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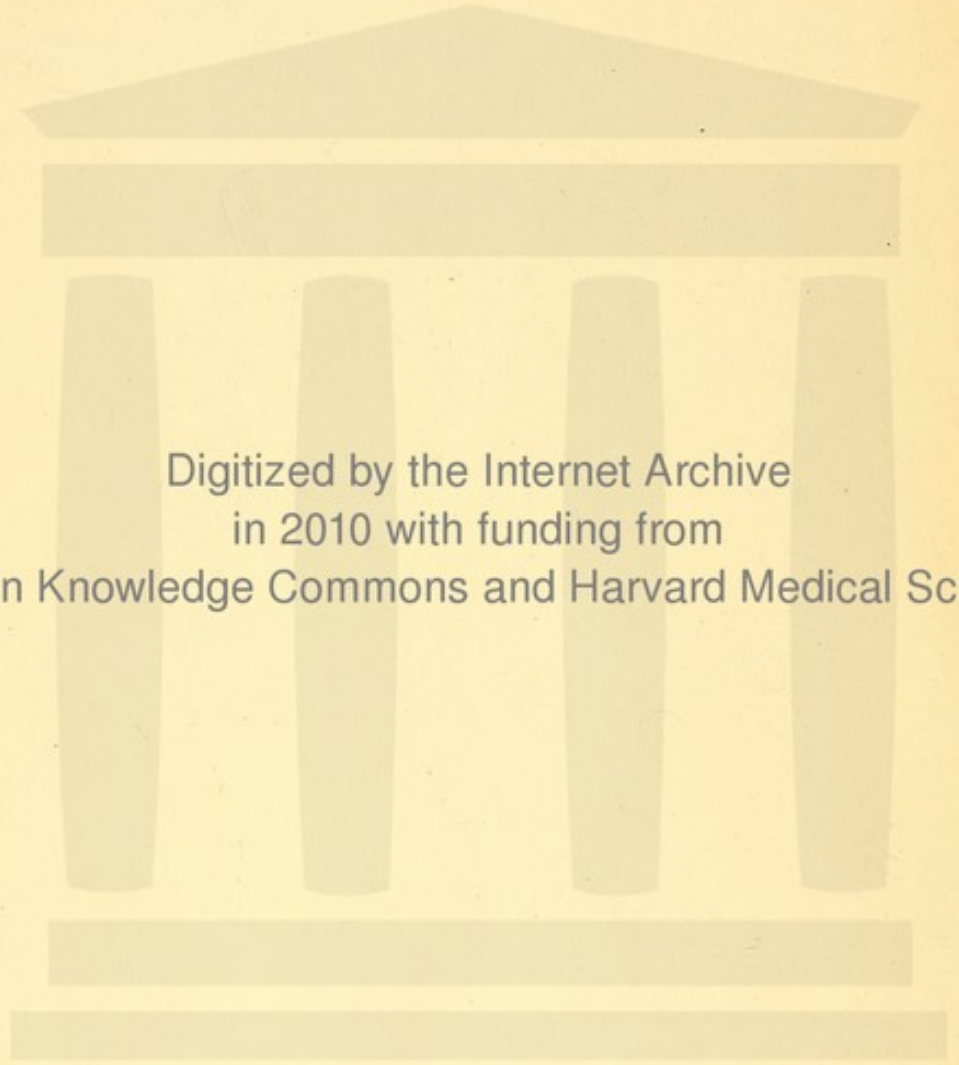
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Phillips Coombs Knapp

ACCIDENT AND INJURY

THEIR RELATIONS TO DISEASES OF THE NERVOUS SYSTEM

BY

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TO

PROFESSOR STARR,

IN GRATEFUL RECOLLECTION

OF MANY KINDNESSES.

PREFACE.

THE original purpose in this undertaking was to furnish a systematic description of the nervous affections which result from injury and fright, and which, under the generally familiar term of "the traumatic neuroses," exist independently of any as yet demonstrated lesion in the nervous system. These disorders are of sufficiently common occurrence to constitute a comprehensive chapter in internal medicine, and they have obtained a position of legal prominence from the frequency with which they are associated with questions of liability. Yet, in spite of their importance, that they are not always clearly understood by physicians generally is shown by the contradictory views expressed concerning them when they become the subjects of litigation.

While it can not be denied that the nature of the traumatic neuroses is still in many respects obscure, sufficient evidence as to their causes and symptoms has now accumulated to permit them to be recognized as definite clinical types, and to be diagnosticated with reasonable accuracy. This evidence is, however, more or less inaccessible to general readers, existing chiefly as scattered monographs, most of which are in foreign languages. No single book has attempted, in recent years, to present in detail the enlarging views, and the deductions from them, concerning these disorders. More than a year ago it appeared to the writer that an embodiment of such views and deductions, together

with facts of personal experience, might prove of service, not only to practitioners of medicine, but also to those whose interest in the questions involved arises from legal rather than from medical reasons.

This task was accordingly undertaken. As it progressed it became evident that a treatise on the traumatic neuroses would be much more useful were it preceded by some account of injuries to the nervous system of organic character. A disease can only be diagnosticated as functional when evidences of organic lesion are absent, and the physician to recognize the one must be able to exclude the other. Accordingly, instead of proceeding at once to the delineation of neurasthenia, hysteria, and allied conditions, the first parts of the volume are concerned with the simpler methods of examination, with the causes and effects of acute organic injuries to the nervous system, and with a consideration of in how far accidents may be held responsible for the appearance of certain chronic degenerative diseases. The question of malingering is so intimately connected with the traumatic functional nervous disorders that its description also was deemed essential. Treatment does not properly fall within the scope of the work as indicated by the title, but since it is a subject of such importance the few words regarding it may be found not out of place.

In its present form the book might be mistaken for a general treatise on traumatic diseases of the nervous system. So comprehensive a purpose it can not aspire to fulfil. It is merely an attempt to present the views which seem most tenable as to the part played by injury and shock in the more important of these diseases. The writer has drawn freely upon the works of Erichsen, Page, Charcot, Oppenheim, Knapp, Dana, Gilles de la Tourette, Strümpell, Heller, and many others, for which he desires to express his obligations. His warmest thanks are due to Professor Starr

for kindly suggestions given during the preparation of the manuscript, and for his permission to make use of the rich material of the Department for Nervous Diseases at the Vanderbilt Clinic; to Dr. V. H. Norrie for many of the photographs; and to Mr. I. Strauss for help in correcting the proofs and in many other ways.

PEARCE BAILEY.

60 WEST FIFTIETH STREET, NEW YORK,
October 20, 1897.

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ACCIDENT AND INJURY.

INTRODUCTION.

THE CONSIDERATION OF THE CASE.

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 morbid conditions that have been for a long time active. Yet, as every causal factor adds its special *câchet* 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 traumatisms upon 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 struc-

ture, 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 functional traumatic nervous disorders have received careful observation and description by neurologists; but the behavior of the nervous system under injuries which directly affect nervous structure has been too little subjected to special investigation. Most acutely occurring injuries to the brain and spinal cord are received in surgical wards, where the exact character of nervous symptoms and the probable location of lesions only occasionally become the objects of close study. Yet organic injuries to the nervous system constitute a field of investigation full of fruitful promise. There are constantly occurring in all large cities selective injuries to nervous tissue, which result from all varieties of causes and which occur under all possible conditions. Were they systematically examined at the bedside, and were the clinical picture, when possible, amplified by a microscopic study of the pathological conditions to which the symptoms had been due, these injuries could be made to subserve the purposes of experimental pathology, and could be relied upon to furnish more valuable results than have been obtained by experiments on animals.

It is from the observations of morbid conditions as they occur in man that we must look for the answers to such questions as concussion of the spinal cord, or the localization of special functions, or the understanding of the factors of predisposition to nervous disease, or the indications for

operative treatment. That the subject has been, and still is, treated with less attention than it deserves, there is, unfortunately, no question of doubt. Injuries to the spinal cord are as old as the art of medicine itself; yet it is only within the last decade that there has arisen any definite knowledge as to the localization of function of the spinal-cord segments. Such knowledge as we now possess is largely due to the study of traumatic 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 can often diagnosticate a fracture of the spine from the seat of the ambulance, 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 and rarest skill 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 fracture of the skull 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 traumatism 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.

A *functional*, as opposed to an *organic* disease of the nervous system, is one in which the anatomical integrity of nervous structure remains unimpaired. There may be serious or even alarming symptoms, yet the evidences which indicate destruction of special centers or conducting paths are regularly absent; also, the course of the disease is benign, tending to recovery. Although there is no reason to doubt that every departure from health is a result of changes of some character in physical structure or composition, the morbid alterations in nervous tissue which underlie the functional nervous diseases have hitherto escaped identification, and until they are identified these affections must be classed as depending upon no known pathological conditions. They can only be diagnosticated as functional, however, when careful examination has demonstrated that there is absence of those symptoms which are the infallible signs of morbid conditions of recognized structural character.

To the inherent difficulties of diagnosis which are due to the intricacies of the anatomy and physiology of the nervous system, 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.

If the injuries are to 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 can not 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, which figures very prominently in many cases, 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 such as 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 for 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 evident, 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.

In the absence of information as to the exact details there is usually no difficulty in learning the general characters of an accident, from which much may be inferred as to its probable results. The law of averages, or the "law of the long run," as Prof. Mendenhall (Science, December, 1895) calls it, holds good here as elsewhere, so that each variety of casualty has its own ratios of characteristic effects. How unvarying these effects are, when determined by sufficiently extensive observations, is shown by the following tables, which have been kindly furnished

me by an officer of one of the largest railway systems of America.

They relate to accidents and injuries of all characters which have occurred in the past six years on or about the railway or in any of the shops of the company. They have proved to be of invaluable service to the public, to employees, and to the railway company, for, by showing the varieties of accident which are most frequent and the conditions under which they are most liable to occur, they call official attention to faults in construction or operation, and thus furnish the surest means of diminishing the chances of personal injury.

Lack of space prevents me from reproducing all the ratios which this able statistician has generously placed at my disposal. The three following tables, however, illustrate the general character of his work and the inferences which may be drawn from it:

TABLE 1.

INJURIES TO PASSENGERS.

YEAR.	1891.	1892.	1893.	1894.	1895.	1896.
WHERE, ETC.:						
On passenger trains	87.5	90.9	88.9	83.1	80.6	79.1
On freight trains	8.2	4.0	2.5	3.8	8.6	5.3
On station grounds, etc	4.3	5.1	8.6	13.1	10.8	15.6
Postal and expressmen	14.0	6.3	5.0	6.1	2.1	4.2
CAUSE:						
Collisions	27.6	33.8	42.7	16.9	8.0	8.0
Getting on and off moving trains	17.2	28.9	10.3	27.9	28.0	14.5
Getting on and off trains after stop made	3.5	7.7	2.2	7.9	10.8	8.6
Defective and unlighted stations and platforms	0.8	....	0.2	....	2.1	2.2
Miscellaneous	50.9	29.5	44.4	47.3	51.1	56.7
RESULT:						
Death	5.0	7.2	7.8	10.8	1.4	9.5
Loss of limb	1.5	2.7	1.8	1.6	1.4	0.6
Loss of finger or toe	1.5	0.4	0.4	....	0.7	....
Spinal injury	4.7	1.8	2.2	2.3	0.7	1.2
Fracture or dislocation	8.6	5.4	3.5	11.6	8.6	8.7
Sprain	3.9	4.5	5.7	6.6	6.4	12.3
Cuts and bruises	48.3	52.9	63.2	34.3	55.0	44.7
Miscellaneous	26.5	25.1	15.3	32.8	25.8	23.0

TABLE 2.

INJURIES TO EMPLOYEES.

YEAR.	1891.	1892.	1893.	1894.	1895.	1896.
EMPLOYMENT:						
Conductors	5.3	6.4	7.2	7.8	5.0	4.8
Enginemen	2.0	2.6	2.9	2.2	2.6	2.1
Firemen	4.2	3.5	5.0	4.4	4.1	4.3
Brakemen	45.1	45 8	44.3	39.1	30.8	26.0
Mechanics	18.4	16.2	18.8	19.4	28.0	34.6
Warehousemen	1.0	0.7	0.7	0.4	0.4	0.8
Laborers	16.0	16.6	13.8	10.8	15.8	16.7
Miscellaneous	8.0	8.2	7.3	15.9	13.3	10.7
CAUSE:						
Coupling	28.6	27.9	26.3	23.2	17.4	11.1
Collisions	1 9	2.4	3.8	2.5	2.2	1.3
Getting on and off trains	7.3	7.2	6.5	8.0	7.0	7.5
Caught in frogs and switches	0.2	0.1	0.1	0.3	0.1	0.4
Use of tools and machinery	7.8	0.6	3.2	13.8	22.3	28.0
Fell from cars	5.5	4.8	4.9	5.6	4.3	4.1
Defective tools and appliances	1.8	0.6	1.2	0.5	0.6	1.6
Miscellaneous	49.7	56.4	55.2	46.1	46.1	47.6
RESULT:						
Death	3.9	4.0	3.0	2.5	1.3	1.6
Loss of limb	1.4	1.1	1.1	1.2	0.4	0.5
Loss of finger or toe	3.2	3.4	3.4	3.3	2.3	1.3
Spinal injury	0.1	1.3	2.7	0.6	0.1	0.1
Fracture or dislocation	6.2	6.3	6.0	5.3	4.9	3.6
Sprain	14.1	13.0	11.0	16.3	15.2	17.3
Cuts and bruises	47.7	50.0	49.6	39.4	50.1	52.2
Miscellaneous	23.4	20.9	23.2	31.4	25.7	23.4

TABLE 3.

PERCENTAGE OF RESULTS.

YEAR.	1891.	1892.	1893.	1894.	1895.	1896.
RESULT:						
Death	10.3	9.0	8 5	8.6	6.1	5.9
Loss of limb	2.7	2.2	2.1	2.3	1.4	1.0
Loss of finger or toe	3.0	3.0	2.7	2.6	2.0	1.3
Spinal injury	0.4	1.3	2.7	0.6	0.2	0.2
Fracture or dislocation	6.6	6.6	6.6	6 2	5.7	4.8
Sprain	10.9	10.5	9.1	13.5	13.3	15.3
Cuts and bruises	44.9	47.2	47.4	36.6	46.5	48.8
Miscellaneous	21.2	20.2	20.9	29.6	24.8	22.7
TOTAL:						
Passengers	257	221	281	130	139	187
Travelers on highway	190	239	179	195	196	184
Employees	2,488	3,105	3,087	2,339	3,854	3,753
Trespassers	455	492	447	430	433	399
Total	3,390	4,057	3,994	3,094	4,622	4,523

All these statistics are self-explanatory. The first and second tables show the distribution of accidents among passengers and employees respectively; table 3 shows the percentage results of all accidents and the total number of injured persons upon which the statistics are based.

From the study of these tables it appears that the larger number of these casualties are trivial and occur among employees, and are consequently unattended with medico-legal questions, so that the percentage of injuries to the nervous system is very much less than one would suppose by reading court records.

What is true for railway accidents is also true for other kinds of casualties—namely, that their character 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 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 can not, of course, be foretold. On the other hand, it can be positively stated 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.

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. 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:*

* State Hospitals Bulletin, July, 1896.

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.
  - Symelus.
  - Ectromelus.
  - Phocomelus.
  - Excessive length of the arms.
- 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 pre-eminently 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 can not 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. 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 its 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 overcoming visitations, such as a healthy man could easily resist. Accordingly, more reliance is to be placed upon those evidences of alcoholism which the physician may discover, rather 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 may be 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 "traumatic 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

* Die Beurtheilung der Nervenerkrankungen nach Unfall, Stuttgart, 1896.

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. After acute injuries to the nervous system, latent syphilitic processes not uncommonly become active and give the symptoms of focal lesions. Syphilis, like alcoholism, 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.

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 Blackwell's 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 probable 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. 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.

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.

In some cases, and especially if the patients are women, it is impossible to obtain consent to a complete examination. Under such circumstances the physician in his report should

not fail to mention such limitations, and should only give a conditional opinion.

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.
- 7th.
- 8th.
- 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).

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.—After organic injury to the spinal cord or peripheral nerves there are no characteristic mental symptoms unless the brain is injured as well. The mental states in hysteria and neurasthenia will be described with those diseases.

After severe head injuries the patient may be in coma or be stupid and delirious, and no further evidences of the mental state are obtainable. If, after slight injuries, there is decided impairment or perversion of the mental faculties, there is reason to suspect that some disease or poisoning affecting the mind had pre-existed. Eccentricities of manner, carelessness or peculiarities of dress, and in many conditions the facial expression, are to be regarded as evidences of mental states.

In accident cases the condition of the memory is, for many reasons, of special importance. It should be ascertained how much the patient remembers about the accident, and whether he is cognizant of events which occurred just before it, or during it, or just after it. If he were rendered unconscious, it should be determined how soon consciousness returned and memory became re-established. The memory for recent events should be compared with memory for things of the more remote past. The presence or absence of systematized amnesia should also be established. Next to memory, the power of fixing the attention is one of the most important of mental symptoms. In coma and delirium it is of course abolished; but often, when the patient is apparently conscious, he shows by inattention or by confusion that he has very little idea of his surroundings. Delusions, illusions, and hallucinations may be elicited by the physician himself, or may be observed by attendants.

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

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. 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, asafœtida, 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, renders 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 by a competent ophthalmologist has proved an absence of organic disease or defects which might explain it.

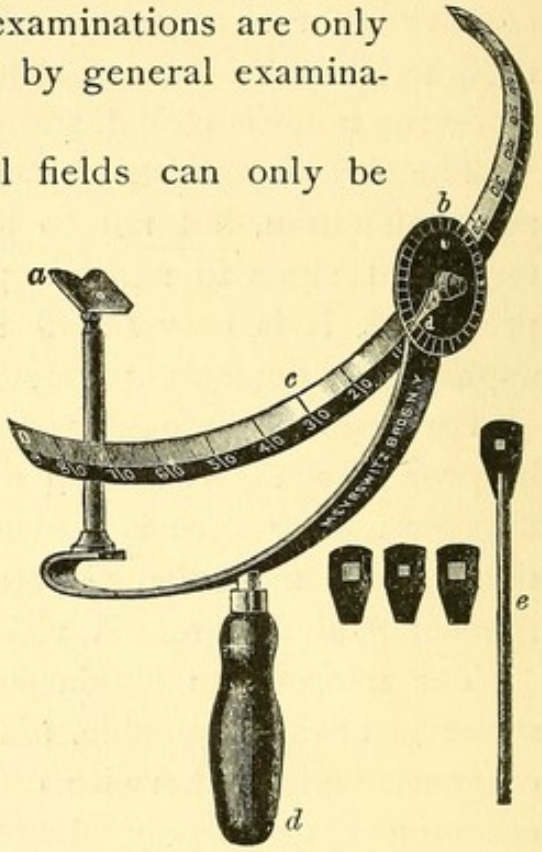


FIG. 1.—Schweigger's perimeter.

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.

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

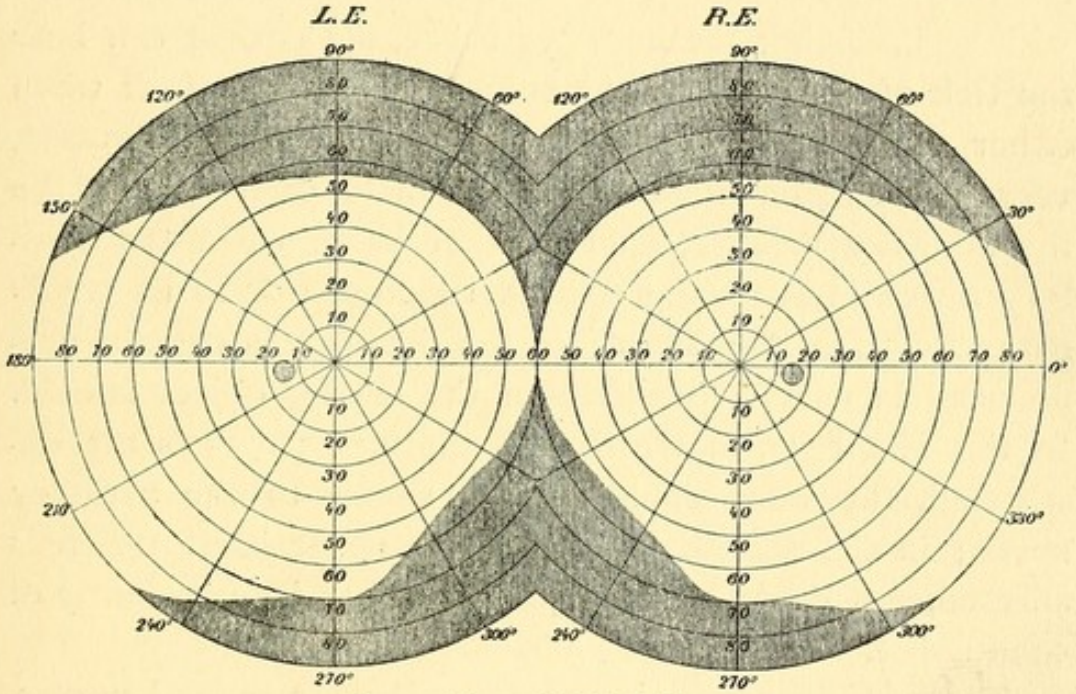


FIG. 2.—Normal field of vision.

in different individuals within normal limits, but a diminution of ten degrees 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 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, 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. 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 can not 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, paralysis from cerebral lesions is rarely absolute. Even when the use of the paralyzed member is very 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 the rule, 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—the physician can usually recognize inequalities of muscular power on the two sides. 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.

The neck is rarely the seat of traumatic paralysis. Its presence 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. The best 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 is seen by impairment of the finer movements of the fingers. The patient has difficulty in buttoning his clothes, in picking up pins or other fine objects, in the effort necessary to 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, when lying at full length, should be requested to raise himself 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

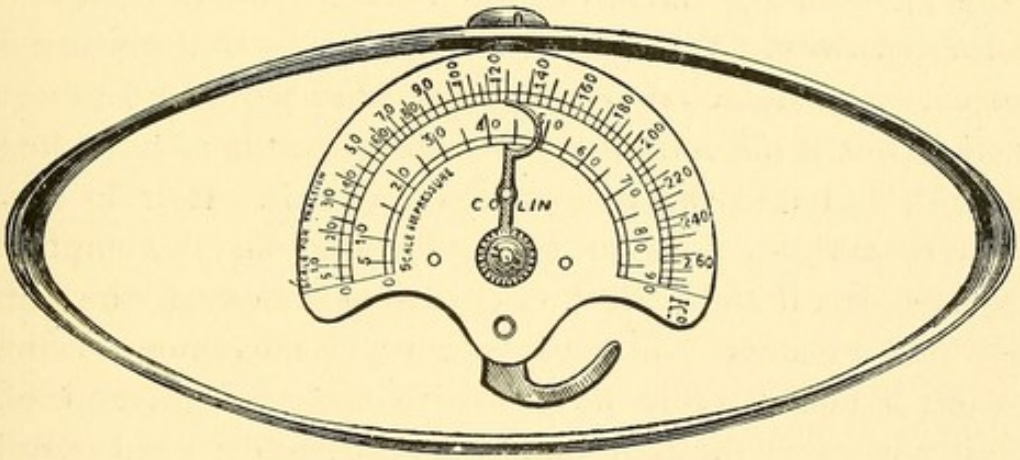


FIG. 3.—Hand dynamometer.

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 ap-

paratus 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.—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; it is shown by the general stiffness of movement, and especially by the gait.

Acute cerebral lesions which produce paralysis cause 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.—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 at trying to touch objects with the toes, or by trying to describe circles on the floor.

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.

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. Unless the injury has been extremely severe, the physician only occasionally has the opportunity of witnessing these symptoms. In making his decision as to their character, 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 can be no 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 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 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 can not 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 his 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 the attempt, when possible; 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 are a variety of instruments which are of greater service to the anthropologist and experimental psychologist than to the clinician. It is not only usually impossible 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 can not 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.

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 or to the first dorsal root, or to the first dorsal segment 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 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).

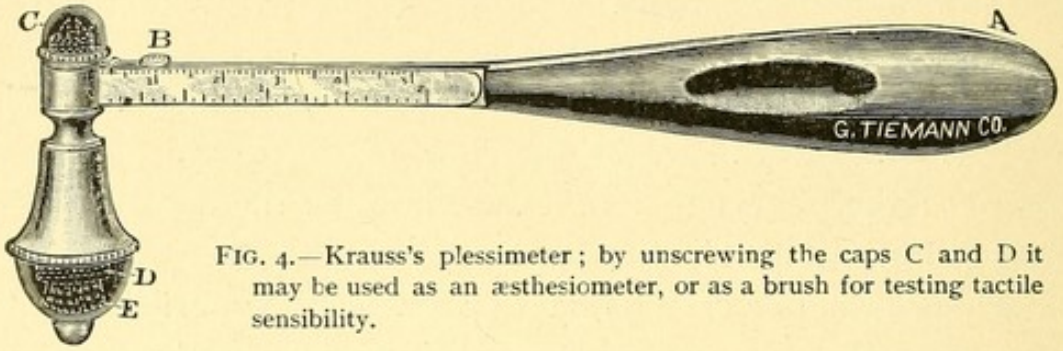


FIG. 4.—Krauss's plessimeter; by unscrewing the caps C and D it may be used as an æsthesiometer, or as a brush for testing tactile sensibility.

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 head 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 frequently associated with ankle clonus, and even in the absence of clonus, when light taps bring about quick and forcible muscular contraction, the condition must be regarded as exaggerated. Since absence 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 employment of the proper means for its elicitation would



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

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 this reflex may be obtained by having the patient sit in a chair, one leg crossed over the

other, while the physician taps the patellar tendon. If there is no response, the Jendrassik (Fig. 6) method of reenforcement is resorted to. The patient, having crossed the legs, pulls forcibly on his closed hands and looks up

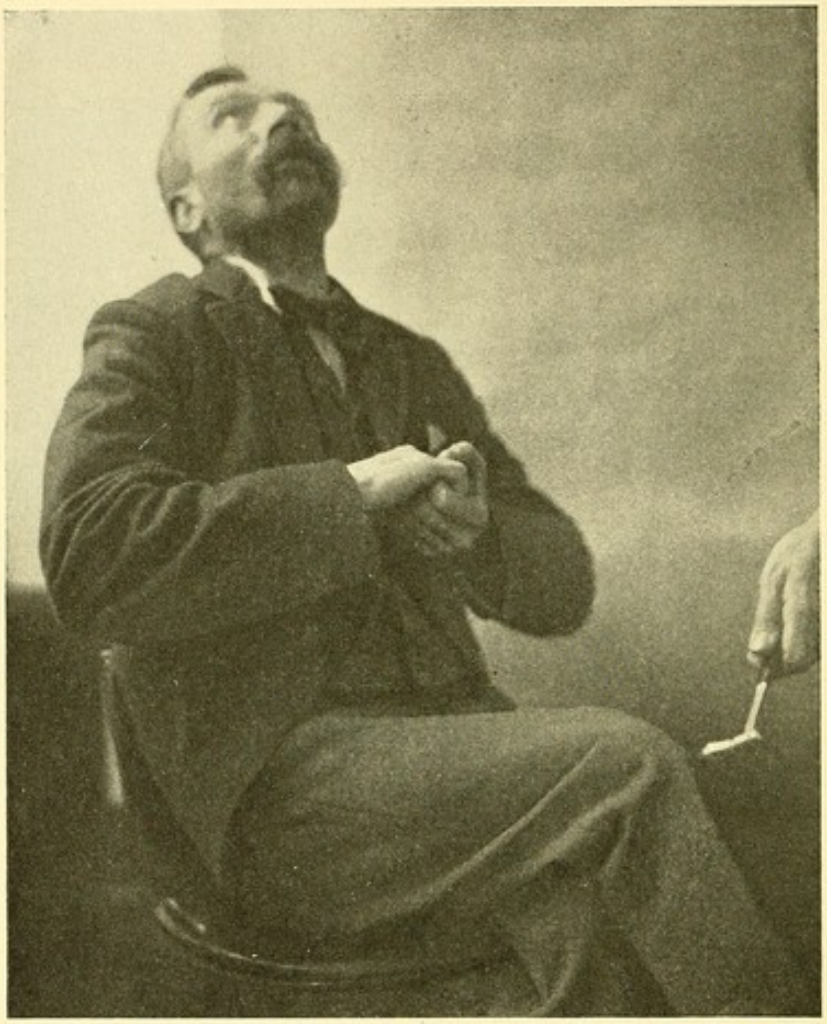


FIG. 6.—Showing the Jendrassik method of obtaining the knee-jerk.

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 ele-

vated 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 can not 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.

Foot or Ankle Clonus is usually regarded as an evidence of degeneration of the pyramidal tracts, although it may rarely occur in functional cases. In these latter, however, it does not present the exaggerated characteristics which stamp it as a symptom of organic disease. 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 become at once apparent when the cutaneous surface is exposed; but by inspection alone it is impossible to determine the presence of atrophy of the muscles, unless it has already reached a considerable degree of advancement. Under such circumstances the affected part, as determined by inspection or by measurement with a steel tape, is found to be smaller than that of its fellow of the opposite side; or, if both limbs are involved, smaller than there is reason to suppose they were before the accident. Also, when the atrophy of muscle is pronounced, the skin over it is loose and flabby, and there is absent the feeling of firmness and resistance characteristic of normal muscle; but in the early stages such unmistakable evidences of atrophy are often wanting, and it is necessary to resort to examination by

electricity. If there are abnormal changes in electrical excitability it follows as an inevitable conclusion that there is also an atrophy of muscle, although it may have escaped detection by other methods of examination.

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. Electrical examination is indispensable for all affections of the lower motor neuron, and no case of paralysis can with justice be diagnosticated as functional unless such means of investigation have been employed.

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 can not be compared with 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

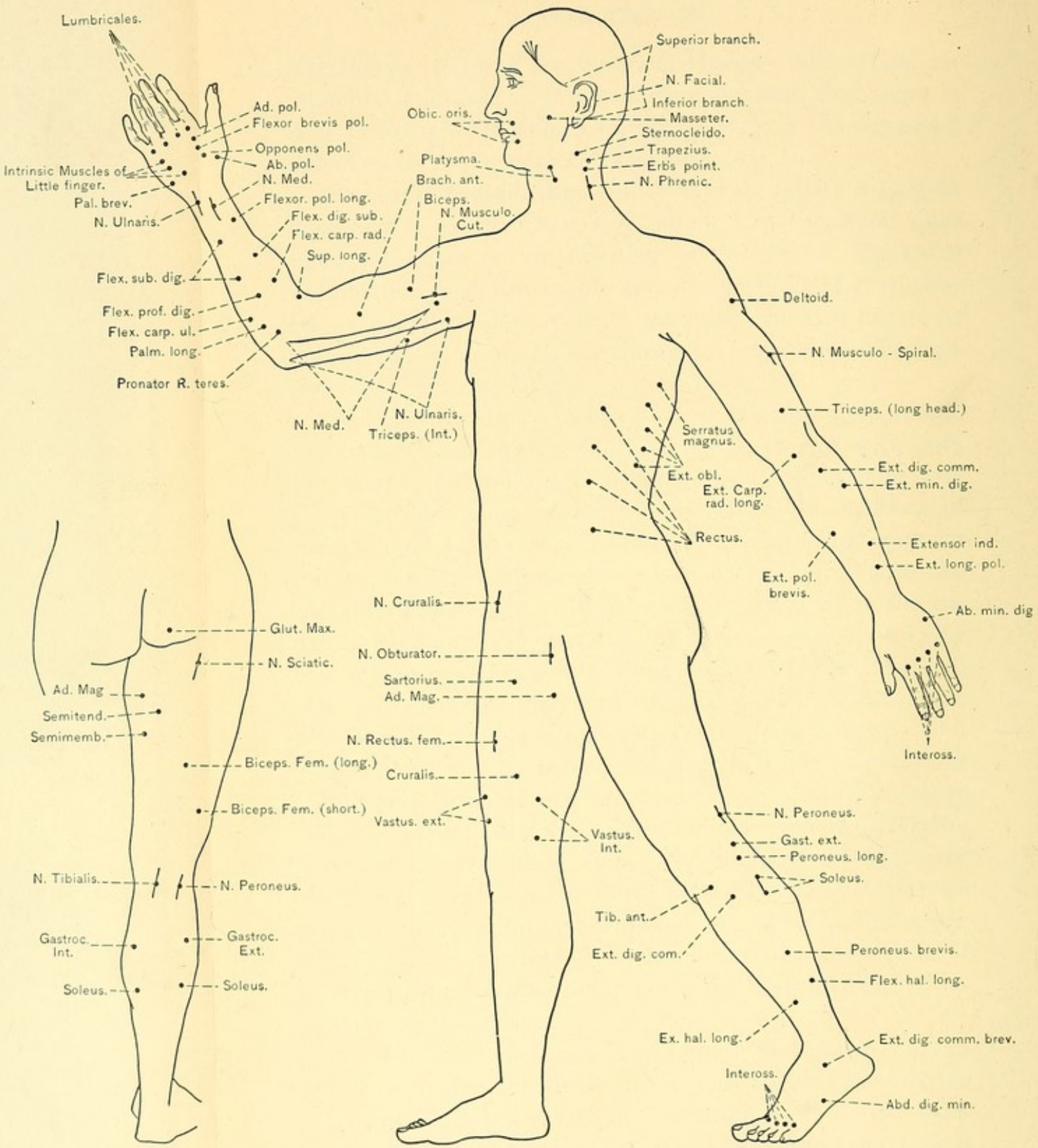


FIG. 7.—Diagram showing the superficial position of the large nerve trunks and the motor points for the muscles.

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. 7).

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 terminations* 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 contractibility to faradism the galvanic response decreases until it reaches and remains at the standard of health.

The accompanying diagram (Fig. 8) may graphically represent the behavior of the electrical reactions for the first four weeks after a moderately severe injury of a periph-

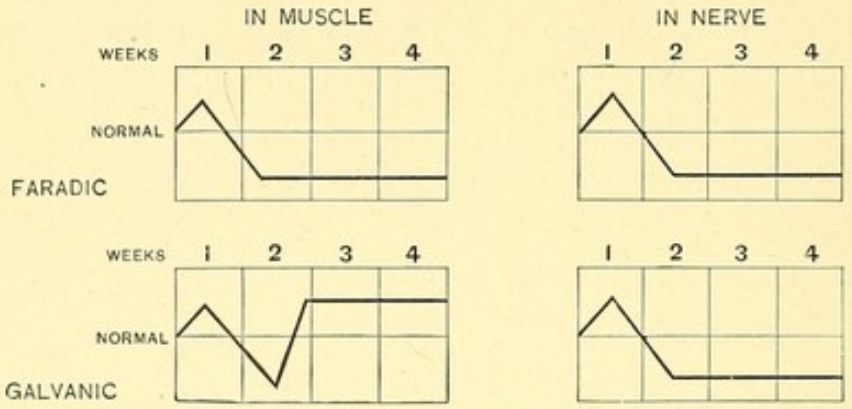


FIG. 8.—Diagram to illustrate the electrical reactions occurring in a nerve after a moderately severe injury.

eral 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 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 wormlike, instead of being quick and tonic, as in health. This vermicular 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.

PART I.

ORGANIC EFFECTS OF INJURY TO THE NERVOUS SYSTEM.

WHILE it is not the chief object of this work to portray the clinical or pathological conditions which may result from organic injuries to the nervous system, a brief mention of nervous symptoms as caused by definite lesions seems imperative as a preliminary to the study of functional derangements. In all cases of traumatic nervous disease the question at once arises whether there is a lesion destroying nerve tissue, or whether the symptoms are merely indicative of a repression or perversion of function of temporary character occurring independently of known lesion.

In functional cases the evidences of structural lesion are not present; to prove them absent, however, requires a painstaking examination directed toward the whole cerebro-spinal axis. It is accordingly the object of the three immediately succeeding chapters to briefly describe the symptoms which are to be regarded as indicative of acute organic injury to the brain, the spinal cord, and the peripheral nerves. In the fourth chapter of this section are considered the possibilities for a traumatic origin of certain of the chronic degenerative diseases.

CHAPTER I.

