fatigue, and exists independently of injury to the spinal column.

Secondly, and more especially characteristic of traumatic neurasthenia, is pain in the back of a different character, which is due to actual back sprain. Our knowledge of this condition, in regard to the cases resulting from railway accidents at least, is largely due to Page. The affection has come to be known as traumatic lumbago, and merits a separate description.

Traumatic Lumbago.—Traumatic lumbago, since it depends upon actual injuries sustained by the vertebral column, cannot be classified among functional nervous diseases. Yet

it is so frequent an accompaniment of neurasthenia, and so rarely fails to become complicated by neurasthenic disturbances, that it may best find its description at this place, among the symptoms of neurasthenia. That it is closely allied to spondylitis traumatica may be seen by reference to page 220. Lumbago is usually the first symptom to appear after the accident, and ordinarily lays the foundation upon which the functional manifestations are superposed.

The confusion which for so many years existed in regard to the nature of the nervous disorders which most frequently follow railway accidents was in large part due to the fact that traumatic lumbago was supposed to be dependent upon injury to the spinal cord. Although the symptoms are often serious, and in some respects similar to those caused by organic injury to the cord, it has been proved, with a reasonable degree of certainty, that in lumbago the spinal cord is not injured at all. Injury to the spinal cord is rarely followed by complete recovery. Complete recovery is the rule in lumbago, a disorder which is never fatal. In lumbago there is no anæsthesia, no loss of sphincter power, and no paralysis that the patient cannot overcome unless the nerves have been stretched or torn. When the spinal cord is injured some or all of these symptoms are present. There is also now little reason to doubt that the kinds of injury which most frequently cause a strain of the muscles and ligaments of the back are essentially different from those which cause disturbance of the contents of the spinal canal. Intraspinal hemorrhage, or laceration, or other damage to the spinal cord is almost exclusively the result of extreme violence. Yet since such conditions have occasionally followed accidents which were apparently trivial, it cannot be positively said in any given case that the spinal cord has escaped injury until examination has shown that its functions remain unimpaired.

Traumatic lumbago itself depends upon strain or lacera-

tion of some of the numerous structures which protect the spinal cord. The vertebræ through which the spinal cord passes are held in place by many ligaments, and between each bone is a disk of cartilage. The bones and ligaments are surrounded by numerous muscles, which give protection, support, and the power of movement to the spine. The essential characteristics of the vertebral column are flexibility and strength. Yet while considerable movement is possible in the spinal joints, especially in the cervical and dorsal regions, these movements are limited by the spinal ligaments, and if they are carried too far the ligaments will be strained or torn.

There is convincing evidence in support of the view that traumatic lumbago depends upon more or less extensive lesion to the ligaments or muscles, or to some other of the structures which are in relation with the vertebræ. From the anatomy of the spinal column it is obvious that these injuries may easily result from sudden twists or wrenches of the spine, such as are so frequently received in railway and similar accidents, or as may occur through blows or falls on the back, or through lifting heavy weights. Also the clinical type of the affection is identical, when due allowance is made for differences in function, with those observed in injuries to ligaments, articular surfaces, and muscles situated elsewhere.

The symptoms appear soon after the injury, though it is several days before they reach their maximum intensity. The chief characteristic of the condition, and indeed the one which is the basis for most of the others, is pain in the back. The pain is usually not spontaneous; that is, if the patient is perfectly motionless he may be fairly comfortable. But the slightest motion in the affected part causes the pain to become active. Now almost every movement of the body causes some motion in the spinal joints. Most movements of the head and neck, any movements of the arms, the use of the muscles

of the abdomen, chest or back, to effect changes in position or for expulsive acts, the movements of the legs—all are associated with more or less extensive changes of position in the spinal column. Similarly, slight jars or shakes are transmitted to the spine, and, by causing movements such as pass unnoticed by healthy persons, quickly induce an increase of pain in parts which are affected. Consequently there are very few moments during waking hours when these patients are comfortable, because it is almost impossible for anyone to remain perfectly still voluntarily. The pain is severe and real. At the slightest movement the patient cries out. The face is drawn and expressive of suffering and of apprehension. In severe sprains of the back the whole vertebral column, from the skull to the pelvis, is hypersensitive. More commonly, however, the pain is limited to certain regions, such as the neck or the dorsal region. It may be limited to the coccyx, causing the condition known as coccygodynia. The skin over the painful places is usually not hyperæsthetic, although sometimes the slightest touches are badly borne. As Page suggests, it is probable that extreme hypersensitiveness of the skin is due to the fear of being hurt. Previous examinations have taught the patient that manipulation of the back is painful, and so he comes to shrink at all contact, and may cry out that the physician has hurt his back before the back has been touched. Deep pressure, however, either of the back muscles or of the vertebral spines, almost always causes pain in traumatic lumbago, although it does not invariably do so in lumbago which is not of traumatic origin. The muscles are tender to the touch, probably through injury to the muscle fibers. When pressure is made on any of the vertebral spines which are in the painful areas, pain, often very severe, is felt at the seat of pressure chiefly, but may also radiate up and down the back. There can be no doubt as to the genuineness of this symptom. If, before

the pressure is made, the finger of the examiner is placed upon the pulse of the patient, it may sometimes be found that the spinal pressure causes a considerable acceleration of the pulse rate. This test—the Mannkopff test, previously described (see p. 383)—is utilized for the purpose of detecting simulation, or the voluntary exaggeration of spinal pain. It is a sign of considerable though not of unfailing value, and is not always present in traumatic lumbago. If the heart beats faster when the pressure is made, it is certain that there is either real pain, or that the patient is genuinely afraid that he is about to be made to suffer; the heart cannot voluntarily be made to materially increase its rate.

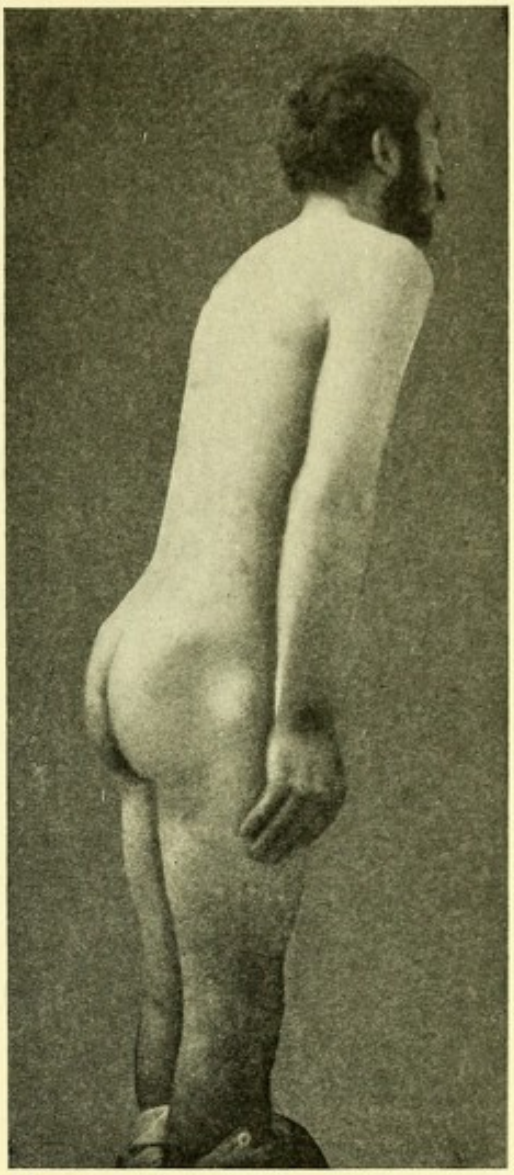

FIG. 81.—Characteristic attitude in traumatic lumbago.

The muscles of the back are usually in a state of spasm, a condition which may be very marked. In a recent Vanderbilt Clinic case, illustrated in Fig. 81, the back muscles were so tense that they felt like boards.

In this case the immobility of the back was extreme. The patient had received a severe fall on the buttocks, soon after which the

symptoms became very marked. Not only was the whole back held as though in a vise, but all movements of the head, arms, and legs were reduced to a minimum. In standing, the body was inclined slightly forward, with the hands pressed against the thighs; there was almost no change of position in the trunk when the patient sat down; and in walking, the legs were dragged forward in short steps, the feet being watched with as much care as the rigid position of the head permitted.

The attitude usually assumed by the patient with traumatic lumbago is due entirely to the pain. He puts himself in the position in which he is least liable to receive any jar or vibration, and which permits the greatest freedom from movement of the back. Only those voluntary acts which involve the least change in position of the vertebral joints are undertaken. The spine is held as rigid as possible, which results in very characteristic attitudes and movements. In standing, the back is seen to be held perfectly stiff. It is usually bent somewhat forward, and, from an increase of sensitiveness on one side, there may be a slight curvature. To look up, down, or around, the patient bends or turns the whole body, holding the neck stiff. Movements of the arms are performed very slowly. If the patient is to stoop down, instead of stooping in the ordinary way, which would involve bending of the back, he usually goes down on one knee, holding the back perfectly straight. In getting up from a chair, or in changing the position of the body when sitting or lying down, the hands and arms are called into service in order that the muscles of the back and abdomen may do as little work as possible. In getting up from the floor the patient may "climb up the legs with the hands," as do the patients with progressive muscular dystrophy. In getting up from the sitting posture, the hands are placed on the thighs, and the body assisted up in this way.

For similar reasons there may be striking interferences with gait. The patient walks with slow, hesitating steps, lift-

ing the feet but slightly off the ground, and sometimes actually dragging one or both feet. The interference with gait, through pain in the back, often has the appearance of true weakness in the legs, although in reality the legs are as strong as ever, and appear weak only because the patient is afraid to move them.

In some patients the power of walking is altogether lost for a time, as is shown by the following case which was referred to me for examination relative to a claim against the Delaware, Lackawanna and Western Railway for injuries received in a collision on January 15, 1894:

> The patient had been bruised about the back and buttocks, and for two weeks was in bed hardly able to move the legs at all, because every movement caused such severe pain. At the end of five weeks he could turn over in bed, and soon after began to get around on crutches, which he was obliged to use for nine weeks. He ultimately made a complete recovery.

The patient often attributes the interference with walking to spinal disease. Thus a gentleman suffering from typical lumbago, not of traumatic origin, because he had noticed that he did not lift his feet from the ground without pain, thought he was suffering from locomotor ataxia. Having read a little medicine, he came to me to have his knee jerks tested. In traumatic lumbago, unaccompanied by any injury to the spinal cord or nerve roots, the reflexes remain unchanged. The leg is often held stiff, but the knee jerk may be elicited, although reënforcement is sometimes necessary. Disturbances of the functions of the bladder and rectum are occasionally observed. They are paralytic in appearance, but not in fact. Through the pain caused by the contraction of the diaphragm and the abdominal muscles, the patient performs the expulsive acts as infrequently as possible. Constipation and temporary retention of urine are the results. It sometimes happens that there may be a little dribbling of urine, caused by an involuntary over-

flow of a too full bladder, which the patient is afraid to empty on account of the pain. There is never, however, any true incontinence or retention of urine or incontinency of fæces.

Pain caused by movements may make it difficult for the patient to go to sleep, or during sleep he may be constantly aroused by the pain.

The duration of traumatic lumbago is variable. Ordinarily the intensity of the pain on movement begins to decrease after three or four weeks; but the back usually remains sensitive for a long time, and in many cases the patient never feels that his back is as strong as it was before the accident.

A switchman, who was struck in the back by the front platform of a cable car, came to the Vanderbilt Clinic four months after the accident. He had become entirely free from neurasthenic symptoms; yet the pain in the back, although much better than it had been, was still very annoying, so that he frequently had sharp twinges on sudden movements, and could not stoop down without considerable discomfort.

The severe and persistent character of the pain renders lumbago a somewhat serious affection. By interfering with sleep, exercise, and alimentary processes, and by causing constant suffering, it may make decided inroads on nutrition. By impairing the general health and by constantly calling the patient's attention to his back, it materially assists in the production of the neurasthenic picture. The patient becomes unhappy and anxious, broods over his trouble, thinks that his spinal cord is injured, and believes himself in danger of becoming paralyzed—a fear which is not surprising when one considers how closely the limitation of movement in lumbago may imitate paralysis.

While lumbago is a particularly frequent accompaniment of traumatic neurasthenia, its occurrence in that disorder is not constant, nor is it usually so severe as the picture which has been drawn would indicate.

But the symptoms are always the same in kind, though they vary greatly in degree. In its exaggerated form, in which the back is held absolutely immobile, and in which all the other symptoms are equally pronounced, traumatic lumbago is distinctly rare, and only occurs as a result of severe injury to the back, such as may cause slight dislocation in the spinal joints, or injury to some of the spinal nerves.

It is much more common for the clinical picture to be limited to pain and stiffness of the back, which do not seriously interfere with nutrition.

The differences between traumatic lumbago and spondylitis traumatica should be borne in mind. In this latter there is usually more evidence of local injury, the pain is more localized and more spontaneous, though it may temporarily remit after the first week or ten days. As soon as the gibbus appears it is evident that a more serious affection than lumbago is present.

Headache is another common sensory symptom of traumatic neurasthenia. It may affect the occipital region, and is then complained of by the patient as "pain at the base of the brain." Or it may be in the forehead and over the eyes, similar in situation and character to the pain of constipation. Besides pain there may be various peculiar sensations in the head. The patient feels as though there were a tight band around it, or as though he were wearing a heavy cap—the *casque neurasthénique* of Charcot. There are frequently subjective sensations which are complained of as noises in the head, or feelings as though the skull were about to fly apart. The mental confusion from which neurasthenics so frequently suffer is associated with a feeling of lightness and peculiar sensations in the head. In addition to these somewhat indefinite pains, people suffering from neurasthenia are frequently subject to neuralgia. The pain under such circumstances is a true neuralgic pain,

which follows the course of the nerve trunks, and presents the painful points and other symptoms of neuralgia due to conditions of anæmia or malnutrition.

If at the time of the accident any parts other than the head or back were bruised or injured in any way, pain persists in them for a longer time than it would in persons who are not neurasthenic. The neurasthenic pays so constant an attention to local troubles that subjective disturbances which accompany them are intensified and made to last longer than they otherwise would.

Besides local pain, there is almost constantly present a general feeling of fatigue which may amount to pain. The patient says he " feels tired all over," a sensation much aggravated by exertion or excitement. Hyperæsthesia is also common. As has been said, it is usually present in the painful spots of the back, so that pressure over the vertebral spines is not well borne and may cause acceleration of pulse. The skin around such areas may also be hypersensitive, although, as Page says, this is probably a hyperæsthesia of education. Other regions of the body may also be the seats of exaggerated sensibility. The scalp is often very tender. It is usually a long time before injured parts may be touched without causing expressions from the patient both of fear and of acute pain. The muscles of the whole body, especially those of the chest, back, and legs, are commonly sensitive to the touch, and tender. Hyperæsthesia becomes more marked after any fatiguing influences.

Anæsthesia does not exist in simple traumatic neurasthenia. It is frequent in hysteria, and occurs sometimes in the serious forms of nerve disturbances which will be described as unclassified. When any case presents this symptom, it is certain that some other condition than simple traumatic neurasthenia exists. Neurasthenic patients often complain of feelings of

numbness, " pins and needles " or tingling, but the examination shows that the situation of these sensations is indefinite, and that all forms of cutaneous sensibility are perfectly preserved. There are also various other subjective sensations in neurasthenia. The complaint may be that there is a feeling of shivering without being cold, or as if cold water were running down the back, or as if electricity were being applied, or of numbness and tingling in the legs or hands, or other peculiar sensations of indefinite character.

Special Senses.—Ocular Symptoms.—The visual disturbances of this neurosis are usually considerable, although there are, apart from possible preëxisting organic defects or disease, no morbid changes in eye structure. The pupils respond quickly to light. The pupils may also dilate and contract under psychic influences. They are usually moderately dilated. When light is thrown into the eye, the iris may contract and then dilate again, and repeat this alternating contraction and dilatation until it finally comes to rest in contraction. Inequality of the pupils does not of itself indicate organic disease. It is observed in neurasthenia, and also exists in a certain proportion of perfectly normal people. The complaints of the patient relate to various disagreeable subjective feelings referred to the eyes, such as pain, burning, flashes of light, black specks, and " everything getting dark about him." The eyes are generally abnormally sensitive to bright light, and they soon become painful from use. The picture of the nervous invalid, sitting with the back to such light as may be admitted through tightly closed blinds, is generally familiar.

Asthenopia, or weakness of vision, is the most constant visual disturbance of the disorder. The eyes quickly become fatigued, so that the patient no longer sees perfectly. After short efforts at reading or writing the letters and words become indistinct. The rapidity with which fatigue follows any at-

tentive use of the eyes interferes very seriously with vision. The patient feels as though there were a veil before the eyes, and he complains that they are blurred. Tests with the perimeter show that the visual disturbances depend, in part at least, upon fatigue of the retina, especially in its peripheral portions. By successive or continuous exercise of its functions the retina loses some of its peripheral power. Thus the visual field is smaller the second time it is tested than it was the first, smaller

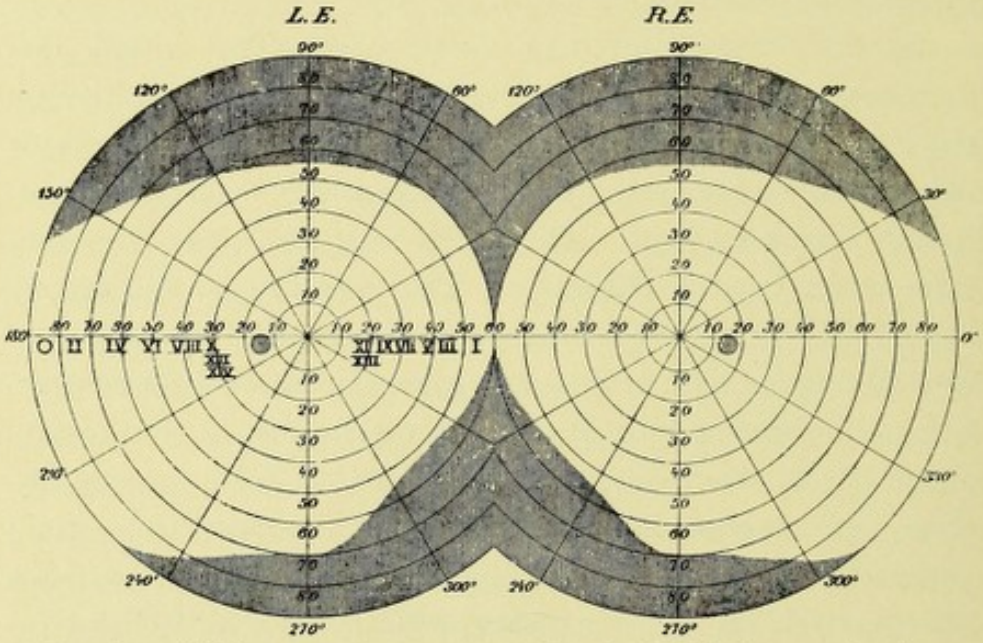

FIG. 82.—Shifting type of contraction of the visual fields in neurasthenia. (After Bass.)

the third time than it was the second. By repeated tests the field may become very much contracted, or the patient may lose the power of concentrating the attention upon the testing object. This symptom of retinal fatigue has been called by Förster the "shifting type of contraction" (Fig. 82). The most convenient method for its determination has been described by Wilbrand and Sänger. By this method the testing object is moved along the horizontal meridian of the perimeter from without inward to the center, and is then continued across

to the limit of the field on the nasal side. The horizontal extent of the field before it has become fatigued is thus ascertained. The object is then moved in the same meridian, across the field, back to the temporal side. If the retina is easily fatigued, the testing object will on its return outward have ceased to be perceived before it has reached the temporal periphery of the original field. A similar limitation will be found on the nasal side when the object is again moved inward. The shifting type of limitation is the same for colors as for form. There is never a reversal of the normal color fields, such as is found in hysteria.

Limitation of the visual fields through fatigue may occur to a certain extent in normal individuals, but the rapidity and extent of the contraction in neurasthenia are characteristic.

In neurasthenia the acuity of central vision is not impaired.

Of the other special senses, TASTE and SMELL are not affected. Organic disease of the AUDITORY APPARATUS forms no part of the neurasthenic picture. Hypersensitiveness of hearing is, however, a prominent symptom, which becomes much intensified by any local ear disturbance. The hearing is often remarkably acute. A neurasthenic patient of Binswanger's is said to have been able, while sitting in the house with the windows closed, to distinguish her husband's footsteps from the noises of the street, and to recognize the individual voices of servants who were talking two stories below. In many cases of neurasthenia slight noises are heard so distinctly that they become sources of considerable annoyance, and may add to the severity of the other symptoms.

The ear is also frequently complained of as being the seat of various abnormal subjective sensations, which are described as ringing, buzzing, and other peculiar noises. They are often very troublesome, and become more intensified by general fatigue or by any temporary disturbances of health. Dizziness

is another symptom which may be referred to the ear. It is of nearly constant occurrence in neurasthenia, but it, too, is subjective rather than objective. The patient complains that his head feels dizzy, that objects spin around before him, that he is in constant fear of falling, and that he is often obliged to sit down or to catch hold of surrounding objects for support. Yet the objective evidences of dizziness are usually wanting. When told to walk or stand, the patient can usually hold himself steadily. Sometimes when standing with the eyes closed there is some swaying of the body, but it is never in any degree proportionate to the amount of dizziness and unsteadiness of which complaint is made.

Reflexes.—Neither the superficial nor the deep reflexes are ever lost in simple traumatic neurasthenia. When the reflexes are either absent or diminished it should excite suspicion of more serious trouble than nervous exhaustion. The deep reflexes may remain normal, but in by far the larger number of cases their activity is increased. This increase affects the two sides equally. If the reflex of one side is more active than that of the opposite side, it is suggestive of organic lesion. The knee jerks are usually very active, so that a slight tap on the patellar tendon induces a quick, forcible, and extreme extension of the leg. But true ankle clonus does not occur. In excessively nervous people there may be a slight trepidation of the leg when the test is first tried; but this can hardly be mistaken for true clonus. The Babinski reflex is also absent. The tendon reflexes of the upper extremity—the extensor, supinator, triceps, etc.—are frequently absent in health. In neurasthenia they are often present or hyperactive. There is no standard by which the normal activity of the superficial reflexes—cremaster, epigastric, abdominal, etc.—can be determined. Some of them may be absent in normal persons. In neurasthenia they are usually lively. If there is pain or hyperæsthesia of one

side of the body, there may be an increase of superficial reflex activity on that side. The reflexes, both superficial and deep, share, with the other symptoms of neurasthenia, the common characteristic of becoming quickly fatigued. With successive blows on the tendon, the knee jerk becomes progressively less, and may finally cease to respond altogether. The superficial reflexes also quickly tire in the same way. Fatigue of reflexes may be induced in health, but in neurasthenia it supervenes much more quickly.

Vascular Disturbances.—Very few sufferers from traumatic neurasthenia escape some disturbances of the circulation, of which the most common is palpitation of the heart. This neurasthenic palpitation is not due to disease of the heart itself, for that organ is normal in size and has no murmurs. Attacks of palpitation are sometimes sufficiently severe to resemble the attacks of angina pectoris, so that during them the whole præcordium is made to vibrate by the tumultuous action of the heart, and pulsation in the superficial vessels is plainly visible. The pulse beats may be as frequent as 130 to 150 to the minute, or it may even be impossible to count them. The pulse is quick, soft, and full, often intermittent and irregular, without any increase of arterial tension. Pain over the heart felt during the palpitation is often very severe. The patient becomes alarmed, his face is anxious and drawn, he gasps for breath, although there is rarely any true dyspnœa, and he experiences a variety of abnormal sensations which he refers to the præcordial region.

These attacks occur in varying degrees of severity. It is only rarely that there is any danger of confusing them with those of true angina pectoris, a condition due to morbid changes in the arteries or walls of the heart. In true angina pectoris the patients are usually of an age when arteriosclerotic changes are most common, some evidences of which may often

be found. In that condition, also, attacks come without apparent cause, and the pain, which exists for some time before there appears any irregularity of the action of the heart, usually radiates from the left shoulder down the left arm. In neurasthenia, pain occurs more frequently after than before the palpitation, and for neurasthenic palpitation there can usually be found some immediate exciting cause, although it is often trivial. Sudden noises, fright, or any slight mishap, often bring on an attack. The mere thought of the accident often causes the heart to beat violently, and the patient may awake from a bad dream to find that even in sleep psychic influences have acted upon the innervation of the heart. In addition to palpitation, the heart action in neurasthenia may apparently remain accelerated for long periods of time, beating 90 or 100 or even more times in a minute.

In connection with long-continued neurasthenic tachycardia arises the question, Does it lead to structural alteration in the heart muscle? Oppenheim reports cases which seem to have resulted in this way, and Knapp inclines to this view. That in neurasthenia, even when the patient is at rest and undisturbed, the heart may beat a little more rapidly than usual, is possible, although difficult of proof; but that such an increase in the pulse rate alone, unaccompanied by other factors, ever leads to hypertrophy and dilatation of the heart seems a matter of considerable doubt. It may assist in the development of a preexistent tendency to cardiac degeneration, or it may accentuate symptoms which had passed unobserved. But it is difficult to believe that a person whose vascular system was previously normal is in any danger of disease of the heart walls from neurasthenia alone. Undoubtedly cases occur in which cardiac hypertrophy and dilatation become apparent while the patient is being treated for neurasthenia. Disease of the blood vessels, which existed before the accident, and which would

have eventually become marked by clinical signs even if the patient had never become neurasthenic, may be one of the explanations of such phenomena. It is also possible, if the period of invalidism is protracted, if the patient worries for years, gets little exercise, is idle and introspective, that arteriosclerosis may show itself.

The other vascular symptoms of neurasthenia may be referred to impairment in the capillary circulation. The patient is frequently subject to sensations of heat and cold and to sudden flushings of the whole body. The face may be observed to become successively red and pale. The hands and feet are often cold, and may be blue and feel numb. Sweat is increased in amount, and the patient is very apt to break out into perspiration as a result of slight excitement.

Digestive Disturbances.—Digestive disturbances are frequent in neurasthenia whatever its origin, and in many cases it seems as though the nervous symptoms were in large part due to, or aggravated by, an auto-intoxication from the absorption of poisonous substances derived from imperfect metabolism. In traumatic neurasthenia the appetite may be impaired for some time after the accident, although it usually soon becomes restored to the normal standard, even when the nervous symptoms are getting worse. The tongue is coated and tremulous and the breath is offensive. The various symptoms of gastric dyspepsia are frequent. There are peculiar subjective sensations referred to the stomach, such as feelings of hunger, or as though the stomach were constantly empty, or as though there were something gnawing at it. Gastric pain, particularly after eating, is the subject of frequent complaint. Vomiting may occur immediately after the accident, but it rarely continues as a symptom later in the disorder. There is usually a considerable production of gas after eating. The gastric symptoms are commonly associated with palpitation of

the heart. A full meal may cause the heart to beat violently for a considerable length of time. The skin may be sallow and yellow, but there is rarely any jaundice. The bowels are constipated, as a rule. Constipation may have originally occurred as a result of lumbago, but it also exists independently of lumbago, and may persist after the pain in the back has in large part disappeared. In rare cases occurring after accidents there is persistent diarrhœa, which cannot be explained by other than nervous causes. In the milder degrees of the neurosis the general nutrition is not materially interfered with. In severe cases, however, there is considerable loss of weight.

The remaining symptoms, though often complained of, are of secondary diagnostic importance. In the female there may be interference with the menstrual function. In men, complaints regarding sexual matters are frequent. The mental state is accountable for the anomalies of the sexual act. Thus, in coitus, by reason of excitement, ejaculation frequently occurs too early; or, by reason of timidity or fear, erections fail at the proper moment. Thus, the anatomical structures being normal and living spermatozoa being present, coitus is defeated by purely mental means. Disturbances of this character are most frequent in bachelors. In some neurasthenics there is a slight mucous discharge from the urethra, and, at stool, mucus may be discharged from the prostate. This is interpreted as spermatorrhea, and is accepted as an explanation of the lost manhood. In litigated cases " loss of manhood " is frequently advanced as a cause for damages. Usually the sexual symptoms are classed among the general results of the accident; sometimes as the result of injury to the sexual organs themselves.

A workman fell in the water when a bridge on which he was working gave way. While under water he was struck across the thighs

and on one testicle by a beam. He brought suit for damages, the chief item of his complaint being "loss of manhood." The man before the accident had been accustomed to the most unbridled sexual excesses. The sexual organs were normal, but the man was a confirmed neurasthenic. The jury gave a small verdict, which the judge promptly cut in two.

The urine shows no characteristic changes. It is often neutral or alkaline in reaction, so that the phosphates precipitate readily. The patient, observing all symptoms, does not fail to ascribe a sinister meaning to this one. He thinks again of his sexual disorders, or believes he has Bright's disease. The urine may be somewhat lessened in quantity.

Diagnosis

Nervous excitement immediately following an accident, although it may present many of the symptoms that have just been ascribed to neurasthenia, does not constitute neurasthenia. It is when these symptoms persist for weeks or months, changing slightly by reason of their chronicity, and being supplemented by others, that they present characteristics sufficiently definite for them to be recognized as constituting a clinical type of nervous disorder. If we review the symptoms given as neurasthenic, we will find few of them strictly objective. In the weeks which must elapse before a diagnosis of neurasthenia can be made, the contusions and wounds which may have been received have healed, and the patient is surgically normal. The nervous symptoms of objective character are tremor, rapid pulse, defective peripheral circulation, certain visual disturbances, and possibly pain. None of these is typical. Neurasthenic tremor does not differ, either in character, distribution, or degree, from that seen in many other nervous states. The circulatory disturbances are also of doubtful value by themselves. Rapid pulse is so common a result of the excitement

incidental to a medical examination that several trials are necessary before a pathological significance can be ascribed to it; and changes in peripheral circulation are often individual peculiarities. The objective visual disturbances of neurasthenia can hardly be called pathognomonic. In how far pain is an objective symptom has already been considered.

So in the diagnosis of neurasthenia, the physician has no thoroughly reliable objective signs to lean upon. Giving a proper value to such objective signs as he finds, he must in the main rely upon his own experience and acumen to determine in how far the patient's appearance, manner, method of relating his symptoms, and the character of them coincide with the classical symptom-complex of neurasthenia.

Organic disease must be eliminated before the diagnosis can be made. This applies to diseases of the viscera (notably of the kidneys and blood vessels) as well as of the nervous system. General paresis is the one organic disease of the nervous system with which neurasthenia can most easily be confounded. The mental symptoms of paresis are often typically neurasthenic at the beginning; but in paresis the changes in the pupils, in the knee jerk, and the tremor of the tongue are present and readily serve as a means of differentiation. Certain psychoses may be confused with neurasthenia. The persistent insomnia, the loss of flesh, the motor unrest, the extreme depression—with suicidal tendencies and sometimes delusions—of melancholia, are usually sufficient to distinguish it from neurasthenia. In hypochondriasis there is usually an hereditary taint, and the source of anxiety is much more limited than in neurasthenia. In hypochondriasis, also, the physical symptoms, usually well marked in neurasthenia, are wanting. As already said, the psychasthenia of Janet has many neurasthenic characteristics, notably fears, morbid impulses, imperative conceptions, obsessions, etc. But, with few exceptions, in such

cases there is a distinct hereditary taint, and in none of these cases can trauma alone be assigned as cause.

Neurasthenia and hysteria are often blended, though the two affections are in themselves quite distinct.

The objective symptoms of hysteria (paralysis, contracture, spasm, anæsthesia, etc.) are wanting in neurasthenia. In hysteria, function is either abrogated or presents itself in an exaggerated degree; in neurasthenia, disturbed function consists in undue irritability and ease of fatigue, without distinct perversion or objective demonstrations. The neurasthenic relates, while the hysteric shows, his symptoms.

Prognosis

The prognosis of traumatic neurasthenia as regards life is good. It is hardly to be believed that a previously healthy man ever dies even from the remote results of an accident in which he received no injury more vital than a sprain of the back or a nervous shock. In persons whose previous health was impaired, or who were suffering from actual disease, the effects of injury and shock are more serious and may even prove fatal. Even though patients have died during the course of traumatic neurasthenia, it is more reasonable to suppose that in such cases the accident added an impetus to the progress of preëxisting disease, or that the shock, injury, and resulting exhaustion proved to be more than a previously enfeebled organism could combat.

It is very difficult to arrive at any satisfactory conclusion regarding the prognosis for recovery. Different men hold different views relative to the percentage of complete recoveries, and to the average duration of the disorder. The railway surgeon regards the prognosis as good in nearly all of the cases. The neurologist, on the other hand, takes a graver

view of the prospects for a return to health. To see how widely opinions on this subject may vary, it is only necessary to turn from the writings of Page to those of Oppenheim. Page believes that most of the patients get well upon settlement of claim; from reading Oppenheim's book one might be led to suppose that recovery was exceptional. This diversity of opinion may be explained, in large part at least, by the kind of cases seen by the different observers.

The corporation surgeon says he sees many cases of neurasthenia following accidents which terminate in complete recovery, either spontaneously or after settlement. If recovery occur spontaneously, the surgeon thinks the patient had little or nothing the matter with him; if it follows payment, he infers, and often correctly, that the symptoms had been largely exaggerated. Such an inference is by no means always justifiable. Neurasthenia generally improves after mental anxiety and worry are removed, and relief from these factors is in many cases as reasonable an explanation of recovery as the payment of the claim. Furthermore, the corporation surgeon, while he often hears of the miraculous cures which the company's gold not infrequently induces, loses from sight the vast majority of the patients whom he examines. Upon adjustment of the claim they vanish into obscurity, and there is no means of discovering whether they continue to be the prey of nervous symptoms or not. He infers that their troubles are over, but he can only be sure of this in a relatively small number of cases; and it is this fact which prevents him from arriving at any definite estimate of the percentage of cases which completely recover.

Page endeavored to decide the question as to the proportion of recoveries and the duration of symptoms. In his tabulated list of 234 persons who had been injured on the London and Northwestern Railway, the inquiries were not made, in the larger number of cases, until two years had elapsed after the

accident. Some of the cases he saw himself, some he heard about from other doctors or from outside sources. The information regarding many of these cases is too meager to be conclusive. But from his inquiries it appears that considerably more than one-half of the patients recovered eventually, although recovery was often delayed.

Unsatisfactory as are such investigations and uncertain as are their results, they constitute the only means we have of determining exactly how serious the condition is. It was originally the intention of the writer to pursue a similar line of inquiry regarding the injuries to the nervous system which have resulted from accidents on American railways; but after considerable reflection, and on the advice from various railway officials, the plan was abandoned as impracticable. To insure absolute accuracy of results, it would be necessary to have the records of the examinations of a large number of patients soon after the accident, as well as thorough personal examinations made after a considerable interval of time.

Such a task, were it undertaken by the railway company, would require an enormous amount of labor; it is practically impossible for a private individual. It constitutes, however, the one means of determining even approximately the number of persons who eventually recover entirely from their injuries. Until some such investigation is carried out, our views of the prognosis of traumatic neurasthenia must be based upon personal observation, and upon the communications of neurologists and surgeons who have the opportunities of seeing large numbers of these cases.

The position of the neurologist is at the other extreme from that of the railway surgeon. The persons who come to him are either plaintiffs seeking an expert opinion or patients desiring treatment. Whichever they may be, they usually present evidences of serious disturbance of nervous function. It

is, however, unquestionable that the serious condition of many of these sufferers is due to the influences by which they have been surrounded since the accident, rather than to the effect of the accident itself. The patients engaged in litigation have become worse through anxiety, uncertainty, and frequent examinations. Those who come with the sole object of getting well have often been subjected to faulty methods of treatment.

This is especially apparent in clinic patients. In my opinion, treatment in clinics is distinctly prejudicial to neurasthenic patients, and I have no doubt that the severity of the symptoms in some of the clinic cases which have been reported to show how rebellious an affection traumatic neurasthenia may be, even when unassociated with litigation, was in large part due to the injurious influences which every clinic exerts on such cases. The patient, while awaiting his turn, discusses his case and his symptoms with other patients. Through the constant association with disease and doctors his attention is continually riveted on personal troubles, and he thus often becomes an incurable hypochondriac.

These patients are rarely satisfied with one physician, but usually try several, either simultaneously or consecutively. One of them confided to me that he had been at one time under the care of two doctors, who gave him different medicines. In order to avail himself of the therapeutic skill of each, he mixed the separate medicines together and took one-half of the amount obtained by adding together the doses of both medicines! The evil influences of clinic treatment are less prejudicial than those caused by waiting for legal questions to be decided, but they are longer continued. A claim is eventually disposed of, and the patient's anxiety is then in large part relieved. But as long as the neurasthenic has strength to work, or money enough to pay car fare, he may prolong his dispensary treatment indefinitely.

However, whatever be the causes of the condition, the neurologist sees many cases of traumatic neurasthenia which last for years, and many which seem incurable. Of 29 cases of "traumatic neurosis" observed by Sänger some time after the accident, 3 were entirely well, 7 had only slight subjective symptoms and could work, 11 were only partially able to work, and 8 were *in statu quo*. Richter reports 3 cases, resulting from accidents, in which the patients were inmates of the Dalldorf Asylum. All of them had originally been regarded as simulators.

A visit to any large neurological clinic will convince the most skeptical that a large number of these complaining hypochondriacal invalids believe themselves to be as ill as they claim to be. To such a clinic come almost daily men who give no evidences of having any grave disease of the nervous system, yet who profess themselves as entirely unable to work. In such cases idleness does not result from any hope of compensation. Litigants rarely come to dispensaries for the outdoor poor. On the contrary, inability to work means suffering for them and their families. Yet many of these men, who previously to the accident had been active wage earners, are unable during months or years to resume their regular employment. A locomotive engineer came from time to time to the Vanderbilt Clinic for over two years. From the time that he was in a collision he was unable to return to his occupation. He was a robust, intelligent man, and was not conscious of any injury at the time of the accident, nor did we ever have reason to suspect that his condition was anything more than traumatic neurasthenia. Yet on the slightest excitement or fatigue this man became so confused and tremulous that he abandoned all idea of trying to work, and went to live in the country for a year to see if he could get back his former strength and self-control. Nammack reports the case of a policeman who in the

discharge of his duty was very much frightened and shaken by a team of runaway horses. The man was not physically injured, and had, of course, no thought of bringing any action, and yet after two years his nervous condition would not permit him to return to duty, and he was eventually pensioned by the police department. Similar cases are of not infrequent occurrence, and several examples of them have been mentioned in preceding pages.

It comes especially to the attention of the neurologist that the seeming exaggeration of traumatic neurasthenia is not necessarily dependent upon financial considerations. Many patients, after their claims have been adjusted and paid or their actions have been decided, continue to present, with very little variation, the same old train of familiar symptoms. There may be improvement when all the legal formalities are at an end, but not infrequently the symptoms continue with but little or no improvement.

The case mentioned on page 398 grew worse, instead of better, after settlement.

The examples just mentioned represent the more serious and the exceptional cases. It is safe to say that of the total number of persons who develop neurasthenia as a result of accident, the majority recover sufficiently to return to work.

In many the restoration to health is complete, so that they are apparently as well as they ever were. It is possible that even those persons who are, as far as outsiders can see, as well as ever, still do not enjoy the same consistent and resistent health they did before the accident. Every wound leaves its scar, and neurasthenia may well leave its scar on conscious personality. Some patients find themselves more irritable and more easily tired than before the accident. The back may become painful on fatigue or when suddenly jarred or moved. There is more or less headache, and the patients do not sleep as

well as they formerly did. Such a condition of nervousness and fatigue may continue for many years, so that the patient never feels himself "the man he used to be." However, in the majority of persons previously healthy such symptoms eventually pass away entirely.

There are other cases, of which examples have already been given, in which the patients, from the time of the injury, show evidence of profound nervous disturbances. All the symptoms of neurasthenia are well marked, and instead of getting better as time goes on the condition remains stationary, or seems to get worse. The symptoms of traumatic lumbago may pass away, but the insomnia, the tremor, the despondency, and the malnutrition remain. If these cases persist, the prognosis for ultimate recovery is doubtful, and some of them are never able to resume their occupations.

The duration of the disorder is consequently variable. It is essentially a chronic affection, requiring a considerable length of time for repair. The larger number of patients who are properly treated can return to work in the course of a few weeks or months, although a longer time may be necessary before they " feel themselves again." It is uncertain just how long the symptoms may last before it can be said that recovery is no longer probable. It varies with individual cases and with associated circumstances. Knapp believes the chances of recovery in any case which has existed over three years are slight.

Several factors very materially affect the prospects of recovery. Perhaps the most important of these, and certainly the one which has received the greatest attention, is litigation.

The worst possible thing to which the patient with traumatic neurasthenia can be exposed are the mental and moral surroundings which are inseparable from litigation.

Litigation, much more than merely entering a claim for

damages with willingness to adjust it, furnishes the very influences from which a neurasthenic should be free. The anxiety, uncertainty, and delay which are the inevitable consequences of lawsuits prevent the patient from having the physical and mental rest which are essential to recovery. His mind is more constantly attracted to self-contemplation; he is made frequently to relate his symptoms, and is required to pass through many medical examinations. From the nature of the proceedings he is not encouraged to maintain that his sufferings are lessening nor urged to go back to his work, even if he is able to do so.

It is entirely unfair to assume that the increase of nervous symptoms during the time that litigation is pending is nothing more than voluntary exaggeration by the patient, or that the symptoms improve after settlement because there is no longer anything to be gained by posing. As has already been said, it is universally characteristic of neurasthenia that the symptoms become worse through disturbing influences of any character. This is observed in cases in which the question of litigation does not enter at all; and in litigated cases the various nervous disturbances may become intensified not necessarily for forensic purposes, but because litigation itself provides the very factors most prejudicial to the patient. Similarly, if the patient improves when the suit is settled it is in part, at least, because the anxiety and uncertainty and fatigue have been removed, not necessarily by reason of the financial gain.

Treatment, which can do much to hasten recovery, cannot be properly carried out as long as the patient remains a plaintiff. The physicians whom the patient sees are experts rather than therapeutists; the important question is detailed diagnosis and evaluation of symptoms rather than cure. As it is usually months or years before a case is settled, the patient is obliged to go without proper treatment, and to be surrounded by de-

pressing factors for a long time and during a period when the symptoms are most amenable to cure. Isolation and rest and moral control, the specifics for neurasthenia, are generally impossible in negligent cases.

Bad as it is, pending litigation is not the absolute bar to recovery from neurasthenia that it seems to be to recovery from hysteria. Few, if any, hysterics make material improvement before the legal questions are terminated. But it is entirely possible for a neurasthenic to be well on the way to restored health, if not actually arrived there, long before the money question is decided.

In conclusion, it may be said that the prognosis for more or less speedy and permanent recovery in any case of neurasthenia is good if the patient is not hampered by litigation complications, and can put himself at once under the care of a skillful physician, although even then recovery is not always assured.

CHAPTER III

TRAUMATIC HYSTERIA

Ætiology—Pathology—Symptoms: Mental, Sensory, of Special Senses, Motor, General Somatic—Medico-legal Relations—Prognosis—After-Histories of Litigated Cases.

It is through the philosophical and clinical studies of Charcot and his pupils that hysteria has come to be regarded as a distinct disease, which, although it may take many forms and show startling powers of mimicry of other diseases, has its own identity and limitations. It is true of hysteria, as of many of the psychoses, that the limitations are often difficult to determine. The manifestations are essentially psychical, and, when not pronounced, it is not easy to decide whether they are to be regarded as symptoms of disease or as mental variations within the limits of health.

Hysteria major and hysteria minor are the divisions of the disorder as classified by Charcot. Hysteria major, or *la grande hystérie*, is the type characterized by convulsive attacks; it is infrequent in America. Hysteria minor embraces all the varieties in which there are no attacks. This term, consequently, is extremely comprehensive, since it might include not only most of the cases of traumatic hysteria, but also those which are characterized by slight mental instability, in which there may never have been any of the stigmata or accidents of the disease.

Just how comprehensive the conception of hysteria minor should be it is impossible to determine. Mental suggestibility is the most prominent symptom of hysteria; but if we are to class as hysterical all persons who display this symptom, it will

be found that a large part of the world's population is a victim of the disease.

Different views on this subject are held by different men. Möbius says that "hysteria is simply the morbid increase of a rudiment which is present in all, and that everyone is a little hysterical. If this were not so," he aptly adds, "it would fare ill with the practice of medicine."

Charcot taught that hypnosis is possible in hysterical subjects only, and yet Bernheim claims that he can hypnotize ninety per cent of the persons he sees. If both propositions are true, the conclusion is inevitable that hysteria is an almost universal disease.

For us it is sufficient to say that although all hysterical persons are suggestible, all suggestible persons do not present other hysterical stigmata, and that more symptoms than mere mental suggestibility are necessary before we are justified in making the diagnosis of hysteria.

Inasmuch as we are entirely ignorant of the underlying anatomical character of the disorder, any definition of hysteria is necessarily imperfect. For practical purposes, however, it may be said that hysteria is a disease of unknown pathology, affecting the whole nervous system. The symptoms, which may be permanent or transitory, are mental and physical. The mental symptoms are the most important, and in them are to be sought the explanations of the physical manifestations. The physical symptoms may imitate very closely those of organic nervous disease, but they do not depend on any structural lesions which have as yet been recognized. They are involuntary expressions of disordered mental states. When called into activity by traumatic agencies, the affection is called traumatic hysteria.

The symptoms of hysteria have been recognized since the days of Plato and Hippocrates. For many centuries they were

supposed to be dependent upon uterine troubles, and to occur exclusively in the female. Charles Lepois, writing in 1618, abandoned the uterine theory and admitted the existence of the disorder in men. The contributions of Sydenham to the subject of hysteria showed more discernment than any which had preceded them, and were more valuable than many of those which were to follow. Sydenham studied most of the hysterical stigmata. He admitted the existence of hysteria in the male, giving it the name of hypochondriasis, and regarded the nervous system as the seat of the trouble. Brodie, in 1837, recognized that functional paralysis may depend upon impairment of will power. The first modern treatise on the subject was written by Briquet in 1859.

It is to Charcot, however, that we owe the complete presentation of the symptomatology of this disease. His able mind and extensive experience in all branches of medicine well fitted him to discern the errors of previous workers in this department of neurology. His rare opportunities at the Salpêtrière were so well employed that he lived to see hysteria occupying a definite and limited place in clinical medicine. It is chiefly through his teachings that hysteria has been shown to be not a mixture of affectation, exaggeration, and deceit, but a condition in which the symptoms are involuntary expressions of disordered mental states. These symptoms are not assumed. The paralyses, anæsthesias, and convulsions of hysteria are as real to the patient as though they had visible underlying causes.

The false statements of hysterical persons are not necessarily willful lies, but may originate honestly from hallucinations or from losses of memory. Many of the physical symptoms can be accounted for by impairment of will power. As Paget aptly put it, in referring to a paralysis which to-day would be recognized as hysterical, "The patient says, 'I cannot'; it sounds like, 'I will not'; it really is, 'I cannot will.'"

Brissaud has well said that hysteria is "one and indivisible," and in employing the term traumatic hysteria there is no intention to imply that hysteria provoked by trauma has any essential differences from the disorder when it is due to other exciting causes. But as all diseases vary under different causal conditions, so hysteria following trauma has some characteristics peculiar to itself. These special characteristics are not sufficiently distinctive to warrant any separation of traumatic hysteria from the protean affection; individual clinical parallels may be found in cases of hysteria in whatever way they may have developed. Inasmuch, however, as there are usually certain circumstances which modify the disorder when it is of traumatic origin, the following description will be limited to those forms of it which are most frequently observed as a result of physical injury and psychic shock.

Trauma as a cause of local nervous disturbance without organic lesion has been recognized by Brodie, Russell Reynolds, Page, Strümpell, and many others; but to Charcot must be given the credit of having established beyond a doubt the possibility of the existence of hysteria excited by injury. He taught that the functional paralyses and anæsthesias which sometimes follow trauma were identical with those induced or made to disappear by hypnosis, and he formulated the theory that in the development of hysteria the influence of trauma is an influence of suggestion. The shock of the injury acts as a hypnotizing agent, the local pain or discomfort calls the patient's attention to the part injured and suggests the symptoms of paralysis, or contracture, or anæsthesia, or whatever the symptoms may be (traumatic suggestion). There are some objections to this theory, but it is the best which has as yet been offered.

As a distinct disease, with its paralyses, contractures, convulsions, and the like, hysteria is less familiar to physicians

generally than it should be. In its pronounced varieties the condition is comparatively rare in this country, and only exceptionally occurs in the exaggerated forms observed in Europe. As a result, many cases of hypochondriasis and neurasthenia are classed as hysterical when they are not hysterical at all. Also it too frequently happens that hysterical mimesis is confused with voluntary simulation, and that true cases of hysteria are regarded simply as frauds. With the increasing general interest in neurology and with the more conservative use of the word "hysterical," hysteria may some day become in America as well recognized a disorder as it is in Europe.

Ætiology

Predisposing Causes.—Charcot taught that hysteria is a disease which only shows itself in persons whose nervous systems are diseased, degenerate, or abused. He classed it with the diseases of degeneracy, and regarded it as a child of the neuropathic family. He believed that predisposition to it depends upon ancestral defects or acquired enfeeblement of the nervous system, and that without such predisposition the disorder could not exist. The profound disturbances of hysteria which may follow insignificant exciting causes certainly indicate that the *agent provocateur* can only produce effects in a nervous system already prepared for their development. Very much less importance is attached by the Germans to the necessity of predisposition in the causation of hysteria, and, indeed, to prove it is not always easy.

As in other nervous disorders, we may in many cases discover evidence of a hereditary disposition, yet it often happens that such proofs are not obtainable. In America, so many of the poorer classes have immigrated as children, and know little or nothing about their families, that we are frequently unable

to gather any reliable information regarding their antecedents. The evidences of ancestral defects, as shown by stigmata of degeneration, aside from such stigmata as may properly be regarded as hysterical symptoms, may be entirely absent.

Just as the history and evidences of inherited impairment of the nervous system may be wanting, so hysteria may develop in persons whose personal life seems to have been free from disease-inducing influences. On the other hand, it can sometimes be demonstrated that the resisting powers of the nervous system have become enfeebled through the effects of disease or of chronic intoxication. Among the diseases which predispose to hysteria, nephritis, diabetes, syphilis, the continued fevers, and many of the affections of the nervous system are prominent; chronic metallic poisoning is an important ætiological factor, and especially poisoning from lead or mercury, the latter of which seems to be associated with such particular frequency with hysterical stigmata that some authors believe the *tremor mercurialis* to be largely, if not entirely, of hysterical origin; hysteria is also very frequently found in victims of chronic alcoholism. When trauma is the immediate provoking agent of hysteria, predisposition appears less necessary than when the disease is of nontraumatic origin. Dana and Knapp both regard hereditary factors as of less importance when the disease follows an injury than when it is due to other exciting causes. Of 21 cases of traumatic hysteria studied by Berbez, only 9 had undeniable neurotic antecedents.

Exciting Causes (*Agents provocateurs*).—Of the various causes by which hysteria may be excited, trauma and shock are the only ones which concern us here. Traumatic hysteria has been recognized only in recent years. Although it was previously known that slight injuries might be followed by functional palsies and contractures it was not until 1886 that trauma came to be recognized as one of the most frequent of

the *agents provocateurs* of hysteria. It was again by Charcot that the causal relationship was made clear. He also showed that not only was trauma to be considered as an ætiological factor, but as one of the most frequent causes. According to Berbez, a pupil of Charcot, one-fifth of all cases of hysteria are of traumatic origin. The injury is usually not severe. That such injury may be followed by functional paralysis, or contracture, or convulsions, has received abundant clinical verification.

A coachman fell from his cab, and, though considerably shaken up, received no severe injury; five days later he developed a brachial monoplegia of the side upon which he had fallen. A glass polisher, soon after a slight injury to the wrist, presented an hysterical contracture of the hand. A mother, in a fit of anger, struck her child with the back of her hand; the hand which had given the blow soon became anæsthetic and powerless. All of these injuries were too slight and inadequate to cause organic changes sufficient to account for the startling symptoms which ensued (Charcot).

In addition to the causes already mentioned, hysteria may result from almost any untoward event in which physical injury, however slight, and fright are combined. Recently a number of cases have been reported as the result of slight shocks received during the use of the telephone; and I have personally observed one case of hysterical brachial monoplegia following the application of the x-ray.

Far more important than the severity of the actual injury are the circumstances under which it occurs, and the patient's mental state at the time it is received. All influences which cause excitement and fright very much increase the probability of hysteria ensuing as a result. If, immediately before the accident, the about-to-be victim has an opportunity to be frightened by what is going to happen to him, the mental impression thus received accentuates the effects of the physical hurt. To both of these factors—trauma and fright—mankind

has ever been constantly exposed, and in our mechanical times the probabilities of injury have become so many times multiplied that we are almost constantly exposed to terrifying accidents.

Hysteria is frequently the sequel of distressing railway and similar disasters, in which the victim may or may not have been injured. It is essential for purposes of justice, as well as for correctness of diagnosis, that it be generally recognized that very startling hysterical symptoms can follow accidents which have caused only slight physical commotion or bruising.

In the hysterical phenomena which follow local injury the influence of suggestion is evident. It is the part injured which becomes the seat of the paralysis or anæsthesia or contracture. A blow on the shoulder is followed by brachial monoplegia of the same side; a blow on the hand causes symptoms in the hand only; if the injury is to the head, the result may be hemiplegia, not crossed, but on the same side as the trauma. The influence of suggestion is particularly well seen in the cases in which hysterical symptoms are superimposed upon organic injury. Charcot records the appearance of hysterical contracture of the forearm and hand, due to splints which had been applied for a fracture of the radius. Nerve injuries are occasionally associated with local hysterical manifestations. In the following case, for the privilege of reporting which I am indebted to Dr. Starr, a lesion of the cauda equina was complicated by astasia-abasia:

A woman, twenty-seven years of age, was injured in a railway collision on July 4, 1894. She was much frightened, felt severe pain in the back, was able to walk for a few minutes, but then the legs gave way and she sank to the ground. She remained unable to walk at all for fifteen months, though she recovered the power of moving her legs in all directions when in bed. Immediately after the accident there was retention of the urine, and since that time the control of the bladder has been imperfect. The catheter was used for eight

months. The action of the bowels has also been irregular. There was considerable numbness in the legs and back, at first with imperfect power of movement and rather rapid atrophy in the muscles of the thighs and legs, but strength and volume have returned to them.

Examination, October 2, 1895, showed areas of anæsthesia (Fig. 84) around the umbilicus and around the anus. Though the patient had perfect command of the legs, and could move them in every

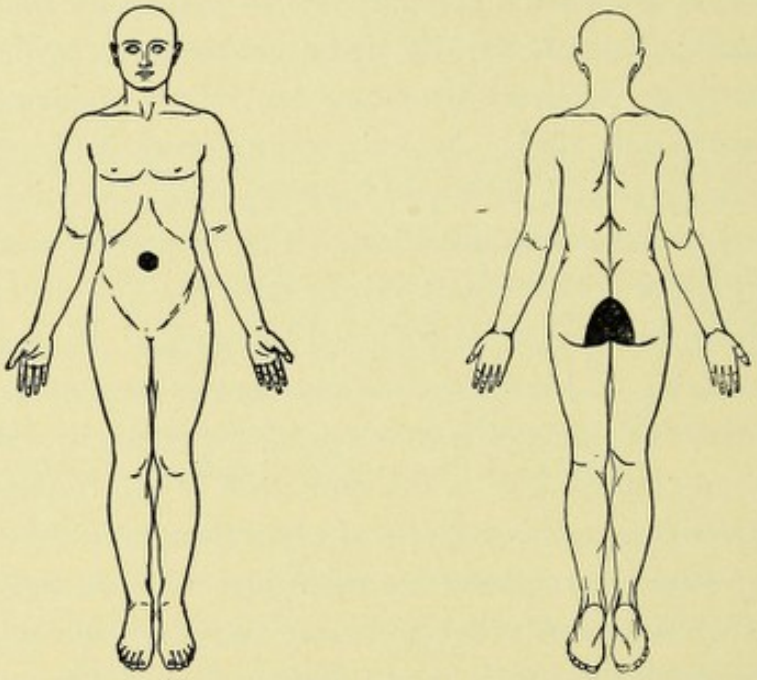

FIG. 83.—Anæsthesia in a case of injury to the cauda equina, associated with astasia-abasia.

direction while lying down, astasia was complete, the legs collapsing immediately when any attempt was made to stand. There were general neurotic symptoms as well. This condition persisted for a long time after the accident, in spite of substantial damages. In this case it seems that the astasia was purely hysterical, while the anæsthesia of the perineum and the bladder and rectal symptoms were evidences of hemorrhage or some organic injury to the cauda equina.

One year later the patient was seen again, having in the meantime been taught to walk with a roller machine. She then walked perfectly, had no general hysterical symptoms, but the area of anæsthesia remained exactly the same as before.

When psychic shock alone is the exciting cause, the symptoms are usually less localized. Under such conditions the hysterical manifestations are more commonly aphonia or convulsions or coma. Among the other factors having certain bearings on the ætiology of traumatic hysteria are sex, age, occupation, race, and sometimes litigation.

Nontraumatic hysteria is more frequently observed in women than in men. The failure to recognize that hysteria may occur in man was for a long time an important obstacle in the progress of knowledge regarding the disease; but in the past few years its frequent occurrence in the male has become amply demonstrated. This advance was largely due to the observation of hysterical stigmata which occurred in men as the results of accident and injury. Although women furnish by far the larger number of examples of nontraumatic hysteria, when the disorder first appears as a result of trauma it seems as though men were more frequently affected than women. Berbez, in 21 cases of traumatic hysteria, found that 14, or two-thirds of them, were men. In Knapp's cases the relative number of men and women was about equal (25 cases—13 women). Greater exposure to traumatic influences, acting as a predisposing cause, may account for the greater frequency with which traumatic hysteria appears in men than in women. However, the increased proportion of men to women is not nearly so large in this disease as it is in traumatic neurasthenia.

Age exerts the same influence upon the development of traumatic hysteria as upon that of traumatic neurasthenia; both are essentially disorders of the active periods of life, although traumatic hysteria may occur at any age, and sometimes occurs in young children. Profession and mode of life, except in so far as they predispose to impairment of health, seem to exercise less influence than race. The American, who is so subject to nervous exhaustion, less commonly develops hysteria after accidents.

In Europe, and especially in France, traumatic hysteria is not uncommon. All over the world Hebrews are a prey to various neuroses, and hysteria is a common disease in male Jews. In fact the largest number of cases of traumatic hysteria which occur in this country are found in Hebrews or in the foreign born.

The effects of litigation have a somewhat different bearing upon the course of hysteria than upon that of neurasthenia. In neurasthenia the uncertainty and anxiety of suit may make a serious affection of what would otherwise be trivial. In hysteria the symptoms are developed soon after the accident, independently of any question of damages and often before there is an opportunity for such a question to arise. Neurasthenia would in many cases not appear at all if the patient could at once be surrounded by proper influences only; but most commonly, when anyone who develops hysteria has once received the fright or the injury, the mischief is already done, and the disease appears in its full development. The subsequent course of the case will depend in large part upon environment, and will be protracted or made more severe by the evil effects of litigation. But there are few cases of traumatic hysteria of which it can be truthfully said that the symptoms would not have appeared except for the question of damages.

Pathology

Into theoretical speculation upon the pathology of hysteria it would be useless to enter here. Nothing is known as to what morbid anatomical changes are responsible for the symptoms of this disease, and, furthermore, it is improbable that the problem will be solved by any of the microscopical methods at present at our disposal. As far as is known, the central nervous system in hysteria is anatomically normal. That some morpho-

logical or chemical changes underlie the alteration in function there is little reason to doubt; but the pathology of hysteria will prove even more elusive than that of neurasthenia. Hysteria is a disorder chiefly affecting the mind, and its location is probably cerebral; but whether it is entirely cortical, or situated as well in other regions of the brain, it is impossible to say.

Symptoms

The symptoms of hysteria are divided into permanent symptoms, or stigmata, and accidental, paroxysmal, or intervallary symptoms. Usually some of the stigmata are constantly present during the disease; they include the mental state, anæsthesia, and the affections of the special senses. The paroxysmal symptoms, as the name indicates, are only of occasional occurrence, and embrace the paralyses, the contractures, the convulsions, and similar manifestations.

As Briquet very truly says, "hysteria attacks the whole organism." Its symptoms may be those of disturbance of function of any organ. Some are very much more frequently encountered than others, and certain symptoms are usually associated. Anæsthesia and contraction of the visual fields are particularly constant, and are generally combined. Paralysis is more infrequent, yet when it occurs it is almost invariably accompanied with loss of sensation. Tremor is common, occurring in a variety of forms. The greater the number and variety of symptoms presented by any one case the more probable it becomes that the patient was an hysteric before the accident. Children rarely have a variety of symptoms. In them, paralysis is the commonest manifestation, and neurasthenia is more frequently absent than present. Occasionally also in adults only one symptom is present, and it is in such cases that the diagnosis is difficult and uncertain, and can only be made by

the exclusion of other conditions. In such cases, when litigated, the exclusion of simulation becomes sometimes impossible. Fortunately, for diagnostic ends at least, traumatic hysteria is most frequently revealed by several characteristic evidences, and often the picture is so clearly defined that there can be no doubt as to its meaning. In a case which has been under my care for some time, the patient tells a characteristic story of hysteria which may or may not be true; but the symptoms he presents are unmistakable, and have been abundantly verified by repeated observations. They are in so many respects illustrative of traumatic hysteria that an account of the case may be properly given here:

P. D., forty-seven years of age; alcoholic, as was also his father; denies all symptoms of nervous disease, except alcoholism, in all the members of his family. Says he is an actor by profession.

On October 19, 1894, while in the flies of a theater, he fell twenty-eight feet, "landing on the head and spine." He was in the hospital for seven weeks, during the first half of which period he was unconscious. When he came to himself he found that he could not move the left side at all, and that the movements of the right hand were very much impaired. There was no loss of control of the bladder or rectum. For many months after the accident he was completely aphonic; he soon recovered considerable power in the left hand, less in the left leg. He was too much disabled to work, and having no money, he was sent to the almshouse hospital, New York City. The patient remained in the almshouse three months and a half, and during that time, the house physician says, he did not utter a sound of any kind, and invariably dragged the left leg in walking. He had no motives for simulation, and I believe that the patient was honest in regard to his symptoms. Eventually leaving the hospital, this man was again seen in a few months at the workhouse, where he had been sent for drunkenness. The left leg was still dragged, but the patient had recovered the faculty of speech. He told us that he had recovered his voice as the result of falling off the rear platform of a railway car. He had been considerably stunned by the accident, but on regaining consciousness, he says, he "was scared to death by finding that he was talking to himself." The aphonia, at least, had been cured! The general condition at this time (October, 1896—two years

after the original accident) appears, from my notes, to be as follows: A well-developed man, slightly anæmic. Heart, lungs, and other vegetative organs apparently sound. There is some weakness of the left hand, shown on attempts at movement. The movements at the left knee and ankle joints are limited by reason of muscular weakness, so that the patient can flex and extend both the foot and the leg to a slight degree only. In walking (Fig. 84), the left leg is dragged, as though it were an inert mass. The toe and inner side of the foot scrape along the floor, and the heel touches the floor only when the patient is standing still. There is a contracture of the wrist and of the fingers of the right hand. The fingers are held in extension and the wrist is slightly flexed. This contracture may be overcome, but attempts at forcible movement in these joints cause considerable pain. There are nowhere any wasting of muscles or changes to the normal reactions of electricity.

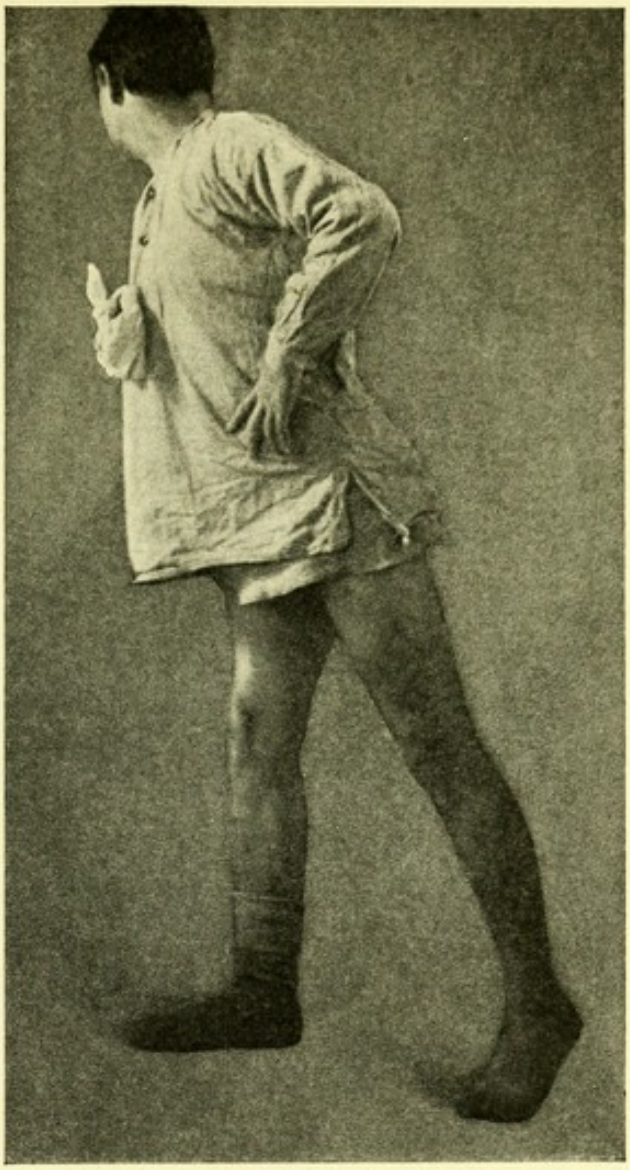

FIG. 84.—Traumatic hysteria, showing characteristic dragging of the foot.

From a point a very few inches above the knee, downward, the left leg is totally anæsthetic. The skin does not bleed to ordinary pin pricks in the anæsthetic area. There is a diminution in the acuity

of cutaneous sensibility over the remaining portion of the left side of the body (Fig. 85). There is slight deafness in the left ear. The patient insists that he sees perfectly well, but the visual fields as marked out by the perimeter (Fig. 86) show a moderate contraction, which follows the periphery of the normal field. It is more marked for the left eye. The color fields are diminished in extent, but there is no reversal of their normal arrangement.

The patient complains of no pain or hyperæsthesia, with the exception of the pain caused by movements of the right wrist. He sleeps fairly well. He is a tractable prisoner, but because he walks better at times than at others, he is generally looked upon as a " fakir " by the guards.

The mental condition presents nothing particularly characteristic. There have never been any emotional manifestations, and although

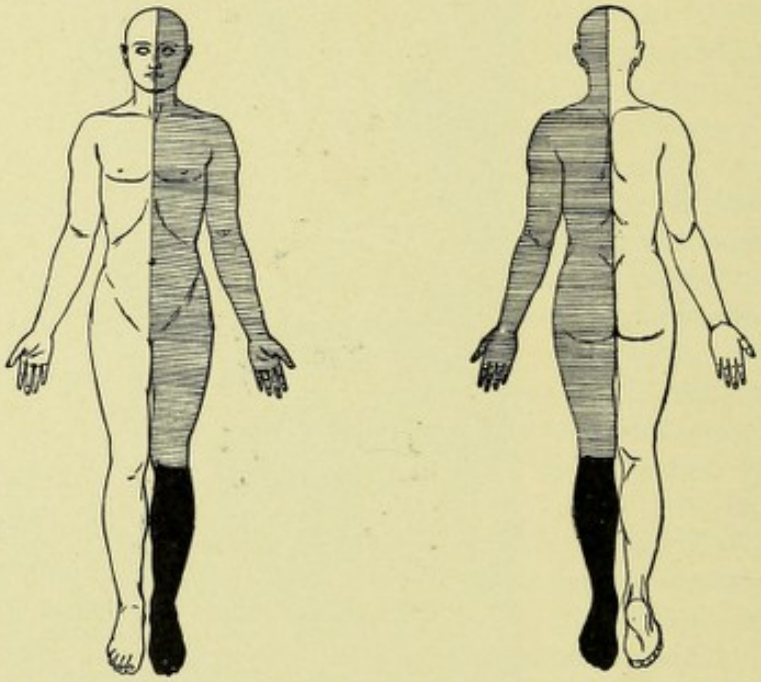

FIG. 85.—Traumatic hysteria. Paralysis existed in the left leg. In the shaded areas, cutaneous sensibility was diminished; in the left leg, it was totally abolished.

at times the patient appears discouraged and depressed, the neurasthenic mental state, often present in traumatic hysteria, is not pronounced in this case. The symptoms did not disappear when the patient was intoxicated. He once came to my office very drunk, but was dragging his foot in the same old way. The long period during

which he has maintained the same chain of symptoms, the absolute lack of motive for deception, and the absence of all evidences of organic disease, render the diagnosis of traumatic hysteria incontestable.

There was a predisposition caused by alcoholism, both parental and personal—a consideration which should have been taken into account had the case been the subject of medico-legal inquiry.

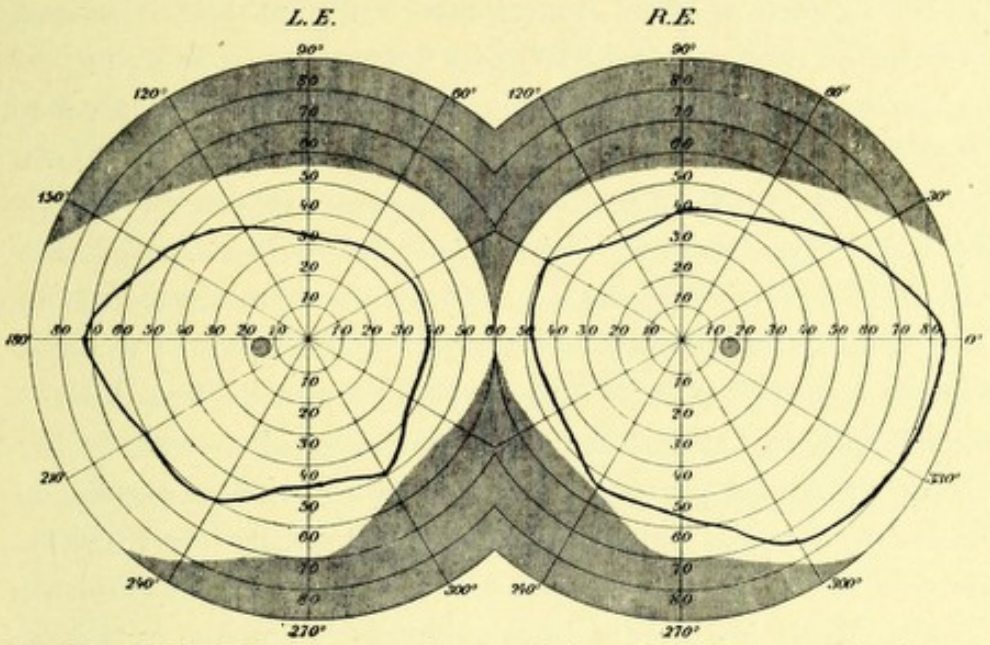

FIG. 86.—Field of vision in a case of traumatic hysteria. The contraction is more marked on the left, which was the side of paralysis and anæsthesia.

MENTAL SYMPTOMS

The most prominent feature of the mental state of traumatic hysteria is that by the mental state physical symptoms are created which imitate conditions such as may be the results of organic disease. The imitation is sometimes so close that it is with difficulty that the true nature of the physical disturbances may be appreciated. The mimetic powers of this affection are far more highly perfected than any to which the voluntary simulator may hope to attain, and differ from attempts at fraudulent and voluntary simulation in that they are, in

large part at least, unconsciously performed. In a disorder which of itself is histrionic and exaggerative the opportunities for voluntary exaggeration and deceit are considerable, and hysterical symptoms become regularly worse when they are observed by others. The patients, who are never so happy as when forming centers of attraction, have a tendency to embellish such of their clinical manifestations as are deemed worthy of special investigation by learned men, or as are received with interest by audiences composed of physicians and medical students. It seems probable that some at least of the astonishing hysterical phenomena which are reported from certain psychiatrical clinics, if they are to be considered as genuine products of mental disease, could occur only under conditions which are most favorable to the full development of the psychosis. Yet, after due allowance has been made for errors of interpretation, the fact remains that psychical mimesis is frequently found in persons who are ignorant of the existence of any such thing as hysteria, and who, uneducated and without motive, closely imitate diseases of which they have never heard. That hysteria is different from simulation is proved by the occurrence of hysterical phenomena among all peoples and in all times, by the general uniformity in the character of the symptoms, and by the lack of motive or ability of simulation in the majority of its victims. Although the disease is essentially simulative and its manifestations unreal, it occurs not only without intention, but without the patient knowing that his symptoms are false. It can best be explained by the theory that it is a morbid emotional mental state which involuntarily and unconsciously provokes a mimicry of the symptoms of other diseases. It is only when the fact is appreciated that hysteria is a serious disorder affecting the mind that the sufferer from it can be treated with the consideration he deserves; even then it is oftentimes difficult to believe in the sincerity of a patient

who is seen to move a limb which was apparently paralyzed, or who, though nearly blind to usual tests, never pays the accidental penalties of organic blindness. It can be proved by optical tests that in hysterical amaurosis sight is in reality not lost; in the hypnotic state the patients may describe sensations which, while awake, passed apparently unperceived; or may be made to move muscles which were paralyzed; similarly, by hypnosis, apparent memory defects may be shown to be not genuine.

When it is thus demonstrated that functional activity, though apparently abolished, exists, we cannot suppose that there are lesions in the organs or in the nerve tracts or in the perceiving centers themselves. Janet, who has studied this subject with rare skill, explains the apparent contradiction of hysterical phenomena by the theory of a limitation of the field of consciousness. Mental actions go on, but are independent of the patient's personal knowledge. Centripetal stimuli may be perceived by the perceiving centers, but they are not transferred to the domain of personal consciousness. Centrifugal impulses may be voluntarily liberated, but such volitional impulses originate independently of the higher ego. By thus assuming a power of subconsciousness, of which the higher consciousness in normal minds is only occasionally aware, and to which, in hysteria, the higher consciousness may become completely oblivious, most hysterical symptoms can be explained.

In nontraumatic hysteria the mental symptoms are somewhat different as they occur in men and in women. Traumatic hysteria, whether occurring in men or in women, has the characteristics of male hysteria, in which the purity of the hysterical picture is blurred by the admixture of symptoms of neurasthenic conditions, so that it is often a composite of hysteria and neurasthenia, a circumstance which has gained for it the

name of hysteroneurasthenia. In traumatic cases neurasthenic disturbances are particularly prominent during the interval between the occurrence of the accident and the appearance of typical hysterical manifestations. A case of Charcot's well illustrates this:

> A man, fifty-three years of age, industrious laborer, witnesses the suicide of his son. He loses consciousness for several moments. From that time the father is a changed man. From being gay and active, he becomes sad and bad-tempered. He shuns the people with whom he used to be on good terms. His sleep becomes restless and filled with annoying and painful dreams. He thinks of his son as a happy child, and then sees him bloody and disfigured. The memory for recent events fails. The man becomes distracted. His head feels as though he wore a heavy cap. The sexual functions are weakened. There are digestive disturbances after eating. He becomes weak and easily fatigued. These symptoms are largely neurasthenic in character, and they continue to be prominent even after the addition of paroxysmal hysterical accidents.

Suggestibility is the underlying characteristic of the hysterical mental state, whether the disorder is induced by trauma or by other causes. The patient is constantly subjected to influences similar to those used in induced sleep. By Charcot's theory, as we have seen, the accident itself exerts its influence by means of suggestion. Hypnotism is the effect of suggestion upon a hysterical mind, or at least upon a mind which is morbidly suggestive—a condition closely allied to hysteria, if not in itself hysterical—and the mental symptoms of hysteria and the manifestations of provoked hypnosis are closely related, if not identical. By this theory may best be explained the genesis of the disorder; by it also may be best understood those symptoms which are purely psychical and the rapid alternations in the manifestations which are physical. The different effects of auto-suggestion under different conditions are well illustrated by the case to which reference has already been made

(p. 448) : The actor who fell from the flies of a theater was apparently but little hurt, but became, a few days later, completely aphonic. For several months he could not utter a sound, although he was perfectly well in other ways, aside from the hysterical symptoms. When he fell from the rear platform of a moving railway train he again escaped with trifling injuries; but the impression which this accident made upon him acted in a different way from that of the first, for he immediately began to talk, and has had no difficulty in talking ever since. In a person once hypnotized, the most trifling occurrences may act as suggesting agents, and in hysteria it sometimes seems as though these agents originated in the patient's own mind, independently of external stimuli (auto-suggestion). Of similar import are dreams and nightmares, which are frequent symptoms of hysteria. When the disorder arises from traumatic causes the details of the accident may in this way be kept constantly in view. In the daytime the patient reflects upon what he dreamed at night, thus continuing the auto-suggestion. Many of the apparently false statements of hysterical subjects arise from dreams which the patients fail to differentiate from actual occurrences.

From dreams at night and from reveries through the day may arise hallucinations. Hysterical hallucinations have attained medico-legal importance in various criminal inquiries in Europe. They are not frequent in traumatic hysteria; when they occur, they usually, but not always, relate to the accident.

Hun reports a case in which they were an important feature:

A dressmaker, who was sitting in the last car of a railway train which was standing before the station, saw another train on the same track rapidly approaching from behind. She was very much frightened, and in trying to get out she was thrown down and hurt her back, although she escaped any grave injury. She was temporarily unconscious, but soon came to herself. In addition to the ordinary

hysterical symptoms of sensorial anæsthesia and paralysis which resulted from the accident, this patient began to have recurring attacks in which she shouted and "raved that she was in a lunatic asylum, and begged not to be sent upstairs to the noisy ones." She also saw knives and blood on the walls of her room. The patient was paid $8,500 by the railway company, without litigation, but after this the gradual improvement which had begun did not seem accelerated. She continued to have from three to six attacks of convulsions each month, and the right leg remained insensible and paralyzed. After hospital treatment she was cured of all her symptoms.

In a recent personally observed case the patient, after a slight injury on a street car, developed recurring attacks, during which she was apparently unconscious. She was delirious, calling out that the car was coming down upon her, that knives were being stuck in her, that she was home again in Ireland, etc.

Loss of memory often gains for the hysteric the reputation of a liar and swindler, and renders the history he tells the physician unreliable by reason of faulty and contradictory statements. Hysterical amnesia usually differs from organic amnesia in its periodicity and in its limitation to certain classes of ideas. Janet classifies it as systematized, localized, generalized, and continued, though the two latter forms are rare. In systematized amnesia the memory is lost for a certain category of ideas, not necessarily acquired at the same time, but relating to any one thing. Thus a patient remembered other things, but forgot all circumstances connected with Janet's personality. In local amnesia the forgetfulness is for certain periods of time. Thus, in traumatic hysteria, the patient may forget circumstances which occurred immediately before or immediately after the accident, or may not know that any accident occurred, although otherwise the memory remains normal.

Hysterical affections of memory are inconstant and contradictory. They are not necessarily attended with deterioration of reasoning power. In traumatic hysteria a frequent

form of memory loss is due to lack of attention and is confined to recent events. The patient remembers the details of the experience of childhood, but forgets the happenings of yesterday.

Aboulia, or impairment of will power, is a constant mental symptom of the disease, whatever its origin. In its most common form it is manifested by a difficulty in performing motor or intellectual acts at will. The patient feels himself incapable of doing the simplest things or of undertaking the most ordinary intellectual exercise. Diminution of volition is particularly frequent. Instead of being active and emotional, the patient is quiet and subdued. There is hesitancy in the performance of voluntary acts, which may amount to complete loss of will power. Attempts by the physician to arouse activity of mind or body may be successful, but the patient quickly becomes fatigued and confused and inattentive.

Some of the more striking psychological accidents, such as have been frequently described in ordinary hysteria, are usually wanting in traumatic hysteria. Somnambulism, catalepsy, and the like are rarely mentioned as occurring in cases which arise from injury or fright.

Yet, although the occurrence of exaggerated psychical disturbances is not the rule when the psychosis is of traumatic origin, they are sometimes observed. Laudun has described the case of

> A girl, eleven years old, who was very much frightened while swinging. Soon afterwards she had several attacks of coma and paralysis, with cataleptic flexibility of the muscles. In the first attack the pupils were widely dilated, and did not respond to light. After twenty-five hours the patient awoke from the stupor, but the muscular movements remained for some time hesitating and slow. The memory for the time which elapsed from the beginning of the attack until the patient awoke was entirely lost. The succeeding attacks, of which there were several, lasted a shorter time. In eight

days the patient was entirely well again. During the attacks there was sugar in the urine, which disappeared when the patient recovered.

Throughout the whole symptomatic range of hysteria we are constantly confronted, and often in the same patient, with clinical contradictions. We witness demonstrations which indicate both a loss and an increase of irritability, defects of function and excesses of function. Anæsthesia and hyperæsthesia of the skin often exist side by side, and skin hyperæsthetic to some stimuli may be anæsthetic to others. The muscles may show a loss of irritability, as in paralysis; or a heightened irritability, continuous, as in contracture; or intermittent or rhythmic, as in morbid movements. In many of the anomalies of the muscular mechanism, notably in those of the eye and of the viscera, it is difficult or even impossible to distinguish whether loss or excess of muscular excitability is responsible for the clinical condition. But for them all the psychological basis is the same, and, with all its variety, hysteria is "one and indivisible." In hysteria there is profound disturbance in the sphere of the emotions, and personality undergoes manifold changes. In clinical manifestations and course, therefore, there are encountered as many variations as there are in personality itself.

The general appearance and manner of a patient suffering from traumatic hysteria are the results of the mixture of hysterical and neurasthenic symptoms. He is inattentive, inactive, and indifferent, and shows a diminution of interest in his surroundings. He is credulous, and may seem fond of exaggeration and imposture, but his credulity is childlike, and his exaggeration and deception lack the cunning of the paranoiac. In relating the history of the accident and the symptoms which have flowed from it, his expression and manner often indicate that he himself is not convinced of what he stoutly maintains to be fact. He is often pictured as a partial maniac, ever ready to

laugh or weep, or to give evidences of exalted mental states. As a matter of fact he is moody, introspective, and depressed, approaching the type of melancholia more closely than that of mania.

Sensory Symptoms

Anæsthesia.—Hysterical anæsthesia is a symptom of which the patient only rarely complains, and of which he is often ignorant until its existence is pointed out to him by medical examination. The subjective difference between hysterical anæsthesia and anæsthesia of organic origin is well illustrated by a case related by Janet:

> A young girl cut her right wrist with a piece of glass, thus injuring the median nerve. There was no paralysis, but she complained of tingling and anæsthesia, total in places, over the palm of the hand and fingers. The physician who examined the case discovered that there was also a complete left hysterical hemianæsthesia, of which the patient not only did not complain, but whose existence she did not even suspect. There could be no more convincing proof of the necessity for thorough sensory examination in nervous cases.