INJURIES TO THE BRAIN.

CONTRASTED to the cerebral injuries in which there is no difficulty in deciding upon the character or the approximate location of the trouble, are cases in which the nature or even

the situation of the lesion can not be determined without an examination of the whole nervous system, and it is in such cases especially that the physician needs to be familiar with the essential characteristics of those symptoms which bespeak loss of continuity in cerebral conducting paths or injury to cerebral cells.

Even the most scrupulous care and widest experience are often unavailing for diagnosis, and the nature of the condition does not become plain until recovery stamps it with characteristics of individual types, or until the pathologist gives the answer to the riddle which the clinician was unable to solve.

The most perplexing cases are those in which unknown persons are found lying senseless. The presence of scalp wounds may prove a head injury, such as could have been inflicted by a blow or a fall; witnesses are wanting, and the physician or surgeon must rely upon his own skill to decide whether the patient lost consciousness as the result of a fall or blow on the head, or whether the fall resulted from dizziness or unconsciousness brought about by some purely medical disease.

It is the old and perplexing question of the differential diagnosis of coma; its consideration here must be limited to a brief description of those symptoms which are usually present when it is due to direct injury of cerebral tissue. They are of three classes:

1. *General*, which may occur after any severe general injury.

2. *Mental*, which indicate interference with intellectual processes.

3. *Focal*, which are due to lesions of circumscribed areas of the brain.

1. **General Symptoms.**—The most important symptom of general injury, either to the nervous system or to other parts, is surgical shock.

It is to be distinguished from psychic or nervous shock, which is the impression made upon the mind by sudden and painful emotional influences. Physical or corporeal shock is a sudden depression of the functional activity of the general nervous system. Its pathology is unknown. It is a constant accompaniment of severe injuries of any part, and it sometimes follows traumatism of a trifling nature. It occurs immediately after the accident, and may continue a varying length of time. Consciousness is not lost, but the patient is depressed and apathetic. The face is pale, the skin is moist and cold, the pulse is soft, rapid and feeble, and the respirations are shallow. Shock often appears to be the sole determining cause of death.

Among other common general symptoms of lesion to the brain more suggestive than shock may be mentioned vomiting, slow pulse, stertorous breathing, the tendency to congestion, œdema or consolidation of the lungs, fever, and incontinence or retention of urine or fæces.

2. **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 cerebral injuries, and in many which are apparently trivial, it is more or less completely lost. 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.

Often, instead of unconsciousness or hebetude, the mental condition is one of confusion. This may even be the case after 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 hæmorrhage on the opposite side of the brain.

Delirium is a frequent complication of head injuries, especially in alcoholic persons. It may be mild or muttering, or may be attended with hallucinations, delusions, and impulsive acts.

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 some irremediable cerebral condition. In some fatal cases of traumatic cerebral hæmorrhage mental symptoms exist without any focal signs.

3. **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. 9 and 10). Although no individual region of the

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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

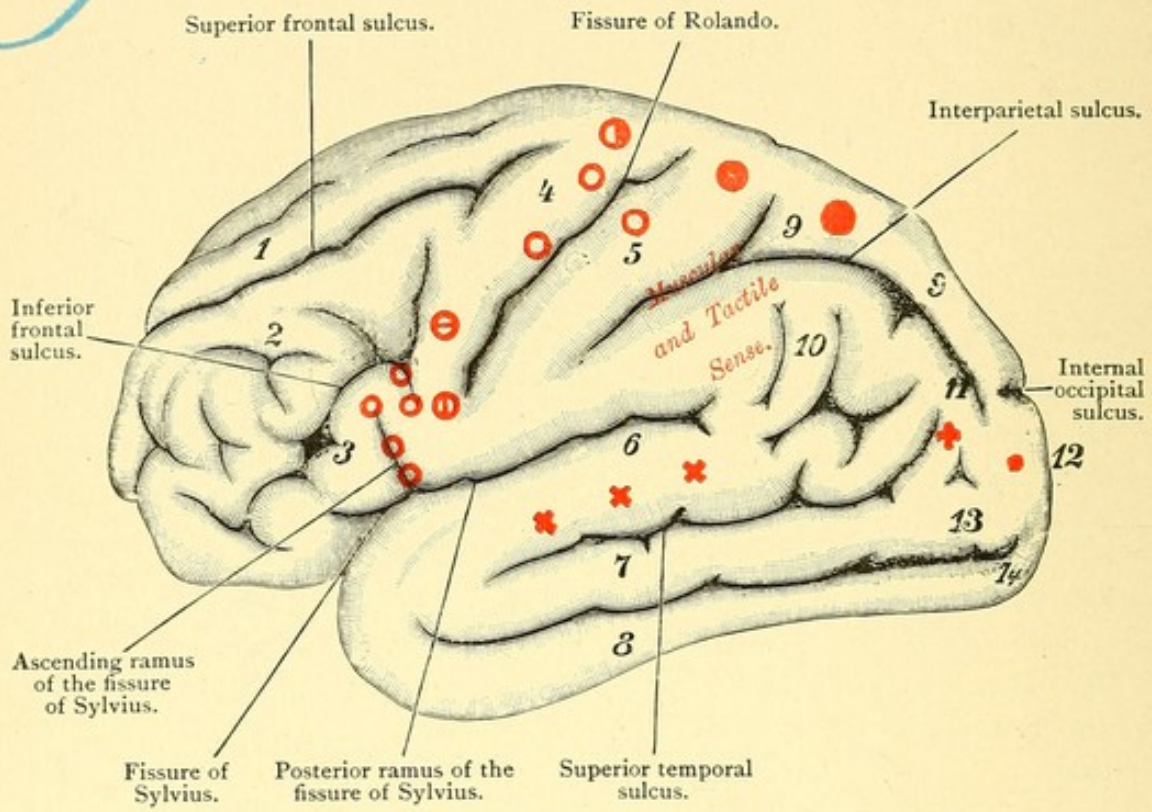


FIG. 9.—Schematic representation of the cerebral cortex and its centers. (After Tillmanns.)

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

4. Anterior.
5. Posterior.
} Central convolution.

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

9. Upper.
10. Lower.
} Parietal convolution.

11. Gyrus angularis.

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

○ 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. 10).

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 traumatisms 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

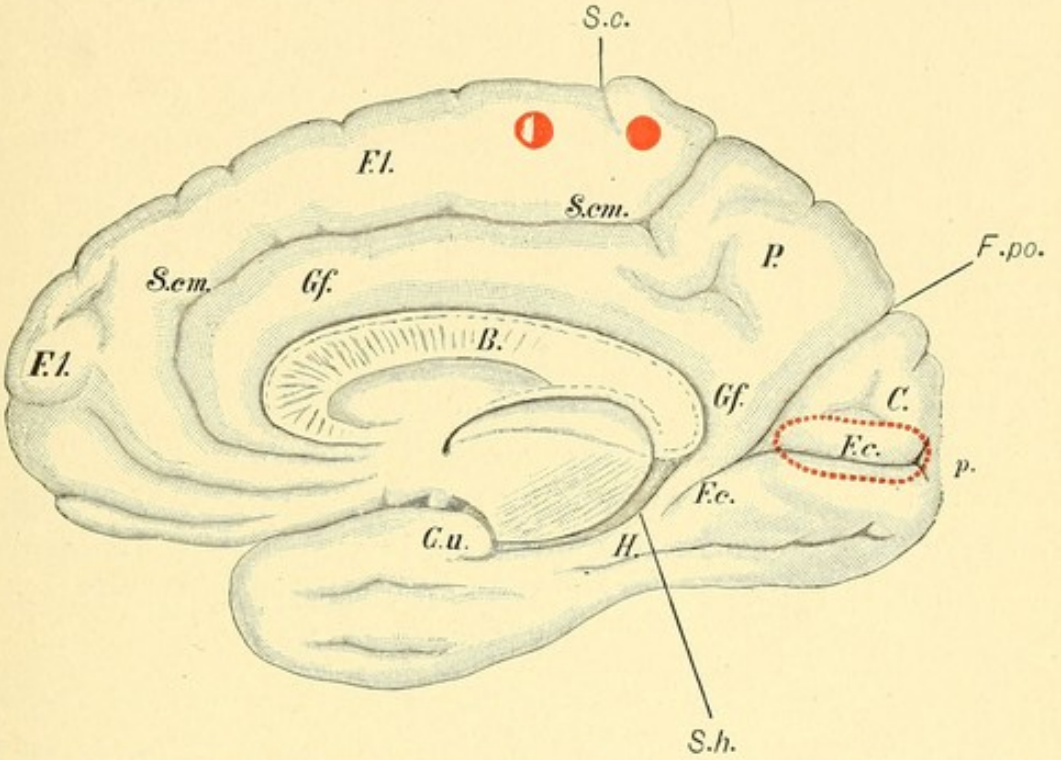


FIG. 10.—View of the right cerebral hemisphere from the median side; *B*, corpus callosum divided longitudinally; *G. f.*, gyrus fornicatus; *H*, gyrus hippocampi; *S. h.*, sulcus hippocampi; *G. u.*, gyrus uncinatus; *S. c. m.*, sulcus calloso-marginalis; *F. 1*, first frontal convolution; *S. c.*, 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.)

mental symptoms that examination for the highly specialized cerebral functions is unsatisfactory or unavailing. However, as such selective injuries sometimes occur without seriously interfering with general intellectual power, and since, 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, the most common forms of cerebral speech disturbances must be mentioned.

The best defined of the cerebral centers for speech may be seen in the accompanying figures 9 and 10. They in-

clude 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 without loss of understanding of spoken or written language. Motor aphasia is the most common of the isolated speech disturbances which may be due to traumatisms. 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 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. 11) 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

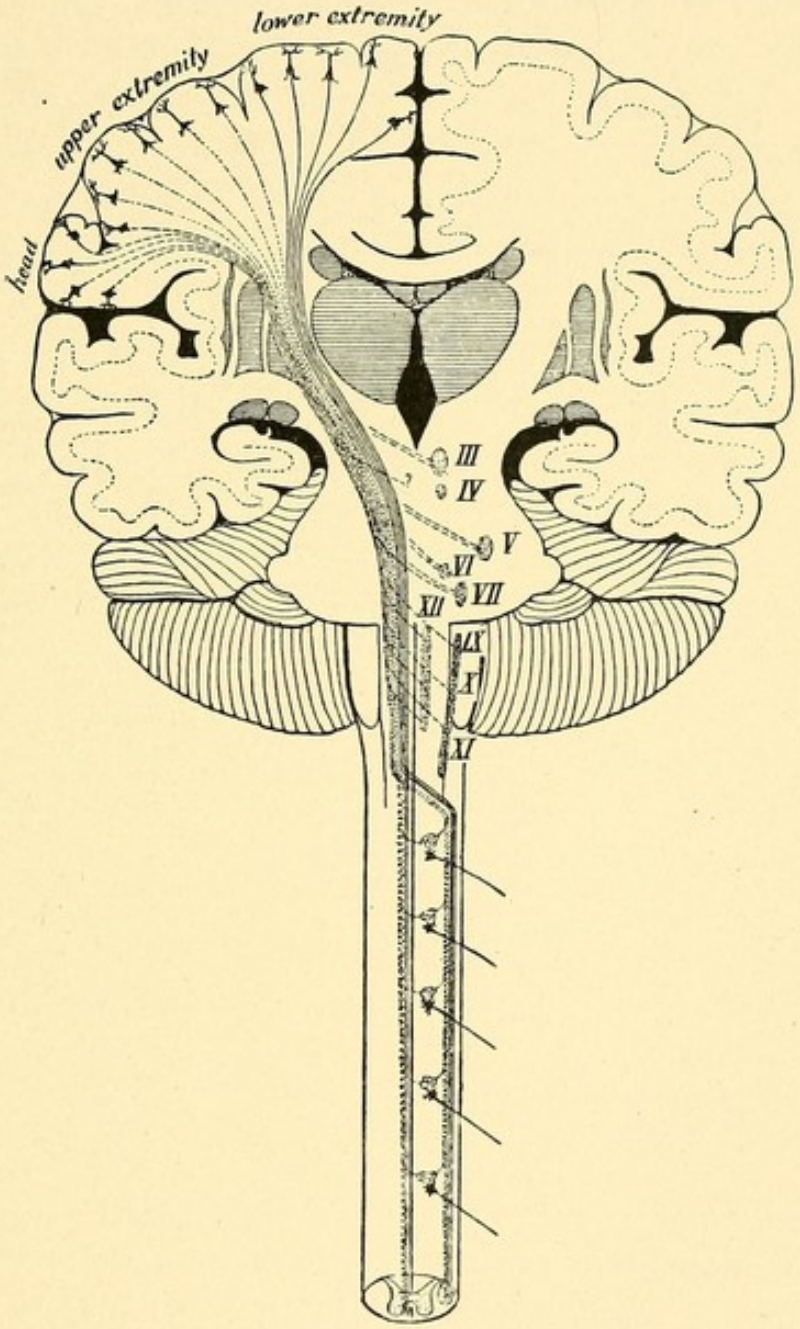


FIG. 11.—Schema illustrating the course of the cerebro-spinal motor path. (After Van Gehuchten.)

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 the (*a*) cell body, the (*b*) protoplasmic processes or dendrites, which are generally supposed to convey impulses *to* the cell and are consequently afferent, and the (*c*) axis cylinder of the cell, or the neuraxon, which is known to convey impulses *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. 12). 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 filaments with the peripheral neuron. The peripheral

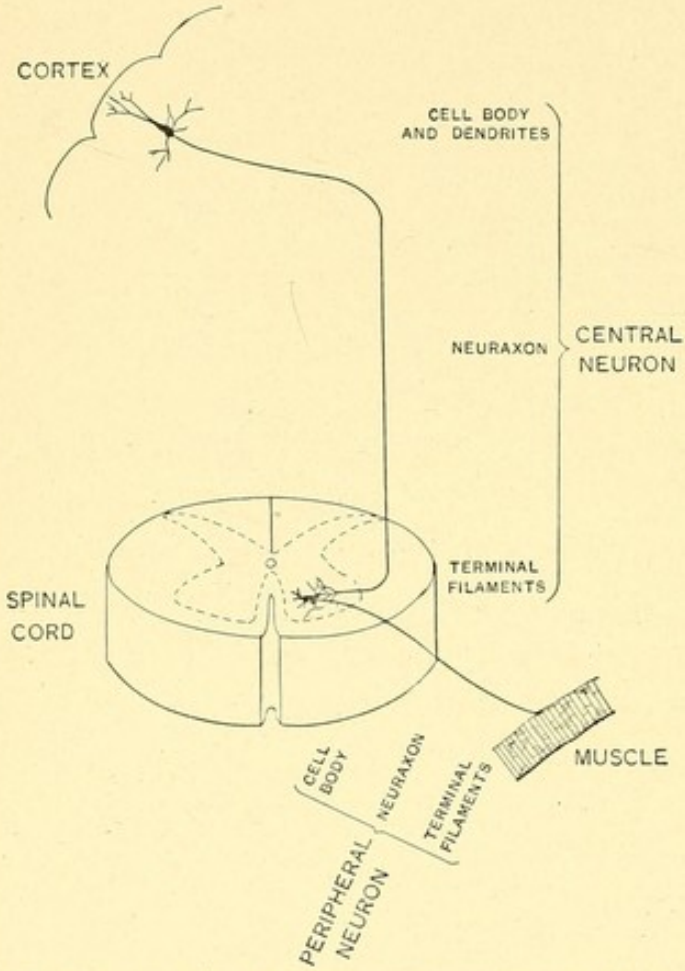


FIG. 12.—Schema to illustrate the arrangement of the motor neurons.

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 can not 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,
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 seen only 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 traumatism of the brain; when it occurs it is due to a selective 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.

If the lesion is in the anterior fossa of the skull, the only *cranial nerves* affected are the olfactory and optic.

Symptoms of direct injury to the *olfactory nerves* are rare. They consist of disturbances of smell, which are usually associated with impairment of taste. Fractures at the base of the skull and penetrating wounds not uncommonly cause laceration or hæmorrhage into the sheaths of the *optic nerves*. If either optic nerve is injured in front of the chiasm, the result is more or less complete blindness in the eye of the same side. The ophthalmoscope shows optic neuritis, which may be followed by atrophy.

Injury of the optic nerve at the chiasm causes bilateral blindness. If the optic tract is injured posteriorly to the chiasm the result is hemianopsia. Palsy of the *third nerve*, 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. 13). Complete third-nerve palsy, however, is comparatively rare in acute conditions. More frequently the nerve shows a partial and selective palsy. Thus there may be a drooping of the upper lid, so that the affected eye can not 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.



FIG. 13. — Double third-nerve palsy, showing outward and downward deviation of both eyes and wrinkling of the forehead. (Incurable Hospital.)

Palsy of the *fourth nerve* is uncommon, and unless pronounced is only demonstrable by means of prisms.

The *fifth nerve* usually escapes intracranial lesion and is never injured alone in this situation. Its affection is revealed by palsy of the masseters and buccinators and by anæsthesia, with pain over the face and forehead. If the fibers to the eyeball are involved there results neuroparalytic ophthalmia.

The *sixth nerve* is frequently the seat of traumatic paralysis. Through the resulting weakness of the external rectus, the eyeball is turned inward and movements outward are limited or impossible. Palsy of any of the ocular nerves causes the subjective sensation of diplopia.

Intracranial facial (*seventh nerve*) paralysis differs from extracranial facial paralysis in that there may be added disturbances of taste and of salivary secretion. When of trau-

matic origin it is invariably unilateral and associated with disturbances of the *auditory nerve*, such as deafness, subjective auditory sensations, and dizziness. Palsy of the nerve will be considered more fully on a later page. By reason of their situation, the glossopharyngeal, pneumogastric, and hypoglossal nerves are never paralyzed by acute intracranial injuries, which are not almost immediately fatal.

Sometimes several cranial nerves are paralyzed together. A characteristic clinical type of such multiple nerve lesions,

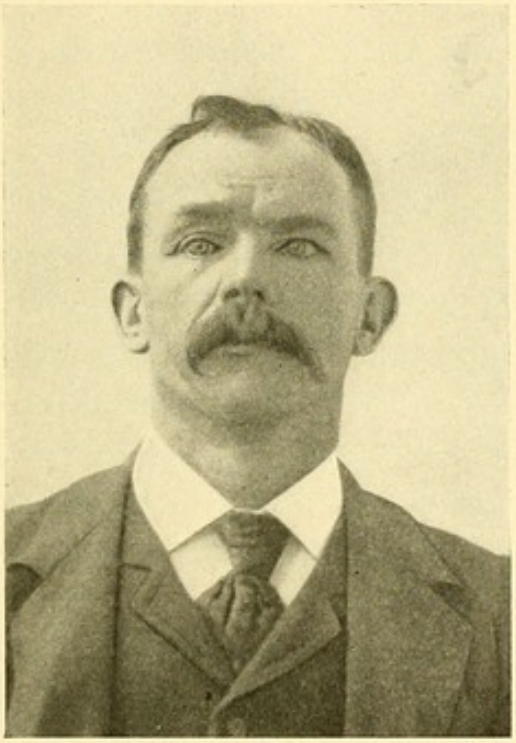


FIG. 14.—Photograph showing paralysis of the left sixth and seventh nerves, due to a fracture of the skull. Patient trying to look straight ahead.

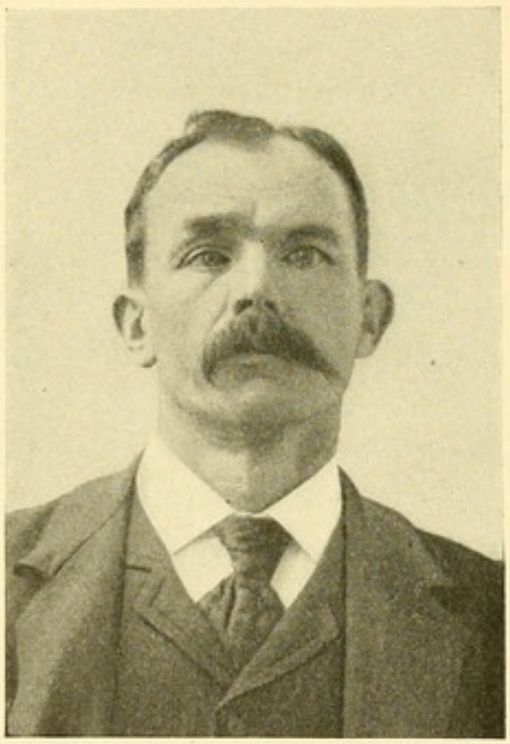


FIG. 15.—Same case. Patient trying to look toward the left. (Vanderbilt Clinic.)

sometimes seen after fractures at the base, is illustrated by the following case (Figs. 14, 15).

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 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.

However, in traumatic affections at the base of the brain there is only rarely the opportunity for that accuracy of topographical diagnosis which is so generally possible in chronic lesions. The former result, in the vast majority of cases, from fractures at the base of the skull which cause such extensive hæmorrhages or lacerations of the cranial nerves or of the cerebral substance that it is usually impossible to determine as to the existence of discrete multiple lesions, and the physician or surgeon generally has to content himself with making the diagnosis of injury at the base of the brain, without definitely ascertaining its exact location.

The symptoms of injury to the *cerebellum* are often indefinate. Lesion of the lateral lobes gives no localizing symptoms. If the middle lobe, or worm, is involved, there is the characteristic cerebellar ataxia, which may be associated with loss of knee-jerks. Injuries to the medulla are always immediately fatal.

Sensory Symptoms.—Pronounced anæsthesia is rarely if ever observed as a focal symptom of traumatisms 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 gives 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 can not be denied, however, that similar symptoms might result from laceration of the brain, or from hæmorrhages 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 traumatism could act in a way to produce so small and so selective a lesion as would be necessary to cause the pure type of hemianæsthesia, such as is produced by interference with the sensory tracts in the pons or in the internal capsule, without at the same time so seriously injuring other parts of the brain that the unilateral character of the sensory loss was not maintained, or that the sensory symptoms became masked by more serious nervous 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.

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 one of the most valuable diagnostic signs of hemiplegia, and may be present when the loss of motor power is very slight.

Bladder and Rectum.—In deep coma there may be the retention of urine, or the involuntary passage of urinary and fæcal discharges, which is the rule. When the patient is conscious there is usually no loss of control of the bladder or rectum as a result of cerebral injuries.

Varieties of Brain Injuries.

The brain is very much less securely protected against injury than the spinal cord, there being wanting the thick cushions of muscles, and the bones which surround the encephalon being thinner and less resistant than those which inclose the spinal cord. A blow which, if applied to the back, would be followed by no evidences of spinal cord lesion, might, if applied to the head, cause serious cerebral injuries. By blows on the head, by falls, or by the entrance of foreign bodies, the skull may be fractured and the brain be contused or lacerated, either directly by splinters of bone or indirectly by transmitted force. Hæmorrhage may occur from vessels which are ruptured, either in the bone, in the membranes, or in the brain substance, or there may be generalized œdema or circumscribed œdema which latter gives focal symptoms.

Without fracture of the skull the intracranial blood-vessels may be ruptured, or the brain substance or cranial nerves may be contused or lacerated. While in the majority of cases the skull is fractured, the fracture is often not recognizable during life, and the diagnosis of the nature

of the injury must be made from the nervous symptoms alone.

Concussion.—In the chapter on injuries to the spinal cord it will be stated that there is still much doubt as to the existence of any such condition as concussion of that organ. The probability that head injuries may be followed by pronounced cerebral symptoms without there being any discernible lesions in the brain is very much stronger. There is, of course, no question but that after many head injuries multiple hæmorrhages occur, especially in the gray matter, which do not give any localizing symptoms, and that in many of these cases the condition must be diagnosticated clinically as that of cerebral concussion. But the results of daily clinical experience leave hardly room for doubt that, after very insignificant head injuries, there occur unconsciousness and other slight cerebral symptoms of too trivial a character to be dependent upon any lesion so gross as hæmorrhage. To cite an example familiar to boxers, a blow on the point of the jaw may deprive the victim of all appreciation of his surroundings. Yet in a few moments he is entirely recovered and ready to continue the fray. He has suffered temporarily from cerebral concussion. From these conditions, when the patient is for a few minutes without consciousness, to those in which he lies for days in coma, stupor, or delirium, there are various intermediate stages; and while, in the severer cases, we may suspect during life and be able to prove after death that the symptoms depended upon multiple though minute hæmorrhages and lacerations, there are many patients who do not die, and who, by the speediness of their recovery, indicate that the brain has received no severe injury. In the absence of localizing signs we are unable to distinguish between the different varieties of cerebral concussion, and we are consequently obliged to designate by the same term conditions which may be very different.

The symptoms of cerebral concussion may be slight or severe. In consideration of the fact that serious organic injuries of the brain may for a long time cause no pronounced cerebral symptoms, the physician can hardly be accused of overcautiousness if he refuses to diagnosticate cerebral concussion until the patient has been under observation for several days.

After the injury there may be deep coma or stupor, or only mental confusion. Delirium is not infrequent, and in alcoholic patients its occurrence, associated with zoöscopic hallucinations, is the rule.

The patient may be induced to answer questions, but his mind immediately wanders again; he may seem partially conscious and yet not recognize his surroundings. The memory of the accident is often entirely lost. There is usually headache, which may be very intense, and associated with intolerance of light and sound. Dizziness, nausea, vomiting, and convulsions are frequent symptoms. The pulse is slow and regular, and the breathing is superficial. The tendon reflexes are exaggerated, especially in severe cases.

With the exception of nystagmus and alternating strabismus there are no focal signs. As soon as any localizing signs appear, such as unilateral increase of reflexes, or paralysis of the limbs, or evidences of injury to the cranial nerves, the diagnosis of concussion must be changed to that of a more definite lesion. Consequently the diagnosis is one by exclusion. Cerebral concussion is an accompaniment of all severe brain injuries, but under such circumstances its symptoms are masked by those of the graver conditions.

The duration of the condition depends on its severity. Usually the patient is able to get up in the course of a few days, although there may be left evidences of mental impairment which are a long time in disappearing, or which may not disappear at all.

Fractures.—In fractures of the cranial vault there are often external evidences of the condition, such as depressions or fissures in the bones. These may be wanting, however, and in fractures at the base they are never present, so that the diagnosis must be made from the nervous symptoms alone, or from such signs of leakage as subconjunctival ecchymosis or discharges from the ears or nose.

When persons are found unconscious, with paralytic symptoms, with or without scalp wounds, it is not always easy to determine whether the coma is apoplectic in origin, or whether it is a result of a fracture of the skull.

In the summer of 1896 one of the female inmates of the 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 hæmorrhage 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 hæmorrhage in the left internal capsule, but no injury to the other parts of the brain, and no fracture of the skull.

In such cases the absence of all external signs of fracture, the complete hemiplegia without involvement of the cranial nerves, and the occurrence at a period of life when apoplectiform attacks are common, are suggestive of primary rather than traumatic brain lesion. But the diagnosis is always difficult, and in many cases can not with certainty be made.

There is no domain in medicine or surgery in which more caution in diagnosis and prognosis is required than in head injuries.

In fractures of the skull the patient may present no evidences other than those of concussion for days or weeks, and then suddenly develops symptoms of an alarming or fatal character. It is a common occurrence for persons whose skulls are fractured to walk to hospitals or to be arrested for disorderly conduct on the street.

In a case communicated to me by Dr. Psotta, House Surgeon of 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.

The most that the physician or surgeon can do in these difficult cases is to ascertain, as nearly as possible, the severity of the accident, and, after severe accidents, to withhold an opinion until sufficient time has elapsed to render the occurrence of further symptoms improbable.

Any head injury is liable to be complicated by intracranial inflammation, and the symptoms of meningitis, abscess, or encephalitis may be added to those of conditions which did not at first seem serious.

CHAPTER II.

Injuries to the Spinal Cord.

Although the studies of recent years have cleared away much of the confusion with which the writings of Erichsen enshrouded spinal-cord injuries, the terminology applied to these affections to-day is far from satisfactory. For this the unfortunate survival of the expression "concussion of the spinal cord" is largely responsible.

As used to-day, this term is extremely indefinite in its meaning. There is, of course, no longer excuse for applying it to the forms of nervous disease which are purely functional in character (traumatic neurasthenia), nor to the cases in which there may be slight injuries to the vertebral joints without affection of the more delicate structures beneath them (lumbago). But aside from its use, or misuse, as applying to affections in which there is no disturbance of the structure of the spinal cord itself, it remains as a designation of lesions for which the names myelitis, softening, and hæmorrhage are alone permissible. Some writers call any injury to the spinal cord "spinal concussion"; others use the term for any spinal affection which may result from concussion accidents; still others make it synonymous with intraspinal hæmorrhage, with or without vertebral lesion. A conservative few restrict it to those cases in which, with absence of injury to the spinal column, there may be evidences of focal injury to the spinal cord which has not been caused by hæmorrhage.

Since it is still a matter of doubt that there exists any

pathological condition such as the term spinal concussion in its most restricted sense would imply, to retain so generally familiar a designation for a condition which, if it exists at all, is extremely rare, will certainly prove an obstacle to the advancement of our knowledge of the causal relations of traumatism to spinal disease. It is, however, much easier to object to than to do away with a term which has gained popularity. I would nevertheless strongly urge that if "concussion of the spinal cord" is to be retained at all in medical nomenclature, it should be restricted to those cases in which it can be shown that the nervous elements of the cord have been directly injured, without having been compressed by bone or by extravasated blood.

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. 16) 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 its other efficient means of protection, the spinal cord is not infrequently 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

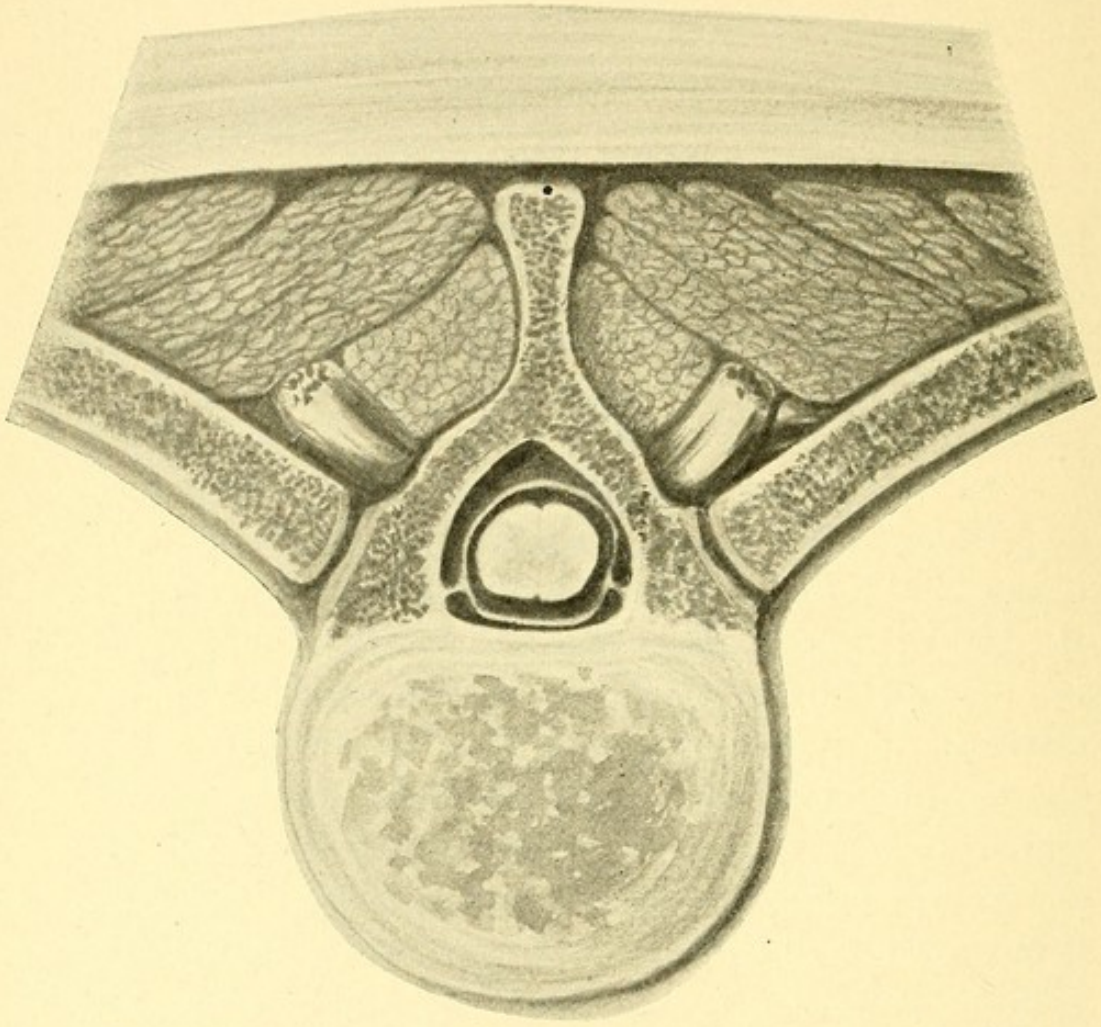


FIG. 16.—Transverse section of the structures surrounding the spinal cord at the line of the ninth thoracic vertebra

fracture. Falls on the buttocks, either from heights or, as occasionally 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 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 established when the symptoms are in accord with those we now recognize as indicative of lesion of the spinal cord.

Although it is almost always possible to determine whether the spinal cord has or has not been injured, the diagnosis of the exact character of the lesion is more difficult. That our knowledge of this subject might be very materially advanced by the careful observation of a large number of accident cases in accordance with some definite classification, seems reasonable to expect; and for the furtherance of that object the following classification is proposed:

I. *Spinal column injured*, by wounds, fractures, and dislocations which cause compression of the spinal cord, and, secondarily, myelitis, softening, or hæmorrhage.

II. *Spinal column uninjured*, but spinal cord the seat of: 1. Compression by blood clots. 2. Hæmorrhage into cord substance. 3. Concussion or commotion.

The clinical applicability of such a classification is, of course, in the present state of our knowledge, restricted, since where there are no external evidences of vertebral injury it is usually impossible to be certain of the exact character of the cord lesion. From the standpoint of pathological anatomy, however, it is justifiable, and it seems not

unreasonable to hope that pathological studies conducted along its lines, when preceded by accurate clinical observations, would so much enlarge our comprehension of the nature of spinal-cord injuries that they could be accurately diagnosticated during the life of the patient. In accordance with this classification injuries to the spinal cord will now be briefly discussed.

I. Spinal Column Injured.

By far the larger number of traumatic lesions of the spinal cord are secondary to injuries of the vertebræ. It is often impossible to determine during the life of the patient whether fracture or dislocation of the bones has or has not occurred, because the integrity of the spinal column may be seriously impaired without there being any irregularity or abnormal mobility of the vertebral spines. External evidences of injury are absent with particular frequency in fractures which involve the bodies of the vertebræ only, or in the dislocations resulting from sudden twists or wrenches, in which the bone or intervertebral cartilage, after having been momentarily thrust forward so as to crush the spinal cord, immediately springs back again into its place without leaving external traces of any change of position.

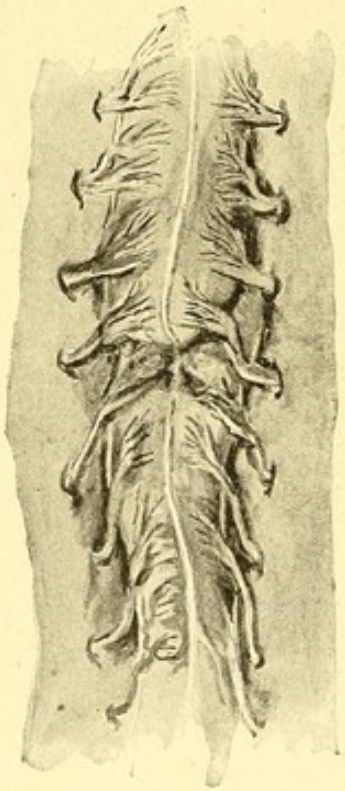


Fig. 17.—Crush of the spinal cord, due to fracture of the cervical vertebræ. (From a drawing kindly furnished by Dr. Van Gieson.)

Vertebral lesions may cause damage to the spinal cord in a variety of ways. The cord itself may be cut in two or lacerated or crushed (Fig. 17) by dislocated bone or by splinters of bone; it may be com-

pressed by hæmorrhage into the spinal canal, coming from the bone, or from the ligaments, or from the membranes (hæmatorrhachis) (Figs. 18 and 19); its blood-vessels may be so torn that blood is poured out into the cord tissue itself (hæmatomyelia). From any of these lesions may immediately result softening or inflammation (myelitis), or, secondarily, degenerations or gliosis of varying degrees. The symptoms of any of these conditions indicate a focal injury to the spinal cord, and will be described on page 90.

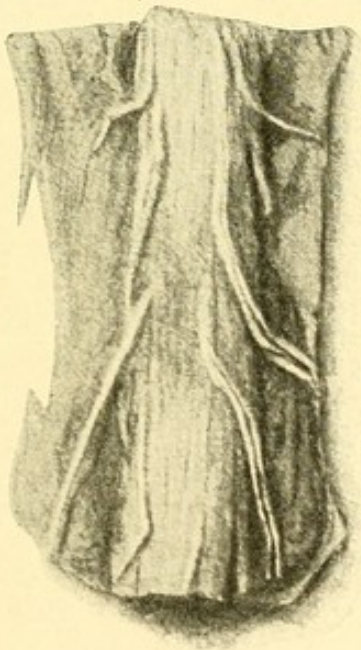


FIG. 18.—Intradural hæmorrhage. (From a specimen kindly furnished by Dr. G. E. Brewer.)

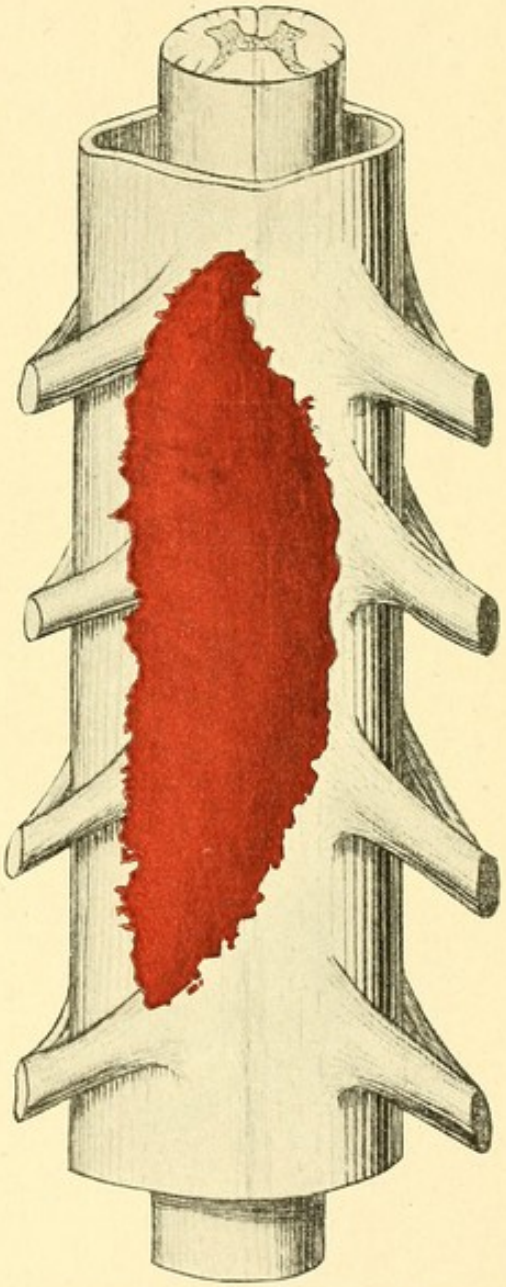


FIG. 19.—Extradural hæmorrhage. (Roosevelt Hospital.)

When the medullary lesion is secondary to injuries to the bones it is usually impossible to decide during the life

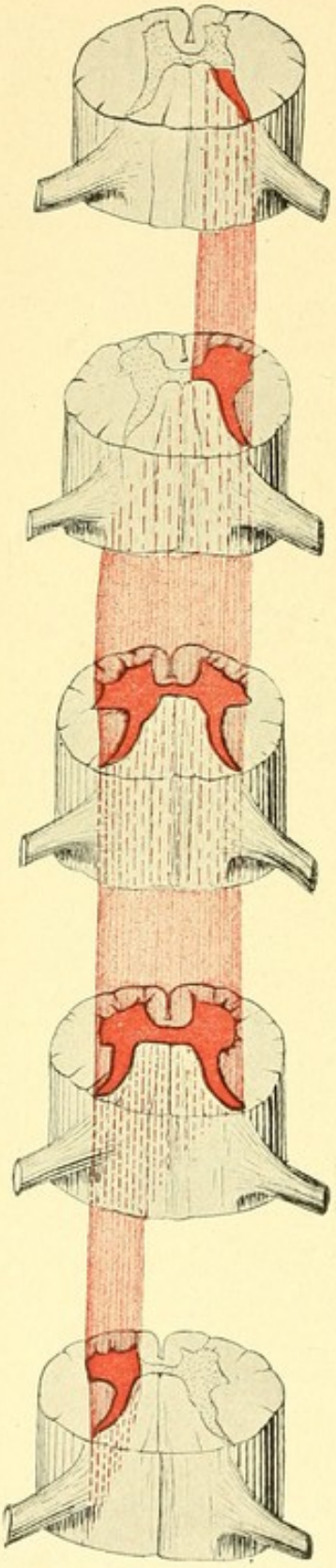


FIG. 20.—Diagram to illustrate the course of the blood in a case of hæmatomyelia.

of the patient the exact nature of the pathological condition within the spinal canal. In such cases the cord is so seriously crushed or compressed at the seat of the fracture that the symptoms are simply those of a transverse lesion, whatever the underlying conditions may be.

In view, however, of the special pathological interest which attaches to hæmatomyelia the following case may be briefly related here. Although the lesion was too extensive to permit any inferences as to the special symptoms of central hæmorrhage, the disposition of the blood was characteristic of that condition:

A man fell on his head, fracturing the cervical vertebræ. He did not lose consciousness, but was immediately paralyzed in all four extremities, and died in a few days. I performed the autopsy at the City Hospital twelve hours after death. *Brain*, normal; *spinal column*, comminuted fracture of the third and fourth cervical vertebræ. On opening the dura, the outline of the *spinal cord* was found to be well preserved, though there was some tendency to bulging at the fifth and sixth cervical segments. On section (Fig. 20) there was a sharply defined hæmorrhage which followed, as nearly as the naked eye could see, the gray matter of these two segments. Farther up and down the cord there was a different disposition of the hæmorrhage. The blood had ascended the posterior horn on the right side as far as the

middle part of the third cervical segment. The path of the descending blood followed the left posterior horn of the cord to the lower part of the seventh cervical segment (Fig. 21). Studied in microscopic sections, the whole of the fifth and sixth cervical segments, with adjacent portions of the seventh and fourth, were found to be in a condition of acute myelitis with its ordinary appearances—viz., destruction of nerve elements with a cellular exudate consisting of leucocytes and large granular cells. In these two segments the blood-vessels were unusually prominent

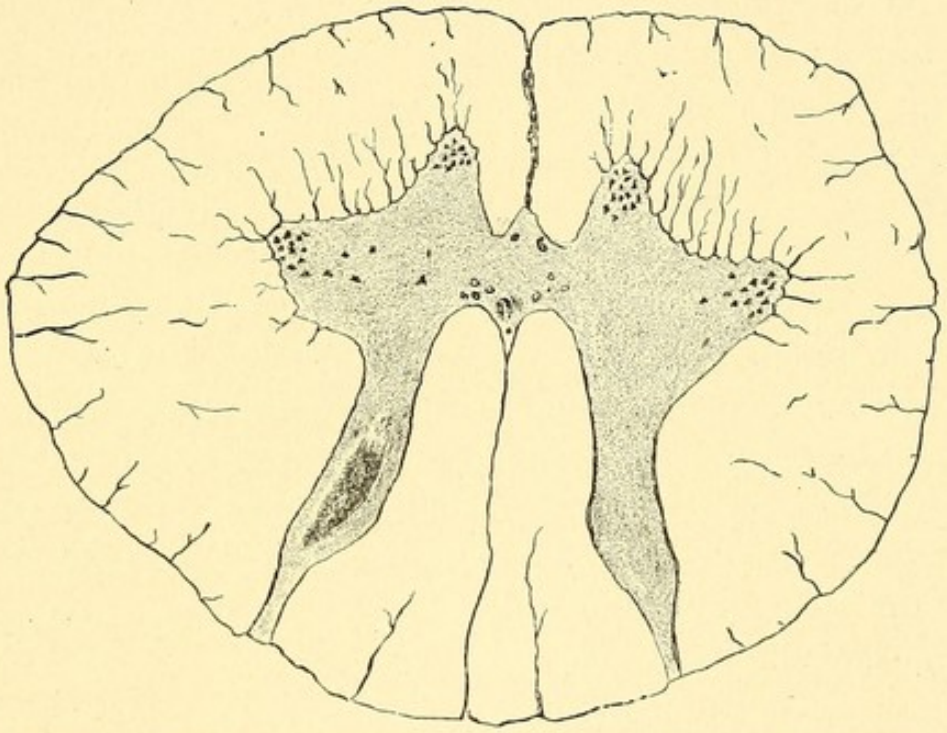


FIG. 21.—Showing inferior termination of the posterior column of blood in a case of traumatic hæmatomyelia. (City Hospital.)

and dilated, while at the same time there were many free red blood cells. By far the larger number of the red blood cells were collected in the gray matter, so that the outlines of the anterior and posterior horns and the gray commissure were brought out in sharp relief. In the other segments the red blood cells were still more closely confined to the gray matter, though they could be seen as scattered cells throughout all the sections. With the exception of the fifth and sixth segments, there was little inflammation or softening, and the nerve fibers were well preserved.

The amount of extravasated blood in this case being so large, it seems probable, although it can not be positively asserted, that the hæmorrhage resulted directly from the injury, rather than

that it was secondary to the softening and inflammation by which it had been surrounded.

II. Spinal Column Uninjured.

The cases in which the spinal cord is injured while the vertebral column remains intact form only a small proportion of the total number of injuries to the spinal cord. It is consequently in regard to them that satisfactory information is difficult to obtain and that our knowledge is most incomplete. Furthermore, as these cases are naturally less dangerous to life than the ones in which the cord lesions are secondary to bone lesions, many of the patients recover more or less completely, and the nature of the affection remains largely conjectural. They result from the same kind of violence as is active in causing fracture and dislocation of the spinal column. In most of the recorded cases of this variety of injury which have come to autopsy the lesion has consisted of hæmorrhage.

If hæmorrhage is the chief pathological factor, its occurrence may be explained by assuming that the physical shock was sufficient to cause a rupture of some of the blood-vessels of the spinal membranes or of the spinal cord. Hæmorrhage may occur without bone lesion (1) within the spinal canal, so that the cord is compressed by blood clots (hæmatorrhachis), or (2) into the substance of the cord (hæmatomyelia). Concussion or commotion will be discussed after these hæmorrhagic conditions.

1. *Hæmatorrhachis* may be illustrated by the following case, reported by Lambret:

> A driver fell from a cart, striking on his head. He immediately developed symptoms of flaccid paraplegia, with anæsthesia and loss of sphincter control, and as a result of his injuries died in a little more than twenty-four hours after the accident. At the autopsy both skull and spinal column were found to be intact, but there was a long extradural blood clot in the spinal canal which had compressed the spinal cord in the upper dorsal region.

2. *Hæmatomyelia* (hæmorrhage into the spinal cord) is one of the most interesting of the present problems of the pathology of the nervous system. Although it may occur either with or without fracture of the vertebræ, its chief clinical interest centers about the cases in which the spinal column remains intact, so that the hæmorrhage takes place independently of crushes of the cord. In hospitals and, less commonly, in clinics, cases are met with, ninety per cent of which are traumatic, in which the symptoms, by the suddenness of their onset and by their distribution, point to the existence of this condition. Many of the patients recover more or less completely, so that the diagnosis remains unsubstantiated by post-mortem demonstration. In others, death ensues so rapidly that pathological examination, although showing the anatomical condition, leaves undetermined what the ultimate clinical effects would have been had the patient lived.

There is considerable anatomical evidence, which is based upon the course which extravasated blood follows in the spinal cord, in favor of the view that central hæmorrhage, if not too extensive, may cause symptoms different from those of a transverse lesion. The gray matter is the least resisting portion, and its vessels are the most liable to rupture; consequently, in many of the cases of primary hæmatomyelia, the hæmorrhage occurs in and remains localized to the gray matter, although it may burrow for long distances up and down the cord.

The anatomical condition is shown by a case of Thorburn's, almost identical with the one already described, except that the hæmorrhage occurred independently of any fracture of the spine.

A man received a severe blow between the shoulders and became paralyzed in the muscles of the leg and abdomen and in the intercostal muscles. After three days of the ordinary symptoms of severe spinal-cord injury he died with congestion of the lungs.

At the autopsy, "in the muscles over the lower cervical and upper dorsal regions was some dark effused blood, but the vertebral column presented no evidences of injury. The membranes of the cord were quite normal, as was the external appearance of the cord itself, but on section there was found to be a dark, black hæmorrhage into the central gray matter in the lower cervical and upper dorsal regions. This hæmorrhage, which measured in its vertical extent from one and a half to two inches, was, in the greater part of its extent, situated centrally, occupying the whole of the central gray matter and extending but little into the white substance, which in its neighborhood was merely softened and of a faintly yellow tinge. At the lower part, for a very short distance, the hæmorrhage was limited to the anterior cornu of the right side, while the corresponding left horn appeared to be perfectly healthy; elsewhere the cord was firm, and presented no abnormity."

In cases in which the patients have partially recovered, the blood extravasation has been absorbed, leaving long cavities in the spinal cord. To such cavities Levier gave the name of "*Röhrenbildung*"; in an elaborate study of this condition, as yet unpublished, Van Gieson has individualized the sequelæ of hæmatomyelia under the term "*hæmatomyelopore*," as indicating a distinct disease of the spinal cord.

The cord may be hollowed out very quickly, as is shown by the following case reported by Parkin:

The patient was standing, on March 29th, beneath a lift, when the chain broke and the lift fell on his head. He was immediately rendered unconscious, and remembers no more about the accident. On admission to the hospital there were no signs of paralysis of the limbs, but in eight or ten hours after admission the left arm was found to be weak, and rapidly became paralyzed. On March 30th there was anæsthesia over both legs and arms, *said to be only partial*. He could move the right arm a little, but not the left. Could not pass his urine. Catheter had to be used twice a day. Motions passed involuntarily. Temperature subnormal. April 2d: Left arm completely paralyzed. Left leg stiff, but moves a little. No knee-jerk on left side. Plantar and epigastric reflexes present. April 6th: Left hemiplegia has become complete; anæsthesia about the same. April 11th: Died in about the same condition.

(The gradual extension of paralytic symptoms in this case was very similar to that frequently observed in hæmorrhage of the brain.)

Post-mortem.—Spinal column intact. No extra-meningeal hæmorrhage. On making section of the cord at a point two inches and a half below the apex of the fourth ventricle, it was found to be hollowed out, having about half an inch of healthy periphery. The cavity contained soft, grumous material, evidently altered blood, which escaped on section. The cavity occupied about three quarters of an inch of the length of the cord.

It still remains to be proved whether these acutely occurring hæmorrhagic cavities can cause the symptoms of syringomyelia, which is a disease characterized by chronic cavity formation in the gray matter. In cases in which the hæmorrhage is associated with a fracture of the vertebræ there is so much direct injury to the spinal cord in addition to the hæmorrhage, that the symptoms indicate a lesion of both the white and the gray matter; and even without bone lesion, if the blood is poured out so extensively that it seriously compresses the white matter, the clinical manifestations will be those of a transverse lesion. But when the white matter has been affected but little or not at all, and the hæmorrhage appears in the gray matter alone, it seems probable that if the patient recovers from the effects of the acute injury he may present symptoms which are the results of lesions limited chiefly to the gray matter. These symptoms will be found in the parts of the body receiving their nerve supply from the segments affected, and consist in localized paralyses of motion, with loss of reflexes and degenerative electrical reactions; atrophy and trophic changes and dissociated anæsthesia, of which the most common variety is a loss of sensibility to temperature and pain, while the sensations of touch are normally transmitted and perceived.

The symptoms of syringomyelia are usually more marked on one side than on the other, and there occasion-

ally is seen the Brown-Séquard type, which consists in paralysis of motion on one side of the body and of sensation on the other side.

Although no autopsy has as yet proved that a central hæmorrhage of the spinal cord can cause symptoms similar to those observed in syringomyelia, there are several clinical cases recently reported which speak very strongly for such a possibility. Of these, one by Minor may be mentioned:

A man, twenty years of age, previously healthy, fell a distance of seven and a half feet, and two days later was brought to the hospital. Examination showed no evidences of lesions to the skull, brain, or vertebræ, but disclosed symptoms which can best be explained by a small hæmorrhage situated in the gray matter of the left side of the spinal cord, and extending from the sixth cervical to the first thoracic segment. There was paralysis of the left arm and leg and paresis of the right arm, with diminution of electrical excitability in both upper extremities. The tendon reflexes were absent on the left side, but were only diminished on the right. Trophic disturbances were present about the shoulders. The sensory abnormities were the most interesting. With complete preservation of the sense of touch over the whole body and of the sense of pain and temperature over the left side, there was on the right side from the jaw downward a profound analgesia and thermo-anæsthesia. The patient recovered almost entirely from the paralysis and from all the other symptoms, with the exception of the loss of sensibility to pain, which remained as long as the case was under observation. (It will be observed that the association of symptoms in this case is very similar to that commonly observed in syringomyelia.)

In some cases it may be inferred from the motor symptoms alone, even when anæsthesia is totally absent, that a small hæmorrhage has occurred and has remained limited to the gray matter.

The following case, a patient at the Vanderbilt clinic whom I recently presented at a meeting of the surgical section of the New York Academy of Medicine, illustrates this:

The patient, who is seventeen years old, was a strong, healthy lad until the summer of 1895. Then, in diving from a dock six

feet above the water, he went too straight and, the water being only four feet deep, struck his head with force against the bottom. He did not become unconscious, but immediately lost power in all four extremities. He was pulled out of the water by other boys, and was taken home and put to bed. For three days he lay in bed, unable to move the limbs at all, with pain in the back of the neck and at the lower part of the spine. There was retention of urine and incontinence of fæces. "Pins and needles" in the left side only. No loss of sensation. No involvement of the face. After three days the right side began to improve and all the pain ceased. In four months the patient could walk, and has been gradually improving ever since. Now (April 7, 1897) there are weakness and stiffness in the intrinsic muscles of the hands of both sides, very much more marked on the left. The left hand is in the pen position through spasm of the interossei, and the interosseous muscle of the left little finger is paralyzed. Electrical reactions are normal except for the extensors of the fingers and for the interossei on the left side; in these muscles electrical excitability is diminished. There is a slight sinking in of the left interosseous spaces. The other muscles of the arms are normal. The right leg is perfectly normal, but the left leg is weak, and its muscles are in a condition of spastic rigidity, with increased reflexes and ankle clonus. The abdominal and plantar reflexes are lost on the left side. As the patient walks the left leg is dragged somewhat and circumducted in characteristic hemiplegic style. There are nowhere any changes in cutaneous sensibility, nor tremor, nor tenderness of the back, nor external evidences of any injury to the spine.

Thus the symptoms which remain are: Slight flaccid paralysis of the intrinsic muscles of the left hand, and to a very slight degree of the muscles of the right hand; spastic paresis of the left leg, no disturbances of cutaneous sensibility, no evidences of injury to the spinal column.

Such symptoms permit a very exact localization of the lesion. In this case there is every probability that there was a slight hæmorrhage into the gray matter of the first thoracic segment of the spinal cord, chiefly anteriorly and on the left side. An œdema or softening quickly developed, so that for a time the patient presented the symptoms of a transverse lesion. These soon went away, however, leaving a focal lesion which involved only slightly the anterior horn of the right side, but destroyed some of the anterior horn on the left side. The absence of sensory symptoms

would indicate that the posterior horn and the central canal were not involved. The hæmorrhage must also, either directly or indirectly, have impaired the conducting power of the left pyramidal tract.

In the cases which have been described here, and indeed, as far as I know, in all hitherto published cases of hæmatomyelia, the hæmorrhage has been focal. Although the blood may have burrowed for long distances up and down the gray matter of the cord, the columns of blood have been extensions from one bleeding point, and not distributed as multiple hæmorrhages.

Through the courtesy of Dr. T. J. Larkin, Assistant Pathologist to St. Francis' Hospital, I have recently had the opportunity of examining a case of hæmatomyelia essentially different from any previously described. In this case there was no single point of extensive hæmorrhage, but the spinal cord was dotted throughout its whole extent with microscopic collections of free red blood cells. Although the spine was fractured at one point, the hæmorrhages were multiple and occurred independently of the bone lesions, so that the case can be properly described in this section. The facts are as follows:

A workman, after receiving a severe fall, was picked up unconscious and brought to the hospital in deep coma and paralyzed in all extremities. He died in three hours from respiratory failure. At the autopsy the skull was found intact, but there was a hæmorrhage in the middle fossa. There was a fracture dislocation of the atlas and axis which had caused extensive mutilation of the second and third cervical segments of the spinal cord. There was no hæmorrhage in the spinal canal. Except at the point of compression, the spinal cord looked in every way normal. The membranes were not lacerated, the contour of the cord was perfectly preserved, and its substance was firm to the touch. On section it could be seen that the blood vessels were somewhat prominent, but the eye could detect no hæmorrhage either at the seat of injury or in lower levels. All the other organs appeared perfectly normal and free from hæmorrhage. Through a misun-

derstanding on the part of the dead-house attendant, the upper portion of the cord was thrown away, so that the microscopic examination was limited to the thoracic region and regions below it. Sections were made of every segment of the thoracic, lumbar, and sacral regions, and of the conus terminalis, which had been hardened in Müller's fluid and formalin, and which were then examined microscopically by means of the picro-acid-fuchsin method. The lesions found were generally distributed up and down the cord, and consisted of recent capillary hæmorrhages, which were so minute that they could not have been detected by the naked eye. No single hæmorrhage contained over two or three hundred blood cells, and the majority of them contained fewer. In some places there were only two or three free cells. In a few sections the blood lay in little cavities, but the most common distribution of it was in the form of an infiltration of a small number of blood cells between the neuroglia and nerve fibers. There was nowhere any tubular 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. Nearly every section contained the hæmorrhages, but no section contained very many, usually not more than two or three. The situation of the hæmorrhages 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 cord appeared normal.

Thus, to briefly resume, a healthy man fell with sufficient violence to break his neck and to cause an intracranial hæmorrhage without fracture of the skull. The force was exerted chiefly on the atlas and axis, as seen by the injury to these bones. But it was also adequate to cause throughout the cord punctate hæmorrhages, which were entirely unassociated with the severe local injury and which were probably due to the general violence to which the whole spinal cord was subjected. That injury can cause minute and multiple lesions in the spinal cord, *without fracture of the vertebræ*, has often been assumed, but, as far as I know, never before been demonstrated.

The proof of such a possibility is of great importance for the possible explanation of the pathology of the obscure cases of suspected spinal disease in which, after severe accidents, the patients give symptoms of serious impairment of spinal-cord function without definite signs of circumscribed injury. In the present case the lesions of vital centers completely masked any clinical symptoms

which the capillary hæmorrhages might otherwise have caused. Had the violence acted so that there had been no gross lesions, it might be that the small hæmorrhages would have given rise to symptoms indicative of multiple and minute foci of structural damage to the spinal cord.

Concussion or Commotion of the Spinal Cord.

As a designation for a pathological entity, the term concussion or commotion of the spinal cord should be limited to such conditions as may occur as a result of injuries to the back, or of general concussion accidents, and which have not been caused by pressure from displaced fragments of the spinal column or by hæmorrhage in the spinal canal or into the substance of the spinal cord. The time has gone by when intelligent physicians were deceived by the supposed resemblance between such general functional affections as hysteria or neurasthenia and concussion of the spinal cord. Concussion, however, continues to be invoked as an explanation for certain cases of organic paraplegia which are probably due to lesions much coarser than molecular disarrangement. The pathological condition with which it is most liable to be confused is hæmorrhage, and to attempt to distinguish between conditions so closely allied may at first seem to be of the nature of a pathological quibble. Recent acquisitions to our knowledge of hæmatomyelia have shown, however, that such a distinction is imperative if we are to get at the truth of the influence of trauma in the pathogenesis of the obscurer forms of spinal-cord diseases. Hæmorrhage usually occurs first in the gray matter, and generally remains localized there. If fibers are injured without hæmorrhage, as the believers in spinal concussion maintain, it would seem reasonable to suppose that the resulting symptoms would be different from those of hæmatomyelia.

Does such a condition as spinal concussion exist? In

other words, can any violence be sufficiently severe to cause direct injury exclusively to the essential elements of the spinal cord? To this question a positive answer is yet to be given. As is well known, lesions of all grades of severity may follow compression by bony splinters or by blood clots in the spinal canal, and intraspinal hæmorrhage may occur without there being any discoverable pathological changes in the nerve fibers, other than those explainable by the effects of pressure. But these are well-recognized conditions, and are the results of laceration and compression, and do not require to be explained by the assumption of concussion.

The question is not whether the fibers which compose the spinal cord are of different function and amenable to different laws than are nerve fibers situated elsewhere, but whether they are not so securely protected that an injury can not reach them without having first fractured or dislocated bone or having caused rupture of blood-vessels.

The nerve fibers of the spinal cord of course differ in no essential respects from those of the brain or of the peripheral nerves. They serve purposes of conduction and obey general physiological laws; yet a blow on the head, without causing fracture of the skull or visible damage to the brain, may cause symptoms of cerebral concussion, and a blow on a peripheral nerve is frequently followed by loss of conductivity in it without there being any gross injury to its structure. With the spinal cord, however, the case is different. The back often receives severe injuries without there resulting any symptoms referable to the spinal cord. The explanation of the fact that violence applied to the back rarely if ever results in symptoms of spinal-cord lesions which can not be explained by laceration or compression, is to be sought for in the immunity from any but the severest injuries afforded that organ by the protecting structures which surround it.

Accordingly, while we are not in a position to unquali-

fiedly assert that spinal concussion can not occur, we can say that if it occurs at all it is certainly one of the rarest of accidents to the nervous system—a fact which is amply established by experience, and which may be explained by the anatomy of the cord itself and of the surrounding parts. In by far the larger number of cases the spine is fractured, and the only cord symptoms are those referable to a nervous lesion in the immediate vicinity of the bone lesion. We never hear of cases of compression of one part of the cord and of concussion in a part higher up; on the contrary, spinal-cord injuries are characterized by the contrast between the conditions below the lesion and those above it.

The literature of spinal concussion is voluminous, but trustworthy facts are few.

In the cases which have been regarded as reliable examples of the condition by A. Westphal, and by Willard and Spiller, the vertebræ were fractured, and there were cord lesions with hæmorrhages in the immediate vicinity of the fracture. These investigators maintained that the cord lesion was the result of "concussion" and not of compression by loosened vertebræ. They based their opinions upon the absence of evidences of external bruising to the cord.

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 after it has been momentarily though seriously compressed. That slight and temporary compression is amply sufficient to cause a loss of spinal function is shown by the frequency with which complete paraplegia results from spinal operations when the spinal cord has been merely touched by the finger of the operator. To disregard the action of so well-recognized a cause as fracture, as is done by the above-mentioned authors, which was in their cases present, and to assume the action of another and unproved cause, appears to me unjustifiable.

Gowers is the warmest partisan of the theory of spinal concussion, and one is tempted to accept as final the opinion of so illustrious a neurologist. Yet the proofs which he adduces are far from satisfactory. Most of the cases he cites date from a period whose neurological contributions must be accepted with considerable reserve. In one of the more recent ones (Fischer) there was an extra-dural hæmorrhage, extending from the cervical to the lumbar regions, of which Gowers makes no mention. Gowers's own case is so interesting that it is to be regretted that it is not reported a little more in detail.

"A lady was severely 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." In the absence of fuller details, this case can hardly be accepted as proof of spinal concussion; the softening might equally well have been the late results of hæmorrhage.

The celebrated researches of Schmaus have been generally received as finally settling the question. Schmaus reports 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. As in all of these cases the patients had survived the injury and had lived with paralytic symptoms for several months, in order to discover the early stages of concussion, Schmaus induced paraplegia in animals by tapping them on the back with a hammer, without inflicting injury to the spinal column. As the result of his investigations, he 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. Hæmorrhage is not concerned in the ætiology, but may appear as an accidental or secondary accompaniment."

Two objections must be overcome before Schmaus's conclusions can be accepted, as proving that the human spinal cord can be concussed. One of them is, that of the human cases which he examined all presented cavities in the gray matter; and inasmuch as they did not come to autopsy till a long time after the injury, the probability is very strong that these cavities were not the results of gliosis, as Schmaus interpreted them, but were spaces which were left after hæmorrhages that had resulted from the original injury; and that the cases were not examples of concussion, but were forms of myelitis secondary to hæmatomyelia.

Schmaus's animal experimentation seems at first sight more conclusive. The animals became paraplegic, and after death, without any hæmorrhages, there was a swelling of the axis cylinders in the parts of the cord opposite the seat of external injury.

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, furthermore, are not identical with those of a subsequent observer, Bikeles, who, 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 hæmorrhage.

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, can not be regarded as having materially added to our knowledge of the subject. 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 evidence is totally insufficient for the question to be accepted as proved, although the weight of evidence is against the existence of the condition. When one considers the volumes which have been written upon the subject, the medical congresses in which discussion of it has held the prominent place, the enormous sums of money which have been paid for it in damage claims, and then turns to the few published cases which are advanced as proof, there seems to be a great disparity between the amount that is known about it and the amount that is talked about it.

If traumatisms ever act upon the spinal cord, so that its essential elements are injured without being compressed by foreign bodies or by blood, such a lesion is, from the published evidence, among the rarest of nervous pathology, and by no means entitled to the position of prominence it has so long occupied.

Symptoms of Spinal-cord Injury.—The symptoms of acute injury to the spinal cord are so generally familiar that they need be but briefly referred to. They result from loss of conductivity and from disturbance of the reflex functions situated in that organ. There are, accordingly, paralysis, almost always bilateral, with atrophy and degenerative electrical reactions, and, as a later result, contractures. At first the paralysis in the affected muscles is flaccid and usually complete. When the limb is grasped by the hand of the examiner there is total absence of all resistance; if lifted up and then let fall, it drops like a dead weight. The reflexes are almost always lost at first. Whether they remain absent, or return and become exaggerated, depends upon the altitude and extent of the lesion, ultimate exaggeration of the tendon reflexes being the rule in lesions above the lumbar enlargement. Priapism is frequent. The fæces may be passed involuntarily, or there may be constipation. Similarly there is retention or incontinence of urine. Trophic and vaso-motor disturbances, in the form of bedsores, cyanosis, and coldness in the extremities, are prominent symptoms in most cases.

Pain, either at the seat of the injury or radiating down the limbs, is in my experience only an occasional symptom. Anæsthesia has always been regarded as a cardinal symptom, but it is through the studies of Starr, Thorburn, Sherrington, Head, and still more recently of Kocher, that we have learned its true value. It has been conclusively shown that the distribution of anæsthesia due to spinal-cord lesions is entirely different from that caused by injury to the brain

or to the peripheral nerves, or from that observed in hysteria. This fact is of paramount importance for purposes of diagnosis between affections of the spinal cord and conditions by which they may be simulated, as well as for the localization of the site of the injury. In fractures or dis-

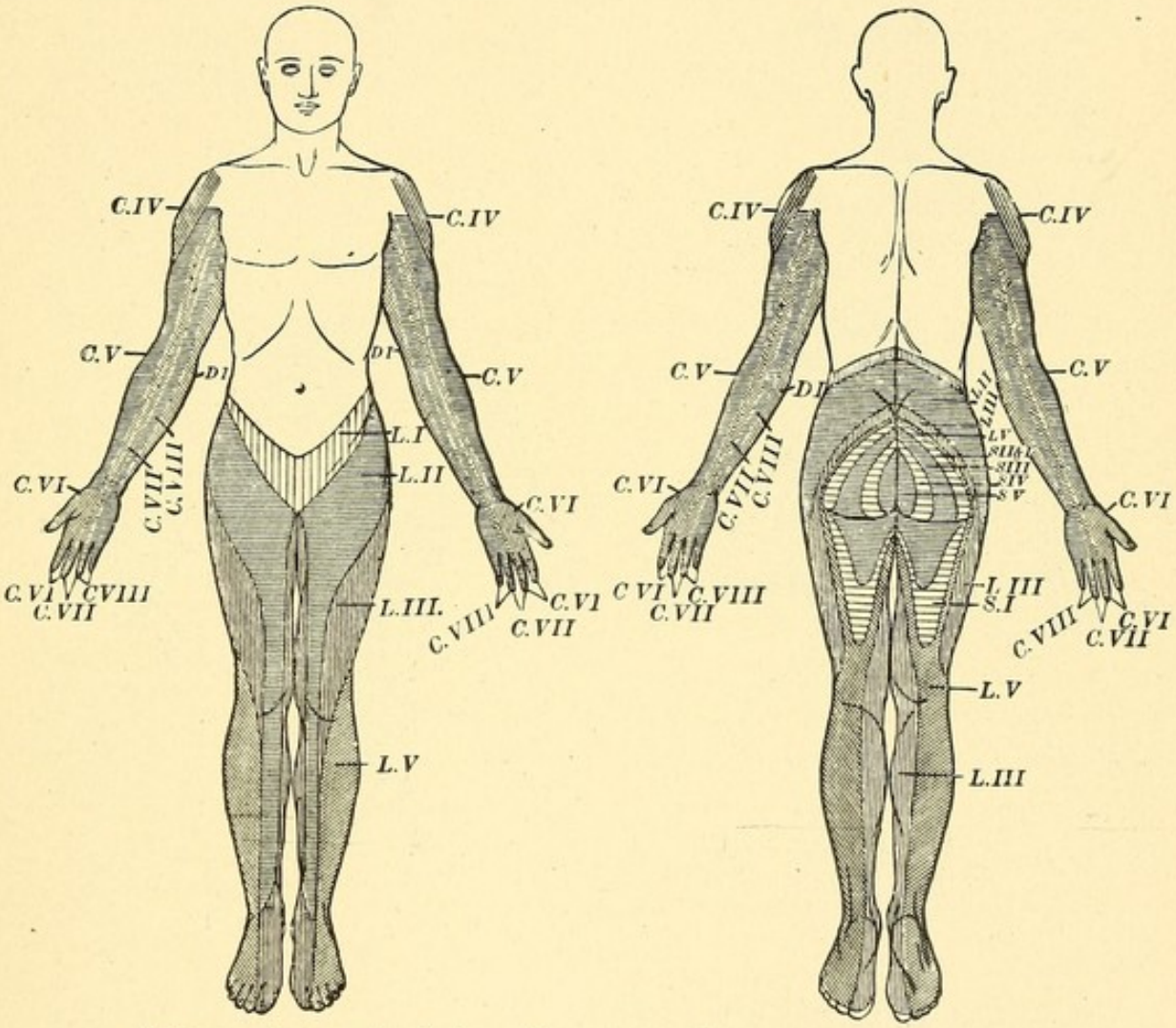


FIG. 22.—Sensory distribution of the spinal-cord segments as determined by spinal-cord lesions. (After Starr.)

locations of the spine it is usually impossible to determine from such external evidences as mobility or deformities of the vertebræ the exact seat of the lesion in the spinal cord. But when cutaneous sensibility is lost, as it almost always is in these cases, the topography of the anæsthesia may be confidently relied upon to furnish a means for the determi-

nation of the highest spinal-cord segment which has lost its power of transmitting sensory stimuli.