Ignorance of the existence of hysterical anæsthesia on the part of the patient is an ignorance of personality only. We have seen that in hysterical anæsthesia sensations are really perceived, although they do not enter the field of consciousness. This fact receives additional proof by the absence of any injury to regions which are hysterically insensible. It is almost unheard of for a hand, the seat of the profoundest hysterical anæsthesia, to be injured, without the patient's knowledge, by cuts or burns. This is in direct contrast to the anæsthesia resulting from injury to the peripheral nerves or spinal cord, for when complete anæsthesia of the hand is of organic origin it almost invariably happens that the patient is unconsciously cut, bruised, or burned.

However, in spite of the fact that until told of it the patients may be unaware of the existence of anæsthesia, loss of some form of sensibility in the skin or mucous membranes is by far the commonest symptom of hysteria. Even in America, where the more pronounced hysterical affections are infrequent, hysterical anæsthesia is constantly met with. The anæsthesia is most commonly total, all forms of sensibility being impaired or abolished. Less frequently the different sensations may be dissociated. This dissociation may be similar to that of syringomyelia. Thus, of 17 cases of hysterical hemithermoanæsthesia examined by Charcot, 11 presented the type of total anæsthesia. In 6 there was a dissociation of sensations. Two of these 6 had loss of temperature sense, with preservation of sensibility to touch and pain. In the 4 others the dissociation was the same as that observed in syringomyelia—i. e., touch sense preserved, pain and temperature sense lost. Analgesia is the most common of any individual sensory defect.

The different forms of sensibility may be entirely lost or merely impaired, so that the ordinary stimuli, applied to the skin or mucous membranes, pass unperceived, but are recognized when the irritation is intensified. Thus, in hysterical impairment of touch sense, slight touches may not be recognized, although deep pressure is felt; or there may be analgesia for ordinary painful stimuli, while strong electrical currents cannot be well borne.

The limits of cutaneous anæsthesia, when other symptoms are pronounced, are usually sharply defined. The commonest topographical distribution is unilateral. In the clinic it most frequently occurs in the form of a left hemianæsthesia extending from the head to the feet, and involving the mucous membrane of the eye and mouth of the affected side (Fig. 87). In traumatic hysteria, however, in which paralysis is so frequent, the sensory loss commonly coincides in its distribution with that of

the parts paralyzed (morphological anæsthesia, see Fig. 85). If there is hemiplegia, there will be hemianæsthesia; if paraplegia, the sensory loss will be from the waist down, usually, though not always, skipping the genital organs. If muscular power is lost in one limb only, the anæsthesia involves the skin which covers the paralyzed member. When the sensory change occurs in segments of limbs only, it is called segmental, a form which is frequently seen in the involvement of areas of skin ordinarily covered by gloves and stockings (glove anæsthesia, stocking anæsthesia).

Sensory loss may occur in scattered areas (Fig. 88) over the whole body, a form easily overlooked if sensory examination is not painstaking. Pronounced anæsthesia of the whole cutaneous surface is usually associated with other unmistakable hysterical symptoms, and is rarely observed except in coma.

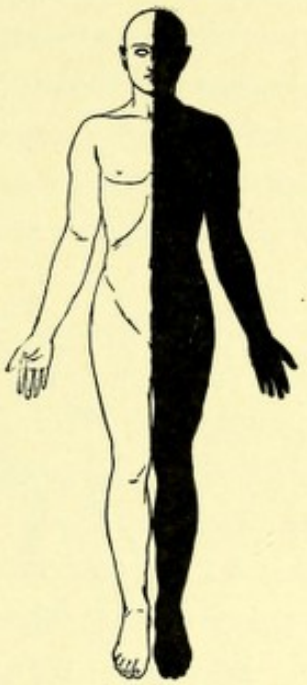

FIG. 87.—The most frequent distribution of hysterical anæsthesia.

The topographical distribution of hysterical anæsthesia differs essentially from that due to other causes. It never follows the distribution of individual nerves. In transverse spinal-cord lesions the resulting loss of cutaneous sensibility is found in the parts supplied by the injured segments; these parts do not correspond to the areas affected by hysterical anæsthesia. Furthermore, the penis usually retains its sensibility in hysteria, but never does in lesions above the third sacral segment which cause anæsthesia. In unilateral spinal-cord lesions (Brown-Séquard paralysis) paralysis of motion and sensation are on opposite sides. The topographical distribution of the anæsthesia of syringomyelia follows the spinal segmental type. In

organic lesions of the brain the resulting anæsthesia is generally slight, and the associated symptoms are usually sufficient to permit of a correct diagnosis.

Hysterical anæsthesia may come and go, be permanent or transitory, or may frequently change its situation. It ordinarily disappears during sleep, and may disappear during the various intoxications. Its locality can frequently be made to change during the hypnotic sleep, and suggestions received during waking hours may cause it to disappear or change its place. In children it is often absent even when paralysis exists.

The various alterations of the sensory functions of the skin

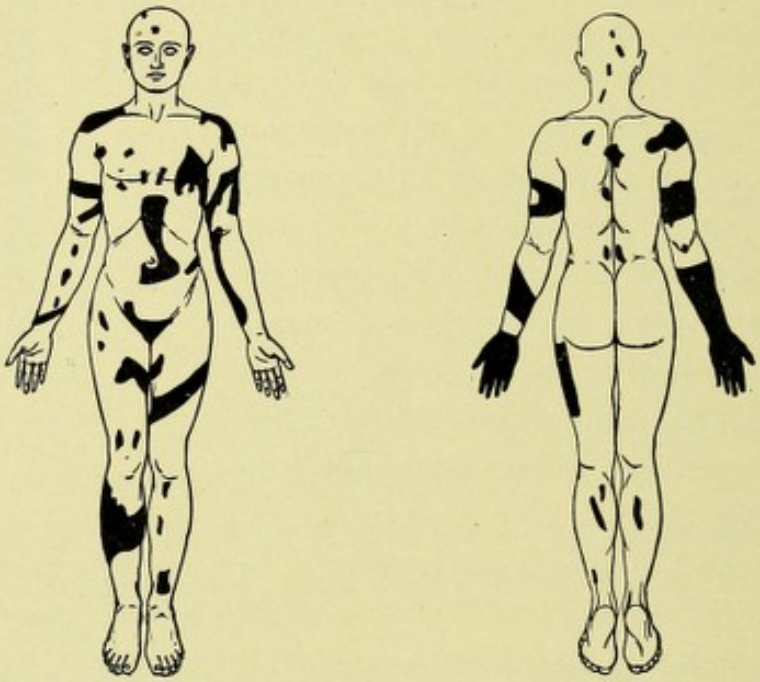

FIG. 88.—Disseminated anæsthesia. (After Pitres.)

and mucous membranes which take place after the hysterical attack, after strong emotions, or after the use of the magnet, are the results of suggestion. Most of the psychiatrical clinics of Europe are supplied with huge horseshoe magnets, which are used to demonstrate the mobility of hysterical anæsthesia.

Since Peterson has shown that there are absolutely no physiological effects from the strongest magnetic currents, it is an inevitable conclusion that the so-called magnetotherapy acts solely by mental impressions.

The cutaneous surface is the most frequent seat of anæsthesia. The mucous membranes, especially those of the eyelids and pharynx, the muscles, joints, and viscera, are also common locations. To the eye and to the touch hysterically anæsthetic skin is normal. It is not paler than normal skin, but for some unexplained reason it has a diminished tendency to bleed when pricked. In the anæsthetic areas the muscular sense is regularly abolished.

Hyperæsthesia and Pain.—These are sensory symptoms of hysteria for which examination is usually unnecessary. The patients complain of spontaneous pain in the hyperæsthetic parts, as well as of the suffering caused by slight jars or knocks. The pain may be very severe and lasting, as in the hysterical joint affections; or it may be transitory and mobile, like hysterical anæsthesia. Hyperalgesia is more frequent than spontaneous pain. The slightest contact may cause expressions of agony, and the patients are very unwilling to permit any touches on the hypersensitive parts. In such cases deep pressure is often well borne. The pain caused by touching the hypersensitive part is much accentuated if the patient knows that the test is about to be applied. It often happens that no pain is felt when the part is brushed or tapped while the patient's attention is fixed on something else.

The value of the Mannkopff sign is questionable in traumatic hysteria.

The situation of hyperæsthesia is variable. General hyperæsthesia is extremely rare. Hemihyperæsthesia may occur. Anæsthetic areas frequently contain hyperæsthetic zones. In the male the testicle, and in the female the ovarian regions, are

common locations of hyperæsthesia. In traumatic hysteria there are usually areas of hyperæsthesia which correspond in situation to the seat of the injury. This is the form of hyperæsthesia described in the local traumatic neurosis of Strümpell. If the back has been injured or twisted in any way there is often the pain of traumatic lumbago, although the spinous processes of certain vertebræ are frequently hypersensitive independently of lumbago. The clavus hystericus—the sensation as though a nail were being driven into the head—is an hysterical symptom generally familiar. In addition to these forms of increased sensitiveness, headache and pains along certain nerves, e. g., the fifth, the sciatic, etc., are common. In paroxysmal hysteria the areas of hyperæsthesia become hysterogenetic zones —that is, pressure upon them may induce a paroxysm, or pressure exerted during a paroxysm may cause it to cease.

Hysterical pain does not usually interfere with sleep, nor is it accompanied with the nutritional disturbances which are such common consequences of pain from other causes. It is a difficult symptom to explain. Janet suggests that it is entirely due to the ideas aroused by association relative to contact. Although, psychologically, the pain is probably not genuine, it is real to the patient, and may cause him much discomfort and suffering.

Special Senses

The Eyes.—Next to cutaneous anæsthesia, disturbances of vision are the most frequent of the hysterical stigmata. Apart from any possible preëxisting organic defects or disease, the eye is found to be anatomically normal. The conjunctivæ are very constantly anæsthetic, and the palpebro-conjunctival reflex is usually abolished. Examination of the pupils, of the optic nerves, of the ocular muscles, and of the refracting apparatus is, with few exceptions, negative. Nevertheless, vision may be

impaired (amblyopia) or there may be complete blindness of one eye (amaurosis). Bilateral hysterical amaurosis, on the other hand, is one of the rarest of hysterical symptoms.

When the eyes are examined with the perimeter, it is found that there is a peripheral limitation of the fields.

In the larger number of cases of hysterical amblyopia the peripheral limitation is slight, not exceeding ten or fifteen de-

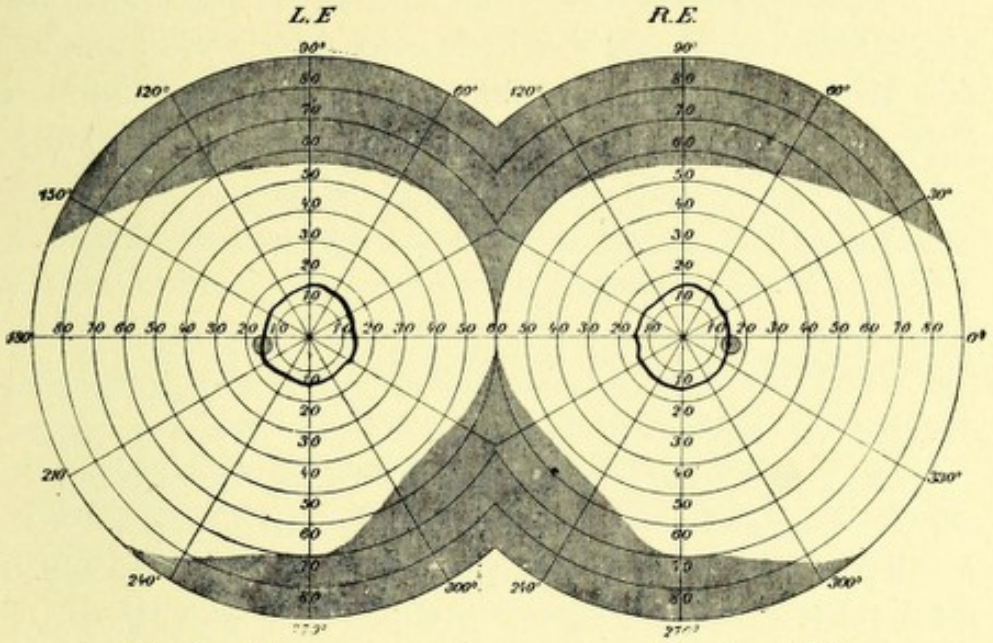

FIG. 89.—Extreme concentric contraction of the visual fields. (After Gilles de la Tourette.)

grees. In some cases, however, it may be very extensive, the perimeter showing that only a few degrees of the field retain their normal seeing power. When slight in degree, the contraction most commonly follows the periphery of the normal field, but when it is excessive it is concentric (Fig. 89). Both eyes usually share in the visual impairment, though it is customarily more marked in one eye than in the other. If there is unilateral paralysis or unilateral cutaneous anæsthesia of the body, the more affected eye will correspond to the paralyzed or anæsthetic side. At different examinations the extent of vision

often varies, but the variation does not follow the regular shifting type observed in neurasthenia. Several examinations are advisable, and they should be conducted with a view to rapidity rather than to minute accuracy. Since the field often changes as the result of any of the various hysterical accidents, or with the general condition of the patient, it has been called by Janet the "barometer of hysteria."

Frequently associated with this limitation of vision for white are characteristic abnormities in the color fields. With contraction of the visual field for white, the perception of the most internal colors may be lost, or when the contraction for white is only moderate in degree there may be a reversal of the normal color fields; thus red may be seen in a larger field than blue.

Visual disturbances, like those of general sensation, are frequently discoveries of the examining room. The acuity of central vision ordinarily remains unimpaired. Subjective ocular disturbances, such as flashes of light before the eyes, muscæ volitantes, etc., occur, but the patients often neither complain nor are conscious of any trouble with sight, and they are not subject to the disabilities resulting from contraction of the visual field which are present when it occurs from organic causes. Hysterical patients in reality see, although visual perception does not become known to the higher consciousness. Thus hysterical amblyopia, like other hysterical symptoms, is actually false, although, when the patient is conscious of it, it is real to him.

Although the patient may be unconscious of any visual disturbances, examination of the seeing power made of each eye separately may show that in one eye vision is impaired or lost. While it seems almost incredible that anyone can be totally blind in one eye without knowing it, examination of many cases has shown that unilateral hysterical amaurosis may exist independently of the patient's knowledge. That the apparently contradictory manifestations of hysterical amblyopia may be genu-

ine, and exist when the patient has no motive to deceive or to be "suggested," has been proved with certainty by Prince. He reports in detail a case in which, instead of there being anything to be gained by simulation, there were the strongest reasons why the patient should appear sound in every way, for he was a very zealous applicant for a position on the Boston police force. The ocular symptoms, together with other hysterical stigmata, were discovered at the time of his medical examination for admission.

Prince also reports unusual symptoms in a man who, as the result of an accident on an electric street car, developed hysterical hemiplegia and hemianæsthesia and ocular symptoms. Vision in the right eye was good, but on the left side (the hemiplegic side) there were amblyopia, color blindness, contraction of the field of vision, polyopia, a retraction of the near point, and an approach of the far point to a distance of about eight feet.

The visual disturbances were noticed only when the right (sound) eye was closed.

Hemianopsia is not a symptom of hysteria.

In addition to the pure disturbances of vision, hysteria presents a variety of anomalies in the motor innervation of the eye. It is a question of doubt and contention as to whether these consist of true paralyses, or of spasms of the individual muscles. Spasm of the eyelids, blepharospasm, one- or two-sided, may occur as an isolated accident, or be joined with other symptoms. It is usually accompanied by anæsthesia about the orbit. The lids are tremulous, thrown into wrinkles, and the spasm increased by attempts to open the eyes. In a peculiar and rare variety, known as ptosis pseudoparalytica, the upper lid alone is affected, thus giving the appearance of a third-nerve palsy. It is due to spasm, and differs from a true third-nerve palsy in that there is no wrinkling of the forehead on the affected side, and in that the lid itself, instead of being flaccid, is contracted. A few cases are on record in which the lid in hysterical ptosis is

flaccid rather than spastic, thus indicating a true hysterical palsy of the levator palpebræ. Of affections of the muscles which move the eyeball, Holden says:

"The extra-ocular muscles often exhibit a lack of balance, and particularly a varying latent divergence or exophoria, which may be regarded as a sign of fatigue of convergence. Again, a spasm of any of these muscles may give rise to diplopia, which might suggest an actual paralysis, but the anomalous behavior of the double images in different directions of the gaze and the presence of corroborative symptoms lead us to the diagnosis of a purely functional disturbance.

"Diplopia of another sort and of a purely psychical nature is absolutely characteristic of hysteria, and is a very valuable diagnostic sign when it exists. This is uniocular diplopia. With one eye closed the patient sees a light double; when it is carried a certain distance away from the eye, and when asked to count fingers, he sees double the number presented."

Holden reports the following case:

A man, thirty-five years of age, complaining of failing vision. The pupils were normal, the fundi were apparently normal, and retinoscopic examination showed the patient to be emmetropic. When asked to read the distant test letters he hesitated. His left eye was then covered, and I held up two fingers fifteen feet away from him and asked him how many fingers he saw with his right eye. He promptly answered "Four." When I held up four, he said "Eight," and so on. When the left eye was used alone he gave similar answers. The sensibility of both corneas was found to be reduced. It was then learned that he was bringing a damage suit against a company for injuries he had received several months before.

True, persistent nystagmus is not a symptom of hysteria, though a previously existing nystagmus is often increased after an injury. The general rule regarding the pupils is that they are normal in size and in reaction. At the same time, with pronounced spasm or cramp in the ciliary muscle, or with weak-

ness of convergence, the reaction of the pupils to light may be slow or absent. That the Argyll-Robertson pupil may occur in hysteria has always been denied. The disturbances in the motor apparatus of the eye just referred to sometimes develop in direct sequence to injury. More frequently, however, they make their appearance some time afterwards, when the psychosis is well developed. As symptoms they are shifting and variable in the extreme.

Hearing.—The commonest hysterical affection of hearing consists in a unilateral partial deafness on the side corresponding with the one which is also the seat of paralysis or cutaneous insensibility. Or there may be a blunting of hearing on both sides, in which case it is more pronounced on the side affected by other symptoms. In either case tinnitus and other subjective sensations may or may not be present. Complete deafness is rare in traumatic hysteria; when present it is usually associated with some organic ear disease. Deaf-mutism is very rare. Its characteristics regarding speech are the same as for simple mutism. The following case by Francotte illustrates the condition:

A man, thirty-five years of age, who had previously had hysterical accidents, was bitten on the leg by a dog. He was very much frightened, and so confused that he was taken in charge by the police. When he arrived at the station he had lost all power of speech, and was completely deaf. Francotte saw the patient nine days after the accident. During that time he had uttered no sound, and could only communicate with others by gestures, or understand what was addressed to him by writing. The case was cured by hypnotism.

The following personally observed case illustrates the method of development of deaf-muteness during the course of traumatic hysteria:

W. P., a previously healthy farmer, of good habits, on September 8, 1899, was in a cart which was run into by a trolley car. P. remem-

bers nothing about the accident. It is doubtful whether he jumped out or was thrown out of his cart; however that may be, he picked himself up and walked to the sidewalk, where he apparently became unconscious. He was carried to a neighboring house, where he had a violent convulsion. He struggled with a number of men who were standing around him, arched himself so that only head and heels touched the floor, and altogether gave a theatrical display. He then apparently became unconscious, and was carried to the hospital, where he remained in a stupid condition for twelve hours.

Examination at this time by the company's surgeon failed to show any contusion, wound, or other evidences of organic injury. The following day the patient complained of great pain down the right side. There were also twitchings of the muscles of the face and of the left arm.

In a day or two the pains switched to the left side, being complained of over the scalp and in the arm. He began to improve until September 21st, when a fire broke out in the hospital. There were great confusion and fright at the time, and the nurse came to P. telling him to get up and get out, or he would be burned up. P. told the nurse to save herself, as he would rather be burned up than get up. She half pushed and half pulled him up, and then he was carried out of the hospital. He declared that he was not frightened by this conflagration. He was taken to his brother's house, and for a week or two afterwards showed no ill effects from the fire. Early in October, however, he became deaf and dumb. He had said that ever since the accident it had hurt him to speak, and there seems to have been some difficulty in speaking before he altogether stopped talking. A week or two after becoming deaf and dumb the pain changed from the left side to the right, the patient stopped using the right hand almost altogether, and also the right leg, and from that time on he has held communication with others altogether by writing. He never lost the power of reading, and learned to write with the left hand very quickly. The above-described condition remained about the same at the time of the trial, in May, 1900. P. was brought to the court sitting in a chair. He looked well, had no paralysis about the face, but had an almost constant twitching of the muscles of the lips, neck, and left arm. The right arm was not moved at all; the right hand was supported in the left much of the time, the fingers of the right hand being held straight and slightly flexed on the metacarpus. When he first came into the court room he had an attendant put his right leg over the left, and he held it in that position without moving it for the two hours and a half he was on the stand. The questions

which were asked him in direct and cross-examination were written on pieces of paper, and P. would write his replies beneath them. He grasped the meaning of the questions at once, and wrote as quickly as could be expected of a man who had only written with the left hand a short time. His answers were inteligent and pertinent. He gave no special evidences of fatigue, and was not emotional. On one or two occasions it seemed as though he had heard what was being said, although I cannot be positive about this.

In May, 1901, there was little improvement. But a report received in 1904 stated that speech and hearing were perfectly restored, and that he walked without difficulty.

Smell and Taste.—Loss of smell or taste may be bilateral, and is usually associated with anæsthesia of the nasal mucous membranes and of the inner surface of the lips and of the tongue. Impairment of these functions is found under circumstances similar to those associated with anæsthesia occurring elsewhere.

In a recent Vanderbilt Clinic case, as a result of a bicycle accident, a young man lost for several months the senses of smell and taste. This case is particularly remarkable, as none of the more common hysterical stigmata were present. The visual fields were not contracted, the cutaneous sensibility was normal, and there were no paralyses. Inasmuch, however, as there had been no injury to the mouth or pharynx, or other gross injuries, we were obliged to consider the ageusia and the anosmia as functional.

Motor Symptoms

Paralysis.—Paralysis is an hysterical accident rather than a permanent stigma of the disease. Slight injuries frequently produce marked local symptoms in persons who are already affected by the disorder, or, as we have seen in discussing the ætiology of hysteria, the psychosis may appear for the first time after any accident in which the factors of fright and injury are prominent.

In the larger number of cases of traumatic hysteria the

local injury is trifling, or at best not severe. Hysterical paralysis may, of course, follow grave injuries, but clinical experience shows that the trauma itself plays only a subordinate part. The loss of muscular power is particularly prone to follow accidents in which the emotions have been actively called into play. This is the "traumatic suggestion" of Charcot. Ordinarily there is some little interval of time between the occurrence of the accident and the appearance of the paralysis. During this "period of meditation" the patient reflects upon his injury, and, through pain or discomfort, his attention is directed to the injured part until gradually or suddenly the power of voluntary motion in it becomes impaired or lost. The sudden paralysis of traumatic hysteria is rarely accompanied by the associated signs of paralysis due to organic lesions of the brain or spinal cord. Unless developed during an hysterical seizure, or unless the shock is extreme, there is no prolonged loss of consciousness.

When there is loss of power in a limb, usually some of the muscles escape, although all may be paralyzed. It sometimes happens that the paralysis affects certain muscular functions only, and that the pseudo-paralyzed muscles can perform other movements perfectly well. This is well illustrated by the condition first described by Blocq under the name of astasia-abasia. In this condition the patient has entire use of the legs for all movements except those necessary for standing or walking. He may be able to move the legs in bed, or to dance, or to hold himself on the toes, while all attempts at standing or walking prove ineffectual. On attempting them the patient falls immediately to the floor. This condition differs from most forms of hysterical paralysis in that there is no anæsthesia.

The possible medico-legal importance of astasia-abasia has been shown by Bremer:

A woman, forty-nine years of age, married, claimed that she had become paralyzed as a result of injuries received in an elevator. She asserted that by a sudden stopping of the elevator she had been thrown forward from the seat, striking her head, and that since that time her lower extremities were paralyzed. She brought suit for $20,000. The physicians who appeared on behalf of the plaintiff admitted that there was no interference with the functions of the bladder or rectum, or loss of sensation; but they asserted that while the patient was capable of moving her legs in every direction, and with apparently normal strength in the sitting or lying position, she was not able to either stand or walk. The expert for the defense accepted the symptoms as described by the plaintiff's physicians; but instead of attributing this peculiar form of paralysis to an injury to the spinal cord, he ascribed to it only such importance as belongs to hysteria. It was then shown that the woman had had previous hysterical accidents, and that the astasia-abasia was only a new symptom of the hysteria from which she had been suffering for years. The jury adopted the views of the expert for the defense, and gave a verdict favorable to the defendant.

The degree of hysterical paralysis varies. It may amount to nothing more than weakness which is universally distributed, a condition to which Charcot gave the name of amyosthenia.

Amyosthenia, however, is a permanent stigma rather than an hysterical accident. It is manifested by general muscular weakness, such as is seen in neurasthenia, but, unlike neurasthenic weakness, it is apparent only. In hysterical paralysis the patients in reality retain their muscular power, as may be proved by the muscular work they do. It is when their attention is drawn to the movements they are about to perform—as in dynamometric tests, or in performing movements at command—that the weakness is chiefly manifested. "It sounds like 'I will not,' but it really is 'I cannot will.'"

The degree of paralysis varies also at different times, being more marked at one examination than at another. As long as the disease remains active the loss of power usually

becomes progressively greater. Electrical reactions in the paralyzed muscles almost always remain normal, although a few cases with diminished electrical excitability have been recorded. Atrophy is rarely present, and never reaches an extreme degree.

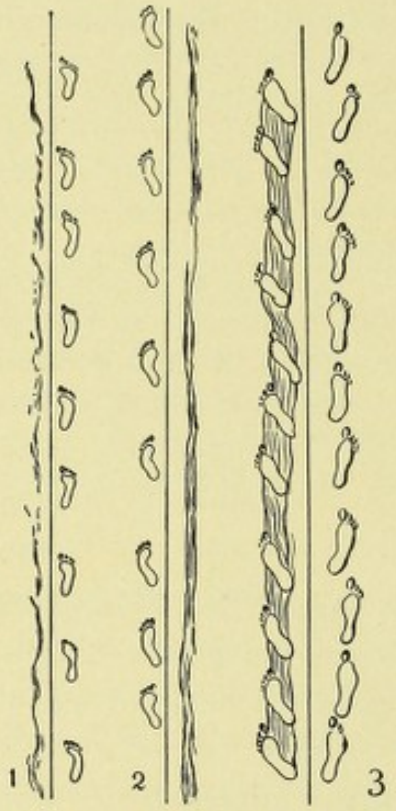

FIG. 90.—Impressions which illustrate the differences in the gait (1 and 2) of hysterical hemiplegia, and (3) organic hemiplegia of the flaccid type. (From Blocq, after Gilles de la Tourette.)

The most common forms of hysterical paralysis are hemiplegia, brachial monoplegia, and paraplegia.

HEMIPLEGIA occurs most frequently on the left side. It has many points of difference from organic hemiplegia—viz., the face escapes, although one buccal angle may droop, and there may be an associated spasm of the facial muscles; the leg is more involved than the arm; the knee jerks are not morbidly exaggerated; and foot clonus is absent, except in some cases in which the foot is contracted in extension. In organic spastic hemiplegia the mowing gait is a sign which the most inexperienced may interpret. The leg is circumducted in a very characteristic way. Even in flaccid hemiplegia, if the patient can walk at all, some use is made of the paralyzed leg. But in hysterical paralysis the leg is dragged as though it were an inert mass. This difference is well seen in the accompanying reproduction of footprints (Fig. 92). In organic hemiplegia anæsthesia is rarely an important symptom, while the anæs-

thesia of hysterical hemiplegia is pronounced, involving the whole of the paralyzed half of the body and usually associated with anæsthesia of the special-sense organs.

MONOPLEGIA is perhaps the most frequent form of paralysis observed in traumatic hysteria. The left side is the one usually affected. The loss of power may involve the whole limb, or, as is more frequent, only certain segments or muscles are paralyzed. The arm is much oftener involved than the leg; it may hang down from the shoulder as an inert mass, or the shoulder may escape, and only the movements of the hands be impaired or lost. The symptomatology of hysterical brachial monoplegia has been established through the work of Miura, who studied 31 cases in which this condition was a symptom. In 15 the paralysis was of traumatic origin, and followed the injury immediately, or was separated from it by a "meditative" period. The extent of the motor loss varied from weakness of wrist and fingers to complete paralysis of the whole arm. When weakness of the leg was added to the brachial monoplegia there was an associated hemianæsthesia. In 9 cases there was hemianæsthesia with pure brachial monoplegia. In other cases the anæsthesia was limited to the paralyzed arm, so that it was bounded superiorly by a line perpendicular to the long axis of the extremity. In 1 case there was no anæsthesia at all.

The following case, reported by Lebrun, is interesting, as it shows both the possibility of hysterical monoplegia developing from slight injury, and the occurrence of convulsive attacks as a complication:

A young man, eighteen and a half years of age, when examined for military service, was found sound in every way and was enlisted. A few weeks later, while being vaccinated, he was accidentally pricked by the lancet on the left elbow. He fell unconscious almost immediately, and on the succeeding day had several more attacks of unconsciousness. In a few days after the injury, which was obviously

very trifling, power in the left arm began to fail. He was released from duty, and, as he was becoming worse, he voluntarily sought the hospital, where he was seen by Lebrun, who made the following notes:

"Drooping of left shoulder. The left arm hangs inert; when told to move it, the patient lifts it a little, with effort, but lets it fall again. He can hold nothing in the hand. Electrical reaction normal. No atrophy. Total anæsthesia of the left arm, extending superiorly a little above the shoulder. There is a concentric limitation of the visual fields, most marked on the left side. The testicles are hysterogenetic zones. The attacks are ushered in by violent headache, buzzing in the ears and throbbing of the temples, and by the globus hystericus. The face becomes congested and anxious in appearance. Inspiration is prolonged and difficult. Expiration is short. This, the first period of the attack, lasts about one minute. It is followed by clonic spasms, the *arc en cercle*, and other histrionic attitudes and movements. This period closes by the patient bursting into tears. In these attacks consciousness was only partially lost. The patient understood what was going on around him, but could not reply."

This patient was apparently a confirmed hysteric, since he had been paraplegic in childhood as the result of a slight injury, and had always been suggestible. In his case the slight injury caused by the lancet was sufficient to call into action the dormant psychosis.

Although the arm is by far the most frequent situation of hysterical monoplegia, other parts may be the seat of localized paralysis. Hysterical monoplegia of the leg is not rare. Also a few cases of hysterical paralysis, with anæsthesia, limited to the one side of the face, have been observed.

Paraplegia.—When hysterical paraplegia results from injury or shock, its onset is usually sudden. It is particularly liable to be complicated by the development of contractures, which may be permanent. The loss of power may involve the whole of both limbs, rendering all motion impossible, although more commonly some motor power is retained, so that the legs or feet may be moved about in bed.

The reflexes are often active, except the plantar reflexes, which are frequently lost. Gilles de la Tourette says that in-

continence of urine and fæces may occur. This is certainly very unusual. Ordinarily the patients retain perfect control of these functions. If control is lost, the presumption of organic mischief becomes very strong.

The anæsthesia in hysterical paraplegia usually extends from the waist downward, involving all parts except the genital organs. This rule is not invariable, for the genital organs may be hyperæsthetic or anæsthetic, or other parts may retain their sensibility. A case detailed by Souques illustrates this:

A man, twenty-nine years of age, without discoverable nervous taint, was very much frightened and then knocked down by a horse. He was thrown against the curbstone, thus receiving a contusion of the hip, and immediately went into coma which lasted two days. In eight days he was able to walk with difficulty, but soon went into coma again, which lasted this time five days. He lost for a time the power of speech. Then he began to have nightmares and hallucinations relative to the accident.

Physical examination at this time showed complete anæsthesia (Fig. 91) below the waist, with the exception that there was only a diminution of sensibility of the genital organs, and parts of both feet were normally sensitive. The muscular sense was lost. Movement was almost entirely abolished in the lower extremities. The patient was unable to flex the thigh or lift the heel from the bed; but he could be seen to attempt these movements, and the muscles also could be seen to contract. Slight power of flexion and extension persisted in the toes. There was absolutely no resistance to passive movement. The knee jerks were diminished, but present on both sides. The scalp was hyperæsthetic. Left glossolabial hemispasm. The superior extremities were weak, but all movements in them possible. Slight tremor. Limitation of the visual fields. Loss of the sense of smell on the right side; the sense of taste abolished on both sides. While under observation this patient had an attack of mutism and dyspnœa.

Polyplegia.—Hysterical paralysis of all four extremities is unusual, but sometimes occurs.

Sérieux reports the case of a young girl, fifteen years of age, who, after a severe fright, fell into convulsions and hallucinatory delirium,

during which her arms and legs received slight bruises. A few days later all four extremities became paralyzed, the degree of the loss of muscular power in any limb being proportionate to the severity of the injuries the limb had received during the convulsive attacks. The paralysis was flaccid, and there was a diminution of electrical reactions and of tendon reflexes. The muscles were the seats of

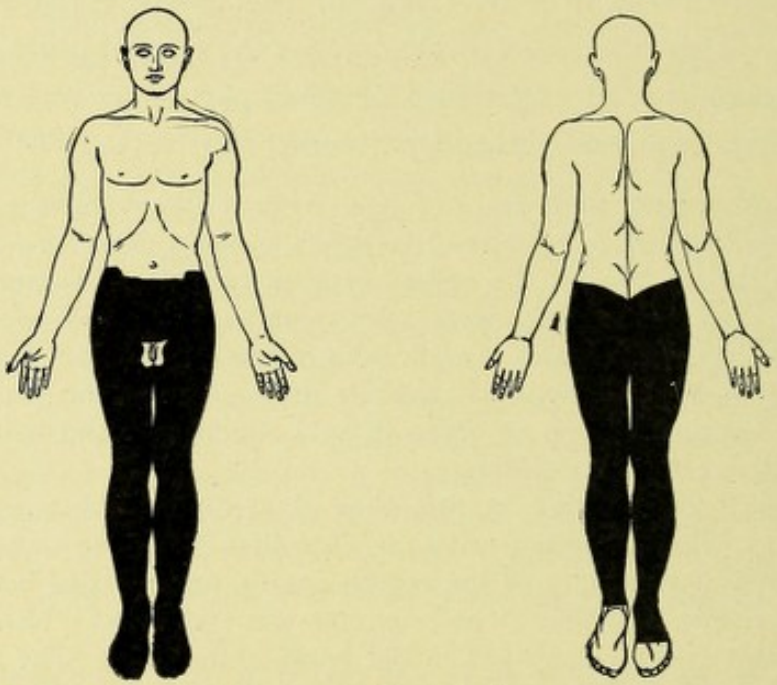

FIG. 91.—Anæsthesia in a case of hysterical paraplegia. (Souques.)

quick, shocklike contractions, resembling those of electric chorea. Anæsthesia was at first generally distributed, but was very variable. The visual fields were contracted, and the smell and taste were lost.

The symptoms eventually passed away, except a left hemi-anæsthesia.

In parts which are the seat of hysterical paralysis or anæsthesia the muscular sense is lost, as may be demonstrated for individual limbs by the ordinary clinical tests. Unsteadiness of standing or walking is sometimes apparent at once. This may take the form of ataxic movements of the leg, such as occur in locomotor ataxia. The ataxia may be more or less pronounced than in tabes. On standing still, with the eyes closed, there may

be considerable swaying of the body. Some hysterical patients cannot stand at all with closed eyes. In a recent case at the Vanderbilt Clinic the ataxia was most marked when the patient tried to get up from a chair. These disorders of movement may occur independently of paralysis. The ataxic and incoördinated movements seen in hysteria often obscure the diagnosis, and render a proper comprehension of the case impossible until examination has shown the evidences of organic disease to be absent, or has revealed the presence of some of the hysterical stigmata.

Thus, in a recent Vanderbilt Clinic case, the patient's chief complaint was of an inability to button his clothes. The movements of the fingers were uncertain and ataxic, resembling in this respect the incoördination seen in general paresis. These symptoms had occurred subsequently to a fall from a horse car. Examination failed to reveal any paralysis or other evidences of organic disease, but the mental condition of the patient was characteristic of traumatic hysteria, and there was a generalized blunting of cutaneous sensibility, which in both hands and over the left side of the body was very pronounced.

Similarly, an active farmer, fifty-five years of age, was thrown from a trolley car, and soon afterwards became depressed, apathetic, and emotional. A suit for a large sum was brought against the company. My examination showed a left hemianæsthesia, weakness in the muscles of the left side, diminution of the visual field, and similar manifestations of hysteria. There were no evidences of structural lesion. Uncertainty in movements of the legs was very marked. The man could not, apparently, walk straight, but would stagger to the right. On standing with closed eyes he would fall toward the right, although he never let himself go entirely. His wife said she had observed him when he was working in the field, and that he would then stagger to the right, though he never fell.

Contractures.—In hysteria, tonic and continued spasm may occur in almost any of the muscles, but is most frequent in the muscles of the extremities. The contracture may develop suddenly as the result of any provoking agent, but usually it comes slowly, becoming gradually more and more extensive and se-

vere. It may complicate paralysis, especially paraplegia, or may occur when there is no loss of power except such as is imposed by the rigidity of the muscles. Hysterical contracture may so closely simulate the contractures due to degeneration of the pyramidal tracts that it is sometimes impossible, from morphological appearances alone, to distinguish between the two conditions. The state of the muscles themselves, however, is very different in the two conditions. In hemiplegia, or in disease of the spinal cord, the contractures come on gradually; it is many weeks before they are very pronounced, and even then they can be overcome by moderate force. It is common in neurological wards to see a hemiplegiac straightening out the fingers of his paralyzed and contracted hand; but in hysteria the contractures are vise-like, not to be overcome by any moderate degree of violence. Both sets of muscles surrounding a joint may be so tense that the joint is held absolutely motionless. Also in organic injury to the pyramidal tract increase of tendon-reflex activity, with ankle clonus, is the rule, and often occurs, soon after the accident, on the unparalyzed as well as on the paralyzed side. On the other hand, there is rarely any great exaggeration of the tendon reflexes in hysteria. A still more valuable sign for differential diagnosis is that hysterical contracture may appear after injuries too trifling to induce any morbid conditions of organic character. The appearance of this form of immobility, after a slight injury, in a strong and apparently healthy man is well shown by a case of Berbez (Obs. 9):

The patient was a robust blacksmith, thirty-four years of age, the father of four children. The man had always been apparently in perfect health. He denied all history of ancestral or personal nervous stigmata. While at work he was slightly burned on the left hand and forearm by a red-hot iron. The wound was not deep, but took six weeks to heal, and left a scar 10 or 12 cm. long and 3 or 4 cm. wide, on the back of the hand and lower part of the forearm. There was

no particular fright connected with the accident, but three or four days later the patient found that the fingers of the left hand were getting stiff and beginning to feel numb. This condition of stiffness and numbness continued for seven weeks, when, on arising one morning after a rather sleepless night, the patient found his hand in the attitude of *main en griffe*. This position was gradually changed into one of flexion of the wrist and fingers, with pronation of the forearm (Fig. 92).

The shoulder and upper arm were not involved; the forearm was pronated. The hand was flexed on the forearm; the four fingers were so tightly flexed that the nails dug into the palm of the hand. The fingers were tightly pressed together, and the thumb was firmly fixed

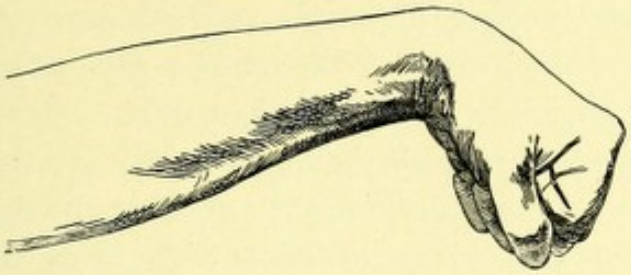

FIG. 92.—Hysterical contracture. (After Richer and Berbez.)

on the external surface of the second phalanx of the index finger. The contractions, which persisted at night, were so pronounced that all efforts at reduction were unsuccessful. Hysterical stigmata were eventually added to these symptoms. The left side of the body became the seat of a hemianæsthesia, and the taste, smell, and hearing were much blunted on the left side. Both visual fields were contracted, but most markedly on the left side. The color fields were, however, not reversed.

At first magnetotherapy was able to induce a return of cutaneous sensibility to all of the left side except the parts involved in the contracture. They remained anæsthetic. A later attempt caused the sensibility to return to the left hand and arm, but was followed by a contracture of the sound (right) hand.

Hysterical contractures may exist only for a short time and disappear suddenly, or they may be persistent, and sometimes they are permanent. They do not relax during sleep, though they may often be made to do so by suggestion, and almost always disappear during ether narcosis. When affecting the

arms or legs they usually assume forms different from those of organic contractures. In a common type of contracture of the arm, when either the arm alone or the arm and leg are both involved, the arm is drawn across the chest, the forearm is partly flexed, the wrist is flexed, and the fingers are tightly closed in the hand. The hand may assume various positions from the preponderance of the contracture in certain muscles. The position for holding the pen, due to spasm in the interossei, is not an uncommon one.

When contracture affects the lower extremities they are usually held absolutely immobile and straight. Not uncommonly a contraction of the calf muscles produces a condition of equino-varus with flexion of the toes.