The sensory supply of the spinal-cord segments is so constant that it is possible to construct charts that indicate

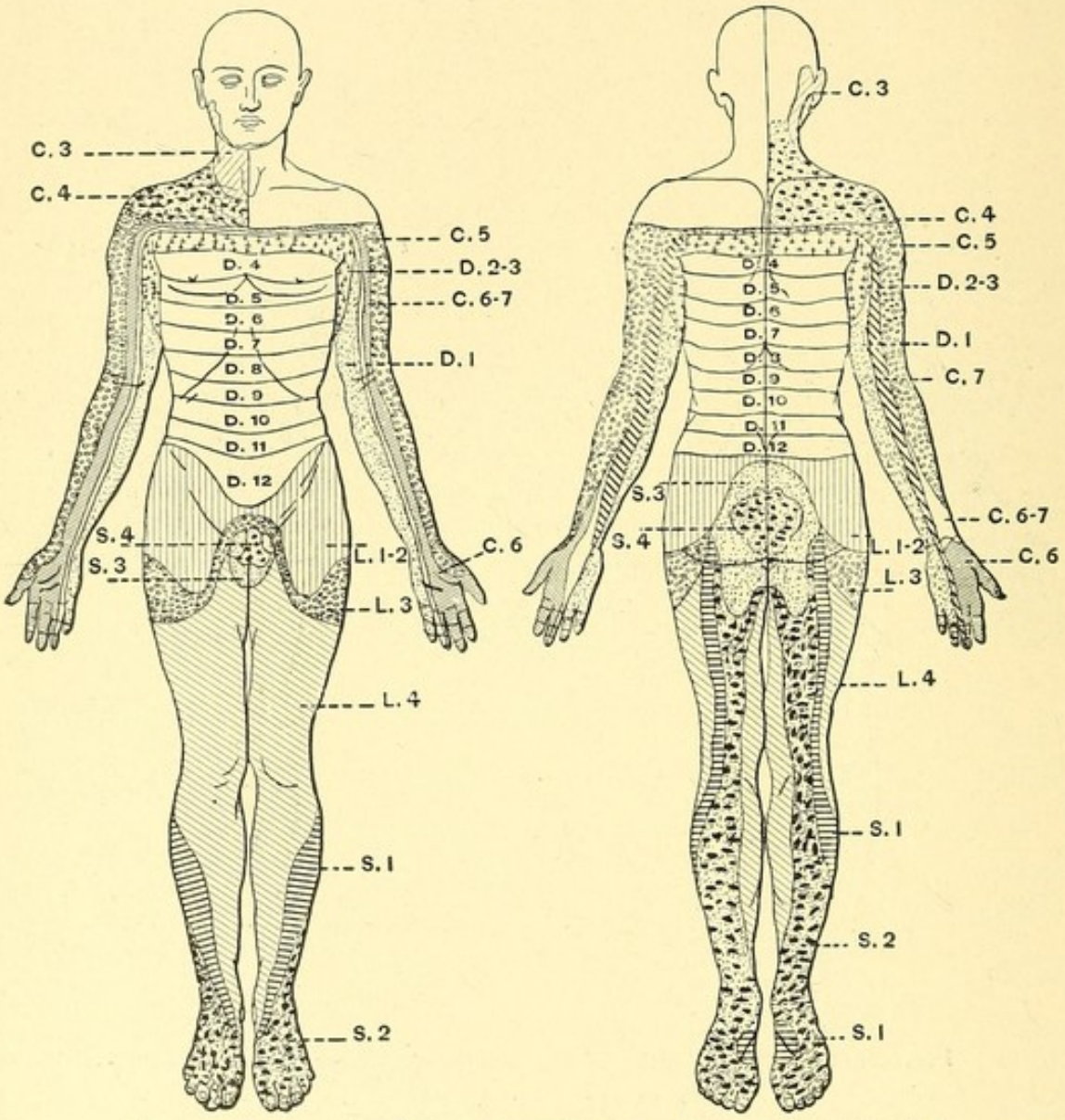


FIG. 23.—Sensory distribution of spinal-cord segments. (After Kocher.)

the distribution of the anæsthetic areas which may be expected in lesions of any one segment. Although the schemes as elaborated by different observers do not agree absolutely, the differences are unimportant and may be explained by the fact that the lesion rarely affects a segment

exactly according to its anatomical boundaries. Figs. 22 and 23 illustrate the areas of segmental distribution of anæsthesia as laid down by Starr and Kocher.

A transverse lesion of one segment involves a loss of conductivity in all the segments below it. Thus Fig. 24 illustrates the areas of sensory loss in a case of fracture of the cervical vertebræ, seen by me in consultation with Dr. P. R. Bolton. There was a transverse lesion at the eighth

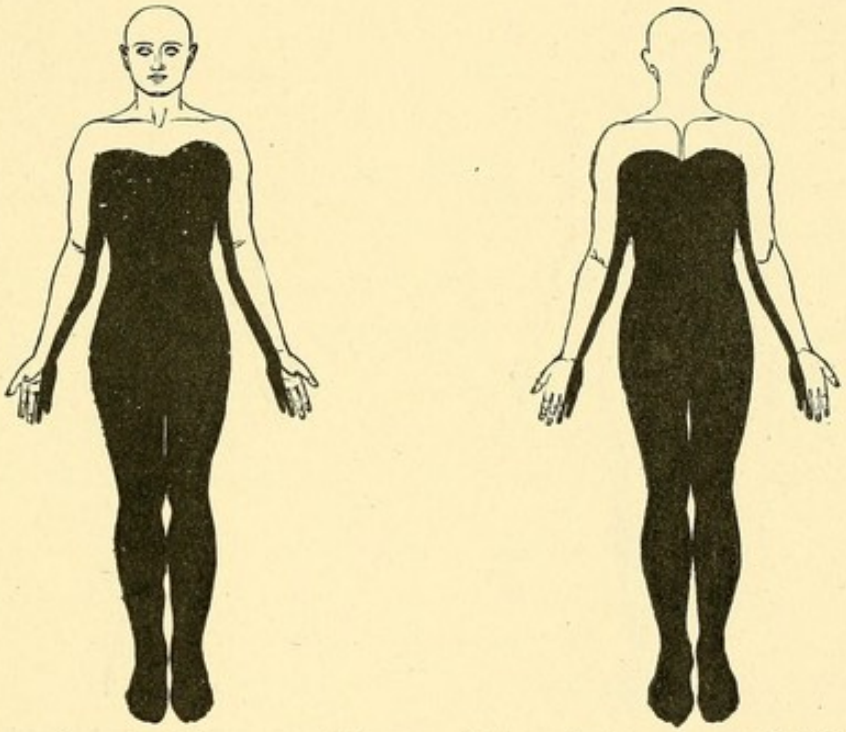


FIG. 24.—Anæsthesia in a case of fracture of the cervical vertebræ, with injury to the eighth cervical segment of the spinal cord. (Hudson Street Hospital.)

cervical segment, with anæsthesia in the distribution of that segment and in all segments below it. Similarly, in a patient of my own who is still living, the anæsthesia, as shown in Fig. 25, indicates a lesion which does not extend above the fifth lumbar segment. The characters of the anæsthesia after severe injuries are constant. With the exception that it is less at the upper limits, the anæsthesia is usually total and complete, so that sensations of touch, pain, or temperature are not perceived at all, and that the sense of position is lost.

The extent and distribution of the various clinical manifestations differ with the extent and character of the lesion. If the cord has been seriously bruised, or if extensive hæmorrhage has occurred, the resulting symptoms appear promptly and are then those of a transverse lesion. If, on the other hand, the damage to the spinal cord has been slight or partial, the symptoms may be somewhat delayed in appearing and may be of a more selective character. Anæsthesia is

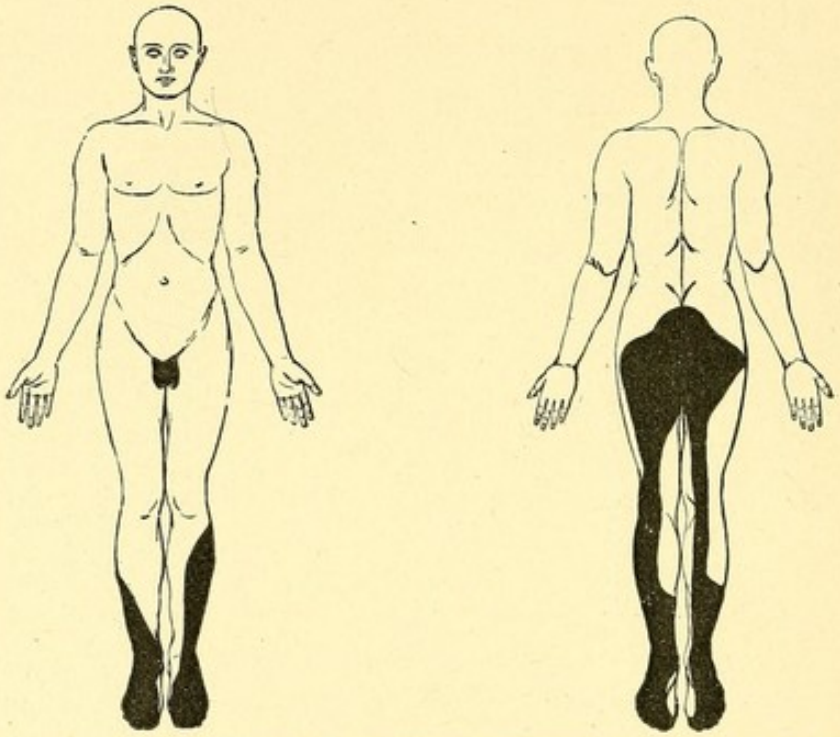


FIG. 25.—Anæsthesia in a case of a fracture-dislocation of the vertebræ, with injury to the fifth lumbar segment of the spinal cord. (Incurable Hospital.)

always present in severe injuries, but when the other symptoms indicate only a partial lesion anæsthesia may be entirely absent.

Thus, in a case seen in consultation with Dr. G. E. Brewer, the patient, a man, forty-five years of age, had fallen downstairs, hitting his back, and being generally shaken up. The left radius was fractured, but there were no external evidences of injury to the bones of the spine. The patient walked about for two hours after the accident, but then "felt his legs giving out." He was obliged on this account to stay in bed at first, but soon partially

regained the use of his limbs. At the time that I saw him, two weeks after the accident, he could walk with support; the knee-jerks were active, especially the right; the abdominal reflexes were absent; there was a large bedsore over the sacrum; there was incontinence of urine and fæces. The most scrupulous examination of cutaneous sensibility failed to reveal any abnormal variation of that function for either touch, heat, cold, pain, or the sense of position. By many this case would be regarded as an example of concussion of the spinal cord; but, for reasons already given, it seems to me more correct to make the diagnosis of compression from a slight dislocation or fracture, or from hæmorrhage.

The character of the lesion in injuries to the spinal cord may usually be correctly inferred if there are evidences of fracture of the vertebræ. It is when there are no external indications of injury to the spinal column that it becomes difficult to determine in what way the function of the spinal cord has become impaired or lost. We are then unable to distinguish between compression by bone, or hæmorrhage. We only know that hæmorrhage is particularly frequent in the cervical region, and that a gradual extension of symptoms may be due to blood finding its way up or down the cord.

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CHAPTER III.

INJURIES TO THE PERIPHERAL NERVES.

DEGENERATION and inflammation of the peripheral nerves may result from a variety of causes, but only those forms of peripheral nerve disturbance which are the effects of direct and immediate violence will be considered here. The pathological changes that occur in a nerve which has been divided, and so cut off from its trophic center, can not be detected with the microscope before the expiration of 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, which may occur in varying degrees as the result of crushes, cuts, blows, or strains, is degenerative rather than inflammatory, and it is only by the addition of infectious elements (as may occur in extensive crushes without external wound, or when there is a wound of the skin as well as injury to the nerve) 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.

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 these cases there may be restoration of function in varying degrees.

Loss of conducting power is the most important symptom of degeneration of nerves. The anterior and posterior roots of the spinal nerves conduct impulses of different kinds, but the union of the motor and sensory roots occurs so soon after their exit from the spinal cord that most peripheral nerves and all spinal nerves are mixed nerves, and consequently peripheral nerve palsies almost always present symptoms of disorder of both sensory and motor function. The trophic centers for the posterior roots are in the spinal ganglia, and so extra-medullary; those for the anterior roots are in the anterior horns of the spinal cord. Thus there are different degenerating paths for the two sets of root fibers. But these are differences which do not appear in the clinical picture, because simple traumatic lesions affect the nerves outside the spinal canal, and consequently external to the trophic centers for both sensory and motor nerves.

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 severer forms of traumatisms 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 conductivity 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 only observed 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. A gentleman was alarmed concerning atrophy of the interossei of one hand and beginning

main en griffe; the atrophy was very marked, and had all the appearance of a progressive muscular atrophy, but the condition was one of pressure palsy of the ulnar nerve, due to injury received while the patient was under ether for an operation for appendicitis a month previously. After injury to a peripheral nerve the muscles may occasionally very soon 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 after traumatisms affecting the muscles around the shoulder joint. It is fine, fibrillary, and very much intensified by movement and fatigue.

Sensory symptoms are not usually prominent in traumatic palsies of nerves; even when a nerve is divided anæsthesia does not invariably result. In very few of the isolated peripheral palsies which have come to my notice have there been any sensory symptoms other than 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 non-septic 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 different 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 disappear 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 (page 42). 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 hyper-excitability is short; after slight injuries it may continue for several weeks 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 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 not be complete. 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.

Vaso-motor and trophic disturbances are common accompaniments of severe injuries to nerves, and consist in the condition 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.

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. From the effects of severe lacerations the return to normal function is slower and more imperfect. Even from complete nerve section fairly good recovery is possible, if the ends become approximated, although it is always tedious.

It is impossible to give a certain prognosis in severe injuries to nerves until sufficient time has elapsed to permit us to be guided by electrical reactions.

The importance of paralysis of the cranial nerves, for purposes of cerebral localization, has already been mentioned. Some of them are, although infrequently, involved by injuries of the head and neck. Any of the superficial branches of the fifth nerve may be paralyzed by blows or cuts on the face. The pneumogastric has been injured, without life being lost, by stabs and bullets. Paralysis of the spinal accessory, with consequent loss of power in the sterno-cleido-mastoid or trapezius muscles, or in both muscles, is an occasional result of stabs in the neck. Lesions of this nerve may be associated with lesions of the hypoglossal or pneumogastric. The facial and hypoglossal are the most important of the cranial nerves frequently injured, and consequently they are the only cranial nerve palsies which will be described in detail.

Facial Nerve.—Traumatic peripheral 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 afterward, 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.

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 can not 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 can not 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 can not 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 occipito-frontalis to a marked degree and does not seriously interfere with the muscular movements in-

volved 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.

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 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 stylo-hyoid 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.

Cervical Plexus.—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. 26). 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 muscle. The prognosis of this condition is grave.

Posterior Thoracic.—This nerve supplies the serratus magnus. Although isolated paralysis of the posterior thoracic is not common, it may result from falls on the shoul-

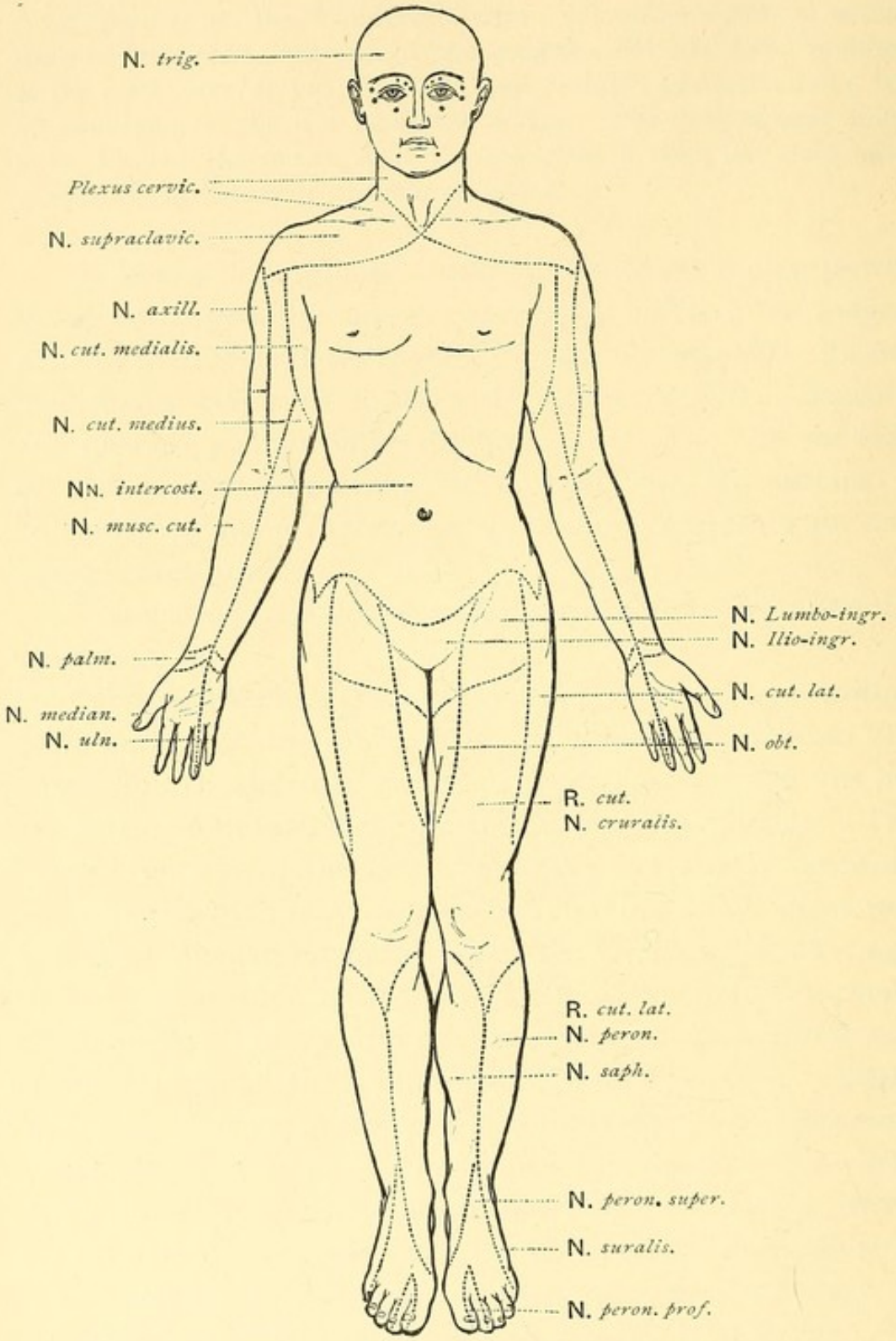
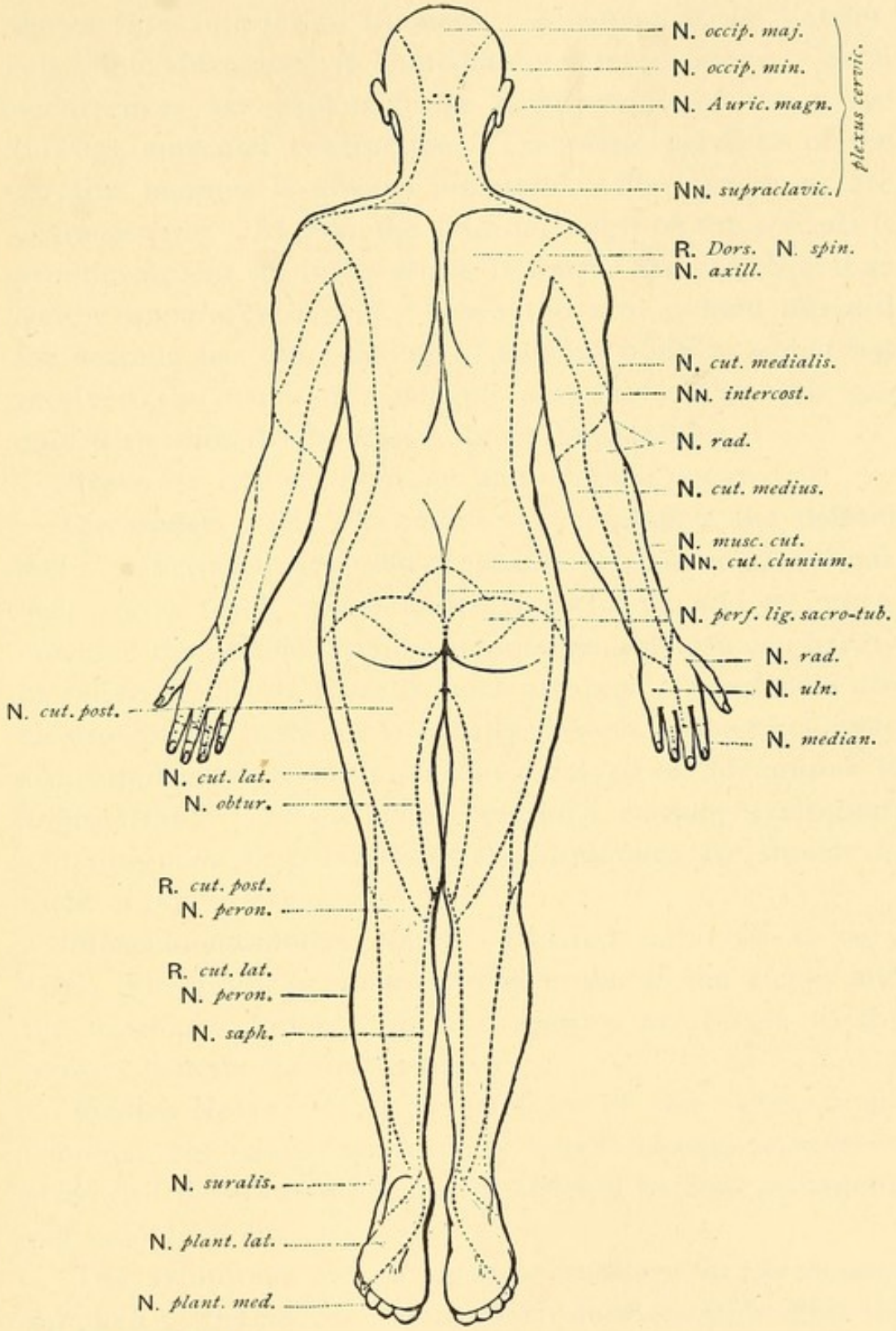


FIG. 26.—Distribution of peripheral



nerves for cutaneous sensibility.

der 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." The arm can only with difficulty be raised above the shoulder.

Recovery is always tedious and may not occur.

Circumflex.—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.

Musculo-cutaneous Nerve.—Isolated paralysis is very rare. The motor symptoms are referable to the biceps and the brachialis 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.

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 sublimis digitorum, and of one half of the flexor pro-

fundus 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 frequently associated with trophic disturbances (atrophy, glossy skin, etc.). The electrical reactions and prognosis have no individual characteristics.

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 the 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 the most

frequent. Unlike the musculo-spiral, 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 can not 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. 27), 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 palsy are chiefly subjective—numbness, tingling, and simi-

lar sensations. There may be some diminution of cutaneous sensibility, but complete anæsthesia is extremely rare, unless the nerve is inflamed as well as injured.*

Prognosis.—Ulnar palsy, when it occurs as a result of pressure from dislocated bones, usually recovers in a few

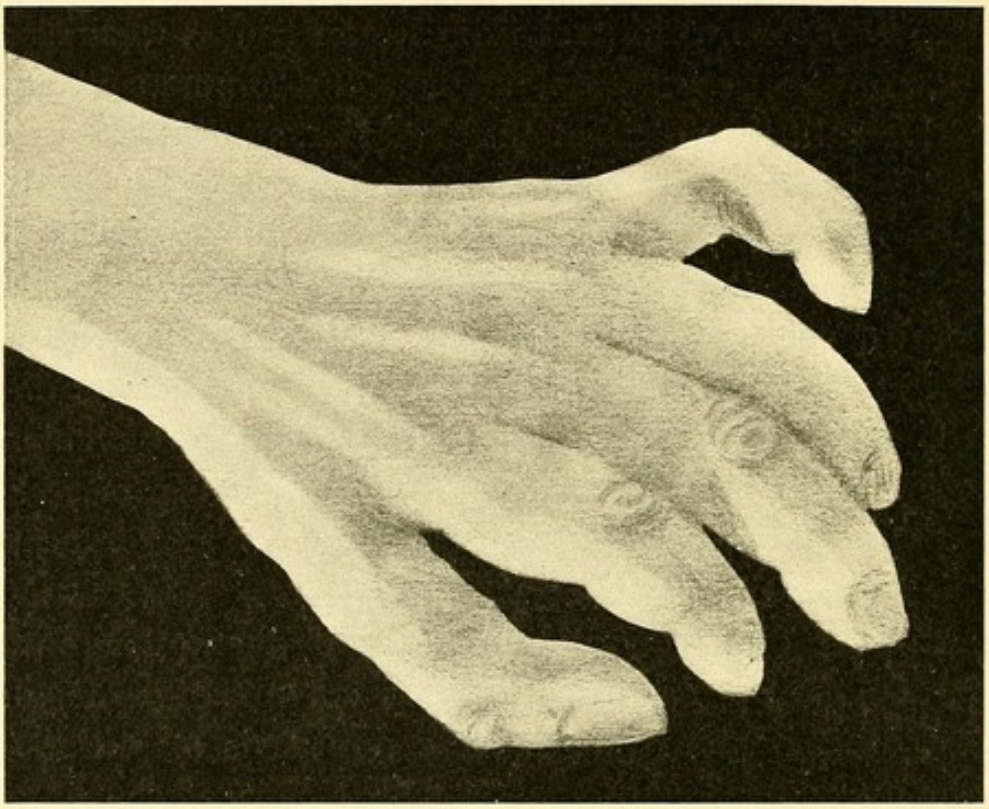


FIG. 27.—Beginning *main en griffe*. From an injury to the ulnar nerve. (Almshouse Hospital.)

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 re-

* 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 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.

serve. 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.

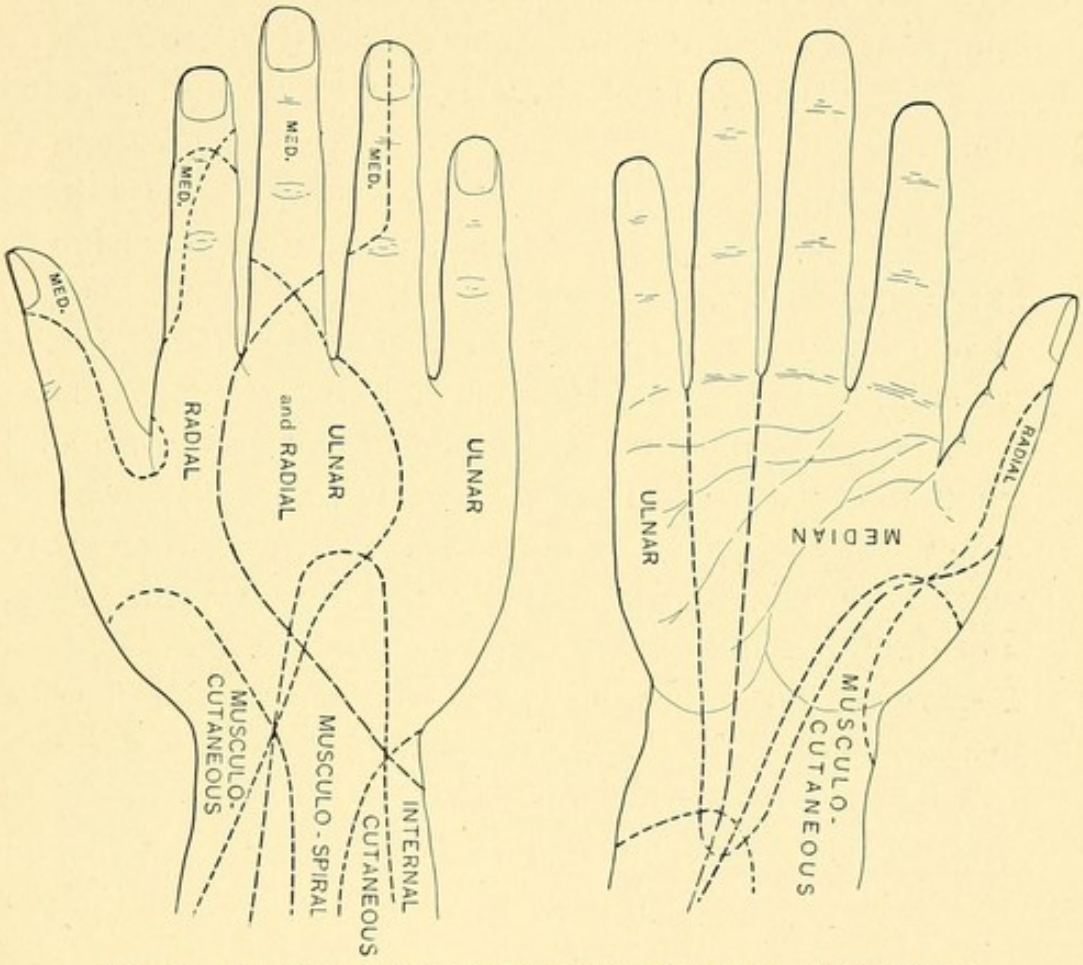


FIG. 28.—Distribution of the sensory nerves in the hand. (After Quain.)

Musculo-Spiral.—The musculo-spiral 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 musculo-spiral 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 musculo-spiral 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. Musculo-spiral paralysis has also resulted from falls on the hand and from sudden and forcible extension of the arm.

The *symptoms* are familiar to every physician. If the nerve is injured in the axilla or upper part of the arm, there will be paralysis 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 musculo-spiral paralysis causes the familar drop-wrist (Fig. 29). 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 at-

tempted. In this way the grip is very much lessened in force. By paralysis of the supinator longus the 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

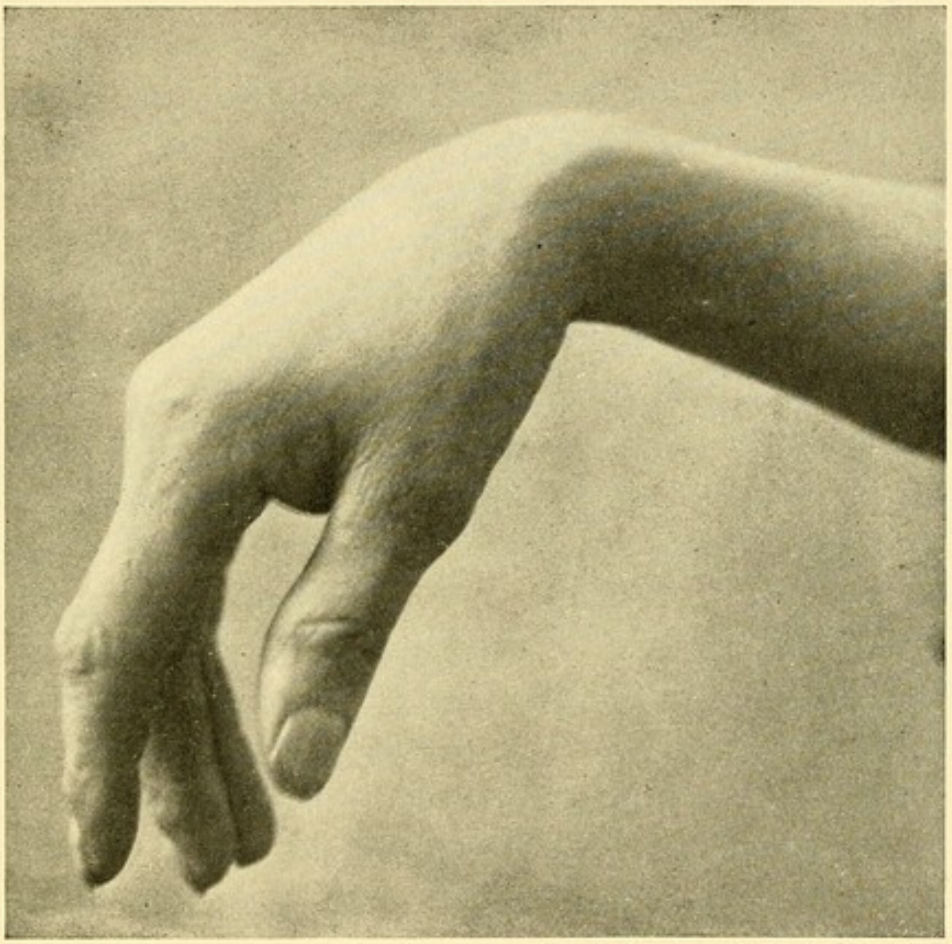


FIG. 29.—Drop-wrist, from palsy of the musculo-spiral nerve. (Vanderbilt Clinic.)

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, and so pronounced reactions of degeneration rarely occur.

The *diagnosis* of traumatic musculo-spiral 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 musculo-spiral 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 musculo-spiral, median, and ulnar nerves, which came on immediately after a fall on the palm of the hand.

The musculo-spiral and ulnar are the nerves most frequently involved together in injuries around the shoulder joint, although paralysis of the circumflex, or the median or the musculo-cutaneous, 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.

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 infra-spinati 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 infra-spinati 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 can not be raised from the side, and the forearm can not 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. 7); after injury disordered electrical reactions soon become manifest at this same point. A Van-

derbilt clinic case after a fall on the right shoulder complained 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.

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 good, in that the patients usually recover. In my experience, however, such recovery has always been tedious, extending over many months.

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 cilio-spinal reflex, sinking in of the eye, and flattening of the face. There are no vaso-motor 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 musculo-cutaneous nerves there was affection of the median and ulnar.

The paralyses of the lumbar and sacral plexuses are relatively infrequent, and result from causes similar to those enumerated for the palsies of the arm. When traumatic, they are usually the results of fractures or dislocations of the lumbar vertebræ or of the pelvis. It may be sufficient to recall that the lumbar plexus supplies the muscles concerned in flexion and adduction of the thigh, and to a slight extent in rotation outward, and in extension of the leg. Consequently the knee-jerk may be lost in paralysis of the lumbar plexus. The other muscles of the hip and thigh, and all the muscles of the leg, receive their innervation through the sacral plexus.

Injuries to the lumbar or sacral plexus, or to any of their ultimate branches, are followed by the same kind of symptoms as have been described under the palsies of the cervical and brachial plexus, and may easily be diagnosticated by remembering the distribution of the nerves.

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CHAPTER IV.

ULTIMATE ORGANIC EFFECTS OF INJURY.

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. It is often difficult to foresee what this termination is to be.

Of cerebral lesions the possible variety of final results is almost unlimited. Death may ensue instantly or after varying periods of time, and may be due to the destruction of vital centers; or the lesion of the brain may be the one additional straw which an already enfeebled constitution is unable to carry. If the patient lives he may recover from the immediate effects of the injury, but may remain permanently paralyzed as regards motion or special sense function, or be left with a serious and incurable impairment of the intellectual faculties. Dementia, associated or not with paralysis, is one of the most frequent of the permanent results of severe injuries to the brain.

This is illustrated by a man at present under my observation, who at the time of the accident was under the care of Dr. Bolton

at the Hudson Street Hospital. The patient was a fireman, thirty-one years of age, who fell through a hatchway, striking on his head and fracturing his skull. When brought to the hospital he was in a stupor, was very irritable and noisy, and presented evidences of paralysis of the left arm and left leg, and of ptosis on the right side.

The physical symptoms disappeared in a few weeks, but although it is now several months since the accident, and although he has regained good general health, the patient remains slovenly, unable to care for himself, or to talk intelligibly or to understand what is said to him. He sits quietly in his chair, oblivious to his surroundings, and though able to perform all movements perfectly well, he does not move unless told to do so. He is at present completely demented, and hopes for his recovery do not seem bright.

Any of the psychoses may develop after severe injuries to the head, whether the skull has or has not been fractured. Mania and melancholia are the most common of the traumatic psychoses. They may appear weeks or months after the accident, and usually present no symptomatic differences from like psychoses of idiopathic origin. Their prognosis is always grave, although some cases, in which there was a depressed fracture of the skull, have been benefited by operation. Neurasthenia and hysteria may also be the result of severe injuries to the head, although these disorders more commonly follow accidents of less gravity.

It is often impossible in an individual case to foresee what the ultimate effects of severe head injuries are to be. In the cases in which the evidences of extensive hæmorrhage or laceration of the brain are pronounced it is usually apparent that the patient has not long to live; but when the symptoms do not unequivocally point to a fatal lesion there are no means by which it may be foretold how complete recovery is to be or if it is to occur at all. Abscess of the brain may develop after injuries to the head in which the only immediate symptoms were scalp wounds or in which any abrasion in the cranial covering was not discoverable.

In acute traumatism of the spinal cord the prognosis is bad, both as to life and as regards recovery. Lesions of spinal-cord structure are irreparable; if nerve fibers or nerve cells are destroyed, they are not replaced. The only hope, in cases of traumatic paraplegia, that the symptoms may entirely disappear, depends upon the loss of function being due to some lesion such as extramedullary hæmorrhage, which temporarily abolishes function without seriously disturbing structure. When the cord is completely cut across there is no chance for recovery, and the patient usually dies in a few days as a result of the injury. In many cases the lesion is not completely transverse, and the patients regain a certain amount of muscular power, and control of the sphincters may be restored. Anæsthesia frequently disappears almost entirely. In general, any one who sustains an injury to the spinal cord, if he does not die, remains more or less of a cripple for life, although this rule has its exceptions. The nearer to the brain the lesion is situated the worse is the prognosis for life. Injuries to the fourth cervical segment, or to segments above it, are almost immediately fatal. The mortality of all cervical lesions is very high. The prognosis is somewhat better when they are in the dorsal region, and is most favorable for those of the lumbo-sacral region and of the cauda equina.

When there is a time interval between the receipt of any injury to the nervous system and the appearance of the symptoms which may be alleged to be its direct result, a causal connection is much more difficult of proof. The question of the traumatic origin of *tumors* of the nervous system is even more obscure than when new growths develop in other parts of the body apparently as the direct consequence of injury. 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. As far as the nervous system is concerned, the evidence for the traumatic origin of tumors can not be regarded as anything more than suggestive.

Thus in a recent autopsy held at the Almshouse upon the body of a woman, seventy-one 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.

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 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.

* Vierteljahrschrift für gerichtliche Medicin, January, 1896.

According to views recently expressed by Strümpell * as to the pathology of *multiple sclerosis*, the genesis of that

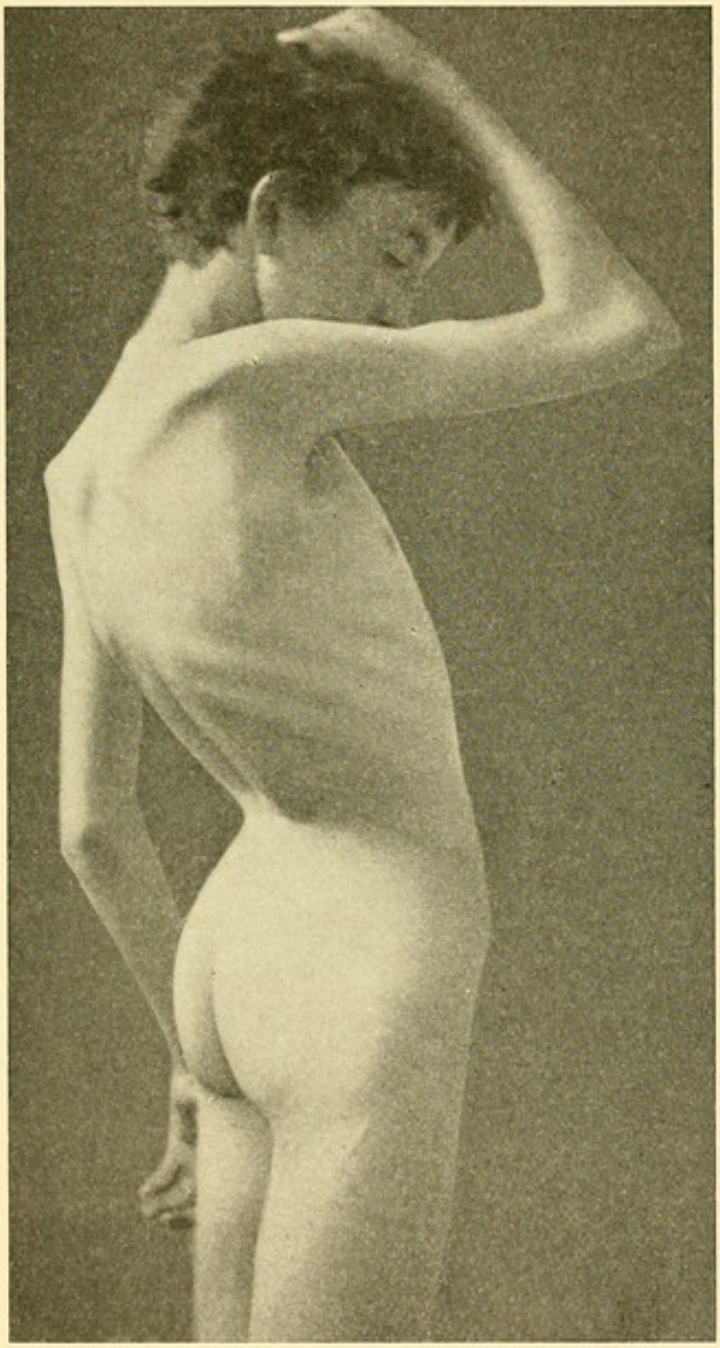


FIG. 30.—Deformity of the back caused by syringomyelia. (Vanderbilt clinic.)

disease depends upon a morbid activity being given to abnormal islets of neuroglia tissue scattered throughout the

* Neurologische Centralblatt, 1896, No. 21.

cerebro-spinal axis. If this theory is correct, the ætiology of multiple sclerosis is probably similar to that of tumors; yet clinical substantiation that multiple sclerosis arises from traumatic causes is still slight. This subject will again be referred to.

So little is known about the pathology of *acromegaly* (Fig. 31), 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.

The relationship of traumatism to diseases of the nervous system is necessarily so obscure that it can not be even approximately decided except for those disorders which are comparatively frequent. Thus, although there is no doubt but that spinal hæmorrhage 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 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.

There is, however, a group of comparatively common chronic degenerative diseases of the nervous system of which traumatism is regularly spoken of as a cause, and which, by reason of the symptoms being often observed for

* Münchener medicinische Wochenschrift, 1895, xlii, 14.

† Char. Annalen, 1895, Jahrg. xx.

‡ Deutsche medicinische Wochenschrift, February, 1897.

the first time after some accident, have important forensic relations. These diseases are *epilepsy, general paralysis of the insane, locomotor ataxia, progressive muscular atrophy, and paralysis agitans.* In some of them the occasional occur-

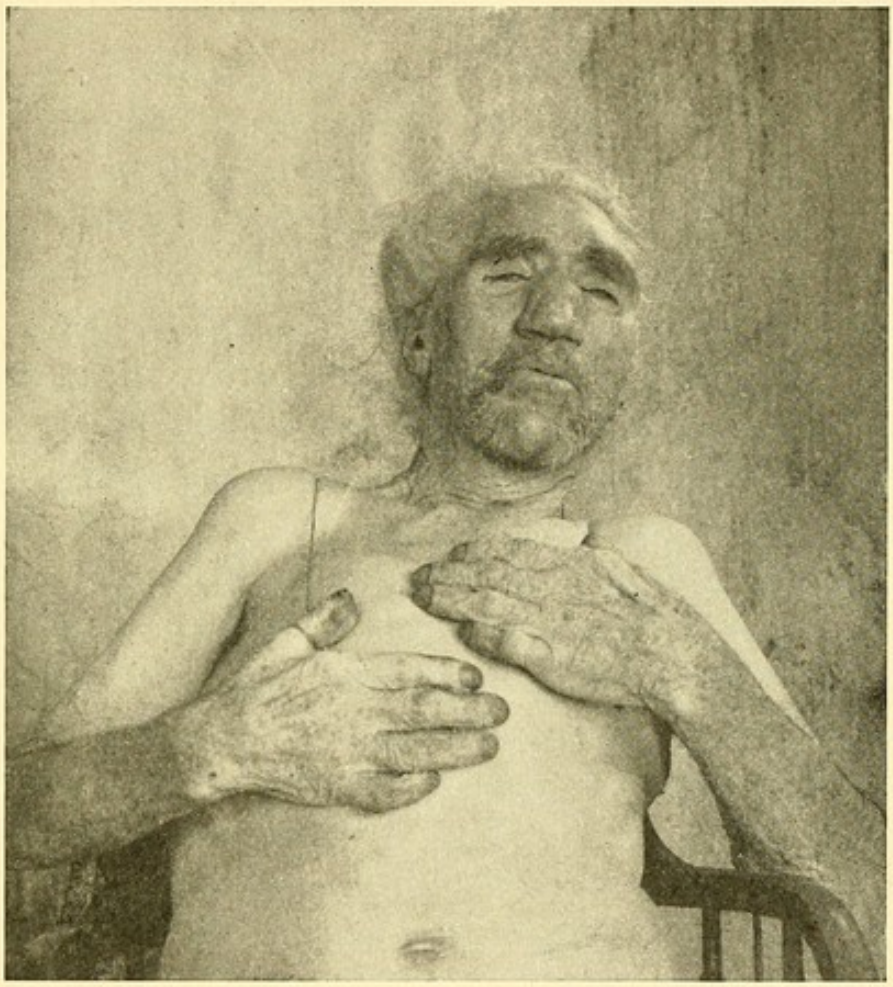


FIG. 31.—Acromegaly. (Photograph of a female patient who died in the Incurable Hospital.)

rence of trauma as a sole cause seems probable; in others the possibility of a traumatic origin is extremely doubtful, as is indicated by the absence of well-authenticated cases which have occurred in this way. Epilepsy is the only one of the group which can be positively proved, both clinically and pathologically, to owe its development, in certain cases, solely to trauma.

So many factors must be taken into account in a consid-

eration of these diseases and their relationship to injury that they require individual description.

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 occuring in one of every five hundred of the population. In the larger 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 trivial traumatisms, 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

have been the direct result of injury. But whatever the true cause of epilepsy may eventually be shown to be, our actual knowledge permits us to recognize as traumatic only such cases as develop within a reasonable length of time after a severe injury, or as indicate by their symptoms that there has been an injury to a limited portion of the brain.

Our knowledge of the *pathology* of traumatic 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 traumatic 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 traumatic epilepsy predisposition plays a subsidiary rôle. General convulsions sometimes develop soon after a slight injury to the head or to other parts in persons who are hereditarily neurotic, or

whose nervous systems, as a result of alcoholism or other forms of chronic poisoning or degeneration, have become less resistant to injurious influences or stimuli. In such cases it is often impossible to determine the causal value of the injury. The general character of the convulsions would seem to indicate that no one portion of the brain had been traumatized, but that the whole cerebral cortex was in a condition of latent disease that required only a slight disturbance of the cerebral equilibrium to become active. We must know very much more about cerebral pathology than we do at present before it will be possible to decide whether such cases can properly be regarded as the direct results of injury, or whether epilepsy would have eventually developed as a consequence of some one of the mishaps of daily life to which every one is exposed.

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.

It seems, however, extremely improbable that wounds or injuries by which the brain is unaffected, occurring to persons who were not already doomed to present manifestations of the disease, can be regarded as causes of epilepsy. At the Vanderbilt clinic, among the records of over one thousand cases, injury to the head is the sole form of physical traumatism which has been alleged as active in the production of the disorder. It is possible that injuries to other parts may be followed by epilepsy, as injury may be followed by any disease, but in such cases the influence of the traumatism must be regarded as inconsequential.

As regards epilepsy, severity is the most important element of a head injury. 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 afterward 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 a 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.

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 a traumatism 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 arm 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 traumatic 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.

Many cases have been reported as traumatic 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 traumatism 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 traumatism 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 if 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 traumatic 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 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 hæmorrhage 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 typical symptoms of traumatic 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 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 general, 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.

Traumatic epilepsy usually becomes in time an exact clinical counterpart of the idiopathic variety, and so we may expect to find in it the various psychical equivalents which are occasionally encountered when the disease arises from unknown causes. Still, while somnambulistic attacks, impulsive acts, temporary amnesias, and similar phenomena may occur, they do not constitute a prominent feature of the traumatic variety of the disease. Many of the morbid impulsions of cerebral neurasthenia and some of the symptoms of traumatic insanity are epileptic in character, being transitory and coming on without definite warning; but unless they have been or are associated with convulsive

seizures, we are not justified by our present knowledge in calling them manifestations of epilepsy.

The *course* of traumatic 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 very materially 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.

Diagnosis.—To establish beyond reasonable doubt a causal relationship in any case between an injury and the development of epilepsy may be very easy, or it may be practically impossible. In persons previously healthy in whom the skull is shown to have been fractured, there will

rarely be any question as to the relationship between the traumatism and the subsequent convulsions. But if external evidences of fracture are not demonstrable, there are several considerations which must be taken into account before it can be stated that the epilepsy is in all probability traumatic:

1. 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.

2. 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.

3. The injury itself is naturally the most important causal element. If it results in hemiplegia or monoplegia, the causal connection is plain; or if the seizures are local, and so indicative of localized brain lesion, there will rarely be any question as to their traumatic origin. Localized muscular spasm as a symptom of idiopathic epilepsy, although it has been observed, is extremely rare, and it would probably never occur in a way for it to be mistaken for the local spasm of traumatic epilepsy. When the seizures are general, whether they depend upon injury or not must be decided by those other considerations which have already been mentioned.

4. The time elapsing between the receipt of injury and the first epileptic manifestations can not be considered as a definite guide in diagnosis. The statistics are derived from those cases which have been operated upon, and in most of them the paroxysmal symptoms have been delayed for a

few weeks or a few months after the accident. 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 in other respects less typical of the traumatic form of the disease.

5. 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. Traumatic epilepsy, on the other hand, is almost always one of the penalties of the exposure to accident which is incident to the active periods of life. Of course, children may receive severe head injuries, and grown men sometimes develop idiopathic epilepsy. But the two diseases belong so essentially to different periods of life that traumatic epilepsy in the child can with justice be diagnosticated only after elimination of all other causes and a scrutinizing study of attendant circumstances. Conversely, the probability is greater that the convulsions that appear after injury to an adult who has never had fits, arise as the direct consequence of cerebral injury.

It may be said, in conclusion, that no case of epilepsy in which there is any question of operation or of medico-legal inquiry, should be accepted as traumatic without the seizures having been witnessed by a physician or by some person trained in the observation of the symptoms of disease. Such observation can best be carried out in hospitals.

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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 of the difficulties which are met with in investigations relative to the traumatic origin of tabes. General paresis, like tabes, 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 non-traumatic 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 absolute certainty that the traumatism 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 one hundred thousand 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.

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 symp-

toms 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.



FIG. 32.—Showing characteristic facial expression in the early stage of general paresis. (Vanderbilt Clinic.)

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		
Mendell and Schnell....	75 per cent	Quoted by Oebeke.
Binswanger.....	49–72 " "	
Ziehen.....	33–43 " "	
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 per cent syphilis probable.	
In France it is given by		
Régis.....	70–90 per cent.	
In America it is given by		
Peterson.....	60–70 per cent;	
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 15 to 20 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 pre-existence 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, occurs 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 to injurious influences.

The causes which are 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 disease 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 fifteen cases of injury to the head in seventy-six cases of general paresis, in which the causes "were clearly made out." In eighty male cases, Krafft-Ebing found cranial injury to be the cause in six. 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 forty-three cases of general paresis in one hundred cases of injuries to the skull. In five hundred cases of insanity Schläger ascribed a traumatic ætiology to forty-nine, of which seven developed general paresis.

Gudden reports forty-five cases in which a history of head injury was prominent. In six of these the trauma had occurred in childhood, years before the symptoms of the general brain disease. In eighteen the trauma and the paralytic symptoms were separated by a period varying from six months to twenty years; and in twenty-one the paralytic symptoms developed in direct sequence (few months) upon the head injury. The accident was generally accompanied by loss of consciousness, and in four cases the skull was fractured. Of one hundred and seventy-five cases reported by Hirschl, trauma had exerted an influence in 7.4 per cent.

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 building. 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 afterward, 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 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 forgetful, 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 witnesses 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 penal-

ties 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 occurrence of the traumatism, 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 any one suffering from it.

If the disease is sufficiently advanced to be diagnosticated, there is no more reason for the physician to accept

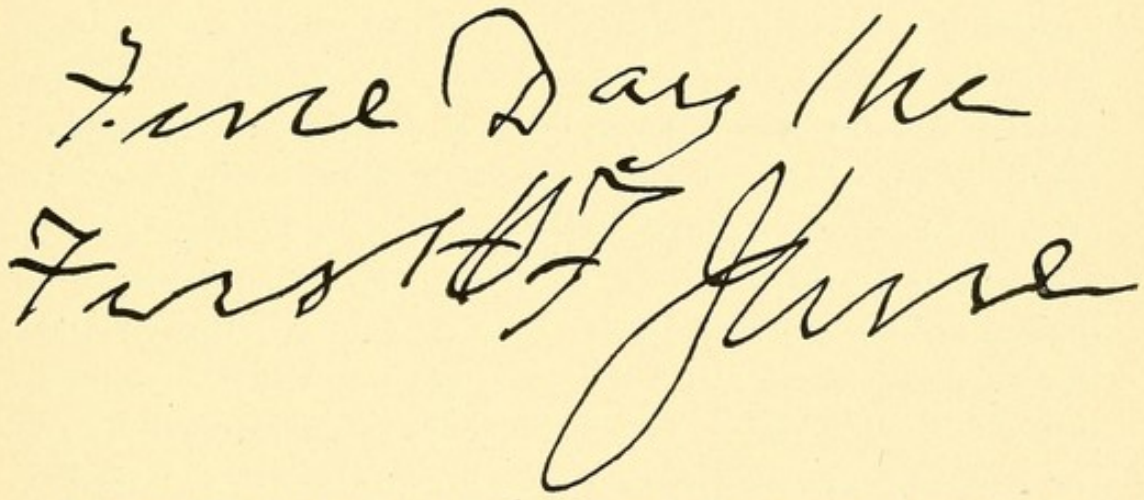


FIG. 33.—"This is a fine day for the first of June." Handwriting of a patient in the early stage of general paresis. (Vanderbilt Clinic.)

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 the traumatism was responsible for the development of the disease.

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 many head injuries, especially if the skull has been fractured or there has been intracranial hæmorrhage or brain laceration, there develops a condition of delirium and excitement, at the subsidence of which the patient passes into a condition of more or less complete 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, although demented, for many years. Some of them present such striking similarities to the cases of general paresis that it is only by the observation of them for considerable periods of time that it can be decided that the underlying anatomical process must be essentially different.

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 pre-existed. The hereditary predisposition has been proved by the family history, by congenital syphilis, or by the stigmata of degen-

eration. The acquired predisposition has consisted in the results of preceding syphilis. Of all these factors syphilis is the most important. In view of the fact that syphilitic processes become much more active after traumatisms, injury is often regarded as of minor ætiological importance if it is soon followed by paretic symptoms in an individual who is undeniably syphilitic, although he may never have presented any cerebral symptoms before the accident. This is hardly just. 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 ascribe to the traumatism 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 afterward he was discharged to 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 has 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 two thousand clinical histories he is forced to believe that traumatism is not to be regarded as a sole cause of the disease. In over one hundred autopsies made on paretics he has only once seen anything which seemed to be correlative to traumatism. In one case, over the region of the right gyrus occipitalis superior, there was a depression, about the size of a silver twenty-five-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 of traumatism being 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 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, but 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, 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 for the existence of traumatism as a sole cause of general paresis, but they are the best evidence we have.

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 only to be accepted after scrupulous inquiries have eliminated all of the many opportunities of error.

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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 for so long a time and has become so advanced and so extensive that it is impossible to tell when it had its beginning. Until we know which nervous element is first affected, we can not hope to know very much about the ætiology of the disease. 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 degeneration of the posterior columns in the spinal cord, with which are usually associated 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 a selective action on certain portions of the spinal cord or of its nerve roots, and that the tabetic degeneration is in reality

syphilitic. Although this view may be correct, it 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.

Pre-ataxic Period.

The clinical difficulties which are met with 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 the most chronic of all 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 become apparent is not definitely known, but certainly for a considerable length of time. A patient of mine, fifty-two years of age, who had syphilis thirty years ago, has been in the pre-ataxic 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 alcoholism, and the tabetic symptoms were discovered during the course of a routine examination.

The pain is usually the cause of the patient seeking medical advice, 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,

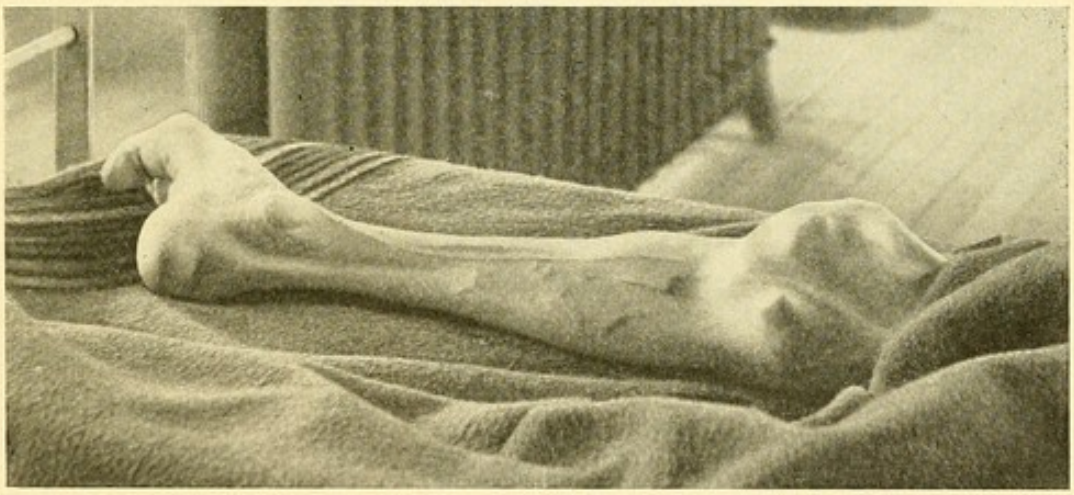


FIG. 34.—Charcot joint, the alleged result of traumatism. (Incurable Hospital.)

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. 34 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 of connecting paths 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 diagnosis of tabes can not 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

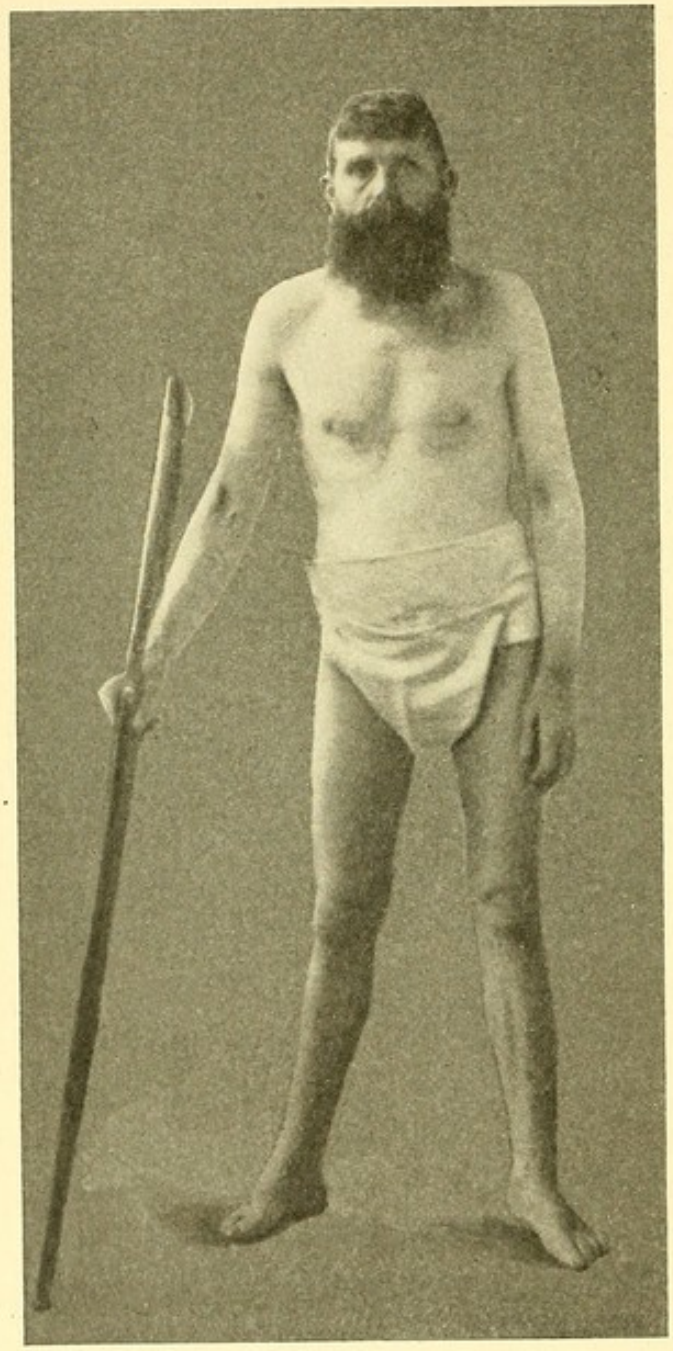


FIG. 35.—Attitude of static ataxia. (Vanderbilt Clinic.)

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.

Fig. 36 shows the tactile anæsthesia chart of a patient whose only other symptoms are slight unsteadiness of gait and loss of knee-jerk.

That the early symptoms of tabes are often overlooked is a fact of common experience, for it occurs not infrequently

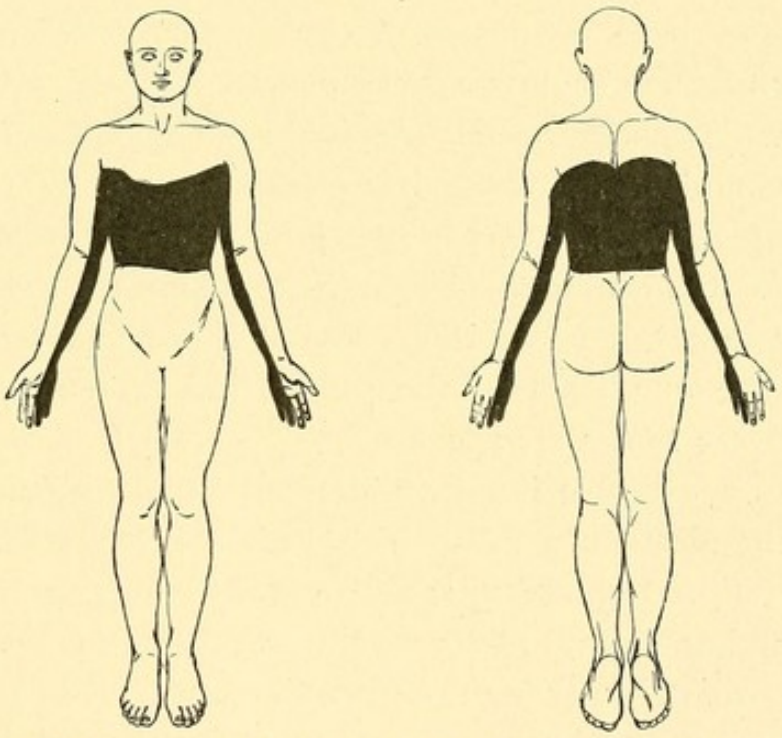


FIG. 36.—Tactile anæsthesia in a patient in the early stage of locomotor ataxia.

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 pre-ataxic stage, have consulted a physician for other troubles, but unless at that time he complained of subjective nervous 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 were 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 were at that time free from beginning posterior spinal sclerosis, because in the routine examination for life insurance there is no attempt at examining for nervous symptoms which are not immediately apparent. On the medical examiner's blank the color of the eyes is noted, but there is no space left for statements concerning the condition of the pupils, and there are no questions relative to the knee-jerk.

That an examination by a competent physician, who is also on the alert for nervous disease, is necessary before any one can be pronounced as 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 any one 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 pre-ataxic 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.

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 ones 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 one hundred and eight cases observed in Leyden's clinic, Storbeck finds only twenty-three who are surely syphilitic, twenty-two which are doubtful, and sixty-three 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 can not 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 with syphilis is large, compared with which the number of persons who develop tabes is ex-

tremely small; 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 to satisfactorily prove any particular predisposition to the spinal disease, or to any disease 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 earlier degenerative 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 only predisposed the spinal cord to degeneration, 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 determine 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 can not 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 two hundred and eighty-one 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 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 one hundred and eighty-five full clinical histories of the disease which are recorded in the books at the Vanderbilt Clinic, in three 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 his 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 his bed 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

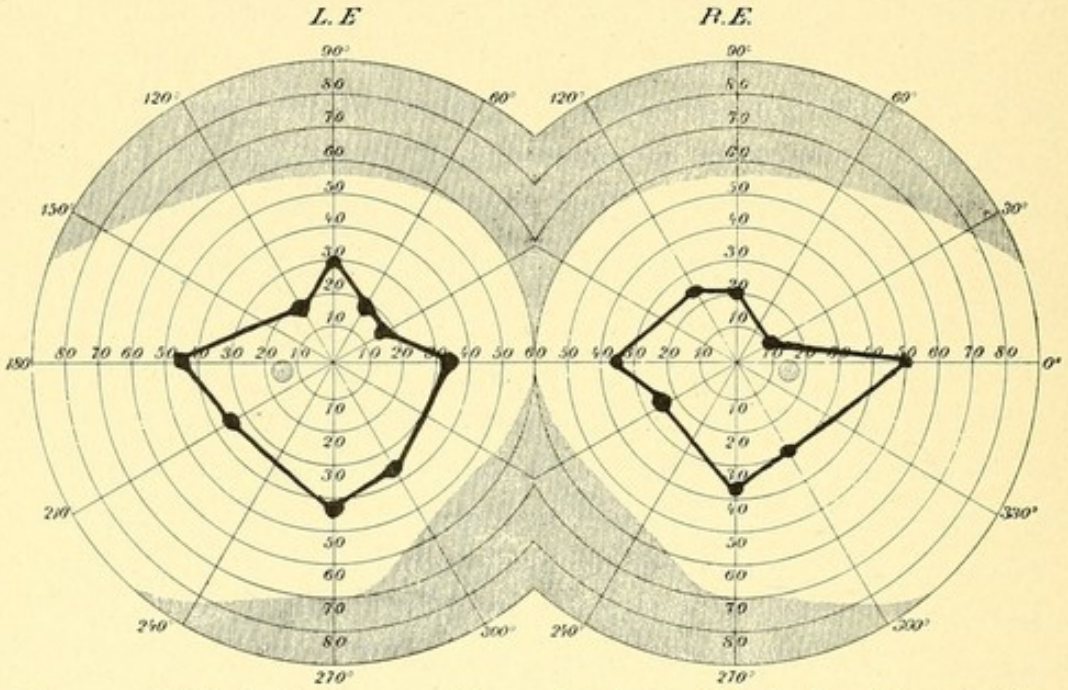


FIG. 37.—Field of vision in a case of locomotor ataxia which developed immediately after an injury. (Vanderbilt Clinic.)

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., $\frac{20}{40}$; L. E., $\frac{20}{50}$ (for field of vision see Fig. 37). 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 can not 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 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 forty cases of tabes which had been published as traumatic, and rejected them all as being insufficient as evidence, although he admits that twelve 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 pre-existed. If this can not 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 can not 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 three 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.

Thus the matter stands as it did in 1895, and we can do no better than to repeat the conclusion of Prince, that "the view that locomotor ataxia may be caused by traumatism *per se*, irrespective of a direct lesion of the cord, is not sustained by the published evidence thus far adduced. If such a relation exists, further evidence is required before it can be accepted."

It seems as though Prince would have been justified in going even still further, and in saying that, in view of the large number of published cases of tabes, compared with the few cases reported as traumatic, and in the total absence of any verification of traumatism as a cause, it is improbable that injury or shock stand in any direct causal relation to the disease.

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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 so as to include atrophies which are progressive, although different in course and pathology from the original disorder described by Duchenne. There are at present two distinct and well-recognized groups of 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.

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 55.) 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 anatomo-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 has followed traumatism. The bladder and the rectum are rarely involved in this disease, and there is almost never 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 type Duchenne-Aran 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 mus-

cles. 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 waste, 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.

A less frequent type of progressive muscular atrophy is the "peroneal," called, after its earliest describers, the Tooth or Charcot-Marie type. Its pathology is obscure, as it still remains to be conclusively proved that it depends upon disease of the anterior horns of the spinal cord.

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 failure 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 relation of traumatism to progressive muscu-

lar 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 were 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. In nerve injuries without severe wounds or fractures, 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, as indicated in Chapter III, 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 symptoms of progressive muscular atrophy are nearly alto-

gether 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 were 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 due to traumatism: 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 the arms and both the 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 only bend the head 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. Deg-

lutition 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 Prof. 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 was 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.

At the time of the examination the patient was a good illustration of the peroneal type of progressive muscular atrophy. 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 hæmorrhage, or an outbreak of localized syphilitic inflammation in the spinal canal in a way to exert pres-

sure 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 has been under our observation at the clinic for many months, developed progressive muscular atrophy three months after a blow on the back:

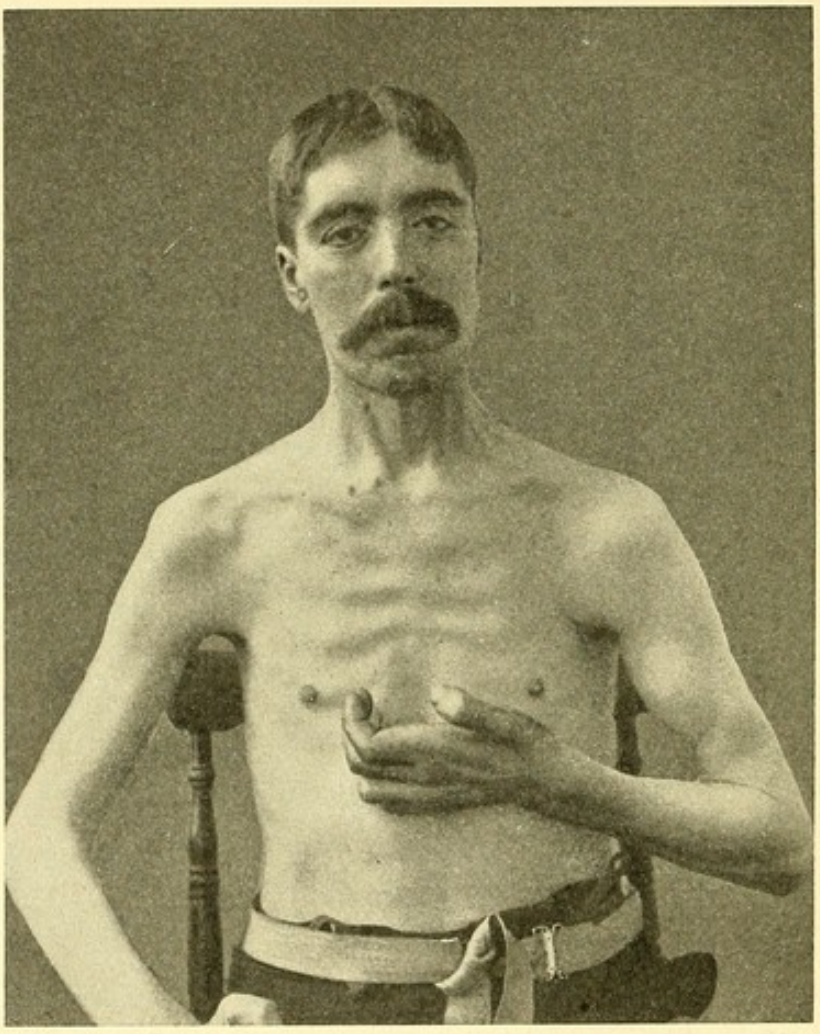


FIG. 38.—Case of progressive muscular atrophy which began three months after an injury to the back. The photograph shows the wasting of the shoulder and chest muscles and the *main en griffe*. (Vanderbilt Clinic.)

The patient (Figs. 38 and 39) 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 symp-

toms of syphilis. He was married at twenty-nine years of age, and has one healthy child. His wife never had any miscarriages.