Although in paraplegia the legs are most frequently held in rigid extension and adduction, they may assume the same type of flexion as is seen in organic disease of the spinal cord.

In some cases the diagnosis of hysterical contracture is very puzzling, although it may usually be made by examining the reflexes (if the contractures do not make this impossible), the condition of the muscles themselves, and by looking for other associated signs of hysteria.

Local and persistent spasms may occur in individual muscles or muscle groups. They are less permanent in character than contractures of the limbs. Some, such as trismus, in which the jaws are held tightly together without injury to the tongue, are rarely seen outside of general attacks.

As mentioned, one side of the face may be the seat of spasm, giving rise to the appearance of paralysis of the other side. Spasm of the tongue occasions difficulties of speech. In the muscles of the neck it may cause the position of torticollis. Spasm or contracture of the back muscles may produce considerable spinal curvature. Contracture and swelling of the abdominal muscles are the causes of phantom tumors.

Tremor is an almost constant symptom of hysteria following accident. In its most common form it is a fine, rapid tremor of the fingers and hands, such as occurs in neurasthenia. It may sometimes imitate the tremor of paralysis agitans or the coarse incoördinated movements of multiple sclerosis. It becomes more marked under the influence of excitement and fatigue, and usually disappears during sleep. In location it may be generally distributed, though it is more commonly limited to certain groups of muscles.

There are also a variety of morbid movements commonly regarded as hysterical in character, although in many of them other hysterical stigmata are absent. Among these may be mentioned the rhythmical chorea of Charcot, paramyoclonus multiplex, and a series of incoördinated movements of forcible and shock-like character.

In Charcot's rhythmical chorea, or chorea major, there are alternating contractions in opposing groups of muscles, especially in those of the hand and trunk. Another variety has been called electrical chorea, from the sudden and forcible choreiform muscular contractions. It differs in course and in geographical distribution from the disorder named, after its first describer, Dubini's disease.

Schütte has recently reported a case of paramyoclonus multiplex which seems to have been one of the ultimate developments of traumatic hysteria, and which shows how long a time may elapse between an accident and the appearance of exaggerated symptoms:

The patient was a man, fifty-two years of age, having no hereditary nervous taint, who had always been well and strong. He fell some little distance, striking his head. The accident was followed by weakness, dizziness, a feeling of pressure in the head, and similar subjective symptoms. The patient, soon after the accident, was pronounced as unable to work, and entitled to the indemnity which the German law allows in such cases. For eight years he presented

the ordinary symptoms of hysteroneurasthenia. At the end of this time he was admitted to the hospital, suffering from the complication of paramyoclonus multiplex. The hysterical stigmata which had been previously observed had now disappeared, and the general condition was one of neurasthenia, to which had been added morbid muscular movements. These movements were more marked in the muscles of the trunk, and consisted of fibrillary twitchings and forcible clonic contractions. The arms and legs were involved to a less degree. On making intended movements many muscles not necessary for the movement were called into play. During sleep, and when the patient was at rest, the clonic spasms ceased, but the fibrillary twitchings continued. The sleep was disturbed. The tendon reflexes were active, and there was a slight bilateral foot clonus. It was an apparently typical case of paramyoclonus multiplex, and is one of the few recorded examples of that symptom-complex resulting from trauma.

Hysterical Joints.—Hysterical joint affections were recognized and described by Brodie, and since his time have become familiar pictures to surgeons. They are frequently the sequelæ of accidents, after which they may appear immediately, although there is usually the intervening meditative period. Their development, when it once begins, is usually rapid. In more than one-half the cases the knee is the joint affected; after it, in order of frequency, come the hip, wrist, shoulder, and ankle.

The most prominent manifestation of hysterical joint affections is pain, which is very much intensified by any movement. Discriminating examination shows, however, that the hyperalgesia is in the skin rather than in the joint itself. The hypersensitiveness is not necessarily limited to the region of the articulation, as in organic disease, but may involve the whole limb. The position in which the limb is held does not always indicate the functional nature of the trouble.

The skin is not reddened, and œdema is rare.

The muscles around the joint are usually the seat of contracture, with or without paralysis, and in this situation also there are geometrical areas of anæsthesia.

In regard to diagnosis I cannot do better than repeat a remark of Pitres's relative to an hysterical coxalgia: "Absence of redness and swelling, little or no spontaneous pain, no sensitiveness on percussing the heel, neither retraction of the muscles nor morbid positions, which is the organic lesion that could persist for nearly two years with a similar category of negative symptoms?"

Hysterical joints are freely movable in ether or chloroform narcosis—a fact, however, which can rarely be demonstrated in medico-legal cases. It is well shown in a case of Charcot's:

The patient was a man, forty-five years of age, with negative personal and ancestral history. While working at a circular saw the machine got out of order, so that he was tossed fifteen feet in the air, coming down on the buttocks. There was no loss of consciousness nor fracture or dislocation of any bone. The left foot was slightly bruised, but the patient got up and walked a short distance. Very soon, however, he found that he could not touch the left foot to the floor without much pain in the whole left leg. The left leg soon became so contracted that the knee jerk on that side could not be obtained, and movement at the hip became very limited. When the patient was sitting, the left lower extremity was extended, not touching the floor. On attempting to rise, the muscles of the arms were called into play; in order to avoid movement of the thigh muscles when standing (Fig. 93), the whole weight of the body rested on the right leg, causing the left side of the pelvis to be tilted up. There was also considerable curvature of the spine, the convexity being toward the right; the left leg and thigh were slightly carried forward, so that the great toe of the left side was 20 cm. higher than that of the right. Measurement showed no real shortening. The movements at the hip joint were very limited and caused expressions of great pain. The surrounding muscles were rigidly contracted, and the overlying skin was insensible to pricks. There were many other hysterical stigmata. The man was put under deep chloroform narcosis, when it was found that all movement at the hip could be freely made. When the patient first recovered from the effects of the anæsthetic, movements at the hip were painless; but when his attention had rested a short time on the affected joint the old train of symptoms returned.

An hysterical joint may remain useless for weeks or months. When of long duration the condition is liable to be seriously complicated by organic anchylosis, so that the joint may remain motionless, through the formation of adhesions, long after all the hysterical symptoms have passed away. Under the influence

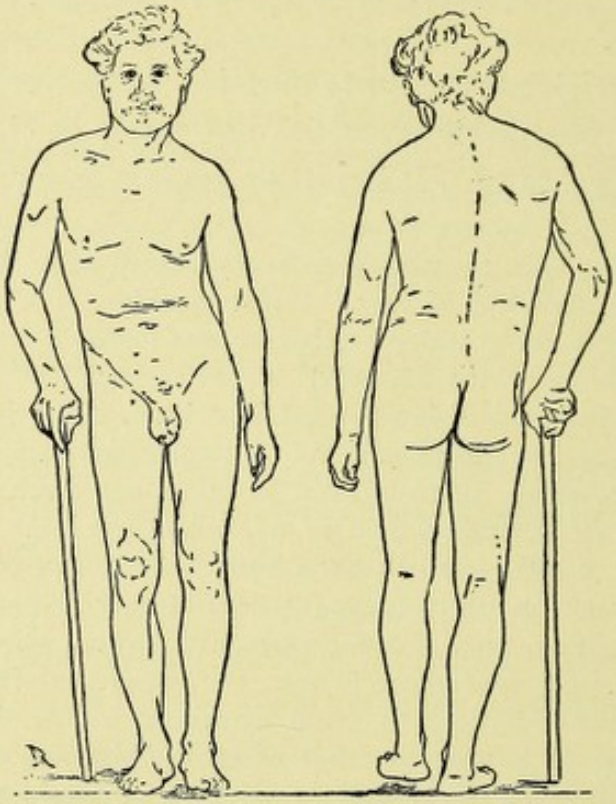

FIG. 93.—Attitude in a case of hysterical coxalgia. (After Richer and Berbez.)

of emotions the hysterical joint affections, if recent, may disappear suddenly. In such cases relapses are frequent.

Reflexes.—Pathological exaggeration of reflex activity is not the rule in hysteria, although the tendon reflexes are often overactive. Foot clonus is rare, yet it may occur in hemiplegia and paraplegia. When present, it is associated with rigidity (usually in extension) of the limbs. Hysterical clonus is usually not difficult to differentiate from organic clonus. The contractions lack the rhythm, the force, and the persistence of the or-

ganic variety, and give evidence of being voluntary movements. Clonus of this character occurs, according to some authors, in twenty per cent of all cases.

Contrasted to this spurious clonus, not uncommon and easy to recognize, is a form seen in rare cases of hysterical paraplegia. This form is identical with organic clonus, and can only be differentiated from it by other symptoms. For such a differentiation the Babinski reflex is most serviceable, though it should not be forgotten that slight organic clonus may exist without the Babinski reflex.

The tendon reflexes may be diminished, though never lost, in hysteria. Diminution is particularly frequent when anæsthesia is present as well as paralysis. The plantar and conjunctivo-palpebral reflexes are commonly absent, but the abdominal and cilio-spinal are sometimes retained, even when there is cutaneous anæsthesia.

Sphincters.—Impairment of sphincter control in hysteria is very unusual. The paroxysm passes without involuntary micturition, and the various forms of paralysis are usually unassociated with either rectal or vesical disturbances. There may be a frequent desire to urinate, due to psychic rather than to local conditions, and spasm of the neck of the bladder, resulting in retention of urine, occurs in some cases of paraplegia. The mucous membranes of the bladder and rectum rarely, if ever, lose their sensibility. If they do, it is easy to understand how involuntary passage of urine or fæces might occur. But the most competent observers admit that loss of sphincter control is among the most unusual of hysterical accidents.

The Hysterical Attack.—The *grande attaque* of the French consists of four periods: (1) Epileptiform; (2) period of clownism; (3) period of passionate attitudes; (4) period of delirium. Although it has been observed in other continental countries, it is only in France that the grand attack is often seen in its full

sequence of development. Bernheim calls it the " attack of culture." It seems probable that the aggregation of so many hysterical patients as are assembled in the Salpêtrière may have furnished suggesting influences for the induction of exaggerated symptoms. In this country the hysterical attack is uncommon. Knapp estimates it as occurring in ten per cent of traumatic cases. Even then it never follows the complete type, but is manifested by the epileptiform period alone, or by emotional excitement, or by other parts of the grand attack. The term hystero-epilepsy, which has been applied to the convulsions of hysteria, is a misnomer which has led to much confusion. The disease is either hysteria or epilepsy, or the two diseases occurring simultaneously in the same individual. Although in many cases these two diseases occur together, and although it may be impossible to distinguish between them, it is better to state that such is the case than to employ a misleading and indefinite nomenclature. There are marked differences between epileptic and hysterical fits. In epilepsy the convulsion is of short duration, is not necessarily preceded by an aura, and occurs very frequently at night. The duration of an hysterical attack may be a half hour or more. An aura is constant, and nocturnal occurrence is exceptional. Biting of the tongue and involuntary passage of urine (almost constant phenomena in epilepsy) do not occur in hysteria. During the seizure the pupil almost always reacts to light in hysteria, not in epilepsy, and after the epileptic attack the knee jerk is lost, which is not the case in hysteria.

Furthermore, the convulsive movements of hysteria have an exaggerated character; the period of tonic contraction is longer than in epilepsy; opisthotonos is common. At the expiration of the epileptic attack the patient commonly goes into the condition known as post-epileptic stupor; from the hysterical epileptiform attack the patient usually recovers immediately.

The attacks of hysteria may follow any of the causes capable

of inducing other hysterical phenomena. They are sometimes very frequent, occurring many times a day, or at certain hours of each day. If they take place once, they will usually recur. After an attack new symptoms may appear, or existing stigmata be made worse. Convulsive hysteria is a grave form of the psychosis.

A not uncommon equivalent of the hysterical attack is the so-called hysterical coma. The cases I have seen were in men who were brought to the hospital absolutely unconscious. They were limp, inanimate masses, incapable of being aroused by any stimulation. The vascular and respiratory functions were normal. After a varying number of hours the patients would wake up and walk home.

From the frequency with which they are found lying senseless in the street, many of them come to be individually known to ambulance surgeons, who, when they pick them up, recognize both the condition and the individual.

Hysterical coma may in many ways resemble apoplexy, especially if it be complicated by hysterical hemiplegia. The functional character of the affection, however, usually becomes evident if all the factors concerned are carefully examined.

In a case of Comby's, a nervous woman, thirty-eight years of age, without previous hysterical accidents, fell senseless as a result of a flash of lightning which killed two of her children who were standing near her. The woman was comatose for four days. When consciousness returned she was hemiplegic and hemianæsthetic on the left side, a condition which disappeared entirely in three weeks. Two years after this, during a thunderstorm, the patient again became comatose for seven or eight hours, awakening to find herself again hemiplegic and hemianæsthetic. In two weeks she was again perfectly well. Three years later a similar sequence of symptoms was observed by Comby. The patient was picked up unconscious in the street and brought to the hospital. The coma lasted four hours. Examination then showed: Facial expression good. No fever. Appetite excellent. The whole left side was completely paralyzed. The

left arm lay immobile by the side of the body, and when lifted up it fell lifelessly to the bed. The patient was unable to raise the hand from the bed or to spread the fingers apart. There was a slight paralysis of the lower branch of the left facial nerve (this is a rather uncommon though not an impossible symptom of hysteria); the movements of the tongue and of other muscles of deglutition and of articulation were also impaired. Knee jerks present. No foot clonus. All forms of sensibility of the skin and mucous membranes were completely abolished on the left side. Vision, hearing, smell, and taste were also absent on the left.

In fifteen days all these symptoms disappeared, and the patient walked out of the hospital apparently as well as ever.

General Somatic Symptoms

Most of the general somatic conditions met with in traumatic hysteria are identical with those already described under neurasthenia. Some, however, have characteristics of their own and merit mention.

In the respiratory apparatus a variety of anomalies occur, generally explainable by weakness or spasm of muscle. Mutism, with or without deafness, is essentially a psychic condition, and operates independently of spasm or paralysis of any of the muscles of phonation. It is an hysterical aphasia, rather than a physical interference with phonation. The patients can make sounds, but have forgotten how to articulate words. They can write and read, and, if deafness is not present, can understand.

Aphonia, on the other hand, is produced by physical intervention. The muscles of the larynx, and especially of the vocal cords, are in a condition of spasm or of weakness. The result is that in complete aphonia the patients can make no sounds; in partial aphonia the voice is hoarse. Both mutism and aphonia generally come on suddenly.

The respiration is ordinarily normal, but may become very rapid from emotional causes.

Attacks of dyspnœa, approaching even to asthma, are also

occasionally seen. The patients breathe very rapidly; they become much alarmed, and complain of oppression and need of air. At the same time there is no cyanosis. In other cases the spasm is essentially expiratory, showing itself in recurring cough or hiccough, or the production of strange noises.

Exaggerated symptoms referable to the gastrointestinal tract, such as phantom tumors, hysterical abdominal pains, etc., are important in nontraumatic hysteria. But in the traumatic variety the chief gastrointestinal symptoms are the same as those of neurasthenia. Constipation is common. The same is true for cardiac disturbances, including the attacks of palpitation and the subjective sensations referred to the heart and throat.

Disturbances referable to the peripheral circulation, such as cyanosis, œdema, etc., are not common. As is well known, hysterics do not bleed easily, severe needle punctures oftentimes, especially in anæsthetic areas of the skin, producing no bleeding.

A variety of skin eruptions have been described by French authors. They are rare at best, and possibly many of them own a deeper underlying cause than hysteria. There seems little doubt, however, that vesicular eruptions may result from hysteria also.

A young woman injured her wrist. The surgeon, undecided as to whether a fracture existed or not, used the x-ray for diagnosis. A few days afterwards the arm gradually lost its strength, and ten days later there appeared a vesicular eruption running up the entire anterior surface of the arm and out upon the breast. My examination showed a typical hysterical monoplegia of the arm, with profound anæsthesia running out to the breast. The eruption, in no way resembling an x-ray burn, and coinciding with the anæsthesia, was regarded as purely hysterical in character.

The urine shows no characteristic chemical changes in hysteria. There may be temporary anuria, but the passage of large quantities of pale urine is more common.

Hysterical fever is a disputed symptom, though many cases

are recorded by reliable observers. It is said to follow the hysterical attacks.

The vertigo of hysteria is somewhat different from that of neurasthenia. It is much less frequently complained of than in neurasthenia, the patient acting rather than complaining.

There are, however, a variety of hysterical disturbances of equilibrium and motion which might be mistaken for objective vertigo, especially in view of the readiness of assent characteristic of hysteria. The patient would readily, if questioned, agree that peculiar cerebral sensations were the cause of the objective vertigo. Thus in hysterical hemiplegia the tendency to stumble, to stagger, or even to fall, toward the paralyzed side is often pronounced. In some patients of this class the mode of progression is perplexingly similar to that of cerebellar disease.

In a case of traumatic hysteria seen several years ago, I was quite unable to make the patient stand steadily with the feet together. He would persistently sway toward the right (the paralyzed side), until finally he was forced to put out the right foot to save himself from falling. In walking, also, he made leeway toward the right. The morbid ideas in this patient were so fixed that his peculiarities of locomotion were practically the same whether he thought himself observed or not.

In some cases of convulsive hysteria the attack is characterized by vertigo. This is the pseudo- or hysterical Ménière's disease.

The nutrition in hysteria may suffer considerably. Often, on the other hand, the patients, in spite of paralysis or other incapacities, remain well nourished.

Medico-legal Relations

Both clinical and social factors render traumatic hysteria, when it is the subject of a personal-injury claim, the most difficult of all diseases to be interpreted with judicial fairness.

Even when every suspicion of dishonesty on the part of witnesses, experts or others, is eliminated, the difficulties standing in the way of arriving at a fair adjustment or verdict are very great.

So unique is it in its medico-legal relations, that a few words regarding these latter seem essential. In the negotiations which take place between the injured person and the responsible party soon after the accident there are so many legal and economic considerations, in addition to the purely medical ones, that it is hardly worth while to speak of the difficulties of settlement. But when a case is finally brought to court it remains to be ascertained, after the liability of the defendant is established, what the nature, degree, and probability of permanency of the injuries are and the amount of financial compensation to which the plaintiff is entitled by them. These are the questions which the jury has to decide.

The psychosis is nourished upon suggestion and introspection, means for which are so profusely furnished by the excitement and observation attendant upon court proceedings. It is entirely consistent with its nature that existing symptoms should become worse or vanished ones return on such occasions. It is not necessary to assume, in explanation, any voluntary exaggeration or simulation on the part of the patient. The effect of this clinical idiosyncrasy on a jury, however, is disastrous to the cause of the defendant. The twelve jurors have heard from the medical experts of the two sides testimony too often directly conflicting. On the one side the opinion has been expressed that the patient's condition is due simply to nervousness aggravated, if not caused, by the suit, and that the symptoms will soon subside when the legal proceedings are at an end; on the other side, the belief has been sworn to that the injury is of organic and irreparable character, or else, if perchance its functional nature is admitted, that the nervous

system has sustained a shock from which it can never recover.

The jurors may be convinced of the honesty of all the views they have heard expressed, and yet they are unable to determine from the character of the testimony which of the opposing opinions is the more likely to be correct. They are therefore obliged to rely upon the impression made upon them by the injured person himself. They see a person in an even worse condition, perhaps, than his doctors had depicted. They see an alleged paralyzed limb absolutely motionless; they become witnesses of an emotional outburst more harrowing than any related in the evidence. And they see these things one or two years after the accident has occurred. Their natural inference is that the injuries are permanent. They find it hard to believe that the outlook for a malady which has so long defied the resources of medical skill is anything but hopeless. They are unwilling, if not unable, to believe in the unreality of physical symptoms. They cannot comprehend a part being the seat of paralysis or insensibility unless there is some grave physical defect behind it; they do not know that a limb which is immobile to-day may be in wonted activity to-morrow. Thrown on their own resources by the contradiction in medical testimony, they render a verdict in accordance with their own impressions as to the plaintiff's injury. Their impression is that of a person severely and probably incurably injured; and their verdict, rendered accordingly, is generally in excess of anything to which the plaintiff is entitled.

Prognosis

That death may occur as a result of hysteria uncomplicated by organic disease appears to be a matter of considerable doubt, although such a possibility is admitted by most authorities.

Gilles de la Tourette quotes several cases in which hysteria is said to have been the cause of death. The immediate causes in the cases least open to criticism were due to spasm of the throat and of the stomach, and from exhaustion induced by long vomiting.

A patient at St. Luke's Hospital several years ago presented the hysterical symptoms of hemianæsthesia, contraction of the visual fields, and epileptiform convulsions, but we could discover no evidences of any organic disease. The man was found one morning dead in bed. The autopsy revealed no causes such as are usually regarded as adequate to induce death.

There are, however, so far as I know, no cases of hysteria reported as fatal in which microscopical examination had excluded visible pathological alterations of nervous tissue. That careful and thorough examination in accordance with modern microscopical methods is necessary before it can be said that a nervous disease is functional, has been thoroughly demonstrated by the history of Landry's paralysis, a disorder which formerly was thought to be characterized by no discoverable lesions, but which has recently been shown to be due to degeneration of the ganglion cells in the spinal cord and in the brain.

We may take it, therefore, as a safe working rule, that hysteria does not cause death. Also, as it does not destroy nervous tissue, the disease is recoverable. This statement does not imply that recovery always occurs, or is even necessary. Such is far from being the case. It simply means that, so far as our knowledge goes, no irreparable destruction underlies the manifestations of hysteria; and that no symptom of hysteria, however serious, cannot completely and permanently disappear at any time. Zein reports a case of hysterical paraplegia in which recovery took place after nineteen years.

Recovery and cure are terms in the use of which caution is always necessary, and at the outset must be defined what is

meant by them in regard to hysteria. For practical purposes we assume a hysteric to have recovered when objective symptoms have been absent for a year or longer, when the patient is able to resume his occupation and to lead his ordinary life in apparently good health, and when subjective complaints have materially diminished. This may be taken to represent substantial recovery.

A variety of factors must be taken into account in considering the prognosis of hysteria. The disease varies greatly in development and in course with different surroundings; so that what is true for one race or for one country, is not necessarily true for others. This is especially evident in traumatic hysteria. In America, for example, a large proportion of the cases of traumatic hysteria develop in sequence to accidents which may be actionable; and while such actions are pending, no patient with traumatic hysteria may be expected to get well. But when the legal part of the case is finished, either by settlement or by verdict, a most potent cause for the continuance of the hysterical symptoms is removed, and within a year or so of the verdict improvement or cure can generally be counted on.

The German system is in direct contrast to this. By the German system of indemnity the money question ceases only when the symptoms do. Traumatic hysteria in Germany, therefore, is a more hopeless malady than it is here.

Predisposition is a much less important factor in traumatic hysteria than when trauma does not enter into the ætiology—a fact which renders the prognosis of traumatic hysteria materially better than in the nontraumatic variety. My experience with traumatic hysteria has been that in a very large proportion of cases no predisposition to nervous or mental disease is to be discovered. It is true that litigation tactics often render inquiry as to such factors unsatisfactory, if not misleading. But when due allowance is made for unavoidable imperfec-

tions in investigation, the fact remains that the larger majority of the victims of traumatic hysteria have as good family records as most people, and were quiet, orderly, healthy citizens up to the occurrence of the accident.

Age is an important factor. In young children the physical symptoms of hysteria almost invariably pass away in the course of a few weeks. In a little girl of five years, whom I saw four years ago in consultation, there developed, in consequence of a fall on the face, a complete right-sided hemiplegia. Recovery was perfect within four weeks, and the child has been well ever since. Contrasted to the almost universally favorable prognosis in extreme youth is the grave one in old age. Hysteria is infrequent in old people, but that it occurs is well attested. In them it is apt to be a rebellious and persistent affection, sometimes showing no amelioration whatever.

Between the two extremes of childhood and old age, if not seriously handicapped by heredity, recovery can be confidently looked for in the vast majority of cases.

The previous condition of health of the victim of the accident has important bearings upon the severity and duration of the hysteria. In the healthy and well nourished there is naturally more hope that the psychosis will be temporary.

Preëxisting chronic diseases, on the other hand, make it more difficult to throw off hysteria.

Alcoholism and arteriosclerosis seem particularly unfavorable to the recovery from all functional nervous diseases. The question of sex is generally supposed to be an important one in traumatic hysteria. Most authorities agree that men recover less frequently than women. In my experience with litigated cases men are much more frequently attacked. But there is hardly sufficient data to decide that in them recovery is less probable. It seems to me that the question of sex is very subsidiary to other factors.

The fundamental cause of hysteria is to be found in the mental state, and it is consequently to the mental state we must look for the most reliable guiding lines to prognosis. The physical manifestations indicate to a certain degree how far-reaching the effect of the accident has been upon the mind. Thus a monoplegia indicates less serious psychic disturbance than hemiplegia, and monoplegias usually recover rapidly. Paraplegia, as a result of trauma, is distinctly rare, but is usually a rebellious affection. Morbid movements, such as twitchings, choreiform spasms, hiccough, cough, and the like, usually yield readily to suggestion. Anæsthesia and limitation of the visual fields are such important symptoms that without them the diagnosis of traumatic hysteria is open to doubt. They give a clew to the mental state by their intensity, by their extent and by their distribution. Shifting, inconstant, and partial anæsthesia is of much more favorable omen than the anæsthesia which is profound, and which settles itself in one place and stays there.

Of the affections of the other special senses our information is too meager to justify any broad generalizations as to prognosis. In my experience disturbances of taste, except when complicating hemianæsthesia, have occurred exclusively in the mildest cases. Hysterical blindness and deafness may be persistent affections. Aphonia is usually of a few days' duration only. From one of the cases described, it will be observed that hysterical deaf-mutism may persist without improvement for four years after all legal questions are at an end.

The physical symptoms just enumerated are without doubt useful indices as to the severity of hysteria. But they are indices only, and may very well have taken their particular form from some trivial causative factor or some chance environmental circumstance. They may change completely in character or in distribution, and still leave the patient as badly off

as before. It is a mistake to be guided too much by physical symptoms in attempting to reach a prognosis in this peculiar malady.

The mental state is the fundamental cause, and the patient's purely psychological characteristics of attention, of memory, of mood, emotion, and temperament, of will, and of conduct, are the most important prognostic considerations.

In every case of traumatic hysteria these mental attributes undergo change. In the extent of the change, and its permanence, is the key to prognosis. In other words, the prognosis of any individual case of hysteria demands a psychological study of the individual rather than a simple enumeration of the physical manifestations he may present.

These factors in the prognosis of traumatic hysteria in America are only of value in nonlegal cases or in legal cases when the law question is at an end; for with pending litigation hysterics improve little or not at all. It is the legal delay, usually of two years or more, which is largely instrumental in making hysteria a chronic affection. With legal questions out of the way, with no marked predisposition, and with the patient still in the active period of life substantial recovery may confidently be looked for in the vast majority of cases, although not in all. Recovery is often slow, requiring a year, or even longer, before the patient can return to work; and cases are on record in which a much longer time was necessary.

If, immediately after an accident, a patient could be isolated, could never hear the question of damages mentioned, and could be cared for by a physician who understood the symptoms, I think we would hear very little of persisting traumatic hysteria. Such ideal conditions, however, are rarely possible. In most cases the patient gets too much sympathy; his complaints are received as though they represented real organic troubles, and instead of being put on a rigid disciplinary rou-

tine, he is allowed to follow all the fancies and vagaries of the confirmed nervous invalid. He is thus fed on indulgence and suggestion, which is the food on which hysteria waxes fat.

After-histories of 14 personally observed cases of traumatic hysteria in which damages for personal injury were claimed:

The following cases are cited in brief for the purpose of advancing, as far as they can, knowledge concerning the ultimate outcome of litigated cases of traumatic hysteria. As in only a few was a personal examination after the trial possible, they cannot be taken as being the final word in prognosis. At the same time, the inquiries were carefully undertaken, and the information obtained is, to the best of my belief, correct. They may be accepted, therefore, as throwing some light on the after-career of hysterics who sue for damages.

I. *A. B. vs. Trolley Company.* Healthy man, fifty-six years of age, thrown from car, November 10, 1896. No serious physical injury. Depressed mental state. Gave up work. Left hemiplegia and hemianæsthesia; contraction of visual fields. Case unsettled four and a half years after accident. No improvement in symptoms.

II. *R. vs. Pennsylvania Railroad.* Healthy woman, thirty-nine years of age. In a collision, May 31, 1897. Badly frightened and jarred, but no discoverable physical injuries. Inattentive mental state; slight left hemiplegia and hemianæsthesia. Generous settlement in 1898. She continued her occupation of actress. Last heard from in season of 1900–1, when she was playing in a stock company.

III. *S. vs. B.* Young girl, twenty-two years of age, in elevator accident on October 25, 1897. Severe fall, but no physical injuries other than bruises. The accident was soon followed by a violent hysterical outburst, followed by left hemiplegia. Violent hysterical outburst at the trial, at which she appeared on crutches. Verdict, $11,500. Six months after trial she was married. A year later said to still use a cane in walking.

IV. *F. H. vs. Erie Railroad.* Examined June 1, 1899. Left hemiplegia, with hysterical contractures, in a previously healthy man, as result of a collision by which he was thrown to floor of a car. Had done no work since accident. Liberal settlement, six months after which the man returned to work.

V. *H. B. vs. Trolley Company.* Young man, twenty-two years of age, touched an electric trolley wire with left hand. Said he received a severe shock, but was not burned. Left hand and arm be-

came the seat of hysterical paralysis. Settlement for a small sum, after which the patient recovered rapidly, and continued perfectly well at last report.

VI. Young Hebrew, twenty-three years of age, touched a live electric wire in August, 1898, with an umbrella he was holding in his hand. No burn, but hysterical contracture developed at once in left upper extremity. Within a year received a verdict of $500, since which time he has been working, and is apparently well.

VII. *Poiner vs. Norfolk (Va.) Street Railroad Company.* This case has been described in full on page 469. Up to May, 1901, there had been little improvement; but in December, 1904, he was reported as having fully recovered speech and hearing, and as walking without difficulty.

VIII. *J. K.* Bohemian, twenty-four years of age, in March, 1900, was thrown from his cart on his head. Scalp wound. No fracture. Depressed and inattentive mental state, with left hemianæsthesia and incapacity for working. In January, 1901, verdict, $1,400. In May, 1901, he looked well and was working at full wages. He still complained of dizziness and backache.

IX. *E. vs. New York Central and Hudson River Railroad.* Girl, twenty years of age, always nervous and delicate, in rear-end collision in November, 1903. Badly shaken up and frightened. Symptoms at time of my examination consisted of anæsthesia and great emotional instability. A physician was in almost constant attendance. Settlement early in 1904, after which time all medical services were dispensed with.

X. *Kanen vs. Central Railroad of New Jersey.* Boy, twenty years of age, in Westfield wreck in January, 1903. Was violently thrown from the car and rendered unconscious. Fracture of third and fourth lumbar vertebræ. Symptoms consisted of pain in the back, a paraplegia of the legs of gradual onset, and anæsthesia, also of gradual onset, which ultimately involved the whole body except the head. The verdict rendered at the first trial, in May, 1903, was reversed, and a new trial ordered. At the second trial, in October, 1904, there was considerable difference of opinion among the medical witnesses, some maintaining that the paraplegia was organic, others (including myself) that it was hysterical. Verdict, $15,000. In December, 1905, there was improvement but patient still unable to walk.

XI. *E. vs. Central Railroad of New Jersey.* Woman stenographer, twenty-eight years of age, also in Westfield wreck. Badly shaken up, and rendered momentarily unconscious, and perhaps had a slight convulsion. Chief symptoms consisted in left hemianæsthesia,

dragging of left leg in walking, and pain in the back. Settlement in July, 1903. Report in February, 1905, that she had entirely recovered, and was doing her work again.

XII. *G. vs. Central Railroad of New Jersey.* Woman, fifty-eight, also in Westfield wreck. Was delirious several days after the accident. Chief symptoms at time of my examination, April, 1903, were pain in the back, pain and tremor of right arm, and paraplegia of the legs, with anæsthesia. Settlement in December, 1903. Shortly after, she married and moved away.

XIII. *Alleson vs. Brooklyn Heights Railroad.* Woman, between twenty and twenty-five, recovered, in June, 1898, at the second trial, $8,000 for injuries sustained four years before. She did not get the money herself, some member of her family being said to have made away with it. She is at present in the Kings County Hospital, under the care of Dr. A. C. Brush, subject to a great variety of disabling hysterical symptoms. She says she has never been able to work since the accident.

XIV. *Bennett vs. Brooklyn Heights Railroad.* Athletic and strong young woman, received a verdict of $17,500 in June, 1901. The injuries consisted of a right hysterical hemiplegia and hysterical amblyopia, consecutive to a trolley-car collision in September, 1899. The plaintiff married in 1903, and for the past year has been, according to her father, in perfect health.

CHAPTER IV

INSANITY FROM PSYCHIC SHOCK

Difficulties of Recognition—Delirious Characteristics—Illustrative Cases.

In insanity, with its complex and little-understood ætiology, it is well-nigh impossible to single out any one factor as the sole cause. When emotional factors, which operate here more effectively than in somatic diseases, are the assigned causes, the opportunities of error are great. Some forms of insanity, such as general paresis, in which we know there is an ætiology distinct from the emotions, begin, in sequence to a nervous shock, with symptoms of delirium. When insanity begins in persons with marked predisposition, any apparent exciting cause loses greatly in importance. Recurrent psychoses set in quite commonly after the most trivial and (in themselves) inadequate causes; and many other emotional disturbances in the predisposed occur in a way to make it evident that the symptoms are not the products of any exciting cause, but are merely the fluctuations of deep-seated disease, which appear either independently of exciting causes, or as the results of such disharmonies of existence as all mankind must undergo.

In studying the literature of this subject it is remarkable how few cases there are in which insanity can legitimately be regarded as the result of nervous shock, the test being absence of predisposition, absence of other causes, and a nervous shock of great intensity. In view of this fact, the effect of nervous shock in the genesis of mental diseases generally must be left

to treatises on psychiatry. Certain cases of delirium, however, seem to result from such causes alone. They are the only ones to receive notice here.

Delirium is characterized by psychomotor unrest, insomnia, clouding of consciousness, incoherence of speech, to which may or may not be added hallucinations, illusions, and unsystematized delusions.

While the pathology of delirium from nervous shock is entirely in speculative domains, there are strong reasons to suppose that it is closely connected with disturbances in the cerebral circulation. Certain it is that evidences of disturbed circulation are always present.

It is plain that the symptoms of delirium from nervous shock are strikingly similar to those of the insanity characteristic of head injuries. As it is highly probable that traumatic delirium is not due to the mechanical disarrangement of brain constituents, but to changes in blood supply and pressure, and in brain pressure, it seems reasonable to suppose that traumatic delirium and the delirium from emotional causes are closely allied in pathogenesis, as they are in their clinical aspects.

The following is a synopsis of a few ILLUSTRATIVE CASES:

CASE I.—This case possesses the advantage of being full in the important details bearing upon the patient before, during, and after the accident. The accident was the tunnel disaster in New York City on January 8, 1902, which was the result of a rear-end collision on the tracks of the New York Central and Hudson River Railroad. The patient, W., had been, prior to the accident, an active man, without hereditary taint, and with no history of syphilis or alcoholism. He was fifty-four years of age, married, with healthy children. He had built up a large and lucrative business, in which he was the senior partner. He had general supervision of the business, made all the estimates for orders, and had personally conducted large exhibits at expositions, the last being the exposition at Buffalo. The following account of the accident is from his own recollection of it. It has been substantiated in all important details by statements of

others. W. was in the rear car near the middle, seated alone, and was absorbed in planning the day's work when the train stopped. In about five minutes there was a terrific crash. The rear end of the car seemed to be lifted up and turned completely over. The top was broken in by the top of the tunnel. The next thing W. knew he was lying by the side of the engine that had run into the train from behind. He was pinned between the brick wall of the tunnel and the boiler of the engine in a space not more than two feet wide. He was lying by the side of the boiler, with his legs pinned down and fastened in the *débris*. His feet were caught between the seats, which were jammed so closely together that he could not put his flat hand between them. The engineer asked if he should not back the engine, but was told that he would tear them all to pieces if he did. The steam escaped in volumes, and the heat was almost unendurable. The fear of being scalded and of fire was frightful. There were two ladies back of W., one of whom was screaming terribly, being badly lacerated, with ear hanging, and nose being driven up into the head. Her head was hanging through one of the arches of the tunnel, and she kept calling for some one to keep her from falling. W. reached back with his left hand, and with great difficulty straightened her up. He held her in this position for at least three-quarters of an hour. There were others near him, screaming, one with the top of the car apparently resting on the top of her head. She screamed that she was dying. The steam came constantly up into her face. W. tried to lift the top of the car, but could not. There were several dead bodies in the *débris* close by. In about an hour two men came with a crowbar and began to pry open the seats. The ladies were taken out first, and the roof of the car cut open to let the steam out. The lower part of both of W.'s legs were crushed, and he could only lift them by taking hold of the lower part of his trousers. He was put on a stretcher and lowered to the tracks, carried up the tracks a way, and then up the steps and into a Bellevue Hospital ambulance. His suffering was almost unbearable, and lasted until he became unconscious. He did not regain consciousness until in the hospital.

At Bellevue Hospital W. was found to have compound fracture of both bones of both legs, without other surgical injuries. There were no scalp wounds or contusions about the head, and there is no reason to suppose that he had sustained any cranial injury. The day after admission to Bellevue Hospital he received a good many visitors, and on the afternoon of that day he went into a condition of partial stupor. After this condition had existed for a day or two he was transferred to a private hospital, in the care of Dr. John B. Walker,

in consultation with whom I saw him on January 13, 1902. While in the private hospital, prior to my examination, his sleep had been much interfered with; he was out of his head most of the time, and muttering constantly, much of which was unintelligible. He was restless, but the crippled condition of his legs prevented him from getting out of bed. He would hold both hands up in front of his face, and say that he saw the headlight coming down on him all the time. There was no vomiting. To the questions I put to him at the first examination he gave no answer, and it was only with difficulty that he could be induced to perform such simple movements as putting out the tongue, etc. He lay in bed with eyes closed most of the time. From time to time he would suddenly open them, raise himself up from the bed, hold his hands out in front of him with face averted, but would say nothing. For two days he had to be catheterized, and on one occasion had an involuntary passage of urine in the bed. The temperature never reached 100° F. There were neither suppuration nor pulmonary symptoms to explain the mental condition. The pulse varied from 80 to 108. There was no paralysis of any cranial nerve nor of the hands. Both legs were in splints, so the knee jerk could not be examined. Two days later the condition was practically the same. In response to questions, the patient would sometimes make inarticulate replies, and sometimes would not answer at all. He still made the movement of putting his hands before his face. He picked at the bedclothes, and his eyes wandered about the room. On January 16th he was somewhat better. He spoke to me, put out his hand, said "How do you do?" and asked what my name was. He said that people were constantly coming to see him, but he did not know who they were. Said that the attendants had tied him down so tight yesterday that he could not make anything out of it. Many of his remarks were incoherent, but he evidently recognized people in the room, and appreciated the fact that his mind had been clouded for some time. On January 18th his mind was somewhat clearer, although he still did not know where he was, and could not remember names. He was tested at this examination for reading and writing. He was unable to read, and could not write intelligently at dictation. I next saw him on January 22d. On that day he did not know the name of the hospital where he was, said he "remembered me from having seen me between two points in the North Shore." He said that one of his legs was splitting up into three branches, and the other had a "tap" on it. He thought he was setting out flowers. On January 29th there was evidence of great physical exhaustion, shown by very low tension pulse and weak systole. The mental state

remained about the same, although at night he was apt to become very much excited. He began slowly to improve, and in the course of a few days his mind became very much clearer. On February 8th, however, he developed erysipelas, and with the fever accompanying this infection the symptoms of delirium returned, and were present for ten days or more. I did not see him again until June 13, 1902. He had left the hospital, had no further symptoms of delirium, and was able to get about a little on crutches. He said at that time he was sleeping well, that he only waked up two or three times during the night, and slept until five o'clock in the morning. This amount of sleep was still below his normal standard. His appetite, he said, was good; he had no headache, but was entirely unable to endure physical or mental effort. He had great tremor of the tongue and of the hands. He was able to read understandingly, but was inclined to slur over many words. Many simple words he did not understand, and some he could not read correctly. Thus, for *colleagues* he read *collect,* for *repudiation* he read *reputation,* for *directorate* he read *directors.* In writing there was evidence of great tremulousness, and in very simple sentences he forgot to dot his i's. He left out letters, he left out words, and some letters he seemed entirely unable to make. Of simple problems given him in addition, multiplication, subtraction, and division, the addition was correct, the multiplication was left incompleted, the subtraction was wrong, and the division he was unable to do. Asked to subtract, mentally, 31 from 66, he said it was 29. In counting small change he made mistakes. Asked what day of the month it was, he said it was June 14, 1892. He then looked around with an air of uncertainty to see if he was right, and corrected himself to 1902. His memory for the time preceding the accident was perfect, and that for the time immediately after the accident good; but from shortly after admission to Bellevue Hospital memory was very defective.

My next examination, made on February 11, 1903, gave practically the same results as that of June, 1902.