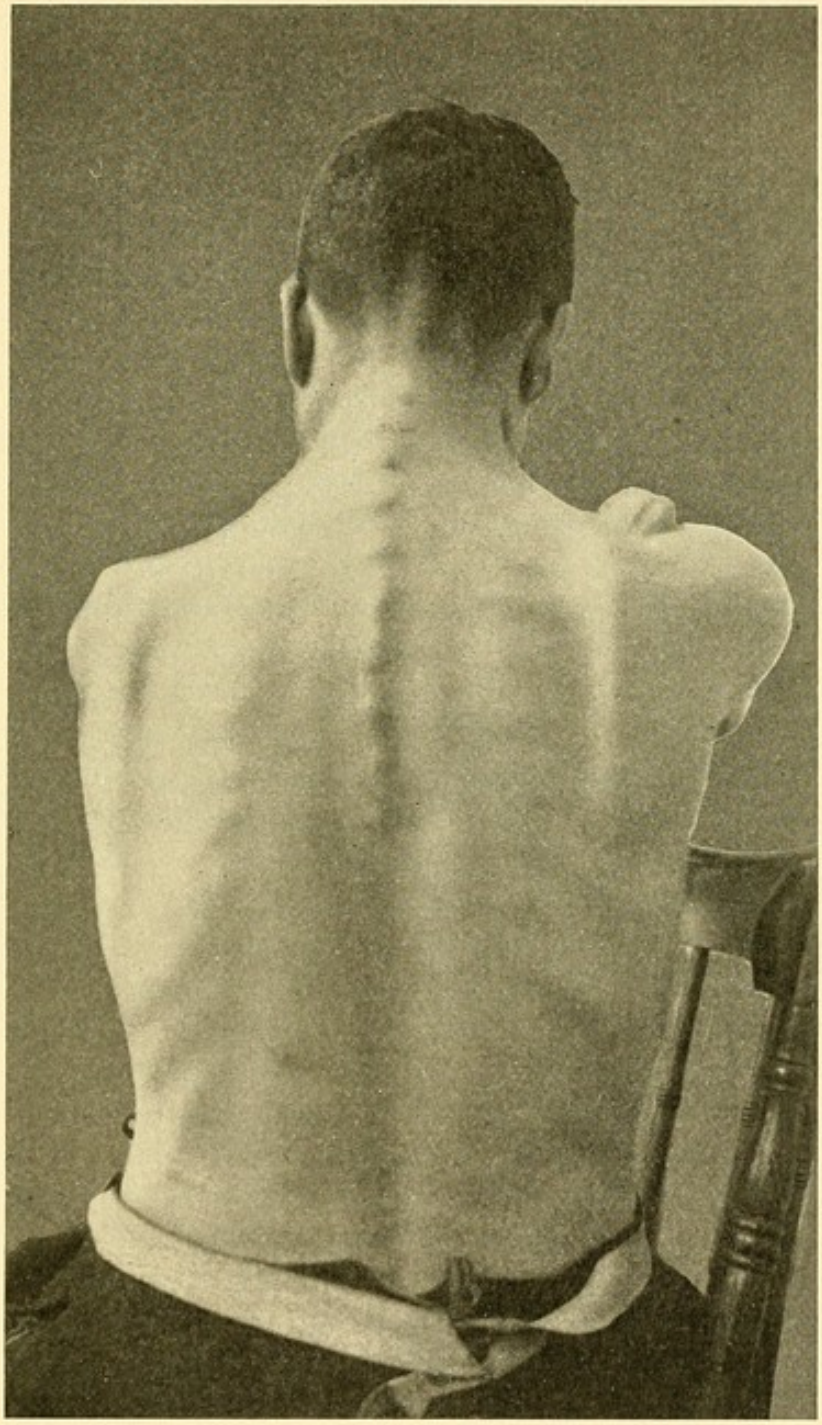


FIG. 39.—Same patient as in Fig. 38, showing wasting of back muscles.

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 eight hundred 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. 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 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 co-ordinated 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, can not 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.

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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. Since, however, disease occurs almost altogether in persons who have reached the age when such degenerations often occur without causing the symptoms of any distinct disease, 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 the disease to be classified without impropriety among the organic nervous diseases.

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 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.

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. 40) 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. 41), 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

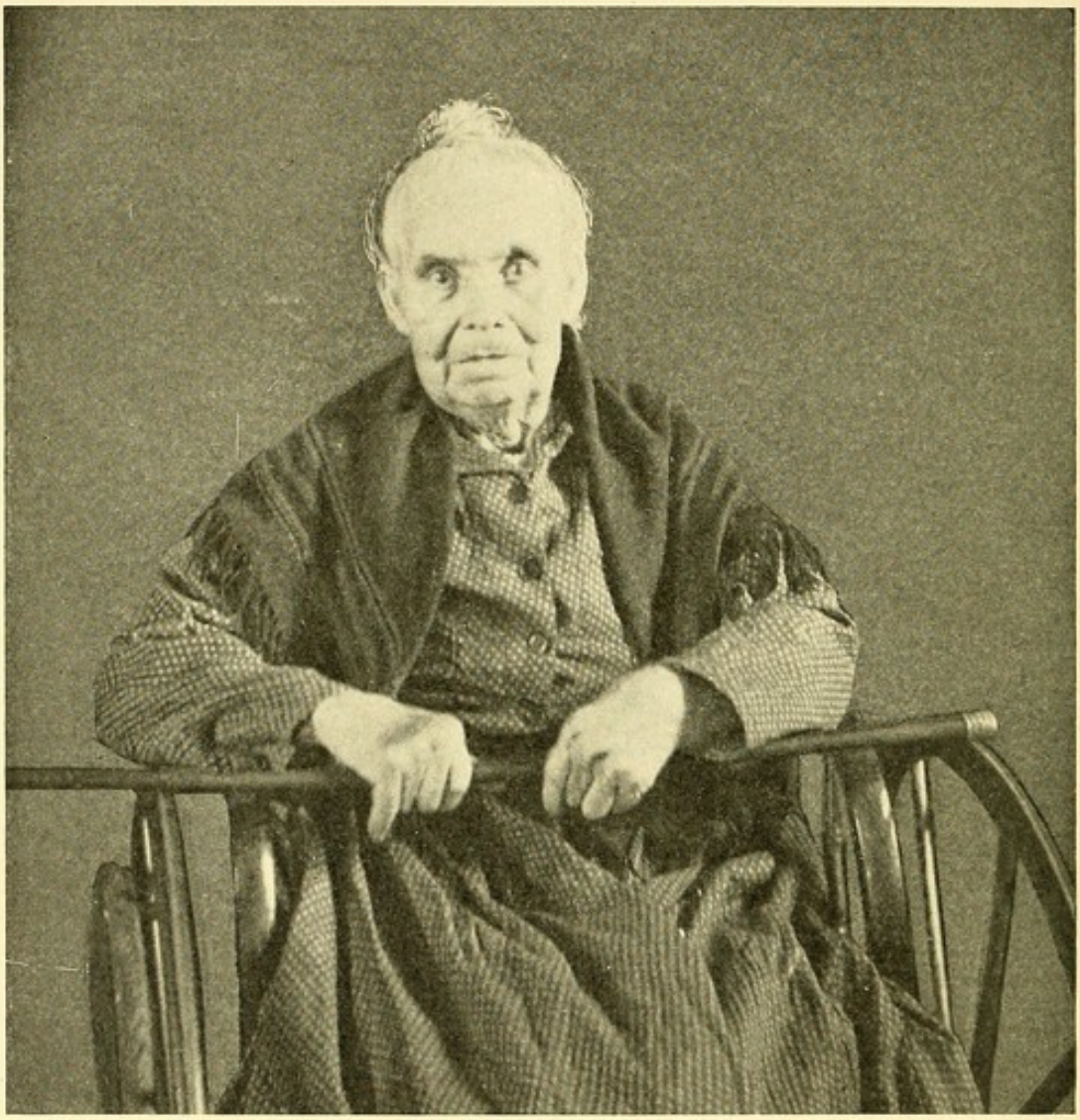


FIG. 40.—Showing facial expression and position of the hands in paralysis agitans. (Incurable Hospital.)

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 sys-

tem 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.

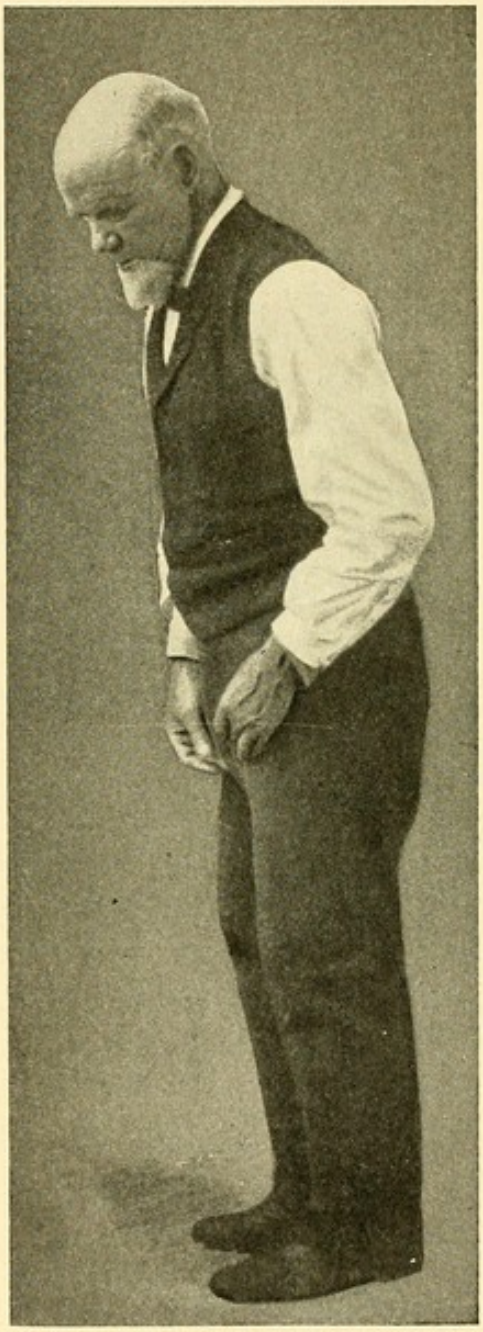


FIG. 41.—Showing characteristic attitude in paralysis agitans. (Vanderbilt Clinic.)

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 per-

sisted, passing to the leg, and afterward 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 one hundred and thirty-nine cases observed at the Vanderbilt clinic, fright is assigned as the sole cause in seven. On the other hand, of twenty-six 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 twenty six cases, general concussion, six; wounds (stabs and cuts), seven; burning and freezing, one; sprains, twists, and fractures, four; contusions, eight.

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 non-traumatic 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—

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 acting similarly to progressive muscular atrophy when it occurs in those parts which had previously been affected through spinal injury.

Thus in a case described by Walz:

Male, aged sixty-one years, 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 hæmorrhages 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 ap-

peared 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 the 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 eight; soon, or in a few days in seven; after one to eight months, in seven; and after one to four years, in four.

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 only appear in persons whose nervous systems are already deteriorated. While the truth of such a theory can not be denied, it can not 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.

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PART II.

FUNCTIONAL EFFECTS OF INJURY—THE NERVOUS DISORDERS WHICH MOST FREQUENTLY FOLLOW RAILWAY AND ALLIED ACCIDENTS—THE TRAUMATIC NEUROSES.

CHAPTER I.

INTRODUCTORY CONSIDERATIONS REGARDING THE HISTORY, NOMENCLATURE, PATHOLOGY, CAUSATION, AND SYMPTOMS OF THE TRAUMATIC NEUROSES.

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, traumatic neurasthenia, and traumatic hystero-neurasthenia. 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 hæmorrhage 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 fifty-three cases only seventeen 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 hæmorrhages 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 any one 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 most 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 were 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 6, 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.

Rigler, 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, hæmorrhages, 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 railway accidents, in which the factors of concussion and fright are so intimately mingled, more frequently caused 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, 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 hæmorrhage 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 severe 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 bed sores 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 tabulated the ultimate results of two hundred and thirty-four 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 perma-

nently 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 these disorders. 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 paralysis.

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. Vaso-motor 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 forty-two 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 associated. 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 can not 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 recently has cast doubt upon the value of many of the individual symptoms of the traumatic disorder. 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.

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 hæmorrhage, 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 "traumatic 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 can not 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 goître, 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.

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 non-traumatic causes. When owing its origin to injury or fright, it is more commonly associated with pronounced neurasthenic symptoms, such as are usually present in non-traumatic hysteria occurring in men. Although traumatic neurasthenia and traumatic hysteria frequently occur together (traumatic hystero-neurasthenia), for purposes of convenience they will be described separately.

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 goitre, 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 has 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 litigated 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.*

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 one hundred successive cases which were seen by Walton, where nervous symptoms were complained of and in which the question of damages had arisen or were 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 seventeen 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, and until 1866 little attention had been given to these disorders. 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

* Causal characteristics special to any individual form will be given under its description.

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 Eighth 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 last eight years:

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	2,146	3,541	4,135	5,823	26,309
1888	2,070	20,148	315	2,138	2,897	3,602	5,282	25,888

After allowance has been made for the increased number of accidents in 1893, the year of the World's Fair, during which travel was very extensive, it will be seen that the total number of persons killed and injured during the past four years is considerably larger than during the four preceding years. Startling as are these figures, many deaths and casualties may properly be classed as occurring on railways, which do not appear in this report.

The extension of the means of rapid surface transportation in recent years has very much widened the conception of the term railway. It now includes elevated roads, electric systems of all kinds, cable roads, and to a certain extent horse-car lines.

Railway collisions or derailments are particularly well

adapted to cause every variety of injury. Decapitation, dismemberment, bruises and crushes of the flesh, fractures, dislocations 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 *ébranlement*, 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 almost 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 occur-

rence 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 ten thousand dollars 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, few are so fortunate as not to be very much terrified and dazed. Fright is as fruitful a cause of some functional nervous disorders as physical injury. It alone may be sufficient to induce paralysis agitans, chorea, or exophthalmic goître, and its influence as a causative factor in the neuroses which most frequently become the subjects of litigation—neurasthenia and hysteria—is very important. Any of the manifestations of hysteria may appear for the first time as a result of fright or nervous shock alone. As we shall see in speaking of the ætiology of traumatic neurasthenia, fright is usually associated with some physical injury in the causation of that disorder, although the physical commotion or shock may be very slight. Both traumatic neurasthenia and traumatic hysteria may sometimes seem to be the result of physical injury alone or of psychic shock alone, but in by far the larger number of cases of these diseases the two causative factors have been active; and the fact that in catastrophes which occur on railways large numbers of persons are subjected to fright and to some physical commotion, explains in part why railway accidents are such fertile causes of these disorders.

Although accidents on railways stand in the most con-

spicuous 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 theatres, in machine shops. The introduction of rapid 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.* 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

* 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.

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 supposes 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 suddenly 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 neuroses, and 'railway-spine' symptoms, such as follow the perceptibly uninjured after railway accidents."

In addition to the character of the accident there are several other considerations of importance in determining the causation of the traumatic neuroses.

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 had 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 in cases which develop hysteria after trauma it is often impossible to discover any predisposition. 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 pre-existed. 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 traumatisms. Perhaps 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 especially susceptible to bad effects from traumatism. 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 afterward 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 vexa-

tions 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 harmless exaggeration of chronic invalidism, the examiner must be on his guard against malingering. To insure 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 should render the physician 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 claimant 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 claimant 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 per-

sons 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 co-operation 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 condi-

tions. 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 perception 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 ap-

plied 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 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 examinations. 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 Chapter II). 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 two hundred and sixteen non-nervous cases and of ten 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 pro-

nounced, 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.

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 can not 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 pre-existing 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.

Glycosuria, as a result of injuries to the nervous system, is a condition about which more information is desirable. The most that can be said of it is that, while it may occur as the result of injuries to the brain and medulla, temporary glycosuria is occasionally found after injuries whose chief results are functional.

The other symptoms of the traumatic neuroses will be so fully described in succeeding pages that they require no

mention 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 for 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.

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CHAPTER II.

TRAUMATIC NEURASTHENIA.

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. Any one who has become over-fatigued by mental

strain or by excessive work, will present most 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; and 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 traumatic 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 can not altogether be explained by the medico-legal aspects of railway accidents, although traumatic neurasthenia has become more frequent with the constant increase 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 the first and most constant symptom of the disorder. In the course of non-traumatic 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 and jammed between the seats or other objects, and the free portions of the body may sustain violent wrenches and twists, which are felt chiefly in 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? Such a result is unusual. If a person is very much frightened by any accident he may in future be morbidly nervous about re-exposing 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 or cause of 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 traumatisms 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 will be 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 sufficiently 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 feature in the clinical picture of neurasthenia, it is to be inferred that the pathology of the disorder is to be sought for in the 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 they can not be ascribed to organic injury of the nervous system, or to functional nervous disease more serious than nervous exhaustion.

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 without thinking too much of claims for

damages. Litigation may help the pocket, but does so at the expense of health. By avoiding it the appearance of neurasthenic symptoms 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 afterward 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 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 only appeared some time after the accident.

The **mental symptoms** constitute the most characteristic feature of the disease. In the milder forms the fretful, querulous invalid may be regarded as hypochondriacal but not as out of his mind. When the symptoms are developed in their highest degree they bring the patient very near to 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. 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 on the 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 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. While solicitude for his family or his business is usually not lost, it is made to yield the foremost place in his thoughts to the anxiety about his own health.

Introspection is an almost constant 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. Any one who, even when in fairly good health, is persistently on the alert for 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 through constant attention they are apt to be 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 pre-existing 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 cases 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.

The will power may become impaired through the combined influences of fear, lack of interest, and lack of attention. The neurasthenic can ordinarily make the simplest decisions only after much hesitation and doubt. Important questions to be acted upon excite him very much, and he frequently declares himself incapable of deciding them. There is, however, no real impairment of intellectual capacity, except such as may not be explained 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. Régis classes the whole group of the morbid fears, which are insane fears, under the head of cerebral neurasthenia. In traumatic neurasthenia, however, the fears rarely take systematized forms, except in so far as all circumstances which relate to the accident are

held in dread. It is unusual to observe fear of any one particular thing, such as fear of contamination (mysophobia) or fear of closed places (claustrophobia), or any of the various systematized forms commonly observed in certain insanities. In traumatic neurasthenia 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.

Some head injuries, however, even when there are no symptoms of fracture of the skull or of cerebral concussion, are followed by effects similar in many ways to the manifestations of cerebral neurasthenia as described by Régis.

Dr. Starr has kindly communicated to me the history 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 every one 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, resembling the speech of the general paralytic. 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 often relate to the accident. 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 præcordial 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, is glad to shun 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 previously the cheeks were red, 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 cheat.

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 is the quickness with which the muscles become fatigued. Single movements may be performed with nearly all their original energy, but muscular power rapidly becomes exhausted. The muscular fatigue can not 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 nervousness 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 often 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 can not, 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. The muscular efforts necessary for the performance of the ordinary duties of the toilet are very fatiguing, and the patient may be obliged to sit down several times while dressing himself. The finer co-ordinated movements of the fingers are imperfectly performed. There is 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.

Tremor is a frequent symptom. It resembles very closely the tremor seen in alcoholism or in confirmed tea-drinkers. 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 neuras-

thenic 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 vertebra, 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 frequently observed in 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, can not 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. 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 can not 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 hæmorrhage, 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 can not 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 laceration 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* of spinal sprain 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 any one 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 past suffering and of apprehension of suffering to come. 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 dreads 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 of 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 page 219)—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 can not voluntarily be made to materially increase its rate.

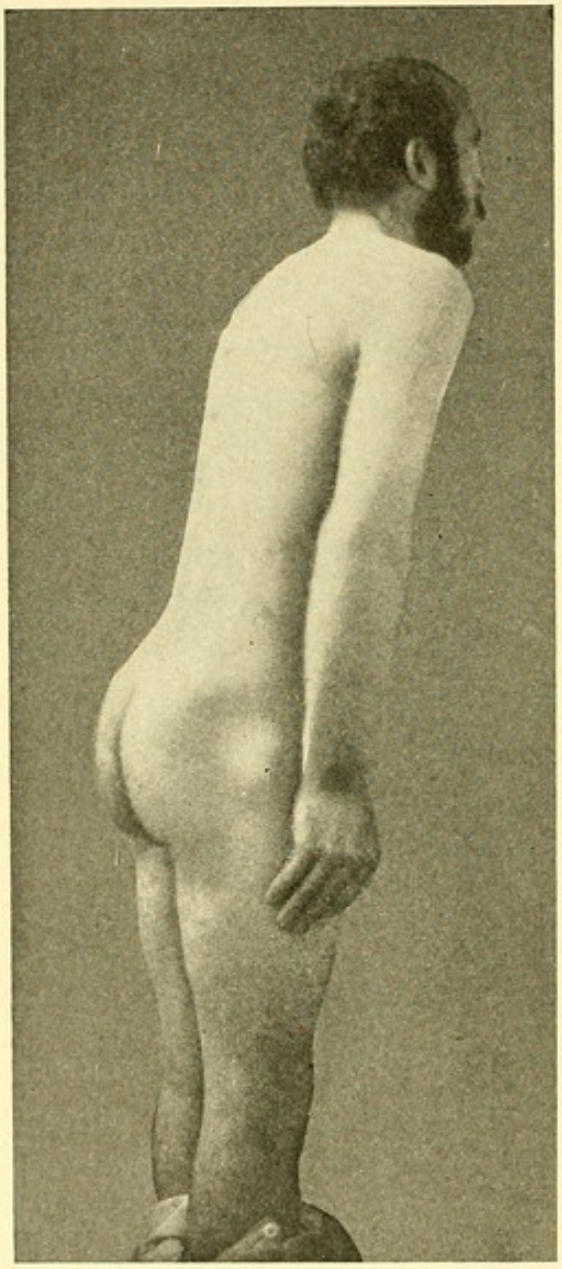


FIG. 42.—Characteristic attitude in traumatic lumbago. (Vanderbilt Clinic.)

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. 42, 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 the patients with progressive muscular dystrophy do. In getting up from the sitting posture, the hands are placed on the thighs, and the body assisted up in this way.

From similar reasons there may be striking interferences with gait. The patient walks with slow, hesitating steps, lifting his 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 upon 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 his 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 re-enforcement 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 overflow 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 incontinence 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 or cause disturbances which might be referred to interference with the function of internal organs.

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 ill borne and may cause some 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 single traumatic neurasthenia. It is frequent in hysteria, and occurs sometimes in the serious forms of nerve disturbances, which will be described as unclassified. For any case in which this symptom is present, the prognosis is graver than that of simple traumatic neurasthenia. 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 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 pre-existing organic defects or disease, no morbid changes in eye structure. The pupils respond quickly to light, but the responses become progressively less prompt and complete by successive stimulation. The pupils may also dilate and contract under psychic influences. They are usually moderately dilated. When light is thrown into the eye, the iris contracts and then dilates again, and repeats this alternating contraction and dilatation until it finally comes to rest in contraction. Inequality of the pupils is usually regarded as indicative of organic disease. Binswanger says, however, that inequality may exist in neurasthenia without indicating organic disease if the pupils continue to react promptly to light. The complaints of the patient relate to various disagreeable subjectives feeling 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 attentive 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 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. 43). The most convenient method for its determination has been described by

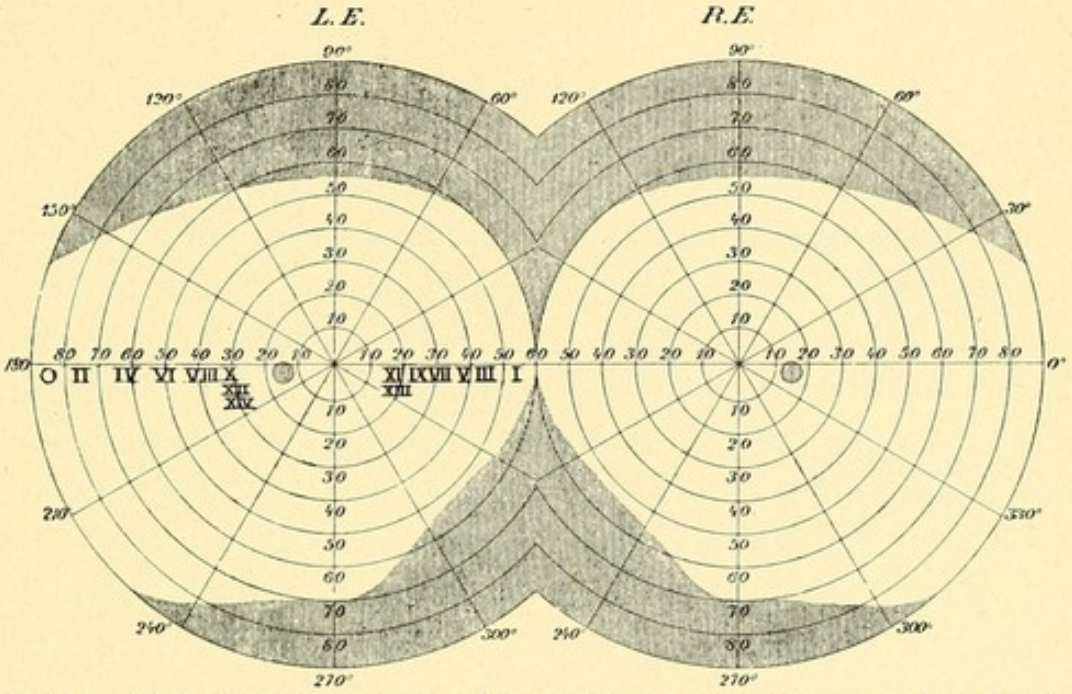


FIG. 43.—Shifting type of contraction of the visual fields in neurasthenia. (After Bass.)

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 dizzi-

ness 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. Ankle clonus occurs very rarely. It is more commonly a sign of organic disease, and, although it may occur in slight degree in neurasthenia, it then lacks the exaggerated character and prolonged duration of the contractions seen in lesions of the pyramidal tracts. 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 one hundred and thirty to one hundred and fifty 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 arterio-sclerotic 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 ninety or one hundred or even more times in a minute.

In connection with long-continuing 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, 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 pre-existent 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, is the most probable explanation of such phenomena.

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 may 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 can not 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 may be considerable loss of weight.

The remaining symptoms, although they may be much complained of, are of secondary diagnostic importance. In the female there may be interference with the menstrual function. Neurasthenic men commonly notice that their sexual power is becoming diminished, and the desire for sexual intercourse is usually lessened. They fear "loss of manhood." The urine shows no characteristic changes. It is often neutral or alkaline in reaction, so that the phosphates precipitate readily. The patient, who observes all his symptoms, does not fail to ascribe a sinister meaning to this. He thinks he has spermatorrhœa or Bright's disease. The urine may be somewhat lessened in quantity.

Prognosis.—The prognosis of traumatic neurasthenia as regards life is very good. It is doubtful if a previously healthy man ever dies from the result of an accident in which he has 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 much more serious and may sometimes prove fatal. Although many patients have died during the course of traumatic neurasthenia, it seems more reasonable to suppose that in such cases the accident added an impetus to the progress of pre-existing disease, or that the shock, injury, and resulting exhaustion proved to be more than a previously enfeebled organism could combat.

Persons who are diseased or enfeebled from any cause are heavily handicapped in the struggle for life, and accidents or other injurious influences which may seem to fatally turn the fate against them must be regarded as contributing rather than as sole causes. So the prognosis of traumatic neurasthenia, as far as life is concerned, is generally good, and only becomes serious in the debilitated or diseased.

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 sees hundreds of 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 two hundred and thirty-four patients 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 communi-

cations 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 claimants 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 usually 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 and offensive 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 claims to be decided upon, 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 carfare, 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 twenty-nine cases of "traumatic neurosis" observed by Sänger some time after the accident, three were entirely well, seven had only slight subjective symptoms and could work, eleven were only partially able to work, and eight were *in statu quo*. Richter reports three 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. Persons who have actions to bring 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 has been coming from time to time to the Vanderbilt Clinic for over two years. From the time that he was in a collision he has been unable to return to his occupation. He is a robust, intelligent man, and

was not conscious of any injury at the time of the accident, nor have we ever had reason to suspect that his condition was anything more than traumatic neurasthenia. Yet on the slightest excitement or fatigue this man becomes so confused and tremulous that he has abandoned all idea of trying to work, for the present at least, and has gone to live in the country for a year, to see if he can 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 making any claim, 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 236 grew worse instead of better after settlement.

However, 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 as well as they ever were. Others 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, and the number of these is considerable, 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. In these cases the prognosis for ultimate recovery is doubtful, and many 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 cases which 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 influences of litigation.

Litigation, much more than merely entering a claim

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 to frequently relate his symptoms, and is required to pass through many medical examinations. From the nature of the proceedings, he is not urged to go back to his work, even if he is able to do so.

It is hardly fair to assume that the increase of nervous symptoms during the time that litigation is pending is entirely due to voluntary exaggeration by the patient, or that the symptoms improve after settlement because there is no longer anything to be gained by exaggeration. 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 also, the various nervous disturbances may become intensified by litigation, not simply 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, can not 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 unfavorable influences for a long time and during a period when the symptoms are most amenable to

cure. Isolation and rest, the specifics for neurasthenia, are impossible in negligent cases.

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 has no claim to bring and can put himself at once under the care of a skillful physician, although even then recovery is not always assured.

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CHAPTER III.

TRAUMATIC HYSTERIA.*

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 mimesis 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 sug-

* To the part of this chapter which was submitted as a competitive essay was awarded the Joseph Mather Smith prize at the annual commencement of the College of Physicians and Surgeons, Columbia University, in June, 1897.

gestibility 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 every one 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, comprehensive learning, 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 pa-

tient says, 'I can not'; it sounds like 'I will not'; it really is, 'I can not 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.

Traumatism as a cause of local nervous disturbance without organic lesion had 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 traumatisms 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 traumatism 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, hysteria is unfamiliar to most American physicians. 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 injurious 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 intoxications. 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 many 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 non-traumatic origin. Dana and Knapp both regard hereditary factors as of less importance when the disease follows a traumatism than when it is due to other exciting causes. Of twenty-one cases of traumatic hysteria studied by Berbez, only nine 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 or hystero-traumatism has only been recognized 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 traumatism 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.

That an injury without being severe 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 a hysterical contracture of the hand. A mother, in a fit of anger, struck her child with the back of the 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.)

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 traumatism. 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 frequently 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. 44) around the umbilicus and around the anus. Though the patient had perfect command of the legs, and could move them in every direction while lying down, astasia was complete, and 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 perinæum and the bladder and rectal symptoms were evidences of hæmorrhage 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

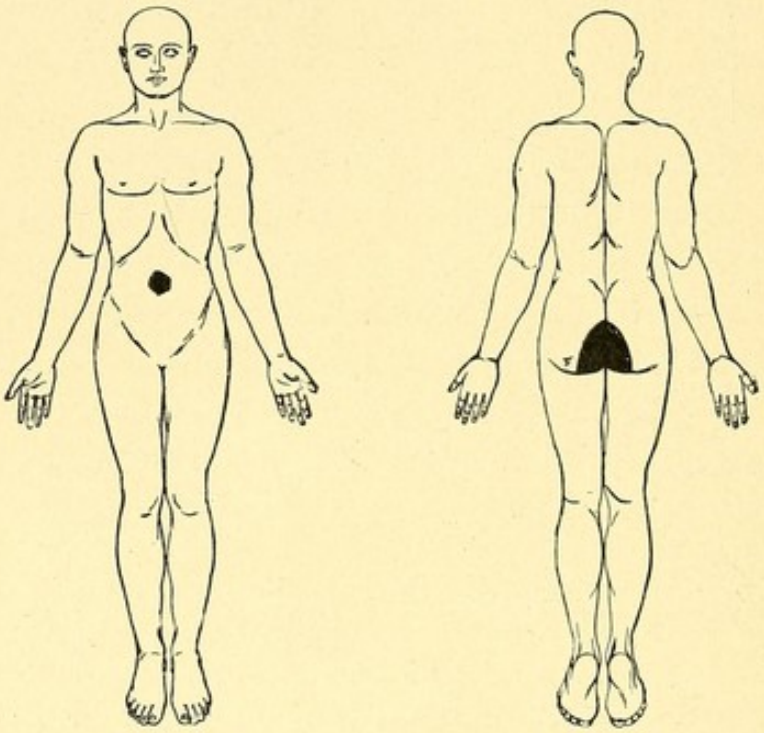


FIG. 44.—Anæsthesia in a case of injury to the cauda equina, associated with astasia-abasia.

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.

Sex.—Non-traumatic hysteria is much more frequent 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 accidents and injury. Although women

furnish by far the larger number of examples of non-traumatic hysteria, when the disorder first appears as a result of trauma, it seems as though men were more frequently affected than women. Berbez, in twenty-one cases of traumatic hysteria, found that fourteen, or two thirds of them, were men. In Knapp's cases the relative number of men and women was about equal (twenty-five cases—thirteen 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 is present in 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 the 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 any one 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 morphological 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, especially in traumatic hysteria. Occasionally only one hysterical 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. It is in mono-symptomatic cases of traumatic hysteria that 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 the 19th of October, 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. 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

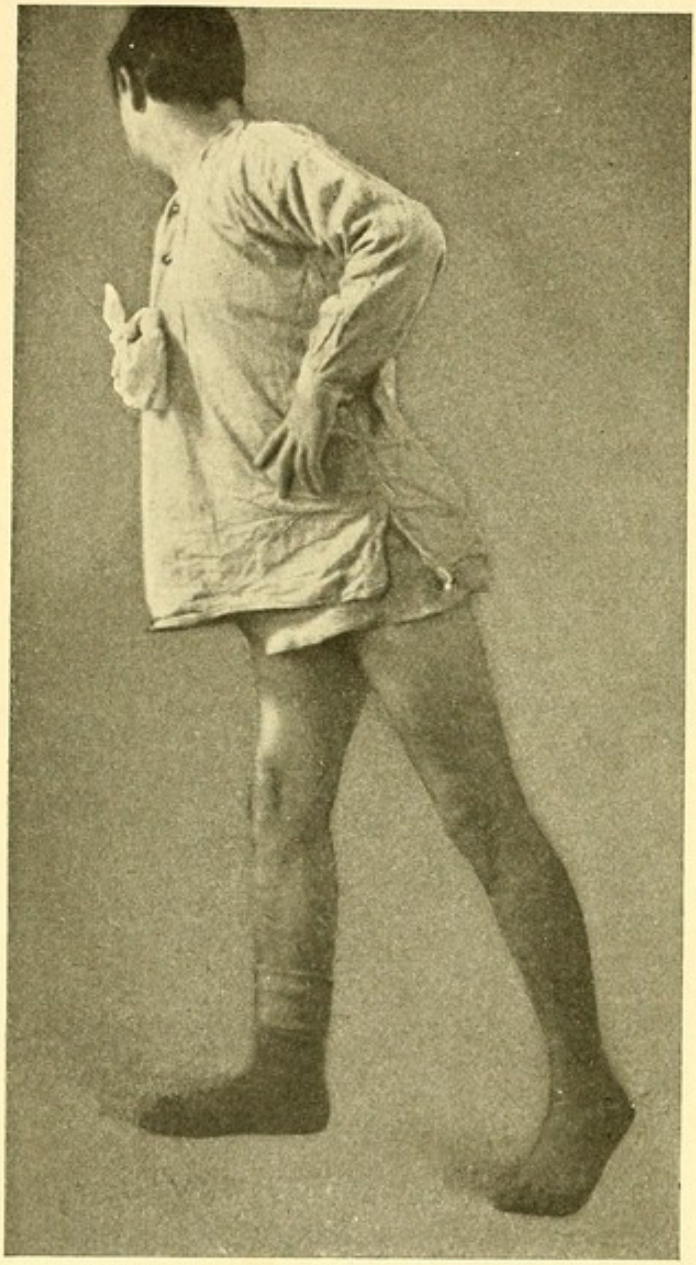


FIG. 45.—Traumatic hysteria, showing characteristic dragging of the foot. (Workhouse Hospital.)