My last examination, made October 15, 1903, showed improvement in many ways, but no real change in fundamental conditions. He made the same mistakes in arithmetical problems, in reading and writing. With the exceptions previously noted, his memory was perfectly clear, although he was uncertain about many of his facts. He had returned to business, but was unable to take any active part in it. He said that he made so many mistakes in writing that he now never wrote a letter unless some one read it over afterwards, and that the making of estimates, which formerly had been his specialty, he was

now entirely unable to do. He was unable to take orders over the telephone, as he had difficulty in comprehending what was said rapidly and could not remember the orders when the telephonic communication was over. From the earliest period of his illness his mental state became worse on any concentrated effort. When he began to clear up a bit from his original delirium, a few moments' conversation would cause him to relapse again into delirium. This condition of ease of fatigue is still present. He can do the first arithmetical problem quickly, and with better chance of being right, than he can the fourth. There have never been any hallucinations other than those incidental to the delirium, and never any fixed delusions. I feel no hesitancy in saying that exaggeration played no part in the case. There was none of the exaggeration of the ordinary neurasthenic type, as he vigorously combated and resented the idea that his mentality was affected. Neither was there any of the exaggeration so common in litigation. He took little interest in his claim for damages, which was settled without trial.

Case II.—Mrs. S., twenty-seven years of age; had always been normal mentally; gave no history of hereditary taint. She had been happily married for five years. One miscarriage, no children. On December 22, 1897, she saw her husband, to whom she was very much attached, walking on the street with a young and comely woman. From the other side of the street she watched the pair until she saw them disappear in a house. She waited, and soon saw a light in one of the upper rooms of the house and her husband come to the window and draw down the shade. When the husband returned to his own house, less than an hour afterwards, he found his wife in tears and unwilling to be comforted by any explanations he had to offer. She said her heart was broken, and she could never trust him again. She did not sleep that night, and the next day was still more disturbed. The condition of insomnia, grief, and agitation became progressively worse. I was called in consultation five days after the occurrence. The patient was in bed, in seemingly good physical condition. There was no fever. She had passed no urine for twenty-four hours. The mental picture was that of confusion. The patient did not know where she was. Did not recognize that Dr. Ely and I were physicians, but took us for some of her former acquaintances. Said at one time that she had not seen her husband (who was in the room) for six weeks; at another, for three months. She had the illusion that strange people were in the room. When asked questions, would make irrelevant replies. The occurrence which was the cause of the attack seemed to have disappeared from her mind. She

could not be aroused to a realization of her surroundings. This condition of affairs lasted for two or three weeks; then the patient began to improve and made a perfect recovery. Up to the present writing she has had no relapses.

Case III (Binswanger, "Ueber den Schreck als Ursache psychischer Erkrankungen," *Charité Annalen*, 1879).—Binswanger reports a case of a woman who, although always timid and easily frightened, had never shown symptoms of mental alienation. There was no insanity in her family, and she had healthy children. On learning that one of her tenants had hanged himself in her house she fainted, but soon recovered herself. She then began to cry and bewail and could not be comforted. She continued to cry and moan, day and night, for five days, and then she began to wander in speech and to throw herself about the room. She neither slept nor ate, and paid no attention to those about her. A little later her attention could not be aroused; the sounds she uttered were no longer intelligible; she sat rigidly with head between knees, offering resistance to any interference, including attempts to feed her. Ten days after the fright the bowels and bladder were evacuated involuntarily; the patient had not slept, but was tired and restless. On the eleventh day she died in coma. The autopsy showed multiple punctate hemorrhages in the cortex, in the cerebellum, and on the base of the brain. Such pathological changes as were present in the cerebral blood vessels were distinctly atrophic in character. There was no local arterial disease. The walls of the arteries were, however, weakened throughout, and Binswanger explains the case by assuming a sudden rise in blood pressure from the fright.

Case IV (Landgraf, *Friedreich's Blätter für gericht. Medecin*, 1885, xxxvi, 458–465).—A little girl of seven and a half years, a normal child apparently in every way, was frightened by two boys dressed up as ghosts. She rushed home, crying, "Oh, my eye! my eye!" Without giving further explanation she sank exhausted to the floor, vomited, and went into severe general convulsions. These continued till the child died, less than ten hours later. The municipal report (Bayreuth) stated that the autopsy showed no anatomical cause of death other than an excessive distention of all the blood vessels of the brain and cerebral membranes.

Case V (Manson, *Lancet*, 1846, ii, 582).—A girl, two and a half years of age, previously normal, was greatly frightened on October 2d by a boy in a mask. She could give no explanation of the cause of her fright. She trembled violently, and all she could say was, "The bogie! The bogie looked through the window at me!" Her

health declined rapidly. On October 14th she was restless, calling "Bogie! bogie!" The next day she did the same, and went into convulsions, which were confined to the diaphragm and fáce; sobbing and sighing, that evening she died. The autopsy showed great congestion of the brain and its membranes.

Case VI (Reibal, *Gaz. Méd. de Strasburg*, 1877, xxxvii, 86–91).—A physician was suddenly forced to leave his house which had been set fire to by a bomb. He soon developed terrifying hallucinations, with insomnia and agitation, which gave place to coma and paralysis. He died in five days.

Case VII (Reibal, *Op. cit.*).—A boy of ten was frightened by a cannon ball which passed close to his head. He ran home, but was immediately taken with chills, vomiting, delirium, and convulsions, and died in a few hours.

The above-cited cases give a general idea of the character of the symptoms, and the variations in their severity, which result from nervous shock in presumably normal persons.

The mental symptoms are essentially delirious. In severity they vary from slight and transient attacks of mental confusion to profound disturbances of consciousness, with the physical symptoms entailed thereby.

In outcome there may be complete and permanent recovery, or recovery with defect, or, as shown in the very instructive case of Binswanger, the symptoms may become progressively worse and end fatally.

Into more specific statements regarding the clinical behavior of these cases it seems impossible to go. They are, in the first place, too infrequent to permit of generalization. Further, the two causal factors, the exciting moment and the individual acted upon, are so extremely variable that a large number of collected and assorted instances would be necessary before any definite deductions could be drawn. As has been said, such a number of cases is not available.

CHAPTER V

UNCLASSIFIED FORMS

Ætiology—Symptoms: Mental; Motor, of the Deep Reflexes; Sensory, of the Special Senses; General—Pathology—Diagnosis—Prognosis.

In the preceding pages it has been repeatedly emphasized that the diagnosis of the traumatic neuroses depends upon the possibility of a clinical separation between organic and functional nervous diseases. When an organic lesion is caused by injury certain definite signs, which are ordinarily absent in functional disorders and which point to injury in definite areas of nervous tissue, usually present themselves at once. Yet while it is generally possible to draw the dividing line with reasonable exactness, and to infer from the circumstances of the accident and from the character of the symptoms the probable nature of the pathological process, there remains a group of cases which cannot be satisfactorily classified. Although they present many of the symptoms of hysteria and neurasthenia their clinical course is more serious than is customarily observed in these disorders, and some symptoms are present which, while not giving positive evidences of definite focal injury, are different from those usually brought about by the impaired mentality of hysteria or the overfatigue of neurasthenia. These are transition types, and bear testimony to the artificial character of the distinction between "organic" and "functional" disease. They have received various names and have been the object of much pathological speculation. By Dana they are called "the grave traumatic neurosis," on account of their unfavorable prognosis. Crocq denominates them as "the grave traumatic neurosis with probable organic le-

sions." Knapp, reasoning from the analogy of the organic character of some of the symptoms, from certain correlated experiments on animals, and from the results of a few human autopsies, goes still farther, and gives to them the name of "traumatic sclerosis."

As clinical occurrences they are, in my experience, distinctly rare. Of the large number of patients with nervous diseases coming to the Vanderbilt Clinic in the past few years, none is recorded as belonging to this class. Dana also considers them infrequent. Knapp, on the other hand, has met with them more often. Some, but not all, of the cases recorded by Oppenheim in his "Traumatic Neuroses" may properly be classed with these unusual forms. Relative to frequency it may be remarked that his cases, although they were collected during a period of eight years from the large nerve clinic of the Charité, in Berlin, were only 42 in number. It is partly owing to Oppenheim's description that so much confusion has arisen concerning the nature of the conditions which most frequently result from injury and shock. Many persons have inferred that the symptomatology, as detailed by the German neurologist, was intended to apply to all the affections embraced in the term traumatic neuroses. Such an interpretation, however, was not Oppenheim's intention. Although cases similar to some of those which he describes are infrequent, that they occasionally occur there can be no doubt. My own experience with them has been slight, and in describing them I shall be obliged to rely chiefly upon facts as reported by other observers.

Ætiology

The accident which gives rise to these cases has usually been severe. Considerable injury of the body is the rule. Psychic shock is sometimes, but not always, present. In a case of

Knapp's, which ended fatally, "a woman was dragged in a railway collision, her head and back bumping over the sleepers." Such accidents as are of not uncommon occurrence in factories, in which the body may be seriously lacerated by revolving machinery, or tossed about by explosions, are most frequently followed by the serious forms. Falls on the head from great heights, or any accidents in which the victims are subjected to very severe concussions, contusions, and wounds, constitute their most frequent starting points. From the comparative immunity from exposure to such injuries among the well-to-do classes the grave traumatic neuroses are most commonly observed in laborers and factory hands, or in persons whose occupations constantly expose them to danger. For similar reasons these disorders are most frequent in men.

Symptoms

The onset of the symptoms is in many respects similar to the onset of simple traumatic hysteria and neurasthenia. On account of the severe injuries to the body there is an increased frequency in the occurrence of physical shock, and the patients often remain unconscious for a considerable length of time after the accident. If there has been extensive laceration of the body, the character of the nervous symptoms may not show itself until some time after the wounds have begun to heal. It then becomes evident that it is more serious than is ordinarily met with in accident cases in which acute organic injuries to the nervous system are absent.

Mental Symptoms.—Of the mental symptoms, depression is often present in a marked degree. The patient sits immobile and inattentive, with many of the evidences of profound depression. He apparently takes no interest in his condition, his prospects, or his family. If spoken to, he replies, but his re-

plies are monosyllabic or brief. He rarely presents the mutism of severe melancholia, yet he is impatient of all interference, and wishes to be left quietly alone. He may be seen crying quietly by himself. Suicide has occasionally occurred. In some cases the picture resembles dementia rather than melancholia. The patient becomes apathetic and childish, with considerable impairment of intellectul capacity, caused by loss of memory and lack of attention. The amnesia is chiefly for recent events, although it may be so pronounced that it is impossible for the patient to distinctly recall happenings of previous years. He forgets names and words, but usually remembers faces. All details of the accident may be entirely forgotten, especially if the head injury has been severe. The lack of attention does not appear to be a lack of attention to outside matters, such as is commonly seen in introspective mental states. The patient appears to be thinking not so much about himself as of nothing at all. It is an inhibition of psychic processes rather than a diversion of thought into morbid channels. The mental state resembles that of hysteria more closely than it does that of neurasthenia, so that the patient is not the querulous, petulant invalid, but presents rather the type of the apathetic man, prematurely old, who is suffering from a serious disorder affecting the mind, which is characterized by a limitation of all intellectual processes.

Such a mental state renders the patient incapable of prolonged mental effort. He is entirely unable to work, and often cannot keep his attention sufficiently concentrated for the performance of the simplest tasks.

The psychic condition is often revealed by the facial expression. The face is pale, often of a grayish appearance, and the lines in the face may be drawn. The expression is indicative of absent-mindedness or of depression.

Disturbances of motion are the most frequent and the most pronounced of the physical symptoms.

Tremor is constant, and appears in various ways. It is usually a tremor which disappears when the muscles are at rest, but is very much intensified during the execution of intended movements and under the influence of excitement or fatigue. It may be nothing more than an exaggeration of the common neurasthenic tremor, in which form it is most commonly observed in the hands. The patient finds it difficult to perform the finer coördinated movements; the hands may tremble so much that it is only with difficulty that he can button his clothes. Similarly, the handwriting is often illegible from the uncertainty and irregularity of the pen strokes. In other cases the tremor of the hands and arms is so coarse, and the excursions are so extensive, exaggerated, and jerky, and so much increased by intended movements, that it is very similar to the tremor which is seen in multiple sclerosis. If the patient attempts to touch the tip of the nose with the index finger when the eyes are closed, he comes very wide of the mark. Tremor of this character renders the individual seriously incapacitated. The movements are so uncertain that the ordinary affairs of daily life can be performed only after deliberation and with care. There may be considerable difficulty in eating; in attempts to raise a glass full of water to the lips, the water may be spilled on the floor or on the patient. The tremor is usually most marked in the hands and arms, although sometimes there is shaking of the head as well.

Nonne has recently reported a number of cases which presented peculiar and characteristic tremor, and in which the movements did not affect single muscles or segments of limbs, but one or both extremities. It was a shaking tremor which, when sufficiently intense, agitated the whole body, so that the patient was unable to either walk or stand. The muscles of the

affected extremities had a marked tendency to contract, so that they stood out hard and tumorlike. This tremor was therefore distinguishable from that of multiple sclerosis, of exophthalmic goiter, of paralysis agitans, or from that due to hysterical, senile, toxic, or other causes. In slight cases the morbid movements ceased during rest. In severe cases they continued as long as the patient thought himself observed or was excited in any way. The gait of these patients was characteristic from their attempts to overcome the contractions of the flexor and extensor muscles of the legs while these extremities, or even the trunk, were being violently shaken by the tremor. Cases similar to those described by Nonne are frequently regarded as simulators. A man presenting these motor symptoms, who was for a time under my observation at the workhouse, was considered by the guards to be malingering in order to escape being made to work. In all of Nonne's cases the symptoms remained unchanged for several years, and the question of simulation, or even exaggeration, could be excluded. In one the patient committed suicide after the symptoms had existed for six years:

Male, fifty-five years of age. Ancestral and personal history negative. A previously healthy man. After a fall on the back and head, by which two ribs were broken, the patient found himself unable to walk. There was no trouble with the sphincters. In six weeks after the accident he could walk a little, but never was able to return to work.

The patient was examined one year after the accident. He then walked with legs apart and knees slightly flexed. There was considerable dorsal flexion of foot in walking, and he pushed himself forward from the hips at the same time, thereby showing vacillation and coarse, shaking tremor in the trunk and all extremities. On standing, a slight tremor of the trunk persisted. No Romberg symptom. On walking, many muscles became the seat of involuntary contractions, which the patient could not voluntarily overcome. There was slight bilateral foot clonus. All the reflexes were active. The strength was diminished in the upper extremities, and still more so

in the lower extremities. There was some diminution of sensibility in the legs. There was slight lateral nystagmus and a moderate limitation of the visual fields. Speech was not interfered with. The intelligence was good, and there was no mental depression. Sleep and appetite normal. The clinical picture, with the exception of some variations in cutaneous sensibility, remained unchanged for six years. However, since at the end of this period the patient took his own life, it is probable that depression had displaced the previous normal mental condition.

Although the gait in the unclassified forms does not present the stiff, dragging, spastic character of the gait of multiple sclerosis, it is rarely the gait of a normal person. The patients walk slowly and hesitatingly, and may drag their feet a little, but the gait has no evidences of true paralysis or of contracture. There may be considerable swaying of the body on standing with closed eyes. In some cases there are fibrillary twitchings in the muscles, a symptom which is especially frequent in the muscles of the shoulders or of the face. The tongue is tremulous as a rule, and disturbances of speech are common. The voice is thick and the enunciation indistinct. The speech defects differ from those of simple traumatic neurasthenia in that they are more or less beyond the patient's control. With the best endeavors and closest attention he may be unable to articulate clearly. The voice also often has a jerky character, another symptom common in multiple sclerosis.

Paralysis cannot be recognized as a symptom of these transitional forms. When there occurs a localized paralysis, it must be explained as a hysterical symptom or as an unequivocal indication of organic disease.

Indeed, the characteristic which renders the classification of these cases so difficult is, that they present no evidences which permit of the diagnosis of a focal lesion such as may be made in cases of organic injury to the nervous system,

and that the symptoms differ in character and in association from those of the functional disorders. Some of the clinical manifestations of hysteria, such as anæsthesia and contraction of the visual field, may be found, but paralysis is absent. Paralysis may occur in certain forms of hysteria which have other severe symptoms, but in such cases the whole symptom-complex would be usually explained by the assumption of hysteria or hysteroneurasthenia.

Although no muscle or muscle groups are paralyzed in the unclassified forms, there is a weakness in all the muscles. The patients are loath to make voluntary movements, they become tired quickly, and muscular exercise and fatigue cause an increase in the other symptoms.

As there is no paralysis, so there is an absence of muscular atrophy. The whole muscular system may share in the general malnutrition, but there is no selective wasting of muscles. The electrical reactions usually remain normal, although diminished excitability of the muscles to electricity has been observed.

Deep Reflexes.—The deep reflexes, as a rule, are exaggerated. The knee jerks are usually so active that taps on or above the knee induce very quick and forcible muscular contractions. In some cases a slight foot clonus has been observed, but this symptom is rare. A few cases have been described in which the knee jerks were diminished, but in no case have the knee jerks been said to have been lost when Jendràssik's method of reënforcement was used. The superficial reflexes usually present nothing characteristic, though they sometimes give sluggish or imperfect responses to stimulation. The abnormal changes in reflex activity affect the two sides of the body equally.

Sensory symptoms may be similar to those observed in simple traumatic neurasthenia or hysteria, but they are much less prominent. If traumatic lumbago is present it presents the ordinary symptoms characteristic of that condition; apart from

lumbago, pain in these forms is not very frequently or very emphatically complained of.

Headache is usually present in slight degrees, but it ordinarily lacks the exaggerated character of neurasthenic headache. The patient feels a tightness or constriction around the head, the existence of which he admits when questioned, but of which he only rarely complains. There is usually some pain in the back, and pressure over painful areas may be poorly borne, but a characteristic of the pain in these forms is that it is passive rather than active. There are rarely heard the constant complaints (so common in neurasthenia) of terrible pain and hyperæsthesia, which are made worse by every movement and by every jar. If the patient is asked if he has pain in certain parts he may reply in the affirmative, but the complainings of constant pain and exaggerations of sensibility are usually absent.

Anæsthesia, which has frequently been observed, differs in important particulars from the anæsthesia of typical hysteria. In extent, distribution, and selection it resembles the loss of cutaneous sensibility such as is found in the depressive and demented forms of insanity, more closely than the typical loss of sensibility commonly occurring in hysteria. The sensory abnormities are in accord with the mental state of inattention, indifference, or depression, and most frequently exist as a general blunting of cutaneous sensibility. Slight touches on any part of the cutaneous surface are imperfectly perceived, and pricks with sharp instruments cause only slight pain.

Less frequently the touch sense may be fairly well retained while there is a generalized analgesia. In some of Oppenheim's cases the analgesia was limited to the hairy scalp.

While it is more common for the impairment of sensibility to be general than for it to be limited to circumscribed areas, such limitations are occasionally observed. They are most frequently found on the back and legs, but they are never sharply

defined like the local anæsthetic areas of hysteria. Even in the localized areas of anæsthesia occurring in these rare forms, the sensibility to any of the various forms of stimuli is rarely completely abolished. It is diminished rather than lost, and the areas of diminution merge insensibly into the regions where sensations are better perceived. It is consequently very difficult to make diagrams of anæsthesia in these cases. The patients give unsatisfactory and contradictory answers, and it is oftentimes impossible to determine just where the boundaries of the anæsthetic areas are.

Special Senses.—Symptoms referable to the special senses are common, of which the most prominent are disturbances of vision. Nystagmus, an important symptom in multiple sclerosis, is not infrequently encountered. It is usually limited to lateral movements and is rarely pronounced. There may be weakness or paralysis of one or more of the extrinsic ocular muscles, thus causing double vision. Inequality of the pupils, as well as the failure of one or both pupils to contract under the stimulation of light, have also been recorded. The condition of the pupils is essentially different from that found in simple traumatic neurasthenia, in which the dilatation and the quick response to light, and the alternating contraction and dilatation, are frequent. There is, in many cases of the unclassified forms, some slight limitation of the visual fields. Optic atrophy has been rarely observed. Case X, of Oppenheim's, apparently developed atrophy of the optic nerve as a result of a railway accident. The history of the case, briefly told, is as follows:

A man, forty-eight years of age, received in a railway accident severe injuries of the back of the head and (probably) of the back. He was rendered temporarily unconscious, but soon came to himself and helped in clearing away the wreck. From that time onward he complained of pain in the back of the head, a tired feeling in the

back, and a constantly increasing numbness and weakness of the whole body. A year after the accident the sight in the right eye became impaired. The patient was first examined by Oppenheim two years after the accident. The symptoms at that time consisted of depression, anxiety, diminution of intellectual power, dizziness, and attacks of unconsciousness. The visual field for the left eye was normal, but the right visual field was considerably contracted. The right optic nerve, seen with the ophthalmoscope, showed a pallor of the whole papilla and a narrowing of the retinal vessels.

He was examined again after two years. None of the symptoms had improved, and some had grown worse. The patient was very depressed and wished to be alone. There were occasional temporary losses of consciousness, the exact nature of which is not evident from the history. The visual impairment was more marked than at the previous examination, and the optic-nerve atrophy had become pronounced, especially on the temporal side. There was a tremor which had many characteristics in common with the tremor of multiple sclerosis. The speech was thick. There were various disturbances of sensory function.

Next to vision, hearing is the most frequently affected of any of the special senses in the unclassified forms. There may be dizziness, subjective auditory disturbances, such as buzzing and ringing in the ears, and other annoyances similar to those observed in traumatic neurasthenia. Diminution of hearing is more frequently observed than the hypersensitiveness of the auditory apparatus which is common in neurasthenia. It may affect one ear more than the other, or there may be a comparative deafness on both sides.

Taste and smell are rarely abolished. If these senses are affected at all, there is usually nothing more than a slight diminution of their functions.

General Symptoms. — The general symptoms resemble those of traumatic neurasthenia. The sleep is interrupted and disturbed by bad dreams. The patients often have the grayish, lifeless color of the skin commonly seen in chronic nervous disease. The appetite is poor, and there may be a variety of

gastric disturbances. The bowels are constipated. Nutritional disturbances are often well marked. The patients lose in weight and sometimes become considerably emaciated. The vascular symptoms are not constant. The heart may remain apparently normal in structure and function; in some cases there is a persistent tachycardia, and there may eventually appear evidences of hypertrophy and dilatation of the heart. The urine shows no characteristic changes. If there is a marked degree of arteriosclerosis the urine may be pale and of lowered specific gravity. Sexual desire and power are very constantly diminished and may be entirely lost.

Pathology

From the complexity and peculiar character of their symptoms, and from the absence of any definite knowledge regarding their pathology, it seems to me more advisable to simply record the occurrence of such cases, without, for the present at least, claiming that they all have a common pathology, or without insisting too strongly upon what the most probable nature of their pathology is. Some of the cases may owe their symptoms to scattered foci of morbid tissue change in the brain, and perhaps the spinal cord, such as occur in the brain in cerebral endarteritis, or in the whole cerebro-spinal axis in multiple sclerosis. Most of the symptoms have occurred in men who are in the middle or later decades of life, a time when degeneration of the vascular system is encountered with particular frequency. Two autopsies (Sperling and Kronthal, Bernhardt and Kronthal), made upon patients who died during the period when these grave symptoms were marked, have disclosed thickened vessels, with areas of degeneration in the cerebro-spinal axis. It seems more reasonable to suppose, however, that in these cases the injury added activity to a process already

existent, rather than that it caused a sclerosis of vessels which were previously healthy.

The probability and possibility of the occurrence of intracranial or intraspinal injury, without there being any reason to suspect gross contusions, lacerations, or hemorrhage, has already been discussed in preceding pages.

There can be no doubt but that minute and multiple lesions may acutely occur in the brain as a result of injury, and that they may furnish the general evidences of cerebral commotion, without indicating which portions of the brain are chiefly affected. Of the nature of these lesions we are ignorant, though it is probable that the larger number of them are hemorrhagic. That such disseminated lesions can occur in the spinal cord without causing the death of the patient still remains to be proved, although there is reason to suppose that they can.

The existence of disseminated areas of hemorrhage seems the most plausible pathological explanation for the types which present the exaggerated forms of tremor and marked increase of reflex activity, with notable inhibition of psychical function.

In other instances it seems as though hysterical and organic symptoms occurred together. Also many of the cases of Oppenheim, in which the symptoms both of neurasthenia and hysteria occur together in severe degrees, may be accounted for by the simultaneous existence of those two disorders.

It is probable that it will some day be possible to make the group of unclassified forms more exclusive. Some of the cases may, when our clinical and pathological knowledge is more advanced, be enrolled as variations of some organic disorders which to-day are but imperfectly understood, others may be shown to be the results of too long exhaustion of nerve cells,

and still others may be found to be consistent with the clinical course of the multiform psychosis hysteria.

From the observation of the profound nervous disturbances which result after physical injury in heavy drinkers, and in persons whose vascular systems are in advanced stages of degeneration, I have become convinced of the force of Saenger's suggestion that some of these forms may be properly regarded as owing their existence to organically degenerated nervous systems quite as much as to the accidents which are supposed to have caused them.

Diagnosis

The diagnosis of the unclassified forms is a diagnosis by exclusion. It can only be made when the symptoms are of a character to prevent them from being regarded as surely indicative of organic disease, or when they present distinct variations from the more common types of the functional nervous disorders which follow accidents.

As has been said, it seems highly improbable that all of the cases which must for the present remain unclassified have a common pathology. The symptoms present too wide a variation to make such a supposition tenable. The mental state of these cases may be nearly normal, or it may be that of depression, or intellectual power may be so much diminished as to give a picture similar to that of senile dementia. The motor disturbances also vary greatly. In the cases which most closely resemble multiple sclerosis the symptoms are rarely identical with those commonly observed in multiple sclerosis. In the unclassified forms also diplopia is uncommon, nystagmus is only slight, and the gait has not the marked spastic character of multiple sclerosis. In the group of cases described by Nonne, the motor symptoms differ essentially from the motor symptoms of any disease hitherto described. The sensory disturb-

ances of the unclassified forms resemble those of hysteria, but they also differ in essential particulars from the hysterical anæsthesias which are commonly met with. Consequently, when we assign any given case of nervous disease following injury to the category of unclassified forms, we can go no farther than to say that the case is one of severe nervous disorder. We cannot definitely specify upon what pathological basis it rests, or by what theory its symptoms may best be explained. Nevertheless, although the diagnosis cannot for the present be precise, it is usually easy to see that the patient is suffering from an affection more serious than neurasthenia or hysteria are generally supposed to be. The general appearance and manner, and the exaggerated character of many of the symptoms, indicate that there exists pronounced disturbance of nervous function. Until the existence of these forms becomes more familiar to physicians, many of these patients will continue to be regarded as simulators. Such an opinion is manifestly unjust, as has been amply shown by the long periods of time during which the symptoms have frequently remained practically unchanged, and in many cases the entire absence of any motive for simulation. For certainty of diagnosis it is usually necessary for the injured person to be constantly under observation for a considerable length of time. It is only by such means that error can be avoided in the diagnosis of disorders whose pathology and symptomatology are so uncertain and so variable.

Prognosis

Of all the factors which are concerned in a consideration of these unclassified forms, the most secure place is held by prognosis. It is a fairly well-established fact that few of these cases recover completely. In traumatic neurasthenia most of the patients eventually return to work, and in traumatic hys-

teria the individual manifestations vanish, eventually leaving the patient apparently well, freed from disabling stigmata and able to do his work. But it is a fact common to all observers that the patients who fall within the group of unclassified forms are only occasionally able to resume all the duties which the disorder obliged them to lay aside. Most commonly the symptoms attain a certain degree of development and then remain stationary. The patient gets neither permanently better nor rapidly worse. By rest and quiet he may seem to improve, but any injurious influence, such as excitement or fatigue, or attempts to work, cause the disturbances to return with their former or with an increased intensity. Some of the patients get progressively worse and die, without any cause other than the effect of the accident becoming apparent. In several, suicide has been reported as the cause of death. A good many patients become eventually demented, or show evidence of other forms of mental disease. Cases have been reported as chronic lunatics which had previously been regarded as simulators.

The prognosis as to recovery is consequently very bad. The prognosis as to duration or as to life must necessarily vary with the individual case.

CHAPTER VI

TREATMENT OF THE TRAUMATIC NEUROSES

The Rest Cure—Other Forms of Rest—Suggestion—Hydriatics—Occupation—Diet—Drugs—Symptomatic Treatment

It has been frequently asserted in the preceding pages that the prognosis of the traumatic neuroses depends in large part upon the ability and willingness of the patient to subject himself to proper treatment. As we have seen, the traumatic neuroses, with the exception of insanity caused by nervous shock, for which the treatment is that for delirium, comprise traumatic neurasthenia, traumatic hysteria, and mixtures of the two. The treatment for all is essentially the same. It consists, in addition to attention to individual symptoms, in the regulation of the mode of life in such a way that the patient is guarded against the social and mental influences which, in his particular state, work him injury, and in supplying such essentials of normal living which, by reason of fatigue or perverted ideas, he can no longer gain for himself. It is the natural method, rather than a treatment by drugs. Nowhere so forcibly as in the functional nervous diseases is the fact brought out that treatment should be directed toward the individual rather than toward the disease. So variable in their effects are the disorders which primarily affect personality that it will never be possible to write how they should be managed in their details. It is the physician's duty to ascertain, as nearly as he can, the kind of individual he has to deal with, and to suit, as wisely as he can, the various remedial measures to particular needs. He must assume the psycholog-

ical control of his patient. He must, by encouraging suggestions, or by devising mental and physical occupation, divert the thoughts of the patient from morbid channels to healthy ones. He must, at the same time, see to it that the patient does not lean upon him too much. Frequent visits and mental and moral support are imperative at first: but as the treatment proceeds the visits should become more separated, and the patient made to rely more and more on himself. If this is not done, the sick man, while he may regain his physical health, becomes one of those unfortunate fetich worshipers who can never free themselves from the thraldom of medical control.

Regularity of performance by the patient in all the physician prescribes is absolutely essential. Without being assured of this the physician is seriously handicapped, and if he cannot obtain it, he would far better give up the case. Regularity is best obtained by giving the patient a written schedule, with a definite something, be it rest, or exercise, or baths, or amusements, or work, for each hour of the day. The nurse, if one is necessary, reports how the duties are gone through with. When a nurse is not necessary, the patient can keep a written journal of how his days pass.

The question of returning to work is of great importance. As a general rule, the sooner work can be resumed the better. After severe accidents the patient is best in bed for a few days. If at the end of that time he is still incapable of work, as shown by great nervousness, tremor, insomnia, etc., a change of scene is advisable, work to be resumed, partially at least, on the return. In severe cases, with great shock and profound mental depression, such a course is not possible, and the rest cure, more or less stringent, is necessary. But even in the rest cure the physician should be constantly on the alert for signs of growing strength, and should advocate a resumption of regu-

lar occupation as soon as possible. In cases which have resisted the ordinary methods of treatment, change of scene and climate, with the throwing off of all medical care and direction, may finally prove efficacious.

The physician is sure to be more or less hampered by the question of damages. He knows that the patient's best interests demand that this question be ruled out altogether, or be kept very much in the background. At the same time it is more or less beyond his control, and in many cases the social conditions demand that the injured person be compensated for loss of time or for curtailment of wage-earning capacity. The most the physician can do is to keep the question of damages in the background as much as possible, and to do his best to effect an early settlement.

The most important aids in the treatment of functional nervous diseases are rest—given as the rest cure and in other ways—suggestion, hydriatics, occupation, dietary restrictions, and drugs.

The Rest Cure

Fatigue being a salient feature in all functional nervous diseases, and the chief one in some, rest stands high among remedial measures. This fact was best appreciated by Weir Mitchell when he first devised his now celebrated rest cure. The rest cure has many disadvantages, and is often employed when it is not needed. At the same time it stands accepted as the most perfect method of giving complete rest. It gives more than rest: it gives the patient into the absolute control of the physician. As such it will always live. The physician, if he be wise and judicious, can better direct the mental operations of the patient while administering the rest cure than in any other way. Carried out in most perfected form it is an expensive form of treatment, as it implies that the patient be

away from home for weeks or months, that he be constantly under the supervision of a nurse, and frequently visited by a physician. It is always also a difficult method of treatment to carry out. It is not always easy to convince either the patient or his friends of its necessity and reasonableness. The friends sometimes regard the patient as more frightened than hurt; or, as more frequently happens, the friends are too solicitous for his welfare. They are unwilling to give him over entirely to the charge of strangers, where he is no longer amenable to their wishes or the recipient of their sympathy. The patient himself often resents being deprived of his liberty; or, acceding for a time, becomes impatient of the forced seclusion, insists upon returning home, or of taking up some less exacting form of treatment. Before beginning the rest cure, therefore, it is advisable to have a full understanding on all sides; for when it is interrupted or abruptly terminated the physician loses greatly his control, and the patient becomes more than ever the prey of uncertainty and indecision.

The class of cases for which the rest cure is most indicated are cases of traumatic neurasthenia which are either serious from the first, with loss of weight, excitability, and insomnia as prominent symptoms, or cases which have existed for some time and have failed to improve. It is indicated in traumatic hysteria, chiefly by reason of the opportunity it gives the physician to exercise psychological control of the patient; for the cures of hysteria are psychological. The fundamental principles of the rest cure depend upon isolation and rest in bed. By means of the first it is intended to permit a restoration of normal vitality to nerve cells whose functions have become disordered, and by the second to supply to the whole body the rest it needs, but which, without artificial means, it does not obtain. These two means operate conjointly, the action of one enhancing and supplementing the effect of the other. Com-

plete isolation implies a banishment of family and friends, the giving up of all business questions, and of the writing and receiving of letters. In other words, the patient is completely cut off from the outside world. In severe cases it is wisest, at first at least, to have the isolation complete, so that physician, nurse, and masseur (if the latter is necessary) are the only persons the sick man sees. In many cases the complete isolation is never necessary; and in most, after one or two weeks, it is safe to have the patient receive short visits from the more discreet members of his family, or to take up some outside interests. In serious cases of hysteria, however, the complete isolation should be continued until the physical symptoms disappear or the method of treatment is abandoned. The only really effective method of isolation is away from home. For this purpose quiet hotels or boarding houses or private hospitals are generally utilized. Large general hospitals, even when a private room is obtainable, are much less suitable. The setting aside of a room in the patient's own house, to which none but the physician and his assistant are admitted, may be tried when complete seclusion is not necessary. Under the latter circumstances it is never completely successful, and under all circumstances is less desirable than for the patient to be away from familiar sounds and scenes. The one constant companion of the patient during the time that the isolation is more or less complete is the nurse. Male nurses are not fitted for this kind of work, and the female nurse must have some special qualifications, and, if possible, some experience in such cases. It is more important, however, for the nurse to realize that the neurasthenic patient is really ill than it is for her to be intimately familiar with every detail of the treatment, for the way that the rest cure is to be carried out in an individual case will be described to her by the physician when the treatment begins; but unless she realizes that the un-

reasonableness, impatience, and fretfulness of the patient are the voicings of disease, she will be unable to maintain the gentleness, firmness, and patience upon which qualities of the nurse the success of the rest cure depends. No task is more trying than the care of such patients. It requires good health, an even temper, and a cheerful disposition. Many nurses are distinctly unqualified, and should never undertake it. It is easiest for those who by their cheerful manners and ability to read and talk well, or by similar accomplishments, can make the time pass most pleasantly and quickly for their bedridden companions.

Next to isolation, the most important feature of the rest cure is rest in bed, the completeness of which varies with the severity of the case. In grave cases of neurasthenia the patient is kept as nearly motionless as possible. He does not feed himself. When the bed is made he lies on a sofa, and uses portable urinal and the bedpan unless a toilet directly adjoins his room. Like isolation, the enforced rest may be irksome for the first few days, but the patient soon becomes used to it and ceases to complain, or even comes to like it. When the time comes for complete inactivity to be abandoned, the change is to be effected gradually. At first the patient may sit up in bed for a few minutes at a time; then sit with the feet out of the bed resting on a pillow; then sit in a chair, and gradually begin to walk, doing a little more each day than he did the day preceding.

During the rest cure something must be done in the way of exercises. For this, massage and electricity are the chief reliance. Massage may be given by the nurse, if she is a skilled operator. To be able to give massage well, however, is an art in itself, of which only a few of the graduates of our training schools are mistresses. If the passive movements are to be given by some one other than the nurse, it adds to the expense of treatment; but massage to be beneficial must be systemati-

cally and skillfully applied. It should be begun gradually, and it is generally wise to delay it until the patient has been in bed several days. One part or one extremity only should be treated at first in severe cases, as the early applications are often extremely fatiguing. The rubbing can then be increased in point of time, so that at the end of a week or so the whole body surface, except the head and neck, are being exercised for forty minutes or an hour each day.

Electricity is the other means by which the patient obtains exercise while in bed. Its effects, though less marked, are similar to those of massage. If the massage is successful, electricity is not absolutely essential; but without massage, electricity forms the best substitute. It has this advantage over massage, that any intelligent nurse can be readily taught to use it gently and effectively, and that "electrical treatment" generally appeals to the mind of the patient. Of the two currents, the faradic is easier of application and produces good muscular contractions, and is generally preferable, though the galvanic current may be used. The electrodes should be wet with warm salt water, and the sponges be frequently changed, or else covered by pieces of white gauze at each application. At first the current is applied in very weak strength and to small areas of the body; as the patient becomes accustomed to it the current strength may be increased until the muscles react promptly, and all of them are in turn stimulated. The *séance* should last about half an hour.

The diet during the rest cure is controlled by two conditions, viz., that it should be so arranged that the patient may receive plenty of nourishment in spite of any gastric irritability he may have; and, secondly, in cases in which there is any failure in nutrition, that the amount of food taken be excessive, and out of proportion to the work done (forced feeding). If, at the beginning of treatment, dyspeptic symptoms, such as

anorexia, nausea, vomiting, and eructations, are present, the alimentation should consist exclusively of milk, in order that as soon as possible the stomach may be brought to a condition in which it can retain large quantities of more substantial food. To this end the milk is given in small quantities, repeated at short intervals during waking hours. For the first few days one or two ounces of milk given every hour, or slightly larger quantities given at longer intervals, are sufficient. Administered in this way milk can be borne by almost anyone, although he may think it does not agree with him, and although, if given in larger quantities, it might induce vomiting. If the gastric irritability is extreme, the milk may be peptonized. Koumiss, on account of the alcohol it contains, is not recommended as an exclusive diet.

This method of feeding, together with the rest in bed, is usually sufficient to quiet the more prominent gastric symptoms, so that in a short time the quantity of milk may be increased, or, better, it may be alternated with more palatable articles of food. At the end of a week it is usually possible for the dietary to be amplified, so that while the patient continues to be fed every two hours, he receives substitutes for the milk several times a day. These substitutes should at first be limited to fluids such as soups or broths, or some of the prepared foods; but they may soon be made to include eggs, custards, and jellies, until finally, by the addition of meat juice, chops, chicken, etc., with the simpler vegetables, the patient is receiving a regular diet.

As long as he remains in bed the plan of some nourishment every two hours should be adhered to, although no large quantity is taken at any one time. Even after he gets up and begins to walk around, and is taking three fairly full meals a day, he should have some nourishment between meals, in order that the amount of food taken remains excessive.

Stimulants are to be avoided. Tea and coffee can generally be excluded without difficulty. To relieve the tedium of the dietary, a cup of cocoa or chocolate may be occasionally permitted. If the patient is markedly alcoholic, with symptoms of cardiac weakness, it may be necessary to provide some means for the improvement of the heart's action. This may be done by prescribing small quantities of alcohol, or, better, some of the heart tonics, such as strychnine, strophanthus, or digitalis. Tobacco, while the patient is in bed, is to be forbidden. Later, the advisability of its use depends upon the effect it has upon the individual, his dependence upon it, and the length of time he has been habituated to its use. If it renders him more nervous, it should be given up. Often, however, it calms rather than excites; then one or two cigars a day can be allowed with safety.

The general management of a neurasthenic man or woman during the one or two months that the only persons he or she sees are comparative strangers is a matter of considerable delicacy. It is the physician's duty to make the period of confinement as little irksome as is consistent with the ultimate object in view. Every latitude and privilege consistent with the sick man's welfare should be allowed. When, however, it has once been decided what the patient may and may not do, the plan of treatment should remain unaffected by unreasonable protestations and complaints. By acceding to demands which are the results of fretfulness and impatience the physician or nurse loses in personal influence, and postpones the time when such nervous manifestations are to permanently disappear. Gentle firmness on the part of both physician and nurse is absolutely imperative to the success of this treatment. Objections and complaints are best forestalled and obviated by the immediate institution of the daily *régime*, to which the patient is to conform as regularly as though he were

keeping business appointments. By this means the patient comes to look forward to the hours given up to special things with expectancy and often with pleasure, and the time passes. When the daily or rather hourly schedule is once determined upon, it should only be changed for purposes of enlargement. Its special character varies with individuals, but its principle remains essentially the same. Thus, if the patient does not leave the bed at all, he has milk at seven; at eight he is assisted at his toilet by the nurse, and receives a sponge bath; at nine more milk, and is read to for a short time; at ten the physician calls; at eleven more nourishment, followed by rest and quiet, and so on throughout the day, there being definitely determined hours for the physician's visit and that of the masseur, for reading, talking, resting, eating, etc. As the patient begins to improve, the hours are changed to fit different requirements, but the rule of regularity remains undisturbed. The length of time necessary for the observance of the more stringent requirements of the rest cure is in large part determined by the rapidity with which improvement occurs. The more prominent symptoms should begin to yield in a few days or a few weeks. The dyspeptic symptoms disappear and appetite improves; the sleep improves in quality and in duration; the pains are less frequently complained of; the tremor and excitability are less evident; the nutrition is raised, as is shown by an improvement in the color of the skin and by a gain in weight. There is no absolute rule as to when the patient may get up and see his friends again; this must be determined by the physician, who has learned the capabilities of the person under his care, and who, by permitting him to gradually do more and more, can readily ascertain how rapid the return to normal living should be.

Valuable as is the rest cure, it counts its failures, and the question arises, if the patient improves but little or at all, How long shall the treatment continue? In general it may be said

that if at the end of two months the benefit is not plainly perceptible, the method of treatment should be much modified or changed altogether. The prognosis in such cases is not good; recovery, if it occurs, being certain to be tedious. Exception to the above must be made for obstinate cases of traumatic hysteria. In them, two months' isolation may be too short to overcome the fixed ideas which cause the physical symptoms. In such cases six months, or even longer, may be necessary to cause the symptoms to yield.

When the rest cure is reasonably successful, at the end of from one to three months the patient is comparatively free from the distressing symptoms from which he suffered at the beginning, and can endure a moderate amount of physical and mental exercise fairly well. He is still somewhat nervous and excitable, however, and in much the same condition as many neurasthenics who are hardly ill enough to need the rest cure, but who require some especial form of treatment for the reëstablishment of health.

Various Forms of Rest

In persons who no longer require the rest cure, and those who were never ill enough to make such a procedure necessary, rest, more than is necessary for healthy individuals, is still a primary requisite. This is secured in persons without employment by having the breakfast served in bed, and by lying down for thirty minutes or an hour two or three times during the day. If the patient has employment, it should be taken, when possible, from working hours; or, if that is not feasible, he should retire earlier than usual, so that he passes at least ten hours in bed. Rest in the daytime should be regarded as a serious prescription. The room should be darkened, free from noise or confusion, and the patient lie absolutely still, and do

nothing. The clothes should be loosened, and everything arranged to secure the most perfect relaxation and repose. Many neurasthenics suffer from what they call tension. Their muscles seem constantly contracted; they find it difficult to relax. They should be shown the positions in which the trunk and limb muscles have the greatest support when lying down, and be told to assume these positions when taking their rest. The effect of complete relaxation of the muscles as a relief to mental fatigue is quickly apparent. No less important than physical rest is the interruption and variation of mental activity. The rest cure tends to bring mental activity to its lowest ebb. This is by no means always necessary; but the physician should see to it that the neurasthenic does not keep his mind too long on any one subject, whether it be his own troubles or his business affairs. The life of the neurasthenic, therefore, while it should be equable, with avoidance of all that tends to fatigue or excite, should be varied. He should have a certain amount of work, a certain amount of rest, a certain amount of diversion. Diversity in all is rest.

Suggestion

By the term hypnosis is usually understood the peculiar psychical condition brought about by various means, during which the patient's higher consciousness is in abeyance. In the hypnotic state the subject may be made to do things of which on awakening he has no recollection; also, suggestions made during this state may be subsequently acted upon independently of his volition. It is largely by means of hypnosis that the falsity of hysterical symptoms has been shown. Contrary to popular belief, the induction of hypnosis implies no particular gift on the part of the operator. Almost anyone can be taught how to induce it. Whether a person can or cannot be hyp-

notized depends upon himself rather than upon accessory circumstances. Certain types are readily hypnotizable, others with great difficulty only, and some few are apparently unhypnotizable. As a therapeutic measure it has never obtained popularity in the medical profession. It has never been freed from the penumbra of the black art and witchery; and once its use meets popular sanction the doors are thrown open to all kinds of charlatanism and imposture. In formal hypnosis suggestions are given the patient in the hypnotic state, the effects of which are intended to make themselves felt in the waking state. It has no part in the treatment of neurasthenia, and in hysteria it can only cause a disappearance of the individual symptoms without materially benefiting the fundamental mental disorder. When the physician succeeds by means of hypnosis in bringing about a disappearance of any of the physical manifestations of hysteria, he does so by bringing about an alteration of personality during which the subconscious self of the patient receives and reacts to impressions which would not affect the conscious self. But since the nature of hysteria can best be explained by assuming that the physical manifestations are dependent upon alterations of personality, it seems an unavoidable conclusion that by contributing additional phases to the underlying morbid psychical conditions, the whole disorder would be rendered more fixed even though the temporary physical manifestations are made to disappear. Consequently hypnotism in hysteria is a last resort. When the patients have failed to benefit from the rest cure, from hydrotherapy, or from other means, hypnotic suggestion is not only permissible, but remains as the one therapeutic measure from which benefit may be hoped. Tried under these conditions, it has much greater chance of success if the patient is isolated. The *séances* may be given daily. Profound sleep is neither necessary nor desirable, it having been found that suggestions

are equally effective if given in the half waking or hypnoid condition. The suggestions should be given both toward the general mental state and toward the individual symptoms. For further details as to technics the reader is referred to the works of Bernheim, Forel, Wetterstrand, and Binswanger. While formal hypnosis is only occasionally advisable in the management of the traumatic neuroses, these disorders may be greatly influenced by suggestions given in other ways. In neurasthenia these suggestions take the form of encouragement and reassurance, and of argument; in hysteria they need to be more subtle, and directed in a way that the patient is constantly forced to see that many of his ills are imaginary. To directly deny to the patient the reality of the symptoms is worse than useless. But the physician may dismiss many symptoms as unimportant, and say that others, while annoying, are certainly transitory. He may pass without remark certain demonstrations, leading the patient to believe that they are unimportant. By various devices he can demonstrate that the patient is capable of many things of which he says he is incapable. Suggestions of this kind, repeated day after day, cannot fail in their effect. The patient more and more comes to see that the medical man is right. But no less important than that the physician should make good suggestions is that he should prevent the patient being controlled by suggestions which tend to keep up the symptoms. Hence isolation, and hence the various devices of occupation, etc., to interrupt the patient's own current of thought.

Hydriatrics

Water, taken internally, causes an increased activity in the kidneys and skin, and by diluting the toxic metabolic products which are circulating in the blood it exerts a beneficent influence upon nutrition. It may be administered with advantage

in large quantities during the whole course of neurasthenia. The patient is directed not to drink fluids with his meals, but to take two or three glasses between each meal. It is the external application of water, however, which is attended with the most brilliant therapeutic results in functional nervous diseases. Success depends on the proper choice of the individual procedure, and upon whether it is properly carried out. The sponge bath, given daily during the rest cure, is chiefly for purposes of cleanliness. Different portions of the body are sponged off with water at 70° F. Each segment is dried and wrapped up before another is bathed.

The drip sheet is chiefly used for patients who are up and about. The best time for its application is upon rising in the morning. It has this advantage that it may be given at home, and is less alarming to the patient than some of the more elaborate hydriatric measures. The sheet, after having been partially wrung out in water at 70° F., is applied as follows: The nurse, standing behind the patient, whose head has been wet with cold water, puts one corner of the sheet under the patient's right arm and carries it across the chest; the rest of the sheet is then carried across the back, over the left shoulder, and continued over the front of the right shoulder until the end can be tucked in at the back, so that the body is entirely covered. By slapping, and by light and rapid rubbing over the sheet from periphery toward the center, the patient soon becomes warm. When the reaction is fully established, which occurs in one or two minutes, the patient is dried quickly and wrapped in blankets. In succeeding applications the temperature of the water may be reduced a few degrees at a time until it finally reaches 50° F.

The wet pack is only occasionally necessary in the functional nervous disorders. When necessary, the patient is wrapped in a sheet wrung out in water at 70° F., which may

be gradually reduced to 60° F., and then rolled in a blanket, in which he is left for an hour or more. This procedure is of special service when insomnia is pronounced, when alcohol is present, and in states of excitement generally. The desired results may also be obtained by the local wet pack, which is one of the most efficient means of allaying mental excitement and inducing sleep. It is also useful in the treatment of constipation. It is applied by placing a piece of sheet folded several times, which has been wrung out in water at 60° to 70° F., and then folded in the form of a compress across the patient's abdomen. A piece of flannel bandage of the same length as the compress, but two inches wider, is then put around the body to hold the compress in position. The whole is applied just before going to bed. It may be renewed in half an hour, or, if the patient falls asleep, may be left on.

The footbath may be hot or cold, the effects being similar. In the cold footbath the water is at a temperature of 40°, and the patient rubs the feet energetically one against the other for five minutes; the friction keeps the feet comfortably warm. The hot footbath at 110° F. is perhaps somewhat less efficacious. Both hot and cold footbaths are efficient sleep-inducing agents, and are valuable aids for the relief of mental irritation and excitement, and of the disagreeable subjective cerebral sensations.

The douche excels all other hydriatic measures in the constitutional treatment of neurasthenia and hysteria. For it to be successful two conditions must be fulfilled. These relate to temperature and pressure. The temperature of the water must be accurately regulated by the thermometer, so that the patient does not receive a shock from too cold water, or that he may not fail to react promptly, as often occurs when the water is too warm. The force with which the douche is delivered determines the degree of mechanical irritation, which is nearly as

important a factor as temperature. A douche, even when of the proper temperature, which comes to the patient with only a few pounds' pressure, is followed by a prompt reaction in robust persons only; in anæmic and exhausted individuals it is a long time after the contraction caused by the cold before the capillaries dilate again; the desired result of a quick alternation of vascular tone is therefore not obtained. Proper facilities for giving douches are rarely found in private houses, or even in general public baths. They should be sought for in properly equipped hydriatric institutes where the requirements as to pressure and temperature can be obtained, and where the physician's directions can be carried out by skilled attendants. The treatment should be taken every day, or every other day. The patient is placed for two or three minutes in a chamber containing hot air (130° to 150°). On emerging, he immediately gets under a rain bath of from 60° to 70°, and of from 15 to 20 pounds' pressure. The degree of temperature and the amount of pressure vary according to the stimulation desired for the individual case. The colder the water and the greater the force the more pronounced the effect. Cold and pressure are both gradually increased in succeeding treatments. At the end of one or two minutes the water is turned off and the patient briskly rubbed. He may then lie down, or, better, dress, and at once go out in the open air, in order to take advantage of the deepened respirations which follow the douche. For the rain or Charcot douche may be substituted the fan douche, or the spout, delivered on the back only. The fan douche or spout is often serviceable in the treatment of the manifestations of hysteria. Under the local cold douche of from 20 to 35 pounds' pressure, anæsthesia or contracture often disappears. Local and general douches may be alternated. It is frequently impossible for patients needing water treatment to have access to properly equipped institutes. The cold sheet,

the footbath, and the packs can be given perfectly well at home. In robust patients the cold shower bath and cold footbath and cold spinal spraying may be tried. They should not be persisted in unless the reaction is prompt.

Occupation

To the patient who is able to return to work it is generally unnecessary to prescribe any additional occupation. But it is imperative that something be given to do for those who are unable to work, and for those who have no occupation. The occupation should be preferably out of doors. In the line of recreation are golf-playing, walking, riding, etc.; more in the line of tasks are gardening, botanizing, and the care of animals. It is always desirable that the patient have tasks with a definite purpose. Wood carving, modeling, carpentry, basket making, weaving, etc., are all excellent, and have the advantage that the patient may turn to them and thereby divert and calm himself during an access of nervous symptoms. Whatever tasks are determined upon should be carried through with regularity and persistence.

Diet

The question of diet is one requiring supervision both as to the quality and quantity of the food; the time of eating, rapidity, thorough mastication, etc. Alcohol and stimulants generally are to be given up. Never, except in the rest cure, is an exclusive milk diet indicated. But when the nutrition is poor, an addition to the regular diet of one or two glasses of milk taken between meals is very beneficial. In other cases a partial elimination of the starches and sugars is indicated. Some neurasthenics and hysterics show a tendency to grow fat. For them reduction of flesh is beneficial, which may be obtained by diet, and by the careful administration of the thyroid extract.

Drugs

The use of drugs in the treatment of neurasthenia is subsidiary to the application of some or all of the procedures which have been described. Neurasthenics are especially fond of dosing themselves. They are apt to think many of the advertised cures and specifics would cure them and to wish to try them. To combat this tendency, the physician should give his remedies with careful directions, and assure himself that the patient is not at the same time taking others.

There are a few drugs which render unquestionable assistance. To combat anæmia and malnutrition, general tonics such as iron, arsenic, cod-liver oil, etc., are generally useful. When gastric irritability is pronounced these may be given in the form of some of the mineral waters, notably Levico water. The most popular nerve tonic is strychnine. This may be given by mouth or hypodermatically. The advantage of the hypodermatic administration of strychnine and of the glycerophosphate of soda is that it brings the patient regularly to the physician, and thus forms a legitimate excuse for encouraging suggestions. The glycerophosphate may also be given in capsules, or simply in water. The bromides may do much harm, and their use is deprecated; but in small doses they allay nervous excitability, especially when given with ergot. They also are recommended for relief of dizziness and headache. The following has proved useful in the hands of the writer:

℞ Natrii bromid. ..... ℥vj;
Ext. ergot. ..... fl.℥j;
Aq. destillat. ..... ℥jv.
Sig.: One teaspoonful in water one-half hour after meals.

Morphine has no place in the treatment of the traumatic neuroses.

Symptomatic Treatment

The treatment of the traumatic neuroses is not a treatment by symptoms. It is the construction and carrying out of a general plan, directed toward fundamental conditions. According to it individual manifestations are overlooked as much as possible. To vigorously attack individual symptoms rarely makes them disappear; or, if they do so, they soon reappear again or are replaced by others. The sooner the patient is made to understand that many of his subjective disturbances are more or less beyond the reach of medicines, and that he can best banish them by overcoming them himself, the sooner will he find himself on the return road to health. At the same time there are some symptoms which benefit by treatment directed toward them on ordinary medical lines; and some of the local manifestations of hysteria show the effects of special effort if combined with general measures.

Insomnia can usually be helped by some of the hydrotherapeutic measures. When they fail to induce sleep, recourse may be had to small doses of bromides or some of the coal-tar products.

Digestive disturbances, and especially constipation, are to be treated along general lines.

Pain is the symptom which most calls for individual treatment. The first thing to determine (always difficult) is as to whether it is the result of actual injury, or fatigue, or toxic or similar causes. Under these conditions material treatment is necessary. The suffering from traumatic lumbago may be so intense as to require immobilization. The immobilization is rarely necessary for more than a few days. At its termination the muscles of the back should be actively massed. The cautery is also useful for the pain of lumbago. Faradism is of but little use. Headache and indefinite pains are best allayed by rest and

general measures. If true neuralgia exists, it may be relieved by hot fomentations, counterirritation, by some of the analgesics, by galvanism, or the sinusoidal current, or by vibration. If pain is entirely hysterical, no analgesic drugs should be given. A placebo may be necessary, but better in some form of local treatment, such as hot water, or faradism or counterirritation, always combined with forcible suggestion.

Some other local hysterical manifestations benefit by special treatment. In paralysis, without contracture, passive movements and faradism cause the muscles to move; also, the patient may often be enticed to make movements with the paralyzed part. In this way he may slowly come to see that the part is not paralyzed after all. When contractures are present this is more difficult. Then, if the legs are involved, by means of supports such as crutches, or movable stools, or walking machines, the patient, being left to himself for certain periods every day, is put in positions so that some use is bound to be made of the paralyzed parts.

Aphonia is often relieved by local measures, such as faradization of the larynx, the patient being told to make a sound with each artificial contraction of the laryngeal muscles. Mutism is more rebellious. Intermittent spasms of any muscles are rarely amenable to any local treatment. Anæsthesia and contraction of the visual fields require no special treatment.

PART III

MEDICO-LEGAL CONSIDERATIONS

CHAPTER I

PERSONAL-INJURY CLAIMS AND EXPERT WITNESSES

Frequency—Medical-Expert Witnesses

Frequency

At the present time it is a rule, with few exceptions, that persons who suffer real or supposed injury in railway or other accidents demand compensation from the companies or individuals responsible. Mr. Lawrence Godkin, in Hamilton's "System of Legal Medicine," states that "probably half the jury cases tried in the courts of the State of New York alone, in any one year, are actions for personal injuries resulting from alleged negligence."

Mr. E. Parmelee Prentice has carefully examined the records of the Chicago courts which have jurisdiction of these cases. The results are somewhat startling. He says: "In 1875 there were altogether about 200 personal-injury suits pending in Cook County. During the first six months of 1890 the number of these suits brought in Cook County was 346, the total damages claimed being $2,814,860. During the corresponding six months in 1896 the number of such suits brought in Cook County was 893, and the total amount of damages claimed was $13,510,000. It would be reasonable to as-

sume from these figures that there are now pending in Cook County 3,600 of these cases, and that the damages claimed are between $50,000,000 and $60,000,000." It is needless to add that this enormous increase is out of all proportion to the increase in the number of accidents.

Thus personal-injury claims constitute a very important feature of modern life. Not only transportation companies, but private individuals as well, fully expect to pay for injuries which are received through actionable negligence. Similarly, few receive injuries traceable to the negligence of others without promptly demanding compensation. In our mechanical times the frequency of accidents is enormous. Consequently, the evaluation of injuries received and the compensation to which the injured person is entitled are matters of prime importance. Greater interests are involved than in any other medico-legal question. This becomes plain as soon as we reflect upon the large sums which are annually paid out in such cases.

The general claim agent of a Western railway, whose system covers about 5,000 miles, told me that in the year 1896 the company paid $350,000 in personal-injury claims alone. This sum was entirely exclusive of the judgments in cases which were brought to trial and which were decided against the company. Another large American railway pays between $200,000 and $400,000 damages every year for personal injuries.

From the report of the Brooklyn Rapid Transit Company for the year 1901 it appears that in that year more than $1,000,000 was paid for personal injuries and expenses incident thereto. This sum represented nearly ten per cent of the gross receipts of the company for the year named. Individual verdicts are also often very high. As much as $35,000 has been paid for a personal injury, and for a death claim resulting from the tunnel accident of the New York Central and Hudson

River Railroad of February 8, 1902, a verdict of $60,000 was returned by the jury. Verdicts varying from $10,000 to $20,000 are not at all unusual, and anything under $1,000 is considered virtually a victory for the defendant. Court calendars are overcrowded with these cases, which form the bulk of jury trials to-day. But the calendar is not a complete index of the degree of activity in this branch of law, as for every case that comes to trial it is safe to estimate that ten are settled by mutual agreement out of court. If the magnitude of the interests at stake are taken into consideration, it is not surprising that trial lawyers should be on the alert, or that there should be great competition for plaintiff's cases. As a result, "runners" or "ambulance chasers," representing legal firms which specialize in accident cases, are constantly stationed about centers of traffic; they rush to the scene of accident, and make their appearance at the hospital door almost simultaneously with the injured person. Thenceforth the claim is prosecuted on the contingent-fee plan. The system has doubtless been much abused, and has been made the object of much attack and ridicule. It is made possible solely through the poverty of the plaintiff, who is generally unable himself to carry on the great expense of trial at law, and who consequently is forced to accept professional services which are to be paid for, on a percentage basis, out of the damages awarded. It has many very objectionable features. By such a system the lawyer is made more than an advocate, and the expert medical witness more than a mouthpiece of science. These sometimes so far exceed the limits of their respective callings as to become partners with the litigant.

The salient differences in the system as applied here and the system of German accident insurance (see p. 55) is that the damages under the German law are determined by a harmonious principle, applied whether the employer or employee is

negligent. In Germany every factor except the extent of the injury is fixed and constant. In the United States, nothing is fixed except the abstract principles of law as set forth in the judge's charge to the jury. An hysterical girl, without responsibilities and without the capacity for self-support, may receive a verdict for some trifling mishap much in excess of that given a workingman for injuries which disable him for life. By the German law, also, the insurance allowance is diminished or increased according as the injured person gets better or worse with time. In America, when the plaintiff gets his money, his case is judicially at an end; his disease may become worse, but he is entitled to no further indemnity, or better, without his being required to make any return of the proceeds. Yet the verdicts are notoriously capricious, often unjustly reflecting the sex and personality of the injured person.

Medical-Expert Witnesses

The medical question for the jury to decide in personal-injury suits is the extent of injury and the probabilities of its permanency. The extent of injury is a question of fact, and is usually testified to by the regular attending physician. Men especially experienced in the particular variety of injury at issue are often called in to testify as to facts, but more especially as to the probable permanency of the injury. These latter qualify as experts. The court usually accepts as an expert any physician or surgeon who is a graduate of some years' standing of a reputable medical school, and who states that he has had experience in the particular subject in hand. Their opinion, based upon the facts in evidence, is the most important part of the testimony given by experts. Being an opinion, it can only have weight when the jury is convinced that it is an honest conviction based on careful observation and experience, and

given without special bias. Probably no expert in a personal-injury suit is credited by the jury as being absolutely impartial. It would be well for experts to bear more prominently in mind that from the moment they take the stand they are assumed to be more or less prejudiced; that if by their testimony, or by their manner of giving it, they strengthen such an assumption, they injure more than they help the side for which they appear.

Medical testimony should be given in the simplest language possible. Technical terms are to be shunned, and when used, their meaning should be explained or illustrated. The witness should be chary about engaging in a contest of wits with the opposing counsel, and under no circumstances is he to lose his temper. His answers should follow the questions promptly, but he should not permit himself to be led into hurried answers in his cross-examination. Many questions are put which do not permit of a direct "Yes" or "No" answer. Under such circumstances the witness should state that he can only answer qualifiedly. He is entirely justified in maintaining this position, however insistent the cross-examiner may be on having a categorical answer.

If the expert has personally examined the plaintiff, he is expected to give the details of his examination, and then his opinion. He is permitted to testify as to statements made by the plaintiff in so far as they had reference to actual suffering or disability at the time of the examination. He is permitted to refresh his memory by notes, provided these were made at the time of or shortly after the examination. Very thorough examinations are advisable even when the condition is plain; for if the expert can be shown to have been hurried or superficial, his testimony can be greatly weakened on cross-examination. Often when the expert has examined the plaintiff, and always when he has not, his testimony is brought in relation to

the case by means of a hypothetical question. The hypothetical question is founded on the facts in evidence, which have been testified to by the various witnesses. The expert is required to accept as true the assumptions made in the question, although he is not responsible for their truth. Thus it may happen on cross-examination that he is obliged to express an opinion based on facts he believes to be untrue. This loophole, also, makes it easily possible for unscrupulous experts to give favorable testimony based on assumptions they know to be untrue.

The hypothetical question looks for answers as to whether the accident described were adequate to cause the symptoms subsequently complained of, what these symptoms indicate, and whether or not they will be permanent.

The question of causation is the most difficult, especially in the many nervous diseases whose causes are not well known. For many of them the most that can be said is that the injury was an active causal factor. The basis of an action must be some physical injury. Fright alone, unaccompanied by physical injury, is not a cause of action (Mitchell *vs.* Rochester R. R. Co., 151 N. Y., p. 107), but when associated with physical injury, fright, and other emotional elements, may contribute to the disability for which recovery at law may he had. Brush quotes the following pertinent decisions:

"'The existence of a predisposition does not excuse the defendant, as such disease might never have occurred had it not been for the injury' (Stewart *vs.* City of Ripon, 38 Wis., 584), and that 'the disease might never have developed to its present extent had it not been for the injury' (L. N. A. R. R. *vs.* Snyder, 117 Ind., 435). But if any casual or unexpected cause intervene, then the defendant is not liable (Godkin). Neither is the defendant excused from the results arising from malpractice on the part of the plaintiff's physician. It has

been held 'that a person who is injured through the act of another is required to act in good faith for his cure, but he is not responsible for any error of a physician in the treatment of the injury, nor will such error shield the party through whose fault the injury was occasioned' (Lyons *vs.* E. R. R. Co., 57 N. Y., 489; Sauter *vs.* N. Y. C. R. R. Co., 66 N. Y., 50; Hope *vs.* Troy Elec. R. R. Co., 40 Hun., 438). In testifying as to the permanency of the conditions found in the plaintiff, the law does not require the witness to state beyond a reasonable certainty, the court holding 'that medicine is far from being an exact science. At its best its diagnosis is little better than a guess, enlightened by experience. The chances of recovery in a given case are more or less affected by unknown causes and unexpected contingencies, and the wisest physician can do no more than form an opinion based upon a reasonable probability'" (Griswold *vs.* N. Y. C. R. R. Co., 115 N. Y., 61; Alberti *vs.* N. Y. C. R. R. Co., 118 N. Y., 77).

CHAPTER II

MALINGERING

Varieties—Exaggeration of Symptoms—Substitution of Origin—Simulation

Varieties

Since corporations find it cheaper to settle small claims than to contest them; since partiality of juries toward the plaintiff, though much less pronounced than formerly, still exists; and since both legal and medical services are obtainable on the contingent fee basis, it is to be expected that the feigning of disease or the exaggeration or substitution of actual injuries is a feature in personal-injury cases.

Malingering is chiefly limited to disease of the nervous system. In visible physical injuries there is little chance for fraud except by substitution. Fractures, dislocations, and similar purely surgical affections cannot be successfully feigned. Few care to voluntarily injure themselves in a way to cause any misinterpretation. Instances of serious wounds self-inflicted by malingerers may be found in medical and criminal annals, but in these cases the underlying motives were stronger than the hope of financial reward. In litigation cases self-disfigurement is limited to abrasions or similar slight personal injuries, of which the trivial character is readily apparent and about the means of whose production there can be no great doubt. With the nervous system the case is different. In popular opinion, nervous function is indefinite, and nervous diseases obey no recognized laws.

Fraud is attempted in accident cases in three ways:

1. Exaggeration of symptoms actually present. 2. The substitution of origin, or the allegation that preëxisting disease was caused by the accident. 3. Entire simulation.

1. Exaggeration of Symptoms Actually Present. — Of the different ways the first is by far the most common. It is rarely seen in organic injuries. The patient who has sustained a fracture of the skull or an injury to the spinal cord is too seriously hurt to think about exaggerating his symptoms. It would hardly seem possible that uncomplicated injury to one of the peripheral spinal nerves could in these days be so construed as to serve as the basis for a claim that the spinal cord had been injured, and that as a result the patient was suffering from incurable, progressive, and ultimately fatal spinal disease. Yet such a case has recently come to my knowledge. The musculospinal nerve had been injured by a blow which had caused a peripheral and probably temporary weakness of the muscles supplied by the nerve. The physician for the plaintiff must have counted upon the incapacity of the experts for the defense and the gullibility of the jury, or else have himself been very ignorant of nervous diseases. Fortunately, the injury was recognized at its true value.

It is most commonly the functional conditions, and especially neurasthenia, which are fraudulently exaggerated. The number of persons who appear before a claim agent professing great suffering and disability, although they know that there is little or nothing the matter with them, is certainly large. It is impossible to determine the proportion of dishonest claims, but it is unquestionably large enough to make the subject one of the most important in medical jurisprudence. Many claimants allege injuries so trivial and so obviously exaggerated that they are readily persuaded to desist, or to accept a small sum in settlement. The extent and frequency of exaggeration

in the cases examined by the corporation surgeon must be in part determined by the surgeon himself. It is often a matter very difficult to decide. When the reality of some of the symptoms cannot be questioned, it is frequently impossible to ascertain just how far the real symptoms are voluntarily magnified. Although he cannot always give reasons for his opinion which prove satisfactory to juries, an experienced surgeon can usually tell from the appearance and manner of the patient whether he believes himself to be as ill as he claims to be.

But although voluntary and dishonest exaggeration of subjective nervous symptoms is frequent, it should be remembered that not all neurasthenics who may be suspected of exaggeration are themselves conscious of any misrepresentation. What appears at first sight as exaggeration may be only a symptom of disease. This fact is too little recognized outside of the medical profession. Lawsuits on the part of persons suffering from slight subjective nervous symptoms have won for the neurasthenic among claim agents and corporation lawyers the name of swindler and extortioner. It is not surprising that a claim agent, whose medical knowledge must at its best be but superficial, and who is so constantly a witness of attempted frauds, should be skeptical as to the justice of all claims for injuries which cannot be objectively proved. He is unaware that the same kind of exaggeration he so often sees in his commercial calling is daily observed by physicians in cases which are uncomplicated by " litigation symptoms." As has been stated in previous chapters, the patient with traumatic neurasthenia, even when no legal questions are in the balance, describes his symptoms as more severe than there is any reason to suppose they are. However robust or careless he may previously have been, exhaustion of the nervous system has rendered him easily fatigued, worried, and fearful about himself. By the immediate discomfort they occasion, and by

arousing fears of trouble in the future, the pains he feels are double causes of suffering. He cannot keep his mind from his own sufferings, and thereby makes himself worse, in accordance with the law of psychology that attention causes an intensification of sensations. Could he divert his thoughts to other channels he would be on the highroad to recovery. An individual who is occupied, and who has neither time nor inclination to think about his own physical ailments, is not worried by such trifling symptoms as cause suffering to a nervous man who has given up his work and who thinks only about himself. The tendency of introspection which is a prominent feature in neurasthenia, and which is invariably detrimental to recovery, is very much increased when the financial question enters.

Furthermore, a person who is claiming damages for injuries cannot afford to make light of any symptoms which he may observe. All the circumstances by which he is surrounded tend to cause him to repress, until the case is decided, any effort on the part of nature, or of his physician, directed to dispel the physical annoyances and suffering to which he is subjected. By frequently rehearsing his case to lawyers and to experts he comes to believe that he is seriously injured; by constantly bewailing the permanency of old troubles, or the advent of new ones, he concludes that he is " ruined for life." The symptoms of a patient who has arrived at this stage are so out of proportion to the physical injuries he has received that the representative of the corporation which is to settle for them thinks they are voluntarily invented, while the patient himself believes in their reality absolutely.

The discrimination between exaggeration of this character, which is the voicing of disease and consequently sincere, and the variety of exaggeration which is the outcome of dishonest greed, often cannot be effected by the medical aspects of the case alone.

As has been said in speaking of the symptoms of traumatic neurasthenia, the disorder, when well marked, constitutes a fairly typical clinical type. In such cases there is an agreement between the story the patient tells and the way he looks and acts which leaves no doubt in the physician's mind that he is really ill. But in mild cases the examiner has to depend solely on the patient's symptoms, a means none too reliable in litigation cases. It is then usually impossible to decide this question from an examination of the patient alone. If a man maintains that he has a headache or a pain in the back, although he may not present the physical appearances which ordinarily accompany such disturbances, it is impossible to prove that his head or his back do not ache. Even to show him to be a liar in other ways would not necessarily prove that he was lying about these symptoms. It is but natural that the feigning of symptoms of this character, which are subjective, and which do not necessarily reveal themselves by objective and tangible evidences, should prove a tempting bait to persons who wish to turn their presence in an accident to good account, or to swindlers who may invent the history of the accident as well as the symptoms.

The decision as to the merits of such cases usually lies beyond the province of the physician, and must be decided by means of information from outside sources. It should be ascertained if the patient conducts himself at all times in a manner which is consistent with the existence of the symptoms from which he is alleged to be suffering; if he is bringing the claim himself, or if he is the victim of "speculation." The professional and social position of the claimant is not always a valuable aid in arriving at a just conclusion. Physicians, and even the clergy, have been known to bring claims of more than doubtful justice. An officer of a large corporation has communicated to me the case of a priest who was in a collision in

which he received no external injuries whatsoever, and who, although experiencing no ill effects immediately after the accident, brought a claim against the company, alleging various subjective nervous symptoms. The man's occupation was of a character that demanded excessive mental and physical work, which he has continued to do without interruption since the accident. An examination by a distinguished neurologist two years after the accident failed to reveal any evidences of organic disease, and while the physician could not deny that the patient was still suffering from the results of the accident, he stated that there was no proof of injury other than the claimant's own story.

The same gentleman told me of a clergyman who was struck in the back by a mail sack, but who received no visible injury. He threatened suit for $5,000, and compromised for $150.

The question may be one of extreme difficulty. It is one thing to say that there are no evidences of disease discoverable, but it is an entirely different matter to prove that a man does not suffer. When, however, the physician fails to find evidence of disease, and when it can be proved that the patient by his daily life shows himself to be strong and active, there is every probability that his suffering is slight at best.

In hysteria, voluntary exaggeration is much less important than in neurasthenia. Nearly all the cases of traumatic hysteria present definite physical symptoms, such as paralysis, contracture, or anæsthesia. These are practically beyond the realm of successful simulation. The patient may, and often does, voluntarily intensify them when under examination. But he does not change their fundamental character; he merely modifies their degree. As we have seen, the degree of any single physical symptom of hysteria is of much less significance than the fundamental mental state which causes it.

2. Substitution of Origin.—The allegation that preëxisting disease or deformity was the direct result of accident or injury is a form of fraud often difficult of detection, and consequently the most frequent and important in negligence cases. If a person maintains that he was in every way normal prior to an injury, and that only since its occurrence have been observed the symptoms for which damages are claimed, the proof of this assertion depends upon facts relative to his previous physical or mental condition, or, in case such facts are not obtainable or are unreliable, upon whether the disability or disfigurements from which he suffers are of a character such as may result from the kind of injury which has been received. The injuries may very well have been caused by an accident, though not by the particular one at issue. Numbers of cases are on record in which the plaintiff has made defects and deformities received long before serve over and over again as a basis for damages. The difficulties encountered in obtaining sufficient knowledge of the claimant's previous life are illustrated by a case reported by Prentice:

> A corporation was presented with a person apparently an idiot, his condition being represented as the result of a blow on the head. The company's attorney took the precaution of having his picture taken. No record of the accident could be found, and after the man had left the attorney's office he could not be found. The picture, however, was sent to all parts of the country and Europe, where information might be expected; detectives were employed in mines in Montana and in cities on the Atlantic coast, with the final result that the defendant's attorney learned the man's birthplace in Europe, discovered his name upon the army registers, with the record of his physical condition, showing that his condition as a youth had been such as it was when he was presented as a claimant. It was subsequently shown that he had first come to America six months after the accident.
>
> A similar case, though with less fortunate outcome, occurred in my own experience. A liability insurance company defended one of its policy holders in a suit in which mental incapacity was alleged as

the result of an injury to the head. The plaintiff, a Bohemian, twenty-one years of age, had evidently been an imbecile from birth. There were no scars on the head. But as the plaintiff spoke no English and was closely guarded by his family and friends, the defendant gained absolutely no information in regard to his condition prior to the accident. Liability being admitted, and witnesses for the plaintiff testifying to his good physical and mental health before the accident, the only defense was hypothetical. The jury naturally returned a large verdict for the plaintiff.

These cases illustrate the difficulties, but still more significantly the importance, of obtaining knowledge of the preceding condition of the patient in accident cases. It is a necessity to which corporations are rapidly becoming alive, and for which they are, as far as possible, providing. Most corporations now demand a physical examination of applicants for employment. The adoption of this rule is in large part due to the unusual frequency in the past with which herniæ have been alleged by employees to have resulted from blows or falls. The frequency of such claims was so out of proportion to the percentage of traumatic herniæ in general surgical practice that the companies concluded that they often paid for disabilities which had existed for a long time before the injury, and for which they were not liable. By adopting the system of physical examination of proposed employees they have materially diminished the number of unjust claims which are brought against them, and have at the same time established means for securing valuable statistics for traumatic surgery.

The chronic degenerative diseases of the nervous system are particularly liable to misconception as to the influence of trauma in their causation. In many of them the evidences as to the possibility of a traumatic origin are still very slight, although we are unable to assert that they cannot directly result from injury independently of the coöperation of other causes.

In traumatic epilepsy and general paresis, which sometimes

develop in immediate sequence to blows on the head, it may often require both delicacy of diagnosis and a very full knowledge of the patient's condition prior to the receipt of the injury before it is possible to determine the appropriate causal value which should be ascribed to it. As has been stated, in these diseases particularly there are many opportunities for error. If a previously healthy person, a few weeks or months after a fall on the head, by which the skull may or may not have been fractured, begins to have convulsive attacks, which at first are localized to certain muscles, but which later become general, the contention is unavoidable that the epilepsy is traumatic; that the patient would never have had the disease if he had not been injured. Very much more conservatism is necessary in ascribing a traumatic origin to general paresis when it first appears after a head injury. But in both of these disorders it is essential to have considerable evidence that before the injury the patient had been free from any symptoms of either; for epilepsy frequently appears for the first time in adolescence or in early adult life, and the fall on the head which is alleged to have caused it may be the result of the first epileptic attack; in general paralysis, also, even when there is no question of injury, the epileptiform attack may be the first symptom noticed by the friends of the patient, and it may be by it that attention is called to the fact that the patient had been for some time different in many ways from what he used to be. In such cases, of course, the mental symptoms are not in any way the results of the fall. The attack and the fall simply form another link in the chain of symptoms.

But if the circumstances of the fall were such as to make it a negligence case, the claim might be made that the mental disease was traumatic, and that the fall was *propter hoc,* when in reality it was *post hoc.*

If the symptoms of locomotor ataxia or progressive mus-

cular atrophy are observed for the first time after an injury, it is sometimes claimed that the injury was the sole cause of the disease.

As has already been shown there is no evidence that locomotor ataxia may be entirely due to injury. When trauma is alleged as a sole cause, there is a far greater probability that the disease had existed, perhaps unperceived, before the injury. The affection is always of extremely insidious onset, and often passes unrecognized in its earlier stages. These facts should make one chary of too hastily ascribing the development of any case to traumatic causes alone.

Progressive muscular atrophy has only infrequently become the subject of medico-legal inquiry. The evidence necessary for the establishment of a traumatic origin of the disease has already been mentioned.

In paralysis agitans the chances of error are fewer. Cases of paralysis agitans which follow injury or fright only exceptionally reach a rapid general development; so that if a patient has marked tremor or stiffness of the muscles, or characteristic attitude or expression soon after an accident, it is highly probable that the disease had existed before.

It must be clearly understood that a person who asserts that any of these diseases is the direct result of injury may do so with perfect honesty. He may himself have been ignorant of their preëxistence. Thus, a patient of Prince's sued for damages, alleging that locomotor ataxia, from which he was suffering, was the direct result of a fall from the back platform of a railway car. The plaintiff frankly acknowledged in the court room that he had suffered from shooting pains in the legs of a typically tabetic character for six years previously. By this admission he vindicated himself from any suspicion of fraud, but he also gave almost certain proof that the disease had antedated the accident.

There is great opportunity for error in these cases. Dr. Peterson has communicated to me a case (already referred to) of progressive muscular atrophy in which a woman sued for injuries received by a blow on the head caused by the falling of a revolving fan. The plaintiff alleged that since the accident she was nervous, tremulous, and could not sleep well. She was examined by the defendant's physician, who failed to observe the atrophy and loss of power in the hands, and apparently found symptoms of nothing more serious than simple traumatic neurasthenia. After twelve months, when the case came to trial, the plaintiff's counsel frankly admitted that his client had suffered for several years from a chronic and incurable nervous disease, which had been rendered worse by the accident. There was, of course, no attempt at deception; but nothing could illustrate better the possibility of fraud by claiming that preëxisting disease had been caused by the trauma. The time elapsing between the receipt of the injury and the trial was long enough for progressive muscular atrophy to develop, and had this patient been unscrupulous, and her physician dishonest, she could undoubtedly have recovered a large sum on the complaint that the chronic spinal disease had been the direct result of injury.

What has been said concerning the possible preëxistence of the chronic organic diseases of the nervous system may be repeated, with certain modifications, for the functional nervous disorders. It is very essential to know something about the previous history of the patient. Neurasthenia very commonly develops in persons who had given no symptoms before the accident, but the fact that they were well before should be proved. It is particularly important to know if there had been no manifestations of hysteria before the appearance of the particular ones which were complained of soon after the accident. Thus, in Bremer's case (p. 472) of astasia-abasia, the woman

who sued for damages, alleging that the paralysis of the legs would not have occurred had she not been in an elevator accident, was shown to have been for years a victim of various hysterical accidents, and to have given many proofs of an extremely disordered mental state.

3. Simulation.—Much confusion has arisen from an indefinite use of the term simulation. It has been made to apply to cases which were exaggerated as well as to those which were entirely created. It will be used here exclusively as a designation for those cases in which there is no foundation in fact for the symptoms which are alleged.

FREQUENCY.—It is now generally admitted by railway officials, and believed by neurologists, that fraudulent simulation is rare. Simulation of all kinds is frequent in armies, in prisons, and in institutions for insane criminals, and in the earlier periods of the history of traumatic nervous affections it was thought to be very frequently associated with them. Riegler was the first to observe the great increase in the number of railway claims after the passage in Germany of the law by which injured persons were to receive payments, graded in amounts in accordance with what they had previously earned, for the time they were unable to work. He was led to believe that feigning was very frequent, and he stated that all the nervous symptoms resulting from railway accidents were either simulated or else due to organic injury of the nervous system.

The opinions as to the extent of simulation have varied a great deal in Germany, where the subject has received the most thorough scientific attention. Hoffman at one time reported simulation in thirty-three per cent of his cases. From several of the alleged patients he obtained confessions. One man had been coached by a physician for the simulation of epilepsy; another for the pretense of anæsthesia. Seeligmüller found simulation in twenty-five per cent of cases; Oppenheim in four

per cent. Shultze regarded it as frequent. Golebiewski, whose observations on this point must command great respect, says simulation does not exceed two per cent of all cases. In this country absolute simulation is generally considered as unusual. Knapp, Dana, Putnam, and others agree in believing that few persons could carry out, under severe investigation, a system of successful feigning. Judd and Walton, on the other hand, think the percentage of simulation is not small. However, as Seguin says, in this country "claimants are very rarely subjected to scientific watching and to repeated examinations; the physician or expert is expected to deliver an opinion after one or two interviews with the patient, so that the chances of detecting simulation are much reduced." In my own opinion, simulation of disease by healthy persons is distinctly rare. Substitution is much easier than creation, and my experience teaches me that most of the frauds in negligence cases are attempted by clever swindlers who get hold of a sick man or an injured man, teach him the story of some accident, and have him swear that that accident caused his disability.