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. 45), 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

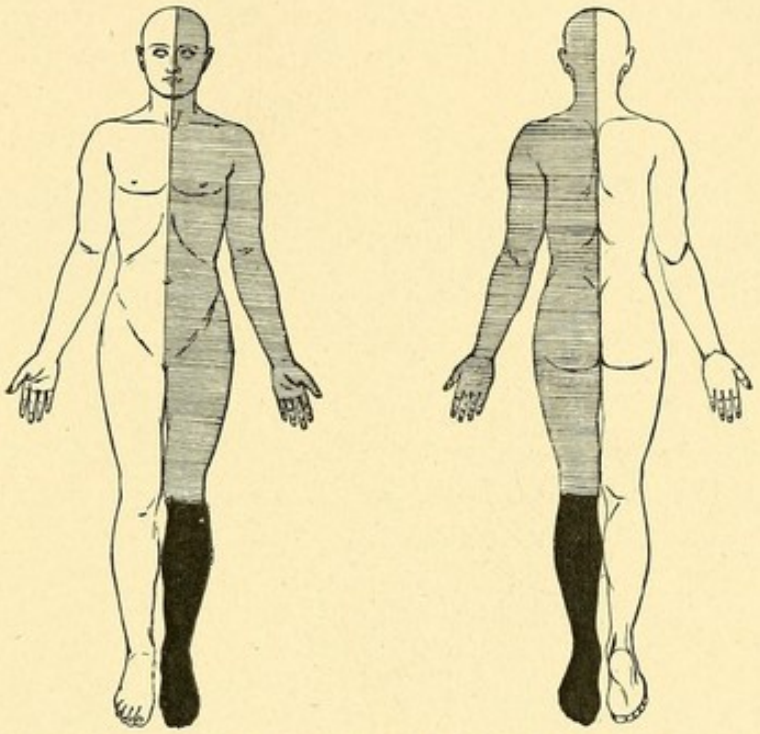


FIG. 46.—Traumatic hysteria. Paralysis existed in the left leg. In the shaded areas, cutaneous sensibility was diminished; in the left leg it was totally abolished. (Workhouse Hospital.)

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.

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. 46). 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. 47) 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, and 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

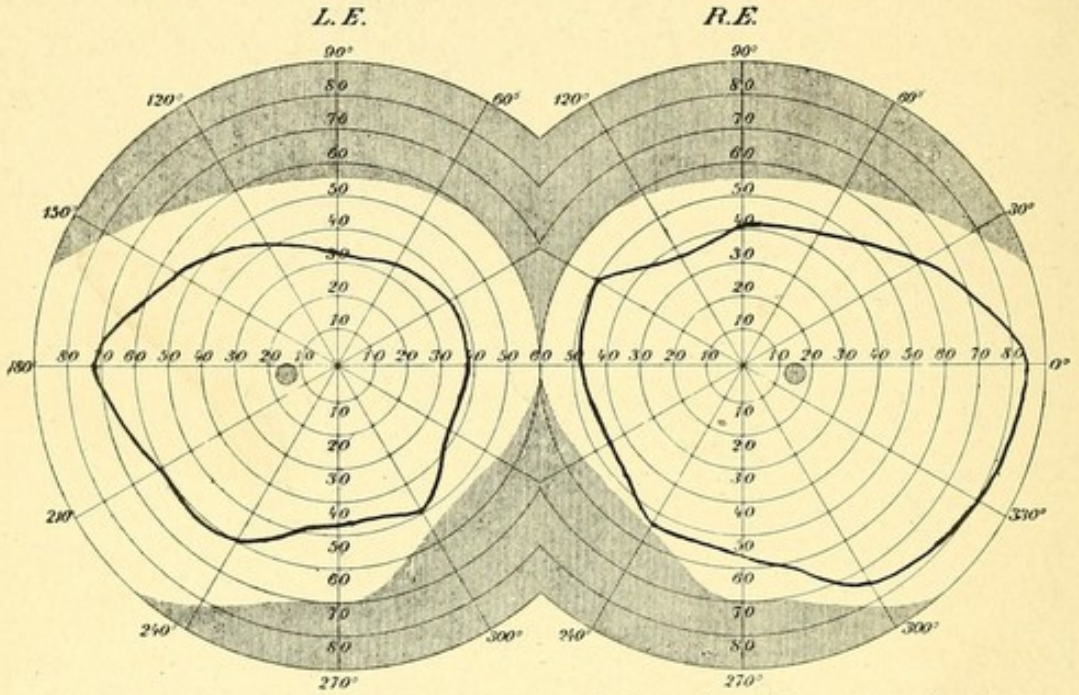


FIG. 47.—Field of vision in a case of traumatic hysteria. The contraction is more marked on the left, which was the side of paralysis and anæsthesia. (Workhouse Hospital.)

although 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.

Mental Symptoms.—The most prominent feature of the mental state of hysteria is the causation of physical symptoms 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 only occur 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 in-

tention, but without the patient knowing that his symptoms are false. It can best be explained by the theory that it is a morbid 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, memory loss may be shown to be not genuine.

When it is thus demonstrated that functional activity, though apparently abolished, exists, we can not 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 non-traumatic 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 hystero-neurasthenia. 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 traumatism 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 suggestible, 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 (page 281): 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 influences. (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 eight thousand five hundred dollars 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.

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 experiences 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 afterward 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.

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 lacks the cunning of the paranoiac. 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.

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 seventeen cases of hysterical hemi-thermo-anæsthesia examined by Charcot, eleven presented the type of total anæsthesia. In six there was a dissociation of sensations. Two of these six had loss of temperature sense, with preservation of sensibility to touch and pain. In the four 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 can not be well borne.

The limits of cutaneous anæsthesia, in my experience, have usually been sharply defined. The commonest topographical distribution of hysterical anæsthesia 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. 48). In traumatic hysteria, however, in which paralysis is so frequent, the sensory loss commonly

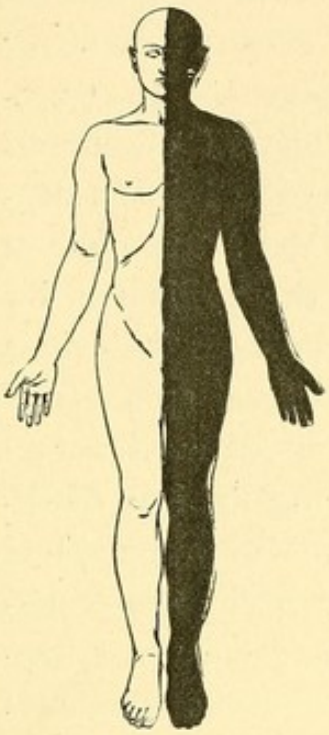


FIG. 48.—The most frequent distribution of hysterical anæsthesia.

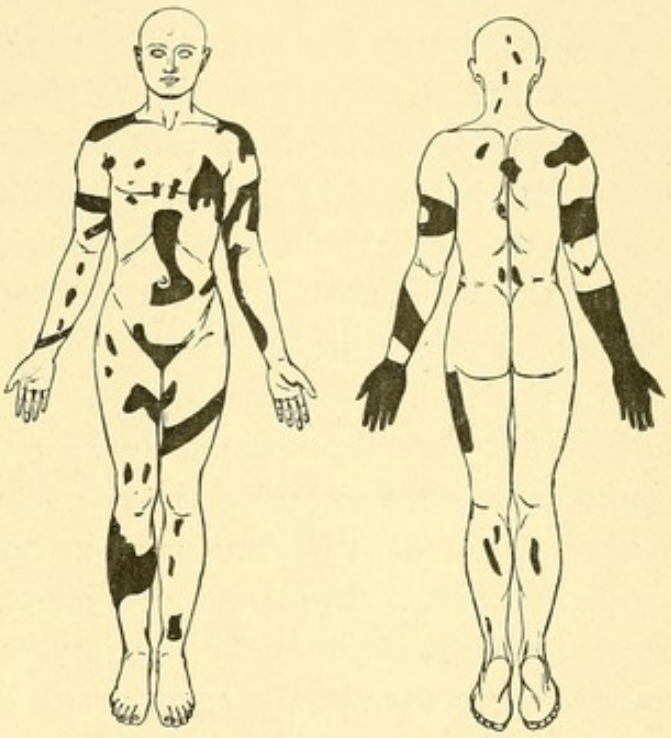


FIG. 49.—Disseminated anæsthesia. (After Pitres.)

coincides in its distribution with that of the parts paralyzed (morphological anæsthesia, see Fig. 46). 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 changes occur in segments of limbs only, it is called segmetal, 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. 49) 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.

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 fifth lumbar segment. 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 (see page 60).

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 to assume different topographical distributions.

The various alterations of the sensory functions of the skin 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 magneto-therapy 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.—Hyperæsthesia is a sensory symptom 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 Striimpell. If the back has been twisted or injured 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 a hysterical symptom generally familiar. In addition to these forms of increased sensitiveness, headache and peculiar sensations in the head 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.

Vision.—Next to cutaneous anæsthesia, disturbances of vision are the most frequent of the hysterical stigmata. Apart from any possible pre-existing 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). Bilat-

eral 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 degrees. In some cases, however, it may be very extensive, the perimeter showing that only a few degrees of

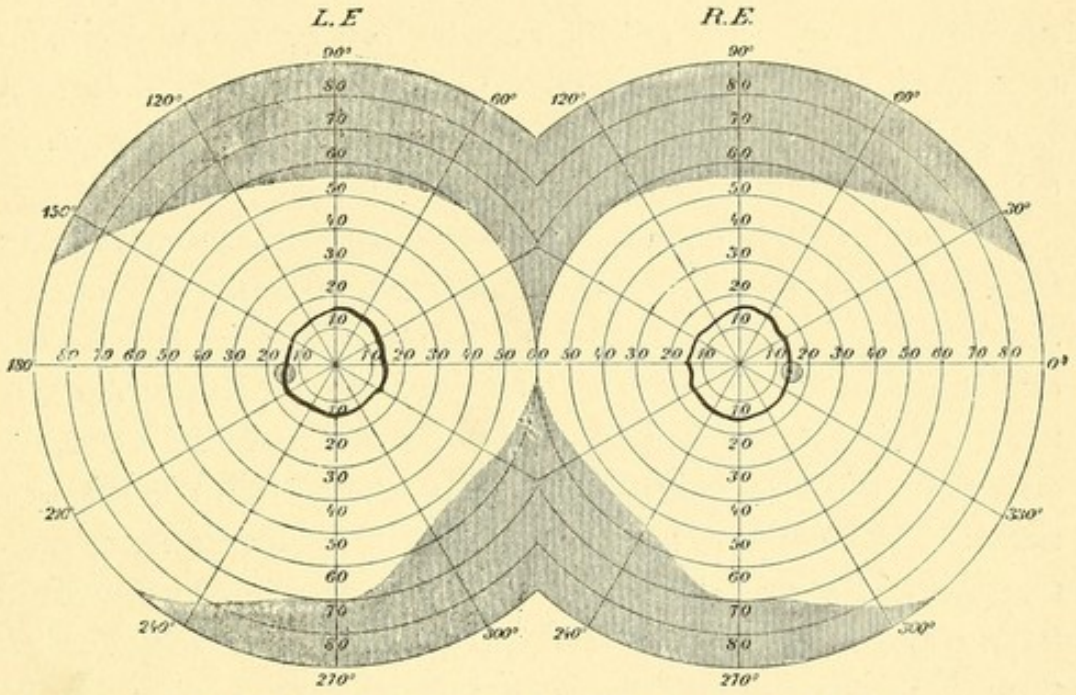


FIG. 50.—Extreme concentric contraction of the visual fields.
(After Gilles de la Tourette.)

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. 50). 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 form are characteristic abnormities in the color fields. With contraction of the visual field for form, the perception of the most internal colors may be lost, or when the contraction for form is only moderate in degree there may be a reversal of the normal color fields; thus red is often 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 any one 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 genuine and exist, when the patient has

no motive to deceive or to be "suggested," has been proved with certainty by Prince. He recently examined 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.

Disorders of the other special senses are less frequent. If there is impairment of **hearing** it is almost always unilateral, corresponding to the anæsthetic side of the body. Bone conduction is interfered with in such cases. 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.

Hyperæsthesia of hearing, smell, or taste may also occur, and is evidenced by subjective disturbances of function of these senses.

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 twenty thousand dollars. The physicians who appeared on behalf of the plaintiff admitted that there were 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 a 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 can not 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.

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 is practically never paralyzed, although 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. 51). In organic hemiplegia, anæsthesia is rarely an important symptom (see page 60), while the anæsthesia 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 thirty-one cases in which this condition was a symptom. In fifteen 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 nine 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 one case there was no anæsthesia at all.

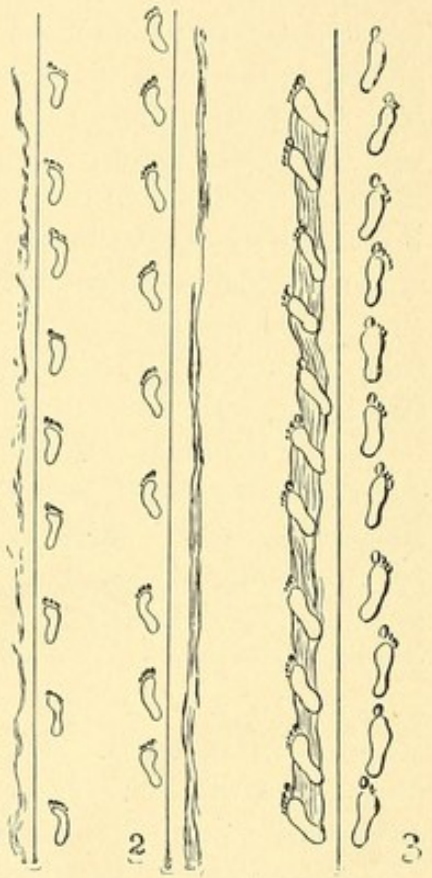


FIG. 51.—Impressions which illustrate the differences in the gait (1 and 2) of hysterical hemiplegia, and (3) organic hemiplegia of the flaccid type. (From Blocq, after Gilles de la Tourette.)

The following case, reported by Lebrun, is interesting, as it shows both the possibility of hysterical monoplegia devel-

oping 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 unchanged, except the plantar reflexes, which are frequently lost. Gilles de la Tourette says that incontinence 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. 52) 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

glosso-labio 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.

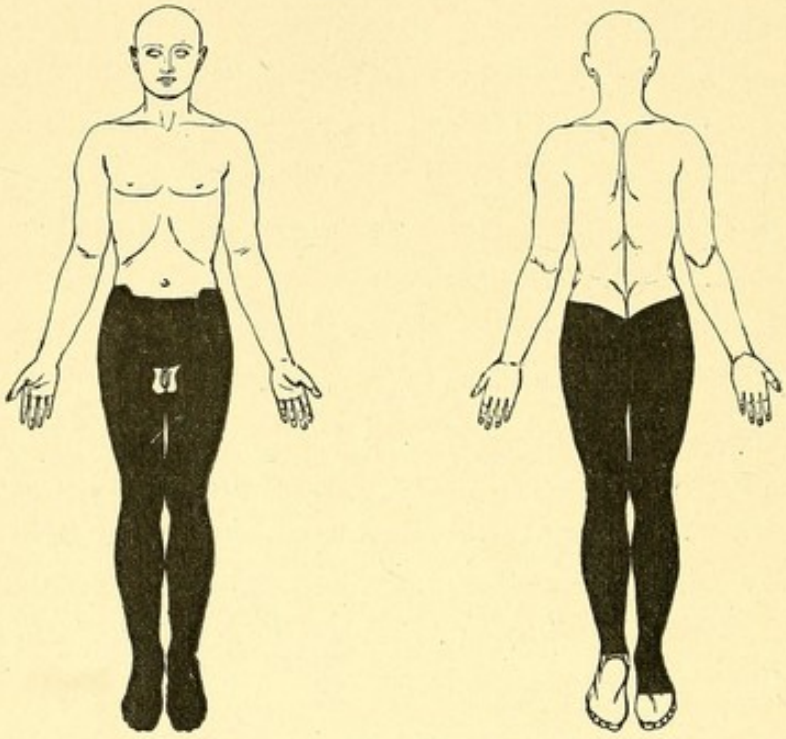


FIG. 52.—Anæsthesia in a case of hysterical paraplegia. (Souques.)

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 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 hemianæsthesia.

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 can not 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 inco-ordinated 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 inco-ordination 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 afterward 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 severe. 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 ten or twelve centimetres long and three or four centimetres 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. 53).

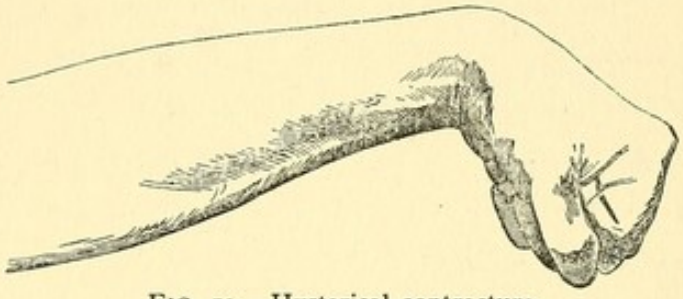


FIG. 53.—Hysterical contracture.
(After Richer and Berbez.)

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 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, magneto-therapy 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 unusual.

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. The extrinsic eye muscles may be involved. Hysterical blepharospasm, which is not infrequent, may be unilateral or occur on both sides. It gives the appearance of ptosis. Spasm of the muscles of the eyeball is

less frequent. 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 accidents. 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 inco-ordinated 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 inco-ordinated movements of forcible and shocklike 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.

Schutte 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 hystero-neurasthenia. 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 traumatism.

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 can not 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. 54), 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 twenty centimetres 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.

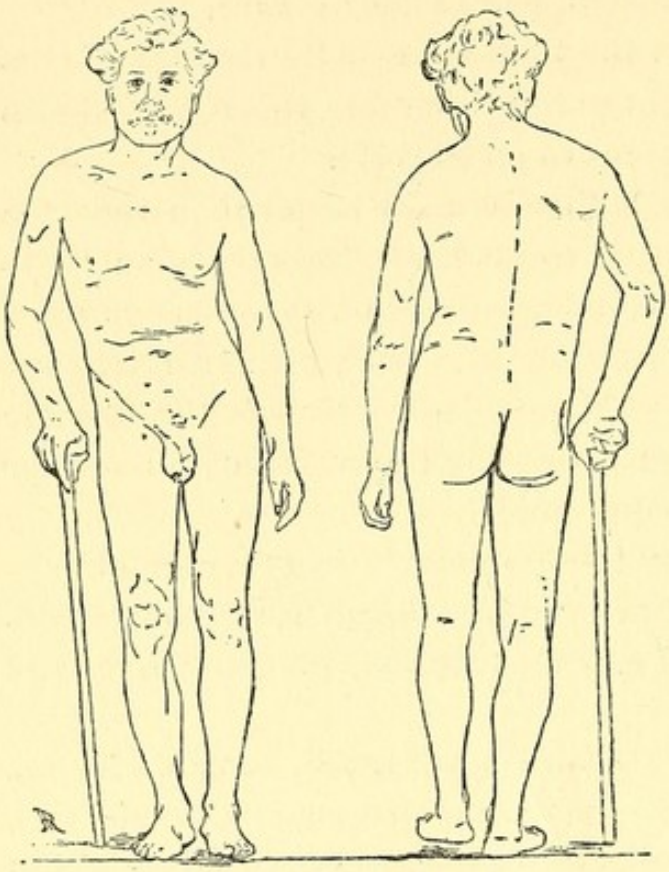


FIG. 54.—Attitude in a case of hysterical coxalgia. (After Richer and Berbez.)

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 of emotions, the hysterical joint affections 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 extremely rare, yet I have seen foot clonus in a case of paraplegia which presented every other hysterical symptom, and which eventually recovered.

The tendon reflexes may be diminished, though never lost, in a paralyzed limb. This 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 a 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 necessarily 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.

Deaf-mutism is a rare accident of hysteria. That it may occur as a result of injury is shown by the following case, reported by Francotte:

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.

Hiccough and cough are common symptoms of hysteria. Blood is sometimes vomited or coughed up immediately after the accident. Anuria may occur, but the passage of large quantities of pale urine is more frequently observed. Ordinarily the urine shows no characteristic chemical changes, although occasionally temporary glycosuria has been found. Nutrition may be seriously affected.

The other symptoms of traumatic hysteria resemble those described under neurasthenia. There may be a variety of subjective symptoms, of which, perhaps, the commonest is the globus hystericus. Cardiac irritability and digestive disturbances are frequently observed. There may be continued vomiting. Constipation is the rule. The respiration is ordinarily normal, but becomes very rapid through emotional influence. After nervous shocks, or local irritation of the throat, the voice may be lost. Either aphonia, in which the patient loses the power of uttering any sound, or mutism, in which the power of speech is lost, are frequent hysterical symptoms. As Pitres has pointed out, hysterical mutism is often an isolated accident, which may be observed in persons entirely free from previous nervous symptoms or active hysterical manifestations.

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.

As we have seen, many cases of traumatic hysteria are somewhat different from hysteria when it occurs from causes other than traumatisms. In traumatic cases, impairment of general nutrition may render the patient less capable of resisting the ordinary injurious influences of daily life. Nevertheless, even in them, the prognosis of hysteria in regard to life is very good. As regards complete recovery, it is much less favorable than that of traumatic neurasthenia.

The patient with traumatic hysteria may be relieved of his paralyses or contractures more speedily than the neurasthenic is rid of his lumbago. Yet the disease often remains,

ready to show itself again under the influence of suggestion or emotion, although its paroxysmal symptoms have disappeared. Although some cases get entirely well in a few weeks or months, never to relapse, there is a general agreement of authority that the disordered mental state of hysteria, which is the cause of the symptoms, is an extremely tenacious condition, and that the prognosis in any given case is always attended with uncertainty.

There are some considerations, however, which to a certain extent are of value in reaching a prognosis. Among the most important of these is predisposition, which has occurred through enfeeblement of the resisting powers of the nervous system by transmitted ancestral defects or by acquired exhaustion. Such predisposition is only occasionally discoverable in traumatic hysteria, and the chances for recovery are naturally best, as in certain forms of insanity, in the patients whose personal and ancestral record is free from nervous stigmata. As regards age, it may be said that the younger the patient the better are the hopes for ultimate recovery. It must not be forgotten, however, that when hysteria appears in children there may often be found a strong hereditary predisposition to nervous disease. This fact may more than counterbalance the favorableness of the prognosis caused by youth. Hysteria in men is attended with a more serious prognosis than in women.

The prognosis is better for the well-to-do classes, for in them proper treatment can usually be instituted. The treatment, which is a regulation of the mode of life rather than a treatment by drugs, is expensive, and can only occasionally be properly carried out in the working classes.

In regard to litigation, what is true for the prognosis of traumatic neurasthenia may be made still more emphatic for traumatic hysteria. The constant questionings and examinations by lawyers and doctors are most potent suggesting influences. They render the consciousness still more lim-

ited, and give an increased permanency to the fixed ideas of the hysterical patient.

Repeated examinations of a limb which is the seat of hysterical paralysis render the loss of power more profound and more permanent; repeated tests for sensation cause anæsthesia to deepen, to spread, or to appear in new places; it is well known that the symptoms of hysteria often owe their genesis to the suggesting influences of hospital life; all these agencies are most prejudicial to ultimate recovery. It is the evil results which attend suggestion of any kind upon the hysterical mind that has led to the general abandonment of the use of hypnotism—a procedure which may cause a symptom to temporarily disappear, but which involves the danger of aggravating the mental state.

The hysterical patient is very suggestible even when not hypnotized, and constant examinations almost invariably suggest to him that the symptoms remain stationary or become worse.

Recovery from hysteria is possible, even when the disorder has occurred in an aggravated form. But, as a rule, the more numerous and severe the symptoms, the less it is probable that the patient will ever be free from the disease.

The exaggerated psychical phenomena are of especially serious import, as they denote a profound disturbance of mental function. The attacks, catalepsy, trance, and coma, indicate that the disease is severe, and that it has probably developed in a predisposed brain.

Some of the stigmata, such as the anæsthesias and visual disturbances, are particularly tenacious.

Recovery from the joint affections is usually slow. They may frequently relapse or last for several years, and may eventually cause permanent organic disabilities.

There are no means of foretelling how long a hysterical paralysis may last.

In a case of Page's, paraplegia existed for four years and

a half, and in a patient of mine, whom I have mentioned, the left leg has been paralyzed for two years. Zein reports a case of hysterical paraplegia in which recovery took place after nineteen years; and Morton Prince has seen three cases of hysterical paralysis (two monoplegias and one hemiplegia and hemianæsthesia) which began in the War of the Rebellion and which had existed, one for twenty-eight and two for twenty-nine years.

The brachial monoplegias seem, as a rule, the least persistent of the individual forms of paralysis.

The prognosis of mutism, or aphonia, is nearly always good; speech is usually restored in a few weeks or months. But all of these symptoms are liable to relapse, and it is never justifiable to consider the hysteria as cured because all the paroxysmal symptoms disappear.

The relapses, which may be more, or less severe than the conditions which preceded them, follow very shortly after apparent cure, or are separated by considerable periods of time.

The rapidity with which hysterical symptoms may vanish, and become re-established, is well shown by a case seen with Dr. G. E. Brewer at the City Hospital:

A man, thirty-eight years of age, alcoholic, fell in the street and found both legs paralyzed. When brought to the hospital he presented the classical symptoms of hysterical paraplegia—viz., great rigidity of the legs, inability to walk, and anæsthesia, with perfect control of the bowels and bladder. He was put on appropriate treatment, and recovered so completely that in a few days he walked out of the hospital in a perfectly normal way. About ten days later I was asked by Dr. Frank Markoe to see a case in his wards at Bellevue Hospital. It was of a man who had been found lying in the street with both legs paralyzed. The patient was the same man that had been seen at the City Hospital.

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(The reader is especially referred to the comprehensive treatise of M. de la Tourette.)

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CHAPTER IV.

UNCLASSIFIED FORMS.

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 can not 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 over-fatigue 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 further, 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 are 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 Charity, in Berlin, were only forty-two 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. 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 replies 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 intellectual 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 can not 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 co-ordinated 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 his index finger when his 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 only be performed 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 goître, 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 mus-

cles 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 mul-

tiple 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 can not 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 hystero-neurasthenia.

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.

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 are diminished, but in no case have the knee-jerks been said to be lost when Jendràssik's method of re-enforcement has been 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.

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 and 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 are not evident from the history. The visual impairment was more marked than at the previous examination, and the optic-nerve atro-

phy 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.

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 arterio-sclerosis, 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 hæmorrhage, 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 hæmorrhagic. That such lesions (see case on page 82) may 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 hæmorrhage 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 disturbances 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 further than to say that the case is one of severe nervous disorder. We can not definitely specify upon what pathological basis it rests, or by what theory its symptoms may best be explained. Nevertheless, although the diagnosis can not, 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 hysteria the individual manifestations vanish, leaving the patient apparently well, although commonly branded with some of the hysterical stigmata. 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.

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PART III.

MALINGERING.

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 recently 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 two hundred 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 assume from these figures that there are now pending in Cook County 3,600 of these cases, and that the damages claimed are between fifty and sixty million dollars." It is needless to add that this enormous increase is out of all proportion to the increase in the number of accidents.

As is well known, the number of suits which come to trial forms only a small fraction of the total number of claims that are entered. Mr. Prentice estimates that for each claim

that appears in court it is probable that eight or ten claims are settled without suit.

The chances of successful issue, which are notoriously good, seem to be the most prominent consideration in bringing claims for damages. The extent of injury, or the social position of the claimant, are matters of secondary importance. The larger number of claims are brought for trivial injuries, and among the claimants may be found the poor and the rich, the capitalist and the pauper, men of all professions, of all creeds, and of all classes of society. An injury received through another's negligence has come to be regarded as capital, which is to be converted as soon as possible into cash. This view has received judicial recognition in Illinois, for by a recent decision of the Supreme Court of that State a claim for personal injuries is property, which may be put on the market and transferred or trusteed.

The larger portion of the population is thoroughly cognizant of its rights, and loses little time after the accident in calling the attention of the responsible parties to the injuries which have been received. If no thought of financial recompense occurs to the injured person at first, this is an omission which will soon be remedied, either by sympathizing friends or by persons experienced in the value of a personal injury as the basis for a damage claim. The press exerts an important influence upon the number of claims and suits for damages. It is especially noticeable in rural districts, where the publication of a large verdict rendered in a negligence case is commonly followed by a very appreciable increase in the number of personal-injuries claims brought in the neighborhood of the original accident. This is partly due to the increased attention given to injuries when it becomes generally known that they may be so easily turned into financial benefit, and partly because the lawyers who make a specialty of scanning the papers in search of casualties have their attention thus called to a promising field for their activities.

Any press mention of an accident occurring in a large city is sufficient to secure for the injured person an abundance of gratuitous and unsolicited counsel. A man was recently injured in New York late in the afternoon. Before eleven o'clock the next morning eight lawyers had called at the hospital to see him. The last hours of the unfortunate man were passed in listening to the arguments of the attorneys who were going to secure damages for him, had they gotten the chance.

The prosecution of personal-injury suits has become a systematized business. Mr. Prentice says that many law firms and brokers employ "runners," who have business relations with saloon-keepers, policemen, and surgeons living in the parts of the city where accidents are frequent. The runners are constantly on the lookout for accidents, and as soon as one occurs they discover it themselves, or are notified by their associates, and lose no time in trying to persuade the injured person to intrust his claim to the firms or brokers whom they represent.

In New York city the value of information regarding an accident is so generally recognized that witnesses carry the news voluntarily to the offices of "ambulance chasers," satisfied with the small fee which they are almost certain of receiving in return for their services.

It is, of course, simple justice that persons who have been injured through the negligence of others should receive reimbursement for the losses entailed by absence from their business, and that they should be compensated for the suffering they have been made to pass through, as well as for any resulting disability. And if, as sometimes happens, the injured person is ignorant of his rights in such matters, it is essential and proper that these rights should be explained to him. Nevertheless, although the larger number of actions which are brought for personal injuries are in all probability based upon some actual, though often slight, loss of health,

it is inevitable, with the enormous increase in recent years in the number of accidents, that many cases which appear in the courts be attended with a certain amount of absolute fraud. And the number of persons who demand compensation from corporations for trivial injuries, or whose claims are absolutely fraudulent, and who, either by compromise or by failure to prosecute, or for any other reason, do not appear in the court, must be very large indeed.

The enormous sums paid every year by large corporations as indemnity for personal injuries renders the temptation to this kind of fraud very strong.

The general claim agent of a Western railway whose system covers about five thousand 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.

It is, furthermore, a generally recognized fact that corporations prefer to settle small claims, even when convinced that they are fraudulent, to defending suits. They are forced to adopt the policy of settling claims rather than of letting them go to trial, from the well-known attitude of juries toward the defendant when the latter happens to be a corporation. It is often flagrantly apparent in such trials that the jury, in reaching a verdict, has concerned itself less with the evidence than with a consideration of the relative financial resources of the plaintiff and of the defendant. This was recently shown in Brooklyn, when Justice Wilmot M. Smith set aside the verdict of $11,000 obtained by Christian F. Stark against the City of Brooklyn and the South Brooklyn Terminal Railroad Company. Stark sued for damages for personal injuries received by falling down an embankment at Thirty-eighth Street. In a previous trial Stark received

a verdict of $10,000, which also was set aside. Justice Smith said: "Prejudice or passion must have influenced a verdict so at variance with a result which should have been reached upon a sober, impartial, and rational consideration of the evidence. A verdict of a jury should not be disturbed unless to allow it to stand would result in a clear failure of justice. I set aside the verdict because of a settled conviction in my mind that to allow it to stand would be a serious reflection upon the administration of justice, and a confession that the courts do not furnish a tribunal to which all citizens may repair with confidence that their contentions will receive rational and impartial consideration."

As long as prejudice, in the minds of the jury, against corporations can be counted upon, the temptation to bring damage suits upon insufficient grounds will remain very great. The inducements for exaggeration and deceit which are brought about by these means are furthered by the speculation in damage claims, which has become so serious a sociological problem. Many negligence cases are taken on the contingent-fee plan by lawyers who associate with themselves self-constituted medical "experts." The lawyer supplies the witness, the doctor formulates his opinion, and the result is one of two: The corporation settles, if it can do so on not too extravagant terms; or it decides to "fight"; it then engages in an unequal contest.

In this way many claimants and plaintiffs receive much larger sums than any to which their injuries entitle them, or are paid by the company even when they are entitled to no compensation whatsoever.

The rectification of this question lies with the corporations themselves and, in a way, with the medical profession.

By showing themselves more zealous in promoting the comfort and privileges of their patrons, and by insisting upon more courteous behavior on the part of employees, transportation and other companies which have to do with

large numbers of the public, have their surest means of gaining the good will of the community, and of thus doing away with the general prejudice against them which becomes so apparent when they engage in litigation against private individuals.

The part to be done by the medical profession will only be possible when legislation has provided an answer to the vexed question of experts. At present, when there is no legal criterion by which the value of a medical opinion may be estimated, the testimony of a charlatan may carry as much weight to the minds of a jury as that of a physician who has shown himself skilled in his subject. It is the contradiction of professional opinion in the witness chair which has brought medical expert testimony into disrepute, and which has caused it to be so often regarded as of questionable value as evidence.

With the existing state of affairs it is not surprising that the feigning of disease alleged to be the result of accidents, or the exaggeration of symptoms due to actual injuries, are difficulties against which corporations must constantly contend and are means by which they are not infrequently defrauded; yet from a purely medical point of view it should be generally easy to unmask the malingerer.

Simulation is of necessity limited chiefly to diseases of the nervous system. In visible physical injuries there is little chance for fraud. Fractures, dislocations, and similar purely surgical affections can not 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. Nervous structure is hidden and protected; no part of it can be felt, and the optic nerve is the only part of it that can be seen, and then only by means of the ophthalmoscope. In popular opinion, nervous function is indefinite, and nervous diseases obey no recognized laws.

It is generally known that participators in accidents often receive large sums of money for injuries to the nervous system, although no visible injury be sustained and although the claimants present no symptoms more definite than those popularly supposed to constitute "nervous prostration." Consequently it is to the imitating of diseases of the nervous system that the simulator naturally turns, and it is with them that he has the greatest chance for success. Neurology is the most recently developed branch of clinical medicine, and is the one least familiar to physicians generally. As the study of it becomes more popular, successful simulation will become more rare; for, notwithstanding the complexity and minuteness of the architecture of the nervous system, and the mystery which must ever surround nervous function, nervous disease causes characteristic and fairly constant symptoms. The detection of simulation depends upon a knowledge of them on the part of the physician, and the inability on the part of the malingerer to exaggerate or create the products of disease.

Fraud is attempted in accident cases in three principal ways:

1. Exaggeration of symptoms actually present. 2. The substitution of origin, or the allegation that pre-existing 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 musculo-spinal 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 withdraw the claim without any medical examination. 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 there is no question as to the reality of some of the symptoms, it is frequently impossible to ascertain just how far the real symptoms are voluntarily magnified. Although he can not always give reasons for his opinion which prove satisfactory to juries, an experienced surgeon can usually tell from the appearance and manner of the pa-

tient 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. The bringing of claims by persons suffering from slight subjective nervous symptoms has 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 can not be objectively proved. He is unaware that the same kind of exaggeration he so often sees in the claim-bureau 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 he has no claim to bring, describes his symptoms as more severe than there is any reason to suppose they are.

A person who has become neurasthenic is tired and weak. However robust or careless he may previously have been, exhaustion of the nervous system has rendered him easily fatigued and 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 can not 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 can not 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 efforts 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, when 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 can not 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 claims to have 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 profession 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 to demand excessive mental and physical work, which he has continued to do without interruption since the acci-

dent. 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 five thousand dollars, and compromised for one hundred and fifty dollars.

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.

2. Substitution of Origin.

The allegation that *pre-existing disease or deformity were the direct results of accident or injury* is a form of fraud often difficult of detection. If a person claims that he was in every way normal prior to an injury, and that only since its incurrence 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 difficulties in obtaining sufficient knowledge of the claimant's previous life is 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 this 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 defendants' 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.

This case illustrates 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 responsible. 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.