Nevertheless, simulation must be considered in all accident cases. "'Drag your leg, you fool! don't you see the doctor coming?' was called out by a workman to his fellow who had been in an accident, and heard by the doctor as he was crossing the yard to see him." Godkin tells an amusing story of a plaintiff who testified "that his right arm had been so injured that he was unable to raise it any longer to a horizontal position. Upon cross-examination the defendant's counsel asked him to indicate how high he could now raise his arm. 'Only so high,' replied the witness, lifting the arm with apparent difficulty a few inches from his side. 'And how high could you raise it before this unfortunate occurrence?' asked the lawyer suddenly. 'So high,' replied the witness, raising the same injured arm above his head with ease."

Not so very long ago a man brought a claim against a Western railway for injury to the spinal cord which was the alleged result of a collision. The patient was apparently paralyzed in both legs, and for two years was never seen to walk without crutches. One day at the end of this time, while sitting with the claim agent in the office of the corporation, a settlement was agreed upon, and the man signed the release and received his check. He arose briskly from the chair and commenced to walk rapidly out of the office. "Halloo!" said the claim agent, "have you not forgotten something?" The satisfied claimant could not repress a blush at seeing that he had left his crutches standing idly in the corner.

During trial terms of the courts there are constantly being published in the newspapers accounts of litigated cases in which the complaints are dismissed and the plaintiffs shown to be open to indictment for conspiracy and attempt to defraud.

That simulation may come to be a systematized occupation and a means of livelihood is well shown by the Freeman family. In an interesting and cleverly written pamphlet entitled "Paralysis as a Fine Art," published in 1895 by the Association of Railway Claim Agents, some of the adventures of the Freemans are described. The family consisted of a father, mother, and eight children, English (or Polish) Jews, all of unsavory reputation (Fig. 94). There is no definite information concerning the male members; but the mother, Mary Freeman, and the two daughters, Jennie and Fannie, between January, 1893, and December, 1894, entered no fewer than nine claims for damages against railway companies. That the claim was fraudulent and the pretenses false in their last attempt each and all swore before a notary in Chicago at the time of their final exposure. There is convincing proof, also, that all the claims that they ever made were fraudulent. It may be well to briefly outline how these frauds were committed:

Mary Freeman, the mother, was forty-three years of age, slovenly in appearance and dress. "She was dirty, and infested with vermin." Hers was the ruling spirit of the family, and she directed the operation of all the frauds. She had been several times arrested for theft.

JENNIE FREEMAN.

On September 11, 1894, she brought a claim against the Chicago City Railroad Company for injuries caused to her right arm by the sudden starting of a car. She received $100.

Jennie Freeman, the eldest daughter, was between eighteen and twenty-two years of age. Her morality was questionable. She was usually on good terms with some physician or attorney. She had been ar-

ESTHER FREEMAN. FANNIE FREEMAN.

FIG. 94.—Some members of the Freeman family.

rested for theft. Her appearance and manner were pleasing and gentle.

On January 9, 1893, she brought a claim against the Chicago City Railroad Company for injuries received in a collision between two cable cars. "She alleged total paralysis from the thighs downward,

loss of sensation, want of control of the bowels and urinary organs, and pretended that she was a cripple and ruined for life." The company's physician "thought the girl was shamming, although the symptoms were so closely simulated that it was apparently a real case of paralysis." The company gave her $500, and Miss Jennie is said to have recovered a few days after the damages were paid.

This paraplegia followed a course of unusual benignity. On October 5, 1893, the patient brought claim against the Manhattan Elevated Railroad Company, of New York, for injuries received by falling against the car door of a Second Avenue train while it was rounding the curve at Twenty-third Street. Settlement of $100 to her physician and $125 to Jennie Freeman.

On May 16, 1894, claim against the Boston and Maine Railroad of having been injured by slipping on a banana peel when stepping out of a car at the Prospect Hill Station. The banana peel was produced in evidence. Jennie Freeman received $125 in respect of these injuries, though the claim agent suspected fraud. She was next heard of in Chicago, where on June 28, 1894, she brought claim against the Illinois Central Railroad Company for injury received by being thrown against the back of a seat through the sudden stopping of a train. "She alleged total insensibility of the lower portion of the body, practically amounting to paralysis. Had a sore on her backbone, immediately above the top of the corsets. Alleged an inability to control the function of the bowels, etc. The examining physician made every possible test, but she seemed totally insensible to all pain. The claimant was settled with for $200."

On September 10, 1894, Jennie Freeman alleged to have fallen from her seat while rounding a curve on one of the lines of the West Chicago Street Railroad. She represented to the company, through her mother, that she was paralyzed. Fraud discovered by claim agent, who, by an unexpected visit, discovered the alleged paralytic sweeping her room.

Fannie Freeman, younger than Jennie, was untidy in her dress, quiet, and had little to say. She had been arrested for theft.

On April 20, 1894, she claimed to have been injured by slipping on a banana peel on the West End Street Railroad Company's car in Boston. She complained of paralysis of motion and sensation from the waist downward, and an inability to control bladder or rectum. Over the lower thoracic vertebræ there were traces in two places where the skin had been abraded. The company's physician says, in his report: "Tenderness to pressure and percussion over the lumbar and dorsal vertebræ. Can't stand, walk, or sit unless completely sup-

ported, and then can only be held in the half-sitting and half-reclining posture, all the time evidencing great agony. When I stuck pins into her feet and legs, and touched them with my hands, she declared she could not feel any sensation, and I couldn't surprise her into any painful expression." The prognosis was given as unfavorable, and Fannie Freeman was paid $325 [or $425].

On June 6, 1894, Fannie Freeman, under an assumed name, brought claim against the New York, New Haven and Hartford Railroad Company at Boston. This case was an identical reproduction of the preceding, even to the patient's maintaining that it was "her first accident." But she was unfortunate in being visited by the same examiner who had seen her a month previously for the West End Street Railroad Company, for the physician this time recognized the fraud, which he had failed to do in the first instance.

On December 24, 1894, Mary Freeman claimed to the general superintendent of the Chicago, Rock Island and Pacific Railroad Company, Chicago, that her daughter Fannie had been injured by falling on her back in a car of the company. She alleged that her daughter was paralyzed from the waist down, and had lost all sensation in the legs; that there was no power over the rectum or bladder, and that the young girl was ruined for life. There were so many suspicious circumstances about the case that the family were watched. Before the expected visit of the company's doctor the alleged cripple was seen, from a hole through the floor of a room above hers, to get nimbly out of bed and put her feet in a tub of iced water, in order that they might feel cold and lifeless to the examiners.

Some of the results of the medical examination, as embodied in the surgeon's report, are as follows:

"Pulse at first 104, but it changed so that at the last of the examination it was 132. Her back was marked by a slight spot about the top of the sacrum, entirely superficial and movable over the underlying tissues, which may have been produced by some injury, or by the abrasion of some part of her clothing. Sensation existed about halfway down the thighs, but below this point it was alleged to be absent. There were no evidences observed of incontinence of urine or fæces, and no girdle sensation was complained of. An unexpected test of raising the foot in the air caused it to stop there, though tests of physical endurance were applied and successfully withstood."

The doctors, satisfied of fraud, reported that there were no objective evidences of the conditions complained of.

The following day the three women were arrested. Fannie Free-

man, "the paralyzed lady, jumped out of bed, cursed, and marched around the room with a tramp like a grenadier."

The Freeman cases have been quoted in some detail because there seems little reason to doubt that all the claims were absolutely fraudulent, and that the symptoms in each case were entirely fictitious.

The cases in which there is a detailed report of the medical examination show how far and in what way the symptoms of nervous disease may be simulated. Certain of the symptoms of the Freemans will be referred to again.

DIFFICULTIES.—The person who makes a claim for personal injuries, when in reality he has not been injured and is not ill, sets himself a task of no little difficulty. In the first place, he must give some account of himself and of his social relations. He may be able to satisfactorily conceal his past if he has anything to conceal. It is often difficult or impossible to obtain information as to the antecedents of foreigners, who speak no English, and who live in non-English speaking communities. A notorious rogue would probably be detected at once; and the appearance and methods of an impostor who has been caught by any large corporation might be recognized when he brings claim against some other company. Fannie Freeman was on one occasion detected in Boston because she was examined by the same physician who had seen her a month previously for another company. Claim agents also exchange for mutual benefit any information which they may gather in regard to suspicious characters. The system with them is not so highly elaborated as is the system of exchange by life-insurance companies of the names of rejected candidates, but it is usually sufficient to brand a man who has been detected shamming several times.

Even if the simulator is able to furnish satisfactory proof of his honesty and respectability there still remain for

him serious difficulties. To closely imitate nervous disease requires no mean order of intellect. A clever simulator must have quickness in adapting himself to new situations, a plausible and ingenuous manner, the power of close attention, a good memory, and some knowledge of the symptoms of disease of the nervous system. These are mental qualifications all of which are rarely present in the individuals who resort to the feigning of disease for financial purposes; yet all of them must be present if a physician who is reasonably skillful and who is on the lookout for shams is to be made to believe that the symptoms are genuine when in reality they are entirely assumed.

The simulator may be successively submitted to the observation of physicians of different ability who make examinations of different characters in diverse ways. To deceive them all, he must have his wits about him. The general appearance and manner, which are among the most reliable signs for medical diagnosis, must to a certain extent correspond with the bearing of persons who really suffer from the symptoms he is trying to imitate. These evidences are the outward expressions of the morbid agencies which are at work; they are difficult to describe and still more difficult to counterfeit. A knowledge of their significance and characteristics in disease is acquired only by long experience in general medical practice, a fact which constitutes perhaps the most serious stumbling-block to the simulator. Even if he had the necessary knowledge of the general appearances in nervous diseases, they are symptoms almost impossible to successfully imitate. They reveal the condition of the whole organism and are largely beyond the power of voluntary control. When the simulator tries to copy them he overdoes it. His manner is exaggerated and theatrical; his symptoms are severer than real symptoms usually are; his sufferings are all in the superlative and his prospects are ruined. It is the exaggerated character of the

story that often at once excites the physician's suspicion and leads him to apply searching tests which result in exposure.

Without remarkable powers of attention the simulator cannot hope to meet with much success. He must be always ready to be examined, and constantly on the alert even when he does not suspect himself of being observed. When under direct examination or observation he must be ever on his guard not to be caught by tricks nor to be betrayed by his own emotions. Page tells of "a man who based a large demand for compensation from a railway company on stiffness of the elbow and inability to move the arm, the result of a collision. A verdict incommensurate with his expectations having been recorded, he threw up his arms and exclaimed, 'My God! I'm a ruined man!'"

To sham any form of disease requires that several symptoms must be counterfeited at the same time. To imitate any one objective symptom successfully for the length of time usually taken for medical examination requires a very close attention; to simulate several simultaneously is for the majority of persons impossible. To keep constantly before the mind that the legs must not move, that pin pricks must not hurt, and that movements of the back are painful, is an intellectual exercise of considerable activity. If these symptoms do not really exist, the attention, while one of them is being examined, is usually concentrated on it at the expense of the others. As a result, alleged paralyzed legs may be seen to move, movements of the back may no longer cause suffering to the subject, or he may be startled into involuntary expressions of pain when pricked in areas said to be anæsthetic. A long and thorough examination, especially if the examiner be suspicious of fraud, will usually disclose some or all of the symptoms to be false. Even when the simulator is entirely conversant with the character of the disturbances he is shamming he is rarely able to

keep himself from being confused by their examination in rapid succession. Fannie Freeman, who was undoubtedly, by experience and by teaching, thoroughly conversant with the symptoms of paraplegia, forgot to let her leg drop when it was lifted up by the examiner. Instead of falling lifelessly to the bed, as it should have done, it was held in the air, and, by successfully resisting the efforts of the doctors to push it down, showed a high degree of muscular strength.

A qualification closely allied to power of attention is a good memory. It is very essential, if a story is to be believed, that when retold it remains essentially, though not minutely, the same. Many claimants excite suspicion by the variations and inconsistencies which appear when they relate the details of the accident to different persons. Genuine symptoms have a certain permanency. A clumsy simulator will often forget, when talking about his case to one examiner, just what he has complained of to the claim agent or to the physician whom he had seen before. On the other hand, it is not always just to infer, because a history of injury or symptoms is indefinite or contradictory, that the claimant is an impostor. Both the memory and attention are impaired to a certain extent in neurasthenia, and still more so in hysteria; but all such inconsistencies will cause a claimant to be regarded with suspicion until it can be proved with a remarkable degree of certainty that they are not the results of clumsy and voluntary attempts at deception.

Knapp believes that simulation must be rare because the larger number of persons who would be willing to fraudulently feign disease are unable to obtain a sufficient knowledge of symptoms to successfully imitate them; that simulators are of a class rarely able to refer to medical books, and that physicians who might be able to give instruction in this kind of fraud could not be persuaded to do so. With this view I do not agree. Inmates, attendants, and hangers-on of large institu-

tions, such as city hospitals, almshouses, penitentiaries, etc., often become, through long experience, well acquainted with the various forms of human suffering. Their moral code is lax enough to let them turn this knowledge to profit, if occasion offers. When a large claim is made and the company decides to contest it, the chances of successful simulation are always small. The simulator fails then, both because his motives are very thoroughly investigated and because he is subjected to a series of rigid examinations. But in by far the larger number of instances the case does not come to trial, because the claim made is generally small, and the companies find it cheaper to settle it than to go to the expense of trial and expert opinion. In this way a simulator, without being particularly expert, may succeed in a small way, although both claim agent and physician suspect him of being a swindler.

The form of disease most commonly simulated is neurasthenia. For it no special knowledge is necessary. The general symptoms of "railway spine" are familiar to most physicians, even though they be unacquainted with general nervous diseases. Physicians can very easily simulate railway spine, or teach how it should be simulated, and experience shows that they sometimes do so. The condition can also be feigned with moderate accuracy by persons who know nothing of general medicine. There are no positive objective symptoms by which the fraud can be discovered, and there are a great many persons who, without any medical degree, have acquired sufficient familiarity with various forms of disease to give a fairly good reproduction of the picture of neurasthenia. Page remarks upon the knowledge possessed by persons in the humbler walks of life regarding the history and symptoms of the kind of injury which is popularly supposed to be inevitable to a railway collision. Knowledge of the symptoms of neurasthenia may be easily acquired. If the simulator cannot learn

them himself, there is little reason to suppose that he would have any serious difficulty in finding a physician ready and able to instruct him. Knowledge of the symptoms of organic nervous disease is more difficult to obtain, and most physicians who would impart it for fraudulent purposes do not themselves know very much about them. But simulation of organic nervous disease can be successful only when the examiner is himself careless or unfamiliar with the symptoms and the means of demonstrating them, for organic nervous disease cannot be feigned with any degree of resemblance. With assumed symptoms indicative of organic disease, if the simulator passes undetected, it is less of a testimonial to his knowledge than it is a proof that the examining physician has been superficial, careless, or ignorant.

Detection of Simulation.—The physician's part in the detection of simulation is to show that the clinical picture is not produced—a task that is rendered easy by an appreciation of the various factors which of themselves make the simulator's part so difficult. While some simulators are so clumsy that they are immediately turned away from the claim agent's door, well-trained impostors like the Freemans merit the best skill of the medical man. For the exposure of such as they, the physician must not only be on his guard, but he must constantly keep on the defensive the person he is examining. By bearing in mind the disadvantages under which the simulator is placed, the examiner can in many ways render them still more embarrassing. The detection of skillful simulation of any one symptom depends upon diverting the simulator's attention. He must be taken off his guard. When he does not know that he is being watched, the physician may find that the facial expression undergoes a marked change. Complaints of pain may be forgotten if he can be made to talk of something not directly relating to the accident. If the examination is

sufficiently long, it is generally found that there are periods during which the expressions of suffering momentarily cease. These lapses may be short, and the simulator may soon collect himself again. But a momentary forgetfulness of this character should at once excite the physician's suspicion. It is by attracting the patient's attention away from the symptom, the true nature of which the physician desires to assure himself, that the fraud frequently becomes apparent. It is very hard to think for any length of time on two things at once, and if the examiner is in reality examining for one symptom while he appears to be testing another, he may often be able to prove that one of the symptoms exists only when the person under examination has his attention fixed upon it.

Detection of the simulation of those disorders which may result from accidents is in most cases possible. Clinical possibilities are limited, and when a man says he is suffering from the effects of an accident the question immediately arises, What is the pathological condition to which the symptoms are due? By eliminating disorders with which the symptoms do not agree at all, the physician who is familiar with the various morbid conditions which cause disturbance of nervous function can reduce the possibilities in any case to the one or two conditions with which the symptoms agree most closely. It then remains for him to determine if the resemblance to either or both of these conditions is sufficiently close to justify the symptoms being regarded as genuine and not feigned. For example, an allegation that the arm is injured is not enough. It should be shown how it is injured, and if careful examination shows that it is surgically intact, and that the symptoms are at variance with those of any known variety of nervous condition, it is entirely justifiable to infer that the arm is not injured at all. Page reports a case in which the dribbling of urine caused by a large prostate was alleged to be due to injury to the spinal

cord. The man was not paralyzed, and he was at once regarded as an impostor, on the ground that any injury to the spinal cord which could cause weakness of the vesical sphincter would cause paralysis or some other symptom of spinal-cord injury. This was not absolutely conclusive, as lesions of the conus medullaris (see page 205) may cause disturbances of the bladder without paralysis of the limbs. In such cases a local examination of the prostate is desirable. In a recent case, a young man was in a railway collision in California, as a result of which he alleged a dribbling incontinence of urine. Examinations by several local physicians agreed in the diagnosis of injury to the spinal cord. The absence of other spinal-cord symptoms, and the absence of a history pointing to spinal-cord injury, led to a local examination. The prostate was found very much enlarged, and its condition explained all the symptoms. Its condition was in no way dependent on the accident.

Fannie Freeman, in her last attempt, might have been detected at once, from the fact that she failed to make her paralysis agree with any known form of paraplegia; for, with alleged complete loss of power of the lower limbs and of the bladder and rectum, the feigned anæsthesia only reached to the middle of the thighs. If the paralysis had been due to injury to the spinal cord, the anæsthesia would have had a very different distribution. If the whole condition had been hysterical, the sphincters would have remained unimpaired.

The choice of diseases for simulation is restricted, inasmuch as the variety of nervous disorders which may result from injury is not large. Acute organic injury to the brain, spinal cord, or peripheral nerves causes symptoms which cannot be feigned. General paresis, progressive muscular atrophy, and tabes cannot be copied with even a shadow of resemblance. Feigned paralysis agitans would quickly be pronounced spurious by any medical student who had examined one genuine

case of that disease. Epilepsy is not uncommonly advanced as a cause of action in negligence cases. But usually the fits have antedated the injury. The simulation of epileptic seizures, or "dummy chucking," not rare in penal institutions, is not often seen in court. If epilepsy is alleged as the result of accident, the evidence of nonmedical witnesses of the fit should be regarded as insufficient proof.

Neurasthenic disturbances are the ones most frequently complained of by simulators, who are quick to take advantage of a condition which is practically devoid of objective symptoms. It is in these cases especially that, as Page says, "it is only by a consideration of every feature and aspect of the case —clinical, pathological, social, and moral—that you can rightfully estimate the kind of exaggeration or malingering with which you have to do." The physician may believe that a case of neurasthenia is absolutely feigned; but unless he has some definite proof upon which to base his opinion, his testimony to that effect would not carry much weight. If a claimant restricts himself to assertions of pain in the back, loss of energy, inability to work, and similar complaints of purely subjective character, the physician may be sure that the symptoms are exaggerated, but he cannot show that they are false. The truth in regard to claims based upon symptoms of such a character must be determined by general considerations, and by information gained from sources other than the medical examiner.

Hysteria is probably never simulated in America.

A claim agent, in telling me of many cases from his experience in which the symptoms had speedily disappeared after settlement, said that there was one case he had never been able to understand. A man had received a slight injury in a collision, as a result of which he alleged that his legs were paralyzed. In spite of many circumstances which appeared suspicious, the claimant was compensated. But the injured man, instead of getting well, remained a paralytic, and it be-

came generally believed that his spinal cord really had been injured. Two years after the damages were paid he suddenly regained full power of the legs. The fact that this case was misunderstood is in accord with the common opinion, both lay and medical, held in America in regard to hysteria.

It is not generally appreciated that hysteria is a disease, and not premeditated swindling. Until the subjective reality of hysterical symptoms becomes more generally believed in, the disease cannot be popular with simulators. To feign a disease which is itself regarded as fraud would be worse than useless. But the failure to recognize that symptoms are hysterical, and not voluntarily created, may cause much injustice to the hysterical patient. He may be immediately branded as an impostor the moment the objective symptoms are shown to be not organic. There is more probability of a hysterical person being classed as a simulator than of a simulator attempting to feign hysteria. The diagnosis between hysteria and simulation, however, should not be difficult to anyone who is familiar with hysterical symptoms. Hysteria is the cleverest simulator of all, and is adroit and consistent when the simulator is clumsy and confused. The hysteric copies, while the simulator caricatures. Hysteria causes its victim to involuntarily mimic organic disease much more closely than organic disease can be imitated voluntarily. But even the mimicry of hysteria is not perfect, and by the irregularity and association of its symptoms it can readily be differentiated from organic disease.

The forms of disease which have been described as unclassified are accompanied by symptoms too serious to permit them to be misinterpreted or imitated.

An individual or a corporation which are being sued for damages for personal injuries should be entitled to ascertain the character and extent of the injury for which they are held liable. Viewed from a purely commercial standpoint one has

a right to know what one is paying for, or, in other words, to insist upon a definite diagnosis before payment is made. Such diagnoses, if they cannot be made at once, usually become possible after the observation of a few days or weeks. When the possibilities of exactness in diagnosis of disorders of the nervous system are more generally recognized, and when "shattered nerves," and similar meaningless designations, come to be regarded as insufficient claims in negligence cases, the detection of simulation will be rendered very much easier. The simulator would then be forced to choose some definite condition to feign, and to have all his symptoms in accord with it.

The symptoms most difficult to simulate, or even to exaggerate, are those known as objective. They are of sufficient importance, however, to be considered in detail.

Simulation of Individual Symptoms

Motion.—The most difficult objective symptom to counterfeit is the loss of voluntary motor power. If the simulator decides to enlarge upon the simpler complaint of pain in the back and general nervousness, he is very apt to be led away by the attractive picture of paralysis. By refusing to move his limbs, or by giving them the appearance of lifelessness when they are handled by the examiner, it seems to him comparatively easy to substantiate the statement that certain muscles have lost their power of voluntary movement. He loses sight of the fact, however, that true paralysis has certain signs which are impossible of voluntary imitation, and that it is well-nigh useless to attempt to impose in this way upon a careful physician who is familiar with the various phases and causes of impairment or loss of muscular power.

There are two reasons which seem to justify the statement

that true paralysis cannot be well feigned. One is that the condition of the muscles themselves—the flabbiness, softness, and lack of tone—cannot be voluntarily counterfeited with any great degree of similarity. The other reason is that organic paralysis does not occur as an isolated symptom, but is invariably accompanied by others which are completely beyond the control of the will.

If paralysis is simulated, when the limb is lifted up and suddenly released it may be held a moment before it is dropped again; or if the examiner tries to move the limb his hand may detect that there is a moment in which the muscles are held stiff, although they relax again as soon as the subject discovers the object of the test; or the limb may be seen to move under the influence of painful stimuli, or when the person under examination forgets to hold it motionless.

Paralysis is rarely isolated. Appearing acutely, it can take place only as the result of organic injury to the nervous system or of the fixed ideas of hysteria. If the brain is the seat of a lesion there may be a resulting hemiplegia or monoplegia, but the paralysis is ushered in with considerable cerebral disturbance. Paralysis due to primary intracranial lesions rarely occurs without unconsciousness or convulsions or severe headaches, or some similar severe cerebral symptoms. The functions of the cranial nerves cannot be voluntarily suppressed. A man may claim that he does not taste, see, or hear, statements which may be difficult to verify; but he cannot voluntarily cause permanent deviation of the eyes, such as results from palsy of the nerves to the ocular muscles, nor can he keep his face drawn to one side so that it does not become apparent, during movements of talking or laughing, that the facial nerve is not paralyzed. Hemiplegia is usually quickly followed by unilateral changes in reflex activity, which, as we shall see, cannot be imitated in a way to deceive anyone familiar with

these important evidences. The symptoms of hemiplegia, as the result of injury, are too complex for the average simulator, and are pictures with which he is usually unfamiliar. Any attempts to copy them must be unusual, if they are ever made.

Loss of power in the legs seems to the simulator the clinical type most consistent with the general theory of paralysis which follows accidents. Injury to the spine with resulting paralysis of the legs is a comparatively common condition, and the average simulator is rarely a sufficiently profound pathologist to recognize the fact that traumatic paraplegia is most frequently the result of fracture or dislocation of the vertebræ, such as occur only after very severe accidents. Furthermore, organic injury to the spinal cord is without exception accompanied by many symptoms which do not permit of voluntary imitation. Trophic disturbances are beyond the realm of imitative art; by soaking the legs in iced water they may be made to feel cold and look blue, but there is nothing but actual nerve injury which can cause the appearance of a beginning bedsore, and the cleverest actor cannot make his muscles waste or his nerves give degenerative electrical reactions. In recent organic paraplegias there is a softness and flabbiness of the muscles which is characteristic; and as the paralysis of some muscles passes away, as it usually does in time, they resume their normal tonic action. But the muscles remaining paralyzed offer no resistance to their opponents, and contractions and deformities are the results. Organic paraplegia is usually accompanied with some trophic symptoms, together with anæsthesia and changes in reflex activity. Even without the aid of them, it is usually possible in any case to demonstrate whether the paralysis is true or feigned.

The simulation of organic monoplegia is still more difficult. It is often alleged that the arm is injured, but these are cases which are characterized by peripheral rather than central

injuries. The neurologist can at once decide if there has been any injury to peripheral nerves, and a surgical examination should quickly discover the integrity of the bones and joints. In the absence of any such evidences of peripheral injury it is justifiable to infer that the case is feigned or hysterical. Traumatic monoplegia from any cause is very rare, and as a result of spinal injury it is practically unknown, which may be explained by the fact that the spinal localization for the movements of any one limb extends over several segments of the cord. Any injury which affects a sufficient number of segments to cause a monoplegia will be accompanied by paralyses of other parts.

Hysteria can imitate better than any simulator the organic paralyses, and, as has been said, there is more danger that the involuntary mimicry of hysteria be regarded as deliberate imposture than that the impostor who feigns paraplegia may pass undetected. The decision of the question between hysterical paralysis and simulation may be very difficult. If the plaintiff's physician asserts that his client is suffering from organic paralysis, the truth of the statement may be determined with certainty. But if the claim should be made (it rarely is) that the paralysis is functional, it may be impossible to refute it by objective signs. The characteristics of hysterical paralysis have already been described in such detail that it will be sufficient to recall the fact that they differ in essential particulars from the paralyses arising from organic causes. But the differences are very similar to those which exist between organic paralysis and feigned loss of muscular power. The hysteric, like the simulator, suffers neither from bedsores nor from loss of sphincter control; he drags his foot on the side rather than at the toe; there is no circumduction of the leg; there are usually no changes in the reflexes; hysterical symptoms are more marked when the patient knows that he is being watched.

In a word, although hysteria cannot imitate the motor symptoms which do not permit of voluntary imitation, there are many cases in which, from the character of the motor symptoms alone, it cannot be inferred whether the condition is the result of premeditated deceit or of the unconscious mimicry of hysteria. To decide this question recourse must be had to other symptoms.

In the larger number of the cases of hysteria the associated signs are sufficient to permit the disorder to be recognized. Contracted visual fields, the mental state, and the anæthesias, if associated with a paralysis not characteristic of organic lesion, are sufficient to establish the diagnosis. But occasionally paralysis occurs as the only symptom of hysteria, and in such cases the elimination of shamming is extremely difficult. Hysterical paralysis presents the one difference that, although it may be shown by hypnotism to be unreal, it does not fall into the traps which so frequently catch the simulator. The hysterical patient may move his leg better when unobserved, but even then there is not the complete preservation of muscular power which the simulator may be shown to have.

Tremor.—Tremor is a symptom of nervous disease which is very difficult of simulation. Attempts to imitate the tremor of multiple sclerosis are overdone. It is doubtful if the tremor of paralysis agitans can be successfully counterfeited for any length of time. Even if it were, there are so many other symptoms which accompany tremor in paralysis agitans that it may be regarded as practically impossible to feign the complete clinical picture of that disease. The gait, the facial expression, the voice, the muscular rigidity, are all as important symptoms of paralysis agitans as tremor. They are too numerous and too complex to permit of consistent and simultaneous mimicry. The other pronounced tremors, such as are observed in paramyoclonus multiplex, tic convulsif, etc., are too intricate to be

voluntarily copied with any degree of similarity. Fine tremor, either in the face, tongue, or hands, is a common symptom of functional nervous disorders. It is almost identical in character with the trembling observed in alcoholism or in excessive tea drinkers. Although it cannot be voluntarily imitated with success, it is well to bear in mind, in examining any case which presents this symptom, that it may depend on conditions which antedated the accident.

Anæsthesia.—In organic diseases of the nervous system anæsthesia may be a most valuable and certain symptom. In diseases of the peripheral nerves, in spinal-cord lesions, and, to a less extent, in cerebral affections, blunting or loss of one or all forms of sensory function is of the highest value for purposes of diagnosis and localization. It may also be a pathognomonic evidence of hysteria. The significance of this symptom can, however, be easily misinterpreted. Loss of cutaneous sensibility is extremely changeable in functional disorders, and even in organic disease it may vary considerably, so that examinations on different days or at different times of the same day do not yield identical results. The value of anæsthesia as an objective symptom is further impaired by the fact that sensibility varies so extensively in degree in normal individuals. Women are less sensitive to pain than men. Analgesia, partial or complete, is not a rare occurrence in apparently healthy criminals. Certain races, especially the Polish Jews, have a variety of anomalies in pain perception. In persons who naturally do not feel pain acutely the simulation of analgesia becomes a comparatively easy matter. There are also a good many individuals who can suppress any evidence of pain as long as their attention is fixed upon this object. Many of the "human pincushions" in museums really feel pain, but, in consideration of the salary they receive, agree to permit an inquisitive public to stick hat pins through the calves of their

legs, or resort to similar attempts at causing them to give expressions of discomfort or suffering. Knapp quotes from Pitres a case of this kind in which the fraud was exposed: "A man had for several years taken the part of the anæsthetic man in a show, enduring pricks, cuts, etc., without the slightest manifestations of pain. In order to obtain the shelter of hospital life he feigned a disease, one symptom of which was anæsthesia; but when he was subjected to the unexpected application of painful stimuli, Pitres proved at once that the anæsthesia was feigned."

Persons whose sensibility to pain seems normally not highly developed, and those who have educated themselves to repress any of the ordinary manifestations of pain, have naturally the best chance of escaping detection in the simulation of anæsthesia, and, when the examination is not thoroughly and thoughtfully made, may lead the examiner to believe that insensibility really is present; but even for these cases there are several tests which will generally prove the unreality of the alleged sensory symptoms.

In all sensory examinations the eyes should be blindfolded. Tests for analgesia are best made when the subject thinks he is being examined for something else. Thus, if the legs are alleged to be anæsthetic, a sudden thrust of the needle into one leg, while the operator is examining the other, may cause it to be quickly drawn up. Electrical currents are usually considered of value for the detection of feigned analgesia. They may be applied by means of a strong faradic battery, whose current strength should be suddenly increased without the knowledge of the subject. Knapp suggests that this test be applied during the course of the ordinary routine of electrical examination of the muscles. The individual muscles may be examined in the usual way, and then, with one of the electrodes over the alleged analgesic area, the full force of the battery is sud-

denly turned on. Strong faradic currents are very startling and very painful, and this test usually results in some sudden expression of surprise and suffering if the insensibility is not genuine. Detection of shamming of the loss of sensibility to touch is more difficult. The person whose tactile sensibility is normal will know if there has been no contact, as well as recognize light touches; but he can deny said sensations when he really feels them. To prove his statements false is not always possible. The test usually employed is to mark out the areas in which insensibility to touch is said to exist, and, after an interval of a few minutes, to repeat the examination and note if the results of both examinations agree. This test appears to me to be of very slight value for the detection of simulation. If the anæsthetic areas measured at different times agree perfectly, it is certain evidence that there is some serious sensory disturbance; but it is not a proof of feigning if the limits of the loss of sensibility to touch vary somewhat at different times. Anyone accustomed to making examinations of cutaneous sensation will remember how often he has been baffled in trying to make out the anæsthetic limits in cases of unquestionable organic disease. They vary so widely at different examinations that variation alone is not sufficient cause for the assertion that they are not genuine. Sensation to touch is essentially a subjective symptom and its reality is very difficult to prove. Since, however, in simulation its loss is nearly always associated with analgesia, if it can be shown that the patient feels pain when he says he does not, we are justified in assuming that the loss of touch sense is also false.

Thermo-anæsthesia is rarely feigned. To test for its reality, the patient, while being examined with the test tubes filled with cold water and warm water, may suddenly be touched with a tube of boiling water.

None of these tests for sensation decides positively between simulation and hysteria. Through the suggestion imparted by manipulation and examination hysterical anæsthesia may change its boundaries, or change its situation, or entirely disappear.

It can, however, usually be decided in accident cases whether the anæsthesia be true or false without the employment of these tests, which are, after all, suggestive of the methods of the Inquisition.

In every nervous disorder of which it is a symptom, anæsthesia has certain peculiarities of character and distribution as well as of the way in which it is associated with other symptoms. These are individual characteristics, familiar only to those who have devoted considerable study to the physiology and disease of the nervous system. Were a simulator familiar with them—which he rarely is—it is extremely improbable that he could put his theoretical knowledge to practical use during a thorough medical examination.

When anæsthesia follows the course of the peripheral nerves or the sensory distribution of the spinal segments, or occupies areas which are known to be favorite locations for the insensibility of hysteria, it furnishes by its distribution very strong presumptive evidence that it is not feigned; but when, instead of being characteristic, it assumes a form not in agreement with the other symptoms present, it should at once arouse suspicion as to its genuineness. Such a suspicion may then be verified by the tests commonly employed to discover if the anæsthesia is real in the places in which it is alleged to exist.

The anæsthetic areas, and the characteristics of anæsthesia resulting from injuries to the peripheral nerves or to the central nervous system, and from the mental state of hysteria, have already been described and illustrated. It has been emphasized

that complete loss of sensibility rarely follows peripheral-nerve or cerebral injuries. Since the sensory distribution of the spinal cord has become almost absolutely established, it is just as possible to tell from the anæsthesia what segment of the spinal cord is injured in a case of spinal compression, as it is to refer the numbness and tingling which may follow a peripheral injury to the nerve upon whose lesion it depends.

If in any case the group of alleged symptoms points to compression or injury to the spinal cord, it may be regarded as practically impossible for the simulator to have sensory motor and reflex symptoms all agree, so that they could be referred to injury of the cord at or below any one segment. Thus, to again quote Case III, of Fannie Freeman, the fraud was evident from the anæsthesia alone, for with alleged total paralysis of the legs the anæsthesia only reached the middle of the thighs. Such a localization for insensibility resulting from any injury to the spine is entirely without precedent.

The irregular areas of hysterical anæsthesia obey certain general laws. In traumatic hysteria the loss of cutaneous sensibility is very commonly found only in limbs which are paralyzed.

To distinguish between simulation and hysteria by the examination of sensation alone is often impossible. As in feigned anæsthetic areas, so in parts hysterically anæsthetic, the effects of accidental injuries to the skin, so common in organic anæsthesia, are rarely observed. In feigned anæsthesia the skin bleeds readily; in hysterical anæsthesia the skin has little tendency to bleed when pricked. But usually, by reason of the uncertainty and variability of the sensory disturbances of hysteria, it is necessary to look for associated signs before one can be certain that the patient really believes he does not feel. In traumatic hysteria such additional signs are usually present, and it is by appreciating the character of hysterical paralysis

or contracture, or by demonstrating a functional limitation of the visual fields, that the functional character of the anæsthesia may also be made plain.

Pain and Hyperæsthesia. — It is very difficult to determine the extent and true character of alleged pain. In many cases disturbances of nutrition and the general bearing of the patient permit no doubt of its reality; but because the visible evidences of suffering are absent it cannot always with certainty be inferred that the patient does not suffer. If he is so situated that he can be constantly watched in order that it may be determined whether the sleep is disturbed, or the expressions of pain cease when the patient thinks himself unobserved, the question may be decided with certainty. In negligence cases such opportunities are rarely at hand, and to decide how much the patient suffers (or if he suffers at all), the physician must usually consider each of the many factors in the case, of which the possible motive for simulation, the general bearing of the patient, and the agreement of his description with the ordinary clinical types of pain, are the most important. The examination should be as long as is feasible, in order to find out if the pain appears to be continuous, or if by distraction of the attention the patient can be made to betray himself.

Hyperæsthesia presents many of the same difficulties of diagnosis as pain. Even when due to actual nervous disease, it is more intense when the patient's attention is fixed upon it. However, when anyone who makes assertions of extreme sensitiveness in any part does not show evidences of pain when the part is touched without his being forewarned, it is very probable that the hyperæsthesia is exaggerated or feigned, although we cannot always deny positively that it exists because the patient does not cry out when the sensitive part is disturbed. The Mannkopff symptom is not sufficiently certain in its action to permit the conclusion that pain is not caused

by pressure on alleged sensitive parts because it is not followed by an increase in the heart's action.

Ocular Symptoms.—Visual disturbance is frequently alleged by simulators, but they usually limit themselves to the feigning of general impairment of sight or of blindness in one eye. In any case in which the question arises as to the genuineness of blindness, examination by an ophthalmologist is desirable. Bilateral blindness imparts such characteristic features of attitude, movement, and facial appearance that a physician who is at all familiar with morbid ocular conditions cannot be imposed upon for any length of time. It might be possible for a simulator to imitate the deportment of a blind man were he permitted to keep his eyes closed, but this he may not do. Blind men keep their eyes open unless there is paralysis of the ocular muscles, and a claimant who kept both eyes closed would immediately arouse suspicion. Furthermore, in true blindness there are important alterations in the pupillary reflex and in the fundus of the eye. If both eyes are blind, the response of the pupils to light is impaired or lost. If one eye is blind, light thrown into it does not cause the pupil to contract; but if light is thrown into the sound eye, the pupil of the blind eye contracts through the agency of the consensual reflex. The ophthalmoscope renders valuable information, because total blindness rarely occurs in one or both eyes without there being some demonstrable lesion of the eye itself. If such a condition could occur, it would be accompanied by other evidences of disease. Although in locomotor ataxia there may be loss of perception of light before the optic atrophy becomes apparent, examination shows a loss of knee jerk, or ataxia, or other signs of tabes.

Atropine or allied drugs are sometimes used by impostors to cause a dilatation of the pupil or a loss of the pupillary reflexes. Applied either locally or taken internally atropine may

cause mydriasis, with immobility of the pupil to all stimuli, without inducing any constitutional symptoms. From the paralysis of accommodation, vision for near objects is lost. The patient cannot read, and is uncertain in gait. The pupils are dilated and do not change their size under the influence of light. By the dilatation of the pupil, however, is furnished an opportunity for the more thorough examination of the fundus of the eye. Bilateral mydriasis and pupillary immobility, if not artificially induced, are almost always accompanied with significant changes in the fundus, discoverable by the ophthalmoscope, or by paralysis of the oculomotorial nerves, or by evidences of constitutional conditions.

The feigning of monocular blindness may be discovered in a variety of ways. If, with both eyes open, a prism is placed before the sound eye so as to cause diplopia, and the patient admits seeing two objects, it is evident that he sees one of them with the eye alleged to be blind.

This test may be rendered more delicate by bringing the edge of the prism over only one-half of the pupil of the sound eye while the other eye is closed. The patient at once admits seeing double. Then if, after uncovering the alleged blind eye, the prism is held directly in front of the sound eye, the patient admits diplopia, it is certain proof that he sees with the eye alleged to be blind. Again, when in a normal person one eye is closed, and a pencil is held two or three inches from the printed page, a part of the field of vision is obscured so that he cannot read a line of the print continuously, although with both eyes open it can be read perfectly well; consequently, if a pencil or a similar object be held before the sound eye, and the patient reads the lines continuously, it may at once be inferred that he sees with the eye alleged to be blind. Or again, if, while the patient is reading with both eyes open, a strong convex lens be placed before the sound eye so that vision with it

becomes impossible, sight may be proved to exist in the alleged blind eye by the person under examination continuing his reading.

There are several tests with colors. Thus, if a patient with both eyes open, and with a red glass over the sound eye, can read a line of letters made with a red pencil on a white ground, he does so with the alleged blind eye, since solid red, without individualization of letters, is all that is seen with the sound eye. Similarly, with both eyes open, a spectacle frame containing red glass on one side and green glass on the other is placed before the patient's eyes. He is then asked to read the test cards on which are alternate red and green letters, large at one end and growing small at the other, on a black ground. A patient who has binocular vision can read these letters continuously, but a patient blind in one eye would miss every other letter, the eye which is covered with a red glass seeing only the red letters, the green letters being neutralized by the red glass, and *vice versa.*

There are a number of optical arrangements which are resorted to for the detection of simulation. The stereoscope may prove of service. Also the *boîte de flées*, by which test the subject looks with both eyes into a box containing an optical contrivance by means of which the object that is apparently seen by the right eye is in reality seen by the left one, and *vice versa;* so that a simulator may admit seeing an object which appears to him on the same side as his sound eye, but which is in reality seen by the eye said to be blind. If a clever simulator makes a study of any of these tests he may become sufficiently expert to avoid being caught by them.

As has repeatedly been emphasized the sight is in reality not lost in hysterical blindness, and the hysterical patient may be regarded as a simulator because he does not conduct himself during the above tests as a person would who was really blind.

The question arises, How is simulated blindness to be distinguished from hysterical blindness? It may often happen that such a diagnosis presents many difficulties, and depends for its solution upon associated symptoms rather than upon any local conditions. Interferences of vision of which the patient is himself conscious are among the more serious incidents of hysteria, and are usually accompanied with other pronounced hysterical manifestations. If in any case of blindness which is not organic in origin there are associated and well-marked stigmata of hysteria, we may infer that the blindness also depends upon actual impairment of vision and is not fraudulent; for to prove fraud we would have to prove that the general symptoms were also feigned, and to successfully feign the complicated clinical picture of hysteria may be regarded as impossible.

Limitation of the visual field is an ocular sign of great diagnostic value in hysteria, and it may properly be regarded as objective. It is typical of hysteria, and probably, in this country at least, is never feigned. There has been much discussion among European neurologists and ophthalmologists as to the possibility of simulating this symptom. It seems possible that an old hospital patient who had assisted at many perimetric examinations, or a physician who had studied visual disturbances, might feign limitation of the visual fields. But the perimeter is not generally popular in this country, and even among neurologists is infrequently used. It is consequently improbable that any simulator would take the trouble to learn enough about this symptom to counterfeit it with any degree of success. The characteristics of hysterical contraction of the visual fields have already been described, and need not be repeated here. If simulation of it were attempted, there would probably result irregularity and inconsistencies which would

immediately arouse suspicion that the condition was not genuine. Knapp suggests, if the case is in any way doubtful, the use of perimeters of different diameters, claiming that if by different instruments the fields remain the same the contraction is not feigned.

If the patient complains that his eyesight is impaired, but not lost, it is more difficult to determine how much truth there is in these statements. If examination of the eye, as well as of vision, shows no abnormities, the decision as to whether vision really is impaired, and, if so, how much, must be guided by the other aspects of the individual case.

The truth can usually be arrived at by observing the behavior of the patient during protracted examination, in which the various tests for acuity and extent of vision are repeatedly employed.

Hearing.—Deafness is rarely feigned as one of the various nervous symptoms which follow accidents. Hysterical deafness usually occurs in the form of impairment of hearing. If the patient declares he is absolutely deaf, and there are no evidences of any structural disturbance in the auditory apparatus, the deafness is either functional or feigned. If feigned, its character may usually be disclosed by resorting to the various tests which depend upon confusing the simulator or taking him off his guard. Knapp proposes the following modification of Coggins's stethoscope test: A binaural stethoscope, with tubes several feet long, is placed in the ears of the person to be examined. The tubes should be so arranged that either of them may be closed at the will of the examiner without the knowledge of the subject. Thus the sound may be conducted, as the examiner pleases, to either the normal or the alleged deaf ear, which may lead the simulator to make contradictory statements.

Convulsive Attacks.—The convulsive attacks of hysteria are not frequent when the disorder results from traumatic

causes, and when they occur they are accompanied with pronounced permanent stigmata, and can easily be recognized at their true value. The feigning of epilepsy is a frequent device among criminals, and in a few cases epilepsy has been proved to be simulated in actions for personal injuries. There are several characteristics about epilepsy which make it very difficult to simulate in accident cases. In the first place, epileptic fits only occasionally occur in public. Although from 10 to 15 cases of epilepsy are treated daily at the Vanderbilt Clinic, an epileptic fit rarely takes place there, and we are obliged to treat many cases of epilepsy in which there is no proof, except the statement of the patient or his friends, that epilepsy exists. With an interned population of nearly 1,000 at Craig Colony, epileptic seizures are rarely seen in the grounds. Unusual surroundings and excitement also tend to suppress rather than bring on the attacks. The epileptic paroxysm follows a regular cycle. The patient falls, irrespective of any danger of hurting himself; the initial muscular contractions are intense; the face is blue; the tongue is bitten; the breathing is abdominal; the pupils do not react to light; the urine is passed involuntarily; the knee jerks are lost for some time after the convulsion is over.

To feign epilepsy, the simulator is forced to have his attacks when he can be observed. That fits can be shammed so skillfully that they might pass for true epilepsy if made before a witness who was not a physician is unquestionable. In such cases it is impossible to tell whether the person really has epilepsy, without having him observed by some one familiar with the disease; then the fraud can usually be discovered. When the simulator falls he usually falls in such a way that he is in no danger of being hurt. He takes good care that his tongue is not bitten; there are no changes in the reflexes or in the pupils; the muscular contractions are exaggerated and theatri-

cal; the tonic stage is usually very short; and in the clonic stage the movements have a more varied and unnatural character than in epilepsy.

Vascular Disturbances.—Voluntary control of the action of the heart is possessed by so few persons, and always in so slight a degree, that the simulation of tachycardia merits no particular attention. It should be remembered, however, that rapid and tumultuous heart action is frequently observed in persons who have no serious trouble with the heart, but results from various emotional and toxic causes. So many exciting influences attend the physical examination of the chest, especially in women, that considerable care and repeated examinations are necessary before any serious significance can be attributed to tachycardia when it is unassociated with other symptoms of vascular disturbance. In simulation, fear of detection may cause the heart to beat much more rapidly; thus, at the end of the last examination, Fannie Freeman's pulse rose to 132 to the minute, although at the beginning it was only 104.

In all cases in which there is any tendency to rapid or tumultuous heart action the examination should be repeated after the patient's first excitement has passed away, or after rest in the recumbent posture has permitted a reëstablishment of the normal cardiac rhythm.

The other vascular symptoms, such as flushings, coldness and blueness of the extremities, cannot be counterfeited in a way to deceive an examiner who is on the alert for deception.

Paralysis of the Sphincters.—A person who has lost voluntary control of the bladder or rectum is in so miserable a condition that a superficial examination is usually adequate to reveal the genuineness of the symptoms. Paralysis of the bladder results in retention or incontinence of urine. In retention,

the urine collects until it is drawn off by a catheter, or until the filling of the bladder causes a relaxation of the vesical sphincter, so that the urine overflows either continuously by drops, or in larger quantities at short intervals (overflow incontinence). In incontinence, the lips of the meatus are constantly wet from the continuous dribbling of the urine. Both conditions are liable to be quickly complicated by excoriations on the scrotum and sides of the legs, and in both there is almost unexceptionally a strong urinary odor about the bedding and about the patient. These secondary results are usually absent in simulation, although, of course, the simulator may produce them if he will.

If retention is alleged, it may be impossible to prove it genuine unless the patient can be watched for twenty-four hours, in order that it may be observed whether or not he passes water involuntarily and exhibits the physical signs of a distended bladder. Such observation is often impossible. Without it, the absence of the ordinary secondary effects of urinary incontinence, the absence of distention of the bladder as shown by percussion above the pubes, and the incongruity of the associated symptoms with the alleged vesical condition, are usually sufficient for diagnosis.

False incontinence is more easily detected than false retention. In the absence of an almost constant moistening of the meatus, and without the excoriations and urinary smell which are the unavoidable consequences of the incontinence of urine, the existence of that condition can be positively denied.

Incontinence of fæces is easily recognized. In addition to the fæcal odor and frequent soiling of the linen, the anal orifice is large; and when the examiner inserts the finger in the rectum there is a total absence of the normal muscular contraction of the sphincter.

Reflexes.—The reflexes are beyond voluntary control. The skin reflexes cannot even be modified. In cutaneous areas which are insensitive to touch or pain through organic disease or through hysterical ideas the superficial reflexes are lost. It is this latter fact which reacts to the simulator's disadvantage, for if they are not lost in places alleged to be anæsthetic, it should excite suspicion of shamming. The ciliospinal reflex and the tendon reflexes, on the contrary, continue to act, unless their spinal centers or their conducting paths have been destroyed. The tendon reflexes often appear increased in suspicious cases. The tap on the patellar tendon or on the wrist may be followed by a contraction which is exaggerated beyond the usual limits. If such exaggeration is not genuine, it usually is at once apparent by the momentary pause between the application of the stimulus and the resulting contraction. A person who is feigning exaggeration of the tendon reflexes may be discovered at once by testing them when his eyes are covered. Deprived of vision, he can no longer tell when to expect the tap, and the pause, which may have been only momentary when his eyes were open, becomes so apparent, and the contraction is so evidently unnatural, that there is no difficulty in saying that the increase is feigned. It may sometimes happen that the tap causes the natural reflex at once, and that after a few moments, when the brain has had time to send its message, a second contraction occurs, which is manifestly voluntary. Foot clonus cannot be imitated in a way to deceive. It must be remembered that in some persons the knee jerks are naturally very active, and the elbow and wrist jerks are present; such an increase is always bilateral and is of little significance.

Like the superficial reflexes, the tendon reflexes cannot be decreased. In testing for the knee jerk, if it fails to respond it will be found that the knee is held immobile by the muscles.

If such is the case, the flexor tendons of the leg will be felt firm and hard, preventing any extension of the leg. The Jendrássik mode of reënforcement may overcome this. If it does not, it must be said that the patient does not permit the knee jerks to be tested, but not that they are lost.

BIBLIOGRAPHY

Methods of Examination, Etc.

Ehrnzooth, "Zur Kenntniss der Bedeutung des Traumas als aetiologisches Moment der Entstehung infectiöser Cerebralerkrankungen." *Deut. Zeit. für Nervenh.*, 1901, Bd. xx.

Hitchcock, quoted by Hall, "Adolescence." New York, 1904.

Janeway, "The Clinical Study of Blood Pressure." New York, 1904.

Peterson, "The Stigmata of Degeneration." *State Hospitals Bulletin*, 1896.

Saenger, "Die Beurtheilung der Nervenkrankheiten nach Unfall." Stuttgart, 1896.

Thayer, "On the Late Effects of Typhoid Fever on the Heart and Vessels." *Amer. Jour. Med. Sci.*, 1904, vol. cxxvii, p. 391.

Injuries to the Brain

Abel, "A Case of Puncture at the Base of the Brain," etc. *Brit. Med. Jour.*, 1895, vol. i.

Anger, "Mal. Chirurg." Paris, 1886.

Bailey, "Fracture at the Base of the Skull." *N. Y. Med. News*, 1903.

Bergmann, V., "Handbuch der praktischen Chirurgie." Stuttgart, 1899.

Bollinger, "Traumatische spät Apoplexie." Berlin, 1891.

Bullard, "The Permanent or Later Results of Fracture of the Skull." *Bost. Med. and Surg. Jour.*, 1897, vol. cxxxvi.

Courtney, "Psychic Epilepsy, with the Report of a Case." *The Med. News*, 1901.

Cramer, "Patholog. Anat. Befund in einem acuten Fall der Paranoia Gruppe." *Arch. für Psych.*, Bd. xxix.

Cushing, "The Blood-Pressure Reaction of Acute Cerebral Compression." *Amer. Jour. med. Sci.*, 1903, vol. cxxv.

Daiher, "Ueber Hirnabscess." Strassburg, 1900.

Donath, *Pester med. Presse*, 1902.

English, "The After-Effects of Head Injuries." *Lancet*, 1904, Feb. 20 *et seq.*

Friedmann, "Ueber eine besondere schwere Form von Folgezustand nach Gehirnerschütterung," etc. *Arch. für Psych.*, 1891, Bd. xxiii.

Frost, "The Final Chapter in the History of an Extensive Injury to the Head." *Amer. Jour. of Insanity*, 1903, vol. lix.

Gowers, "Epilepsy." London, 1903.

Gray, "Epilepsy" in "Text-book of Nervous Diseases by American Authors." Philadelphia, 1895.

Guder, "Die Geisterstörungen nach Kopfverletzungen," etc. Jena, 1886.

Heer, "Ueber Schädelbasisbrüche." *Beit. j. kl. Chirurg.*, 1892, Bd. ix.

Higgins and Ogden, "Traumatic Glycosuria." *Bost. Med. and Surg. Jour.*, 1895, vol. cxxxii.

Kaplan, "Kopftrauma und Psychosen." *Allg. Zeit. für Psych.*, Bd. lvi.

Knotz, "Seh- und Hörstörungen, sowie über Augenmuskellähmungen nach Schädelverletzungen." *Wien. med. Presse*, 1900, 30, 31, 35.

Laplace, "The Surgical Treatment of Insanity." *Med. and Surg. Rep.*, 1896.

Lloyd and Deaver, "A Case of Focal Epilepsy Successfully Treated," etc. *Amer. Jour. Med. Sci.*, 1888.

Macewen, "Pyogenic Infective Diseases of the Brain and Spinal Cord." Glascow, 1893.

Meyer, "Traumatic Insanity." *Amer. Jour. of Insanity*, 1904, vol. lx, 3.

Näcke, "Ein Dämmerzustand mit Amnesie," etc. *Neurolog. Cblatt.*, 1897.

Oppenheim, "Die Encephalitis und der Hirnabscess." Wien, 1897.

Phelps, "Traumatic Injuries of the Brain." New York, 1897.

Reverdin et Vallette, "Absces traumatique du lobe occipital droit." *Revue Med. de la Suisse Rom.*, 1902.

Roncali, "Absces traumatique du lobe temporo sphen." Chipault's Travaux, 4ème année.

Siemerling, "Traumatische Epilepsie." Tübingen, 1895.

Starr, "Brain Surgery." New York, 1893.

Stierlin, "Schädelbasisfractur mit Lähmungen im Gebiete des X. und XII. Hirnnerven." *Arch. für kl. Chirurg.*, 1900, vol. lxi.

Van Gieson, "A Contribution to the Pathology of Traumatic Epilepsy." *N. Y. Med. Rec.*, 1893.

Van Nes, "Ueber Schädelbasisbrüche." *Deut. Zeit. für Chirurg.*, Bd. liv.

Wagner, "Ueber Trauma, Epilepsie und Geisterstörungen." *Jahrb. für Psych.*, 1888-89, Bd. viii.

Werner, "Ueber die Geisteskrankheiten nach Kopfverletzungen." *Vierteljahrs. für gericht. Med.*, 1902, xxiii.

Wildermuth, "Die epileptische Geistesstörungen," etc. *Allg. Zeit. für Psych.*, vol. lii, p. 1087.

Injuries to the Spinal Cord

Bailey, "Primary Focal Hæmatomyelia." *N. Y. Med. Rec.*, Nov. 19, 1898.

Bailey, "Traumatic Hemorrhages into the Spinal Cord." *N. Y. Med. Rec.*, April 7, 1900.

Barling. *Birmingham Med. Review*, 1893.

Bikeles, ref. in *Schmidt's Jahrb.*, 1895, Bd. ccxlviii.

Bowlby, "The Reflexes in Cases of Injury to the Spinal Cord." *Lancet*, 1890, vol. i, p. 1071.

Burrell, "Fracture of the Spine." *Bost. Med. and Surg. Jour.*, vol. cxvii.

Chipault, quoted by Starr, "Organic Nervous Diseases." New York, 1904.

Déjérine, "Hæmatomyélie compliquant une section médullaire." *Arch. de Neurologie*, vol. viii, No. 44.

Fisher, quoted by Gowers.

Gowers, "Diseases of the Nervous System." London, 1899.

Gurlt, "Handbuch der Lehre von den Knochenbrüchen." Hamm, 1862.

Kümmel, "Ueber die traum. Erkrankungen der Wirbelsaüle." *Deut. med. Woch.*, No. 11, 1895.

Leyden, "Klinik der Rückenmarkskrankheiten." Berlin, 1874.

Müller, "Untersuchungen über die Anatomie und Pathologie des untersten Rückenmarksabschnittes." *Deut. Zeit. für Nervenh.*, 1898, Bd. xiv.

Parkin, "Cases of Hæmatomyelia." *Guy's Hosp. Reps.*, 1891, xlvii.

Prewitt, "Gunshot Injuries of the Spine." *Annals of Surg.*, 1898, vol. xxviii.

Roeseler, "Die Stichverletzungen des Rückenmarks," etc. *Friedreich's Bl. f. gericht. Med.*, 1901.

Schmaus, "Path. Anat. des Rückenmarks." Wiesbaden, 1901.

Schmaus, "Beit. zur path. Anat. der Rückenmarkserschütterungen." *Virchow's Archiv*, 1890, Bd. cii.

Seiffer, "Das spinale Sensibilitatsschema zur Segmentdiagnose der Rückenmarkskrankheiten." *Arch. f. Psych.*, 1901, vol. xxxiv, p. 648.

Stewart, "A Case of Severed Spinal Cord, in which Myelorrhaphy was followed by Partial Return of Function." *Phila. Med. Jour.*, June 7, 1902.

Stolper, "Ueber traum. Blutungen um und in das Rückenmark." *Monatsch. für Unfallheilkunde*, Feb. 15, 1898.

Thorburn, "A Contribution to the Surgery of the Spinal Cord." Philadelphia, 1889.

Wagner and Stolper, "Verletzungen der Wirbelsäule und des Rückenmarks." Stuttgart, 1898.

Watson, "An Experimental Study of Lesions arising from Severe Concussions." Philadelphia, 1890.

Weisenburg, "A Clinical Report of Three Cases of Injury to the Lower Spinal Cord and Cauda Equina." *Amer. Jour. Med. Sci.*, May, 1904.

Westphal, "Ueber einen Fall von traum. Myelitis." *Arch. für Psych.*, 1896, Bd. xxvii.

Whitman, "A Treatise on Orthopædic Surgery." Philadelphia, 1901.

Willard and Spiller, "Concussion of the Spinal Cord." *N. Y. Med. Jour.*, March 6, 1897.

Vibert, "Étude medico-legale sur les blessures." Paris, 1888.

Injuries to Nerves

Bailey, "Operative Paralysis of the Spinal Accessory Nerve." *Annals of Surg.*, 1901.

Ballance and Stewart, "The Healing of Nerves." London, 1891.

Bruns, ref. in *Neurolog. Cblatt.*, 1902.

Clark, "The Surgical Treatment of Chronic Facial Palsy." *Proceedings N. Y. Neurolog. Soc.*, March 3, 1905. Also, "Brachial Birth Palsy." *Proceedings N. Y. Neurolog. Soc.*, April 6, 1904.

Hartmann, "Zwei bemerkenswerthe Fälle von Erkrankung der Nerven aus dem Plexus Sacrolumbalis." *Jahrb. für Psych. und Neurol.*, 1900.

Hector, "Die Erfolge der Nervennaht," etc. *Inaug.-Diss.*, Berlin, 1901.

Hirsch, "Traumatic Injury of the Pneumogastric Nerve," etc. *N. Y. Med. Jour.*, 1897, vol. lxvi.

Hoedemaker, "Ueber die von Erb zuerst beschrieben combinirte Lähmungsform an der oberen Extremität." *Arch. für Psych.*, 1879, ix.

Klumpke, "Contribution à l'étude des paralysies radiculaires du plexus brachial." *Revue de Méd.*, 1885, No. 7.

Litten, "Ueber die Normaliter bei jeder Respiration am Thorax sichtbaren Zwerchfellbewegungen." *Deut. med. Woch.*, 1893, No. 13.

Quain, "The Nerves." London, 1895.

Traumann, "Schussverletzung des Vagus," etc.; "Stichverletzung des Hypoglossus," etc. *Deut. Zeit. für Chirurg.*, 1893, Bd. xxxvii.

Wallenberg, "Stichverletzung des dritten dorsal Nerven am Ganglion Spinale." *Neurolog. Cblatt.*, 1901. Also, "Chronic Degenerative Diseases."

Trauma in Chronic Diseases

Acromegaly

Unverricht, "Akromegalie und Trauma." *Münch. med. Woch.*, 1895, Bd. xlii.

Syringomyelia

Laehr, "Beiträge zur forensischen Bedeutung der Syringomyelia." *Charité-Annalen*, 1895, xx.

Huisman, "Ein Fall von Syringomyelie nach Trauma." *Deut. med. Woch.*, 1897.

General Paresis

Bechholm, "Maladies mentales d'origine traumatique." *Thèse de Paris*, 1896.

Bannister, *Am. Jour. of Insanity*, 1893–94, p. 477.

Christian, "Des traumatismes du crâne dans leurs rapports avec l'alienation mentale." *Arch. de Neurol.*, Juillet et Sept., 1889.

Fournier, *Gaz. méd. de Paris*, Nov. 3, 1894.

Fox, "Unusual Cases of General Paresis." *Jour. Ment. Science*, 1891, p. 393.

Gudden, "Zur Aetiologie u. Sympt. der prog. Paralysie mit besondere Berück. des Trauma," etc. *Arch. f. Psych.*, Feb., 1894.

Hamilton, "System of Legal Medicine." New York, 1894.

Hirschl, "Die Aetiologie der prog. Paralysie." Abs. *Neurol. Cblatt.*, 1896, p. 369.

Jacobson, "Traumatische Psychosen." *Nord. med. Ark.*, 1893, iii, 13, p. 1.

Kaplan, "Kopftrauma und Psychosen." *Allg. Zeit. für Psych.*, Bd. lvi.

Mickle, "General Paralysis of the Insane." London, 1886. Also, "General Paralysis from Cranial Injury." *Jour. Ment. Sci.*, Jan., 1883, p. 545.

Oebeke, "Zur Aetiologie der allgemeinen fortsch. Paralysie." *Zeit. für Psych.*, 1891, xlix, p. 1.

Peterson, "Relation of Syphilis to General Paresis." *N. Y. Med. Rec.*, Dec. 9, 1893.

Régis, *Manual of Mental Med.*, Syracuse, 1894. (Trans.)

Schläger, quoted by Gudden, *op. cit.*

Locomotor Ataxia

Bernhardt, "Zur Lehre von der traum. Tabes." *Monats. für Unfallheilkunde*, 1895, vol. ii, p. 193.

Craig, "Case of Tabes from Injury." *Lancet*, 1895, vol. i, No. 8.

Erb, "Syphilis und Tabes." *Berl. klin. Woch.*, 1896, No. 11.

Hitzig, "Ueber traum. Tabes u. d. Pathogenese der Tabes im Allgemeinen." Berlin, 1894.

Klemperer, "Ueber traum. Tabes." *Zeit. für kl. Med.*, 1890.

Lammers, "Ein Fall traum. Tabes." *Cblatt. für innere Med.*, 1897.

Prince, "Traumatism as a Cause of Locomotor Ataxia." *Jour. Nerv. and Ment. Dis.*, Feb., 1895.

Storbeck, "Tabes Dorsalis und Syphilis." *Inaug.-Diss.*, Berlin, 1895.

Soukanoff, "Contribution à l'étude des changements du systema nerveux dans la polynévrite." *Arch. de Neurol.*, p. 177.

Progressive Muscular Atrophy

Ballet and Dutil, *Revue de Médicine*, Jan., 1884.

Bullard, "A Case of Progressive Muscular Atrophy following a Blow on the Head." *Bost. Med. and Surg. Jour.*, 1885, p. 369.

Charcot, J. B., "Contribution à l'étude de l'atrophie musc. prog." Paris, 1895.

Clarke and Jackson, "On a Case of Muscular Atrophy, with Disease of the Spinal Cord and Medulla Oblongata." *Medico-Chirurg. Trans.*, 1867, vol. l, p. 489.

Eisenlohr, "Klinische und anatomische Beiträge zur progressive Bulbärparalysie." *Zeit. für klin. Med.*, 1880, vol. i, No. 3, p. 435.

Hirsch, "Amyotrophic Lateral Sclerosis following Old Poiomyelitis." *Med. News*, 1895, vol. lxvii, p. 500.

Mann, "A Case of Traumatic Progressive Muscular Atrophy of Long Duration, complicated by an Attack of Left Hemiplegia, due to Embolism." *Alienist and Neurologist*, 1886, vol. vii, p. 430.

Raymond, "Atrophies musculaires." Paris, 1889, p. 138.

Zielen, "Aerztliches Gutachten über einen forensischen Fall von progressiven Muskelatrophie." *Vierteljahrs. für gericht. Med.*, 1894, vol. viii, p. 286.

Paralysis Agitans

Charcot, "Œuvres complètes." Paris, 1891, i, p. 155.

Vandier, "De la paralysie agitante de cause traumatique." *Thèse de Paris*, 1886.

Walz, "Die Traumatische Paralysis Agitans." *Vierteljahrs. für gericht. Med.*, 1896, xii, p. 323.

Gowers, "Diseases of the Nervous System." London, 1893.

Multiple Sclerosis

Jelliffe, "Multiple Sclerosis: Its Occurrence and Ætiology." *Jour. Nerv. and Mental Dis.*, July, 1904.

Tumors

Carara, "Ein mit Exitus letalis nach Kopfverletzung beendeter Fall von Hirn tumor." *Vierteljahrs. für gericht. Med.*, 1896, Bd. xi.

Loewenthal, "Ueber die traumatische Entstehung der Geschwülste." *Arch. für kl. Chirurg.*, Bd. xlix.

Syphilis

Nonne, "Syphilis und Nervensystem." Berlin, 1902.

Saenger, "Die Beurtheilung der Nervenerkrankungen nach Unfall." Stuttgart, 1896.

Diabetes

Ebstein, "Traumatische Neurose und Diabetes Mellitus." *Arch. für kl. Med.*, 1895, Bd. liv.

Naunyn, "Diabetes Mellitus," in Nothnagel's "System of Medicine."

Reibal, *Gazette Méd. de Strasburg*, 1877, xxxvi, 86, 91.

Sternberg, "Die traum. Entstehung innerer Krankheiten." Jena, 1900.

History of the Traumatic Neuroses, Etc.

Abercrombie, "Treatise on the Brain and Spinal Cord." 1828.

Angell, "Railway Spine." *Medical News*, 1905, vol. lxxxvi.

Bezald and Dainilewsky. See Werner.

Booth, Critical Digests in *Sajous's Ann. of the Univ. Med. Sci.*, 1892, 1893, 1894.

Bruns, Critical Digests in *Schmidt's Jahrbücher*, ccxx, p. 142; ccxxi, p. 210; ccxxx, p. 81; ccxxxi, p. 21; ccxxxiv, p. 25; ccxxxviii, p. 73; ccxlii, p. 191; cclv, 8.

Clevenger, "Spinal Concussion." Philadelphia and London, 1889.

Conty and Charpentier. See Werner, *Vierteljahrs. für gericht. Med.*, xxiii, 1902.

Crocq, fils, "Étude pathogénique et clinique des névroses traumatiques." Bruxelles, 1896.

Dana, "Concussion of the Spine and its Relation to Neurasthenia and Hysteria," *N. Y. Med. Rec.*, Dec. 6, 1884; *ibid.*, article, "Traumatic Neuroses," in Hamilton's "System of Legal Medicine," New York, 1894.

De la Tourette, "Traité clinique et therapeutique de l'hystérie." Paris, 1891 and 1895.

"Discussion über Nerven-Unfalls Erkrank." Aerzt. Verein zu Hamburg. *Neurolog. Cblatt.*, 1896, pp. 569 and 617.

Eisenlohr, "Bemerkungen über die traum. Neurose." *Berl. kl. Woch.*, 1889, No. 52.

Erb, Ziemssen's "Encyclopædia of the Practice of Medicine," New York, vol. xiii, 1878.

Erichsen, "On Railway and Other Injuries of the Nervous System," London, 1866. Also, "Spinal Concussion," London, 1875 and 1882.

Förster, "Gesichtsfeldmessung bei Anæsthesiæ Retinæ." *Kl. Monatsbl. für Augenheilk., Beilage*, 1877.

Graefe, von, "Anæsthesiæ Retinæ mit concentr. Gesichtsfeldbeschränkung." *Kl. Monatsbl. für Augenheilk.*, 1865, iii.

Hamilton, "Railway and Other Accidents." New York, 1904.

Hodges, "Concussion of the Spine, So-called." *Bost. Med. and Surg. Jour.*, 1880, vol. cii.

Hughes, "Cyclone Neuroses and Psychoses." *Alienist and Neurol.*, Jan., 1897.

Knapp, "Accidents from the Electric Current," *Bost. Med. and Surg. Jour.*, April, 1890, pp. 17 and 24; *ibid.*, "Traumatic Nervous Affections, an Attempt at their Classification," etc., *Am. Jour. of the Med. Sci.*, 1892, p. 629; *ibid.*, "Nervous Affections following Railway and Allied Injuries." "Text-Book on Nervous Diseases by American Authors," Philadelphia, 1895.

Koenig, *Deut. Zeit. für Nervenh.*, 1895, vol. vii, p. 263.

Mannkopf. See Rumpf.

Müller, "Zur Frage der Ermüdbarkeit des Gesichtsfeldes." *Arch. für Psych.*, 1896, p. 225.

Nammack, "Case of Traumatic Neurasthenia." *Trans. N. Y. Neurolog. Soc.*, 1895.

Ollivier, "Traité des mal. de la moelle épinière." Paris, 1837.

Oppenheim, "Weitere Mittheilungen über die sich am Kopfverletzungen und Erschütterungen anschliessenden Erkrankungen des Nervensystems." *Arch. für Psych.*, 1885.

Oppenheim, "Die traumatische Neurosen. Berlin, 1889 and 1892.

Outten, "Railway Injuries," in Witthaus and Becker's "Medical Jurisprudence, Forensic Medicine, and Toxicology," New York, vol. iii, 1894.

Page, "Railway Injuries in their Medico-Legal and Clinical Aspects." London, 1883 and 1891.

Peters, *Deut. Zeit. für Nervenh.*, 1894, 3 and 4.

Porcher, "Influence of the Recent Earthquake Shocks in Charleston on Health." *Med. News*, Dec. 11, 1886.

Putnam, "Recent Investigation into the Pathology of So-called Concussion of the Spine." *Bost. Med. and Surg. Jour.*, Sept. 6, 1883.

Reibal, *Gaz. Méd. de Strasburg*, 1877, xxxvi.

Riegler, "Ueber die Folgen der Eisenbahnverletzungen." Berlin, 1879.

Rumpf, "Beiträge zur kritischen Symptomatologie der traumatischen Neurosen." *Deut. med. Woch.*, 1895, p. 165.

Saenger, "Die Beurtheilung der Nervenerkrankungen nach Unfall." Stuttgart, 1896.

Schultze, "Traum. Neurose." *Neurol. Cblatt.*, 1889, p. 402; also, *Samml. kl. Vort.*, N. F., 14.

Seguin, Critical Digests in *Sajous's Ann. of the Univ. Med. Sci.*, 1889, 1890, 1891.

Squires, *N. Y. Med. Rec.*, 1887, xxxii, 190.

Strauss, "Ueber den Werth des Mannkopffschen Symptomes bei Nervenleiden nach Trauma." *Berl. kl. Woch.*, 1892, p. 1222.

Strümpell, "Die traumatische Neurosen," *Berliner Klinik*, 1888; also,

Münch. med. Woch., Dec. 3 and 10, 1895; *ibid.*, "Ueber Untersuchung Beurtheilung und Behandlung von Unfallkranken," Munich, 1896.

Thomsen and Oppenheim, "Ueber das Vorkommen und die Bedeutung der sensorischen Anaesthesie bei Erkrankungen des centralen Nervensystems." *Arch. für Psych.*, 1884.

"Verh. des XII. Internat. Med. Kongress," 1893.

Walton, "Spinal Irritation: Probable Cerebral Origin of the Symptoms." *Bost. Med. and Surg. Jour.*, Dec. 27, 1883.

Walton, "Hysterical Anæsthesia Brought on by a Fall." *Bost. Med. and Surg. Jour.*, Dec. 11, 1884.

Walton, "Contribution to the Study of the Traumatic Neuropsychoses." *Jour. Nerv. and Ment. Dis.*, 1890, p. 432.

Westphal, "Einige Fälle von Erkrankung des Nervensystems nach Verletzungen auf Eisenbahnen." *Charité-Annalen*, Berlin, Jan. 6, 1880.

Traumatic Neurasthenia

Bass, "Das Gesichtsfeld." Stuttgart, 1896.

Beard, "A Practical Treatise on Nervous Exhaustion." New York, 1880.

Binswanger, "Die Pathologie und Therapie der Neurasthenie." Jena, 1896.

Förster, "Gesichtsfeldmessung bei Anæsthesiæ Retinæ." *Kl. Monatsbl. für Augenheilk., Beilage*, 1877.

Hodge, C. F., "A Microscopical Study of the Changes due to Functional Activity in Nerve Cells." *Journal of Morphology*, 1892.

Janet, "Les obsessions et la psychasthenia." Paris, 1903.

Knapp, "Traumatic Neurasthenia," in "Nervous Diseases by American Authors." Philadelphia, 1895.

Lugaro, "Sur les modifications des cellules nerv. dans les divers états fonctionels." *Arch. Ital. de Biol.*, 1895, xxiv.

Nammack, *Trans. N. Y. Neurolog. Soc.*, 1895.

Oppenheim, "Die traumatische Neurosen." 2te Auf., Berlin, 1892.

Page, H. W., "Railway Injuries in their Medico-Legal and Clinical Aspects." London, 1887.

Richter, *Berliner Klinik*, August, 1895.

Sänger, "Die Beurtheilung der Nervenerkrankungen nach Unfall," Stuttgart, 1896; *ibid.*, *Neurolog. Cblatt.*, 1892, p. 121.

Vibert, "La Névrose traumatique." *Annales d'hygiène pub. et de méd. leg.*, 1892, vol. xxviii, p. 139.

Wildbrand and Sänger, "Ueber Sehstörungen bei functionellen Nervenleiden." Berlin.

Traumatic Hysteria

Babinski, "Contractures organiques et hystériques." *Soc. Méd. des Hôp.*, May, 1893.

Bailey, "A Case of General Analgesia." *N. Y. Med. Rec.*, Dec. 28, 1895. *Ibid.*, "The Diagnosis of Idiopathic Epilepsy." *Am. Med. Surg. Bul.*, Aug. 8, 1896.

Berbez, "Hystérie et traumatisme." *Thèse de Paris*, 1887.

Bernheim, "Hypnotisme, Suggestion, Psychothérapie," 1891.

Bremer, "A Case of Hysterical Astasia-abasia Suing for Damages." *Jour. Nerv. and Ment. Dis.*, 1893, p. 13.

Briquet, "Traité clinique et thérapeutique de l'hystérie," 1859.

Blocq, "Sur une affection caractérisée per de l'astasie-abasie," *Arch. de Neurol.*, 1888; *ibid.*, "Les troubles de la marche dans les maladies nerveuses," Paris.

Brodie, "Lectures Illustrative of Certain Local Nervous Affections." London, 1837.

Charcot, "Œuvres complètes." Paris, 1890.

Comby, J., "Apoplexie hystérique avec hemiplégie gauche survenue pour la première fois a la suite d'une fulguration." *Union méd.*, 1894, No. 60.

Dana, C. L., Hamilton's "System of Legal Medicine." New York, 1894.

Francotte, "Sourd-muettité hystérique," etc. *Mercredi médical*, 1894, No. 40.

Holden, "Ocular Symptoms of the Traumatic Neuroses." *Medical News*, 1904.

Hun, "Two Cases of Traumatic Hysteria." *Med. News*, April 11, 1891, p. 403.

Janet, "État mental des hystériques." 2 vols., Paris.

Knapp, "Traumatic Hysteria," in "Text-Book of Nervous Diseases by American Authors." Philadelphia, 1895.

König, "Weitere Mittheilungen über die functionellen Gesichtsfeldanomalien mit besonderer Berücksichtigung von Befunden an normalen Menschen." *Deut. Zeit. für Nervenh.*, 1895, vol. vii, p. 263.

Laehr, "Ueber Störungen der Schmerz und Temperaturempfindung in Folge von Erkrankungen des Rückenmarkes." *Arch. für Psych.*, 1896, iii, p. 773.

Laudun, "Ein Fall von Catalepsie." *Wien. med. Presse*, 1894, Nos. 30 and 35.

Lebrun, "Monoplégie brachiale hystéro-traumatique." *Arch. méd. Belges*, 1893, vol. i, p. 382.

Lepois, quoted by Gilles de la Tourette, *op. cit.*, i, p. 13.

Miura, "Trois cas de monoplégie brachiale hystérique." *Arch. de Neurol.*, May, 1893.

Möbius, "Ueber den Begriff der Hysterie." *Neurolog. Beiträge*, i, 1894.

Page, H. W., "Injuries of the Spine and Spinal Cord, without Apparent Mechanical Lesion and Nervous Shock in their Surgical and Medico-Legal Aspects," London, 1883; *ibid.*, "Railway Injuries in their Clinical and Medico-Legal Aspects," London, 1891.

Paget, "Clinical Lectures and Essays." Second edition, London, 1879.

Peterson, "Physiological Experiments with Magnetism at the Edison Laboratory." *N. Y. Med. Jour.*, Dec. 31, 1892.

Pitres, "Leçons cliniques sur l'hystérie et l'hypnotisme." Paris, 1891.

Prince, "Three Cases of Traumatic Hysterical Paralysis of Twenty-nine, Twenty-eight, and Twenty-nine Years' Duration Respectively, in Males," *Amer. Jour. of Med. Sci.*, 1892, p. 63; *ibid.*, "Hysterical Monocular Amblyopia," *Am. Jour. of Med. Sci.*, Feb., 1897.

Richter, "Études cliniques sur la grande hystérie," etc. Paris, 1885.

Reynolds, Russell, "Paralysis, and Other Disorders of Motion and Sensation Dependent on Idea." *Brit. Med. Jour.*, 1869, vol. ii, p. 483.

Schutte, E., "Ein Fall von Paramyoclonus Multiplex bei einem Unfallskranken." *Neurolog. Cblatt.*, 1897, No. 1.

Sérieux, "Note sur un cas de paralysie hystéro-traumatique des quatre membres." *Arch. de Neurologie*, July, 1891, p. 31.

Souques, "Étude des syndromes hystériques 'simulateurs.'" Paris, 1891.

Strümpell, "Ueber die traumatische Neurosen." *Berlin. Klin.*, 1888, Heft 3.

Sydenham, quoted by Gilles de la Tourette, *op. cit.*, i, p. 15.

Tourette, de la, Gilles, "Traité clinique et thérapeutique de l'hystérie." 3 vols., Paris, 1891, and 1895.

Zein, quoted by Gilles de la Tourette, *op. cit.*, vol. ii, p. 110.

Insanity from Psychic Shock

Binswanger, "Ueber den Schreck als Ursache psychischer Erkrankungen." *Charité-Annalen*, 1879.

Landgraf, *Friedreich's Blätter für gericht. Med.*, 1885, xxxvi, 458–465.

Manson, *Lancet*, 1846, ii, 582.

Reibal, *Gaz. méd. de Strasburg*, 1877, xxxvi, 86–91.

Unclassified Forms

Bernhardt and Kronthal, "Fall von sog. traum. Neuros. mit Sect. Bef." *Neurolog. Cblatt.*, 1890, No. 4.

Crocq., "Les névroses traumatiques." Brussels, 1896.

Dana, "The Traumatic Neuroses," in Hamilton's " System of Legal Medicine." New York, 1894.

Knapp, "Traumatic Sclerosis," in "Text-Book of Nervous Diseases by American Authors." Philadelphia, 1895.

Nonne, "Ueber pseudospastische Parese mit Tremor nach Trauma." *Neurolog. Cblatt.*, 1897, Nos. 20 and 21.

Saenger, "Die Beurtheilung der Nervenerkrankungen nach Unfall." Stuttgart, 1896.

Sperling and Kronthal, "Eine traum. Neurose mit Sect. Befund." *Neurolog. Cblatt.*, 1889, Nos. 11 and 12.

Treatment of the Traumatic Neuroses

Baruch, "Hydrotherapy," in Hare's "System of Therapeutics." Philadelphia, 1891.

Bernheim, "Suggestive Therapeutics." Translated by Herter, New York, 1889.

Forel, "Der Hypnotismus." Stuttgart, 1889.

Mitchell, "Fat and Blood." Philadelphia, 1891.

Peterson, "Vibratory Therapeutics." *Med. News*, Nov., 1897.

Wetterstrand, "Hypnotism." Translated by Peterson, New York and London, 1897.

Winternitz, "Vorträge über Hydrotherapie."

Medico-Legal Considerations

Bailey, "Simulation of Nervous Disorders Following Accidents." *The Railway Surgeon*, Feb. 8, 1896.

Brush, "The Medical Witness." *Brooklyn Med. Jour.*, June, 1903.

Dana, *op. cit.*

Godkin, "Accident Cases," in Hamilton's "System of Legal Medicine." New York, 1894.

Golibiewski, "Atlas and Epitome of Diseases Caused by Accidents." Trans. by Bailey, Philadelphia, 1900.

Heller, "Simulationen und ihre Behandlung." Leipzig, 1890.

Hoffman, *Berl. kl. Woch.*, June 21, 1891.

Judd, "Legal Aspects of Spinal Concussion." *Bost. Med. and Surg. Jour.*, 1890, vol. cxxiii, p. 219.

Knapp, "Simulation in Traumatic Nervous Diseases," *Bost. Med. and Surg. Jour.*, September 28, 1893; *ibid.*, "Feigned Diseases of the Mind and Nervous System," in Hamilton's "System of Legal Medicine," New York, 1894.

Page, *op. cit.*

Prentice, "Speculation in Damage Claims." *North Am. Rev.*, Feb., 1897.

Prince, "Traumatism as a Cause of Locomotor Ataxia." *Jour. Nerv. and Ment. Dis.*, Feb., 1895.

Schmidt-Rimpler, "Zur Simulation concentrischer Gesichtsfeldeinengung mit Berücksichtigung der traumatischen Neurosen." *Deut. med. Woch.*, 1892, p. 561.

Schultze, *Deut. med. Zeit.*, Oct. 16, 1891 ; *ibid.*, "Weiteres über Nervenerkrankungen nach Trauma," *Deut. Zeit. für Nervenh.*, 1891, p. 444.

Seeligmüller, *Deut. med. Zeit.*, Oct. 16, 1891.

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