It is, however, the chronic degenerative diseases of the nervous system which 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 can not directly result from injury independently of the co-operation 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 may later become general, it is in entire accord with our present medical knowledge to assume that the epilepsy is traumatic, and that the patient would never have had the disease if his head 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. It is essential to have considerable evidence that before the injury the patient had been free from any symptoms of either of these disorders; 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 men-

tal 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 muscular 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 been shown (page 138), 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 is generally unfamiliar, in the earlier stages at least, to the larger number of American physicians. 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 (page 167).

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 claims 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 pre-existence. 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 him-

self from any suspicion of fraud, but he also gave almost certain proof that the disease had antedated the accident.

There is great opportunity for fraud 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 pre-existing disease had been caused by the traumatism. 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 trauma.

What has been said concerning the possible pre-existence 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 (page 301) 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 as a designation for those cases only 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 it is not very common for persons who have no symptoms at all to simulate disease as a result of accidents, on account of which they demand compensation. 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 a 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. 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."

But although simulation is probably not very common, it is sufficiently frequent for it to be an important factor in the consideration of 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." (Page.) 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 his 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. "Hullo!" 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.

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. 55). 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.

On September 11, 1894, she made claim on the Chicago City

Railroad Company for injuries caused to her right arm by the sudden starting of a car. She received a hundred dollars.

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 arrested for theft. Her appearance and manner were pleasing and gentle.



JENNIE FREEMAN.

On January 9, 1893, she made claim on 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



ESTHER FREEMAN. FANNIE FREEMAN.

FIG. 55.—Some members of the Freeman family.

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 five hundred dollars, and Miss Jennie is said to have recovered a few days after the damages were paid.

If this paraplegia were genuine, it must have followed a course of unusual benignity, for on October 5, 1893, the patient made claim on 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 one hundred dollars to her physician and one hundred and twenty-five to Jennie Freeman.

On May 16, 1894, claim on 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 one hundred and twenty-five dollars 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 on 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 two hundred dollars."

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 supported, 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 three hundred and twenty-five [or four hundred and twenty-five] dollars.

On June 6, 1894, Fannie Freeman, under an assumed name, made claim on 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 the 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 doctors 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 were satisfied of the fraud, and reported that there were no objective evidences of the conditions complained of.

The following day the three women were arrested. Fannie

Freeman, "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.—In accident cases simulation is attempted with the sole object of gain. Reference has already been made to the temptations which lead to this particular form of dishonesty. Yet, while the bait is alluring, the person who makes a claim for personal injuries, when in reality he has not been injured at all, 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. Of course, 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.

The past record, however, the successful simulator may

be able to conceal, if concealment is necessary. It is more difficult for him to prove himself of a character and position, the possessors of which do not ordinarily resort to flagrant and absolute imposture. In this respect the absolute simulator is very different from the person who exaggerates actual though slight injuries. Symptoms may be, and occasionally are, dishonestly magnified by persons whose reputations for integrity have escaped suspicion, and who are regarded as estimable members of society. But such persons are rarely absolute simulators. The simulator crosses the border land of deceit and takes his place among criminals. If he can not prove himself as following some respectable calling, the success of his venture is hazarded. Claim agents show an inclination to know as much as possible about personal habits and modes of life of the claimant before they consider adjustment, and the impossibility of furnishing satisfactory details of this character undoubtedly deters many who otherwise would cheerfully undertake to defraud by feigning disease.

Even when 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 must hold himself ready for examination at any time. Examinations made at the most unexpected

hours or in rapid succession should find him always prepared to reproduce an essentially unvarying chain of symptoms. He 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, the simulator 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 only acquired 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 prospects are ruined. Yet with it all are wanting the objective evidences which almost invariably accompany such severe symptoms. The eye is bright, and the face, seen in repose, belies the assertion of constant physical and mental torture. 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 can not 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. It seems to me that lack of medical knowledge, in accident cases at least, is one of the minor difficulties against which the simulator has to contend. 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 gen-

erally 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 escape exposure, although both claim agent and physician suspect him of being a swindler.

The form of disease most commonly simulated is neurasthenia, for which 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. Inmates, attendants, and hangers-on of various hospitals and public institutions generally become, through long experience, well acquainted with the different forms of human suffering. 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 can not 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 only be successful when the examiner is himself careless or unfamiliar with the symp-

toms and the means of demonstrating them, for organic nervous disease can not 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 experienced swindlers as they were, 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 recognized as an impostor, because 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.

Similarly, 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 can not be feigned. General paresis, progressive muscular atrophy, and tabes can not 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 rarely feigned in accident cases, and usually appears in the form of a genuine epileptic alleging that the disease was caused by injury. If epilepsy is alleged as the result of accident, the evidence of non-medical 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 he is not justified in calling the man a swindler. If a claimant restricts himself to assertions of pain in the back, loss of energy, inability to do his work, and similar complaints of purely subjective character, the physician may be sure that the symptoms are exaggerated, but he can

not show that they are false. The truth in regard to claims which are 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 became 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 can not 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 not to be 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 any one 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 is being sued for damages for personal injuries should be entitled to ascertain the character and extent of the injury for which he is held responsible. Viewed from a purely commercial standpoint, he has a right to know what he is paying for, or, in other words, to insist upon a definite diagnosis before payment is made. Such diagnoses, if they can not 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 simple 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 can not be well feigned. One is that the condition of the muscles themselves—the flabbiness, softness, and lack of tone—can not 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 only take place 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 can not be voluntarily suppressed. A man may claim that he does not taste, see, or hear, statements which may be difficult to verify; but he can not voluntarily cause permanent devia-

tion 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, can not be imitated in a way to deceive any one 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 only occur 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 can not 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 assert 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 can not 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 can not 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 anæsthesias, 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 rigid-

ity, 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 can not 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 suddenly 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. Any one 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 decide 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 hyste-

ria 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 point 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 addi-

tional 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 can not 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 appear 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 any one 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 can not 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 can not 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 im-

postors 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 can not 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 oculo-motorial 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 can not 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 ten to fifteen 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. 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 pupils do not react to light; the urine is passed involuntarily; and 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 theatrical; 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 rate 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 re-establishment of the normal cardiac rhythm.

The other vascular symptoms, such as flushings, coldness, and blueness of the extremities, can not 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 simu-

lation, 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 total absence of the normal muscular contraction of the sphincter.

Reflexes.—The reflexes are beyond voluntary control. The skin reflexes can not 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 cilio-spinal 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 con-

traction 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 at once discovered 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 can not 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 can not 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 re-enforcement 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.

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PART IV.

TREATMENT OF THE TRAUMATIC NEUROSES.

It has been frequently asserted in 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. Although the present volume concerns itself with causes rather than with cures, it seems so essential that there should be a greater familiarity with methods which can often prevent a reasonably healthy man or woman from becoming a permanent invalid, that these closing pages may be appropriately devoted to treatment. As we have seen, these disorders comprise traumatic neurasthenia and traumatic hysteria, and mixtures of the two. The treatment for all is essentially the same. It consists in the regulation of the mode of life in such a way that the patient is shielded from those influences which experience has shown work him the greatest detriment, and in supplying, as far as possible, the essentials of normal healthy living which, by reason of fatigue or perverted ideas, he can no longer gain for himself. It is the natural method, rather than treatment by drugs, and it is based upon principles of physiology and psychology, rather than upon any to be found in the materia medica. Under the name of the *Rest Cure*, as described by Weir Mitchell,* one form of treatment for neurasthenia and hysteria is generally familiar. When these disorders result from injury or shock it undergoes slight modifications.

* Fat and Blood, sixth edition, Philadelphia, 1891.

THE REST CURE.—It is by no means necessary for every case of traumatic neurasthenia or hysteria to be put to bed for weeks or months. It is advisable for any one who has been severely frightened or shaken up to rest quietly for a few days or a week, during which time he may be free from effort and annoyance and have his thoughts diverted as much as possible from the accident. But at the end of that time, if the shock and bruising were not excessive, he can usually take up his work again. When, however, neurasthenia or hysteria is pronounced, and especially when the patients have been harassed by frequent examinations and have found no benefit from various therapeutic methods, the rest cure, in its most comprehensive extent, remains as the best means of speedy if not of ultimate recovery.

Before proceeding to an enumeration of the various procedures incident to the rest treatment, emphasis must be laid upon the one condition, without the fulfillment of which success is imperilled or rendered impossible. This condition is the complete control of the patient by the physician. The amount of rest, both physical and mental, should be determined by him, and he should be kept constantly informed of the most trivial happenings which go on in the sick-room. He should have within his jurisdiction the regulation of the patient's whole mode of life; he should have the right to determine whether complete isolation be necessary, or if interviews with friends or with counsel are to be permitted, and he should decide as to their frequency and their length.

While litigation is pending it is practically useless for the patient to undertake the rest cure. Under existing conditions any person who is really the worse for an accident can usually obtain, by settlement, reasonable compensation for his injuries without going to court. If the claim for damages can not be amicably settled without suit, the patient may as well decide at once between the two alternatives, viz.—whether he wishes to relinquish the chances of a gen-

erous verdict, or, secondly, to indefinitely postpone his recovery. When the injuries are slight, and the amount claimed is large, this decision is quickly reached; but if the functional disorder is well marked, the patient is often unable to decide for himself. Under such circumstances the most that the physician can do is to explain the nature of the treatment, and to show how impossible it is to fulfill its requirements and at the same time to grant to attorneys the proper facilities for preparing their case. When he has emphasized the unquestioned truth that the longer a traumatic functional disease exists the more difficult it is to cure, when he has explained that during the months or years that these suits are dragging through the courts before a final verdict is reached the disease is taking on a phase for which no money can be considered adequate compensation—when the physician has made these facts plainly evident to the patient and his friends, he can do no more.

Although litigation constitutes the most serious obstacle to the institution and carrying out of the rest cure, it is not always easy to convince the patient or his friends of the reasonableness of the method, even when the powerful factor of litigation is absent. The friends of the patient often regard him as more frightened than hurt, and the patient himself resents being deprived of his liberty. He may accede to the physician's demand for a time, but becoming tired of forced seclusion, eventually insists either upon returning home or of undertaking some more radical form of treatment. Or his friends may be unwilling to give him over to the entire charge of strangers and to place him where he is no longer amenable to their wishes or the recipient of their sympathy; or, having consented to the separation, soon change their minds and insist upon some less exacting method. Yet, unless the patient's surrender is unconditional, and unless he agrees to continue the treatment for a reasonable length of time, the hopes of cure of confirmed

neurasthenia or hysteria are slight. If treatment is begun and given up at the end of a few days or weeks, the patient goes back to his original environment or falls into the hands of quacks, and loses what little he may have gained.

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 brain cells, whose function 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. **Isolation,** in many cases, should be so absolute that the nurse, and perhaps the masseur, are the only persons, except the physician, whom the patient sees. The most effective method (the one advocated by Dr. Mitchell) is obtained away from the patient's home. For this purpose quiet hotels or boarding houses, or private hospitals, or public hospitals in which a quiet room can be obtained, are frequently utilized. The setting aside of a room in the patient's own house, to which none but the physician and his assistants are to be admitted, is sometimes feasible, although it is rarely advisable when strict seclusion is to be enforced, and is altogether less desirable than for the patient to be placed away from familiar sounds and scenes. If complete isolation is necessary, it should be so effected that there is no communication whatsoever with the outside world. Then both family and friends are excluded, and there is naturally total banishment of all questions of business; the patient neither writes nor receives letters; the meals are brought to the door of the room by an attendant and handed in to the nurse. Such absolute imprisonment is only occasionally desirable and is rarely necessary for a long time. The physician must decide by the character of the patient and by the gravity of the symptoms when to institute it and when to stop it. He must also use his judgment

as to the kind of persons he permits the patient to receive, taking care to admit last such relatives or friends as are conspicuous by lack of tact or by emotional tendencies.

The one constant companion of the patient during the weeks when 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 the management of 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 indicated to her by the physician when the treatment begins, but, unless she realizes that the unreasonableness, 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 and an even temper; it is easiest for those nurses who are enduring and unemotional, and who by their cheerful manners and ability to talk or read well, or by similar accomplishments, can make the time pass most pleasantly and quickly for their bedridden companions.

Next to isolation, and together with 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 is not permitted to feed himself, and should not even turn over in bed without the assistance of the nurse. The use of the bedpan and of the portable urinal renders it unnecessary for him to get up to empty the bowels or bladder. Like isolation, this enforced rest generally proves disagreeable to the patient for the first few days. However, he ordinarily becomes soon accustomed to it, and

ceases to complain. When the time arrives 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.

Although the re-establishment of the exhausted nervous system is best secured by means of isolation and prolonged rest from voluntary effort, the fact remains that the circulation is more active and nutrition consequently better when the muscles do some work. To secure muscular activity without making serious demands upon the nervous system, the methods at our disposal are massage and electricity. **Massage** may be given by the nurse if she is a skillful masseuse, and then no outsider need be introduced, and the isolation of the patient remains undisturbed. 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. The most skillful masseurs and masseuses that we have in this country have been educated in this special branch in Sweden, and, in New York at least, confine themselves solely to giving massage. If the passive movement is to be given by some one other than the nurse, it adds, of course, additional expense to the treatment, and diminishes the seclusion of the patient; but the value of systematic and skillfully applied massage is so great that its proper administration more than counterbalances these drawbacks. Systematic rubbing of the whole body, for purposes of general nutrition, must be instituted gradually, as the idea of having the body touched by others is repulsive to many persons, and most patients only become accustomed to it after several *séances*. It is best to delay it until the treatment has lasted several days, and to begin it by merely rubbing an arm for a few minutes. It may then be gradually extended, so that at the end of a

week or ten days the *séances* last an hour, and the arms, legs, abdomen, and back are being systematically exercised every day.

Electricity is the other means by which the patient may obtain exercise. Its effects, although less pronounced, are similar to those of massage. If a good masseur or masseuse can be obtained, there is rarely any necessity for using electricity. When for any reason, however, it is impossible to secure the services of a skilled massage operator, the faradic battery can be successfully substituted. It has the advantage that an intelligent nurse can be easily taught to use it gently and effectively, and that "electrical treatment" often has a beneficial mental effect on the patient. The electrodes should be wet with warm salt water, and as the sponges get very dirty, it adds a touch of neatness to the procedure if they are either frequently changed, or else covered by pieces of white gauze at the time of each application. At first the current is applied in very weak strengths, and to small areas of the body; as the patient becomes used to its action 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 regulation of the **diet** during the rest cure is controlled by two considerations—viz., that it should be so arranged that the patient may receive plenty of nourishment in spite of any gastric irritability that he may have; and, secondly, that the amount of food taken should be excessive and out of proportion to the work which is done (forced feeding). If at the beginning of the treatment dyspeptic symptoms—such as anorexia, nausea, vomiting, and eructations—are present, the alimentation must consist exclusively of milk, in order that the stomach may be brought as soon as possible to a condition in which it can retain large quantities of more substantial food. Under these circumstances the milk is given in very small quantities which are repeated

at short intervals during waking hours. Thus, one or two ounces of milk given every hour, or slightly larger amounts given every two hours, are sufficient for the first few days. Administered in this way, milk can be borne by almost any one, although he may think that it does not agree with him, and although, if given in larger quantities, it might immediately induce an attack of vomiting. In rare instances it may seem necessary to have the milk peptonized. Koumyss, on account of the alcohol it contains, is not to be recommended as an exclusive diet. This method of feeding, combined with the rest in bed, is usually sufficient to quiet the more prominent of the gastric symptoms so that in a very short time the quantities of milk may be increased, or 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 two or three 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 very soon be made to include soft-boiled eggs, custards, and jellies, until finally, by the addition of chops, steaks, chicken, etc., 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 may be 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 remain excessive.

Stimulants should be as far as possible 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 remains in bed, is to be avoided. Later the advisability of its use must be determined by the effect it has on the individual. If it renders him more nervous, and if it is desired to use it in excess, it should be cut off altogether. When, however, its effect is to calm rather than to excite, one or two cigars a day can with safety be allowed.

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. Although the patient is in a certain sense a prisoner, he has undertaken the treatment of his own accord, and he can terminate it when he wishes. It must accordingly be the physician's care to make the period of confinement as little irksome as is consistent with the ultimate object in view. Every possible latitude and privilege consistent with the sick man's welfare he should endeavor to provide. 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 will seriously lose in personal influence, and will postpone the time when such nervous manifestations are to permanently disappear. Gentle firmness on the part of both physician and nurse is essential to the success of this kind of treatment. Objections and complaints are best forestalled and obviated by instituting at once a daily *régime* to which the patient is to conform as regularly as though he were keeping business appointments. By this means he comes to look forward to the hours given up to special things with expectancy if not with pleasure, and the time passes with rea-

sonable rapidity. 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 individual cases, 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, these 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 results from the treatment. As a general rule, the more prominent symptoms begin to yield in a few days or weeks. As a result of the milk diet the dyspeptic symptoms disappear and the patient begins to realize that he has an appetite; the sleep becomes improved 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 increase in the color and the firmness of the skin, and by a gain in weight. There is no absolute rule by which it may be determined just when the patient may get up or begin to see his friends again. As in convalescence from general diseases, this question must be decided by the physician who has learned the capabilities of the person under his care, and who, by gradually permitting him to do more and more, can readily ascertain how rapidly the return to normal living should be.

Valuable as is the rest cure, it is not infallible; and the

question arises, If the patient improves but little or not at all, how long should the treatment continue? To this different answers are given by various authors. 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 very much modified or changed altogether. The prognosis for recovery in such cases is far from bright.

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 its beginning, and can endure a moderate amount of exertion fairly well. He is still somewhat nervous and excitable, however, and in very much the same condition as many neurasthenics who are hardly ill enough to need the rest cure, but who require some special form of treatment for the re-establishment of health.

Carried out in its most perfected form, the rest cure is expensive, as it implies that the patient be away from his work for several weeks or months, that he be constantly under the supervision of a nurse, and frequently visited by a physician. An effort is being made, however, to make it more accessible to persons who are in moderate circumstances. In the Philadelphia Hospital beds are set aside for this treatment, and some few of the hospitals connected with the railways have recognized its value, and undertake to treat patients in accordance with its principles.

In persons who no longer require the complete rest cure, and in those who were never ill enough make such a procedure necessary, rest, more than is necessary for healthy individuals, is still a primary requisite. This may be secured, if the patients have no employment, by not rising until after breakfast, and by lying down for thirty minutes or an hour two or three times during the course of the day. If it is necessary that the patient return to his employment—and

in many cases such a course is extremely desirable—rest may sometimes be obtained during working hours; or, if not, the patient should go to bed earlier than usual, so that he passes at least ten hours in bed. The whole mode of life of a neurasthenic should be moderate and equable. He should shun as far as possible all that may fatigue or excite. What has been said in regard to litigation in its relations to the rest cure may be repeated with almost equal force for the patient who does not need to be isolated or put to bed. In the first case it is a bar to recovery from a serious condition; in the second it exposes the patient, who under proper conditions might soon be well, to the danger of becoming an irritable and hopeless invalid.

There are a variety of procedures besides the rest cure which are efficacious in the amelioration and cure of neurasthenic and hysterical conditions. Some of them are applicable during the rest cure itself, and some are only resorted to as a termination of that form of treatment or in cases which are not sufficiently severe to demand seclusion and forced rest. For purposes of convenience they will all be considered together.

HYDRIATRICS.—Second only in value to the rest cure in the treatment of neurasthenia and hysteria is the scientific application of water. Taken internally, by causing an increased activity in the kidneys and skin, and by diluting the toxic metabolic products which are circulating in the blood, it exerts a most beneficent influence upon nutrition. It may be administered with advantage in large quantities during the whole course of neurasthenia or hysteria, with the exception of that period of the rest cure during which the patient is on a fluid diet. A not unpleasant method is a glass of very hot water, taken one hour before each meal. It is the external application of water, however, which is attended with the most brilliant therapeutic results in functional nervous diseases.

Thirty years ago the water treatment, whose ignorant exponents were called "hydropaths," had won a well-deserved reputation for quackery. To-day, largely through the researches of Winternitz, of Vienna, and of his pupil, Simon Baruch, of New York, it has taken its place among the most efficient of therapeutic agents. Its importance is universally admitted in Europe, where hydrotherapeutic establishments are numerous. Among the most prominent of these may be mentioned: In Austria, Dr. Winternitz's, at Kaltenleutgeben, near Vienna; the Centralbad, in Vienna; Dr. Koehler's, in Eisenbach; Dr. Pospischl's, in Krems; in Germany, at Elgersberg and Königstein; in France, at Divonne and Lamalou; in England, at Malvern.

In America, although the recognition of the usefulness of hydriatrics was less prompt than on the other side of the ocean, its value is now becoming rapidly appreciated. Some of the sanitaria in this country are prepared to administer many of the procedures of the water treatment. Prominent among these institutions are the ones at Dansville, Clifton Springs, and Watkins, in the State of New York, and at Battle Creek, Mich. The Hydriatric Institute in New York city, and two institutions at Louisville, Ky., are fitted out with complete apparatus for all hydriatric procedures, the various douches being the chief applications made in them.

The institutions at Louisville, the Hydriatric Institute and the Riverside Public Baths in New York, give treatment to ambulant patients who may return to their homes after each treatment.

The beneficial effects of wisely chosen hydrotherapeutic measures are especially evident in the functional nervous diseases. Since the opening of the hydriatric department of the Riverside Public Baths in New York city, most of the Vanderbilt Clinic cases of neurasthenia and hysteria are referred to that institution for cold douches. It is only jus-

tice to state that our success in the treatment of these conditions is now much more satisfactory than it previously had been. The improvement in nervous symptoms which often follows a sojourn in a sanitarium is largely due to the water treatment received there. Some patients with neurasthenia and hysteria, in whom but little benefit was derived from the rest cure, have returned, apparently well in every way, after a few months' stay at a sanitarium.

The success of hydrotherapy in any case depends in large part upon whether the proper procedure is chosen for the individual condition it is meant to benefit, and upon whether it is properly carried out. The methods which have proved most successful in the treatment of the functional nervous diseases may be briefly outlined here, although for a thorough comprehension of this important subject the reader must be referred to special treatises.*

The **sponge bath,** which is given daily during the period of the rest cure when the patient is in bed, is chiefly for purposes of cleanliness. Different segments of the body are sponged off with water at 70° F. and then dried. Each segment is dried and wrapped up before another segment is bathed.

The **drip sheet** is to be used when the patient, after the rest cure, is beginning to walk around the room. The best time for its application is upon rising in the morning. It has the advantage that, as it requires no special apparatus, it may be given perfectly well 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 seventy degrees, 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

* See Baruch on Hydrotherapy, in Hare's System of Therapeutics, Philadelphia, 1891.

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, he is dried off quickly with a towel and returned to bed. In succeeding applications the temperature of the water may be reduced a few degrees at a time until it finally reaches fifty degrees.

The Wet Pack.—The full 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 seventy degrees—which may be daily reduced one degree in temperature until sixty degrees is reached—and then rolled in a blanket, in which he is left for an hour or longer. This procedure is of especial service when insomnia is pronounced or when the nervous symptoms are in large part dependent upon alcoholism or upon inadequate elimination by the kidneys, and in erethetic conditions. More frequently the desired results may be obtained by the local wet pack, which is one of the most efficient agents for allaying mental excitement and inducing sleep. It is also often useful in the treatment of chronic constipation. It is applied by placing a piece of sheet folded two or three times, which has been thoroughly wrung out in water at sixty to seventy degrees, 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, so that it holds the compress in position. The abdominal compress is usually applied just before going to bed, and should be left for half an hour. If at the expiration of this time no effects from its use are apparent, the sheet may be wrung out in cold water and again applied.

The Foot Bath.—In hydriatric institutions the water for the foot tub enters and leaves in a constant and rapid stream, so that there is the stimulus of mechanical irritation as well as that of heat or cold. In private houses it is usually necessary to be content with an ordinary foot tub filled with water at the desired temperature. The patient sits with the feet in the water, which comes above the ankles. Either hot or cold water may be used, the effects of each being similar. In the cold foot bath the water is at a temperature of forty degrees, and the patient rubs the feet energetically one against the other for about five minutes. By the friction the feet are kept comfortably warm. While in the water, and when they are taken out, their capillaries undergo marked dilatation, as is shown by the rosy color of the skin and the sensations of warmth in the lower extremities. The hot foot bath is on the whole less efficacious, although often very useful. In it the water is at a temperature of one hundred and ten degrees, and the feet are immersed for five or eight minutes.

Both the cold and hot foot bath are efficient sleep-inducing agents and are valuable aids for the relief of mental irritability and excitement, and of the disagreeable subjective cerebral sensations.

The Douche.—The douche excels all other hydriatric measures in the constitutional treatment of neurasthenia and hysteria. Although it can not be applied during the rest cure, it is a seasonable post-graduate course to that procedure, and in many cases of functional nervous disease of moderate severity, when for any reason the rest cure is not undertaken, the patients recover while taking cold douches, without their ordinary mode of life being seriously interfered with. For the successful operation of this form of treatment two conditions must be scrupulously fulfilled. These relate to temperature and pressure. The temperature of the water must be accurately regulated by means of

the thermometer, in order that the patient may not receive too great a shock from the water being too cold, or that he may not fail to react quickly, 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 proper temperature, which comes to the patient with only a few pounds of pressure behind it, is followed by a prompt reaction in robust persons only; in an anæmic and exhausted individual 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 consequently not obtained. As these conditions are difficult of fulfillment in private houses, it is desirable for the patient to receive this treatment in institutions in which the requirements as to pressure and temperature can be obtained and the physician's directions can be carried out by skilled attendants. The treatment should be taken every day, or at least every other day. The ordinary method in hydriatric establishments is for the patient to be placed for two to three minutes in a chamber filled with hot air (130° to 150°). On emerging he immediately gets under a rain bath of sixty to seventy degrees of temperature and of thirty to seventy pounds of pressure. The degree of temperature and the amount of pressure will vary according to the amount of stimulation necessary for the individual case, The colder the water, and the greater the force with which it strikes the body, the more pronounced is the effect. The water is turned off at the end of one or at most two minutes; the patient is then quickly dried by towels and by rubbing. It is best to send him at once into the open air, unless he be very feeble, in order to take advantage of the deepened inspirations which follow the douche. As the general or rain douche is often too severe at the beginning of treatment, a way may be paved for it by using for a week

or two the spinal fan douche, in which the back of the patient is played upon by water coming from a hose fitted with a quarter-inch nozzle, the stream being scattered into the shape of a fan by placing a finger upon the nozzle. The requirements as to temperature, pressure, and duration of the rain douche and the spinal douche are the same. Although the spinal douche is local in application, its effects are general. When applied to other circumscribed areas, this form of treatment may be followed by improvement in local symptoms. It often acts well in the isolated physical manifestations of hysteria. Under the local cold douche of twenty to thirty-five pounds pressure the anæsthesia, paralysis, or contracture, when of functional origin, often disappears. Local and general douches may be alternated.

It is so frequently impossible for patients needing water treatment to have access to properly equipped hydriatric institutions, that it is often necessary for them to get along with such facilities as the ordinary dwelling house supplies. This is only feasible, however, when the nervous disease has not reached a high degree of development. When the patient is very anæmic or very much exhausted he is liable to receive more harm than good from measures which can not be accurately regulated and whose effects can not be carefully observed. The cold sheet, the foot bath, and packs are, of course, given as well at home as anywhere, provided the nurse is intelligent and skillful. The douche, however, is rarely practicable in private dwellings, as neither its temperature nor its force can be properly controlled. Still, when the patient is reasonably robust, certain substitutes for it are possible. These are the ordinary cold shower bath, provided it is delivered with considerable force; or the full cold bath, which may begin with the water at eighty degrees, a temperature which is gradually lowered; or, if the patients react well, the alternate sponging of the nape of the neck and spine with hot (110°) and cold (65°) water.

The local douches can not be satisfactorily carried out at home.

HYPNOTISM.—If hypnotism is understood to include every variety of suggestion by which the mind can be influenced, it is a procedure which will never be eliminated from the practice of medicine. The effect of mental prompting is evident in all social intercourse, and the peculiar relations existing between physician and patient necessarily render it an agent of great power. A medical man is supposed to be acquainted with the solutions of all the problems of physiology, and the patient is as ready to believe what the physician says as he is to trust to his own sensations. On the one hand is a person whose judgment, will, and self-control have been weakened by illness; on the other is a man with unimpaired faculties, conversant with human nature, and presumably conversant with the character of the symptoms of which the sufferer complains. Nothing could be more natural under such circumstances than that the mental actions of the patient be to a certain extent directed and controlled by those of the more vigorous personality. In general diseases, in which the brain is apparently unaffected, the influence of mind upon body is too far reaching to permit of neglect; and in the functional disorders of the nervous system, whose symptoms are in large part mental, the patient is so susceptible to the appeals to the emotions and to the suggestions or counter-suggestions of certain trains of thought, that the physician who neglects these methods can hardly hope to be successful in his treatment. Contrasted with this form of suggestion, which every physician uses almost unconsciously, is that which is made during the condition of induced sleep.

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.

As a therapeutic measure it has been generally abandoned by the school of the Salpêtrière. It is still used in Germany, but its special European exponents are Bernheim, of Nancy; Forel, of Zürich; and Wetterstrand, of Stockholm. In America it has never attained popularity among the members of the medical profession, partly because few American physicians have had the opportunity of systematically studying its *modus operandi* and its effects, and partly because in this country it has never been freed from the reputation of quackery. Whatever may be its efficacy among the ignorant peasantry of Europe, in this country, whose people are matter-of-fact and comparatively free from superstition, the therapeutic uses of hypnotism are very restricted. It has no part in the treatment of neurasthenia, and in hysteria it can only cause a disappearance of the individual manifestations, without benefiting the fundamental conditions from which the morbid mental states arise. Even did we not accept the theory of Charcot, that hypnotism is only possible in the victims of hysteria, the ease with which hysterical persons, who have once been hypnotized by an operator, can again be brought under this influence, and the readiness with which auto-hypnosis comes to take an important place among hysterical manifestations, are sufficient evidences that in *la grande névrose* the reaction to suggesting influences occurs more readily and is accompanied by more permanent effects than in normal individuals. When the physician succeeds by means of hypnosis in causing a disappearance of any of the physical manifestations of hysteria, he does so by bringing about an alternation 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 alternations of personality and upon subconscious mental states, it would seem to be an unavoidable conclusion that by contributing additional phases to the underlying morbid psychical conditions the whole disorder would be rendered worse, even though the temporary physical manifestations are made to disappear. Consequently hypnotism is to be resorted to only when other therapeutic means have proved useless. When the patients have failed to receive benefit from the rest cure, or from hydriatric measures, or from other forms of treatment, hypnotic suggestion is not only permissible, but remains as the one therapeutic measure from which benefit may be hoped for. However, as Gilles de la Tourette says, "Hypnotism is a potent modifier of hysterical strata and may be dangerous even in skilled hands. It should only be used when the dangers incurred are less serious than the symptoms which it is intended to cure."

DRUGS.—The use of drugs in the treatment of the neuroses is subsidiary to the application of some or all of the procedures which have been described. The patients are, however, more contented if they are taking some medicine, and there are a few drugs which unquestionably render material assistance in the treatment of these conditions. To combat anæmia and for purposes of general nutrition, some form of iron or arsenic is generally useful. When the gastric irritability is pronounced, these tonics may be administered in the form of some of the natural waters, such as Levico, which contain proportionately large amounts of these minerals. The most popular nerve tonic is strychnine. It may be given by mouth in the form of the sulphate, gr. $\frac{1}{60}$ to $\frac{1}{30}$, three times a day after meals, or hypodermatically, as the nitrate, gr. $\frac{1}{40}$. Strychnine is to be especially

recommended when the clinical manifestations indicate depression, rather than irritability, of the nerve centers. A new drug, which in many cases yields very satisfactory results, is the glycero-phosphate of soda. It may be prescribed in three- or five-grain capsules (Chapoteau), or according to the following formula:

℞ Glycero-phosphate of soda (Schering).. ʒ vj;
Distilled water..... ℥ viij.

M. Sig.: One teaspoonful in hot water, three times a day, half an hour after meals.

The bromides may do a great deal of harm in neurasthenia, and their indiscriminate use is to be deprecated. In small doses they are often efficacious in allaying extreme nervous excitability, especially when exhibited in conjunction with ergot. They may also be recommended for the relief of dizziness and headache. The following prescription has proved useful in the hands of the writer:

℞ Sodii bromid..... ʒ vj;
Ext. ergot. fl..... ℥ j;
Aq. destil.....q. s. ad ℥ iv.

M. Sig.: One teaspoonful in hot water half an hour after meals.

Insomnia can usually be allayed by some of the hydrotherapeutic measures. When they fail to induce sleep, recourse may be had to small doses of the bromides, or sulphonal, or trional, of which the latter is the most efficient and least harmful. Morphine has no place in the treatment of the traumatic neuroses.

For constipation much can be done by massage, although drugs are often necessary. A teaspoonful of magnesium sulphate in a full glass of cold water, taken on rising in the morning, is in many cases sufficient to insure one or two movements daily. If constipation is rebellious, more active measures are necessary. When possible, the natural aperient waters, such as Hunyadi or Francis Joseph, are pref-

erable to other laxatives. The fluid extract of cascara, gtt. x to xxx once or twice a day, or at bedtime, gtt. xlv, is often serviceable. When these measures fail, recourse must be had to the more energetic laxatives or purgatives, which need not be enumerated. If they are given every night, it is advisable to have a variety, so that, by taking a different one each night in the week, the patient does not come to rely too much on any one of them.

Whatever drugs are used, they should be given for definite purposes, and should be taken according to implicit directions. Neurasthenic patients are especially fond of dosing themselves on the appearance of every new symptom, and of accepting as genuine, and as applicable to their cases, the cures effected under this or that specific as recorded in the advertising columns of the newspapers. To combat this tendency the patient should be told the nature and the dose of the drugs he is to take, and when he is to take them; and he should be particularly cautioned against taking anything in the way of medicines without the physician's orders.

Such are the most important of the measures at our disposal for the treatment of the traumatic neuroses. Varying conditions may occasionally demand additional methods. If the suffering from lumbago is intense, it may be relieved by the application of a plaster jacket. It is rarely necessary to continue the immobilization for more than a week or ten days. At its termination the muscles of the back should be actively masséd. The cautery is often used in this condition, since it alleviates the pain, but it renders the back sensitive and postpones the possibility of massage. Except in so far as it supplies exercise for the muscles, faradism is of but little service. The indefinite pains are best allayed by rest and by the general tonic treatment. If true neuralgia exists, it may be improved by hot fomentations, by some of the analgesics, by the application of galvanism,

or, better, by ten-minute daily *séances* with the sinusoidal current. Also the vibrator, as devised by Peterson, is useful in some cases.

Passive movements are the most rational means for causing the disappearance of hysterical paralyses and contracture. By causing movement in limbs which are apparently paralyzed, the patient sees that they are movable, and gradually comes, when sufficiently encouraged, to move them himself. In hysterical paralysis of the leg the power of walking may often be speedily restored by having the patient push a chair in front of him, or by means of a "roller machine." By distracting the patient's attention, motion can usually be obtained in hysterical contractures, and gentle and persistent passive movements are the most efficacious means of treating this condition. By massage, also, sensibility may be made to return to skin which was the seat of hysterical anæsthesia. These means, the beneficial results of which are often permanent, are much to be preferred to those, such as the faradic brush or the horseshoe magnet, which depend for their action upon fright, or upon utilizing the credulity of the patient. Some general exercise should be insisted upon as soon as the patient is able to undertake it. During the rest cure, as soon as complete rest is abandoned, the patient should be made to perform light exercises, such as opening and closing the hands, or flexing and extending the elbows and wrists, as a part of the daily schedule. Similarly, he should make use of the legs, practising getting up on and down from a chair, and walking prescribed distances around the room at regular times. For the rest-cure patient, as he gets better, and for all neurasthenic and hysterical patients who do not go to bed, the exercise may include light gymnastics and short walks or drives, but may be gradually extended until it includes some of the moderate athletic sports for which the facilities are now becoming so generally accessible.

Change of climate, involving as it does diversion of thought and modification of home habits, is almost always beneficial. As a general rule, the seashore is particularly adapted for the irritable forms of disease, while patients who are depressed and apathetic do best in high altitudes, such as may be found in Colorado or in the elevated regions of southern California.

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